

**Extended Comments of Richard A. Lemen, Ph.D., M.S.P.H.,
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My name is Dr. Richard Lemen and I am a former Assistant Surgeon General of the United States Public Health Service as well as former Acting Director and Deputy Director of the National Institute for Occupational Safety and Health. Currently I am a private consultant and as such, I have testified on behalf of plaintiffs in asbestos litigation. I have researched the epidemiology of asbestos-related diseases for the past 37 years and have consulted extensively on asbestos with United States Governmental agencies, the World Health Organization, and various Governments around the World. I have also written multiple papers in the peer review literature and chapters for textbooks on the epidemiology of asbestos-related diseases.

I would like to thank Chairman Wynn and the entire Subcommittee on Environment and Hazardous Materials

committee for the honor and opportunity to testify today before you. I am here to support the efforts of both the United States House of Representatives and the United States Senate to ban asbestos in the United States. This Ban will represent a monumental public health achievement for the United States and its citizens in preventing asbestos-related disease to workers and the public and I commend the efforts of the United States Congress for their work in this endeavor.

Asbestos is a killer.

It often kills in what appears to be a random pattern affecting one and leaving another unharmed even though they have similar exposures. We do not know why this happens, but it probably has to do with individual susceptibility or other circumstances unknown to science today. We do know that asbestos-related diseases are dose-response diseases and as the dose increases, the risk of developing asbestos-related diseases increases. We also

know there has not been a dose identified below which, some individuals, are not at risk of disease.

As we address asbestos during this hearing, over the next two to three hours, approximately 3 to 4 people will die of an asbestos-related disease. These deaths are preventable.

Unfortunately, these numbers represent only an estimate and are one that is clearly an underestimate, because there are no nation-wide surveillance systems that adequately capture the true nature of asbestos-related diseases. For example, one of our premier surveillance systems, the Surveillance Epidemiology and End Results (SEER) database of the National Cancer Institute (NCI) has found to under-report mesothelioma in some areas by as much as 80%.¹ I am glad these Bills provide language to address these deficiencies so

¹ Pinheiro GA, Antao VCS, Bang KM & Attfield MD, 2004. Malignant mesothelioma surveillance: A comparison of ICD 10 mortality data with SEER incidence data in nine areas of the United States. *Int J Occup Environ Health*: 10; 251-255.

that we will eventually have data to measure the true impact of asbestos and to determine if our public health efforts to prevent asbestos-related diseases are effective.

As we see in countries that have banned or placed strict regulations on the import and use of asbestos the trend of asbestos-related diseases are beginning to slow down.

However, that is not true in the United States, according to the National Institute for Occupational Safety and Health (NIOSH) where asbestosis is the only occupationally induced dust disease of the lungs that continues to increase each year, this is also true for mesothelioma, a signal tumor related almost exclusively with exposure to asbestos.²

While this County is still experiencing an asbestos-induced disease epidemic, that continues to grow worse, it is shifting

² McDonald JC, 1985. Health implications of environmental exposure to asbestos, *Environ Health Perspect.* 62: 319-328; Mullan RJ, Murthy LI, 1991. Occupational sentinel health events: An up-dated list for physician recognition and public health surveillance. *AJIM.* 19: 775-799.

from occupational to claim non-occupationally exposed victims.

Proponents of continued asbestos usage are trying to influence the regulatory agencies with efforts to exclude some forms of asbestos as well as re-write the definition of asbestos to exclude exposures to non-asbestos materials often contaminated with fibrous forms of asbestos.

It is clear that all forms of asbestos, including chrysotile, the “so-called-safe” form of asbestos, cause all asbestos-related diseases. While chrysotile appears less potent on a fiber-by-fiber basis for the induction of mesothelioma when compared to the other commercial fiber types, the amphiboles, it represents the most commonly used asbestos today and historically represents over 95% of asbestos usage. Chrysotile fibers tend to spit longitudinally as well as partially dissolve, resulting in shorter fibers within the lung.³

³ 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.

I am pleased these Bills make no distinction and include all fiber types in the Ban, and recognize the shaky science base for the proposing the continued use of what some say about chrysotile the “so-called-safe” form of asbestos.

