Extended Comments by Richard A. Lemen, Ph.D., M.S.P.H. to the United States Senate Subcommittee on Environment and Public Works, United States Capitol, Washington, DC, June 12, 2007.

I would like to thank Chairman Boxer, Ranking Member Inhofe and the entire EPW Committee for the honor and opportunity to testify today.

My name is Dr. Richard A. Lemen. I am retired from the United States Public Health Service where I was an Assistant Surgeon General of the United States. At the time of my retirement I was also Deputy Director and had been Acting Director of the National Institute for Occupational Safety and Health (NIOSH). I have spent my entire career, since 1970, studying the epidemiology of asbestos-related diseases and have conducted numerous epidemiology studies, written many scientific papers, advised the World Health Organization, various other National governments, and have testified before the United States Congress on several occasions concerning the health risks from exposure to asbestos. I am an adjunct professor of environmental and occupational medicine at Emory University and a donsultant in occupational health and epidemiology. I also testify in asbestos-related litigation on behalf of plaintiffs. My CV, which I have supplied the Committee, will give you further information concerning my studies on asbestos.

Often asbestos is referred to as the "magic Mineral" having at least 3000 or more uses, such as being woven into cloth, with vegetable fibers; for wrapping the corpuses, referred to by Pliny as the funeral dress of kings prior to cremation in order to help collect the ashes; in making clay pots some 4000 years ago; and was even mentioned by Marco Polo, during his travels to the far east, where he found it called "salamander" skin

which was mined from the mountains, extracted then crushed, by subjects of the Great Khan, into a fibrous like wool that was then spun and made into cloth of which some were used for table cloths, that when soiled, were thrown into the fire and came out "white as snow" for use again; one was sent to the Pope, in Rome, "in which cloth he keeps the Sudarium of our Lord." Benjamin Franklin even bought a purse from the "northern part of America" made from woven asbestos.[1]

Our modern knowledge of asbestos usage and asbestos-related disease began in the early 1900s, with reports of lung diseases among asbestos workers in the United Kingdom as well as the United States. By 1930, the disease asbestosis was well established as a lung disease contracted from exposures to asbestos. Unfortunately, by the mid-1930s it was suspected that, in addition to asbestosis, cancer may also result from exposure to asbestos. Today we know that various cancers, including lung cancer, gastrointestinal cancers, and mesothelioma are all causally associated from exposure to asbestos. We know that all forms of commercially used asbestos, including chrysotile, as well as the amphiboles cause all of the asbestos-related diseases including asbestosis, lung cancer, mesothelioma and gastrointestinal cancers.[2]

Asbestosis is a progressive disease which can eventually result in death after much disability and suffering, even after occupational exposures have ceased. Asbestosis does not respond to medical treatment, only palliative care can be given.[3]

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[1] Lemen, RA, 2005. Epidemiology of Asbestos-Related Diseases and the Knowledge that Led to What is Known Today. In: ASBESTOS Risk Assessment, Epidemiology, and Health Effects, Eds. RF Dodson, SP Hammar. CRC Taylor & Francis, 201-308.

[2] Lemen, RA, 2005. Epidemiology of Asbestos-Related Diseases and the Knowledge that Led to What is Known Today. In: ASBESTOS Risk Assessment, Epidemiology, and Health Effects, Eds. RF Dodson, SP Hammar. CRC Taylor & Francis, 201-308.

[3] ATSDR, 2001. Agency for Toxic Substances and Disease Registry Questions and Answers Exposure to Asbestos. Department of Health and Human Services, Atlanta, GA, July 26.

Asbestos-induced cancers are not confined to just the workers exposed at work, but asbestos exposures can be brought home to family members, as a result of contamination of their work clothes, prompting asbestos-induced disease in them as well. Asbestos-related diseases can also occur to residents living near asbestos sources.[4]

In the United States it is estimated that between 189,000 and 231,000 deaths have occurred since 1980 due to workplace exposure to asbestos. Another 270,000 to 330,000 deaths are expected to occur over the next 30 years and for those workers exposed, over a working lifetime, to the current Occupational Safety and Health administration (OSHA) standard of 0.1 fibers/cc - 3.4/1000 workers are estimated to die as a result of asbestos-related diseases.[5] A more recent study suggested the use of linear extrapolation, as used by OSHA, from high exposure levels may underestimate the risks at low doses (Gustavsson et al., 2002).[6] Unless asbestos use in the United States is not banned there is no end of its ability to exposure workers and consumers to its dangers.

