THE NEED FOR CONTROLLING ASBESTOS IN THE INDUSTRIAL ENVIRONMENT AND THE PROBLEM OF STANDARDIZING THIS CONTROL

October 22, 1971

Occupational Health 211

by

Craig Barrow
Donald Davis
Joel Gaydos
Daniel Geary
Likhit Kongsiri
Carl Nim

Graduate School of Public Health
University of Pittsburgh

THE NEED FOR CONTROLLING ASBESTOS IN THE INDUSTRIAL ENVIRONMENT AND THE PROBLEM OF STANDARDIZING THIS CONTROL

Asbestos refers to a group of minerals which possess the common property of being fibrous silicates and forming flexible fibro-crystals which are refractory to heat. There are many asbestos minerals but only six are of commercial importance: chrysotile, amosite, crocidolite, actinolite, anthophyllite, and tremolite. Of these, chrysotile accounts for 95 percent of the world's production. Industrial use of asbestos varies because of differences in surface properties, metallic elemental content, tensile strength, fiber diameters, and flexibility. It would seem reasonable to assume that these same differences could also affect respirability, deposition in the body, retention, translocation, and biologic reactivity. In fact, in vitro hemolysis by chrysotile and the absence of marked hemolytic properties in the other asbestos types have been demonstrated (Schnitzer, et al, 1970).

Asbestos has been in use for more than 2,000 years. The Romans mined it in the Italian Alps and the Ural Mountains, and Pliny wrote of weaving asbestos in 50 A.D. The term asbestos is probably derived from a Greek adjective meaning unextinguishable or unquenchable (Brodeur, 1968). The idea of an unextinguishable or unquenchable flame is related to the use of asbestos as lamp wicks by the Vestal Virgins in 70 A.D. (Cooke, 1927). Despite the early observations of the curious nature of this substance, the asbestos mining industry did not actually begin until the late 19th century. However, since its inception, the industry has grown at an astounding rate, the

production for 1970 (approximately 5 million tons mined) equalling that for the period 1879 to 1926 (Panel on Asbestos, 1971). This industrial boom is a reflection of the fact that an almost unlimited number of uses have been found for asbestos, ranging from marine paints and linings for chemical vats to insulation products, floor tile, and textiles (Panel on Asbestos, 1971).

Industrial exposure to asbestos occurs not only in the open and deep mining operations and processing, but also in the application of asbestos products and in the demolition and repair of structures containing asbestos materials.

The first published report of the possible adverse effects of asbestos appeared in the "Charing Cross Gazette" in 1900. Dr. Montague Murray, a British physician, described the case of a 33-year-old man who had worked in an asbestos carding room for 10 years prior to his death. This man was the sole survivor of 10 men who had started to work at the same time and examination of his lungs at post mortem showed "pulmonary fibrosis" and "spicules of asbestos." (Cooke, 1927.) A preliminary report of a second case appeared in the British Medical Journal in 1924. The complete report appeared in 1927. This second case was a 33-year-old female who began working in an asbestos factory at age 13. She was forced to quit at age 31 because of a severe cough and deteriorating physical condition. Autopsy, following death at age 33, showed pulmonary fibrosis and the presence of "curious bodies" suspected to be iron containing asbestos fibers in the lungs. (Cooke, 1927. McDonald, 1927.)

Clinical pulmonary asbestosis was seen by physicians prior to 1927 but autopsies were not obtained, and it is presumed that many asbestosis deaths were attributed to tuberculosis. (Oliver, 1927.)

Exposure to asbestos is evidenced by the appearance of ferritin coated fibers in the lungs. These "ferriginous bodies" (also called asbestos bodies and asbestosis bodies) have been demonstrated in the lungs of many city dwellers not occupationally related to asbestos. However, if the dose of fibers inhaled is overwhelming, severe pathological effects occur in the lungs and the resulting disease is asbestosis, one of the most serious of the industrial pneumoconiosis. (Panel on Asbestos, 1971.) Asbestosis (also called asbestotic pneumoconiosis) is characterized by X-ray changes consistent with diffuse interstitial fibrosis and calcified or non-calcified pleural plaques. (Panel on Asbestos, 1971.) The individual with asbestosis will suffer from shortness of breath and will exhibit physiological characteristics consistent with a restrictive pulmonary disease. (Panel on Asbestos, 1971.)

If the association between asbestos and disease stopped with asbestosis, the situation would have been serious enough. However, in 1935, Drs. K. M. Lynch and W. S. Smith of the Medical College of the State of South Carolina, reported the case of a 57 year old man with a 43 year history of exposure to dust, the last 21 years being in an asbestos factory. Post mortem examination revealed not only the "asbestosis bodies" in association with phagocytes, but also the presence of epidermoid bronchial carcinoma. (Lynch and Smith, 1935).

Unfortunately, this association has withstood the test of time. Selikoff, Hammond, and Chung conclude that asbestos workers who smoke have approximately 92 times the risk of dying from bronchogenic carcinoma as men who neither smoke cigarettes nor work with asbestos. (Selikoff, et al, 1968.)

As fate would have it, the association between asbestos and fatal disease continued. In 1960, Wagner, Sleggs, and Marchand published the complete report of 33 cases of diffuse pleural mesothelioma--all but one case had probable exposure to crocidolite. (Wagner, et al, 1960.) Crocidolite has since also been associated with mesothelioma of the peritoneum. (Panel on Asbestos, 1971.) Wagner's group suggests a possible incubation period of 20 to 40 years. (Wagner, et al, 1960.) McDonald and his co-workers reported no great relationship between chrysotile and malignant mesothelioma (McDonald, et al, 1970), and it may be that crocidolite is the only asbestos related to this disease[1/]. (Panel on Asbestos, 1971.) McEwen, et al believe there is a genuine increase in mesothelioma in Scotland despite the fact the diagnosis has become more acceptable in recent years. (McEwen, et al, 1970.) Since only two of McEwen's 80 cases showed evidence of asbestosis the exposure required for this tumor may be quite small.