I would like to provide data, which will shed light on the reasons for keeping the fiber definition as is in these Bills. From my years with NIOSH, I know researchers have found among talc miners and millers’ mesothelioma from two counties in Northern New York and new cases continue.⁴ Data also indicate talc miners and millers also experience excess parenchymal fibrosis and pleural changes. Rohl and Langer, at the time from the Mt. Sinai School of Medicine in New York, have stated “Talc because of its composition, conditions of formation and geological occurrence, is frequently contaminated with asbestos fibers.”⁵

⁴ 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

NIOSH's Dement and co-workers 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 5 μ m in length. "Such short fibers would not be included in a NIOSH count scheme since fibers below 5 microns are not counted even if detected by light microscopy as per assessment for determining air content of fibers as related to the PEL.⁶ Their

⁵ Rohl AN, Langer AM, 1974. Identification and quantitation of asbestos in talc. *Env Health Perspectives*, Dec., 9; 95-109

⁶ Dement J M, Zumwalde RD, Gamble JF, Fellner W, DeMeo MJ, Brown DP, Wagoner JK, 1980. Occupational exposure to talc containing

finding of asbestiform tremolite, anthophyllite and in a couple of samples chrysotile fibers when using Analytical transmission electron microscope (ATEM) as well as PCM in a mine labeled non-asbestiform talc dictates the need for more thorough and comprehensive analyses and as well as inclusion in the asbestos ban.

The exclusion of fibers less than 5 μm in length is not scientifically justified for three reasons. First, because of the previous definitions excluding fibers less than 5 μm have limited the ability of epidemiology to study populations solely exposed to fibers at these short lengths. This is because the method of choice was the PCM analytical method and was chosen based on its ability to count fibers only and not on a health effect basis.⁷ While PCM has been the international

asbestos-Morbidity, Mortality, and environmental studies of miners and millers. NIOSH Technical Report-DHEW (NIOSH) Publication No. 80-115, Feb.

⁷ “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 its diameter. This decision was taken in the light of evidence to the effect that the

regulatory method for analysis, it is not able to detect thin diameter fibers [$<0.2\mu\text{m}$ in diameter]. The evidence suggests that PCM may underestimate exposures and the health risks as found, for example, in the analysis of brake residue,⁸ and because of this, the transmission electron microscopy [TEM] should be an adjunct to PCM. Second, a reanalysis by NIOSH of analytical samples previously taken and using the greater than five micron length definition found, when using transmission electron microscope methodology, that on

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.

⁸ 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.

average 90% of the fibers were actually below 5 μm in length.⁹ This indicates epidemiology studies using the PCM method only have no basis to imply that only those fibers over five microns were the causative fibers. A new study of the NIOSH cohort of textile workers, predominately exposed to chrysotile asbestos, in South Carolina, has added new information to this second reason. This is the finding, when using TEM analysis, a strong correlation shows “. . . cumulative exposure to all fiber size indices, including fibers $\leq 5 \mu\text{m}$ in length, were highly statistically significant predictors of lung cancer or asbestosis mortality.” Mesothelioma was not examined as only 3 cases were observed at this period of latency.¹⁰ Third, pathological studies dating back to 1933 have shown that fibers most likely to penetrate into the lung tissue and to move to the areas where mesothelioma occurs are these short

⁹ 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.

¹⁰ Stayner L, Kuempel E, Gilbert S, Hein M, Dement J, 2008. An epidemiologic study of the role of chrysotile asbestos fiber dimensions in determining respiratory disease risk in exposed workers. *OEM Online Firs*, Published on December 20, 2007, as 10.1136/oem.2007.035584.

fibers.¹¹ In summary, science has not exonerated fibers below five μm in length from being a health risk and on the contrary, what little science that exists would indicate the opposite.

I would suggest, in addition to the Bills direction that the EPA develops analytical methodologies that they also include NIOSH who has been instrumental in developing the most used analytical methods to date.

Cleavage fragments of asbestos should be included in these Bills. The cleavage fragment of a mineral is comprised of the same chemical composition as the form of the mineral defined by shape as a fiber. Cleavage fragments, in the form of dust, are as readily inhaled as a fiber of the same mineral. The finding of disease including mesothelioma in both New York talc miners and Minnesota iron miners where cleavage

¹¹ Gloyne SR, 1933. The morbid anatomy and histology of asbestosis. *Tubercule*, 14: 447-451; 550-557; July, September; 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.

fragments were at issue confirm their need for inclusion in the asbestos Ban bill.¹²

