Comments by Richard A. Lemen, Ph.D.
Public Hearing on Asbestos and Other Mineral Fibers: A Roadmap for Scientific Research
– May 4, 2007 -

I would like to commend NIOSH on their Draft Roadmap for Scientific Research on Asbestos and Other Mineral Fibers released in February 2007. Important issues are addressed in this document and NIOSH has set forth a good agenda to address them.

I would like to make some personal observations before I go into specifics about issues in the NIOSH Roadmap. First, I encourage NIOSH to evaluate all data input it receives with full disclosure of the source. I am here today as in my role as Co-chairman of the Science Advisory Board of the Asbestos Disease Awareness Organization (ADAO), a non-profit asbestos victims advocacy organization. I also testify on behalf of plaintiffs in asbestos litigation. I am also an Adjunct Professor with the Department of Occupational and Environmental Health of the Robbins School of Public Health of Emory University in Atlanta. I am here today at my own expense. I would hope all who testify here today will disclose their own affiliations and potential conflicts of interest and who is supporting their attendance here today.

Since the passage of the OSHAct of 1970, NIOSH has been the leader in prevention methodologies for asbestos-related health issues. The first criteria document from the newly formed NIOSH of 1971, 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®. Dr. Key recommended an immediate lowering to 2 fibers/cc. NIOSH was the first Federal agency, in its 1976 Revised Criteria Document, to call for a ban on asbestos. NIOSH has maintained this position to the present while suggesting in the interim that the only reliable and practical analytical method, at that time, was 0.1

fiber/cc using the NIOSH 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 since most individual "fibrils" 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.[1] This is something NIOSH suggests it intends to address in the new Roadmap.

NIOSH's new Roadmap represents its continued leadership role in occupational safety and health by addressing asbestos-related issues needing clarification and further elucidating and/or addressing questions still unresolved. By so doing NIOSH is fulfilling its congressionally mandated role under the Occupational Safety and Health Act of 1970.

NIOSH should not back away from including all respirable asbestiform fibrous minerals, including cleavage fragments

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[1] 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 y 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."

which appear to be in a fibrous habit and thus fitting the asbestos definition by light microscopy and that are clearly respirable[2] dusts. This should only be changed if there exist irrefutable data, both human and animal, showing the safety of any such fibrous mineral being excluded since the only difference of these entities from the structures of the same mineral in true asbestiform habit is the structural morphology with all other characteristics being the same.

NIOSH should develop valid methodologies to sample for all size fibers, including those less than 5 um in length, now not addressed by OSHA regulatory standards. Both animal and human data support such an inclusion as can be seen by the attached Appendix - 1.[3]

NIOSH should address and refine their current surveillance of fiber-related diseases. For example it is well known that the National Cancer Institutes SEER data base underreports mesothelioma.[4] NIOSH should continue its Respiratory Disease Surveillance System and should assure that other NIOSH surveillance systems become more comprehensive and inclusive, and analysis should not relying solely on Proportionate Mortality/Morbidity Analysis (PMR) for determining mortality or incidence data, as this type analysis underreports low incidence and rare diseases, albeit important diseases i.e. mesothelioma.

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

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[2] 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.

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

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

consumption of asbestos has gone up so have background rates of asbestos-related diseases.[5]

NIOSH should review the epidemiology literature on all fibrous materials, not just those related to the currently regulated asbestiform fiber types. Such research should address all respirable fiber types and all size parameters, including short respirable fibers.

Since biopersistence has been used as a surrogate by identifying lung fiber burden as a critical factor in causation, toxicological studies should evaluate whether external airborne concentrations are representative of the fiber concentrations and morphologies once the fibers have been inhaled into the lung. Data suggest that the breathing zone samples of chrysotile may not represent the actual fiber burden of chrysotile fibers in the lung as they break apart from fiber bundles and multiply once within the lung, while the amphiboles do not.[6] This is important as it means a higher dose of chrysotile in the lung as well as higher rate of translocation of chrysotile from the lung. 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. This translocation of chrysotile asbestos indicates a more specific role for chrysotile in the etiology of mesothelioma. 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[7]. Since

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[5] See Appendix 3 – Mesothelioma Background, Richard A. Lemen, Ph.D.