The association between asbestos and asbestosis was made about 1900, the relationship of asbestos to bronchogenic carcinoma occurred in 1935, and 1960 may be called the asbestos-mesothelioma year.

If this continues, then we can expect the announcement of a rather unfortunate asbestos-related disease in 1975. What could it be? Some of Selikoff's work has already alluded to increased carcinoma of the stomach, colon, and rectum in asbestos workers. (Selikoff, et al, 1968.) Or perhaps some new element will achieve industrial prominence and asbestos will be the vehicle for transporting it to a site in the lungs or gastrointestinal tract where it may exert considerable damage. The idea that asbestos itself may not be the agent of disease but may merely carry the culprit into the body is not unique. Work has already begun in the area of metals associated with asbestos fibers. (Cralley, et al, 1968.)

This discussion leads to one important fact--the need for control of asbestos in the environment. In 1968, Selikoff's group reported that 15 of 94 deaths in asbestos workers were due to asbestosis. (Selikoff, et al, 1968.) Considering that these men often worked outdoors and used materials containing only 5 to 15 percent asbestos, one wonders how much our present day standards for control are actually accomplishing. With regard to mesothelioma, the possibility of no safe exposure may be the real situation. Another area that must be considered is the obvious differences in the six types of asbestos. Perhaps each of the six types requires a different set of standards for control. Do we really know enough about asbestos to intelligently control it? In 1967, Dr. Clark Cooper posed several questions concerning asbestos. (Cooper, 1967.) Four years later they are still significant questions: Are all forms of asbestos equally hazardous

with regard to pneumoconiosis? Are all types of asbestos equally implicated in the association of asbestos with bronchogenic carcinoma? Could mixtures of asbestos, especially those containing amosite be related to mesothelioma? Thus, we will proceed with a discussion of man's attempt to determine a safe level for asbestos in the environment.

First of all, we would like to take a look at the various counting and sampling methods that have been utilized in the determination of asbestos concentration in the work environment. This is of significant importance because the sampling technique has a direct bearing on the establishing of a threshold limit value. This point will be further clarified in the discussion of Great Britain's and the United States' standards for asbestos exposure.

In the U.S. the impinger method has been adopted as a standard by those concerned with prevention of asbestosis. In the epidemiologic studies which correlated dust exposure with asbestos it was found that the incidence and severity of asbestosis increased with increasing dust exposure. Using the impinger to evaluate effectiveness of environmental controls, it was noted by the 1960's that asbestosis had decreased in incidence since the 1940's, but was still occurring at a significant rate.

In Great Britain a customary procedure in monitoring asbestos processing plants has been to count only the fiber content collected in air samples. The most common air sampling method at the present time is membrane filtration. Fiber concentrations determined by these methods have not been compared to criteria developed in the United States,

because these fiber-counting methods do not yield results directly comparable to those obtained by the impinger sampling and counting method. This section is concerned with the relationship of dust counts by impinger sampling to fiber counts from membrane filters.

The ratios are presented as a rough indication of the relationship of impinger counts to fiber counts reported from abroad and to give some indication of the relationship for fiber counts from personal samplers taken from the worker using membrane filters. (Ayer, Lynch, 1965.)

Chrysotile, crocidolite, and amosite are widely different in chemical properties and there is a distinction geologically and crystallographically between chrysotile, a serpentine type of sheet silicate, and crocidolite and amosite, which are chain-structure silicates of the monoclinic amphibole type. However, there is a common factor that all occur in long crystals which are so fine as to be fibrous in structure. The individual chrysotile crystal, by electron microscope observations, is believed to be from 0.02 to 0.04 microns in diameter. These observations and also X-ray studies have led to the conclusion that chrysotile is based on a sheet-like structure and the sheet is folded into a hollow tubular form. By contrast, although less work has been done on crocidolite and amosite, they are probably based on a chain silicate structure and are rod-like crystals. (Addingly, 1966-67.)

Counting and sizing under the microscope is a difficult problem. At one time an attempt was made to count everything visible under an oil-immersion objective. More recently, an entirely different basis has been adopted; specifically, a count being made of particles between 5 to 100 microns in maximum dimension. The main reason was it greatly

simplifies counting and minimizes error between different observers. Secondly, it was based on rather limited data, that most processes in asbestos textile factories produced dust which had a similar size range. More recent measurements have shown this to be not true.

The third reason is that asbestosis is mainly associated with the break-up of asbestos bodies containing fibers of 10 microns and upward in the lungs.

The impinger method was developed for the evaluation of atmospheres in silicosis-producing occupations. The impinger is a very efficient collector of quartz and particles of a similar density, 3/4-micron diameter and larger. The associated counting method uses a microscope with a 10 X objective to examine particles which have settled in a shallow cell (0.1 or 1-mm depth) and capable of detecting quartz particles to this same minimum size. The impinger method is the one described by PHS Bulletin 217 and subsequently by the American Conference of Governmental Industrial Hygienists, the American Public Health Association, and the Asbestos Textile Institute. In general, either water or alcohol may be used as a sampling medium. When the method is applied to air sampling in asbestos textile plants, few asbestos fibers are seen in the samples, a fact which has undoubtedly been observed by all those who have used the method. Even when all particles with a length-to-width ratio of 3 or greater are counted as fibers, the fiber count is usually less than 10 percent of the total impinger dust count. Electron micrographs of samples collected by other methods reveal that most airborne asbestos fibers are very small in diameter (hundredths to tenths of a micron) even when they are many microns in length.

Fibers of such small diameter may not be collected by the impinger; if collected many of them would not settle to the bottom of the counting cell in the allotted time, and those that did settle would usually not be visible by the microscopic technique employed. (Ayer, Lynch, 1965.)

For the environmental study of the asbestos textile industry, it was desired to have another routine sampling and counting method for asbestos fibers. A medium for dust sampling that has come into wide use in recent years is the membrane filter. Membrane filters will collect particulates as small as 0.1 μ in diameter with practically 100 percent efficiency. The particles are collected on the surface and the filter may be examined directly with incident light, rendered transparent and examined by high resolution transmitted light microscopy, or the particles may be transferred to an electron microscope grid and examined by electron miscroscopy.