Products containing asbestos can still be found in things found in the home such as lamp sockets, floor tiles, cat box fill, braking mechanism in washing machines and cars, furnaces, and other products. Because these products are not only manufactured by workers, but are also used, maintained, and repaired by workers – they (workers) suffer additional

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[4] NIOSH, 1995. Report to Congress on Workers' Home Contamination Study Conducted Under The Workers' Family Protection Act (29 U.S.C. 671a). U.S. Department of Health and Human Services, Public Health Service, Centers For Disease Control And Prevention, National Institute for Occupational Safety and Health (NIOSH), Cincinnati, OH 45226, September. See sections on Asbestos p. 6-11; 45-46; 55; 62-63; 86-87; tables 2-6 (pp. 145-159).

[5] OSHA, 1986. OSHA, 1986. Final Rule: Asbestos. 51 FR 22612. U.S. Department of Labor. Occupational Safety and Health Administration, Washington, D.C., June 20.

[6] Glustavsson P, Nyberg F, Pershagen G, Schéele P, Jakobsson R, Plato N, 2002. Low-dose exposure to asbestos and lung cancer: Dose-response relations and interaction with smoking in a population-based case-referent study in Stockholm, Sweden. Am J Epi, Vol. 156 (11); 1016.

exposure from consumer products as do the consumers using these products.

The most recent Criteria Document from the World Health Organization's (WHO) International Programme for Chemical Safety (IPCS) states in 1998 that no threshold has been identified for carcinogenic risks to chrysotile asbestos.[7] Chrysotile is the main commercially used asbestos in the World. This 1998 WHO statement is consistent with the WHO's earlier conclusion in 1989 *"[T]he human evidence has not demonstrated that there is a threshold exposure level for lung cancer or mesothelioma, below which exposure to asbestos dust would not be free of hazard to health"*.[8] The WHO recognizes what NIOSH concluded 31 years ago, in 1976, that ". . . (only a ban can assure protection against carcinogenic effects of asbestos)".[9] I cannot tell any of you, on this Committee, why some will develop asbestosis or other asbestos-related cancers and why others won't. But what I can tell you is that asbestos-induced diseases are preventable. Each and every one!

The first criteria document from the newly formed NIOSH of 1970, was on asbestos, after NIOSHs first Director Dr. Marcus Key had sent a letter to OSHA stating the inadequacy of OSHAs new start-up standard for asbestos, based on the then ACGIH TLV®. NIOSH was the first federal agency to call for a ban on asbestos in its 1976 Revised Criteria Document. NIOSH has maintained this position to the present, while suggesting in the interim that the only reliable and practical analytical method, in 1976, was 0.1 fiber/cc using the NIOSH

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[7] IPCS, 1998. **Environmental Health Criteria 203: Chrysotile Asbestos**, International Program on Chemical Safety, World Health Organization.

[8] WHO, 1989. Occupational Exposure Limit for Asbestos. WHO/OCH/89.1, Office of Occupational Health, World Health Organization, Geneva.

[9] NIOSH, 1976. Revised Recommended Asbestos Standard. DHEW (NIOSH) Publication No. 77-169. U.S. Department of Health, Education, and Welfare. Public Health Service. Centers for Disease Control. National Institute for Occupational Safety and Health. December.

Phase Contrast Method (PCM) 7400 asbestos analytical method. Unfortunately chrysotile cannot be seen in the light microscope when it occurs in the fibril form and thus most chrysotile is not counted in an air sample using a NIOSH 7400 count scheme-diameter resolution of approximately 0.25 microns where as most individual fibers of crocidolite and chrysotile are 0.02-0.05 microns in diameter. OSHA describes the advantages and disadvantages of the Phase Contrast Microscope (PCM) as can be seen in the footnote.[10]

Any definition of asbestos should include all respirable asbestiform fibrous minerals, including fibrous cleavage fragments which are respirable.[11] This should only be changed if there exist irrefutable data, both human and animal, showing the safety of any such fibrous mineral being excluded. Valid methodologies now exist to sample for all size fibers, including those less than 5 um in length, not currently

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[10] Rules and regulations-Dept Labor-OSHA 29 CFR Parts 1910, 1915, 1926-Occupational Exposure to Asbestos- Final rule-Aug 10, 1994

59FR4096
"1.3 Advantages and Disadvantages
There are four main advantages of PCM over other methods:

(1) The technique is specific for fibers. Phase contrast is a fiber counting technique which excludes non-fibrous particles from the analysis.
(2) The technique is inexpensive and does not require specialized knowledge to carry out the analysis for total fiber counts.
(3) The analysis is quick and can be performed on-site for rapid determination of air concentrations of asbestos fibers.
(4) The technique has continuity with historical epidemiological studies so that estimates of expected disease can be inferred from long-term determination of asbestos exposures.

