

My name is Ann G. Wylie. I hold a baccalaureate degree from Wellesley College and a PhD from Columbia University. I am Professor of Geology at the University of Maryland. I have spent more than 30 years studying asbestos and the minerals that compose it.

I am here today to discuss the both the scientific and the federal regulatory definition of asbestos.

REGULATORY HISTORY

In the early 1970's the United States lagged behind the rest of the world in the strict regulation of occupational exposure to airborne asbestos. Regulation of asbestos was one, if not the first, major initiative of both EPA and OSHA when they were formed at this time. Needless to say, these two agencies were in a hurry.

OSHA wrote a **definition of asbestos** and specified a **method for its measurement**; both were incorporated into law. Together these comprise the federal regulatory definition of asbestos.

The federal regulatory definition was written without any consultation with the mineral experts at the United States Geological Survey or the US Bureau of Mines, and, consequently, it was not mineralogically correct.¹

OSHA's regulatory definition identified mineral names without specifying the asbestiform character. This is the same as saying that hail and snow are the same thing. Both are ice, but everyone knows that they are not the same and that have different potentials for harm.

The measurement method, called the membrane filter method², compounded the definitional problem. The foundation for the membrane filter method was developed in the 1960's in British factories that utilized asbestos. The particles included in exposure estimates were specified by both a minimum length and a minimum length to width ratio. A length of >5 micrometers was chosen to reflect an acceptable level of reproducibility among analysts.³ A length to width ratio of 3:1 was also specified, but its choice was not explained. Whatever the reason, 3:1 was arbitrary. It is not a scientific definition of a fiber, it does not reflect the length to width ratio of asbestos fibers, and it was not chosen because of any studies linking it to health effects.

¹ OSHA's list of asbestos is also incomplete. One very public effect of the latter mistake is that most of the asbestos occurring at Libby Montana is not technically covered by asbestos regulations. (Verkouteren and Wylie, 2000)

² Leidel et al., 1979

³ Addingley, C.F., 1966; Lynch et al., 1970

Because of the membrane filter method, particles longer than 5 micrometers with a length to width ratio of 3:1 or higher meet what has become known as the Regulatory Fiber Definition (RFD). They are also referred to as “federal fibers.”

The effect of these two specifications, a mineralogically incorrect definition of asbestos and the development of an arbitrary Regulatory Fiber Definition (RFD), is that sometime during the 1970’s, rock fragments, sometimes called cleavage fragments, became fibers and fragments of six minerals became *de facto* asbestos.

In 1992, OSHA examined this issue in detail. They concluded that there was no scientific evidence that cleavage fragments have the same health potential as asbestos. OSHA removed them from the asbestos standard.⁴ I am not aware of any epidemiological, animal or cellular studies that have been done since the OSHA decision that would change this conclusion.

NIOSH disagreed with OSHA, and up to this time, it has been the practice of NIOSH to assume that the RFD describes the size and shape of fibers that correlate with their potential to cause human disease⁵. The RFD was also recently applied by EPA in the El Dorado Hills, CA, study. It is clear that there is disagreement within the regulatory community of the appropriateness of the RFD in the protection of health.

NIOSH has just opened this question for study.⁶ This year, NIOSH issued a White Paper outlining in detail a research agenda to examine this question and held public hearings on it last month. The adverse health effects of asbestos are widely known and, with the exception of the differences between chrysotile-asbestos and amphibole-asbestos, are not in dispute. What the NIOSH White Paper addresses is the need to examine the health effects of nonasbestos particles that meet the RFD.

While the NIOSH White Paper does not provide evidence that challenges OSHA’s 1992 decision, it calls for study of the issue, including, animal inhalation studies, epidemiological studies of miners, and cell culture studies. These are necessary before the health effects of nonasbestos particles that meet the RFD can be understood fully.

Why is this issue still in debate after the 1992 OSHA decision? Partly, I believe, that it comes from 1) lack of knowledge about the nature of asbestos, 2) acceptance of the hypothesis that **only** the size, shape, and durability of mineral particles affect their carcinogenic potential, and 3) a reluctance to change.