[6] 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.

[7] 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

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 cause mesothelioma. This is also shown by many mechanistic and molecular studies that 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.[8] Some of these abnormalities include the deletion of the P53 gene that controls cell growth.[9]

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.[10] It has been suggested by some that the fibrous tremolite contamination of chrysotile, usually less than 1%, is the cause of mesothelioma

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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.

[8] 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.

[9] 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.

[10] 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.

among predominately chrysotile exposed persons.[11] New evaluation of the South Charleston chrysotile exposed population of textile workers has confirmed a dose-response relationship between asbestosis and lung cancer.[12] This is important as entities suggesting that chrysotile is the "safe asbestos" base their conclusions on only one outcome, 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; however, this does not appear the case for other asbestos-induced disease. Therefore, future NIOSH research should continue to look at other asbestos-induced diseases when determining recommended regulatory actions for the prevention of asbestos-related diseases.

The current OSHA regulations govern exposure to entities 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).[13]

Next I will provide some data which may shed some light on the arguments for including a broad fiber definition when it

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

[12] 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.

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

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.[14] Rohl and Langer (1974) have stated "Talc because of its composition, conditions of formation and geological occurrence, is frequently contaminated with asbestos fibers."[15] 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.[16]

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 average (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 and 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 transmition

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[14] 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

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

[16] 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.

electron microscope (ATEM) as well as Phase contrast microscope (PCM) and not cleavage fragments.[17]

I would submit for the record today some correspondence you may not be aware of dealing with this issue. These data show how Johns-Manville analysis determined that R.T. Vanderbilt's talc products contain tremolite, chrysotile, and anthophyllite and that CP talc's contain up to 30% tremolite and that because of this Johns-Manville determined the necessity for labeling such fibrous containing talc's as containing asbestos. I recommend that NIOSH maintain its position that all fibrous asbestiform minerals and that all other minerals or materials contaminated with fibrous asbestos be treated as hazardous.

Finally when NIOSH conducts or contracts out epidemiology studies strict criteria must be followed to assure the best quality studies possible. These criteria should include, but not be limited to such areas as:

1 – Determine actual exposure to the fibrous material and not allow dilution of any finding because non -exposed were included in the cohort non-exposed;

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 that vital status is 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;

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[17] 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.

5 – Identify and account for any possible confounders or co-factors that may skew or alter the outcome of the study; and

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

Last, I would encourage NIOSH support the Ban Asbestos Act introduced by Sen. Murray to ban all commercial uses and importation of asbestos. I would also add my assistance should NIOSH need it as I have had an intimate role in NIOSH asbestos research and policy recommendations dating since the agencies inception and until my retirement in 1996.

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.[18] 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[19] and thus are less harmful.[20]

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

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[18] USEPA, 1986. Guidance for Preventing Asbestos Disease Among Auto Mechanics. United States Environmental Protection Agency. EPA-560-OPTS-86-002, June.
[19] 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.
[20] Hatch, D, 1970. Possible alternatives to asbestos as a friction material. Ann Occup Hyg, vol. 13, p. 25.

phase contrast method [PCM] which counts only fibers greater than 5 um in length, epidemiology studies therefore have been forced to compare doses in their cohorts only to fibers greater than 5 μm in length and capable of being seen based on diameter of the fiber. The later point is most critical since the majority of chrysotile released from brake components are fibrils (0.02-0.05μm) and thus not resolvable by light microscopy regardless of length. It must be noted that the PCM analytical method was chosen based on its ability to count fibers only and not on a health effect basis.[21] While PCM has been the international regulatory method for analysis, 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

[21] "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.

brake residue,[22] and because of this, it has been suggested that transmission electron microscopy [TEM] should be an adjunct to PCM.