41066 The main disadvantage of PCM is that it does not positively identify asbestos fibers. Other fibers which are not asbestos may be included in the count unless differential counting is preformed. This requires a great deal of experience to adequately differentiate asbestos from non-asbestos fibers. Positive identification of asbestos must be performed by polarized light or electron microscopy techniques. A further disadvantage of PCM is that the smallest visible fibers are about 0.2µm in diameter while the finest asbestos fibers may be as small as 0.02µm in diameter. For some exposures, substantially more fibers may be present than are actually counted."

[11] Dement J M, Zumwalde RD, Gambel JF, Fellner W, DeMeo MJ, Brown DP, Wagoner JK, 1980. Occupational exposure to talc containing asbestos-Morbidity, Mortality, and environmental studies of miners and millers. NIOSH Technical Report-DHEW (NIOSH) Publication No. 80-115, Feb.

addressed in regulatory standards. These smaller fibers should be included in any asbestos definition. Both animal and human data support such an inclusion as can be seen by the attached Appendix - 1.[12]

Federal and State governments should work together to address, refine, and/or develop surveillance of fiber-related diseases, including those from asbestos. For example it is well known that the National Cancer Institutes Surveillance Epidemiology and End Results (SEER) data base underreports mesothelioma.[13] NIOSH should be funded to continue its Respiratory Disease Surveillance System and should assure that other NIOSH surveillance systems become more comprehensive and inclusive. None of the systems should rely solely on Proportionate Mortality/Morbidity Analysis for determining mortality or morbidity data, as this type analysis underreports low incidence diseases, albeit important diseases i.e. mesothelioma.

Research should determine how much of background mesothelioma and other asbestos-related diseases are related to the increased consumption of asbestos in any reference populations used for comparison and thus adjust expected rates accordingly in order to determine the true risk of asbestos-related diseases.

Epidemiology literature on all fibrous materials, not just those related to the currently regulated asbestiform fiber types should be reviewed and new research conducted when necessary. Such research should address all respirable fiber types and all size parameters of a respirable nature, including short respirable fibers less than 5 microns in length.

Since biopersistence has been used as a surrogate for exposure and fiber type of exposure through identifying their persistence in the lung as a critical factor in causation,

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[12] See Appendix 1 - Short Fibers, Richard A. Lemen, Ph.D.

[13] See Appendix 2 – Mesothelioma Surveillance, Richard A. Lemen, Ph.D.

toxicological studies should evaluate whether the external airborne concentrations of fibers are actually representative of the fiber concentrations and morphologies once the fibers have been inhaled into the lung. Data suggest that the correlation of breathing zone samples of chrysotile may not represent the actual fiber concentration of chrysotile fibers once in the lung as they break apart from fiber bundles and multiply within the lung, while the amphiboles do not.[14] This is important not only as it means a higher dose of chrysotile within the lung but a higher number of fibers that can translocate from the lung to other parts of the body, such as the pleura. Because dose plays a significant role in the toxicity of chrysotile as compared to amphiboles such findings would be important in determining the actual role of chrysotile in asbestos-related diseases such as mesothelioma. Translocation of chrysotile asbestos from the lung indicates a specific role for chrysotile in the etiology of mesothelioma since the chrysotile fibers reach the areas where the tumor develops. Mesotheliomas develop in the pleura, peritoneum and other serosal surfaces of the body. It is universally accepted that chrysotile is a cause of cancer in the lung and migrates to and is concentrated in the pleura[15]. Since chrysotile is carcinogenic and is present in high concentrations in the pleura where the mesothelioma is induced, it is biologically plausible that it causes or contributes to the cause of mesothelioma. This is also shown by many mechanistic and molecular studies that

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[14] Bellman B, Muhle H, Pott F, Konig H, Kloppeel H, Spurny K, 1987. Persistence of man-made fibers (MMF) and asbestos in rat lungs. Annals of Occup Hyg, 31: 693-709.

[15] Suzuki, Y. & Kohyama, N., 1991. Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues. Am J Ind Med, Vol. 19, p. 701-704; Kohyama, N. & Suzuki, Y., 1991. Analysis of asbestos fibers in lung parenchyma, pleural plaques, and mesothelioma tissues of North American insulation workers. Ann N Y Acad Sci, Vol. 643, p. 27-52; Suzuki, Y., Yuen, S., Ashley, R. & Calderaro, A., 1998. Asbestos fibers and human malignant mesothelioma. Advances in the Prevention of Occupational Respiratory Diseases, Eds. Chiyotani, K., Hosoda, Y., & Aizawa, Y., Elsevier Science B.V., p.709 and Sebastien, P., Janson, X., Gaudichet, A., Hirsch, A. & Bignon, J., 1980. Asbestos retention in human respiratory tissues: comparative meas urements in lung parenchyma and in parietal pleura. IARC Sci Pub, Vol. 30, p. 237-246; Dodson RF, Graef R, Shepherd S, O'Sullivan M, Levin J, 2005. Asbestos burden in cases of mesothelioma from individuals from various regions of the United States. Ultrastruct Pathol. Sep-Oct;29(5):415-33.

