THE STATE OF SOUTH CAROLINA
In The Supreme Court

APPEAL FROM SPARTANBURG COUNTY
Court of Common Pleas

Jean H. Toal, Acting Circuit Judge

Appellate Case No. 2017-002611

Beverly Dale Jolly and Brenda Rice Jolly, ... Respondents,

v.

General Electric Company, et al., ... Defendants,

Of whom Fisher Controls International LLC and Crosby Valve, LLC are the..... Petitioner.

APPENDIX – VOLUME VII

C. Mitchell Brown A. Mattison Bogan James B. Glenn Nicholas A. Charles Nelson Mullins Riley & Scarborough LLP Post Office Box 11070 Columbia, SC 29211 (803) 799-2000 *Attorneys for Petitioner Fisher Controls International LLC and Crosby Valve, LLC*	Lisa W. Shirley (admitted pro hac vice) Jonathan M. Holder DEAN OMAR BRANHAM, LLP 300 N. Market Street, Suite 300 Dallas, TX 75202 Telephone: (214) 722-5990 Theile B. McVey John D. Kassel KASSEL MCVEY ATTORNEYS AT LAW P.O. Box 1476 1330 Laurel Street Columbia, SC 29201 *Attorneys for Respondents*

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THE STATE OF SOUTH CAROLINA
In The Court of Appeals

APPEAL FROM SPARTANBURG COUNTY
Court of Common Pleas

Jean H. Toal, Circuit Court Judge

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Case No. 2016-CP-42-1592

Beverly Dale Jolly and Brenda Rice Jolly, ... Respondents,

v.

General Electric Company, et al., ... Defendants,

Of whom Fisher Controls International LLC and Crosby Valve, LLC are the ..... Appellants.

RECORD ON APPEAL
VOLUME VII

C. Mitchell Brown A. Mattison Bogan James B. Glenn Nicholas A. Charles NELSON MULLINS RILEY & SCARBOROUGH LLP 1320 Main Street / 17th Floor Post Office Box 11070 (29211-1070) Columbia, SC 29201 (803) 799-2000 Attorneys for Appellants Fisher Controls International LLC and Crosby Valve LLC	Theile B. McVey, Esq. John D. Kassel, Esq. KASSEL McVEY ATTORNEYS AT LAW Post Office Box 1476 1330 Laurel Street (29201) Columbia, SC 29201 Jonathan M. Holder, Esq. Lisa White Shirley, Esq. (*Pro Hac Vice*) DEAN OMAR BRANHAM, LLP 302 North Market Street, Suite 300 Dallas, TX 75202 jholder@dobllp.com Attorneys for Respondents

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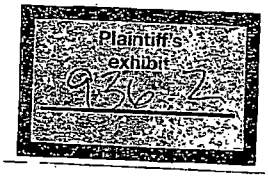
STATE OF PENNSYLVANIA §

COUNTY OF PHILADELPHIA §

AFFIDAVIT OF ARTHUR L. FRANK, M.D., Ph.D.

I am a Physician and Professor of Public Health at Drexel University where I hold the position of Chair Emeritus of Environmental & Occupational Health. I am also a Professor of Medicine at the Drexel University College of Medicine. I am also a Professor of Civil, Architectural and Environmental Engineering. I hold various adjunct professorships at various other universities. I am a Board Certified medical doctor, having received my medical degree in 1972, from the Mt. Sinai School of Medicine. I have been Board Certified by the National Board of Medical Examiners since 1973; have been a Diplomat of the American Board of Internal Medicine since 1978 and with the American Board of Preventive Medicine (Occupational Medicine) since 1979. I received my Ph.D. in 1977 from the City University of New York, where I studied in its Biomedical Sciences Doctoral Program. I have performed cancer research at the National Cancer Institute, participated in epidemiologic studies of asbestos-exposed populations, taught asbestos medicine and public health to medical students and doctors, and have devoted much of my professional life to the study and prevention of asbestos-related disease. I have published numerous peer-reviewed papers, book chapters and presentations on the topic of the causes and prevention of asbestos-related disease. In 2016, I received the Ramazzini Award from the Collegium Ramazzini for my "distinguished record of occupational health and safety research as well as his advocacy and service in the promotion of better occupational safety and health in developing countries and in the international fight to ban the use of asbestos." My current CV is available upon request. The opinions herein are based on my own work, experiences, publications and those cited. I have provided expert opinion in numerous jurisdictions, mostly for plaintiffs, on the causation between asbestos exposure and the development of mesothelioma and other asbestos-related diseases. While my employers have traditionally charged for much of my professional time working on such matters, I personally have not received any direct compensation for my medical-legal consulting. I have, however, used some of the revenue generated to support my university departments. I have not charged for any of the work on this affidavit.



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I hold the following opinions to a reasonable degree of medical and scientific certainty:

I. Outside of Court, the Mainstream Scientific Consensus, Based on a Weight-of-the-Evidence Approach, is that All Forms of Asbestos Can and Do Cause All of the Asbestos-Related Diseases

1. There is overwhelming, generally accepted evidence that inhalation of asbestos fibers of any type, from any source or product, causes mesothelioma (in all known locations), lung cancer, asbestosis, pleural plaques, and other cancers. In 2007, I joined fifty-one (51) other scientists in expressing my opinions about the hazards of asbestos in the article by Welch et al.,[1] entitled *Asbestos exposure causes mesothelioma, but not this asbestos exposure: an amicus brief to the Michigan Supreme Court.* I continue to hold the opinions set forth in that peer-reviewed, published paper, along with the other opinions expressed here, to a reasonable degree of medical and scientific certainty. Since 2007, I have co-authored a scientific amicus brief to Maryland's highest court, further setting out my medical and scientific viewpoints.[2] Similarly, I have reviewed and considered a scientific amicus brief filed by a group of fifty-eight (58) scientists from around the world in support of my medical and scientific opinions.[3] The 58 Scientists "reviewed the document, support the contents and asked to have their names listed. The signers received no compensation for their participation in [the] paper."[4] Most recently, in December 2015, I joined with sixty-six (66) other physicians, scientists, and scholars in a scientific amicus brief outlining the mainstream approach to assigning causation for asbestos-related disease.[5] The Welch (2007) and these other concerned scientists briefs set forth the broad consensus about the asbestos of asbestos and attribution of disease to asbestos exposure. On November 22, 2016, the Supreme Court of Pennsylvania affirmed judgment in *Ford Motor Company v. Rost*, citing the 58 Scientists for the "irrefutable scientific fact" that "each exposure to asbestos contributes to the total dose and increases the person's probability of developing mesothelioma or other cancers."[6] Furthermore, the *Rost* court, citing the 58 Scientists, recognized that I use "a generally accepted

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[1] Welch et al., *Asbestos exposure causes mesothelioma, but not this asbestos exposure: an amicus brief to the Michigan Supreme Court.* Int. J. Occup. Environ. Health. 13:318–327 (2007). This peer reviewed paper is a reliable review of the medical and scientific literature. Welch et al. (2007) presents the peer reviewed scientific position of the signatories and is a scientific – rather than legal – review of the mainstream approach to causation of asbestos disease.

[2] Brief of Amici Curiae Interested Physicians And Scientific Researchers In Support Of Appellant, *Dixon v. Ford Motor Co.*, September Term 2012 No. 82, Court of Appeals of Maryland (January 25, 2013).

[3] Brief of Akpinar-Elci, et al, Amici Curiae, *Rost v. Ford Motor Co.*, No. 56 EAP 2014, Supreme Court of Pennsylvania (filed April 7, 2015) ("58 Scientists").

[4] 58 Scientists p. 2, note 2.

[5] Amici Curiae Brief of Sixty-Seven (67) Concerned Physicians, Scientists, and Scholars Regarding Causation, *Scapa Dryer Fabrics, Inc. v. Knight*, No. S12C1278, In the Supreme Court of Georgia (December 29, 2015) ("67 Scientists").

[6] Opinion, *Rost v. Ford Motor Company*, In the Supreme Court of Pennsylvania – Eastern District, No. 56 EAP 2014 (Decided November 22, 2016) at page 17.

methodology [for attributing causation of an individual's disease], taking into consideration exposure history, individual susceptibility, biological plausibility, and relevant scientific evidence (including epidemiological studies)."[7]

2. Welch et. al. (2007) presents a weight-of-the-evidence review of the scientific data and information regarding the basis of knowledge supporting the conclusion that asbestos exposures from working with and around automobiles and automobile parts that contained asbestos, like all other kinds of asbestos exposure, can and does cause diseases. The published, peer-reviewed Welch article, and other similar weight-of-the-evidence reviews are a type of information routinely and customarily considered and relied upon by practicing occupational medicine physicians, such as myself, in our day-to-day practice outside the context of litigation. Indeed, Welch et. al. (2007) has been cited by other authors in the peer reviewed scientific literature.[8]

3. Outside the courtroom, there is little or no dispute in the medical literature that all asbestos fiber types, including chrysotile, cause asbestosis, lung cancer, and pleural plaques/thickening. The methodology and bases for the opinions as stated herein are not novel, and for the reasons set forth are generally accepted in the medical and scientific community.

4. There are numerous epidemiology, registry and case studies clearly linking all types of asbestos, including chrysotile asbestos, to mesothelioma of all sites including the pleura, peritoneum, pericardium and tunica vaginalis.[9]

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[7] Opinion, *Rost v. Ford Motor Company*, In the Supreme Court of Pennsylvania – Eastern District, No. 56 EAP 2014 (Decided November 22, 2016) at page 20.

[8] *See e.g.*, Ameille et al., *Asbestos-Related Diseases in Automobile Mechanics*, Ann. Occup. Hyg. 56(1):55-60 (2012); Collegium Ramazzini, *Asbestos Is Still With Us: Repeat Call for a Universal Ban*, Am. J. Ind. Med. 54:168-73 (2011); Lin et al., *Increased Standardised incidence Ratio of Malignant Pleural Mesothelioma in Taiwanese Asbestos Workers: A 29-Year Retrospective Cohort Study*, Biomed. Res. Int., Vol. 2015, Article ID 678598 (2015).

[9] Kanarek, *Mesothelioma from Chrysotile Asbestos: Update*. AEP Vol. 21, No. 9, pp. 688-97 (2011); Hein et al., *Follow-up study of chrysotile textile workers: Cohort mortality and exposure-response*. Occup. Environ. Med. 64:616–625 (2007); Loomis et al., *Lung cancer mortality and fiber exposures among North Carolina asbestos textile workers*. Occup. Environ. Med. 66:535–542 (2009); Silverstein et al., *Developments in asbestos cancer risk assessment*. Am. J. Ind. Med. 52:850–858 (2009); Finkelstein et al., *Malignant Mesothelioma among employees of a Connecticut factory that manufactured friction materials using chrysotile asbestos*. Ann. Occup. Hyg. 54(6):692–696 (2010); Egilman et al., *A case of occupational peritoneal mesothelioma from exposure to tremolite-free chrysotile in Quebec, Canada: A black swan case*. Am. J. Ind. Med. 54:153–156 (2010); Pira et al., *Mortality from cancer and other causes in the Balangero cohort of chrysotile asbestos miners*. Occup. Environ. Med. 66:805–809 (2009); Mirabelli et al., *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy*. Occup. Environ. Med. 65:815–819 (2008); Turci et al., *Role of associated mineral fibres in chrysotile asbestos health effects: The case of Balangeroite*. Ann. Occup. Hyg.; 53:491–497 (2009); Everatt et al., *Occupational asbestos exposure among respiratory cancer patients in*

5. "There is general agreement among scientists and health agencies . . . [e]xposure to any asbestos type (i.e., serpentine [chrysotile] or amphibole) can increase the likelihood of lung cancer, mesothelioma, and nonmalignant lung and pleural disorders."[10] Many other reviews support this conclusion, such as those from the American Conference of Governmental Industrial Hygienists,[11] the American Thoracic Society,[12] the Environmental Protection Agency,[13] the International Agency for Research on Cancer (IARC),[14] the National Toxicology Program,[15] the Occupational Safety and Health Administration,[16] the Consumer Product Safety Commission (CPSC),[17] the World Health Organization,[18] the Collegium Ramazzini,[19] and the World Trade Organization.[20] This

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Lithuania. Am. J. Ind. Med. 50:455–463 (2007); Madkour et al., *Environmental exposure to asbestos-response relationship with mesothelioma.* Eastern Mediterranean Health J. 15:25–38 (2009); Yano et al., *Mesothelioma in a worker who spun chrysotile asbestos at home during childhood.* Am. J. Ind. Med.; 52:282–287 (2009); Baumann et al., *Pleural mesothelioma in New Caledonia: An acute environmental concern.* Cancer Detect Prev. 31:70–76 (2007); Baumann et al., *Pleural mesothelioma in New Caledonia: Associations with environmental risk factors.* Environ. Health Perspect. 119:695–700 (2011); Nishikawa et al., *Recent mortality from mesothelioma, historical patterns of asbestos use, and adoption of bans: A global assessment.* Environ. Health Perspect. 116:1675–1680 (2008); Stayner et al., *The Worldwide Pandemic of Asbestos-Related Diseases.* Annual Rev. Public Health, 34: 4.1 – 4.12 (2013); Lin et al., *Cause-Specific Mortality in a Chinese Chrysotile Textile Worker Cohort.* J. Japanese Cancer Ass'n (2012); Gao et al. *Asbestos Textile Production Linked to Malignant Peritoneal and Pleural Mesothelioma in Women: Analysis of 28 Cases in Southeast China,* Am. J. Ind. Med., 58 (10): 1040-49 (2015).

[10] U.S. Public Health Service, U.S. Department of Health & Human Services. Toxicological profile for asbestos. Atlanta: Agency for Toxic Substances and Disease Registry; (2001).

[11] American Conference of Governmental Industrial Hygienists. Asbestos: TLV Chemical Substances 7th Edition Cincinnati OH: ACGIH; Report No.: Publication #7DOC-040 (2001).

[12] American Thoracic Society. *Diagnosis and initial management of nonmalignant diseases related to asbestos.* Am. J. Respir. Crit. Care Med.;170(6):691-715 (Sept. 15 2004).

[13] Environmental Protection Agency ("EPA"). Airborne Asbestos Health Assessment Update. Springfield VA: NTIS; Report No.: EPA/600/8-84/003F (1986).

[14] IARC. *Asbestos: Monograph on the Evaluation of Carcinogenic Risk of Chemicals to Man.* Lyon: International Agency for Research on Cancer; (1988); Straif et al., *A review of human carcinogens–part C: metals, arsenic, dusts, and fibres.* Lancet Oncol.; 10(5):453-4 (May 2009).

[15] National Toxicology Program. Report on Carcinogens, Thirteenth Edition. U.S. Department of Health and Human Services, Public Health Service (2014).

[16] Occupational Safety and Health Administration. Occupational exposure to asbestos; final rule. Federal Register; 59:40964-1162 (1994).

[17] Consumer Product Safety Commission. CANCER HAZARD! CPSC Warns About Asbestos in Consumer Products: Safety Alert. Report No.: CPSC Document #5080 (1994).

[18] World Health Organization. Environmental Health Criteria 203: Chrysotile Asbestos. Geneva: World Health Organization (1998); World Health Organization. Elimination of asbestos related diseases. Ref Type: Generic (2006); World Health Organization. Environmental Health Criteria 53: Asbestos and Other Natural Mineral Fibres. Geneva: World Health Organization (1986).

scientific consensus is also reflected in the Consensus Report of the 1997 Helsinki Conference,[21] the Consensus Report of the 2014 Helsinki Conference,[22] publications from the American Cancer Society,[23] publications from the National Cancer Institute of the National Institutes of Health[24] and Position Statement on Asbestos from the Joint Policy Committee of the Societies of Epidemiology (JPC-SE) June 4, 2012.

6. "There is a broad consensus that chrysotile asbestos causes human malignant mesothelioma. . . . The scientific basis for the mesothelial carcinogenicity of chrysotile is an established body of published epidemiological studies, animal carcinogen assays, and pleural fiber burden studies."[25] This is not a new concept: in 1986, Lilis' chapter on mesothelioma in NIOSH's Division of Respiratory Disease Studies, *Occupational Respiratory Diseases*, DHHS (NIOSH) Publication No. 86-102 (September 1986) observed that "[w]hile the number of mesothelioma cases from populations exposed only to chrysotile has been small, *an association with chrysotile exposure has been definitively established.*"

7. Recently, IARC published an update on asbestos which concluded "all forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite) . . . cause mesothelioma and cancer of the lung, larynx, and ovary. Also positive associations have been observed between exposure to all forms of asbestos and cancer of the pharynx, stomach, and colorectum."[26] IARC described its approach to assessing "causality":

> After the quality of individual epidemiological studies of cancer has been summarized and assessed, a judgment is made concerning the strength of evidence that the agent in question is carcinogenic

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[19] Collegium Ramazzini, *Asbestos Is Still With Us: Repeat Call for a Universal Ban.* Am. J. Indust. Med. 54:168-173 (2011).

[20] World Trade Organization. European Communities – Measures Affecting Asbestos and Asbestos – Containing Products. Report No.: WT/DS135/R (2000).

[21] Consensus Report, *Asbestos, Asbestosis, and Cancer: The Helsinki criteria for diagnosis and attribution.* Scan J. Work Environ Health, 23:311-6 (1997).

[22] Wolff et al., *Consensus Report: Asbestos, Asbestosis, and cancer, the Helsinki Criteria for Diagnosis and Attribution 2014: Recommendations*, Scand. J. Work Environ. Health 5, 41(1) (2015).

[23] American Cancer Society. Malignant Mesothelioma. 10-19-2006. Ref Type: Pamphlet (2006).

[24] National Cancer Institute. Factsheet - Asbestos: Questions and Answers. Bethesda, MD, National Institutes of Health. Ref Type: Pamphlet (2003).

[25] Markowitz et al., *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015).

[26] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012). In 2012, the IARC Working Group was evenly divided on whether the human evidence alone was strong enough to call the evidence sufficient for linking cancer of the colorectum, but it was agreed that "[t]there is sufficient evidence in experimental animals for the carcinogenicity of all forms of asbestos." Page 294.

to humans. In making its judgment, the Working Group considers several criteria for causality (Hill, 1965). A strong association (e.g. a large relative risk) is more likely to indicate causality than a weak association, although it is recognized that estimates of effect of small magnitude do not imply lack of causality and may be important if the disease or exposure is common. Associations that are replicated in several studies of the same design or that use different epidemiological approaches or under different circumstances of exposure are more likely to represent a causal relationship than isolated observations from single studies. If there are inconsistent results among investigations, possible reasons are sought (such as differences in exposure), and results of studies that are judged to be of high quality are given more weight than those of studies that are judged to be methodologically less sound.