Stanton and Wrench (1972)[23] and Stanton et al. (1981)[24] found that the longer, thinner fibers were more carcinogenic, but 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 fibers, individually of low tumorogenic probability, might be more hazardous than fewer long fibers, individually of high probability.[25]

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.[26] Research has found in typical occupational environments fibers shorter than 5 μm

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[22] 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.

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

[24] 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.

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

[26] 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.

in length outnumber the longer fibers by a factor of 10 or more.[27] Shorter fibers must be studied in more depth and they should not be disregarded especially when clearance is retarded.[28] 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.[29]

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.[30] The Davis papers show that: 1) long fibers produced 6 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

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[27] 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.

[28] 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.

[29] 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.

[30] 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 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.

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 appendix, 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 or body burden of either chrysotile or short-fibers, as they are cleared from the lung rapidly compared to longer non-chrysotile fibers.

This same criticism is applicable to the Butnor et al.,[31] 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,[32] of man-made vitreous fibers, as their proof for this contention. While there is clear proof of the longer 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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[31] 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.

[32] 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.[33] 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 ICD 10 codes are inaccurate and underestimate the true incidence of mesothelioma in the U.S.[34]

The new ICD-10 codes for mesothelioma are C45.0 for pleural and C45.1 for peritoneal.[35] 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

[33] 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 &
[34] 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.
[35] World Health Organization, 1992. ICD-10 International Statistical Classification of Diseases and Related Health Problems Tenth Revision: 1; 201.

problematic but much less so than in the past.[36] The new ICD 10 codes have only been in existence for the past 5 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 within the U.S. It is important that NIOSH address this 20% underreporting gag as suggested even with the introduction of the new ICD-10 codes and within the SEER reporting system.

Since it has been generally reported that the incidence of mesothelioma in women is much less associated with asbestos exposure, Steenland et al.[37] 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

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[36] 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.

[37] 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.

mesothelioma over the next few decades.[38] 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, and pipefitters.

[38] 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.

Appendix - 3

Mesothelioma Background Rates

Dr. Richard A. Lemen

Two recent papers have concluded the beginning of a decrease in mesothelioma rates in the United States.[39] Their data analyses bring to the fore additional questions about the reliability of cohort studies based solely on mortality data, without pathological confirmation for mesothelioma. SEER data for example prior to the implementation of the ICD 10 codes are most likely inaccurate and most likely underestimate the true incidence of mesothelioma in the U.S. 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.[40] The findings of this multicentric study have direct implications to the risk of mesothelioma from exposures to asbestos among brake mechanics as their exposures have generally been of a lower magnitude that those encountered by the various highly exposed

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[39] 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 &
[40] 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.

and predominately studied trades including insulators, construction workers, and pipefitters.

The new ICD-10 codes for mesothelioma are C45.0 for pleural and C45.1 for peritoneal.[41] Since it has been generally reported that the incidence of mesothelioma in women is much less associated with asbestos exposure, Steenland et al.[42] 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. 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 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 mortality data will still be

[41] World Health Organization, 1992. ICD-10 International Statistical Classification of Diseases and Related Health Problems Tenth Revision: 1; 201.
[42] 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.

problematic but much less so than in the past.[43] The new ICD 10 codes have only been in existence for the past 5 years and any trends based on this data are unwarranted and it will be many years until an accurate picture can be seen as to the real mesothelioma trends within the U.S.

[43] 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.

File: Talc

Johns-Manville

Internal Correspondence

To: [redacted]

Date: February 17, 1977

From: J. P. Leineweber 1-04

Copies: R. Lamar 3-05 W. C. Streib R&D File Chrono

Subject: PAPER BY C. S. THOMPSON
"ASBESTOS IN YOUR FUTURE"

I believe there is sufficient information in our files concerning our opinions on Dr. Thompson's campaign to convince the regulatory authorities that a fiber is not a fiber. This type of thinking is one of the reasons that industry in general will continue to have a bad reputation in the eyes of those who sincerely want to protect the worker.