indicate how chrysotile may cause mesothelioma. Fiber penetration can rearrange the cytoskeletal apparatus of the cell and this could indicate an interaction between the chrysotile fibers and the normal mitotic process, since giant multinucleated cells are formed. These studies indicate that chrysotile penetrates the cell, enters the nucleus and induces abnormal chromosome formations in dividing cells.[16] Some of these abnormalities include the deletion of the P53 gene that controls cell growth.[17]

Additional research should include evaluation of the synergistic effects between amphibole and serpentine fiber exposures, since it is highly unlikely that uncontaminated serpentine exposures exist in occupational and environmental settings. To date such findings have suggested such a synergistic action between the mixed fiber types.[18] It has been suggested by some that the fibrous tremolite contamination of chrysotile, usually less than 1%, is the cause of mesothelioma among predominately chrysotile exposed persons.[19] New evaluation of the South Charleston chrysotile exposed population of textile workers has confirmed a dose-response relationship between asbestosis and lung cancer.[20] This is important as entities suggesting that chrysotile is the "safe asbestos" are basing their conclusions on only one outcome,

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[16] Malomi, W., Loai, F., Falchi, M., and Donnelli, G., 1990. On the mechanism of cell internalization of chrysotile fibers: An immunocytochemical and ultrastructural study. Environmental Research, Vol. 52, No. 2, pages 164-177.

[17] Levresse, Renier, Fleury-Feith, Levy, Moritz, Vivo, Pilatte, Jaurand, 1997. Analysis of Cell Cycle Disruptions in Cultures of Rat Pleural Mesothelial Cells Exposed to Asbestos Fibers. Am J Respir Cell Mol Biol, 17: 660-671.

[18] Nicholson WJ, Landrigan PJ, 1994. The carcinogenicity of chrysotile asbestos, In : The Identification and Control of Environmental and Occupational Diseases : Asbestos and Cancer. Eds. M Mehlman, A Upton: Princeton Scientific Publishing Co., Inc. Vol XXII; Acheson ED, Gardner MJ, 1979. Mesothelioma and exposure to mixtures of chrysotile and amphibole asbestos.

[19] McDonald J.C., McDonald AD, Chrysotile, Tremolite and Mesothelioma. Letter published in Science, 10 Feb 1995, Vol. 267:775

[20] Hein MJ, Stayner L, Lehman E, Dement JM, 2007. Follow-up study of chrysotile textile workers : cohort mortality and exposure-response. Occup Environ Med (published online 20 Apr. 2007), 031005.

that being mesothelioma. While it is generally recognized that chrysotile on a dose-by-dose basis is less potent than the amphiboles in producing mesothelioma; this does not appear the case in its ability for causing other asbestos-induced disease. Therefore, future research should continue to look at all asbestos-induced diseases when determining recommended regulatory actions for the prevention of asbestos-related diseases.

The current OSHA regulations govern exposure to minerals defined in the regulations as asbestos; however, formations that contain tremolite asbestos also have tremolite cleavage fragments. Thus, just because the cleavage fragments are not covered under the current OSHA regulations, as regulated fibers, does not mean that they are biologically inactive. The emphasis of the fiber pathogenicity being related to the fact that any asbestos structure is a fiber is only one explanation of how it causes disease. The fact is that the non-asbestiform cleavage fragment is an analog of the fibrous asbestos structure and is chemically made of the same composition. The complexity of asbestos induced lung disease/injury includes a wide array of issues other than just physical features (Kamp and Wiseman, 1999).[21]

Next I will provide some data which may shed more light on the arguments for including a broader fiber definition when it comes to materials contaminated with asbestos. As former Deputy and Acting Director of NIOSH I know the agency has been dealing with the issue of talc contaminated with fibrous asbestos for many years. Researchers found among miners and millers from two counties in Northern New York eight talc miners identified as having mesothelioma and now Hull, Abraham and Case (2002) have added five new cases.[22] Rohl and Langer (1974) have stated "Talc because of its composition, conditions of formation and geological

[21] Kamp DW, Weitzman SA, 1999. The molecular basis of asbestos induced lung injury. Thorax.54:638-652