⁴ OSHA, 1992

⁵ NIOSH, 2007

⁶ NIOSH, 2007

THE NATURE OF ASBESTOS

Asbestos is unusual.⁷ It is a mineral habit, like snow and hail are habits of ice. Habit is a form of “growth”.

Asbestos grows as bundles of single fibers, (referred to as fibrils), that are easily separated from each other by hand pressure. The geologic environment that enables asbestos to form is limited and involves the presence of warm, water-rich conditions and open underground spaces.

Fibrils have narrow widths and extraordinary tensile strength imparted to them by their strong outer layers. They are difficult to break and their strength makes them flexible and almost impossible to grind. They are able to enter the body because of their narrow widths and they are retained because their lengths (as much as several hundred micrometers) thwart the body’s mechanisms to remove them.

Asbestos can form from a number of different minerals. A mineral name implies **only** a particular atomic arrangement of a fixed set of elements in particular proportions. Mineral names are not synonyms for asbestos, just like ice is not a synonym for snow although snow is made of ice. To specify asbestos, the mineral name is followed by the term asbestos, e.g., tremolite-asbestos. Two forms of asbestos have a specific name, e.g., crocidolite is riebeckite-asbestos, and amosite is cummingtonite-grunerite asbestos.

The dimensions of asbestos fibrils found in occupational air and in the lung of asbestos workers are published in the literature, providing the basis for a **dimensional definition of asbestos fibers**. Although accurate dimensional definitions of asbestos may have been unnecessary in monitoring asbestos factories, mills and mines where what was in the air was only asbestos, they are essential in a mixed dust environment, essential when dealing with environmental exposures, and essential if asbestos were to be banned in the United States

Published data on the width of asbestos fibers found in bulk samples, on air monitoring filters, and in lung tissue show that asbestos is composed of mineral fibrils that are less than 1 micrometer in width.⁸ Fibrils wider than 1 micrometer are brittle (lack tensile strength) and cannot be used as asbestos.⁹ The widths vary somewhat within and among asbestos deposits, but the range is narrow. The dimensions of the most abundant forms of asbestos are similar: crocidolite fibrils are about 500 to 2000 Å in width, amosite and anthophyllite-asbestos are about 2000 to 10,000 Å in width, and chrysotile-asbestos is about 200-650 Å.¹⁰

⁷ Wylie, 1979, 1993, 1988; Verkouteren and Wylie, 2002

⁸ Wylie et al., 1993

⁹ See Zoltai, 1981, for an excellent discussion.

¹⁰ Polygonal serpentine fibers may have diameters up to 10,000 Å. Baronnet and Devouard, 2005.

Other types of asbestos have equally narrow widths. Actinolite-asbestos has fibril widths of 600-2000 Å and tremolite-asbestos fibrils range from about 2000 to 6000 Å. At Libby Montana, mean widths are about 5000Å and the range is 2000 to about 10,000Å.¹¹

Studies of the lung burden of asbestos workers also report very narrow fibers. Martha Warnock measured 3723 fibers from lung tissue from 27 mesothelioma cases and identified them as crocidolite, tremolite-asbestos, anthophyllite-asbestos, actinolite-asbestos, chrysotile-asbestos, amosite, or other by TEM. More than 60% of the fibers are either amosite or chrysotile-asbestos. The mean width of the entire population was 2600 Å; for amosite it was 2300 Å and for chrysotile-asbestos, 600 Å. Similar dimensions were observed by Warnock in asbestosis and lung cancer cases.¹²

The width of asbestos fibers is independent of length.¹³ Width is the same no matter how long the fibers because width is an independent characteristic imparted during the “growth” of the fibers.

Berman et al.¹⁴ extensive and careful evaluation of the 13 different rat experiments conclude that the fibers that contribute to tumor risk are <4000Å in width or they are bundles and aggregates of such fibers. Stanton and others also find that fibers less than 5000 or less in width are most likely to be carcinogenic. The NIOSH White Paper states: “Fibers and particles with diameters less than 0.5µm (5000 Å) are more likely to cross membranes and translocate to pleural and peritoneal spaces and are more likely to enter the lymphatic and circulatory systems.” Thus, not only is the width of asbestos a defining characteristic, it is key to its carcinogenicity.

