ASBESTOS, ALL FORMS

CAS number: 1332-21-4

ACTINOLITE

CAS number: 77536-66-4

AMOSITE

CAS number: 12172-73-5

ANTHOPHYLLITE

CAS number: 77536-67-5

CHRYSOTILE

CAS number: 132207-32-0

CROCIDOLITE

CAS number: 12001-28-4

TREMOLITE

CAS number: 77536-68-6

TLV–TWA, 0.1 fiber/cc, Respirable fibers*

A1 — Confirmed Human Carcinogen

* Fibers longer than 5 µm and with an aspect ratio ≥ 3:1, as determined by the membrane filter method at 400 to 450 times magnification (4-mm objective), using phase-contrast illumination.

Summary

A TLV–TWA of 0.1 fiber/cc is recommended for occupational exposure to all forms of asbestos. This value is intended to minimize the potential for asbestosis and also to minimize the risk for development of lung cancer. The recommended Threshold Limit Value should also serve to reduce the risk of mesothelioma in asbestos-exposed workers. Based on the significant data associating human exposure to asbestos with subsequent lung cancer and mesothelioma, an A1, Confirmed Human Carcinogen, notation is assigned all forms of asbestos. The recommended TLV for the various forms of asbestos is for fibers > 5 µm in length, with an aspect ratio of ≥ 3:1, as determined by the membrane filter method at 400 to 450 times magnification (4-mm objective), using with phase-contrast illumination. As for all substances designated as an A1 carcinogen, worker exposure by all routes should be carefully controlled to levels as low as possible below the TLV. Sufficient data were not available to provide the basis for recommending a SEN notation or a TLV–STEL.

Chemical and Physical Properties

According to the mineralogical definition,[1] asbestos is "1) A collective mineralogical term encompassing the asbestiform varieties of various minerals; 2) An industrial product obtained by mining and processing primarily asbestiform minerals." For the purpose of considering a TLV recommendation for asbestos dust in the workplace, only the second definition is applicable.

Asbestos is a commercial term that covers a number of naturally occurring silicate minerals whose crystals grow in the fibrous habit.[2] Although there are four types of natural asbestiform mineral fibers that have been in industrial use, only three have been used in the United States: amosite, chrysotile, and crocidolite. The fourth, anthophyllite, is mined and used in Finland. Of the three asbestos

types that have been used in North America, Canadian chrysotile has formed 95% of all natural mineral fibers used, with amosite and crocidolite (both imported from South Africa) constituting the other 5%. Chrysotile is classified as a serpentine mineral, whereas the other three asbestos types are amphiboles. Tremolite is an amphibole that is found as a contaminant in chrysotile and talc deposits.

Asbestos fibers exist in parallel bundles that tend to split longitudinally when subjected to pressure, forming finer and finer fibers, which maintain a high length-to-width aspect ratio. Crocidolite and amosite fibers tend to be straighter and stiffer than chrysotile fibers.

Major Uses

The asbestos minerals show high tensile strength, good thermal and electrical insulating properties, and resistance to chemical attack. Asbestos also possesses inherent flexibility enabling it to be woven into yarns and textiles. Asbestos is used in friction materials such as brake linings and as reinforcement in concrete and synthetic resins. It has also been used in filtration applications.

Animal Studies

In 1951, Vorwald et al.[3] demonstrated that inhalation and the intratracheal injection of chrysotile asbestos produced peribronchial fibrosis of the lung in guinea pigs, rabbits, and rats, similar to that seen in human asbestosis. In 1974, Wagner et al.[4] published the results of an inhalation study in specific pathogen-free rats dosed with amosite, crocidolite, anthophyllite, and chrysotile. These rats developed asbestosis, lung cancer, and mesothelioma with all four forms of asbestos.

Since the 1970s, many studies have been carried out in experimental animals by means of intrapleural and intraperitoneal routes of exposure. These routes were useful in evaluating the effects of fiber size and fiber properties directly on target tissue, but they were not representative of worker exposure. The delivery of a bolus dose of fibers may overwhelm normal physiological clearance mechanisms. Thus, fibers, which may not normally reach the pleural space, may produce mesotheliomas when administered directly into the pleural or peritoneal cavities. The intrapleural studies in rats by Stanton and Wrench[5] and Stanton et al.[6,7] showed that tumor development was most closely associated with long (> 8 µm) and thin (< 0.25 µm) fibers — the so called Stanton fibers.