[22] Hull MJ, Abraham JL, Case BW, 2002. Mesothelioma among workers in asbestiform fiber-bearing talc mines in New York State Ann Occ Hyg, 46, (Supplement 1):132-135

occurrence, is frequently contaminated with asbestos fibers."[23] "The data, however, support earlier studies that indicate that talc miners and millers experience excess parenchymal fibrosis and pleural changes. The data also suggest that individuals in the paper industry and construction trades may be at risk."[24]

Dement et al., in 1980 found from one mine and mill, reported by the company to be producing non-asbestiform talc, air samples of 5 fibers/cc as time weighted averages (TWA) in six job categories containing 48% mineral talc, 37-59% tremolite, 4.5-15% anthophyllite, and 10-15% serpentine, lizardite, antigorite. Thus the TWA exposures to asbestiform amphiboles (anthophyllite and tremolite) were found to be in excess of the present U.S. Occupational Safety and Health (OSHA) and Mine Safety and Health Administration (MSHA) occupational exposure standards. They also found that in many mine and mill operations more than 90 percent of the total airborne fibers were less than 5μm in length. They found asbestiform tremolite, anthophyllite and in a couple of samples chrysotile and found they were fibers when using Analytical Transmission Electronic Microscope (ATEM) as well as PCM and not cleavage fragments.[25]

I recommend that that all fibrous asbestiform minerals and that all other minerals or materials contaminated with fibrous asbestos be treated as hazardous and regulated as asbestos.

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[23] Rohl AN, Langer AM, 1974. Identification and quantitation of asbestos in talc. Env Health Perspectives, Dec., 9; 95-109.

[24]Fitzgerald EF, Stark AD, Vianna N, Hwang S-A, 1991. Exposure to asbestiform minerals and radiographic chest abnormalities in a talc mining region of upstate New York. Archives of Environmental Health. May/Jun, 46 (3); 151-154.

[25] Dement J M, Zumwalde RD, Gambel JF, Fellner W, DeMeo MJ, Brown DP, Wagoner JK, 1980. Occupational exposure to talc containing asbestos-Morbidity, Mortality, and environmental studies of miners and millers. NIOSH Technical Report-DHEW (NIOSH) Publication No. 80-115, Feb.

Finally when new epidemiology studies are conducted strict criteria must be followed to assure the best quality studies possible. These criteria should include, but not limited to areas such as:

1 – Determine actual exposure to the fibrous material and not allow dilution of any effect finding by including those in the cohort not exposed to the fibrous material;

2 - Allow sufficient size of the study population to assure sufficient power to detect adverse effects if they exist;

3 – Conduct sufficient follow-up to assure that at least 95% of the cohort is traced and vital status known and evaluated;

4 – Allow sufficient latency to determine if adverse effects do develop, this is important since known traditional latency periods may be extended due to lower level cumulative exposures experienced today;

5 – Identify and account for any possible confounders that may affect the outcome of the study;

6 – If case-control analyses are conducted make sure that all matched controls are selected so that confounding factors will not skew the outcome, including adequate occupational histories to rule out other causative agents or past occupational exposures; and

7 – Dose-reconstruction should not be allowed unless adequate data points exist, from actual exposure samples taken at multiple points during the entire exposure period, as extrapolation from more recent exposures will often reflect control technologies not in place earlier in the persons exposure history, thus resulting in an under estimate of the individuals true exposure. Dose-reconstruction should never be applied from one work situation to another without adequate working conditions

being explained and/or described by the affected worker or from actual witnesses to the workers exposure conditions, including an explanation of both environmental or personal control-technologies applied in the specific workplace(s).

I would hope all who have testified here today have disclosed their own affiliations and potential conflicts of interest. Since my retirement I have testified numerous times for plaintiff's attorneys in asbestos litigation, I am also Co-Science Director to the Asbestos Disease Awareness Organization (ADAO) which has covered some of my expenses to attend this hearing today, and no expenses for my testimony or preparation for it have been covered by plaintiff attorneys or any other entity other than myself.

Last, I would encourage members of this Committee to support the Ban Asbestos Act introduced by Sen. Murray to include a ban on all commercial uses and importation of asbestos to or within the United States. I look forward to be of assistance should further questions arise.

Appendix 1

Short Asbestos Fibers

Richard A. Lemen, Ph.D.

EPA reported that millions of asbestos fibers can be released during brake and clutch servicing and that such asbestos can linger around the garage long after brake jobs are done and can be breathed in by everyone inside the garage which can present a hazard for months or years. Grinding of used brake block linings has been shown to release up to 7 million fibers per cubic meter and beveling new linings up to 72 million fibers and even light grinding of the new linings up to 4.8 fibers.[26] It has also been reported that during this decomposition process the majority of fibers that remain are of small diameter as well as below 5 micron in length[27] and thus are less harmful.[28]

Any assumption that short fibers, less than 5 micron in length, are not hazardous cannot be justified based on the available science. Because the analytical method of choice, for regulatory purposes, has been the phase contrast method [PCM] which counts only fibers greater than 5 um in length, epidemiology studies therefore have been forced to compare doses of exposure within their cohorts only to fibers greater than 5 μm in length. It must be noted that the PCM analytical

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[26] USEPA, 1986. Guidance for Preventing Asbestos Disease Among Auto Mechanics. United States Environmental Protection Agency. EPA-560-OPTS-86-002, June.