Keep in mind that the potential for diseases to occur from inhalation of fibrous dust or any dust is not just related to its shape. To the contrary, most dust-induced diseases are due to the inhalation of non-fibrous dusts. Certainly fibrous dusts carry some risk for inducing disease once inhaled by virtue of their shape. However increasing numbers of publications have shown that various features associated with the surface and chemical features of inhaled dusts can trigger deleterious chemical events in biological systems such as the formation of charged chemical structures- radicals as well as immune responses that are shown to be harmful to cells in the body.¹³

Presently a fiber, for purposes of various counting schemes

¹² Hull MJ, Abrahm JL, Case BW, 2002. Mesothelioma among workers in asbestiform fiberbearing talc mines in New York State. *Ann Occup Hyg*, 46 (Supp 1): 132-135; Magnan S, 2007. Mesothelioma in Northeastern Minnesota and two occupational cohorts: 2007 update. *Chronic Disease and Environmental Epidemiology*, Minnesota Department of Health, December 7, 16 pgs.

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

(NIOSH and AHERA), as defined by its shape, which is not necessarily based on a descriptor of potential for inducing disease. As noted most dusts that cause pneumoconiosis (dust diseases) are not in the form of a fiber. An example of this is silicosis induced by the inhalation of non-fibrous crystalline silica. The fact that, much more is now known about the mechanisms of disease induction from breathing fibrous forms of a given dust since many of the fibrous forms are used in commercial products where human exposures are defined. However, in reality many fibrous dusts of amphibole minerals also contain cleavage fragments of the same mineral. Thus, distinguishing the potential “the various shapes of the inhaled dusts offer”, as individual “contributors” to induction of disease from such mixed exposures are difficult to distinguish. The debate as to the distinction of a short fiber from a cleavage fragment, as seen in the light microscope, shouldn’t be confused with health related issues. We do not know what fractions of those mixed dusts are capable of being inhaled and their roles individually or cumulatively may act as contributors to the development of disease in man.

The Senate Bill's exemption of asbestos materials containing less than 1% asbestos along with the House version that only exempts specific aggregate products containing less than 0.25% asbestos have no health basis and will pose grave risks to workers and consumers using these exempted products. The language should read that the presence of any asbestos, using the most sensitive analytical methods, is an indication of contamination and thus banned. Since the prevailing scientific consensus remains that no safe concentration for exposure to any form of asbestos has been identified, setting a percentage concentration or exempting any use as an integral part of a product is contrary to current health-based consensus.¹⁴ If either of these exemptions

¹⁴ Cook MB, 2004. Memorandum: Clarifying cleanup goals and identification of new assessment tools for evaluating asbestos at superfund cleanups. To: Superfund National Policy Managers, Regions 1-10, United States Environmental Protection Agency, Washington, D.C. Aug 10; Moatamed F, Lockey JE, Parry WT, 1986. Fiber contamination of vermiculites: A potential occupational and environmental health hazard. *Env Res*, 41: 207-218; Addison J, Davies LST, Robertson A, Willey RJ, 1988. The Release of Dispersed Asbestos Fibres From Soils. Report No. TM/88/14, UDC 553.676.614.7, Institute of Occupational Medicine, Edinburgh, September: 56 pgs; IPCS, 1998. Environmental

remain there will still be persons at risk of developing asbestos-related diseases and will result in less than a ban on asbestos.

The House Bill requires disposal of all asbestos containing products within 3 years but has no provisions for stopping sale or other distribution of these materials. The Bill should call for an immediate embargo of these products upon enactment of the bill with disposal no later than 6 months after the enactment of the bill; or embargoed until approval of application for exemption has been completed. All requests for exemptions must be submitted within 6 months of the Bill enactment.

Any exemption to the Chlorine Manufacturing industry must recognize the inherent dangers for both the worker and the public from the continued use of asbestos in the diaphragm-cell process. New non-asbestos using process are available

and can be used and with a reduction of energy requirement of 15-20%.¹⁵ I urge that, if an exemption is granted, it stipulate

¹⁵ Testimony of Dr. Barry Castleman before the U.S. Senate Committee on Environmental and Public Works, June 12, 2007: Asbestos has long been used in the diaphragm-cell process for making chlorine. This process and the old mercury-cell process are still operated, although a newer and more environmentally and technically superior membrane-cell process has been the only type built anywhere in the world for the past 20 years. Some diaphragm and mercury cell plants have been converted to membrane cells. Power requirements are substantial for chlorine manufacture, and the membrane cell process requires 15-20% less energy than diaphragm cells.