Pott et al.[8–10] carried out a series of experiments in rats given intraperitoneal injections of fibers. The peritoneum proved to be a very sensitive target tissue for studying fiber-induced mesothelioma. Nearly all mineral fibers tested by this method have produced mesothelioma, although some very soluble fibers have not (e.g., wollastonite). Because of the high sensitivity of the intraperitoneal test, a tumor response of at least 10% was considered necessary by Pott and associates to establish a positive result. Sometimes even the saline control injection produces positive results.

Long-term toxicology and carcinogenesis studies published by the U.S. National Toxicology Program have been limited to feeding studies, which included chrysotile, amosite, and crocidolite.[11] Chrysotile[12,13] and amosite[14,15] were studied alone and in combination with dimethylhydrazine. These forms of asbestos were tested in male and female Syrian golden hamsters and F344/N rats, while crocidolite data were available only from rats. Results were usually negative; however, there was one report of some evidence of carcinogenic activity from chrysotile in male F344/N rats.[13] The combination of dimethylhydrazine with chrysotile and with amosite as tested in feeding studies did not yield a significant increase in tumors above the background levels observed in the dimethylhydrazine group alone or in the untreated control group.

Intratracheal administration studies have also been done. These studies involve the delivery of a bolus of large numbers of fibers into the respiratory tract. Since there is no standardized technique for intratracheal administration, it is difficult to compare results from different researchers. A single, massive dose may overwhelm clearance mechanisms. Dose rates strongly influence the acute response.

The inhalation route is obviously of most relevance to human exposures to fibers.

In summary, studies in experimental animals show that longer fibers were more toxic than equal masses of shorter fibers of the same composition. Intraperitoneal and inhalation studies in rats suggest that short fibers pose little, if any, concern for disease development at any site. Fibers of 10 to 15 µm in length were needed to produce disease in the parenchyma of the lung, but shorter fibers of 8 to 10 µm in length can cause mesothelioma.

There is no biological reason to assume that there is a sharp demarcation in fiber length in terms of health effects. Therefore, for assessing exposures, it is necessary to count fibers deemed capable of deposition within the bronchoalveolar regions of the lung. This would include fibers < 3 µm in diameter and > 5 µm in length.

Human Studies

Comprehensive reviews have been published relating to the human health effects of exposure to asbestos.[2,16,17] Inhalation of asbestos can lead to pulmonary fibrosis (asbestosis), cancer of the lung, mesothelioma chiefly of the pleura but also of the peritoneum, pleural disease (diffuse pleural thickening), and a benign condition of the pleura known as pleural plaques. All forms of asbestos were implicated in these diseases, but there were differences in degree among the different asbestos fiber types.

Although identification of asbestos dust as a cause of fibrosing inflammation of lung tissue occurred as early as 1907,[18] it was not until 1930 that a more definitive study by Merewether and Price[19] resulted in the regulations which greatly improved hygienic conditions in asbestos factories in the United Kingdom. The development of lung cancer in asbestos workers, first reported in 1934 by Wood and Gloyne[20] in the United Kingdom and in 1935 by Lynch and Smith[21] in the U.S., was not firmly established until 1955 by Doll[22] in a published study of workers in an English asbestos textile factory. This was followed by the 1964 paper of Selikoff et al.[23] concerned with cancers in U.S. insulation workers. The relationship between the inhalation of asbestos dust and mesothelioma was demonstrated in 1960 by Wagner et al.[24] in South African amosite miners exposed to crocidolite.

Asbestosis is a diffuse, nonuniform, interstitial fibrosis of the lungs that is generally most severe in the basilar portions. As a result of the fibrosis, some of the air spaces (alveoli) are not perfused with blood, and the alveoli that are perfused with blood may not be adequately ventilated because of stiff, thickened alveolar walls. The fibrosis makes the lungs less compliant, thereby increasing the energy requirement for breathing. There is increasing impairment in diffusion of gases leading to increasing breathlessness.