[27] Rohl, AN, Langer, AM, Wolff, MS & Weisman, I, 1976. Asbestos exposure during brake lining maintenance and repair. Environ Research, Vol. 12, p. 110; Sheehy, J. W., Cooper, T. C., O'Brien, D. M., McGlothlin, J. D., & Froehlich, P. A., 1989. Control of Asbestos Exposure During Brake Drum Service. National Institute for Occupational Safety and Health, Public Health Service, Centers for Disease Control, U. S. Department of Health and Human Services, August; & Yeung, P, Patience, K, Apthorpe, L, & Willcocks, D, 1999. An Australian study to evaluate worker exposure to chrysotile in the automotice service industry. Appl Occup Environ Hyg, Vol. 14, No. 7, July, p. 448.

[28] Hatch, D, 1970. Possible alternatives to asbestos as a friction material. Ann Occup Hyg, vol. 13, p. 25.

method was chosen based on its ability to count fibers only and not on a health effect basis.[29] While PCM has been the international method for analysis, it should also be noted that it is not able to detect thin diameter fibers [<0.2μm in diameter]. The evidence suggests that PCM may underestimate exposures and the health risks as found in the analysis of brake residue,[30] or other such exposures where short fibers may be found and because of this, it has been suggested that transmission electron microscopy [TEM] should be an adjunct to PCM.

Stanton and Wrench (1972)[31] and Stanton et al. (1981)[32] found that the longer, thinner fibers were more carcinogenic, but they could not identify a precise fiber length that did not demonstrate biological activity. It must be kept in mind that Dr. Stanton has never said long fibers are bad and short fibers are good. In fact, he appreciated that a large number of short

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[29] "The first decision made concerned that part of the dust spectrum which should be counted and it was agreed that only fibers or fiber bundles having a minimum length of 5 microns and a maximum of 100 microns should be counted, the definition of a fiber being arbitrarily taken as a particle whose length was at least three times it diameter. This decision was taken in the light of evidence to the effect that the particle size distribution or spectrum of an asbestos dust cloud was reasonably constant over a wide range of textile processes, although later work has suggested that this might not be strictly true." This decision represent the conclusions made for use of the Thermal Precipitator Method in collecting asbestos-containing dust and when the Membrane Filter Technique came into use, the basis for the method referred to as the PCM method, it was determined that the 5 micron in length would remain the standard as "The filter on the other hand, having a pore size in the region of 0.45 micron, would appear to be quite adequate for trapping fibers in the length range 5-100 microns." While it was thought the Membrane Filter Technique would be more representative in assessing the "true health hazard to which an operative is subjected" it did not rely upon knowledge that fibers less than 5 micron in length had been shown harmless. Holmes S, 1965. Developments in dust sampling and counting techniques in the asbestos industry. Ann NYA Sciences: 132(1); 288-297.

[30] Yeung, P, patience, K, Apthorpe, L, & Willcocks, D, 1999. An Australian study to evaluate worker exposure to chrysotile in the automotice service industry. Appl Occup Environ Hyg, Vol. 14, No. 7, July, p. 448.

[31] Stanton, M.F., and Wrench, C., 1972. Mechanisms of mesothelioma induction with asbestos and fibrous glass. J. Natl. Cancer Inst., Vol. 48, p. 797.

[32] Stanton, MF, Laynard, M, Tegeris, A, et al. 1981. Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals. JNCI, Vol. 67, No. 5, November, p. 965.

fibers, individually of low tumorogenic probability, might be more hazardous than fewer long fibers, individually of high probability.[33]

Studies have also found that the majority of asbestos fibers in lung and mesothelial tissues were shorter than 5 μm in length, thus indicating the ability of the shorter fibers to reach the tumor site, remain there, and therefore their role in the etiology of disease is implicated.[34] Research has found in typical occupational environments fibers shorter than 5 μm in length outnumber the longer fibers by a factor of 10 or more.[35]

Shorter fibers must be studied in more depth and they should not be disregarded especially when clearance is retarded.[36] That chrysotile fibers tend to spit longitudinally as well as partially dissolve, resulting in shorter fibers within the lung, was reported in a review of several articles.[37]

Davis et al., 1986, 1988 and the Berman et al., 1995 reanalysis of the Davis data and the McDonald et al., 1989 papers examine both the toxicity or lack thereof for short fibers.[38] The Davis papers show that: 1) long fibers produced 6

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[33] Greenberg, M, 1984. S Fibers. Am J Indust Med, Vol. 5, p. 421-422 & Personal correspondence from Dr. Morris Greenberg, 23 May 2003.