The minimum fiber which is routinely counted is believed to have a diameter of one-half μ.

A point which should be mentioned is that any comparisons between impinger dust counts and fiber concentrations as determined by some other method can only reflect present conditions. Although the manufacturing processes and equipment used now are essentially the same as those used in the 1930's, the quality of the fiber is said to have improved greatly in the past 10 years--the proportion of the asbestos received at the textile plant which is "dust" is much smaller than previously. This undoubtedly would have some effect on the ratio of fibers to nonfibrous particulates, but the degree of effect cannot be readily determined. (Ayer and Lynch, 1965.)

C. G. Addingley made a comparison between the midget impinger and the membrane filter, counting only particles or fibers of 5 microns and upwards in maximum dimensions. The membrane gave a count of 78 percent higher than the midget impinger. This is in the same ratio as for particles 1 micron and over.

In another experiment, a membrane filter was put in the exit line from a midget impinger and particles 1 micron and above were counted. It was found that for every 87 particles counted on the impinger record, 13 particles passed through and were collected on the membrane filter.

Addingley also found that for fibers greater than 5 microns, it gives a count as high or higher than any other method. The results are consistent for particles of 1 micron and upwards. It almost doubles the counts from impinger methods. (Addingley, 1966-1967.)

The U.S. Public Health Service collects samples on airborne mineral dusts by the membrane method. This method is suitable for experimental studies of most mineral dusts. (Edward, Lynch, 1968:), and at present it has been adopted by the American Conference of Governmental Industrial Hygienists as the recommended technique for counting the asbestos fiber concentration in the work environment. (American Conference of Governmental Industrial Hygienists, 1971.)

From the work done by Ayer and Lynch, and Addingley, they concluded that the membrane filter is much more efficient than the impinger. But to compare earlier work done by the United States and Great Britain, a relationship was found between the membrane filter (counts are given as fibers per cc) and the impinger (counts are given as mmppcf).

At the present time the overall ratio appears to be 1 mppcf by impinger to 10 fibers per ml, 6 fibers per ml longer than 5 microns, or 3 fibers per ml longer than 10 microns when the membrane filter samples are counted with phase contrast illumination on the transparental filter using a 4-mm (43 X) objective.

Size selective sampling using horizontal elutriators ahead of the sampling device indicated that the major portion of the fibers cojnted would reach the lung if inspired. (Ayer and Lynch, 1965.)

The following is a brief resume of other sampling devices and their shortcomings:

Owens Jet Counter: This im of value taking snap samples. Its efficiency falls off with increasing size of particle and also with increasing amounts of air sampled, since the high air velocity on the second and subsequent strokes of the pump tends to disturb the sample deposited during the first or previous strokes.

Konimeter: This also is designed to take snap samples. It suffers from defects and disadvantages similar to those of the Owens instrument. Records taken on a glycerine or vaseline coated glass slip cannot, of course, be ignited to remove smoke particles.

Thermal Precipitator: This a generally approved method in England and Europe for estimation of airborne dusts. It has been used extensively in asbestos textile factories. Recent results suggest that its efficiency falls off for long asbestos particles.

Gravimetric Method: Addingley does not favor this method. Roach (1965) reports favorably. Ayer and Lynch (1965) discuss gravimetric methods and suggest they provide "poor to fair" index of exposure. This method has a number of limitations. It gives no indication of particle of fiber size distribution.

Tyndallometric Method: It operates on a light-scattering principle, using a very small chamber of 0.002 cc in volume, so that one particle at a time passing through generates a single pulse in the photoelectric detector. This cell feeds an amplifier with a pulse height discriminating and digital counting system.

Comparisons were carried out with membrane filter counts. With the Royco counter set to count particles of 5 microns and upward, it was found to give a count 25 percent lower than the membrane filter. (Addingley, 1966-67.)

At this point,we shall examine the British criteria for their asbestos standard. The purpose of this approach is more than just a mere examination of Great Britain's threshold limit value for asbestos. On the contrary, the United States' work is closely associated to work done in Great Britain. Thus, before taking an in-depth look at the TLV for asbestos in America, it is important to examine the standard in Great Britain.

British TLV'S: Chrysotile was used in the commercial manufacture of asbestos products in England in 1880. Asbestos products containing crocidolite and amosite were first produced in 1895 and 1907, respectively. (Newhouse, 1969.)

In 1929, Merewether completed a 1-year study of 363 workers employed in asbestos textile factories. Twenty-five percent of those workers were suffering from pulmonary fibrosis. He suggested a connection between the inhalation of asbestos dust and the incidence of asbestosis. The results of this study initiated legislation in the form of the Asbestos Industry Regulations of 1931.

The Regulations of 1931 were intended to reduce the asbestos textile worker's exposure to asbestos dust in England's factories. Control measures recommended in the legislation were of a qualitative rather than a quantitative nature. Attention was focused on making particularly dusty factory areas less dusty. The regulations applied only to particularly dusty areas in the asbestos textile factory. Employers were required to provide ventilation in the specified areas and to take

whatever steps necessary to reduce the concentration of airborne dust. Machinery that generated heavy dust clouds in its operation had to be enclosed. Employees who worked in dusty areas were to receive periodic medical examinations. The results of these examinations were recorded and filed. By making asbestosis a compensable disease, additional records became available for further study.

As a result of the required medical examination records and claims for compensation, a means for further assessing the hazard of asbestos exposure had been established. The records began to show a drop in the death rate due to asbestosis. This decrease was primarily due both to the increasing use of antibiotics and the control measures initiated by the Asbestos Industry Regulations of 1931. Pulmonary infections were frequent complications that arose with asbestosis. The use of antibiotics reduced deaths due to these infections. At the same time, the Asbestos Regulations were providing a considerable amount of protection for the asbestos worker (Ellison, 1970).