It is not uncommon to find thickening of the visceral pleura, sometimes very severe, by extension of the parenchymal inflammation. This causes an additional increase in the effort of breathing.

The parietal pleura may show patches of severe thickening, particularly over the diaphragm and the lower portions of the chest wall, resulting in the so-called pleural hyaline plaques. These may become visible in X-ray films of the chest, particularly if these plaques become impregnated with calcium salts. Such pleural plaques may develop from asbestos exposure in the absence of asbestosis. They cause no symptoms.

A study published in 1964 of the members of an asbestos insulators union revealed that deaths from lung cancer in this population were much greater than expected.[18] A later investigation published in 1973 by Hammond and Selikoff[25] of a much larger number of these workers (17,800) showed that nearly all cancers occurred in cigarette smokers. These authors concluded, "It seems clear, then, that lung cancer is uncommon among asbestos insulation workers who have no history of cigarette smoking, and that if the risk is increased such an increase is not great."[25]

The total lung cancer rate in this cohort of workers was 4.8 times the expected. The asbestos insulators who had a history of cigarette smoking had a lung cancer rate 5.4 times the expected rate; however, compared with the lung cancer rate of the nonsmoking workers, the lung cancer rate was 14 times greater for insulators who smoked.

All types of asbestos are known to cause the asbestosis, pleural changes, lung cancer, and mesothelioma described above. However, there is experimental and epidemiologic evidence that there may be differences in the potential of the different asbestos types to produce disease. Thus, it has been suggested that crocidolite has the greatest potential to produce disease; chrysotile, the smallest; with amosite occupying an intermediate position.[26] In a study of 1348 retirees from the asbestos industry by Enterline and Henderson,[27] the respiratory cancer rate of men exposed only to chrysotile was 2.4 times the expected, whereas this rate was 5.3 times the expected for men who had been exposed to a combination of chrysotile and crocidolite. In the asbestos cement industry, a similar difference was observed. Workers exposed only to chrysotile and cement (shingles and sheets) had a respiratory cancer rate of 1.4 times the expected, whereas workers exposed to both chrysotile and crocidolite in combination with cement (asbestos cement pipes) had a respiratory cancer rate 6.1 times the expected.

Mesotheliomas are rare, usually rapidly fatal cancers that originate from the surface lining the chest or abdominal cavity. From 1960 through 1975, 4539 mesotheliomas were reported worldwide.[28] The vast majority of these cancers were in people exposed to crocidolite alone or in combination with other types of asbestos. McDonald and McDonald[28] tabulated those reports of mesothelioma where the type of asbestos exposure was known. Although the number of such cases was small, where the exposure was to crocidolite alone or in combination with other types of asbestos, death from mesothelioma constituted 6.1% of the deaths from all causes (range, 2.42%–16.07%). In contrast, deaths from mesothelioma in workers exposed only to chrysotile constituted only 0.3% of the deaths from all causes (range, 0.24%–0.87%). An even greater contrast is found in the Finnish statistics of workers exposed to anthophyllite. Meurman et al.[29] investigated 216 deaths that occurred among approximately 900 miners and millers of anthophyllite during the 32-year period from 1936 to 1967 and found not one case of mesothelioma.

Not all mesotheliomas result from asbestos exposure. There is a background of "naturally" occurring mesotheliomas that has been estimated to be about ten for males and four for females per million persons aged 45 years and older.[25] Furthermore, it is not uncommon in the various epidemiologic studies reported that 15% or more of the mesothelioma cases have no history of ever having been exposed to asbestos. Perhaps the most important indication that mesotheliomas may result from causes other than asbestos exposures (in this instance, also environmental) comes from a report by Artvinli and Baris[30] who described a mesothelioma incidence of 2.3% in 1974 in the

village of Karain in Turkey (population, 604). This population has been exposed for many generations to dust from the soil that contains kaolin, mica, and volcanic glass particles but no asbestos.

A small excess of deaths from gastrointestinal cancers have been noted in several epidemiologic studies of asbestos workers.[25,31] An association of laryngeal cancer with asbestos exposure has been claimed; pancreatic cancers and lymphomas have also been mentioned in this connection. However, proof of a causal association to asbestos exposure rests, as yet, on an insecure basis.