[34] Suzuki, Y. & Yuen, SR., 2002. Asbestos fibers contributing to the induction of human malignant mesothelioma. Ann NY Acad Sci, Vol. 982. pp. 160-176 & Dodson, RF, O'Sullivan, MF, Brooks, DR & Bruce, JR, 2001. Asbestos content of omentum and mesentery in nonoccupationally exposed individuals. Tox Indust Health, Vol. 17, p. 138.

[35] Dement, JM & Wallingford, KM, 1990. Comparison of phase contrast and electron microscopic methods for evaluation of occupational asbestos exposures. Applied Occ Env Hyg, Vol. 5, p. 242.

[36] Oberdorster, G, 2001. Fiber characteristics, environmental and host factors as determinants of asbestos toxicity. 2001 Asbestos Health Effects Conference, May 24-25, Oakland, CA, U. S. Environmental Protection Agency.

[37] Dement, JM & Brown, DP, 1993. Cohort mortality and case-control studies of white male chrysotile asbestos textile workers. J Occup Med Toxic, Vol. 2, No. 4, p. 355.

[38] Davis JM, Addison J, Bolton RE, et al. 1986. The pathogenicity of long versus short fibre samples of amosite asbestos administered to rats by inhalation and intraperitoneal injection. Br J Exp Pathol 67: 415-430; Davis JM, Jones AD. 1988. Comparisons of

times more fibrosis and 3 times more tumors than the short fiber preparations after inhalation; 2) injection studies, at the highest dose levels 25 mg, found little difference in the numbers of tumors produced by both long and short-fibre chrysotile, while at lower levels there was a significant difference between the long and short-fibre preparations with the longer fibers being more carcinogenic; 3) the mean tumor induction period was longer for the short-fibre preparation in producing mesotheliomas at both the 25mg and 2.5mg dose level and the authors conclude "...would probably have been seen with the 0.25mg dose if the short-fibre chrysotile had produced any mesotheliomas at this level."; and 4) the authors state that the alteration of the short-fibre chrysotile produced by ball-milling is subject to a level of crystal damage which is sufficient to make results difficult to interpret in relation to hazards resulting from short fibres produced during the manufacture of asbestos products or during the subsequent usage of these materials. Berman et al., 1995, using a risk analysis model of their choice choose to eliminate all fibres less than 5 μm in length as "Structures <5 μm in length do not appear to make any contribution to lung tumor risk." Such an assumption is unwarranted given the conclusions of the Davis et al. papers along with the other data, discussed in this affidavit, showing toxicity for the short asbestos-fibers.

McDonald et al., 1989 examined 78 cases of mesothelioma from autopsy between 1980 through 1984 with matched referents to evaluate the lung burden of long vs. short fibers, concluded that the role of short-fibers was nil. Looking only at lung burden analysis for chrysotile short-fibers is not the only way nor is it the most appropriate analysis to determine the role of either chrysotile or short-fibers, as they are cleared from the lung rapidly compared to longer non-chrysotile fibers.

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the pathogenicity of long and short fibres of chrysotile asbestos in rats. Br J Exp Pathol 69: 717-737; Berman DW, Crump KS, Chatfield EJ et al. 1986. The sizes, shapes, and mineralogy of asbestos structures that induce lung tumors or mesothelioma in AF/HAN rats following inhalation. Risk Analysis 15: 181-195; & McDonald JC, Armstrong B, Case B et al. 1989. Mesothelioma and asbestos fiber type: Evidence from lung tissue analyses. Cancer 63: 1544-1547.

This same criticism is applicable to the Butnor et al.,[39] analysis of 10 cases of mesothelioma among brake exposed workers where analysis was only made of lung tissue.

Butnor et al. also dismiss the 'hit-and-run' hypothesis for chrysotile as 'flimsy' and having no solid scientific support and cite Hesterberg et al., 1994, 1995, 1996 studies,[40] of man-made vitreous fibers, as their proof for this contention. While there is clear proof of the biopersistence for amphibole asbestos, the lack of such biopersistence of other fibers, as shown in the Hesterberg et al papers, provide support to the contrary, and are an indication that pathogenicity of a fiber is dependent upon more than simply the dose, dimension, and the durability of the fibers found with in the lung. It is also important to note that chrysotile asbestos produced fibrosis, lung tumors and mesothelioma in rats after inhalation studies as shown in the Research and Consulting Company (RCC) studies cited in the Hesterberg et al., 1995 paper.