If the risk increases with the exposure, this is considered to be a strong indication of causality, although the absence of a graded response is not necessarily evidence against a causal relationship. The demonstration of a decline in risk after cessation of or reduction in exposure in individuals or in whole populations also supports a causal interpretation of the findings.

Several scenarios may increase confidence in a causal relationship. On the one hand, an agent may be specific in causing tumours at one site or of one morphological type. On the other, carcinogenicity may be evident through the causation of multiple tumour types. Temporality, precision of estimates of effect, biological plausibility and coherence of the overall database are considered. Data on biomarkers may be employed in an assessment of the biological plausibility of epidemiological observations.

...

When several epidemiological studies show little or no indication of an association between an exposure and cancer, a judgment may be made that, in the aggregate, they show evidence of lack of carcinogenicity. Such a judgment requires first that the studies meet, to a sufficient degree, the standards of design and analysis described above. Specifically, the possibility that bias, confounding or misclassification of exposure or outcome could explain the observed results should be considered and excluded with reasonable certainty. In addition, all studies that are judged to be methodologically sound should (a) be consistent with an estimate of effect of unity for any observed level of exposure, (b) when considered together, provide a pooled estimate of relative

> risk that is at or near to unity, and (c) have a narrow confidence interval, due to sufficient population size. Moreover, no individual study nor the pooled results of all the studies should show any consistent tendency that the relative risk of cancer increases with increasing level of exposure. It is important to note that evidence of lack of carcinogenicity obtained from several epidemiological studies can apply only to the type(s) of cancer studied, to the dose levels reported, and to the intervals between first exposure and disease onset observed in these studies. Experience with human cancer indicates that the period from first exposure to the development of clinical cancer is sometimes longer than 20 years; latent periods substantially shorter than 30 years cannot provide evidence for lack of carcinogenicity.[27]

Based on this well-reasoned approach to assessing causality, the failure of a study or even multiple studies to detect a statistically significant increased risk of a rare disease such as mesothelioma in a particular group often provides little insight into whether a real risk actually exists. This is especially true where the studies were not designed to investigate the specific question of mesothelioma risk in a particular population (e.g. vehicle mechanics or drywallers), where the studies do not have sufficient statistical power (e.g. vehicle mechanics), or suffer from methodological weaknesses of the various studies, such as non-specific job or other exposure surrogate classifications.

8. After defining asbestos as all forms of this fibrous mineral, IARC stated that the "causal association between mesothelioma and asbestos has been well established." *Id.* IARC also discussed some unresolved questions such as potential differences in relative potency by fiber type and the issues of fiber length.

9. Recently, one hundred (100) scientists involved in the IARC Monograph process responded to industry criticism of IARC's approach to causality.[28] These scientists reaffirmed their support for IARC's approach to causality. They explained the process of assessing whether a substance is carcinogenic:

> IARC assessments of carcinogenicity are based on, and necessarily limited to, scientific evidence available at the time of the review. The evidence comes from epidemiologic studies, animal bioassays, pharmacokinetic/mechanistic experiments, and surveys of human exposure. The aim is to include all relevant papers on cancer in humans and experimental animals that have been published, or accepted for publication, in peer-reviewed scientific journals and also any publicly available government or agency documents that

[27] IARC. Preamble to Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012).
[28] Pearce et al., *IARC Monographs: 40 Years of Evaluating Carcinogenic Hazards to Humans*, Envir. Health Perspectives 123:6 507-514 (June 2015).

provide data on the circumstances and extent of human exposure. To that end, the search of the literature takes a comprehensive approach.

10. Mesothelioma is a tumor of the serosal linings of the chest (the pleura), the abdomen (peritoneum), the heart (pericardium) and testes (tunica vaginalis). The cells of the serosal membranes surrounding the lungs, abdomen, heart and testes are essentially the same, at a cellular level, and react to the presence of asbestos in the same manner. All forms of diffuse malignant mesothelioma, in any location in the body, can be caused by all forms of asbestos. The 2015 Italian Consensus statement agreed, stating "[a]ll types of asbestos fibres cause extrapleural, as well as pleural, MM."[29]

11. By virtue of its membership and process, the statements of the Collegium Ramazzini are synonymous with general acceptance within the medical and scientific community. I agree with the Collegium Ramazzini's most recent reaffirmation on the hazards of asbestos:

> The Collegium Ramazzini reaffirms its long-standing position that responsible public health action is to ban all extraction and use of asbestos, including chrysotile. Every mined fiber is indestructible. Since 1993, the Collegium Ramazzini has repeatedly called for a global ban on all mining, manufacture, and use of asbestos. The Collegium has taken this position based on well-validated scientific evidence showing that all types of asbestos, including chrysotile the most widely used form, cause cancers (such as mesothelioma and lung cancer) and additionally that there is no safe level of exposure. In 2006, the World Health Organization (WHO) called for the elimination of ARD, taking the position that the most efficient way to eliminate such diseases is to cease using all types of asbestos. The 2014 update of this statement, which was attached to the WHO document "Chrysotile Asbestos", published in response to the continuing widespread production and use of chrysotile, emphasized that all forms of asbestos, including chrysotile, are causally associated with an increased risk of cancer of the lung, larynx and ovary, mesothelioma and asbestosis. These observations are in line with the recent evaluation by the International Agency for Research on Cancer (IARC). Occupational exposure to asbestos causes an estimated 107,000 deaths each year worldwide. These deaths result from asbestos-related lung cancer (ARLC), mesothelioma and asbestosis. The WHO recently advanced a risk ratio of 6:1 for contracting lung cancer versus mesothelioma following chrysotile exposure.

[29] Magnani et al., *III Italian Consensus Conference on Malignant Mesothelioma of the Pleura. Epidemiology, Public Health and Occupational Medicine Related Issues,* Med Lav (106) 5:325-332 (2015).

> Regardless, the ARD burden is more likely to be under-than overestimated because ARD are well known to be under-diagnosed and reported. The ARD epidemic will likely not peak for at least a decade in most industrialized countries and for several decades in industrializing countries. In addition to workers, there should be monitoring of household members of workers if they bring asbestos into their homes. All countries with a history of asbestos use are experiencing an epidemic of ARD, with the stage of the epidemic being a function of the country's past asbestos use.[30]

12. Recently, yet another study recognized all forms of asbestos cause mesothelioma: "Malignant pleural mesothelioma (MPM), an aggressive cancer of the membranes lining the lungs, is strongly associated with inhalation of all types of asbestos fibres., and occurs after a long latency (median 44.6 years among males; 45.2 years among females)."[31]

13. Based on the evidence available, more than fifty (50) countries have now banned the use of all forms of asbestos.[32, 33, 34, 35] While the United States has not banned all uses of asbestos, OSHA most recently recognized that "[t]here is no "safe" level of asbestos exposure for any type of asbestos fiber. Asbestos exposures as short in duration as a few days have caused mesothelioma in humans. Every occupational exposure to asbestos can cause injury of disease; every occupational exposure to asbestos contributes to the risk of getting an asbestos related disease."[36]

14. I follow the same weight-of-the-evidence methodology used by IARC, WHO, NIOSH and ATSDR among others, in reaching my conclusions about the health effects of asbestos.[37] I, like those entities and many others, have considered the scientific and medical evidence in its totality, and I reach the same conclusions reached by the

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[30] Collegium Ramazzini, *The 18th Collegium Ramazzini statement: The global health dimensions of asbestos and asbestos-related diseases*, Scand J Work Environ Health 42(1):86-90 (2016).

[31] Merler, et al., *Residual fibre lung burden among patients with pleural mesothelioma who have been occupationally exposed to asbestos*, Occup. Environ. Med. doi:10.1136/oemed-2015-103382 [E-pub ahead of print] (2016).

[32] Stayner et al., *The Worldwide Pandemic of Asbestos-Related Diseases*. Annual Rev. Public Health, 34: 4.1 – 4.12 (2013).

[33] LaDou et al., *The Case for a Global Ban on* Asbestos. Environ. Health Perspectives 118:7 (July, 2010).

[34] Collegium Ramazzini, *Asbestos Is Still With Us: Repeat Call for a Universal Ban.* Am. J. Indust. Med. 54:168-173 (2011).

[35] Markowitz et al., *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015)(indicating 55 countries have banned asbestos).

[36] Occupational Safety & Health Administration ("OSHA"), *Safety and Health Topics – Asbestos*, https://www.osha.gov/SLTC/asbestos/ (accessed September 9, 2016) (citation omitted).

[37] IARC, NIOSH and ATSDR are not policy organizations; they offer scientific weight-of-the-evidence evaluations of the hazards of substances, including asbestos.

mainstream, including 58 other scientists. The following chart is helpful to understand the weight-of-the-evidence approach:

	Amosite	Crocidolite	Chrysotile
Cellular Damage	X	X	X
Genetic Damage	X	X	X
Animal Fibrosis	X	X	X
Animal Cancer	X	X	X
Human Asbestosis	X	X	X
Human Lung Cancer	X	X	X
Human Mesothelioma[38]	X	X	X

Considering all the forms of scientific evidence on causality of asbestos disease, as I have consistently since I began my scientific training, is not a new phenomenon. For example, in revisiting asbestos standards in 1976, NIOSH considered all of the evidence for causality of asbestos disease. In discussing the updated recommended standard in 1976, NIOSH observed that

> all commercial forms of asbestos are carcinogenic in rats, producing lung carcinomas and mesotheliomas following their inhalation, and mesotheliomas after intrapleural or ip injection. Mesotheliomas and lung cancers were induced following even 1 day's exposure by inhalation.
>
> . . .
>
> There are data that show that the lower the exposure, the lower the risk of developing cancer. Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure.[39]

Based on the available evidence in 1976, NIOSH stated:

> the standard should be set at the lowest level detectable by available analytical techniques, an approach consistent with NIOSH's most recent recommendations for other carcinogens (ie, arsenic and vinyl chloride). Such a standard should also prevent the development of asbestosis.

[38] This chart is for demonstrative purposes only. The scientific community has recognized that asbestos is a cause of lung cancer for 60+ years, but asbestos is also an accepted cause of other cancers as discussed in IARC. IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012).

[39] DHEW (NIOSH), Revised Recommended Asbestos Standard Publication No. 77-169 (1976).

> Since phase contrast microscopy is the only generally available and practical analytical technique at the present time, this level is defined as 100,000 fibers > 5 microns in length/m^3 (0.1 fibers/cc), on an 8-hour-TWA basis with peak concentrations not exceeding 500,000 fibers >5 microns in length/m^3 (0.5 fibers/cc) based on a 15-minute sample period. Sampling and analytical techniques should be performed as specified by NIOSH publication USPHS/NIOSH Membrane Filter Method for Evaluating Airborne Asbestos Fibers - T.R. 84 (1976).
>
> This recommended standard of 100,000 fibers > 5 microns in length/m^3 is intended to (1) protect against the noncarcinogenic effects of asbestos, (2) materially reduce the risk of asbestos-induced cancer (only a ban can assure protection against carcinogenic effects of asbestos) and (3) be measured by techniques that are valid, reproducible, and available to industry and official agencies.
>
> However, some difficulties arise in that specific work practices and innovative engineering control or process changes are needed. But because of the well-documented human carcinogenicity from all forms of asbestos, these difficulties should not be cited as cause for permitting continued exposure to asbestos at concentrations above 100,000 fibers >5 microns in length/m^3.

In December 1976, NIOSH issued its first Revised Recommended Asbestos Standard, recommending a lowering of the exposure limits for asbestos. "Primary responsibility for development of this document was shared by Richard A. Lemen and John M. Dement. With technical consultation provided by Dr. Joseph K. Wagoner."[40] NIOSH explained the reasons for the recommended change as follows:

> When the asbestos criteria document was first published in 1972, the National Institute for Occupational Safety and Health (NIOSH) recommended a standard of 2.0 asbestos fibers/cubic centimeter (cc) of air based on a count of fibers greater than 5 micrometers ([microns]) in length. This standard was recommended with the stated belief that it would "prevent" asbestosis and with the open recognition that it would not "prevent" asbestos-induced neoplasms. Furthermore, data were presented which supported the fact that technology was available to achieve that standard and that the criteria would be subject to review and revision as necessary. Since the time that the asbestos criteria were published in 1972, sufficient additional data regarding asbestos-related disease have been developed to warrant reevaluation.

[40] DHEW (NIOSH), Revised Recommended Asbestos Standard Publication No. 77-169 (1976).

15. In addition to being the mainstream methodology for assessing carcinogenicity, my approach to causation has been subjected to peer review.[41] Markowitz wrote:

> The risk of malignant mesothelioma due to asbestos is dose dependent, as amply demonstrated in many occupational cohort studies across a range of industries. Malignant mesothelioma is known to occur at lower levels of exposure to asbestos, and *no dose has been established below which there is no risk of malignant mesothelioma; that is, no "safe" threshold of cancer risk has been demonstrated.*
>
> Table 1 summarizes findings from several large case–control studies of malignant mesothelioma undertaken in numerous countries that evaluated the risk of mesothelioma by estimated occupational asbestos exposure. . . .
>
> *All studies in Table 1 show a sharp rise in mesothelioma risk with increasing asbestos exposure. This pattern signifies that at any level of occupational asbestos exposure, adding additional occupational exposure to asbestos increases the likelihood of developing malignant mesothelioma.*
>
> There are three consequences of this observed exposure–response pattern. First, for the individual who has had some occupational asbestos exposure, it is essential to avoid additional exposure to asbestos, because it adds, often dramatically, to their risk of developing malignant mesothelioma. Second, *in terms of attribution of malignant mesothelioma to prior asbestos exposure, it is clear that each increment in occupational asbestos exposure contributes significantly to the development of mesothelioma in the group*. For example, as shown in Table 1, in the two French and the German case–control studies, adding up to 1 fiber/mL-year of exposure to the lowest category of asbestos exposure elevates the exposed persons to the next higher category of asbestos exposure and doubles or triples the risk of malignant mesothelioma. [footnotes omitted] Third, *finding excess cancer risk at low levels of occupational exposure to asbestos supports the notion that there is no safe level of exposure to asbestos.*[42]

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[41] Markowitz et al., *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015).

[42] Markowitz (2015)(emphasis added). The publications summarized in Table 1 from Markowitz (2015), include: A. Lacourt et al., *Occupational and Non-Occupational Attributable Risk of Asbestos Exposure for Malignant Pleural Mesothelioma*, Thorax 1 (2014); Offermans et al., *Occupational asbestos exposure and risk of pleural mesothelioma, lung cancer, and laryngeal cancer in the prospective Netherlands cohort study*, J. Occup. Environ. Med. 56(1):6–19 (2014);

Table 1 Risk of malignant mesothelioma according to levels of occupational asbestos exposure: results of case–control studies

Cumulative exposure (fiber/mL-year)	Cases/controls	Odds ratio	95% CI
French study[32] no. 1			
Not exposed	95/154	1.0	–
0.001–0.49	77/109	1.2	0.8–1.8
0.5–0.99	29/12	4.2	2.0–8.8
1–9.9	80/27	5.2	3.1–8.8
≥ 10	49/10	8.7	4.1–18.5
German study[33]			
Not exposed	11/67	1.0	–
> 0–0.15	14/12	7.9	2.1–30.0
> 0.15–1.5	38/25	21.9	5.7–83.8
> 1.5–15	46/16	47.1	11.5–193
> 15	16/5	45.4	8.1–257
French study[23] no. 2 (males only)			99% CI
Not exposed	28/327	1.0	–
> 0–0.1	54/181	4.0	1.9–8.3
> 0.1–1	68/121	8.3	3.8–17.7
> 1–10	115/68	22.5	10.4–48.7
> 10	97/27	67.0	25.6–175.1
Intensity of exposure	Cases/Controls	Odds ratio	95% CI
Spanish study[42]			
Not exposed	30/127	1.0	–
Low	35/70	3.35	1.72–6.52
Medium	25/18	9.96	4.38–22.7
High	22/6	27.1	9.28–79.3

Abbreviation: CI, confidence interval.

16. As the data in Markowitz's Table 1 indicate, there is a clear dose response with all levels studied showing substantial, statistically significant, elevated risk. For example, in Lacourt et al. (2014), for the cumulative exposure estimate of more than zero but less than 0.1 f/cc/years, there was a substantially increased risk of mesothelioma (from nearly double and up to over eight (8) times the risk in unexposed population). This is the remaining risk from one year of exposure at the current Occupational Safety and Health Administration ("OSHA") Permissible Exposure Limit ("PEL"). It is possible for an individual patient to receive exposures in excess of 0.1 f/cc/years in a single day of certain work practices.[43]

Rödelsperger et al., *Asbestos and Man-Made Vitreous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results from a German Hospital-Based Case-Control Study*, 39 Am. J. Ind. Med. 262 (2001); and Iwatsubo et al., *Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study*, 148(2) Am. J. Epidemiol. 133 (1998).

[43] The 67 Scientist Brief (page 23, note 40) gave the following example:

> For example, the Health and Safety Laboratory (UK) has reported that dry removal of asbestos insulation (without control methods) can result in exposures as high as 358

17. The epidemiological evidence that all types of asbestos cause mesothelioma, lung cancer and other cancers discussed herein, is supported by evidence that all forms of asbestos cause numerous "mechanistic events" associated with carcinogenesis, including "[i]mpaired fibre clearance leading to macrophage activation, inflammation, generation of reactive oxygen and nitrogen species, tissue injury, genotoxicity, aneuploidy and polyploidy, epigenetic alteration, activation of signaling pathways, [and] resistance to apoptosis."[44] This evidence strongly supports the biologic plausibility that all forms of asbestos cause cancers, including mesothelioma. Because of these mechanistic events, the greater the exposure to asbestos, the greater the patient's chances of having the misfortune that target cells will become a cancer.