Suggestions that lung cancer might be associated with asbestosis appeared in England's literature in 1934 (Stumphins and Myer). Lynch and Smith suggested an association between lung cancer and asbestosis in 1935. Merewether (1949) reported a 13.2 percent incidence of lung cancer among 235 individuals found to have asbestosis at autopsy. The data for his study were available primarily through the provisions of the Asbestos Industry Regulations of 1931. Gloyne (1951) and Doll (1955) also reported incidences of lung cancer in individuals who had died of asbestosis that were much higher than the incidence of lung cancer in

the general population. These reports were too limited in number and scope to indicate a definite association between lung cancer and asbestosis, but did cause concern.

Maximum allowable concentrations for a variety of materials were published by the Factory Inspectorate of Great Britain in 1960. Asbestos was included, with a maximum allowable concentration of 5 mppcf (187 particles per cc). This value was taken directly from the Annual List of Threshold Limit Values recommended by the American Conference of Governmental Industrial Hygienists (ACGIH). It was based largely on Dreesen's study of 1938. The limitations of Dreesen's study in setting a hygienic standard for asbestosis had already fallen under severe criticism (Roach, 1970). More than 50 percent of the workers Dreesen studied were under 30 years of age. Other evidence indicated that the amount of time these individuals had been exposed was insufficient for asbestosis to develop. Of the 105 workers he studied, 82 had been employed less than 5 years. Only 4 had received more than 10 years of exposure to less than 5 mppcf of asbestos dust. Furthermore, of less than 600 employees, 150 had been discharged from the factory prior to the study because they were suspected of having asbestosis. All of these limitations of the Dreesen study and the suggested link between asbestos exposure and lung cancer, caused considerable skepticism of the hygiene standard of 5 mppcf for asbestos.

To investigate effects on health and establish a more effective approach to the control of asbestos dust exposure, a subcommittee on asbestos was appointed by the British Occupatioal Hygiene Society in 1965.

This subcommittee was one of the committees subordinate to the newly formed Hygiene Standards Committee.

The Hygiene Standards Subcommittee on Asbestos chose 290 men employed in one of England's asbestos textile factories for investigation. These men worked in an area of the factory in which a prevalence of chronic bronchitis had been found. Because the Asbestos Industry Regulations of 1931 had caused a sharp change both in death rate and prevalence of asbestosis, those individuals employed before or during the transition period were eliminated from the investigation.

The period of time involved in the investigation extended from January 1, 1933 through June 30, 1966. Only men with 10 years or more service since January 1, 1933,were investigated. The number of years of service each of the 290 men had, ranged from 10 to 34 years.

Although the presence of asbestosis was determined with a variety of methods, the members of the subcommittee considered basal rales to be the earliest observable indication of the disease. Asbestosis was observed to be most prevalent among those workers exposed to high concentrations over a long period of time. The workers that were being investigated had been exposed to asbestos dust concentrations up to nearly 30 fibers/cc. Few workers had been exposed more than 30 years. Fibers greater than 5 μ in length with a length to breadth ratio of at least 3 to 1 appeared to be the most hazardous. The subcommittee reasoned that the lungs might be better able to rid themselves of smaller fibers. However, tabulations of total exposure were based on the assumption that the lungs could not eliminate any inhaled asbestos.

The material could then be assumed to accumulate in the lungs. The subcommittee decided this assumption, though not correct, would provide a built-in safety factor and also make calculations more simple. The dust concentrations were measured with a thermal precipitator and membrane filter used in conjunction with an electric pump or Draeger hand pump. Phase contract microscopy was employed for counting collected fibers.

After all the data had been collected and analyzed, a threshold limit value of 100 fiber years per cc, not to be exceeded over a working lifetime, was recommended. By expressing the TLV in terms of fiber years the subcommittee provided a value that was firm in its limits but flexible in its application. The TLV could be interpreted as 2 fibers/cc for a working lifetime of 50 years, 4 fibers/cc for 25 years, and so on. The product of fibers multiplied by years of exposure,however, remained at 100 fiber years/cc. The subcommittee estimated that less than 1 percent of asbestos workers would be affected by exposure to asbestos if the recommended lifetime limit was observed. However, they stressed that even if the limit was observed, some workers might be adversely affected. A balance had to be found between a reasonable amount of control and a tolerable number of health problems. A TLV of zero would present no health hazard, but would cost too much to maintain. Until new evidence proves otherwise, the recommended TLV is considered to be a reasonable balance between costs to health and costs of control. Little information is available concerning the costs of asbestos dust control. The subcommittee suggested more information was needed and should be acquired as soon as possible.

The membrane filter is considered the preferred method for measuring asbestos dust concentrations, particularly so in instances where large amounts of other dust are present in the air with asbestos. However, additional methods of measurement were also provided for in the Regulations of 1969. Less complicated and less expensive methods can adequately provide measurements of dust concentrations that will provide a foundation for a sound control program. The subcommittee, therefore, did not consider equipment to be a deterrent in the establishment of an asbestos dust control program.

The data investigated by the subcommittee involved only chrysotile asbestos dust clouds produced in asbestos textile factories. In other asbestos industries, or where other types of asbestos fibers are present, caution must be exercised in applying the standards recommended by the subcommittee. Many believe the TLV for crocidolite should be one-tenth that recommended for chrysotile (Ellison, 1970). The subcommittee recognized that crocidolite may be a greater health hazard than chrysotile but, due to the paucity of pertinent health data concerning crocidolite, could only recommend that much greater care be taken in estimating risk where crocidolite is involved. The possible association between asbestos and lung cancer was also recognized by the subcommittee. Since very little is knows of this association, the subcommittee could only express their belief that a reduction in the prevalence of asbestosis should have some effect on reducing the prevalence of lung cancer among asbestos workers.

The asbestos-cancer association requires further research before threshold limit values can be recommended that include risks due to cancer.

The 1969 regulations are applicable to any type of industry in which asbestos or asbestos products are used. This is a much more comprehensive coverage than was provided in the 1931 regulations. The employer is responsible for providing protection to his employees. Before he can require the employee to wear protective equipment, the employer must do everything possible in providing ventilation and other dust control measures. Only when such protection cannot be attained through plant engineering, can the employee be asked to wear protective equipment. The subcommittee on asbestos has set higher standards for protective equipment. Better protective equipment is especially needed in those industries in which asbestos products are used.