The quality of this paper is typified by the illustrations of the asbestiform and non-asbestiform varieties of chrysotile and amosite. Obviously, a competent mineralogist would not let an error of this magnitude slip through.

J. P. Leineweber

J. P. Leineweber
/pa
Attachment

CRMC-ELS-005022

Johns-Manville

Internal Correspondence

To: H. R. Keefe - 2W

Date: January 7, 1975

From: R. S. Lamar

Copies: See end of correspondence

Subject: YOUR MEMO OF JANUARY 2, 1975 TO P. KOTIN, " TALC AND ASBESTOS "

I cannot help but feel that some additional input from Research is needed here. Several points:

1. The National Paint and Coating Association is simply a trade organization. It carries no legal weight. We are still bound by the law as described in OSHA documents. It seems to me that Stender, in his letter of October 9, 1974, destroys his own organization. Until this confusion within OSHA is resolved, we have no choice but to comply with the law as written.

2. As I read the NP&CA Bulletin No. 20, I get two strong impressions:

   a. Rather than clarifying the matter, as stated, they only add to my confusion.

   b. Asbestos is not really asbestos, but it can be anything you want to call it, e.g., "commercial talc."

3. I am greatly concerned with the point raised by A. Finkbiner at our last meeting. This has to do specifically with the fact that we are "sitting" on information which shows quite conclusively that R. T. Vanderbilt's talc products contain not only tremolite, but significant amounts of chrysotile and anthophyllite as well. What might be J-M's legal responsibility by withholding such information? Refer to:

CRMC-ELS-004702

Memo October 9, 1974 V. E. Wolkodoff to R. S. Lamar
Report No. 414-T-33, October 4, 1974
Memo October 10, 1974 K. L. Jaunarajs to R. S. Lamar
Memo October 23, 1974 V. E. Wolkodoff to R. S. Lamar
Memo October 11, 1974 R. S. Lamar to V. E. Wolkodoff

Do we still intend to provide OSHA with this information on Vanderbilt's talc products? I also feel that we should provide this information to R. T. Vanderbilt.

4. Finally, the real question is medical in nature. From information I have from Kotin and Fenner, there is more than ample reason why we should label regardless of what the law might say. And, this could in time have a great deal to do with J-M's legal as well as its moral obligations.

I guess what I am really trying to say, Harry, is that the decision to label was right and should not be subject for reconsideration. We have to learn to live with it in a business sense regardless of how difficult this appears.

R. S. Lamar

kjm
attachment

cc:
P. Kotin - 4N
F. J. Solon - 4N
E. M. Fenner - 4N
A. C. Finkbiner - SW
P. A. Martinson - 2W
H. Kranich - 2W
W. C. Streib
File: 259-6.1

Below Not Sensitive

Johns-Manville

Internal Correspondence

To: J. H. Swensen

Date: August 31, 1976

From: R. S. Lamar

Copies: E. M. Fenner, G. R. Kinzer, H. R. Keefe, T. E. Remmers, W. C. Streib, S. R. Speil, G. Coombs, D, C

Subject: Meeting with Pfizer personnel at R&D Center August 31, 1976. Asbestos Minerals in Talc and Test Methods

We met with Robert E. Norwood, Research Manager for Pfizer's Minerals, Pigments, and Metals Division and with Harold D. Stanley, Jr.; Group Leader, Testing.

This was a good meeting in that both Pfizer and J-M are essentially in agreement as to test methods and interpretation of data. They use all of the same procedures we do: XRD, TEM, SEM, DTA, TGA, etc. but seem to rely most heavily upon TEM and XRD.

We showed them our data on samples of their CP talcs indicating up to 30% tremolite by XRD. They couldn't understand this and we gave them portions of the same samples for testing. They promised to get back to Ed Fenner with their results.