18. Cumulative exposure consistently is recognized, along with some additional factors such as time since first exposure, as the most important indicator of mesothelioma risk.[45] A British report explained that mesothelioma "has essentially only one cause – i.e. asbestos exposure."[46] Others recognize the same:

> Cumulative asbestos exposure, either directly or indirectly, remains the leading cause of mesothelioma. It has been previously determined that cumulative asbestos exposure leads to a proportional increase in mesothelioma risk. Mesothelioma can result from non-industrial environmental contact with asbestos fibers, and para-occupational exposure occurs; for example, women who have laundered their husband's work-related clothing. Cumulative asbestos exposure, either directly or para-occupational, remains the most common factor related to the development of mesothelioma.[47]

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fibers/cc. Health and Safety Authority, *Asbestos-containing Materials (ACMs) in Workplaces Practical Guidelines ACM Management and Abatement* (2013). It would take approximately three days at this level of asbestos exposure to reach the 4.5 f/cc/years permitted under the OSHA regulations.

[44] Straif et al., *A review of human carcinogens--part C: metals, arsenic, dusts, and fibres*. Lancet Oncol.; 10(5):453-4 (May 2009).

[45] Magnani et al., *Italian Consensus Conference on Malignant Mesothelioma of the Pleura. Epidemiology, Public Health and Occupational Medicine-related* Issues, Med. Lav. 106, 5:325-332 (2015) (observing that "[c]umulative exposure is a useful summary exposure index, successfully employed on various fields of cancer research (including etiological research and risk assessment). . . ").

[46] Darnton et al., *The burden of occupational cancer in Great Britain – Mesothelioma*, Health and Safety Executive Research Report RR861 (2012).

[47] Frontario et al., *Primary Peritoneal Mesothelioma Resulting in Small Bowel Obstruction: A Case Report and Review of Literature*, Am J Case Rep 16:496-500 (2015).

19. Darnton et al. (2012) concluded that "[w]orkers with the highest risks today are likely to be those subject to incidental exposures during the course of their work, for example, building maintenance workers."[48]

20. It is important to understand that most people, if not all, have been exposed to asbestos at some point in their lives. This is because of the widespread use of asbestos and, to a lesser degree, from naturally occurring asbestos. Numerous investigators have identified ranges of asbestos in ambient air. Sometimes scientists refer to this as "background" exposure to asbestos. According to the Agency for Toxic Substances and Disease Registry (ATSDR), "there typically would be 0.00001 fibers/mL of asbestos in air in rural areas.[49] Recently, the ATSDR noted rural ambient asbestos levels two orders of magnitude lower, ranging from 0.0000003 - 0.00000003 f/cc.[50] To put this into context, an exposure of 1.0 f/cc is one hundred thousand times (100,000X) the highest rural ambient level reported and is thirty-three million times (33,000,000X) the lowest rural ambient level reported by ATSDR. The graph below illustrates the significance of occupational exposures to asbestos as compared to ambient levels.[51] Thus, it takes less than two hours at the current OSHA PEL (0.1 f/cc) to inhale a lifetime's worth of asbestos fibers in the rural ambient air.

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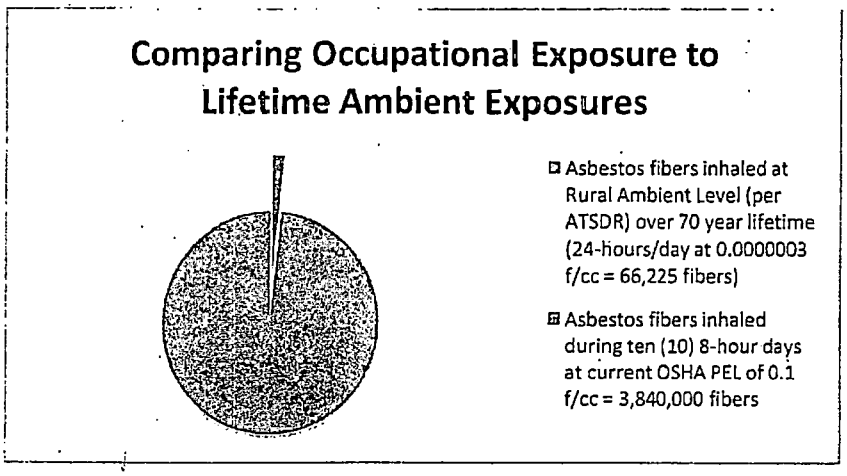
[48] Darnton et al., *The burden of occupational cancer in Great Britain – Mesothelioma*, Health and Safety Executive Research Report RR861 (2012).

[49] U.S. Public Health Service, U.S. Department of Health and Human Services. Toxicological profile for asbestos. Atlanta, GA: Agency for Toxic Substances and Disease Registry (2001).

[50] U.S. Department of Health and Human Services. Health Consultation Evaluation of Community-Wide Asbestos Exposures EL DORADO HILLS NATURALLY OCCURRING ASBESTOS SITE EL DORADO HILLS BOULEVARD, EL DORADO HILLS, CALIFORNIA EPA FACILITY ID: CAN000906083 AUGUST 16, 2011. Atlanta, GA: Agency for Toxic Substances and Disease Registry (2011).

[51] The following assumptions were used to produce ten (10) day exposure at the OSHA PEL of 0.1 f/cc: Working respiration rate = 16 breaths/minute; one breath = 500 cc; 16 breaths/minute X 500cc X 0.1 f/cc X 60 minutes X 8 hours X 10 days = 3,840,000 fibers in 10 days at OSHA PEL. The lifetime ambient fiber number was calculated based on the following assumptions: Resting respiration rate = 12 breaths/minute; one breath = 500 cc; rural ambient per ATSDR = 0.0000003 f/cc; 12 breaths/minute X 500 cc/breath X 60 minutes/hour X 24 hours/day X 365 days/year X 70 years X 0.0000003 = 66,225 fibers.



21. Ambient exposure to asbestos has not been shown to be without risk of mesothelioma.

22. "Using conservative assumptions, a person breathing 12 times per minute with each breath being 500 mL of air, that person would inhale approximately 86 fibers per 24 hour day at an ambient background level of 0.000001 f/mL."[52] In stark contrast, assuming an 8-hour TWA exposure of 0.1 f/cc (the OSHA Permissible Exposure Limit ("PEL")) for one day would inhale 384,000 fibers/8-hour work day.[53] Thus, at the current OSHA PEL, a person will inhale 4,465 times (4,465X) the amount of asbestos inhaled during a 24-hour period at the higher rural ambient levels reported by ATSDR.

23. The Collegium Ramazzini recently observed – in response to an asbestos-industry expert's hypothesis that only the most distant exposures contribute to the risk of mesothelioma – that "the risk of malignant mesothelioma is related to cumulative exposure to asbestos in which all exposures – early as well as late – contribute to the totality of risk."[54]

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[52] 67 Scientists page 23 (note 39).

[53] This calculation was made assuming a working respiration rate of 16 breaths/minute, 500 cc/breath, 480 minutes/day (0.1 f/cc X 16 breaths/minute X 500 cc/breath X 480 minutes/8-hour day = 384,000 fibers per day).

[54] Collegium Ramazzini, *Comments On The Causation Of Malignant Mesothelioma: Rebutting The False Concept That Recent Exposures To Asbestos Do Not Contribute To Causation Of Mesothelioma*, http://www.collegiumramazzini.org/download/Causation_Malignant_Mesothelioma_Comments(2015).pdf accessed December 16, 2015. "The Collegium derives its name from Bernardino Ramazzini, the father of occupational medicine, a professor of medicine of the Universities of Modena and Padua in the late 1600s and the early 1700s. The Collegium is comprised of 180 physicians and scientists from 35 countries, each of whom is elected to membership. The Collegium is independent of commercial interests." *Id.* I am a fellow of the Collegium.

24. Recently, fifty-eight (58) uncompensated concerned physicians and scientists described my approach to attributing cause to asbestos-related disease in a patient as "the generally accepted scientific methodology of reviewing the diagnosis, medical and occupational history, individual susceptibility, biological plausibility, and relevant case reports, case series, and epidemiological studies in order to" attribute causation.[55] In addition, I was a co-author of the Welch et al. (2007) peer reviewed, published amicus brief with 51 other scientists and of an amicus brief filed with the highest court of Maryland in *Dixon v. Ford Motor Co.* 70 A.3d 338 (Md. 2013). I co-authored this brief with 26 other scientists. Through those briefs, 108 physicians and scientists have endorsed my approach to causation. Appendix A is a list of the signatories who explained the mainstream, outside-the-courtroom scientific approach to causation in an asbestos injury case. This list contains many well-known asbestos researchers – none of whom were compensated for their participation in writing the briefs – including the current head of OSHA, Dr. David Michaels; the deceased former head of the IARC Monograph program, Lorenzo Tomatis; Eula Bingham (former head of OSHA); Dr. John Dement, epidemiologist and co-author of the National Institute of Occupational Safety and Health's (NIOSH) first Criteria Document on Asbestos; and Dr. Richard Lemen, epidemiologist and former Assistant Surgeon General who co-authored NIOSH's first Criteria Document for asbestos with Dr. Dement. Although some of these world-renown experts have testified in the U.S. legal system, many have not. These other physicians, scientists and scholars and I agree on the content of these publications and briefs because they are well-supported scientific documents written to enlighten the courts to which they were submitted.

25. My approach to attribution of asbestos-related disease in an individual, sometimes referred to as "specific causation" in the legal setting, has been recognized by legal commentators as the preferred method for attributing causation in a specific individual.[56] In explaining a reasonable approach to medical causation testimony, the Reference Manual provides:

> Determining external causation also generally occurs in a stepwise fashion. In the first step the physician must establish the characteristics of the medical condition. Second, he or she carefully defines the nature and amount of the environmental exposure. The third step is to demonstrate that the medical and scientific literature provides evidence that in some circumstances the exposure under consideration can cause the outcome under consideration. This step is synonymous with establishment of general causation. As part of this step, the clinician attempts to establish the relationship between dose and response, including whether thresholds exist, ultimately defining the clinical toxicology of the exposure. The fourth step is to apply this general

[55] 58 Scientists.

[56] Federal Judicial Center's Reference Manual on Scientific Evidence, Medical Testimony, at pp. 468-69 (West 2d Ed. (2000).

knowledge to the specific circumstances of the case at hand, incorporating the specifics of exposure, mitigating or exacerbating influences, individual susceptibilities, competing or synergistic causes, and any other relevant data.

26. For more than forty (40) years, I have used this same approach to ascribing causation of cancer to a substance. In this regard, it is important to note that with respect to asbestos and disease, in over forty (40) years of researching asbestos diseases outside of the courtroom, examining patients in clinical settings, and in participating as an expert witness in thousands of cases of asbestos disease, I have never had contemporaneous dust measurements from any specific worker who had later developed asbestos disease or had been exposed. Neither in my clinical and research practice outside of the context of litigation, nor in the court cases in which I have been an expert, has this prevented me or other experts from formulating scientifically valid opinions regarding causation in individuals with asbestos diseases and histories of exposure. In forming those opinions, I have always used, and continue to use, the same analysis and methodologies in court cases that I have used for decades in my clinical and research practice, regarding asbestos or other exposures.

27. Scientists do not require epidemiological studies of every job category or every product to conclude that the toxic ingredient caused a signature injury of that toxin.[57] As Dr. Selikoff properly stated, "[t]he floating fibers do not respect job classifications."[58] For example, scientists and physicians will have no trouble linking an individual lung cancer to cigarettes in a 5 year Marlboro smoker, even though there are no epidemiological studies of Marlboro-only smokers, even though we know that different cigarettes have different ingredients and even though that individual also smoked Winston, Pall Mall and or other brands at various other points in their life. Similarly, it's unlikely that a physician would think twice about attributing a poisoning death to arsenic in coffee even though there are no epidemiologic studies of people who ingested arsenic in coffee. Thus, even though there are no well-designed epidemiological studies of workers who worked with chrysotile asbestos joint compounds, or other such exposures, the mainstream medical and scientific community has no trouble attributing the patient's mesothelioma to this chrysotile exposure.

28. Radiographic or other evidence of asbestos exposure, such as asbestosis, is not required to link mesothelioma or lung cancer to asbestos exposure; a history of exposure is sufficient. This concept has been discussed extensively and is supported by numerous well-reasoned scientific papers.[59] Finkelstein (2010) found an elevated risk of lung

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[57] Lemen, Asbestos: Risk Assessment, Epidemiology, and Health Effects. 2d Ed., Chapter 5 *Epidemiology of Asbestos-Related Diseases and the Knowledge that Led to What is Known* Today, Boca Raton: Taylor and Francis (2011) at page 170.

[58] Selikoff et al., *Asbestos Exposure and Neoplasia.* JAMA 22- 26 (1964).

[59] Finkelstein, *Absence of Radiographic Asbestosis and the Risk of Lung Cancer Among Asbestos-Cement Workers: Extended Follow-Up of a Cohort*, Am. J. Indus. Med. 53:1065-1069 (2010); Wolff et al., *Consensus Report: Asbestos, Asbestosis, and cancer, the Helsinki Criteria*

cancer among asbestos cement workers as follows: Among asbestos-cement workers without radiographic asbestosis at 20 years latency the lung cancer SMR was 3.84 (2.24–6.14). Among workers without asbestosis when examined at 25 years latency the SMR was 3.69 (1.59–7.26)" and concluded that "workers from an Ontario asbestos-cement factory who did not have radiographic asbestosis at 20 or 25 years from first exposure to asbestos continued to have an increased risk of death from lung cancer during an additional 12 years of follow-up."[60]

29. In a large epidemiological study of sheet metal workers, Welch et al. (2015) found significant excess mortality for mesothelioma and asbestosis.[61] According to the authors,

> Significant excess mortality was seen for mesothelioma and asbestosis. Controlling for smoking, a strong trend for increasing lung cancer risk with increasing chest x-ray profusion >0/0 was observed. With a[] profusion score <1/0, FEV1 /FVC <80% was associated with lung cancer risk. COPD risk increased with increasing profusion score. This study demonstrates asbestos-related diseases among workers with largely indirect exposures and an increased lung cancer risk with low ILO scores.[62]

This shows increased lung cancer risk without evidence of asbestosis on X-ray. The great weight of the evidence supports my opinion that no clinical markers of asbestos exposure are required to link a cancer to asbestos exposure.

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for Diagnosis and Attribution 2014: Recommendations, 41(1) Scand. J. Work Environ. Health 5 (2015); Egilman et al, *Lung Cancer and Asbestos Exposure: Asbestosis is Not Necessary*, Am. J. Indus. Med. 30:398-406 (1996).

[60] Finkelstein, *Absence of Radiographic Asbestosis and the Risk of Lung Cancer Among Asbestos-Cement Workers: Extended Follow-Up of a Cohort*, Am. J. Indus. Med. 53:1065-1069 (2010)

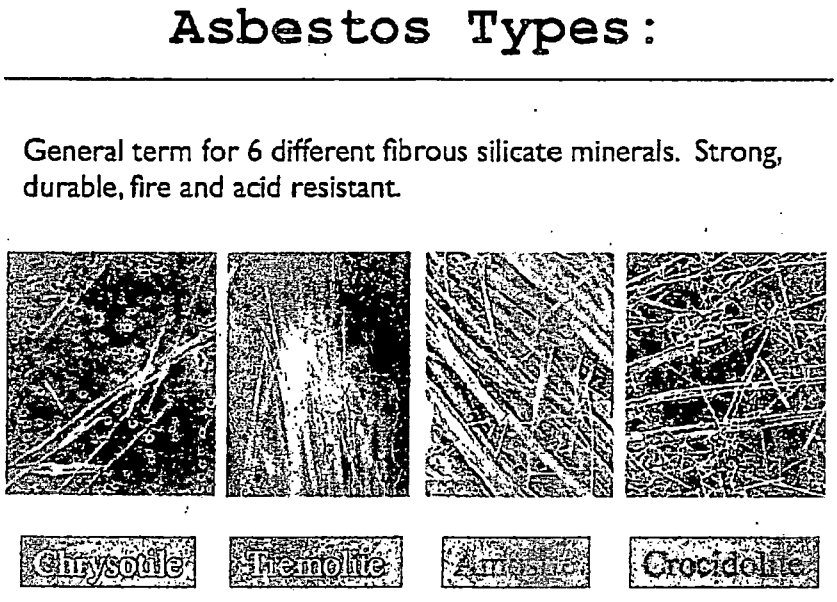
[61] Welch et al., *Mortality among sheet metal workers participating in a respiratory screening program*, Am. J. Industr. Med. 58(4):378-91 (2015).

[62] *Id.*

II. Information About the Hazards of Asbestos was Available from the Early Twentieth Century.

30. I have authored a textbook chapter entitled *The History of Extraction and Uses of Asbestos* in Dodson & Hammar, *Asbestos: risk assessment, epidemiology, and health effects*, CRC Boca Raton (1st Ed. 2006). Much of this section is taken directly from that chapter.

31. Asbestos is a commercial term used to describe two families of naturally occurring minerals. Amphiboles, containing five fiber types, and the serpentine variety, chrysotile, were materials known to the ancients. The following accurately depicts the appearance of the four types of asbestos most commonly found in products:



32. While these different types of asbestos have different elemental makeup, they all cause asbestos-related diseases. Claims by industry-aligned scientists that chrysotile is a less potent carcinogen due to its relative lack of iron content, are unsupported. As Stayner et al. (1996) explained,

> comparison of the carcinogenic potencies of fibers in the rat in vivo does not support the hypothesis that carcinogenic potency is related to iron content. As discussed above, Wagner et al. observed similar numbers of tumors in rats with crocidolite, amosite, and chrysotile, even though these fibers have an elemental iron content of 40%, 28% and less than 1%, respectively. The

> nonasbestos mineral erionite does not include iron as a constituent but is nonetheless a potent mesothelioma inducer in rats.[63] . . .
>
> Therefore, no obvious correlation between iron content and carcinogenicity is apparent in the rat.[64]
>
> Erionite is also recognized as a potent cause of mesothelioma in humans, despite its lack of iron.