Cleavage fragments are different. Cleavage fragments, formed by crushing rock, get wider as they get longer and width is therefore dependent on length¹⁵. They do not possess the asbestos characteristic of high tensile strength and their surfaces are different in fundamental ways. While a 40 micrometer asbestos fiber could easily have a width of 0.2 micrometers, such dimensions could never be formed by breakage and no cleavage fragments have such dimensions.

SIZE AND SHAPE HYPOTHESIS

The hypothesis that only dimensions and durability (biopersistence) determine a mineral particles potential to cause mesothelioma, lung cancer, laryngeal cancer, and asbestosis is known as the Stanton Hypothesis. It was based on a large number of experiments in which Stanton and coworkers at the NCI implanted a number of different fibrous materials in rats.¹⁶ They found that the number of long thin fibers highly correlated with

¹¹ Wylie et al., 1993

¹² Warnock, 1989

¹³ Siegrist and Wylie, 1980

¹⁴ 1995

¹⁵ Siegrist and Wylie, 1980

¹⁶ Stanton et al., 1981

the sarcomas that developed after implantation. Other researchers have found similar results¹⁷.

If the Stanton Hypothesis is correct, then any biopersistent particle that has the dimensions of real asbestos should have the same carcinogenic potential as asbestos. In fact, we know that this is often the case for asbestiform fibers. Long thin fibers of erionite, a mineral not regulated as asbestos, are thought to be responsible for a high incidence of mesothelioma among several small villages in Turkey.¹⁸ Furthermore, the long, thin fiber (not specifically regulated as asbestos by the federal government) from Libby, Montana, has been identified as the agent in a number of mesothelioma cases among those occupationally exposed¹⁹.

However, we also know from the experience of miners exposed to other durable long, thin fibers such as fibrous talc²⁰ that all durable long, thin fibers are not the same. Many studies have shown the importance of the surface in the biological activity of mineral fibers.²¹ Understanding the basis of the carcinogenicity of mineral fibers requires further study.

Can the Stanton Hypothesis be used to justify concern for nonasbestos, durable, RFD particles? If the RFD corresponds to a high carcinogenic potential, then many mineral particles would be potential carcinogens. Many common durable minerals break into elongated particles that conform to the RFD even though they are not asbestiform and do not have the dimensions of asbestos fibers. These include pyroxenes, feldspars, zeolites, some sheet silicates, and many other mineral groups. In fact, the Appalachian and Rocky Mountain Chains contain abundant minerals that would form particles meeting the RFD when crushed.

What does the epidemiology tell us? The studies that have examined the epidemiology of workers exposed to dusts that contain nonasbestos amphibole particles that meet the RFD have found no asbestos-related diseases. Amphiboles make up 5% of the Earth's crust and, although a large group of minerals of variable chemical composition²², most amphibole fragments exceed 3:1 in length to width ratio if they are longer than 5 micrometers. These studies include miners and millers from a talc mine in New York, gold miners from Lead, South Dakota; vermiculite workers at Enoree, South Carolina; and iron miners from the Minnesota taconite iron district.²³

¹⁷ Bertrand, and Pezerat, 1980, Davis et al., 1991, Smith et al., 1979, Pott et al., 1974

¹⁸ Baris, 1987, Wagner et al., 1985

¹⁹ Amandus et al., 1987; Sullivan, 2007.

²⁰ IARC, in press; Honda et al., 2002; Gamble, 1993; Stille and Tabershaw, 1982

²¹ For example: Chamberlain and Brown, 1978; Feuerbacher et al., 1980; Flowers, 1980; Marchisio and Pernis, 1963; Schlipkoter et al., 1963; Brown et al., 1990; Weitzman and Graceffa, 1984; Weitzman and Weitberg, 1985; Hochella (1993) provides an excellent discussion of the variability of surface chemistry, structure and reactivity of mineral surfaces that may affect biological activity.