They had complete analytical data on three samples of Penhorwood which they recently obtained from some unknown source. By XRD they show no asbestos minerals. They believe that their lower limit of detectability by XRD is 0.1% or less. By TEM all three Penhorwood samples showed platy talc with an occasional fiber - less than one fiber per field. They don't regard this as significant and would interpret this as no asbestos mineral by OSHA requirements. We gave them three additional Penhorwood samples from our files for additional studies.

We also discussed commercial talcs from other sources. They feel that R. T. Vanderbilt is completely "off the track" in their stand on OSHA regulations concerning asbestos minerals. They think it is only a matter of time until this stand completely destroys Vanderbilt.

They described Jack Bartel's "emotional" interest in buying Desert Minerals Co. from J-M but believe that this will not happen because they view this business the same way we do - the tremolite is too great a hinderance to marketing.

R. S. Lamar

R. S. Lamar
RSL/imb

CRMC-ELS-004502

Johns-Manville

Internal Correspondence

A00500

To: See below*

Date: November 7, 1974

From: Paul Kotin, M.D., 4N

Copies: F. J. Solon, Jr.
R. Carter

Subject: TALC LABELING

*W. R. Goodwin, 5W	F. L. Pundsack, R&D
F. H. May, Jr., 5W	C. J. Sulewski, 2W
J. A. McKinney, 5W	W. L. VanDerbeek, 2S

We have been informed by Mr. Robert Bacon, Assistant to the President of R. T. Vanderbilt Company (a major talc competitor) that J-M's decision to insert asbestos caution labels on all talc shipments will result in "irreparable damage" to Vanderbilt. Mr. Bacon requested that this matter be brought to the attention of the highest level of management, and he stated that he was asking Mr. Hugh Vanderbilt, President of R. T. Vanderbilt, to call a senior officer at J-M to voice their concern and their belief that it is J-M's intention to hurt their company.

The purpose of this memorandum is to alert you to the situation in the event you are contacted by Mr. Vanderbilt. Our decision to label talc was based on our conviction that J-M's talc contains fibrous asbestos and in no way reflects any intention to hurt or destroy the Vanderbilt Company. If you would like additional details on this matter, please call me.

CRMC-ELS-004031

Johns-Manville

Internal Correspondence

To: V. E. Wolkodoff

Date: October 11, 1974

From: R. S. Lamar

Copies: See end of correspondence

Subject: YOUR MEMO OF OCTOBER 9, 1974
OPTICAL AND TEM COUNTING OF ASBESTOS MINERALS IN ASBESTINE 3X, ASBESTINE 325, NYTAL 200, AND NYTAL 400 AS CURRENTLY PRODUCED BY R. T. VANDERBILT CO.

Thank you Vlad for your very fine report covering your microscopic examination of these samples. Your data confirm analyses we have had made on these same samples by J. P. McGourty using X-ray diffraction techniques (Report No. 414-T-33) and by K. Jaunarajs using differential thermal analysis - primarily for the detection of serpentine mineral.

These present samples are really no different from previous samples of Nytal and Asbestine that I have examined periodically over the years. They all contain very substantial amounts of both tremolite and chrysotile and in two cases substantial amounts of a third asbestos mineral, anthophyllite.

In numerous discussions I have had with R. T. Vanderbilt people, they have readily admitted to having tremolite ("which is not an asbestos mineral and is not fibrous"), but they have never admitted to the presence of either chrysotile or anthophyllite. They do admit that years ago, when they were deliberately mining certain sections of their deposits for fiber, some of the products did contain chrysotile. This product, "Mouldene" was sold as a direct replacement for asbestos in the manufacture of vinyl tile. I have examined "Mouldene" in the past and it was in fact almost entirely chrysotile. "Mouldene" is no longer being made, but all of the International Talc Company and R. T. Vanderbilt Company talc products always have and continue to contain chrysotile as a significant mineral component (in addition to tremolite and anthophyllite).