More rigid worker medical examinations were required. Careful records of occupational history, smoking habits, the presence of nasal obstructions, gross deformities of the chest, bronchial trouble, hypertension, and rheumatoid arthritis were required for each employee. Results of the medical examination could be used as a basis for excluding from employment those individuals considered to be susceptible to the effects of asbestos exposure. According to Davies (1970), most asbestos workers are discouraged from smoking.

The Asbestos Regulations of 1969 were put into effect on May 14, 1970. Provisions were made to review the asbestos problem within 3 years of the date they were proposed (1969).

The American and British threshold limit values for asbestos differ in several respects (Roach, 1970). The British TLV makes distinctions among asbestos types, while the American TLV makes no such distinction.

The period of time over which the concentration must not exceed the TLV is different for the two countries. The American TLV is based on an 8-hour average compared to a 3-month average in England. The difference in the American and British TLV's are primarily due to the weight given risk, control costs, and other considerations in each country. All economic, social, and health aspects must be carefully considered on the basis of existing conditions before a TLV can be recommended (Ellison, 1970).

The regulations specify that an average of no greater than 2 fibers/cm^3 should be maintained over each 3-month period. Since the Factory Inspectorate of England must check these concentrations periodically, attempts are being made to limit the time involved in determining the average concentration. The 3-month standard is intended to take into account day-to-day fluctuations in concentration. If shorter time periods are accepted in determining compliance with the regulations, they will have to allow for these expected fluctuations. Luxon (1970) has suggested a standard of 2 fibers/cm^3 over a 4-hour/time period with a ceiling value (not to be exceeded) of 12 fibers/cc during any 10-minute period. Hickish and Knight (1970) reported that concentrations of asbestos dust in brake system servicing operations they examined were not likely to exceed the 2 fibers/cc average over a 3-month period. However, if those same measurements were averaged over 4-hour time periods, the ceiling value of 12 fibers/cc for any 10-minute period would be exceeded. In another report, Hickish and Knight (1970) presented data obtained when measurements were averaged over 45-minute periods. Even though the areas

in which the measurements were taken were known to have low concentrations of airborne asbestos, the 2 fibers/cc average was sometimes exceeded. They suggest that brake servicing operations such as filing or grinding may greatly exceed the permissible average concentration.

The Asbestos Regulations of 1969 present a comprehensive guideline for the establishment of effective control programs. However, many facets of asbestos health problems require further research. And before reviewing the yet unsolved problems in establishing a meaningful relevant TLV for asbestos, a look at the United States' standard has priority.

This particular section of our survey will review the historical evolution of the TLV standard for asbestos in America. Reasons for changing from one standard to another will be discussed at length, and today's standard will be examined in an effort to discern its validity as a relevant and meaningful threshold limit value.

Work done by Dreesen, et al, in 1938, on 541 employees of three different asbestos textile plants recommended a threshold limit value of 5 mppcf. The three plants in question utilized chrysotile. Dreesen's recommendation was based on the revealing evidence that only three doubtful cases of pneumoconiosis were discovered in those workers exposed to dust concentrations under 5 mppcf. In comparison, numerous well marked cases were found above 5 mppcf.

This TLV for asbestos was in effect until 1969, upon which time the value was revised. As late as the early 1960's, data and new evidence were still sparse as to the biological effects of asbestos in relation to a dose-response curve for the worker. As a result, the standard of 5 mppcf of air for an 8-hour daily exposure over 40 hours per week was

reaffirmed by the American Conference of Governmental Industrial Hygienists in 1964.

However, in the mid-1960's, a new interest in the asbestos standard of 1938 was initiated. New studies, such as that by Selikoff in 1962 and 1963, revealed the striking evidence that asbestosis was still very much a problem for those engaged in its manufacture and use. Also, the increasing incidence of lung cancer and mesotheliomas among asbestos workers was reported. Examining the work history and health records of 632 men who were members of the Heat and Frost Insulators and Asbestos Workers Union of New York on January 1, 1943, Selikoff reported the following in 1964:

Of those men who had been exposed to asbestos and other insulating dusts, there were 255 deaths. This figure is 50 more than the expected number based on comparable U.S. mortality figures. Analysis of causes of death revealed 12 due to asbestosis, 42 deaths attributed to lung cancer, and 4 from mesothelioma, a malignant tumor rarely reported among the general population. (Selikoff, et al; 1964.)

In an updated examination of the men in this same union, Selikoff reported that the 632 original members had the following statistics:

By December 31, 1968, 300 were dead from various causes, including 30 from asbestosis, 72 from lung cancer, and 22 from mesothelioma. (Selikoff, et al; 1969,)

As a result of the increased interest generated from these and other new studies, attention was directed to reexamination of Dreesen's original standard. In 1965, a conference on the biological effects of

asbestos concluded that the 5 mppcf limit was in all probability inadequate to give complete working lifetime protection against all forms of asbestos.

The basis of Dreesen's work was re-examined and in 1965, E. L. Schell published a revealing critique on the thrshold limit values of 5 mppcf. The method of counting the particles was criticized. This was done by an impinger which collected both fibrous and nonfibrous particles. This can be expected to give only an indirect measure of the risk of asbestos, because of the great importance of long fibers.

The study done by Dreesen was limited to the disease, asbestosis, and no consideration was given to other diseases which might also be the result of long-term exposure to asbestos.

Limiting the study to textile plants was unfortunate because by the 1960's, asbestos was utilized in other industries as well. These other industries might use different grades, sizes, and varieties of asbestos. Schell also pointed out that textile work utilizes cleaner fibers and more flexible fibers in their processes.

Dreesen's study was limited entirely to the American textile industry. Therefore, conclusions to be drawn from other textile plants, such as in Britain and other countries was not done. These valuable comparisons could have been utilized in order to arrive at a reasonably safe TVL for asbestos.

The particles were counted with an ordinary microscope and calibrated to count dust particles, using the light-field technique. Types of particles were not separated (e.g, stone dust or cotton dust) and small particles beyond the resolving power of the optical microscope were not counted.