Asbestos exposures in the chlorine industry arise from transport and storage of sacks of asbestos, typically involving tears in the sacks that must be identified and sealed, with spillage cleaned with high-efficiency vacuum filters. Cutting open and emptying sacks of asbestos and transferring asbestos into slurry mixing tanks can cause additional exposures. The empty sacks are an additional exposure source, they must be carefully gathered up, placed in sealed containers, and landfilled at approved sites. Storage and handling of partially used sacks are also sources of exposure. If the slurry is spilled, this has to be meticulously cleaned up right away, because once it dries it becomes a source of airborne asbestos exposure. Handling and storage of prepared or purchased pre-deposited asbestos diaphragms can cause additional exposures. Hydro-blasting for removal/replacement of asbestos diaphragms is another possible source of area contamination, drying, and airborne exposure. The water used for hydro-blasting has to be contained and the asbestos filtered from it. The waste asbestos from this water and the spent diaphragms have to go to a landfill that accepts asbestos.

To some degree, workers can be protected against these asbestos exposures if they wear respirators that will remove some of the asbestos from the air they breathe, and if they wear personal protective clothing such as disposable coveralls. But these safeguards are partial. The respirators must be fit-tested and properly maintained; and even the protective clothing is a hazardous waste that requires special precautions for disposal. Chlorine Institute pamphlet 137, *Guidelines: Asbestos Handling for the Chlor-Alkali Industry*, recommends personal protective clothing and respirators only for workers exposed in excess of the

permitted limits in the OSHA standard, which is all that is legally required. But OSHA has admitted that compliance with its limits will not fully prevent deaths from asbestos. Dr. Richard Lemen and NIOSH epidemiologists estimate that exposure at OSHA's permissible exposure limit for asbestos will still cause 5 deaths from lung cancer and 2 deaths from asbestosis in every 1000 workers exposed for a working lifetime. (L. Stayner et al., Exposure-Response Analysis of Risk of Respiratory Disease Associated with Occupational Exposure to Chrysotile Asbestos. *Occ. Env. Med.* 54: 646-652, 1997).

While company manuals may state that the workers are supposed to observe various precautions to minimize asbestos exposure, there is virtually no OSHA inspection of these workplaces, and the usual combination of production demands, Gulf coast heat and humidity, and carelessness will assure that things are not always done "by the book" to minimize workers' asbestos exposure.

In the past 15-20 years, non-asbestos diaphragms have become available for relatively simple replacement in asbestos diaphragm cell plants. These are sold by Eltech/DeNora and PPG Industries in the US. The non-asbestos diaphragms cost more and last longer than asbestos. Although two-thirds of the chlorine made in the US in 2006 was from diaphragm cells, I don't know how many of these used non-asbestos diaphragms. The technology continues to advance, however, and has had wide acceptance in Europe, where the European Union's temporary exemption allowing asbestos use in chlorine manufacturing comes up for reconsideration next year. I understand that there are only 3 chlorine plants in Europe still using asbestos diaphragms.

PPG Industries has been a leader in the development of non-asbestos "Tephram" diaphragms, and PPG is also a major producer of chlorine in the US. I understand that PPG regularly replaces non-asbestos Tephram diaphragms in its asbestos diaphragm-cell units when they are taken down for periodic maintenance. I do not know of any technical reasons why other diaphragm-cell chlorine manufacturers could not do the same thing.

Therefore, if chlorine manufacturers want extra time to convert to non-asbestos technology, perhaps that could be allowed but with the requirement that when the equipment is shut down for maintenance overhauls, the new diaphragms used be non-asbestos. A similar several-year time frame might be allowed for diaphragm-cell units that manufacturers want to convert to membrane cells.

all Chlorine Manufacturing process shall be converted to non-asbestos usage within 6 years. In addition that during this conversion period that strict controls are in place to reduce the asbestos-exposures to workers at or below the OSHA PEL and air-pollution emissions below existing clean air standards as designated by the EPA.

I would conclude by saying that there are organizations purporting to represent constituencies aimed at curing, treating and preventing asbestos-related diseases while supporting both the Senate passed Asbestos Ban Bill and the exemptions that may be allowed in the current House version, which will result in less than a full asbestos ban and allowing multiple persons to remain at risk of asbestos-related diseases. These organizations are either; not in tune with the current science, or have some other agenda contrary to full prevention of asbestos-related diseases. I urge these organizations to re-think their positions and understand that asbestos-related diseases will never cease without a full asbestos Ban.

I would be happy to answer any questions you may have.

Thank you.