33. More than 4,000 years ago, pottery in Africa and Finland contained asbestos, and Finnish homes were known to contain asbestos rock to pack crevices in log huts. The lamps of the Vestal Virgins in ancient Rome had wicks made from asbestos so the lamps would burn continuously, as long as they were filled with oil. Various Roman historians noted slaves working in asbestos mines were not as healthy as others, and were thought to die young.[65]

34. Charlemagne, Emperor of the Holy Roman Empire, was said to have possessed a tablecloth woven of asbestos, and would astonish his guests by cleaning his tablecloth in a roaring fire.[66] Additional history of the early use of asbestos can be found in the paper by Abratt et al.[67]

35. By 1850, chrysotile deposits were known around Thetford, in Canada. These deposits were again appreciated following a forest fire when in the mid-1870s outcroppings of rocks were noted to not have burned. By 1876, some 50 tons of asbestos was being mined in Quebec and brought to market through a specially built railroad. By the 1950s, over 900,000 tons per year were being mined with a value of almost 100 million dollars.[68]

36. In the early 1800s, asbestos was identified in South Africa,[69] particularly in the northwest area of Cape Province, where the name crocidolite was given to a blue-colored stone otherwise known as "wooly stone." Further interest did not occur until the 1880s and the first records of serious production did not take place until early in the twentieth century. The amount of asbestos produced was far less than from Canada, remaining below

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[63] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis,* Am. J. Public Health 86(2) (1996).
[64] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis,* Am. J. Public Health 86(2) (1996).
[65] Selikoff, Irving J., and D.H.K. Lee. Asbestos and Disease. (Academic Press, New York 1978).
[66] Stayner et al., *The Worldwide Pandemic of Asbestos-Related Diseases.* Annual Rev. Public Health, 34: 4.1 – 4.12 (2013).
[67] Abratt et al., *Asbestos and Mesothelioma in South Africa.* Lung Cancer. 45:S3-S6 (Supp.) (2004).
[68] Stayner et al., *The Worldwide Pandemic of Asbestos-Related Diseases.* Annual Rev. Public Health, 34: 4.1 – 4.12 (2013).
[69] Selikoff, Irving J., and D.H.K. Lee. Asbestos and Disease. (Academic Press, New York 1978).

10,000 tons per year until 1940. In the Transvaal of South Africa a different form of asbestos was mined and was called amosite, an acronym for the Asbestos Mines of South Africa. By 1970, some 80,000 tons per year of amosite was being produced. The mines from which the majority of amosite was derived were run by a small number of Europeans with 6,500 local workers of color.

37. Other locations with significant production of asbestos included Italy, Russia, the United States, Brazil, Rhodesia (now Zimbabwe), and more recently, China. Italy was never a major producer of asbestos, not being able to compete with the larger quantities available in Canada. Russian production was substantial, rivaling that produced in Canada. Russian mines produce primarily chrysotile. In the United States, deposits were mined in Vermont, Arizona, and California. Smaller deposits of anthophyllite were mined in North Carolina and Georgia. In Zimbabwe, mines became operative early in the twentieth century and reached a peak production of 95,000 tons.

38. China has become a major producer and rivals Russia in terms of asbestos production. In 2000, Russia led the world with 700,000 tons, followed by 450,000 tons from China and 335,000 tons from Canada. Canada recently halted production of asbestos. In 2000, the United States was producing only some 7,000 tons from mines in California and elsewhere, this out of a worldwide production of 2,130,000 tons.[70] Not surprisingly, Russia and China accounted for most consumption of asbestos followed by Brazil, India, Thailand, and Japan. The United States used about 15,000 tons of asbestos in 2000, down from a peak of 803,000 tons per year in the early 1970s. At the present time, the United States imports less than 2,000 tons.

[70] Tossavainen, *Global Use of Asbestos and the Incidence of Mesothelioma.* Int. J. Occup. Environ. Health. 10:22 (2004).

39. On a per capita basis, as of about 2014, the greatest use of asbestos is in Russia and former Soviet Republic countries, and in Thailand. Among the countries with lowest per capita usage, other than in countries that have now banned asbestos, are Canada, the United States, and several others at one tenth of a kilogram per capita per year. Although on a per capita basis India ranks low, it stands second in the world's total usage. China, while first in the world, also has a relatively low per capita amount, given its large population base. Major use in the United States is for asbestos cement and roofing materials. In much of the rest of the world asbestos containing cement, construction materials, friction products, and textiles are made, used, and exported. The following figure, reproduced from Frank and Joshi, *The Global Spread of Asbestos*, Ann. Global Health 80(4): 257 - 62 (2014), summarized recent data on the use of asbestos:

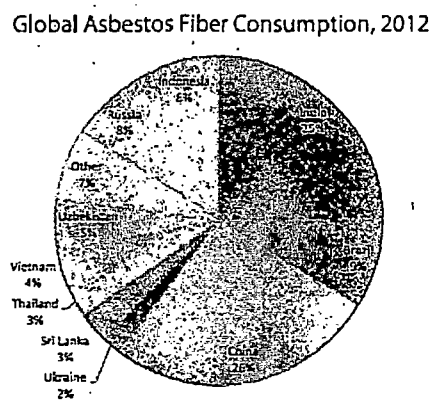

Figure 4. Global asbestos fiber consumption, 2012* *Source International Ban Asbestos Secretariat & Citi Research

Commercial Uses of Asbestos

40. Although there has long been historical use of asbestos, it was originally more a curiosity than a meaningful commercial material. This changed in the last half of the nineteenth century as asbestos began to be used in many commercial settings. For example, with industrialization and the use of steam to drive equipment, it was recognized that asbestos could serve a useful purpose as insulation material.

41. It became increasingly apparent that asbestos, because of its various properties, was extremely useful in many situations. Asbestos resists degradation under heat and cold, does not conduct electricity, and is extremely chemically resistant, including having resistance to many industrial acids. Because of its heat, cold and chemical resistance, asbestos was used in many products. Different types of asbestos were found especially useful for different purposes.

42. In the nineteenth century, the first systematic use of asbestos was for sealing and packing materials, soon followed by its use in the insulation for heat conservation. The

manufacture of asbestos roofing felt and cement came soon thereafter, as did the development of textile made from asbestos.[71]

43. Around the turn of the century asbestos containing cement pipe was produced. The asbestos allowed for added strength, creating lighter and thinner cement materials. The first use of asbestos as a brake lining occurred in 1906, and clutch facings were developed in 1918. In Great Britain, a technique for spraying asbestos as a fireproofing material was developed in the early 1930s, and this technique was imported into the United States a few years later. Considerable use of asbestos was noted during the shipbuilding era in and around World War II. For the first time millions of people, including many women, were exposed to asbestos.

44. After World War II, asbestos was used as a material in plastics, in building materials such as joint compound, spackling, plaster, paint, asphalt, acoustic material, reinforcement for cement siding, and many other new uses. Asbestos was used for filtering wine, beer, and pharmaceutical products. Crocidolite asbestos was even used as a component of one brand of cigarette filters between 1952 and 1956.

45. Asbestos found its way into plasters and stuccos, was used in drilling mud for oil wells and other similar operations, and was used in automobile body under-coatings. Yarns made from asbestos were used in a wide variety of ways, including rope, sewing threads, gas mask filters, wire covering, and for steam hoses, among others. Cloth made from asbestos was incorporated into blankets, mailbags, theater curtains and commercial products such as ironing board covers. Other consumer products, including hair dryers, bowling balls, toasters, play sand, and baby and adult talcum powders were shown to contain asbestos.

46. Construction materials containing asbestos included millboards, cements, laboratory table tops, electrical pump insulation and mountings, and flooring. This listing of products is by no means comprehensive - asbestos was used in 3,000-4,000 commercial products.

47. Increasingly, the use of asbestos is being banned around the world. Even Canada has now effectively closed the Quebec asbestos mines. The current use of asbestos includes building supplies, such as roofing materials and asbestos cement pipes. Automobile brake components continue to contain asbestos, and asbestos cloth is still used in firefighting protective gear. For some countries, the continued sale of asbestos is a significant economic issue. This is in the face of irrefutable evidence of the health hazards of all forms of asbestos, and continuing evidence, especially in developing countries, of no real "controlled use" of asbestos, including chrysotile.

48. With the ban of the use of asbestos in Japan, only developing countries continue to use large quantities of asbestos. China and India, for example, continue to mine and use asbestos, the most frequent use being in construction materials. Thailand, another

[71] Stayner et al., *The Worldwide Pandemic of Asbestos-Related Diseases*. Annual Rev. Public Health, 34: 4.1 – 4.12 (2013).

growing economic power in Southeast Asia, continues to use large quantities of asbestos as well. Encouragement for the use of asbestos in such countries comes from the West, where the hazards are increasingly well recognized and actions are being taken internally to reduce or eliminate the use of asbestos containing products.

Public Health Issues and the Uses of Asbestos

49. The world has a long history of asbestos use, with some suggestions of potential health hazards by the ancients. The real history appreciating the hazards of asbestos begins in the late 1890s.

50. The term pneumoconiosis, having been coined by Zenker[72] in 1867 after examining the lungs and pleura of a man with siderosis, was applied to an increasing number of dust diseases of the lung. In 1924, Cooke coined the term asbestosis.[73]

51. Morris Greenberg, who served as a medical member of the Inspectorate of Factories in Great Britain and is a scholar of the historical aspects of asbestos-related disease, wrote excellent historical overviews of the development of knowledge regarding the hazards of asbestos and the development of mesothelioma.[74] These articles provide an excellent historical account of one aspect of the development of knowledge about the hazards of asbestos and the failings of some in the medical community.

52. In Great Britain, as early as 1898, the Lady Inspector of Factories made note of the fact that asbestos was causing disease among asbestos textile workers.[75] In 1899, Dr. H.M. Murray conducted a post-mortem examination on a young man in his mid-thirties who died of respiratory insufficiency. Dr. Murray reported, during the patient's hospitalization, that the patient was the tenth individual in his particular work area to die, and that his working brethren had all preceded him in death at a young age from similar problems. Dr. Murray noted the man had extensive interstitial fibrosis, and what was described as "curious bodies" in his lungs. In 1907, the autopsy findings, with commentary, were published and optimistically concluded that proper ventilation was now thought to be in place to spare additional workers disease in the future.[76] Unfortunately this was far from correct.

53. In 1915 Collis, after giving a series of lectures, wrote up his findings on pneumoconiosis and discussed the problems of silicosis and asbestos-induced fibrosis, not yet called

[72] Zenker, *Iron Lung-Siderosis Pulmonous*. Dtsc. Arch. Klin. Med. 2:116 (1867).
[73] Stayner et al., *The Worldwide Pandemic of Asbestos-Related Diseases*. Annual Rev. Public Health, 34: 4.1 – 4.12 (2013).
[74] Greenberg et al., *The Doctors and the Dockers*. Am. J. Ind. Med. 45:573 (2004); Greenberg et al., *Mesothelioma Register 1967-68*. Br. J. Ind. Med. 31:91 (1974).
[75] Annual Report of the Chief Inspector of Factories and Workshops for the Year 1898, Her Majesty's Stationery Office, p. 171 (1898).
[76] Murray, H.M. Departmental Committee on Compensation for Industrial Disease, Minutes of Evidence, Appendices and Index, p. 127 (Wyman and Sons, London, 1907).

"asbestosis."[77] The term asbestosis was not used until 1924, when Cooke coined the term to describe pulmonary fibrosis due to the inhalation of asbestos dust.[78] By 1930, Merewether and Price wrote of the principles to protect workers in England,[79] and Lanza in the United States showed that suggested levels of asbestos in the late 1930s were often too high to protect workers.[80]

54. Although previously unnamed, the disease entities caused by exposure to asbestos were not unappreciated. In 1918, a vice president of the Prudential Life Insurance Company, who was a statistician, informed the company there was harm in breathing asbestos dust. At this point in time, Prudential ceased issuing policies on the lives of asbestos workers in the United States and Canada.

55. Although not reported in the scientific literature until many decades later by Tweedale, relatively recent revelations written up revealed at least one major asbestos company in England knew, beginning in the 1920s that its workers were dying of lung cancer and mesothelioma. This company worked diligently to suppress this information.[81]

56. Since 1930, and probably earlier, asbestos dust had been recognized as a hazard wherever visible dust could be seen. In 1930, Merewether and Price stated that "[i]f there is visible asbestos dust, then the invisible dust is in dangerous concentration."[82] In 1935, another insurance company engineer published that "[i]f you can see the dust, you know it to be a terrific hazard."[83]

57. Beginning in 1946, the American Conference of Governmental Industrial Hygienists ("ACGIH") began publishing a list of Maximum Allowable Concentrations ("MAC") and later published Threshold Limit Values ("TLV") for various harmful substances, including asbestos. The first MAC for asbestos was set "without any review of research or data" and the committee wrote that the values were "not to be construed as recommended safe concentrations."[84] This TLV level, designed only to reduce

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[77] Collis et al. *The Pneumoconiosis*. Publ. Health. 28:252-264 (1915).
[78] Cooke, *Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust*. Br. Med. J. 2, p. 147 (July 26, 1924).
[79] Merewether et al., *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry*. Her Majesty's Stationery Office (1930).
[80] Lanza, *Silicosis and Asbestosis*, Etiology, Symptoms, Diagnosis Oxford University Press, page 59 (1938).
[81] Tweedale, From Magic Mineral to Killer Dust: Turner and Newall and the Asbestos Hazard. Oxford University Press (Oxford, 2000).
[82] Merewether et al., *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry*. Her Majesty's Stationery Office (1930).
[83] Johnson, *No Halfway Measures in Dust Control*, National Safety News, (Sept. 1935) (noting a difference between pure silica and asbestos dust but observing "[i]f you can see the dust, you know it to be a terrific hazard.").
[84] Egilman et al., *The origin and development of the asbestos Threshold Limit Value: scientific indifference and corporate influence*, Int. J. Health Serv., 25(4) :667-96 (1995).

asbestosis, was "known to be inadequate when first proposed, was severely criticized between 1946 and 1968, but nonetheless was promulgated annually and remained unchanged until 1971."[85]

58. The protective measures necessary to prevent asbestos disease are the same for asbestosis, lung cancer, mesothelioma or other malignancies. A company that protected its workforce, their families, and bystander co-workers against any asbestos-induced disease would have reduced the risk to its work force from all asbestos-induced diseases. Indeed, if the company used the most basic protective measure – eliminating the use of asbestos in favor of a safer substance – the risk to both the worker and all others could have been eliminated entirely.

59. Since the beginning of the twentieth century, the protective measures a company could take to protect its workforce from exposures to toxic dust have included:

- Warning workers of dangerous health effects and how to avoid harm,
- Instructing workers on hazardous substances and giving out warning literature,
- Repeating instructions frequently,
- Posting warnings and providing constant supervision of working conditions,
- Using proper ventilation and housekeeping,
- Controlling dust at the place of origin to prevent inhalation and ingestion,
- Substituting safer materials for more hazardous materials and/or processes,
- Requiring showers and separate lockers for non-work and work clothing, and frequent cleaning of clothing,
- Routine, periodic medical examination of the workers and notification of findings, and
- Use of respirators, as necessary.

These protective measures have been well known for at least 100 years and continue today to be the backbone of workplace safety when dealing with dangerous substances and processes in the workplace. Moreover, these measures are the same whether the substance at issue is lead, silica, asbestos, cotton or any pneumoconiosis or cancer producing dust.

60. If implemented, these measures would protect the worker, bystander and other workers on the jobsite, and the workers' spouses and children from exposure to toxic substances that might be brought home on workers' clothes. It was reasonably foreseeable this could occur from at least 1930, and probably before.

61. It was for this reason – to give workers the knowledge of the need to protect themselves and their families – that Merewether and Price recommended the workers be given a "sane appreciation of the risk" of working with asbestos.[86]

[85] Egilman et al., *The origin and development of the asbestos Threshold Limit Value: scientific indifference and corporate influence*, Int. J. Health Serv., 25(4) :667-96 (1995).

Twelve (AB)

62. Thirteen years before Merewether and Price wrote about the hazards of asbestos, Alice Hamilton, a pioneer of industrial hygiene and occupational medicine, made clear it is the job of the industrial physician to prevent occupational disease. She also observed and reported that factories very well may be poisoning neighborhoods: In a factory using "litharge and red lead" that was covered "with layers of these poisonous dusts," Hamilton described the plant manager, disappointed about her lack of excitement about the facilities:

> One of them finally brightened up, and said "Come and see this." I saw a wonderful air-washing machine, very expensive. He said "Every cubic foot of air is washed before it comes in." I felt like saying, *"You had better wash it before it goes out, or it will poison the neighborhood."*[87]

As noted above, from the standpoint of the occupational medicine physician, the need for controls to prevent the spread of disease outside the factory from dangerous materials used inside the factory was well recognized and not confined to a particular substance.

63. For example, in 1942, General Electric Co. and the State of Pennsylvania discussed methods to prevent spreading workplace poisons beyond the workplace including shower baths, and separate street clothing and work clothing in a factory that used asbestos felt to insulate wire.[88]

64. By 1953, the Walsh-Healy Act similarly required showers, separate lockers for street clothes and work clothes, and other protections to prevent asbestos from leaving the jobsite and poisoning family members.

65. The first published suggestion of the relationship of asbestos exposure and lung cancer was by Drs. Lynch and Smith, making observations of workers at a South Carolina

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[86] Merewether et al., *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry.* His Majesty's Stationery Office (1930).

[87] Hamilton, *The Fight Against Industrial Diseases – The Opportunities and Duties of the Industrial Physician.* Pa. Med. J. Vol. XXI, No. 6, 378–381 (1918). It was recognized that companies should provide adequate medical facilities at work, that changes of work clothing should be furnished by the employer, that showers should be provided to reduce exposures, and that ventilation to remove hazardous dusts is recommended. *Id.* The need to keep clean work areas, to use wet methods, to use ventilation, to avoid dry sweeping during cleanup and to provide respirators was well known where toxic dusts were present. Miller, *The Health Hazards of Cigar Manufacturing with Suggestions for Obviating Them.* Pa. Med. J. Vol. XXI, No. 6; 360–364 (1918).

[88] Pennsylvania Department of Labor and Industry, Safe Practices Bulletin No. 93, "Occupational Disease Prevention: Exhausting Asbestos Fiber and Dust in Wire Insulation Manufacture," April 1942.

asbestos textile plant.[89] They did not have definitive proof this occurred, but by 1942, Hueper, then director of occupational cancer studies at the National Cancer Institute, concluded the available data was sufficient for him to publish that asbestos caused lung cancer.[90] This was repeated in the scientific literature several times in the 1940s and early 1950s. In 1955, should there have been any question in anyone's mind, Doll reported on lung cancer in excess in Great Britain due to asbestos.[91] Interestingly, this data came from the Turner and Newall Company, where lung cancer cases and pleural cancers had been accumulating since the 1920s, but had not been previously reported.[92]

66. Case reports about mesothelioma began accumulating in the 1940s, and by the early 1950s there were studies relating asbestos to the development of this form of malignancy. The evidence linking cancer to asbestos was strong enough that the Journal of the American Medical Association (JAMA), among the most prestigious medical journals in America, published an editorial on the topic in 1949.[93] The JAMA article serves as a benchmark for general acceptance that asbestos was a carcinogen. By the middle 1950s, asbestos was "known" as a cause of cancer[94] in the industrial hygiene community and it was clearly recognized that the Threshold Limit Values (TLVs) and Maximum Allowable Concentrations (MACs) were not aimed at preventing cancer. By 1958, the American Industrial Hygiene Association (AIHA) published that exposure to asbestos, including during gasket, packing and brake work, was associated with asbestosis and lung cancer.[95] The work of Wagner et al. (1960) in South Africa, clearly related exposure to crocidolite asbestos to the development of this disease, and cited earlier cases.[96] Interestingly, the cases reported by Wagner included not only mineworkers, but also included family members of occupationally exposed workers and environmentally exposed patients.