Processes within the textile plant are difficult to separate. One job could be significantly dustier than other operations. It has been pointed out that counts may vary according to the speed of the machine, grades of material used, nature of weaving requirements, whether it be wet or dry weaving and, finally, the count can even vary with the end product.

Dreesen's work did not take into account the nature of the dust. Gross appearance of the dust differed depending on the plant. Two of the plants were large and two were small. Two converted crude asbestos into yarn and woven goods, while the other two purchased yarn and ended with finished goods. All of these variances were not corrected but rather lumped together. Depending on the occupation the dusts varied. Differences were also found in the proportion of fibers to particulate matter in the dust. Today, chrysotile is supplied cleaner and the percent of particles and fibers in the dust are different than in the 1930's and 40's. A 5 mppcf count today could be altogether different from that in 1938.

It should be appreciated and understood that a 5 mppcf count includes background dust such as cotton and rock dust besides asbestos fibers. The proportions of these ingredients can vary which introduces a very important factor into the credibility of setting the 5 mppcf value as a TLV. Also related to Dreesen's work we find that the dust counts were given as an average. However, the range was vast and biological evidence today suggests that peak exposure may be more important than constant overall background exposure, since on these occasions the defense mechanism of the lungs may be overwhelmed with huge retention at these particular times. One example here should suffice to make this point quite clear.

Picker men were exposed to 34.3 to 74.3 mppcf. But during certain phases of their operations this could go as high as 211 mppcf.

An extremely revealing point which was brought out earlier in our paper, concerns the length of employment of the men whom Dressen studied. Of the 511 employees, 333 had worked for less than 5 years, 73 out of the 511 had worked for less than 1 year and, most importantly, only 66 of the 511 had worked for more than 10 years. Thus, the credibility of the 5 mppcf as a TLV for asbestos is unavoidably questioned when one considers that asbestosis usually has a latent period of 20-25 years before the onset of the disease.

In conclusion, this was a"point-in-time" study and many of the ill were missing as well as the dead being uncounted. Therefore, they were not considered in the overall evaluation of setting the 5 mppcf limit. (Schell, 1965.)

Another factor influencing the need for taking a new look at the asbestos standard was the development of a method by the British Asbestos Research Council for collecting and counting asbestos fibers and their observation that this was a much better index of exposure to asbestos than total particulate count. (Galley, 1969.) The method was termed the membrane filter technique which has been discussed earlier.

As a result of this "reawakening" in the 1960's, the threshold limit value committee, in 1968, reviewed the background of the asbestos threshold limit value of 5.0 mppcf along with the data that had emerged since the study of Dreesen and decided that the asbestos value should be revised.

The committee report was approved by the conference and the new threshold limit value was established to be a time-weighted average. Fiber count limit of 12f/cc greater than 5 μ in length or 2 mppcf. This value was believed to be safe for workers exposed to asbestos for a period of 30 years. (Cralley, 1969.)

However, this was not to be accepted as a permanent TVL for asbestos since the current data were still believed to be too meager and the judgments too subjective. It is also relevant to point out that the committee although suggesting a 12f/cc limit did not completely abolish the old standard of 5 mppcf. Because they felt that there was not enough medical evidence to fully negate the old standard, it remained as a ceiling value,below which all concentrations should fluctuate, and the 2 mppcf was set as a time-weighted average.

During this transitional stage, it was deemed advisable to keep exposure under the 5 mppcf by utilizing the previously mentioned time-weighted average of 2 mppcf. Under these criteria, 5 percent of exposure is permitted to exceed the 5 mppcf. The impinger is used as the sampling instrument under these circumstances. (Cralley, 1969.)

Judgment factors are involved in choosing whether the fiber count is to be used in assessing exposures where asbestos is encountered. Where asbestos is a significant component, the fiber limit is the one recommended. Even where asbestos is only a minor component or exists in trace amounts, if no other highly biologically reactive material is present, fiber count is recommended. Otherwise, applying the particulate count limit may lead to the use of levels more restrictive than necessary. (Cralley, 1969.)

Due to the appearance of new evidence in 1970, the TLV was reviewed and once again it was decided to revise the standard. Health experience in asbestos plants indicated that the 5 mppcf limit was not sufficiently low to protect workers exposed for 30 years. This was supported by Balzer and Cooper who reviewed asbestosis occurring among insulation workers where asbestos exposures were deemed highly unlikely to have exceeded a time-weighted average of 5 mppcf. (Documentation of Threshold Limit Values; ACGIH, 1970.)

A study by Williams and his associates compiled data from two Pennsylvania asbestos textile plants going as far back as 1930. Even though exposures were below 5 mppcf,and in many cases below 2 mppcf, 64 cases of asbestosis were reported from the two plants. (Documentation of Threshold Limit Values; ACGIH, 1970.)

In view of these data it was decided that particles counted by the impinger procedure often include extraneous materials, and a limit of even 1 mppcf might be unrealistic in processes where dust other than asbestos was present, even in relatively low concentrations. Therefore, a limit of 5 fibers/cc, longer than 5 microns, as determined by collection on membrane filters and counted using phase-contrast illumination at 430 X magnification was recommended. (Documentation of Threshold Limit Values; ACGIH, 1970.)

SUMMARY

The Threshold Limit Value standards in America have paralled standards used by the United Kingdom. Each searched for a meaningful set of criteria by which to control the emissions of asbestos dust from mining, manufacturing, and commercial areas where fine fibrous particles might invade the gastrointestinal or pulmonary complexes of mankind. Asbestos requires a Threshold Limit Value as a guideline for quantitative exposure concentrations to protect the workers and passersby alike from inhaling excessive disease contributing fibers.

Table I below gives the 1938 to 1971 standards and the computed reduction of fibers inhaled by innocent bystanders and others living in the community. (Joint American Industrial Hygiene Association; ACGIH, 1963.)