²² Leake et al., 1997, 2004

²³ McDonald et al., 1988, McDonald et al., 1978, Brown et al., 1986, Higgins et al., 1983, Cooper et al., 1992, Honda et al., 2002, Gamble, 1993, Steeland and Brown, 1995, Stille and Tabershaw, 1982

Asbestos fibers **do** meet the RFD. They exceed the 3:1 length to width ratio. But because of their narrow widths, they also exceed a 5:1 and a 10:1 and most exceed a 20:1 ratio. Therein lays the problem. While asbestos fibers conform to the RFD, they are not DEFINED by it, and they cannot be separated from other mineral particles by it. While we know that it is very likely that among amphiboles it is the size and shape that affects their carcinogenicity, the question is “What size and what shape?”

RELUCTANCE TO CHANGE THE REGULATORY FIBER DEFINITION

Neither OSHA nor MSHA consider cleavage fragments to be asbestos. NIOSH has put the issue up for discussion. It is time for this issue to be resolved.

CONCLUSIONS

I conclude by asking you to support the work that NIOSH has proposed to address unanswered questions about the carcinogenicity of nonasbestos mineral particles. I also ask that the National Institute of Standards and Technology (NIST) be funded to develop new analytical methods for identifying and monitoring asbestos, and that NIEHS fund a comprehensive risk assessment. At the present time, these issues are being decided in the courts, not the appropriate venue for scientific discourse.

References.

Addingley, C.F., 1966, Asbestos dust and its measurement. Annals of Occupational Hygiene, v.9, p.73-82.

Baronnet, A., and Devouard, B., 2005, Microstructures of common polygonal serpentines from axial HRTEM imaging, electron diffraction and lattice-simulation data, Canadian Mineralogist, v.43, p.513-542.

Baris, Y.I., 1987, Asbestos and erionite related chest diseases. Publication Somih Ofset Matbaackilik Limited Company, Ankara-Turkey.

Berman, D.W., Crump, K.S., Chatfield, E.J., Davis, J.M. G., and Jones, A.D., 1995, The sizes, shapes and mineralogy of asbestos structures that induce lung tumors or mesothelioma in AF/HAN rats following inhalation, Risk Analysis, v. 15, p. 181-195.

Bertrand, R., and Pezerat, H., 1980, Fibrous Glass: Carcinogenicity and Dimensional characteristics in Biological Effects of Mineral Fibres, Wagner, J.C. Ed., IARC Scientific Publications p.901-911.

Brown, D.P., Kaplan, S.D., Zumwalde, R.D., Kaplowitz, M., and Archer, V.E., 1986, Retrospective cohort mortality study of underground gold mine workers. In Silica, Silicosis, and Cancer, D.F. Goldsmith, D.M. Winn and C.M. Shy, Eds., Praeger Publishers, New York, p.335-350.

Brown, G.E., Carthew, P., Hoskins, J.A., Sara, E., and Simpson, C.F. 1990, Surface modifications can affect the carcinogenicity of asbestos, Carcinogenesis, v. 11, p.1883-1885.

Chamberlain, M., and Brown, R.C., 1978, The cytotoxic effects of asbestos and other mineral dusts in tissue culture cell line, British Journal of Experimental Pathology, v.59, p. 183-189.

Cooper, W.D., Wong, O., Trent L.S., and Harris, F., 1992, An updated study of taconite miners and millers exposed to silica and non-asbestiform amphiboles. Journal of Occupational Medicine, v.34, p. 1173-1180.

Davis, J.M.G., Addison, J., McIntosh, C., Miller, B. G., and Niven, K., 1991, Variations in the carcinogenicity of tremolite dusts samples of differing morphology, Annals of the New York Academy of Sciences, v.643, p.473-490.

Feurenbacher, D.G., Dimataris, G.T., Mace, M., L., Marshall, M.V., and McLemore, T.L., 1980, Comparative cytotoxicity of mutagenicity of organosilane reacted chrysotile asbestos (Abstract) Clay Mineral Society Annual Meeting, p. 34.