Whether or not there is a dose–effect relationship associated with asbestos dust has been answered affirmatively by a number of epidemiological surveys.[31–35] Whereas this relationship is clear-cut with regard to asbestosis and lung cancer, it is less well marked with regard to mesothelioma; but it is, nevertheless, positive. McDonald[36] points to a case–control analysis based on seven cases of mesothelioma at Thetford Mines that includes no case of mesothelioma with < 30 million particles per cubic foot-years exposure and which suggests that the risk increases with exposure. Although fiber equivalents for the dust concentrations in millions of particles per cubic foot are difficult to estimate, McDonald further points out that there is evidence for believing that the conversion factor cannot be less than two. The data of Newhouse and Berry[37] demonstrated a doubling of the incidence of mesothelioma for males who had severe exposures as compared with that of the workers who had light or moderate exposures. This was equally true for those employed < 2 years. In all of the other investigations of mesothelioma incidence, the degree of dust exposure was not indicated, thereby making the determination of any dose–effect relationship difficult.

Earlier exposure data from the asbestos Industry, on which a recommendation for a threshold limit of asbestos exposure could be based, came from studies in England.[35,38] Using the presence of persistent high-pitched rales in the basal portions of the lungs as criterion for the diagnosis of asbestosis, it was determined from a population of asbestos textile workers that less than 100 fiber-years of exposure (2 fibers/cc over a 50-year working period or 4 fibers/cc over a 25-year period) would cause the development of asbestosis in no more than 1% of the workers.[38] This work lead to a departure from the previous dust standard expressed in mppcf to that expressed in terms of fibers/cc. This was in recognition of the variability of the asbestos fiber content of factory dust and that the disease was related to the number of asbestos fibers inhaled and not to the amount of nonfibrous dust particles. Also, it was specified that the counted fibers were to be longer than 5 µm.

The size limit (longer than 5 µm) was placed on the counted fibers because it was not practical to count shorter fibers with an optical microscope (400 to 450 times magnification using phase-contrast illumination with a 4-mm objective). It is recognized that for every asbestos fiber longer than 5 µm, there may be as many as 100 or more fibers shorter and thinner that are not visible under the optical microscope. However, there is considerable experimental evidence to indicate that asbestos fibers shorter than 5 µm are not pathogenic.[39]

A 1976 publication by Gillam et al.[40] suggested that the proposed limit of 2 asbestos fibers/cc longer than 5 µm was inadequate to protect workers against nonmalignant as well as malignant respiratory disease. This conclusion was based on a study of 440 hard rock gold miners who had been exposed to an asbestiform mineral (cummingtonite–grunerite). These investigators found 10 respiratory cancer deaths (including a carcinoma of the maxillary sinus and a mediastinal carcinoma) when only 2.74 such deaths had been expected. Another 5 deaths were from nonmalignant respiratory diseases other than influenza and pneumonia when 1.85 deaths had been expected; these deaths included those from silicosis (the respirable dust contained 13% free silica).

Although the diagnosis of asbestosis was not mentioned by Gillam et al.,[40] it is of interest that these authors emphasized that the ambient air in the gold mine contained an average of 4.82 fibers/cc, 70% to 80% of which were fibrous amphiboles and 60% to 70% of these amphiboles were fibrous grunerite (amosite). Fibers longer than 5 µm averaged 0.36 fibers/cc; approximately 94% of the airborne fibers were less than 5 µm long, averaging 0.13 µm in diameter and 1.1 µm in length.

McDonald et al.[41] investigated the records of the same gold mine as Gillam et al.,[40] but their cohort consisted of 1321 men who had completed 21 years of service with the mining company (in contrast to the 440-man cohort studied by Gillam et al.). The following is a summary of their findings.