TABLE I

TLV Standards for Asbestos Dust

Date	Country	Fibers Inhaled*	Mppcf	Fibers/cc	Years (< 1 % risk)	
1938-69	U.S.	176.5×10^6	5.0	178.0	0.0	**
1969	U.S.	70.6×10^6	2.0	12.0	10.0	***
1971	U.S.	29.9×10^6	0.84	5.0	22.5	***
1970	Britain	11.75×10^6	0.33	2.0	50.0	

*Zero work rate Kg-M/Min.

** Impinger monitoring method

*** Membrane filter monitoring method

During the 31 years from Dreesen's evaluation to 1969, numerous studies have been made resulting in a wealth of statistical information and the promulgation of precaution. (Documentation of Threshold Limit Values; ACGIH, 1971; Brodeur, 1968; Schall, 1965.) Gradually, industries processing asbestos rock, modified operations to reduce in-plant levels of asbestos dust. One company's attempt to so conform (which unfortunately cannot be named) has resulted in its consideration to discontinue operations. This could cause economic turmoil in the community. Others appear to be diligently abating in-plant pollution to more nearly acceptable levels.

Insufficient ambient data of background levels of high population centers gives cause to wonder why the hue and cry for standards that appear to do so little for the worker or others subjected to asbestos inhalation. A reduction of the inhalation rate from 176.5 million to 29.9 million fibers per 8 hours or even to 11.75 million does not, on the surface, portend much relief unless asbestos is not the culprit it is made out to be. Does a tolerance level exist in homo sapiens? How serious to human consumption is asbestos? If asbestos is a serious culprit, as the papers by Dr. Irving J. Selikoff and his co-workers suggest, why should such disdainful appreciation for the dedicated asbestos workers and his neighbors be allowed to continue? Many statements have been made by equally dedicated workers in their attempt to stem the tide of apathy that industry historically seeks to stalk. The innumerable toxicological evaluations indicating extremely long incubation periods during which a myriad of particles, gases, fumes,

pesticides, etc., have been respirated, gives cause to wonder about the part asbestos plays. Some people die or linger in ill health, while others seem to enjoy long productive health lives. The existence of people with a high degree of susceptibility, the unexplainable synergistic capability of various pollution parameters, or some other mystery, excites the scientifically or humanistically minded to expend exhaustive efforts to seek the solution.

How fortunate we are to have the compiled data from numerous individuals in many localities that give information on the asbestos environments, wherein a variety of work loads are performed. Those work loads which exceed the zero work load represented by the above figures, subject individuals to a much greater volume of inhaled asbestos, by multiples of up to eight times the example cited. This would give a maximum asbestos fiber inhalation of 1660 Kg-M/M work rate of from 1412×10^6 at 5 mppcf level to 94×10^6 at the British level of .33 mppcf.

Is the control of asbestos fibers so difficult and costly? Would 10 fibers per 8-hour day be safer? The thinking men in the industry have devised many abatement methods. Greater effort must be expended in this direction in order to assure the asbestos worker of greater freedom from the horrible fate which confronts too many of them. It is in this direction that Unions, industries, and individuals of the medical, epidemiological, toxicological, engineering, and physiological areas of interest must aggressively head. This must be done at a much faster pace to produce a realistic, meaningful standard for the benefit of the asbestos workers and other related industrial workers. Fibrosis, pleural thickening or calcification, cytological and histological

features, peritoneal areas, epithelium-like and mesechymal tumor cells, ferriginous bodies of one type or another do not belong in the pulmonary or gastrointestinal regions of man. It matters not whether asbestos is the cause or the co-factor, the results may be disastrous to the individual whose body is the host.

Now that the problem of controlling asbestos inhalation has been elucidated, we as a group deem it worthwhile to draw some conclusions and consequently, some guidelines in alleviating the problem.

First of all, it is extremely difficult at this time to reduce the risk of asbestosis completely. The reason can be simply stated: Our knowledge and understanding of the dose-response relationship of asbestos in man is minimal. As a result, this barrier in itself presents a problem that needs an answer before we can hopefully establish a meaningful threshold limit value for asbestos.

Our contention is that more effort be placed in the area of solving the petho-physiological mechanism of asbestosis. Although a TLV of 5 f/cc could be the answer, it does not seem ethical to merely sit back and wait until 1980 or 1985 for data to come in as to the validity or not of today's standard. On the contrary, research should be initiated to elucidate the causal mechanism of this particular fibrosis. To this end, toxicology, epidemiology, and other health oriented discipline must make a unified effort to reveal the answers which will in turn solve the problem of how to eliminate asbestosis from the worker's environment.

Secondly, although the membrane filter is a considerable improvement over the impinger, we should not be satisfied. It is extremely time consuming and inconvenient because of the steps involved in actually arriving at a sample count. A TLV is meaningless unless adhered to. It is our opinion that an instrument needs to be constructed which saves time and effort. As a result, fiber counts could be made more frequently and the standard followed more closely. It is only justifiable to negate a standard after closely quantitating the dose-response from it. To insure this, we need to institute means of minimizing deviations from the standard.

Lastly, and possibly of most importance, we need to educate the worker. If asbestos is deadly, the worker must know it is deadly. He should be made aware of the mortality statistics of his occupation and encouraged to have annual check-ups. One example at this time should further prove the need for such a program. Having visited the Asbestos Local No. 24 here in Pittsburgh, we were enlightened to the sobering fact that only once have the men been encouraged to have a chest X-ray in the 65-year history of their union. And health records of the men are virtually nonexistent. We are talking in terms of 273 members of this union. It would appear to be a worthwhile endeavor to encourage these men and their union to initiate an awareness program as to the occupational hazards of asbestos.

BIBLIOGRAPHY

1. Addingley, C.G., Asbestos Dust and Its Measurements, Annals of Occupational Hygiene, 1966-67, Vol. 9-10, pp 73-82

2. American Conference of Governmental Industrial Hygienists. Threshold Limit Values for 1964; American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio.

3. American Conference of Governmental Industrial Hygienists. Threshold Limit Values of Airborne Contaminants and Intended Changes Accepted by ACGIH for 1971; Cincinnati, Ohio, American Conference of Governmental Industrial Hygienists 1971; 27 pp.