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[89] Lynch et al., *Pulmonary Asbestosis III: Carcinoma of Lung in Asbestos-Silicosis.* Am. J. Cancer. 24:56 (1935).

[90] Hueper, Occupation Tumors and Allied Diseases. (C.C. Thomás, Springfield, 1942).

[91] Doll, *Mortality from Lung Cancer in Asbestos Workers.* Br. J. Ind. Med. 12 (2):81-86 (1955).

[92] Tweedale, *The Rochdale Asbestos Cancer Studies and the Politics of Epidemiology: What You See Depends on Where You Sit*, Int. J. Occup. Environ Health 13: 70–79 (2007).

[93] J.A.M.A., Editorial, *Asbestosis and Cancer of the Lung*, (August 13, 1949). This editorial discusses pleural and lung cancer and considers both human and animal data.

[94] Cook, *Symposium on Threshold Limits – Present Trends in MAC's.* Ind. Hyg. Quarterly (Sept. 1956) (recognizing the TLVs hadn't addressed the "perplexing problems" of "cancerigens" and listing asbestos among the known causes of cancer).

[95] AIHA Hygienic Guides, Asbestos (1958). The AIHA Hygienic Guides were available to anyone who wanted them for $0.25/each. It had been recognized that asbestos from brake linings, gasket and packing caused asbestosis as early as 1932. (Merewether, E.R.A. Memorandum on the Industrial Diseases of Silicosis and Asbestos, Her Majesty's Stationery Office (1932)).

[96] Wagner et al., *Diffuse Pleural Mesothelioma and Asbestos Exposure in North Western Cape Province.* Br. J. Ind. Med. 17 (4):260-271 (1960) (reporting on cases of mesothelioma due to occupational, household and environmental exposures to asbestos).

III. Domestic, Household and Non-Occupational Exposure Can And Does Cause Mesothelioma

67. It is well established that take-home asbestos on workers' clothes, shoes, or hair can cause mesothelioma in household members living with the asbestos-exposed worker.

68. OCCUPATIONAL SAFETY AND HEALTH GUIDELINE FOR ASBESTOS POTENTIAL HUMAN CARCINOGEN in 1988, provides appropriate information on how to protect the exposed person and family from asbestos contamination of the home and car:

> **SANITATION**
>
> Clothing which is contaminated with asbestos should be removed at the end of the work period and placed in nonreusable, impermeable containers for storage, transport, and disposal until it can be discarded or until provision is made for the removal of asbestos from the clothing. These containers should be marked "Asbestos-Contaminated Clothing" in easy-to-read letters. If the clothing is to be laundered or cleaned, the person performing the operation should be informed of asbestos's hazardous properties. Reusable clothing and equipment should be checked for residual contamination before reuse or storage.
>
> A change room with showers, washing facilities, and lockers that permit separation of street and work clothes should be provided.
>
> Workers should be required to shower following a workshift and prior to putting on street clothes. Clean work clothes should be provided daily.
>
> Skin that becomes contaminated with asbestos should be promptly washed with soap and water.
>
> The storage, preparation, dispensing, or consumption of food or beverages, the storage or application of cosmetics, the storage or smoking of tobacco or other smoking materials, or the storage or use of products for chewing should be prohibited in work areas.
>
> Workers who handle asbestos should wash their faces, hands, and forearms thoroughly with soap and water before eating, smoking, or using toilet facilities.

69. These types of exposures and their resultant disease manifestations are outlined very effectively in the National Institute for Occupational Safety and Health Report to Congress on Workers' Home Contamination Study that was conducted under The

Workers' Family Protection Act (29 U.S.C. § 671a).[97] In this report NIOSH concludes that:

> families of asbestos-exposed workers have been at increased risk of pleural, pericardial, or peritoneal mesothelioma, lung cancer, cancer of the gastrointestinal tract, and non-malignant pleural and parenchymal abnormalities as well as asbestosis.

This study reviewed twelve epidemiology studies and multiple case reports and concluded "[m]esothelioma has occurred following short term asbestos exposures of only a few weeks, and can result from very low levels of exposure."[98]

70. It has been repeatedly and consistently demonstrated in the medical and scientific literature that family members exposed to asbestos dust from laundering a worker's clothing have a significantly increased risk of developing mesothelioma.[99]

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[97] National Institute of Occupational Safety & Health ("NIOSH"), Report to Congress on Workers' Home Contamination Study Conducted Under The Workers' Family Protection Act (29 U.S.C. 671a) (Sept. 1995) at 6-11, 45-46, 55, 62-63, 86-87, 145-59 tbls.2-6.

[98] National Institute of Occupational Safety & Health ("NIOSH"), Report to Congress on Workers' Home Contamination Study Conducted Under The Workers' Family Protection Act (29 U.S.C. 671a) (Sept. 1995) at 7. This NIOSH report illustrates the generally accepted and scientifically appropriate consideration and use of case reports and case series, along with the other available scientific information regarding asbestos and disease causation, in forming opinions regarding causation of diseases, particularly rare diseases like mesothelioma.

[99] Wagner et al., *Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province.* Br. J. Ind. Med. 17 (4):260-271 (1960); Newhouse et al., *Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area.* Br. J. Ind. Med. 22:261-269 (1965); Lieben et al., *Mesothelioma and Asbestos Exposure.* Arch. Environ. Health. 14:559-566 (1967); Champion, *Two Cases of Malignant Mesothelioma after Exposure to Asbestos.* Am. Rev. Respir. Dis. 103 (6):821-826 (1971); Lillington et al., Letter, *Conjugal Malignant Mesothelioma.* N. Engl. J. Med. 291 (11):583-584 (1974); Greenberg et al., *Mesothelioma Register 1967-1968.* Br. J. Ind. Med. 31 (2):91-104 (1974); Anderson et al., *Household-Contact Asbestos Neoplastic Risk.* N.Y. Acad. Sci. 271:311-323 (1976); Li et al., *Familial Mesothelioma After Intense Asbestos Exposure at Home.* JAMA. 240(5):467 (1978); Vianna et al., *Non-Occupational Exposure to Asbestos and Malignant Mesothelioma in Women.* Lancet. 311 (8073):1061-1063 (1978); Epler et al., *Asbestos-Related Disease from Household Exposure.* Respiration. 39 (4):229-240 (1980); Tagnon et al., *Mesothelioma Associated with the Shipbuilding Industry in Coastal Virginia.* Cancer Res. 40 (11):3875-3879 (1980); Hammar et al., *Familial Mesothelioma: A Report of Two Families.* Hum. Pathol. 20 (2):107-112 (1989); Roggli, *Mineral Fiber Content of Lung Tissue in Patients with Environmental Exposures: Household Contacts vs. Building Occupants.* Ann. N.Y. Acad. Sci. 643:511-518 (1991); Schneider et al., *Pleural Malignant Mesothelioma and Household Exposure.* Review Environ. Health. 11:65-70 (1996); Roggli, *Malignant Mesothelioma in Women.* Anat. Pathol. Chapter 8, pp. 147-163 (1997); Hillerdal, *Mesothelioma: Cases Associated with Non-Occupational and Low Dose Exposures.* Occup. Environ. Med. 56 (8):505-513 (1999); Dodson et al., *Quantitative*

71. In addition to Wagner et al. (1960), Newhouse et al. (1965) reported mesothelioma from household and environmental exposures to asbestos, in addition to occupational exposures.[100] Environmental exposures can also apply to those living near asbestos utilizing facilities. Similar experiences have played out in Japan[101], Italy[102] and elsewhere. Lieben and Pistawka (1967), of the Pennsylvania Department of Health reported several cases from both neighborhood and household asbestos exposures that resulted in mesothelioma.[103] Anderson et al. (1979) and Anderson (1983) reported on familial exposure to asbestos and disease showing both non-malignant and malignant disease occurring in family members not otherwise exposed to asbestos.[104] Vianna and Polan is a particularly interesting epidemiological study documenting a substantially (ten times) elevated risk of mesothelioma in the wives or daughters of asbestos workers, one of whose husband worked as a brake lining worker.[105] Bourdès et al. (2000) found a relative risk of pleural mesothelioma for household exposures ranged between 4.0 and 23.7 and the summary risk estimate was 8.1.[106] Magnani et al. (2000) found domestic exposure was associated with an increased risk (Odds Ratio of 4.81 with a 95% CI of 1.8 to 13.1). [107] Many other publications recognize the real risk from exposure to asbestos carried home from the jobsite.[108] The scientific evidence consistently confirms there is

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Analysis of Asbestos Burden in Women with Mesothelioma. Am. J. Ind. Med. 43 (2):188-195 (2003).

[100] Newhouse et al., *Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area.* Br. J. Ind. Med. 22 (4):261-269 (1965).

[101] Kurumatani et al., *Mapping the Risk of Mesothelioma Due to Neighborhood Asbestos Exposure*. Am. J. Respir. Crit. Care Med. Vol. 178 (2008). Newhouse, in London, showed, among other things, that a number of individuals developed mesothelioma simply from living near an asbestos utilizing facility.

[102] Barbieri et al., *Asbestos Fibre Burden in the Lungs of Patients with Mesothelioma Who Lived Near Asbestos-Cement Factories.* Ann. Occup. Hyg. 56(6) 660 – 670 (2012).

[103] Lieben, *Mesothelioma and Asbestos Exposure.* Arch. Environ. Health. Apr. 14 (4):559, 559-563 (1967).

[104] Anderson et al., *Household Exposure to Asbestos and Risk of Subsequent Disease.* Dusts & Disease. 145-146 (R.A. Lemen and J.M. Dement eds., 1979); Anderson et al., *Asbestosis Among Household Contacts of Asbestos Factory Workers.* Ann. N.Y. Acad. Sci. 330: 387-399 (1979); Anderson, *Family Contact Exposure.* Proceedings of the World Symposium on Asbestos 349-362 (Canadian Asbestos Information Center) (1982).

[105] Vianna, *Non-Occupational Exposure to Asbestos and Malignant Mesothelioma in Women.* Lancet. 311 (8073):1061-1063 (1978).

[106] Bourdes et al., *Environmental Exposure to Asbestos and Risk of Pleural Mesothelioma: Review and Meta-Analysis*, European J. of Epi., 16:411-417 (2000).

[107] Magnani et al., *Multicentric study on malignant pleural mesothelioma and non-occupational exposure to asbestos.* Br. J. Cancer. 83: 104–111 (2000).

[108] Champion, *Two Cases of Malignant Mesothelioma after Exposure to Asbestos*, Am. Rev. Res. Dis. 103(6):821-826 (1971); Lillington, *Conjugal Malignant Mesothelioma* [letter], New Engl. J. Med., 291(11):581-585 (1974); Greenberg & Davis, *Mesothelioma Register 1967-1968*, Brit. J. Med. 31:91-104 (1974); Li, *Familial Mesothelioma After Intense Asbestos Exposure at Home*,

no safe level of exposure to asbestos when it comes to the disease mesothelioma (as evidenced by the report of Ruiz et al. (2011) discussing the wife of an auto brake mechanic with peritoneal mesothelioma).[109] A case-control study in Great Britain suggests that a majority of currently occurring female mesotheliomas are not attributable to identifiable sources of exposure and may be due to unwitting exposures that occurred during the period of heavy asbestos use.[110] OSHA recognizes that "[a]sbestos exposures as short in duration as a few days have caused mesothelioma in humans. Every occupational exposure to asbestos can cause injury of disease; every occupational exposure to asbestos contributes to the risk of getting an asbestos related disease."[111] NIOSH has recognized mesotheliomas have been caused with as little as one day's exposure.[112] In NIOSH's Division of Respiratory Disease Studies, *Occupational Respiratory Diseases*, DHHS (NIOSH) Publication No. 86-102 (September 1986), Dement et al.'s chapter on Asbestosis explained the dose-response relationship between asbestos and diseases such as mesothelioma:

> PREVENTION
>
> Available epidemiologic data support a linear, no threshold dose-response relationship between asbestos exposure and the risk of lung cancer. Additionally, no threshold has been convincingly demonstrated for nonmalignant respiratory diseases associated with asbestos exposure. *Thus, any asbestos exposure carries with it some increased risk of asbestos related diseases. Accordingly,*

JAMA 240(5):467 (1978); Epler, *Asbestos-Related Disease from Household Exposure*, Respiration, 39:229-240 (1980); Tagnon, *Mesothelioma Associated with the Shipbuilding Industry in Coastal Virginia*, Cancer Research, 40:3875-3879 (1980); Hammar, *Familial Mesothelioma: A Report of Two Families*, Human Pathology, 20:1-7-112 (1989); Schneider, *Pleural Malignant Mesothelioma and Household Exposure*, Review Environ. Health, 11:65-70 (1996); Hillerdal, *Mesothelioma: Cases Associated with Non-Occupational and Low Dose Exposures*, Occup. Environ. Med., 56-505-513 (1999); Dodson et al., *Quantitative Analysis of Asbestos Burden in Women with Mesothelioma*, Am. J. Ind. Med. 43:188-195 (2003); Rake et al., *Occupational, domestic and environmental mesothelioma risks in the British population: a case-control study*, Brit. J. Cancer, 1-9 (2009).

[109] Ruiz et al., *Mesotelioma Peritoneal Maligno – Informe de un Caso y Revision de la Lituratura.* Rev. Med. Inst. Mex. Seguro Soc. 49(1):79-84 (2011).

[110] Peto et al., *Occupational, domestic and environmental mesothelioma risks in Britain -- A case-control study.* RR696 (2009). This study indicates that living with an asbestos-exposed person prior to the age of thirty (30) also presents an increased risk of mesothelioma.

[111] Occupational Safety & Health Administration ("OSHA"), *Safety and Health Topics – Asbestos*, https://www.osha.gov/SLTC/asbestos/ (accessed September 9, 2016) (citation omitted).

[112] NIOSH, Revised Recommended Asbestos Standard, DWEW (NIOSH) Publication No. 77-169 (December 1976). Animal inhalation studies have demonstrated a single day's exposure to chrysotile asbestos can cause mesotheliomas. Wagner et al., *The Effects of the Inhalation of Asbestos in Rats*, Br. J. Cancer 29: 252 (1974).

asbestos exposure should be eliminated or reduced to the lowest level possible.

. . .

Appropriate work practices are an important component of any dust control program. These include use of wet methods or high efficiency vacuum cleaners for cleaning of asbestos contaminated areas and proper disposal of asbestos-contaminated waste. Showering and changing of work clothes at the end of the work shift are important in eliminating "take-home" exposures. Respiratory protection is appropriate for short-term jobs or operations where controls may be unfeasible; however, use of respirators is not an acceptable substitute for engineering controls.

72. NIOSH produced an excellent summary of the development on knowledge of the risk to family members from working with industrial hazards: National Institute of Occupational Safety & Health ("NIOSH"), Report to Congress on Workers' Home Contamination Study Conducted Under The Workers' Family Protection Act (29 U.S.C. 671a) (Sept. 1995). This study details some of the early publications regarding the recognition by industry of the need to prevent exposure to industrial poisons in the home. Although the earliest recognition of the need to protect workers and their families came in the context of poisons other than asbestos, the same principles applied from the early 1930s and as discussed above, were applied to asbestos by no later than 1942, when Pennsylvania published its Safe Practice Bulletin No. 93.

73. Over the years, studies have shown other forms of cancer can be caused by asbestos. While there continues to be some controversy, it is generally accepted that gastrointestinal tract cancers, laryngeal cancers, kidney cancers and ovarian cancers are all found in excess following exposure to asbestos, the risk increasing with increasing exposure.[113]

74. As more and more groups of individuals exposed to asbestos have been looked at, more and more evidence of asbestos-induced disease is found. While there clearly appears to be a threshold phenomenon with regard to the development of asbestosis, no such threshold appears to exist for asbestos-related cancers, although a dose-response relationship exists. Time since first exposure and individual susceptibility may also play a role in increasing the risk of mesothelioma.

75. While most studies of asbestos and the development of human disease have focused on individuals occupationally exposed, there is an increasing body of evidence that non-occupational exposure, usually called environmental exposure, can lead to the

[113] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012).

development of asbestos-related disease.[114] This is true for findings such as pleural plaques, where in Finland individuals living near an asbestos mine developed plaques with some regularity, but similar individuals in areas where no asbestos mines existed did not. Wagner et al.,[115] in their classic 1960 paper regarding mesothelioma, spoke to the issue of individuals with environmental exposure developing mesothelioma as fibers were moved from the site of extraction to enter the delivery system, on their way to entering general commerce.[116]

76. In the United States, a current issue of environmental exposure is the situation in Libby, Montana, where a tremolite-containing vermiculite mine has injured workers and townspeople. The product has caused additional disease after entering general commerce.[117]

77. A somewhat more specific phrase, either called household exposure or familial exposure, exists when family members develop asbestos-related disease. Anderson looked at family members of asbestos-exposed workers. Even family members moving into a contaminated household after the worker has stopped bringing in asbestos can lead to the development of the disease.[118]

78. As pointed out by the 58 Scientists Brief, a recent investigation suggested that a germline mutation might increase susceptibility to asbestos-induced mesothelioma:

> The great weight of evidence suggests that there are widely varying levels of susceptibility to asbestos, much as there is with tobacco and lung cancer. For example, even with very high exposures such as those experienced by insulation workers, less than 10% of the insulators developed mesothelioma.[119] On the other hand, just low level chrysotile exposure via home contamination, in the absence of occupational exposures, has been

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[114] Kivoluoto, *Pleural Calcification as a Roentgenologic Sign of Non-Occupational Endemic Anthophyllite Asbestos.* Acta. Radiol. (Suppl.) 194:65 (1960); Newhouse et al., *Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area.* Br. J. Ind. Med. 22 (4):261-269 (1965).

[115] Wagner, *Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province.* Br. J. Ind. Med. 17 (4):260-271 (1960).