All but 10 of the men were traced to the end of 1973 when 651 were still living; cause of death was ascertained for 657 of the 660 who had died. The numbers of deaths observed in various diagnostic categories, with "expected" figures in parenthesis, were as follows: respiratory cancer, 17 (16.5); abdominal cancer, 39 (35.1); other malignant diseases, 37 (39); pneumoconiosis, 39 (0); respiratory tuberculosis or silico-tuberculosis, 39 (3.6); heart disease, 264 (232.5). Silicosis was given as the cause in 37 of the 39 pneumoconiotic deaths and mentioned on the death certificate in 28 of the 264 coded to heart disease. The occurrence of deaths ascribed to pneumoconiosis, tuberculosis, and heart disease was, in each case, related directly to dust-exposure category; deaths coded to respiratory, abdominal, and other cancers showed no such relationship. The pattern of mortality among men with long employment in this industry indicated a serious pneumoconiotic hazard characteristic of hard rock miners, but not of cancer.

An 8-year follow-up of the same population of

asbestos workers, from which the 100 fiber-years exposure was derived as a reasonably safe level,[38] it was found that mortality was increased for lung cancer.[35] There were 31 deaths from this cause where only 19.3 had been expected. From non-malignant respiratory disease, there were 35 deaths where 25 had been expected. In addition, there were five deaths from pleural mesothelioma.

The authors determined that the mean dust level of the workers had been below 5 fibers/cc only in the last decade prior to the date of the study. In 1951, the mean dust level was 10.8 fibers/cc, and 89% of the men had been exposed to mean levels above 5 fibers/cc. In 1972, the mean dust level was 2.9 fibers/cc; only 3% of the men were exposed to a mean level greater than 5 fibers/cc, while 65% were exposed to a mean level between 2 and 5 fibers/cc and 32% to a mean level below 2 fibers/cc.

Because there is a delay of 15 or more years between first exposure and any resulting cancer, Peto et al.[35] considered that the increased mortality demonstrated did not reflect the effects of working conditions over the last 15 or 20 years. They, therefore, proposed to continue the follow-up on workers entering scheduled areas since 1951. In terms of the 100 fiber-year or 2 fibers/cc standard suggested by the British Occupational Hygiene Society (BOSH), the excess mortality reported above could be attributed to asbestos exposures considerably above this level.

The workers in the asbestos textile factory, from which the BOHS standard of 2 fibers/cc was derived, have been studied by highly qualified investigators whose reports were published in 1955,[22] 1965,[32] 1968,[42] and 1977.[35] These workers represented the only cohort of asbestos workers in the world in which health effects were correlated with definitive exposure data defined as fibers/cc.

Dose–Response Relationships for Asbestos-Related Diseases

Asbestosis

The development of asbestosis follows a dose–response relationship with a higher incidence of this disease with increasing levels of exposure.[43] Doll and Peto[16] found it difficult to relate exposure levels to disease in their review because asbestosis is not always a fatal disease. It has been generally agreed that low levels of exposure, even if they initiate a fibrotic reaction in the lungs, will not likely progress to the point of clinical manifestation of asbestosis. Hughes in Weill[44] found very little radiological evidence of asbestosis in asbestos cement workers with a cumulative exposure level below 30 to 40 fibers/cc-years. These authors also noted that one plant in their study, in which crocidolite exposure occurred, had a higher prevalence and faster progression of asbestosis than with chrysotile exposure alone. Becklake[45] reported that the prevalence rates for radiologic evidence of asbestosis were on the order of 20% for cumulative exposure of about 25 fibers/cc-years in a Quebec textile mill using Quebec chrysotile only and 8% for a cumulative exposure of about 60 fibers/cc-years in Quebec miners and millers. It seems clear that fiber type and airborne fiber size distribution influence the dose–response relationship.

Lung Cancer

All types of asbestos are associated with the risk of developing lung cancer. The cancers occur principally in the main bronchi but may occur in smaller bronchi, in the periphery of the lung, and in a variety of histological types. Most occupational exposures involved mixtures of asbestos fiber types. Nevertheless, the World Health Organization task force[2] concluded that crocidolite proved a greater risk of lung cancer than did chrysotile for comparable exposures. Differences have been found in the dose–response curves for lung cancer in mining and milling and in textile, asbestos-cement, and friction product manufacture. The steepest curve was found in textile manufacture. Berry[46] found no significant increases in lung cancer mortality in friction product manufacturers. Exposure to chrysotile in the asbestos-cement industry showed no excess of lung cancer mortality;[43] where amphiboles were used in making asbestos-cement there was an excess of lung cancer.