Flowers, E.S, chemical detoxification of asbestos fibers, in Proceedings, National Workshop on Substitutes for Asbestos, A. Levin and H. Allsbury, eds, EPA-560/3-80-001, Washington D.C., p. 489-496.

Gamble, J.F, 1993, A nested case control study of lung cancer among New York talc workers, International Archives of Occupational and Environment Health, v.64, p.449-456.

Higgins, I.T.T. Glassman, J.H., Oh, M.S. and Cornell, R.G., 1983, Mortality of Reserve mining Company employees in relation to taconite dust exposure, American Journal of Epidemiology, v. 118, p.710-719.

Hochella, M.f., 1993, Surface Chemistry, structure, and reactivity of hazardous mineral dust, in Health Effects of Mineral Dusts, Guthrie, G.D., and Mossman, B.T. eds, Reviews in Mineralogy V.28, Mineralogical Society of America, p.275-308

Honda, Y., Beall, C., Delzell, E., Oestenstad, K., Brill, I., and Mathews, R., 2002, Mortality among Workers at a Talc Mining and Milling Facility. Annals of Occupational Hygiene, v.46, p.575-585.

Hume, L.A. and Rimstidt, J.D., 1992, The biodurability of chrysotile asbestos, American Mineralogist, v.77, pp1125-1128.

International Agency for Research on Cancer (IARC), in press, Report of the Workgroup on Talc, Carbon Black and Titanium Dioxide, Lyon, France.

Leake, B.E. et al. 1997, Nomenclature of amphiboles: Report of the subcommittee on Amphiboles of the International Mineralogical Association, Commission on new Minerals and Mineral Names, Canadian Mineralogist, v.35, p.219-246.

Leake B.E. et al, 2004, Nomenclature of amphiboles: Additions and revisions to the International Mineralogical Association's amphibole nomenclature, American Mineralogist, v. 42, p.883-887.

Leidel, N.A., Bayer, S.G., Zumwalde, R.E., and Busch, K.A., 1979, Membrane filter method for evaluating airborne asbestos fibers, US Department of Health Education and Welfare, NIOSH, USPHS/IOSH technical report n. 79-127.

Lynch, J.R., Ayer, H.E., and Johnson, D.L., 1970, The interrelationships of selected asbestos exposure indices. American Industrial hygiene Journal, v.31, p. 598-604.

Marchisio, M.A. and Pernis, B., 1963, The action of vinylpyridine-polymers on macrophages cultivated in vitro in presence of tridymite dust, Grundfragen Silikoseforsch v. 6, p. 245-247.

McDonald, J.D., Gibbs, G.W., Liddell, F.D.K., and McDonald, A.D., 1978, Mortality to cummingtonite-grunerite. American Review of Respiratory Disease, v. 118, p. 271-277.

McDonald, J.C., McDonald, A.D., Sebastien, P., and Moy, K, 1988, Health of vermiculate miners exposed to trace amounts of fibrous tremolite. British Journal of Industrial Medicine, v.45, p. 630-634.

National Institute of Occupational Safety and Health (NIOSH), 2007, Asbestos and Other Mineral Fibers: A Roadmap for Scientific Research, February 2007 draft, presented at Public Meeting, Washington , D.C, May 4, 2007, 47 pages.

Occupational Safety and Health Administration (OSHA), 1992, Final Rule: Occupational exposure to asbestos, tremolite, anthophyllite and actinolite, Federal Register 57(110), p. 24310-24331.

Pott, F., Huth, F., Friedrichs, K.H., 1974, Tumorigenic effects of fibrous dusts in experimental animals, Environmental Health Perspectives, v.9., p. 313-315.

Schlipkoter, H.W., Dolgner, R., and Brockhaus, A., 1963, The treatment of experimental silicosis, German Medical Month, v. 8, p.509-514.