It is apparent that the R. T. Vanderbilt presentations to OSHA, NIOSH, FDA, MESA, etc. are based on something less than the truth. I find it difficult to believe that they could be so grossly misinformed as to what their materials really are.

How this information is ultimately used is something that will have to be decided at higher levels within J-M. However, it is my belief that R. T. Vanderbilt, with a continuance of their present methods, does nothing but confuse the issue among the talc producers and with the various Federal agencies involved. Any properly informed person would know that they are wrong.

R. S. Lamar

kjm

cc:

R. P. Carter	- 4N	F. L. Pundsack	
E. M. Fenner	- 4N	W. C. Streib	
P. Kotin	- 4N	J. P. Leineweber	
W. B. Reitze	- 4N	S. Speil	
F. J. Solon, Jr.	- 4N	A. J. McArthur	
G. L. Swallow	- 4N		
A. C. F. Finkbiner, III	- 5W		
J. A. McKinney	- 5W		
H. R. Keefe	- 2W		
P. A. Martinson	- 2W		
C. J. Sulewski	- 2W		
W. L. VanDerbeek	- 2S		
R. G. Riede	- 2S		

File 414-C R. T. Vanderbilt

Johns-Manville

Internal Correspondence

To: F. J. Solon, Jr. ✓

Date: Oct. 31, 1974

From: R. P. Carter

Copies: File & C

Subject: TELEPHONE CONVERSATION WITH BOB BACON

Bob Bacon, Assistant to the President, R. T. Vanderbilt Co., called me this morning to advise that our talc labeling decision is causing a "big stink". Vanderbilt's talc customers are very concerned as to why J-M has decided to commence inserting asbestos caution labels on all packages of talc.

R. T. Vanderbilt Co. is of the opinion that it is Johns-Manville's intention to hurt their company. Bob Bacon requested that I bring this matter to the highest level of management within J-M and indicate that Vanderbilt is "very, very upset" with our labeling decision, which they feel will result in "irreparable damage" to them. Bob Bacon indicated that he is going to request Hugh Vanderbilt to personally call a Senior Officer at Johns-Manville to voice their concern.

It has been obvious for some time from my conversations with Bob Bacon that Vanderbilt is very concerned about our talc labeling decision. Without attempting to characterize the motivating factors behind Bob Bacon's frequent telephone conversations to me, their concern may very well be based on a distinct difference between their "corporate conscience" and ours. Certainly, our decision in no way reflects any intention on J-M's part to hurt or destroy Vanderbilt, which is Vanderbilt's current feeling. It is Vanderbilt's opinion that based on John Stender's letter of October 9, 1974, to Hugh Vanderbilt, their talc does not contain asbestiform minerals such as fibrous tremolite. Perhaps that is true, and it is even possible that our talc does not contain asbestiform minerals based on the definition contained in Stender's letter. Basically, Vanderbilt is requesting, if not demanding, that J-M reverse its talc labeling decision based on Stender's letter.

As you will recall, I recommended several weeks ago that we promptly take the following actions in the sequence listed below:

(1) Reconvene a meeting of the talc labeling group which met early this past Summer to review R&D's analyses of Vanderbilt's Nytal and to determine J-M policy and action in response to Stender's October 9 letter.

(2) Arrange a meeting in Denver between certain R. T. Vanderbilt personnel and appropriate individuals from R&D to review our analyses of Nytal and to inform Vanderbilt officially as to J-M policy and contemplated actions on this matter.

(3) Meet with Jon May and perhaps other NIOSH personnel to review the details of their study of tremolitic talc and their recommendations to OSHA, which I have been told were incorporated in Stender's letter.

(4) Arrange for a meeting with OSHA, including Alexander Reis, Dan Boyd and Howard Schulte to discuss the talc labeling situation, and perhaps point out the error in Stender's definition of "fibrous, asbestiform minerals".

I believe this matter warrants our immediate attention.

R. P. Carter

RPC/emr

CRMC-ELS-004603