The most comprehensive study of chrysotile exposure was published by McDonald et al.[43] in 1993. The cohort included 11,000 Quebec miners and millers born between 1891 and 1920 and followed up to 1988. This cohort showed a sharp increase in the standard mortality rate for lung cancer with cumulative exposures above 300 mppcf-years. The conversion of mppcf to fibers/cc presents a problem. The authors suggested that 1 mppcf on average corresponds to 3.5 fibers/cc. Using this conversion, the authors estimated no detectable increase in lung cancer with a 20-year exposure below 50 fibers/cc.

These results were in marked contrast to the studies of lung cancer in chrysotile textile workers.[48–51] The reasons for this difference in lung cancer risk between textile workers and miners and millers are not clear. They may be attributable to differences in airborne fiber distribution and in the estimates of previous exposure. The chrysotile grades used in textiles contain the longest fibers. Dement[51] found a higher proportion of longer airborne fibers (> 15 µm) in textile manufacture compared to other chrysotile users. Doll and Peto[16] calculated nonspecific risk estimates, based on the assumption of a linear no-threshold model, and concluded that a life-long risk caused by 20 to 30 years of exposure in chrysotile textile manufacturing would be about 0.5% for exposures of 0.5 fiber/cc. This prediction was based on particle counts up to

1961, and the reliability of the conversion estimates have been questioned. In addition, the balance of toxicological evidence suggests that the linear no-threshold model may not be appropriate for chrysotile.

Association Between Asbestosis and Lung Cancer

For many years, individuals have pointed out that there is an association between asbestosis and the risk of lung cancer. Both diseases show similar dose–response relationships, similar latent periods, and both require the accumulation of high fiber concentrations in lung tissue. Autopsies of asbestos workers show that asbestosis does not occur until the fiber burden in the lung exceeds 5×10^5 fibers/gram of dried lung (fibers > 5 µm in length). In severe cases of asbestosis, counts were as high as 10^{10} fibers/gram.[52] Studies of asbestos workers with lung cancer show that the lung cancer was seen in association with pulmonary fibrosis.[16,53–56] Hughes and Weill[44] studied 839 New Orleans asbestos-cement workers in which the excess of lung cancer was found only in workers with pre-existing asbestosis (ILO classification 1/0). Animal studies demonstrated that asbestos-induced lung cancer was preceded by pulmonary fibrosis. Some investigators have questioned this association, but the prevailing toxicological view is that there is an association between lung cancer and asbestosis.

Mesothelioma

There is sufficient evidence to show that for a given level of exposure, the risk of developing mesothelioma is far greater with crocidolite and amosite than with chrysotile. Armstrong and co-workers[57] reported that there were 94 cases of mesothelioma out of 1200 deaths in a cohort of Australian miners and millers exposed to crocidolite. The average duration of exposure was relatively brief (3 to 4 months), and most of the cohort was still alive (80%). In contrast, the cohort of Quebec miners and millers of asbestos[47] had only 33 mesothelioma deaths out of a total of over 7000 deaths at the time when most of the cohort subjects had died. It has been suggested that tremolite contamination of chrysotile ore is responsible for the mesotheliomas seen in chrysotile miners and millers.

Overall, it is not possible from the available human and animal data to establish a threshold level of exposure for mesothelioma.

TLV Recommendation

A TLV–TWA of 0.1 fiber/cc is recommended for all forms of asbestos. This value should provide a significant margin of safety in terms of asbestosis prevention which, on the weight of toxicological evidence, supports the view that the prevention of asbestosis should also minimize the risk of lung cancer.[22,31,35,42,44,53,56] In terms of mesothelioma, there are inadequate dose–response data, but the recommended TLV should also serve to minimize the risk of mesothelioma in asbestos-exposed workers. Based on the significant data associating human exposure to asbestos with subsequent lung cancer and mesothelioma, an A1, Confirmed Human Carcinogen, notation is assigned to all forms of asbestos.