[116] Pan et al., *Residential proximity to naturally occurring asbestos and mesothelioma in California.* Am. J. Respir. Crit. Care Med. 172:1019– 1025 (2005).

[117] Peipins et al., *Radiographic Abnormalities and Exposure to Asbestos-Contaminated Vermiculite in the Community of Libby, Montana, USA.* Environ. Health Perspect. 111 (2):1753-1759 (2003).

[118] Anderson et al., *Asbestosis Among Household Contacts of Asbestos Factory Workers.* Ann. N.Y. Acad. Sci. 330:387-399 (1979); Miller, *Mesothelioma in Household Members of Asbestos-Exposed Workers: 32 United States Cases Since 1990,* Am. J. Ind. Med. 47:458-62 (2005).

[119] Ribak et al., *Malignant Mesothelioma in a Cohort of Asbestos Insulation Workers: Clinical Presentation, Diagnosis, and Causes of Death,* 45 Brit. J. Ind. Med. 182 (1988).

> shown to induce high incidences of mesotheliomas in family groups that also have a germ-line BAP1 mutation.[120]

Testa et al. (2014) investigated two families with multiple cases of mesothelioma who shared the BAP1 mutation. Investigation did not identify occupational exposures to asbestos for any of the cases reported, but further investigation demonstrated chrysotile asbestos in five out of five homes where one family lived and chrysotile and tremolite in all of the homes where the other family was raised. This data suggests one explanation for the evidence of individual susceptibility.

79. Another recent study, Cheung et al. (2015), investigating the role of BAP1 mutation concluded "both asbestos exposure and genetic factors have played a role in the high rate of mesothelioma and potentially other pleural or lung cancers seen in this family."[121]

80. Another recent study of mice with BAP1 mutation, Napolitano et al. (2015) further supports the idea that BAP1 mutation makes a patient more susceptible to the mesotheliomagenic effects of asbestos.[122] According to Napolitano:

> We found that, compared with their wild-type littermates, $BAP1^{+/-}$ mice exposed to low-dose asbestos fibers showed significant alterations of the peritoneal inflammatory response, including significantly higher levels of pro-tumerigenic alternatively polarized M2 macrophages, and lower levels of several chemokines and cytokines. Consistent with these data, $BAP1^{+/-}$ mice had a significantly higher incidence of mesothelioma after exposure to very low doses of asbestos, doses that rarely induced mesothelioma in wild-type mice. Our findings suggest that minimal exposure to carcinogenic fibers may significantly increase the risk of malignant mesothelioma in genetically predisposed individuals carrying germline BAP1 mutations, possibly via alterations of the inflammatory response.

81. As reviewed by Røe et al. (2015) "[g]erm-line *BAP1* mutations have been described in families with extraordinarily high incidence of mesothelioma and in 25% of sporadic mesotheliomas, pointing to *BAP1* as the first gene reported to predispose to mesothelioma, but its role in mineral-fibre carcinogenesis has not been established. Furthermore, there are studies showing that *BAP1* mutations are significantly more

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[120] Testa et al., *Germline BAP1 Mutations Predispose to Malignant Mesothelioma*, 43(10) Nature Genetics (2011).

[121] Cheung et al., *An asbestos-exposed family with multiple cases of pleural malignant mesothelioma without inheritance of a predisposing BAP1 mutation* [Abstract]. Cancer Genet. 2015 Oct; 208(10):502-7. doi: 10.1016/j.cancergen.2015.07.004. Epub 2015 Jul 30: 1

[122] Napolitano, *Minimal asbestos exposure in germline BAP1 heterozygous mice is associated with deregulated inflammatory response and increased risk of mesothelioma*, Oncogene. 2015 Jun 29. Doi: 10.1038/onc.2015.243. [Epub ahead of print].

common in epithelial than sarcomatous and biphasic mesotheliomas."[123] The significance of the BAP1 mutation remains unclear, but it is clear that asbestos causes mesothelioma.

82. It has also been long known that a prudent work practice to insure worker safety and health was to restrict exposures to toxic and harmful substances to the workplace where they could be controlled. In 1913, Tolman and Kendall published their manual on methods for preventing occupational accidents and disease, emphasizing the need to segregate work place exposures from the home environment:

> The importance of wearing suitable clothing on the premises should be strongly impressed upon workers in dangerous trades. The ordinary or street-clothes should be taken off and replaced by special suits to be worn during working hours. It is not sufficient for a working-suit, jacket or apron to be put on *over* the ordinary clothing. The working-suit should be taken off before the midday meal and before leaving the factory and exchanged for street clothes. . . . By removing the working-clothes before meals and before leaving the factory the poison is not carried into lunchrooms or into the homes of workers.[124]

Tolman and Kendall also stressed the importance of employers providing their workers with rooms for changing clothing, which should include individual lockers and adequate washing facilities. Again, by no later than 1942, this fundamental necessary protective measure was directly applied to asbestos in Pennsylvania's Safe Practice Bulletin No. 93.[125]

83. At the Fifth Conference of Industrial Physicians and Surgeons in 1918, Henry Field Smyth, another pioneer in toxicology and industrial hygiene, identified asbestos as an industrial hazard to be monitored.[126] At that same conference, all the major industrial hygiene steps mentioned by Merewether and Price (1930) were recommended including teaching the workers about: (1) the hazards of the workplace; (2) the need for cleanliness and post-exposure bathing; and (3) the need for protective clothing.[127] The need to keep work areas clean, to use wet methods, to use ventilation, to avoid dry sweeping during

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[123] Røe et al., *Malignant pleural mesothelioma: History, Controversy and Future of a Manmade Epidemic,* Eur Respir Rev 24:115-131 (2015).

[124] Tolman, W.H. and L.B. Kendall. SAFETY, Methods For Preventing Occupational and Other Accidents and Disease. p. 249-249 (Harper & Brothers Publishers, New York & London, 1913).

[125] Pennsylvania, Department of Labor and Industry, Safe Practices Bulletin No. 93, "Occupational Disease Prevention: Exhausting Asbestos Fiber and Dust in Wire Insulation Manufacture," April 1942.

[126] Smyth et al., *A Preliminary Report on Dust Studies in Various Industries.* Pa. Med. J. Vol. XXI, No. 6; 364-368 (1918).

[127] *See* Schereschewsky, *Some Medical and Surgical Problems and their Solution.* Pa. Med. J. Vol. XXI, No. 6:355–359 (1918).

cleanup and to provide respirators was well known where toxic dusts were present.[128] While some of the early literature discusses the concepts of worker protection in the context of other materials, it is clear the concepts applied to asbestos or any toxic dust.

84. In 1943, in their Manual of Industrial Hygiene and Medical Service in War Industries, the United States Public Health Service emphasized the importance of providing changing rooms for "employees whose clothes are exposed to contamination with poisonous, infectious, or irritating material." The Manual further stated that for workers using toxic substances, "work clothes should be provided and laundered by the employer." The general acceptance of the need to segregate workplace exposures from the home is demonstrated by a memorandum written by Roy S. Bonsib, an industrial hygienist at the Standard Oil Company in 1948. Bonsib wrote:

> Appropriate work clothes, properly fitted and maintained, play a prominent part in an industrial worker's health and efficiency. This is especially true when persons are working with more or less toxic or carcinogenic materials or where cleanliness is a factor in the maintenance of product quality. Consequently, many of the more progressive industrial organizations, such as E.I. DuPont de Nemours & Company, the American Cyanamid Company and Borden Company, have for years supplied their employees with work clothing and have instituted a laundry service.[129]

85. In 1952, the United States Department of Labor issued safety and health standards concerning worker safety and that all contractors performing contract work under the Walsh-Healy Act. Walsh-Healy specifically regulated asbestos as harmful and required the contractor reduce exposures to asbestos below the TLV of 5 MPPCF. Among the health requirements was the provision that:

> Workers who handle or are exposed to harmful materials in such a manner that contact of work clothes with street clothes will communicate to the latter the harmful substances accumulated during working hours should be provided with facilities which will prevent this contact and also permit the free ventilation or drying of the work clothes while they are not in use. In any plant where it

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[128] Miller, *The Health Hazards of Cigar Manufacturing with Suggestions for Obviating Them.* Pa. Med. J. Vol. XXI, No. 6:360–364 (1918).

[129] Bonsib, *Memorandum, Industrial Work Clothes: Their Provision and Laundering.* Medical Department, Standard Oil Company (N.J.) (January 28, 1948). (written for members of American Petroleum Institute Medical Advisory Committee). Eleven (11) years earlier, Bonsib, in a widely published memorandum, discussed the health hazards of working in dust-producing operations involving asbestos. Bonsib, *Memorandum*, Dust Producing Operations in the Production of Petroleum Products and Associated Activities – A Medico-Safety Survey, Medical Department, Standard Oil Company (N.J.) (July, 1937).

> is necessary for both male and female employees to change clothes, separate dressing rooms should be provided.[130]

86. Segregation of work clothing contaminated with industrial dusts and chemicals from the home environment was recommended by the government and industry because it had been known since the 1930s that introducing such substances into the home put the worker's family at risk for contracting disease.[131] For example, in 1935, chloracne from exposure to polychlorinated biphenyl (PCB) was described in family contacts of a chemical worker who was employed in a PCB manufacturing facility. Similarly, in 1949, it was recognized a family member's exposure to beryllium from worker's clothing could result in non-occupational berylliosis.[132]

87. While it may seem like household exposures are low-level exposures, this is not always the case. One asbestos company executive explained the nature of household exposure as he criticized a study discussing household exposures:

> Over and above other deficiencies in this study, is the erroneous assumption that household exposures to asbestos have been minimal in dose relationship concept. The precise opposite is more likely the truth. As recognized by Selikoff and others, the impregnation of drapes, rugs, furniture etc. with asbestos fibers and the constant re-suspension of fiber in the respirable range creates an exaggerated hazard. Once asbestos is carried home by the workman, it accumulates in the home, and its presence in the home is likely to be permanent. Once it gets into the rugs, for example, it becomes re-suspended by movements such as brushing and walking and therefore, family members are getting a 24-hour a day, 7-day a week, exposure, relatively speaking, rather than a partial exposure. Of greater concern is the fact that the entire population of the family, including the very young and the very old, are exposed. Experimental and clinical data on the induction

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[130] Safety and Health Standards For Contractors performing Federal Supply Contracts under the Walsh-Healy Public Contracts Act, United States Department of Labor, at p. 25 (1952).

[131] Commonwealth of Pennsylvania: A Preliminary Report of the Dermatological and Systemic Effects of Exposure to Hexachloro-Naphthalene and Chloro-Diphenyl. Bureau of Industrial Standards, Department of Labor and Industry, Hamsburg, PA, Special Bulletin 43, March 16, 1935 (cited in Anderson, Henry A. Household Exposure to Asbestos and Risk of Subsequent Disease. Dusts and Disease, Pathotox Publishers, Inc., Eds. Lemen and Dement, 1979 at p. 145).

[132] Eisenbud, *Non-occupational Berylliosis*. J. Ind. Hyg. Toxicol. 31:282-294 (1949) (cited in Epler, G.R. *Asbestos-Related Disease from Household Exposure.* Respiration. 39:229-240, 235 (1980)).

> of cancer establish that the very young are more susceptible to carcinogens.[133]

Because of the danger from continued exposure to asbestos carried outside the workplace, it is important to determine if a worker left the workplace in a contaminated state.

88. Anderson et al. (1979) found that living with an asbestos worker led to a seven-fold increase in radiographic evidence of asbestos abnormalities.[134]

89. It has long been known that it is important to prevent toxic substances from leaving the workplace. Given the abundant evidence of the carcinogenic nature of asbestos, the lack of a safe level, and the knowledge that asbestos can contaminate the cars and homes of workers, companies involved in the manufacture, sale and/or use of asbestos or asbestos-containing products should have provided work clothing, showers and change rooms that prevented contamination of street clothing with asbestos.

Historical Recognition that there is No Safe Level of Exposure to Asbestos

90. The concept that there is no safe exposure to a carcinogen is neither a new nor novel opinion in the industrial hygiene, medical and scientific communities; rather the historical scientific literature is loaded with physicians and scientists reaching that opinion. For example, in 1948 American Petroleum Institute recognized that there was no safe level above zero for benzene.[135]

91. At a meeting in 1955, recognizing that the TLVs had not been devised to protect against cancer, Stokinger suggested building a 100 - 500 times safety factor into the TLV that was set for non-cancer outcomes.[136] The TLV for asbestos required counting all particles

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[133] Comments of Johns-Manville Corp. with respect to Notice of Proposed Rulemaking Occupational Exposure to Asbestos, Fed. Reg. (October 9, 1975) (emphasis in original). These comments are supported by the observations of the National Toxicology Program:

> In the past, families of asbestos workers potentially were exposed to high fiber levels from contaminated clothing brought home for laundering. People living in households with asbestos workers were found to have a significantly elevated lung burdens of asbestos, often in the same range as found in individuals occupationally exposed to asbestos, such as shipyard workers.

Department of Health and Human Services. National Toxicology Program. Report on Carcinogens, Thirteenth Edition. Asbestos CAS No. 1332-21-4; pp. 1-3.

[134] Anderson, Henry A., R. Lilis et al., *Asbestosis Among Household Contacts of Asbestos Factory Workers*. Ann. N.Y. Acad. Sci. Vol. 330:387-399 (1979).

[135] American Petroleum Institute Toxicological Review – Benzene (1948).

[136] For the 5 MPPCF TLV for asbestos, this would mean reducing the TLV to 10,000 PPCF – 50,000 PPCF. One company, Dupont, actually did create an in-house TLV for asbestos of

in the air if asbestos dust was being generated – *i.e.* the TLV counted all dust particles in a cloud containing asbestos. If Stokinger's safety factor proposal were applied to the 5 MPPCF TLV for asbestos and converted to the current counting method (f/cc) using the most common "conversion factor" (5 MPPCF ≈ 30 f/cc), the asbestos TLV to protect against cancer would be 0.06 f/cc using the more protective 500 times safety factor.[137] Thus, against cancer, Stokinger's more protective cancer safety factor, if applied in 1955, would have advocated exposures less than today's OSHA PEL. It is important to recognize that by 1955, it was clear that the 5 MPPCF TLV was not fully protective even for asbestosis. As one physician aptly commented, "If you poison your boss a little bit each day it's called murder; if your boss poisons you a little each day it's called the Threshold Limit Value."[138]

92. In 1955, Bonser et al. (1955) reported 2 cancers of the pleura and 4 cancers of the peritoneum (1 man/3 women) in workers at an asbestos textile plant.[139] The authors listed asbestos as "causing cancer" and identified "users" of asbestos as being at risk.[140]

93. In 1956, one asbestos company scientist published his opinion that "it is prudent to set the standard for cancerigenic substances substantially at zero . . . and no considerations can justify allowing inhalation of any concentration which is avoidable."[141]

94. Again in 1964, the widely held belief that there was no safe level of asbestos exposure was discussed by several asbestos company scientists at a major meeting called the

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500,000 PPCF. E.I. Dupont de Nemours & Co., Memorandum, Engineering Department, (May 2, 1968) (setting TLV for "total dust" at 500,000 particles per cubic foot).

[137] This calculation was performed assuming that 1 MPPCF = 6 f/cc and therefore 5 MPPCF = 30 f/cc, direct calculation based on conversion particles per cubic foot (assuming all particles were fibers) to fibers per cubic centimeter. 30 f/cc ÷ 500 = 0.06 f/cc. The direct conversion of MPPCF to fibers/cc ignores the fact that the TLV was a total dust standard and, for any product other than pure asbestos, this calculation would underestimate the safety factor proposed by Stokinger. While I – and most scientists – recognize that the conversion factor adopted by NIOSH was not in any way reliable, the unfortunate fact remains that it was used to create the first OSHA standards. I use this conversion factor to illustrate that even using the least protective interpretation of the historic exposure guidelines, the approach suggested by Stokinger would have required greater protection than went into place for more than a decade under OSHA. This was due, in large part, to political pressure exerted by the asbestos industry in the rulemaking process.

[138] Castleman, Asbestos: Medical and Legal Aspects. 5th Ed. Aspen Publishers (2005) (quoting James P. Keogh, M.D.).

[139] Bonser et al., *Occupational Cancer of Urinary Bladder in dyestuffs operatives and of lung cancer in asbestos textile workers and iron ore miners*, Am. J. Clin. Path. 25:126-34 (1955).

[140] *Id.*

[141] Smyth, Jr. *Improved Communication – Hygienic Standards for Daily Inhalation.* Am. Ind. Hyg. Quarterly. 17(2): 129-185 (1956) (Dr. Smyth was an employee of Union Carbide, which at the time was a major manufacturer of asbestos-containing phenolic compounds and which later became a major miner and distributor of asbestos).

Conference of the Biological Effects of Asbestos. U.S. Rubber's medical officer expressed the opinion clearly and concisely:

> Our own conclusion, as we began seeing what was happening in our own process, was that *the only safe amount of asbestos dust exposure was zero* and that the efforts in terms of achieving that lay basically in engineering, and, secondly, in education. But as far as a safe level of asbestos dust is concerned, our own conclusion in Hogansville, Ga., is that *there is no safe level. The safe level is nil and anything above the safe level represents certain risk.*[142]

95. A British company official offered his own thoughts at that same meeting: "*We do not believe there is any safe limit.* We have our own ideas as to how low we can get and we are always striving to get right down to zero. . . . we know there is no scientific basis for [the asbestos TLV of 5MPPCF] whatever."[143]

96. Dr. Smyth reiterated the widely held view about the danger of carcinogens in a 1962 article, stating "[w]e know so little about the causation of cancer by most substances that it may be prudent to limit the concentration to a form of zero, the smallest amount which can be analytically estimated."[144]

97. Despite the well-discussed weaknesses of the TLV, several companies continued to use the outdated, unsupported values to guide themselves and their customers. For example, despite having had personnel present at the 1964 Conference on the Biological Effects of Asbestos, Union Carbide continued to distribute its 1965 "Asbestos Toxicology Report" into the 1970s. This "Asbestos Toxicology Report" contained several misleading statements and inappropriately suggested that the TLVs mentioned were designed to protect against cancer. Particularly misleading was the following conclusion provided by Union Carbide's Industrial Medicine and Toxicology Department: "In conclusion, while asbestos dust in excess of the Threshold Limit Value is potentially harmful, as are many other dusts encountered in industry, it is readily controlled as other such dusts and it can be used safely with appropriate precautions."[145,146]

98. By the middle 1960s, anyone wanting to discover the hazards of asbestos could have done so by going to a major library. For example, one Union Carbide salesman in the United Kingdom uncovered most of the widely accepted hazards of asbestos by simply

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[142] Wells, Discussion. Ann. N.Y. Acad. Sci. 132 (1)1-766 (1965) (reporting discussion at p. 336) (emphasis added).