Siegrist, H.G. and Wylie, A.G., 1980, Characterizing and discriminating the shape of asbestos particles, Environmental Research v.23, p.348-361

Smith, W.E., Hubert, D., Sobel, H., and Marquet E., 1979, Biologic tests of tremolite in hamsters, Dusts and Disease, p.335-339.

Stanton, M., Layard, M., Tegeris, A., Miller, E., May, M., and Morgan, E., 1981, Relation of Particle dimension to carcinogenicity in amphibole asbestos and other fibrous minerals, Journal of the National Cancer Institute v.67, p.965-975.

Steeland, K., and Brown, D., 1995, Mortality study of gold minerals exposed to silica and nonasbestiform amphibole minerals: an update with 14 more years of follow-up, American Journal of Industrial Medicine, v.27, p.217-229.

Stille, W.T., and Tabershaw, I.R., 1982, the mortality experience of upstate New York talc workers, Journal of Occupational Medicine v.24, p.480-484.

Sullivan, P.A., 3 January 2007, Vermiculite, Respiratory Disease and Asbestos Exposure at Libby Montana: Update of a Cohort Mortality Study, Environmental Health Perspectives, doi:10.1289/eph.9481 at <http://dx.doi.org/>. 38 pages.

Weitzman S.A., and Weitberg, A.b., 1985, Asbestos-catalyzed lipid peroxidation and its inhibition by deferoxamine, Biochemical Journal, v.225, p. 259-262.

Verkouteren, J.R. and Wylie, A.G., 2002, Anomalous optical properties of fibrous tremolite, actinolite, and ferro-actinolite, American Mineralogist v.87, p. 1090-1095.

Virta, R.L., Shedd, K., Wylie, A.G. and Snyder, J.G., 1983, size and Shape Characteristics of amphibole asbestos(Amosite) and amphibole cleavage fragments (actinolite, cummingtonite) collected on occupational air monitoring filters. Aerosols in mining and Industrial Work Environments, v.2, p. 633-643.

Wagner, J.C., Berry, G. and Skidmore, J.W., 1976, Studies on the carcinogenic effects of fiber glass of different diameters following intrapleural inoculation in experimental animals, NIOSH 76-151, p.193-197.

Wagner, J.C., Skidmore, J.W., Hill, R.J., and Griffith, D.M., 1985, Erionite exposure and mesothelioma in Rats, British Journal of Cancer, v.51, p. 727-730.

Warnock, M.D., 1989, Lung Asbestos burden in shipyard and construction workers with mesothelioma: comparison with burdens in Subjects with asbestosis or lung cancer. Environmental Research, v.50, p. 68-85.

Weitzman, S.A., and Graceffa, P., 1984, Asbestos catalyzes hydroxyl and superoxide radical generation from hydrogen peroxide, Archives of Biochemistry and Biophysics, v. 228, p. 373-376.

Werner, A.J., et al., 1995, Asbestiform riebeckite (crocidolite) dissolution in the presence of Fe chelators: implications for mineral-induced disease. American Mineralogist, v.80, p. 1093-1103.

Wylie, A. G., 1979, Optical Properties of the Fibrous Amphiboles, Annals of the New York Academy of Sciences, v.330, p. 611-612.

Wylie, A.G., 1988, Relationship between the growth habit of asbestos and the dimensions of asbestos fibers, Mining Engineering, p. 1036-1040.

Wylie, A. G., 1993, Modeling asbestos populations: a fractal approach, Canadian Mineralogist, v.30, p. 437-446.

Wylie, A. G. and Verkouteren, J.R., 2000, Amphibole asbestos from Libby, Montana: Aspects of nomenclature. American Mineralogist v. 85, p. 1540-1542.

Wylie, A.G., Bailey, K.F. Kelse, J.W., and Lee, R.J., 1993, The importance of width in asbestos fiber carcinogenicity and its implications for public policy, American Industrial Hygiene Association Journal v. 54, p. 239-252.

Yada, K., 1967, Study of Chrysotile asbestos by a high resolution electron microscopy, Acta Crystallographica, v.23²⁴, p.704-707.