4. American Conference of Governmental Industrial Hygienists Committee on Threshold Limit Values, "Documentation of Threshold Limit Values." Third Edition (1971). Available from the Secretary-Treasurer, American Conference of Governmental Industrial Hygienists, 1014 Broadway, Cincinnati, Ohio 45202

5. Asbestos Industry Regulations (1931). Statutory Rules and Orders, No. 1140, H.M.S.O., London, 1931.

6. Asbestos Minerals: The Need for and Feasibility of Interim Air Pollution Controls. Prepared by the Panel on Asbestos, Committee on Biological Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council, Washington, D.C. - Second Draft, February 12, 1971.

7. Ayer, H. E., J. R. Lynch, and J. H. Fanney, Annals of New York Academy of Sciences, 132, Article 1, 1965 - pp 274.

8. Biologic Effects of Asbestos, H. E. Whipple, ed., Annals of New York Academy of Sciences 132, Article 1, 1965.

9. British Occupational Hygiene Society Standards for Chrysotile Asbestos Dust, Ann. Occupational Hygiene 11: 14-70, 1968.

10. Brodeur, Paul, The Magic Mineral, The New Yorker, pp 117-127, October 12, 1968.

11. Cooke, W. E., Pulmonary Asbestosis, British Medical Journal 2 : 1024-1025, 1927.

12. Cooper, C.C., Asbestos as a Hazard to Health

13. Corn, Morton; P. Enterline, and M. Utidjian; Asbestos Dust: A Contaminant in the Work and Urban Environments. Presentation to Occupational Health 211 Class on Sept. 17 and Sept. 20, 1971 (Graduate School of Public Health, Univ. of Pittsburgh).

BIBLIOGRAPHY (Cont'd.)

14. Cralley, L. J.; R. G. Keenan, R. E. Kupel, R. E. Kinser, and J. R. Lynch. Characterization and Solubility of Metals Associated with Asbestos Fibers, Amer. Ind. Hygiene Assoc. Journal, 29, 569-573, 1968.

15. Doll, R.; Mortality From Lung Cancer in Asbestos Workers. British Journal Industrial Medicine - 12; 81-86, 1955.

16. Dreesen, W. V.; J. M. Dallowalle, T. I. Edwards, J. W. Miller, and R. R. Sayer. A Study of Asbestos in the Asbestos Textile Industry; Public Health Bulletin No. 241, U.S. GPO, 1938.

17. Edwards, G. H.; J. R. Lynch. The Method by the Public Health Service for Enumeration of Asbestos Dust on Membrane Filters. Annals of Occupational Hygiene, Vol. 11, pp 1-7, 1968.

18. Gloyne, S. R.; Pneumoconiosis - A Histological Survey of Necropsy Material in 1205 Cases, Lancet, 1 : 810-814, 1951.

19. Hickish, D. E.; and K. L. Knight. Exposure to Asbestos During Brake Maintenance, Annals of Occupational Hygiene 13 : 17-21, 1970.

20. Joint American Industrial Hygiene Association - American Conference of Governmental Industrial Hygienists. Respiratory Protective Devices Manual. Ann Arbor, Mich., 1963.

21. Lynch, K. M.; and W. S. Smith. Pulmonary Asbestosis III: Carcinoma of the Lung in Asbestos-Silicosis; American Journal of Cancer 24: 56-64 (1935).

22. Luxon, S. Technical Implementation of the New Asbestos Regulations. American Occupational Hygiene, 13: 23-24, 1970.

23. McDonald, A. D.; et al. Epidemiology of Primary Malignant Mesothelioma Tumors in Canada. Cancer, 26: 914-919, 1970.

24. McDonald, S. Histology of Pulmonary Asbestosis. British Medical Journal, 2: 1025-1026, 1927

25. McEwen, J., et al. Mesothelioma in Scotland, British Medical Journal, 4: 575-578, 1970.

26. Merewether, E. R. A. Asbestosis and Carcinoma of the Lung, Annual Report of the Chief Inspector of Factories for the year 1947. London: H.M. Stationery Office, 1949.

27. Merewether, E. R. A. The Occurrence of Pulmonary Fibrosis and Other Pulmonary Afflictions in Asbestos Workers. Journal of Industrial Hygiene, 12: 198-223, 239-257, 1930.

BIBLIOGRAPHY (Cont'd.)

28. Newhouse, M.L., A Study of the Mortality of Workers in an Asbestos Factory; British Journal of Industrial Medicine, 26: 294-301, 1969.

29. Oliver, T., Clinical Aspects of Pulmonary Asbestosis, British Medical Journal, 2: 1026-1027, 1927.

30. Roach, S. A., Hygiene Standards for Asbestos, Annals of Occupational Hygiene, 13: 7-16, 1970.

31. Schall, E. L., Present Threshold Limit Value in the U.S.A. for Asbestos Dust: A Critique. New York Academy of Sciences Annals, 132: 316-321, 1965.

32. Schnitzer, R. J.; R. J. Pundsack, Asbestos Hemolysis, Environmental Research, 3: 1-13 (1970).

33. Selikoff, Irving J.; J. Chung, and E. C. Hammond. Asbestos Exposure and Neoplasia. JAMA 188: 22-26, 1964.

34. Selikoff, Irving J.; E. C. Hammond, and J. Chung. Asbestos Exposure, Smoking, and Neoplasia, JAMA, 204: 104-112, 1968.

35. Selikoff, Irving J.; E. C. Hammond, and J. Chung. Mortality Experiences of Asbestos Insulation Workers. Proc. Int'l. Conf. Pneumoconiosis, Johannesburg, South Africa, 1969.

36. Stumphins, J.; and P. B. Meyer. Asbestos Bodies and Mesothelioma, Annals Occupational Hygiene, 11: 283-293, 1968.

37. Wagner, J.C.; C. A. Sleggs, and P. Marchand. Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province. British Industrial Medical Journal, 17: 260-271, 1970.

38. Cralley, Lewis J., Dust Sampling Instruments and Dust Standards in the United States of America for Asbestos. Pneumoconiosis Proceedings of the International Conference of Johannesburg, South Africa, 1969. H. A. Shapiro, Editor. pp 10-12.