[143] Addingley, Discussion. Ann. N.Y. Acad. Sci. 132 (1)1-766 (1965) (reporting discussion at p. 335) (emphasis added).

[144] Smyth, *A Toxicologist's View of Threshold Limits*, American Industrial Hygiene Journal, 23: pp.37-44 (January-February, 1962).

[145] Dernehl and Lane, Asbestos Toxicology Report, (1964) (produced by Union Carbide).

[146] Industrial Medicine and Toxicology Department, Union Carbide Corporation, Asbestos Toxicology Report, (May 8, 1969) (produced by Union Carbide).

going to the library.[147] Mr. Sayers indicated that there was "a growing feeling" that the Threshold Limit Value referred to in Union Carbide's Asbestos Toxicology Report was "no longer tenable."[148] Sayers also observed that mesothelioma "can occur in people with minimal fibrosis, *i.e.* only after a brief exposure, which may be as little as three months. Some authorities even believe that a single brief exposure might be sufficient."[149]

99. Mr. Sayers' library research led him to conclude correctly that, asbestos caused asbestosis, mesothelioma and lung cancer, that the TLVs for asbestos did not prevent disease, and that the TLV for asbestos was, in fact, "an arbitrary choice, and had no experimental foundation." Sayers also recognized the "moral issue" surrounding the need to warn customers and others about the hazards:

> Moral Issues . . . on the basis of the present evidence, we are not entitled under any circumstances to state that our material [chrysotile asbestos] is not a health hazard. What is more, if it is believed that a potential customer would use our material 'dangerously', and that he is unaware of the toxicity question, then it must surely be our duty to caution him and to point out means whereby he can hold the asbestos air float to a minimum.

100. Sayers also was aware of the opinion, expressed at the 1964 Conference on the Biological Effects of Asbestos, that "[t]he M.A.C. (Maximum Allowable Concentration) of 5 million particles per cubic foot is not now acceptable. Industry should aim at 1 million particles, and accept this figure with reservations until our knowledge in this field is extended."[150] On the basis of the foregoing, Sayers observed "[i]t thus appears that the sentence in Dr. Dernehl's Asbestos Toxicology Report: 'It is now generally accepted that a man can work a 40-hour week for a lifetime without developing asbestosis, if the asbestos dust particle count is kept at or below 5 million particles per cubic foot of air' is now no longer held to be true by a number of informed people."[151] Union Carbide's medical director, Dr. Carl Dernehl, agreed, stating that Sayers' memorandum was "reasonably accurate."[152]

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[147] Sayers, Memorandum, Asbestos as a Health Hazard in the United Kingdom, (Dec. 1967) (produced by Union Carbide Corp.).

[148] Sayers, Memorandum, Asbestos as a Health Hazard in the United Kingdom, (Dec. 1967) (produced by Union Carbide Corp.).

[149] Sayers, Memorandum, Asbestos as a Health Hazard in the United Kingdom, (Dec. 1967) (produced by Union Carbide Corp.); NIOSH, Revised Recommended Asbestos Standard, DWEW (NIOSH) Publication No. 77-169 (December 1976).

[150] Sayers, Memorandum, Asbestos as a Health Hazard in the United Kingdom, (Dec. 1967) (produced by Union Carbide Corp.).

[151] Sayers, Memorandum, *Asbestos as a Health Hazard in the United Kingdom*, (1967) (produced by Union Carbide Corp.).

[152] Dernehl, Memorandum to Dr. T. J. Hall, [No Title] (June 7, 1967) (produced by Union Carbide Corp.). In this memoranda, the author points out that testing at the Mellon Institute

Historical Recognition of the Insidious Nature of the Danger of Asbestos

101. It has been documented in medical and scientific literature for many decades that medically significant exposures to asbestos thousands of times above background levels may not be visible to the naked eye. Breathing visible dust, from products containing asbestos reflects asbestos exposures not only well above background levels but also above the highest historic TLV, 5 MPPCF of asbestos-containing dust, a level that is generally not visible to the naked eye.[153] According to one asbestos company, 5 million particles per cubic foot "is generally not visible in the average work area unless a beam of light causing a Tyndall effect is present" and that "[u]sually the dust concentration must be from 8 – 10 million particles per cubic foot before its presence is visible in average lighting conditions."[154] Other experts during the pre-OSHA period reported that concentrations had to exceed 10 MPPCF before they would be become visible in a factory setting[155]:

TABLE 1-8

Sight-Perception Dust Scale

(Visible Dust Concentrations in General Air)

Lighting	Perspective	
	Short Distances (less than 10 to 15 ft)	Long Distances (50 to 200 ft)
	Concentration, Million Particles per cu ft	
Beam of sunlight, background relatively dark	less than 2 (no distance effect)	
Bright sunlight but no beam effect	10–20	2–5
Bright daylight "north" illumination (i.e., interior but no direct sun)	10–20	5–10
Low intensity daylight	20–40	10–20
Dim artificial light (night)	100–200	75–100

revealed that Union Carbide's asbestos produced "the most severe reaction" in animal tests and "that it may be possible that our Coalinga product may be more hazardous to use than long fiber asbestos." Significantly, despite wondering whether 1 MPPCF would protect against mesothelioma, Union Carbide continued – for some time after this memoranda – to recommend to its customers that keeping exposure below 5 MPPCF would prevent disease. The discussion of Union Carbide and Mr. Sayers' memorandum in this regard is included as an illustration of the ready availability of this information and the general acceptance of the conclusions of persons considering the scientific data at the time. This contrasts with the after-the-fact attempts of industry to suggest that there was a meaningful debate at the time regarding the scientific significance of the published literature.

[153] *See*, Johnson, *No Halfway Measures in Dust Control.* National Safety Review. Vol. 32, No. 3:17-18 (1935).

[154] Union Carbide Corp., "Calidria" Asbestos SG-130 and SG-210 for Tape Joint Compounds (October, 1968).

[155] Hemeon, Plant and Process Ventilation. The Industrial Press (1955).

102. As Bonsib et al. (1937) observed:

> any atmosphere in which dust is visible to the naked eye is certainly too dusty to be breathed with safety by human beings, and the wise, farsighted, human employer will immediately start to decrease the dust content in any atmosphere where dust is visible. After he has eliminated the visible dust, there may still remain enough very small invisible dust to cause harm to the health of those who breath it, but in any event if he has exerted sufficient well-directed effort to remove the visible dust it is certain that much of the smaller invisible, and probably most harmful dust has also been removed. Manifestly, if dust is kept out of the air breathed by workers, the latter cannot succumb to dust disease of a respiratory character.[156]

103. The 5 MPPCF TLV was never intended to protect workers from the cancer hazards of asbestos.[157,158] Thus, if someone worked in the presence of visible dust from an asbestos-containing product, the environment was greatly in excess of the TLV for asbestosis alone. Anyone exposed to asbestos at levels that could cause asbestosis is at grave risk of suffering asbestos-induced cancers, including mesothelioma.

104. The first national standards addressing asbestos exposure were promulgated by the Occupational Safety and Health Administration (OSHA), based on a conversion from the TLV for asbestosis. Thus, the first permissible exposure limit (PEL) was recognized as not protective against cancer. Furthermore, the PELs were *never* intended to be the maximum exposure "an employee may be exposed to without incurring the risk of adverse health effects."[159] Indeed OSHA has made clear that even at the relatively low level of today's PEL (0.1 f/cc), the risk of asbestosis probably is eliminated and the risk of cancer is reduced but "a significant risk continues to exist" for mesothelioma and lung cancer.[160]

105. The publically available data would allow non-scientists, who had reason to make simple efforts, to discover the hazards of asbestos and to warn about them. Over the years, I

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[156] Bonsib, *Memorandum*, Dust Producing Operations in the Production of Petroleum Products and Associated Activities – A Medico-Safety Survey, Medical Department, Standard Oil Company (N.J.) (July, 1937).

[157] Schall, *Present Threshold Limit Value in the U.S.A. for Asbestos Dust: A Critique*. Ann. N.Y. Acad. Sci. 132 (1):316-321 (1965).

[158] Stokinger, *International Threshold Limits Values – 1963*. Am. Indust. Hyg. Assoc. J. 25:5 469 – 474 (Sept. 1963) (stating "At present no threshold values for any carcinogen appear in the list of any country.").

[159] Fairfax - Letter from OSHA (Dir. of Compliance Programs) to Ellman (May 13, 1999). See also 59 FR No. 153 at pg. 40966-7 (August 10, 1994).

[160] Fairfax - Letter from OSHA (Dir. of Compliance Programs) to Ellman (May 13, 1999).

have learned companies in the business of making, selling and/or using asbestos products knew what the ancient Greeks knew: working with asbestos can be deadly. Indeed, as discussed above, in 1967, a salesman for Union Carbide learned basically everything a company needed to know from a trip to the library.[161]

Historical Recognition of the Need to Test Products for Safety and Warn of the Dangers of those Products Before Going to Market

106. While many of these events took place many years ago, it has never been acceptable to cause injury to others through commerce. "In 1941, T. Lyle Hazlett of the Westinghouse Corporation published an article in the *Southern Medical Journal* about industrial hygiene practices in companies large and small. The company doctor needed to learn about the potential hazards of the work in that industry, even determining "any health hazards of new processes and materials before they are put into production." He laid out the variety of ways that the workforce could be educated about these dangers "by means of pamphlets, posters, talks by medical personnel and by individual contacts" and by the plant magazine."[162] According to Rosner et al. (2016):

> As the medical director of Union Carbide, Thomas W. Nale, later reflected on that time, "Manufacturers could have waited for the legislative branch of government to write the labels for them to protect the public," but there had been "a great increase in the number of new compounds that had been synthesized and put into production [as the] over-all chemical output had skyrocketed." This meant that "a great many workers in industry were unfamiliar with chemicals which they now had to handle."[163]

107. Indeed, industry, including the asbestos industry, understood the need to test products for safety *before* putting them on the market. For example, in 1942, the Industrial Hygiene Foundation published the presentation of Francis Holden, made at the Seventh Annual Meeting of the Industrial Hygiene Foundation of America, Inc., wherein the author discussed the need for responsible companies to test products for safety: "Every new chemical or product should be investigated as to its toxicity before it is prepared in large amounts and released to the public."[164]

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[161] Sayers, Asbestos as a Health Hazard in the U.K. Memorandum, Union Carbide Corp. (1967).
[162] Rosner and Markowitz. *"Educate the Individual . . . to a Sane Appreciation of the Risk" A History of Industry's Responsibility to Warn of Job Dangers Before the Occupational Safety and Health Administration.* AJPH (106)(1) (2016).
[163] Rosner and Markowitz. *"Educate the Individual . . . to a Sane Appreciation of the Risk" A History of Industry's Responsibility to Warn of Job Dangers Before the Occupational Safety and Health Administration.* AJPH (106)(1) (2016).
[164] Holden, What the Foundation Plant Surveys Are Disclosing, Meeting Report, Seventh Annual Meeting of the Members, Pittsburgh, PA, p. 62 (November 10–11, 1942).

108. According to Rosner et al. (2016) "at an IHF annual meeting the representative from DuPont agreed with Theodore Hatch and noted the importance of animal experimentation in establishing the degree of danger for chemicals and products."[165]

109. Companies involved in the asbestos trade and/or use of asbestos had numerous avenues for testing of products. Henry F. Smyth, Jr. examined the need to research hazards of new chemicals to "prevent injury to the health of workmen" . . . and to what extent the health of the public is being protected. Dr. Smyth discussed his work at one such facility, known as the Mellon Institute of Industrial Research:

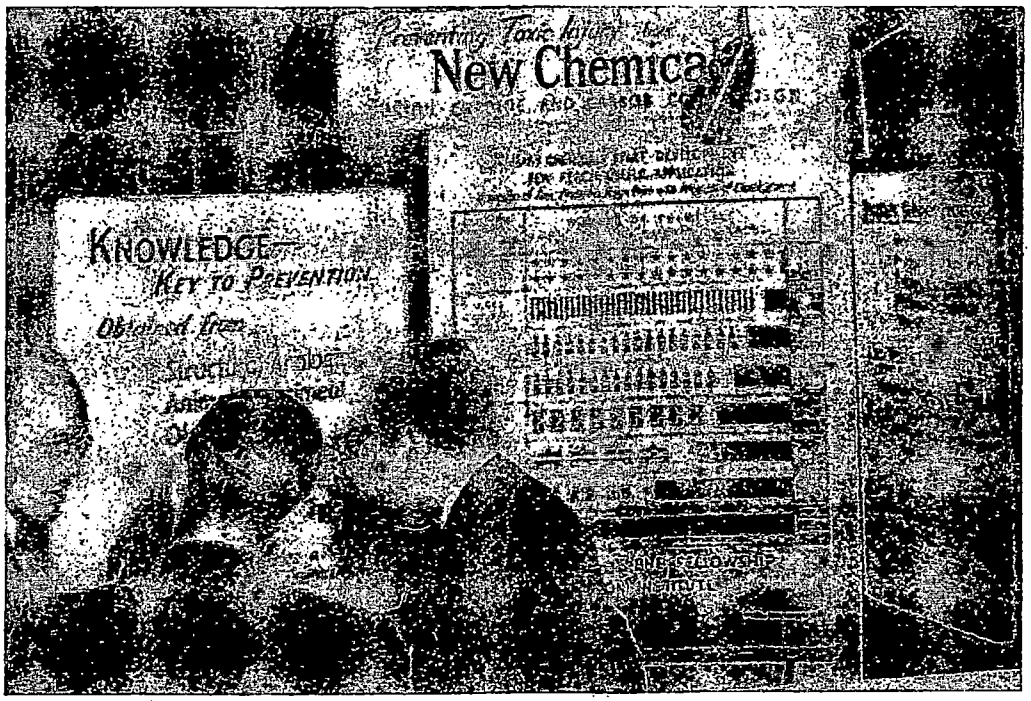
> The prevention of occupational disease requires that knowledge of the potential hazards of the materials handled by workmen shall be readily available to industrial physicians and industrial hygiene engineers. . . .
>
> It is clearly the duty of every manufacturer to delay production of a chemical until the health hazards are well enough defined so that protection of his workmen is possible. It is also his duty not to sell a chemical for an application in which it would endanger the health of the public, and to inform customers, by proper labeling and otherwise, of the hazards of the compounds they buy. . . .
>
> All producers of chemicals are probably aware of the problem which the flood of new materials presents to the industrial physicians and the hygienist. The matter is the responsibility of industry and in only rare instances is it proper to depend upon federal or state agencies to alleviate the situation. Several solutions have been evolved by single manufacturers and it is of interest to examine one of them in some detail. Eight years ago one firm established, at its own expense, an industrial fellowship under my direction at the Mellon Institute of Industrial Research of the University of Pittsburgh. The organization has grown steadily and we now have a staff of 18 technically trained persons, and facilities to house about 3,500 animals, with further expansion visible in the near future.
>
> By means of close contact with the research, production, sales, and medical departments of this manufacturer our group is made aware of all new chemicals which he develops. . . .
>
> Upon all chemicals suspected of being potentially injurious to workmen or about which any doubt is entertained, we at once

[165] Rosner and Markowitz. *"Educate the Individual . . . to a Sane Appreciation of the Risk" A History of Industry's Responsibility to Warn of Job Dangers Before the Occupational Safety and Health Administration.* AJPH (106)(1) (2016).

> perform pharmacological experiments designed to elucidate the situation. By means of tests upon small animals we investigate the hazard of swallowing, of skin penetration, of inhalation, of skin contact and of eye contact. . . .
>
> We refer to this procedure as a range finding test. It is performed in a short time and at a cost of only a few hundred dollars, and the results can be made known to the producer before the stage of pilot plant operation is reached. . . .
>
> After a time it may become apparent that the new material will be made and sold in larger quantities. Not until then is it appropriate to perform more detailed and expensive studies . . . which will reveal more precisely the quantitative hazards which must be guarded against in applications of the chemical, and the nature of the injury which overexposure may produce. When this information is published in the medical literature, our function is fulfilled in respect to the particular material, and the physician and hygienist are thus informed so that they can intelligently safeguard health.[166]

110. The theme Smyth advanced in his 1946 article was continued by Union Carbide in a poster presentation at the 1949 Annual Meeting of the Industrial Hygiene Foundation. Union Carbide's poster, entitled "Preventing Toxic Injury from New Chemicals", had a heading recognizing that "knowledge" is "the key to prevention" and discussed the benefits of studying chemicals. The poster pointed out that toxicity studies protect "research chemists, production men, transport employees, the ultimate consumer and the public." The poster also details how information will reach those who may be exposed to the toxic materials: "Knowledge is disseminated by reports to medical personnel, research chemists, manufacturing departments, shipping departments, sales department, sales literature, answers to inquiries, precautionary labels, manuals for production men, and scientific publications." The Union Carbide poster, pictured below, demonstrates that responsible industry was aware of the need to investigate the hazards of products before exposing the unsuspecting public to those materials.

[166] Smyth, *Solving the Problems of the Toxicity of New Chemicals in Industry*. W.V. Med. J. 42:177–178 (1946).



111. Responsible industry, beginning in the early twentieth century, began to undo the wrongs to workers and to stem the tide of occupational diseases. "It is management's responsibility to make sure that conditions are as safe as it is practicable to make them, and to insure that persons working with new compounds, processes and applications know the hazards involved and the precautions to be taken."[167] As one asbestos industry executive wrote in 1955:

> [t]o be sure, industry's awakening was a slow one. Fifty years ago a few leaders in industry attempted to improve the health and working conditions for employees. But it was just an attempt. The few efforts that were made were crude and isolated. And precious little financial support was offered. It is not surprising that progress was slow. Then the public's voice was heard. The public's growing concern for the health of the employee was forcibly brought to the attention of industry. There were just too many occupational diseases, too many tragedies in the mines, mills and factories. And to the great credit of industry, the problems were recognized.

[167] Hine et al., *Safe Handling Procedures for Compounds Developed by the Petro-Chemical Industry*. Ind. Hyg. Quarterly. 15 (2):141-144 (1954).