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[39] Butnor KJ, Sporn TA, Roggli VL. 2003. Exposure to brake dust and malignant mesothelioma: A study of 10 cases with mineral fiber analyses. Ann Occup Hyg 47: 325-330.

[40] Hesterberg TW, Miiler WC, Mast R, McConnell EE, Bernstein DM & AndersonR. 1994. Relationship between lung biopersistence and biological effects of man-made vitreous fibers after chronic inhalation in rats. Env Health Perspect 102(S); 133-137;Hesterberg TW, Miiller WC, Thevenaaz P, & Anderson R. 1995. Chronci inhalation studies of man-made vitreous fibres: Characterization of fibres in the exposure aerosol and lungs. Ann Occup Hyg 39 (5): 637-653%Hesterberg TW, Miiller WC, Musselman RP, Kamstrup RD, Hamilton RD & Thevenaz P. 1996. Biopersistence of man-made vitreous fibers and crocidolite asbestos in the rat lung following inhalation. Fund Appl Toxico 29: 267-279.

Appendix - 2

Mesothelioma Surveillance

Richard A. Lemen, Ph.D.

Two recent papers have concluded the beginning of a decrease in mesothelioma rates in the United States.[41] Their data analyses bring to the fore additional questions about the reliability of surveillance data for mesothelioma based solely on death certificate analysis or mortality data without pathological confirmation of mesothelioma. SEER data, for example, prior to the implementation of the new ICD 10 codes, are inaccurate and underestimate the true incidence of mesothelioma in the U.S.[42]

The new ICD-10 codes for mesothelioma are C45.0 for pleural and C45.1 for peritoneal.[43] Before the new ICD-10 codes went into effect in 1999 the reporting based on incidence data was likely underreported and thus analysis using such data is likely to have underreported the incidence of mesothelioma. In some cases, SEER data reported only 12% of the mesothelioma cases were accurately reported and even with the new ICD 10 codes it is estimated that only about 80% will be detected through SEER data, indicating that mesothelioma reporting will still be problematic but much less so than in the past.[44] The new ICD 10 codes have only been in existence for the past 8 years and any trends based on this data are unwarranted at this time and it will be many years until a more accurate picture can be seen as to mesothelioma trends

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[41] Price B & Ware A, 2004. Mesothelioma trends in the United States: An update based on surveillance, epidemiology, and end results program data for 1973 through 2003 &
[42] Pinheiro GA, Antao VCS, Bang KM & Attfield MD, 2004. Malignant mesothelioma surveillance: A comparison of ICD 10 mortalaity data with SEER incidence data in nine areas of the United States. Int J Occup Environ Health: 10; 251-255.

[43] World Health Organization, 1992. ICD-10 International Statistical Classification of Diseases and Related Health Problems Tenth Revision: 1; 201.

[44] Pinheiro GA, Antao VCS, Bang KM & Attfield MD, 2004. Malignant mesothelioma surveillance: A comparison of ICD 10 mortalaity data with SEER incidence data in nine areas of the United States. Int J Occup Environ Health: 10; 251-255.

within the U.S. It is important that NIOSH address this underreporting gap.

Since it has been generally reported that the incidence of mesothelioma in women is much less associated with asbestos exposure, Steenland et al.[45] suggest that if take-home asbestos exposure were considered the attributable risks may rise to around 90%. Price and Ware (2004) unjustly suggest that because the female lifetime mesothelioma risk across birth cohorts has remained constant this supports a threshold exposure for mesothelioma, which is yet to be shown and no epidemiological study to date has been able to demonstrate such a threshold. Trends in mesothelioma are on the rise in many countries and a large multicentric study on malignant pleural mesothelioma and non-occupational exposures to asbestos projects that low-doses from the home and general environment may carry a measurable risk of mesothelioma over the next few decades.[46] The findings of this multicentric study have direct implications to the risk of mesothelioma from exposures to asbestos among end-product user of asbestos-containing products, e.g. brake mechanics, as their exposures have generally been of a lower magnitude that those encountered by the various highly exposed and predominately studied trades including insulators, construction workers, shipyard workers, pipefitters to name a few.

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[45] Steenland K, Burnett C, Lalich N, Ward E & Hurrell J, 2003. Dying for work: The magnitude of US mortality from selected causes of death associated with occupation. 43; 461-482.

[46] Magnani C, Agudo A, Gonzalez CA et al., 2000. Multicentric study on malignant pleural mesothelioma and non-occupational exposure to asbestos. Br J Cancer: 83(1); 104-111.