The recommended TLV is for fibers > 5 µm in length, with an aspect ratio ≥ 3:1, as determined by the membrane filter method at 400 to 450 times magnification, using phase-contrast illumination with a 4-mm objective for the various forms of asbestos. Sufficient data were not available to recommend a SEN notation or a TLV–STEL. The reader is expected to be familiar with the section on *Excursion Limits* in the "Introduction to the Chemical Substance TLVs" of the current edition of the *Documentation of the TLVs and BEIs* for guidance and control of excursions above the TLV–TWA, even when the 8-hour TWA is within the recommended limit.

Historical TLVs

Asbestos

1946–1947: MAC–TWA, 5 mppcf
1948–1973: TLV–TWA, 5 mppcf
1968–1969: *Proposed* TLV–TWA, 12 fibers/ml > 5 µm in length, or 2 mppcf
1970–1971: *Proposed* TLV–TWA, 5 fibers/ml > 5 µm in length
1972: *Proposed* TLV–TWA, 5 fibers/ml > 5 µm in length; A1a, Human Carcinogen with an Assigned TLV
1974–1979: TLV–TWA, 5 fibers/cc > 5 µm in length; A1a, Human Carcinogen with an Assigned TLV

Amosite and Tremolite

1978: *Proposed* TLV–TWA, 0.5 fiber/cc > 5 µm in length; A1a, Human Carcinogen with an Assigned TLV
1980–1986: TLV–TWA, 0.5 fiber/cc > 5 µm in length; A1a, Human Carcinogen with an Assigned TLV; Tremolite listing deleted
1987–1997: TLV–TWA, 0.5 fiber/cc* > 5 µm in length; A1, Confirmed Human Carcinogen
1991: *Proposed* TLV–TWA, 0.2 fiber/cc,* asbestos, all forms; A1, Confirmed Human Carcinogen
1997: *Proposed* TLV–TWA, 0.1 fiber/cc,* asbestos, all forms; A1, Confirmed Human Carcinogen
1998–present: TLV–TWA, 0.1 fiber/cc,* asbestos, all forms; A1, Confirmed Human Carcinogen

Chrysotile and Other Forms

1978: *Proposed* TLV–TWA, 2 fibers/cc > 5 µm in length; A1a, Human Carcinogen with an Assigned TLV
1980–1986: TLV–TWA, 2 fibers/cc > 5 µm in length; A1a, Human Carcinogen with an Assigned TLV
1987–1997: TLV–TWA, 2 fibers/cc* ; A1, Confirmed Human Carcinogen
1991: *Proposed* TLV–TWA, 0.2 fiber/cc,* asbestos, all forms; A1, Confirmed Human Carcinogen
1997: *Proposed* TLV–TWA, 0.1 fiber/cc,* asbestos, all forms; A1, Confirmed Human Carcinogen
1998–present: TLV–TWA, 0.1 fiber/cc,* asbestos, all forms; A1, Confirmed Human Carcinogen

Crocidolite

1978: *Proposed* TLV–TWA, 0.2 fiber/cc > 5 µm in length;

A1a, Human Carcinogen with an Assigned TLV
1980–1986: TLV–TWA, 0.2 fiber/cc > 5 µm in length; A1a, Human Carcinogen with an Assigned TLV
1987–1997: TLV–TWA, 0.2 fiber/cc* ; A1, Confirmed Human Carcinogen
1991: *Proposed* TLV–TWA, 0.2 fiber/cc,* asbestos , all forms; A1, Confirmed Human Carcinogen
1997: *Proposed* TLV–TWA, 0.1 fiber/cc,* asbestos, all forms; A1, Confirmed Human Carcinogen
1998–present: TLV–TWA, 0.1 fiber/cc,* asbestos, all forms; A1, Confirmed Human Carcinogen

Asbestos, All Forms

1991: *Proposed* TLV–TWA, 0.2 fiber/cc,* A1, Confirmed Human Carcinogen
1997: *Proposed* TLV–TWA, 0.1 fiber/cc,* A1, Confirmed Human Carcinogen
1998–present: TLV–TWA, 0.1 fiber/cc,* A1, Confirmed Human Carcinogen

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* Fibers longer than 5 µm and with an aspect ratio ≥ 3:1, as determined by the membrane filter method at 400 to 450 times magnification (4-mm objective), using phase-contrast illumination.

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