> Industry no longer considered an employee mere chattel or commodity to be put on the block to be auctioned off to highest bidder. His dignity as a human being was being acknowledged.
>
> Public-spirited men of wealth endowed research institutions to probe into occupational hazards and diseases and to develop methods and procedures to improve the health of the individual, of the employee in industry, and conditions in the community.
>
> Progressive-minded companies established medical clinics. They pushed ahead with health and safety programs. The programs were, and still are, based on taking care of the physical and mental well being of employees, helping and protecting the consumer and promoting the common good.[168]

112. By 1955, the "typical" health and safety program included medical surveillance of workers, control of occupational hazards, on the job medical care, safety precautions and plant "medical, industrial hygiene and safety personnel coordinated by a headquarters health department."[169]

113. By the first half of the twentieth century, industry was "also aware of [its] responsibility to the consuming public. Today, the products of industry are designed to promote health and comfort of the public. . . . Today our industrial research organizations probe every health hazard. . . . [E]very effort is made nowadays to protect the consumer with safer products and better methods of handling them. And every safeguard is insisted upon before the product is marketed to the consumer."[170] Industry recognized that the obligation was to the employee, the users of products and also to protect the families of employees: "...the employee's health is better, his morale is higher. He and his family live longer and more happily."[171]

114. Significantly, industry was well aware by the 1940s of the need to warn anyone and everyone who might be exposed to a risk of harm. The Manufacturing Chemists Association (MCA) issued guidelines for how to appropriately warn users and purchasers of hazardous products.[172] In its "Statement of Legal Principles" in the late 1930s the MCA stated, "The manufacturer or one who holds himself out to be the manufacturer

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[168] Fisher, A. R. The Economics of Industrial Health, Twentieth Annual Meeting, Industrial Hygiene Foundation, Transactions Bulletin No. 29, p. 15 (November 16–17, 1955).
[169] Fisher, A. R. The Economics of Industrial Health, Twentieth Annual Meeting, Industrial Hygiene Foundation, Transactions Bulletin No. 29, p. 16 (November 16–17, 1955).
[170] Fisher, A. R. The Economics of Industrial Health, Twentieth Annual Meeting, Industrial Hygiene Foundation, Transactions Bulletin No. 29, p. 17 (November 16–17, 1955).
[171] Fisher, A. R. The Economics of Industrial Health, Twentieth Annual Meeting, Industrial Hygiene Foundation, Transactions Bulletin No. 29, p. 18 (November 16–17, 1955).
[172] Manufacturing Chemists Association, A Guide for the Preparation of Warning Labels for Hazardous Chemicals, Manual L-1 (1942).

must know the qualities of his product," and further, the "manufacturer cannot escape liability on the ground that he did not know it to be dangerous." Finally, "a manufacturer who puts out a dangerous article or substance without accompanying it with a warning as to its dangerous properties is ordinarily liable for any damage which results from such failure to warn."[173] The MCA, through its members, recognized that the best way to warn end users of chemicals was to label the hazardous materials. The MCA recognized the "need for furnishing the appropriate information in those cases where [the product at issue presents] hazards requiring special precautions. Precautionary information should, so far as practicable, reach every person using, handling, or storing hazardous substances."[174] As the National Paint, Varnish and Lacquer Association observed in 1939, "[t]he manufacturer or one who holds himself out to be a manufacturer must know the qualities of his product and he cannot escape liability on the ground that he did not know it was dangerous."[175] The MCA warning guidelines were well known to industry in the 1940s, as illustrated by this entry in the IHF's annual report:

> It is of primary importance that there be uniformity in labeling hazardous chemicals so that the exact type and degree of danger will be presented. The Manufacturing Chemists' Association Manuals L-I and L-2 provide guidance. For mixtures, only the dangerous constituents need be mentioned. Under no circumstances should chemicals be over-labeled, i.e., the degree of hazard should not be exaggerated.[176]

115. Indeed, responsible "industry inspired, to a very great extent, the movement to label properly certain types of products that might harm the consumer if he were not forewarned."[177]

116. According to Rosner et al. (2016), industry recognized the moral requirement to test products before exposing the public to dangers:

> In 1956, the Chairman of the Manufacturing Chemists Association's Food Committee reiterated that the chemical industry adhered to the principle that companies test their product and inform the government of any potential dangers. The medical

[173] Rosner and Markowitz. *"Educate the Individual . . . to a Sane Appreciation of the Risk" A History of Industry's Responsibility to Warn of Job Dangers Before the Occupational Safety and Health Administration.* AJPH (106)(1) (2016).
[174] Manufacturing Chemists Association, A Guide for the Preparation of Warning Labels for Hazardous Chemicals, Manual L-1 (6th Ed. 1961) at page 7.
[175] National Paint, Varnish and Lacquer Association, Inc., Memorandum Regarding Manufacturing Chemists Association Legal Principles (1939).
[176] Smyth et al., Summary on Conference of Chemistry and Toxicology, Eleventh Annual Meeting of Foundation Members, Transactions Bulletin No. 8 (1946).
[177] Fisher, A. R., The Economics of Industrial Health, Twentieth Annual Meeting, Industrial Hygiene Foundation, Transactions Bulletin No. 29, p. 17 (November 16–17, 1955).

> director of Hercules Powder Company raised the question of "whether industries should tell their employees that the material they are handling is toxic or carcinogenic." L.C. McGee explained that this was not in question and that ethically workers "are entitled to it. . . . I would say that we must give the employee that knowledge so that he can understand and use it in coming to a conclusion as to why he must do certain things to protect himself."[178]

117. By 1957, the MCA's guidelines regarding warnings were so widely known that anyone providing materials to the United States Navy was required, by military specifications, to include warnings, based on Manual L-1.[179]

118. Even before the Navy expressly adopted the MCA's guidelines on warnings, adequate warnings were expected. The Navy regulations[180] included the following:

> **7.8 REGULATION AND STATUTE MARKING.**
> Special handling instructions and warnings shall be shown as required by the Interstate Commerce Commission regulations, U.S. Coast Guard regulations, Civil Aeronautics Board publications, and by statute.

119. One of the motivating factors for warnings was lawsuits: "Indeed the Manufacturing Chemists Association's Medical Advisory Committee, at its meeting in September 1958, "emphasized that manufacturers have a responsibility to give full information on the toxicological hazards of their products."[181]

120. In 1972, using essentially the identical guidelines published first in the 1940s, the MCA issued a proposed asbestos warning as follows:

> WARNING ! HARMFUL IF INHALED MAY CAUSE DELAYED LUNG INJURY (ASBESTOSIS, LUNG CANCER)
>
> Do not breathe dust.

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[178] Rosner and Markowitz. *"Educate the Individual . . . to a Sane Appreciation of the Risk" A History of Industry's Responsibility to Warn of Job Dangers Before the Occupational Safety and Health Administration.* Public Health Then and Now (106)(1) (2016).
[179] MIL-STD-129B (1957) incorporated the MCA Manual L-1, *A Guide for Preparation of Warning Labels for Hazardous Chemicals.*
[180] MIL-STD-129A (1954).
[181] Rosner and Markowitz. *"Educate the Individual . . . to a Sane Appreciation of the Risk" A History of Industry's Responsibility to Warn of Job Dangers Before the Occupational Safety and Health Administration.* Public Health Then and Now (106)(1) (2016).

Use only with adequate exhaust ventilation or approved respiratory protective devices.

Remove dust and fibers from clothing only by vacuum cleaning. Clean work areas only with vacuum cleaners or wet cleaning methods."[182]

121. In 1968, in England, Ford Motor Company researchers found airborne dust measurements during brake drum blow out measuring "in excess of the alternative TLV of 25 particles/cc" recommended a "general instruction that inhalation of dust during brake cleaning should be minimized, and if practical, alternative methods, which would produce less dust, can be devised, their use should be recommended."[183] The alternative TLV of 25 particles/cc was "regarded as equivalent to 2 fibers/cc by membrane filter count."[184] Using the membrane filter method, the Ford industrial hygienists calculated high time weighted averages (TWAs) ranging from 0.68 f/cc to 2.55 f/cc.[185]

122. Despite the fact that guidelines were available, many companies included cautions that did not adequately inform the consumer. For example, Union Carbide, an asbestos mining and milling company, claims to have begun placing the following warning on its bags of asbestos at least by 1969: "WARNING – Breathing Asbestos Dust May Be Harmful. Do not breathe dust."[186] This warning was described by a Union Carbide employee as "the most innocuous warning we [Union Carbide] could devise." This innocuous "warning" led the person in charge of Union Carbide's asbestos business to conclude that Union Carbide asbestos was as dangerous as being behind a plow in a very dry weather.[187] This is proof that such warnings did not adequately inform the consumer of the risks of asbestos.

123. Concerns about providing adequate warnings, such as including the word CANCER as proposed by NIOSH and the MCA, related largely to fear of lost sales of asbestos to less dangerous, substitute materials. For example, one scientist at Union Carbide observed

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[182] Best, G. - Manufacturing Chemists Association Letter to Director Scannell. OSHA (March 15, 1972).

[183] Hickish, *Ford Motor Co Report 41/68 – Exposure to Asbestos during the Servicing of Brakes of Passenger Cars*, (July 1968).

[184] Hickish, *Ford Motor Co Report 41/68 – Exposure to Asbestos during the Servicing of Brakes of Passenger Cars*, (July 1968).

[185] Hickish, *Ford Motor Co Report 41/68 – Exposure to Asbestos during the Servicing of Brakes of Passenger Cars*, (July 1968). The TWAs were as follows: (1) 1.25 f/cc for static sample by the car; (2) 2.55 f/cc in dust cloud and then static; and (3) 0.68 f/cc for a personal sample. The personal sampling (yielding TWA of 0.68 f/cc) was taken "during an entire work shift," during which brake blow out was carried out on eleven (11) vehicles.

[186] Gould, Memorandum re King City – Asbestos Multiwall Bag Specifications (July 11, 1969)(produced by Union Carbide).

[187] Myers, John Dep. *Lester Rice v. Union Carbide Corp.* USDC, District of SC, Charleston Division, Case No.: CA-81-977-9 (9.9.82) at p. 62: 20 – 24.

that "cancer is a very emotional word . . . We cannot predict with certainty what effect the use of the proposed label will have on our business, but the general feeling here is that it is likely to vary somewhere between serious and fatal."[188]

124. Another asbestos product company, CertainTeed objected to NIOSH's proposed CANCER label, testifying that "[w]e feel strongly that any such label on our finished product, particularly pipe used for water transmission, would immediately make it unsaleable. Municipalities subject to political pressures just wouldn't take a chance on a product so labeled. There is no evidence that ingestion of asbestos fiber is in any way harmful. However, all the public and the public officials will see when they see the label is the word "cancer" and the "no sale" sign will go up."[189]

125. All of the information above, with the exception of a few internal documents, was readily available to any company that cared to investigate the hazards of asbestos. As part of membership in the Industrial Hygiene Foundation (IHF), the National Safety Council (NSC), the American Ceramic Society (ACS), the American Society of Mechanical Engineers (ASME), the Illinois Manufacturers Association (IMA), and the Asbestos Information Association/North America (AIA/NA) members received regular copies of those organizations' periodicals. Because these organizations, and many other industry-focused groups, published on medical, industrial hygiene and safety issues, members of these organizations actually received information about the hazards of asbestos.

126. The IHF, for example, regularly distributed abstracts – summaries of national and international medical, industrial hygiene and safety literature – beginning in the 1930s.[190] The IHF Digests summarized more than one hundred articles from all over the world detailing the hazards of asbestos.

127. The IMA, a Chicago-based trade organization, was well aware of the hazards of asbestos in the 1930s and worked to shape the law of Illinois to reduce legal liability of its members resulting from occupational exposure to asbestos and silica.[191] Because asbestos disease and silicosis were so important to industrial concerns in the United

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[188] Rhodes – Letter to Thurber re "OSHA Regulations – Asbestos Use Warnings, May 30, 1975.
[189] Views And Arguments On A Standard For Exposure To Asbestos Dust To Be Promulgated Under The Williams-Steiger Occupational Safety And Health Act Of 1970 To Be Presented To Hearing Examiner Arthur Goldberg On March 14, 1972 By Bruce Phillips On Behalf Of Certain-Teed Products Corporation, Valley Forge, Pennsylvania.
[190] Air Hygiene Foundation of America, Inc., Officers, Committees, Members and Purposes, Information Circular No. 8 (1938) (stating that the organization "[p]rovides monthly abstracts summarizing current literature on occupational health subjects. This is an important labor-saver for company officials. Further, it insures executives against missing vital new developments."
[191] Industrial Review, Occupational Diseases, Heath, Comfort and Safety and Changes in the Blower Act, 9:105 (February, 1936).

States, organizations like the IMA routinely discussed these matters with their members.[192]

128. ASME's monthly publication *Mechanical Engineering* published numerous articles about asbestos and health before 1940.[193] ASME is one of the largest, most popular engineering societies.

129. Given the abundant information available to industry since the early twentieth century, that asbestos could cause disabling and fatal diseases, and including cancer since 1942, it is my opinion companies involved in commerce should have, at a minimum, been warning that asbestos could cause cancer and other asbestos-related disease.

[192] *E.g.*, Industrial Review, Book III on Silicosis, 10:118 (November, 1938) (discussing the IMA's receipt of The Pneumonokonioses (Silicosis) – Literature and Laws, Volume III and stating "[t]he work is invaluable to any industrial concern or professional authority dealing with silicosis.").

[193] *E.g.*, Willson, *Dust in Industry – Shop Methods and Equipment Effective in Controlling Dust Hazards.* Mech. Eng. 55:2 (1933); Dallavalle, J.M, *The Control of Industrial Dust – The Problem of Local Exhaust and General Ventilation.* Mech. Eng. 55:10 (October, 1933); Sayer, *Occupational Diseases – Additional Responsibility Legislation Places on Industry.* Mech. Eng. 60:2 (1938).

IV. All Types of Asbestos Can Cause Mesothelioma of the Peritoneum in Men and Women.

130. The consensus in the non-aligned medical and scientific community is that "all types of asbestos fibres cause extrapleural, as well as pleural, MM."[194] According to the Helsinki Criteria, "all types of malignant mesothelioma can be induced by asbestos, with the amphiboles showing greater carcinogenic potency than chrysotile."[195] Peto, et al. (2016) observed that the risk of mesothelioma from chrysotile asbestos is not "negligible."[196] In a well-documented review, Kanarek (2015) concluded "that all types of asbestos, including amphiboles and chrysotile, are causative for peritoneal mesothelioma. It is clear that there is a causal relationship between all types of asbestos at all dose levels for peritoneal mesothelioma and no threshold of exposure to asbestos appears safe."[197]

131. My opinion that all types of asbestos can peritoneal mesothelioma is based on epidemiological studies, human fiber burden testing showing that all types of asbestos reach the extra-pleural tissues (peritoneum and tunica vaginalis) animal studies showing that all fiber types can cause mesothelioma in the peritoneum of experimental animals and in vitro studies showing that all types of asbestos cause the cellular changes that lead to mesotheliomas.

132. Similarly, Landrigan et al. (1999) concluded "[c]linical and epidemiologic studies have established incontrovertibly that chrysotile causes cancer of the lung, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx and certain gastrointestinal cancers."[198]

133. According to the recent Italian Consensus panel report, "[a]fter asbestos exposure, risk of peritoneal MM shows a continuous increase."[199]

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[194] Magnani et al., *III Italian Consensus Conference on Malignant Mesothelioma of the Pleura. Epidemiology, Public Health and Occupational Medicine Related Issues,* Med Lav (106) 5:325-332 (2015).

[195] Wolff et al., *Consensus Report: Asbestos, Asbestosis, and cancer, the Helsinki Criteria for Diagnosis and Attribution 2014: Recommendations*, 41(1) Scand. J. Work Environ. Health 5 (2015); Consensus Report: *Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution*, Scand. J. Work Environ. Health, 23:311-6 (1997).

[196] Peto, et al., *Authors' reply to letters from Egilman et al. and Oliver, et al.*, Occup. Environ. Med. 73:10 710 – 711 (2016) (stating "There are sufficient grounds for worldwide replacement of chrysotile with safer substitutes. The mesothelioma risk, although less, reinforces the case.").

[197] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).

[198] Landrigan et al., *The Hazards of Chrysotile Asbestos: A Critical Review*, Indus. Health 37: 271 – 280 (1999).

[199] Magnani et al., *III Italian Consensus Conference on Malignant Mesothelioma of the Pleura. Epidemiology, Public Health and Occupational Medicine Related Issues,* Med Lav (106) 5:325-332 (2015).

134. O'Donnell et al. (1966) reported three mesotheliomas of the peritoneum and one mesothelioma of the peritoneum and pleura in workers who worked in a textile plant that "used the Chrysotile type of asbestos fiber almost exclusively."[200]

135. Godwin et al. (1968) reported finding mesothelioma of the peritoneum in a worker who worked for three years (as a young adult) in a factory making chrysotile-containing brake linings.[201] The worker died at age 43 from mesothelioma of the peritoneum.

136. Hillerdal (1999) concluded that, based on animal experimental studies and the epidemiology studies, all types of asbestos cause of peritoneal mesothelioma that there is no threshold for asbestos exposure for both pleural and peritoneal mesothelioma.[202] As IARC explained, Welch et al. (2005) "found a strong association (OR, 5.0; 95%CI:1.2–21.5) between asbestos exposure and peritoneal cancer in a population-based case–control study. This study included a large percentage of men with what were judged to be low exposures to asbestos."[203]

137. One problem with early data on the incidence of mesothelioma of the peritoneum is the difficulty of diagnosis and distinction from other peritoneal malignancies. As discussed by Kanarek:

> Selikoff and Seidman found there was great discordance between the underlying cause of death on the death certificates and more detailed clinical and histopathological evidence (called "Best Evidence"), especially for peritoneal mesothelioma. In fact, 60.5% of the 458 cases of pleural and peritoneal mesothelioma combined would have been missed if just the death certificate was available. In the 2271 deaths recorded among the 17,800 asbestos insulation workers observed from Jan 1 1967-Dec 31 1976, only 24 were listed as peritoneal mesothelioma on the Death Certificate as compared to 112 which were found using "Best Evidence". Kindler in a review of peritoneal mesothelioma emphasized the difficulty of diagnosis, that cytology is rarely helpful, and thus the experience of a pathologist with the help of several immunohistochemical tests is essential.[204]

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[200] O'Donnell et al., *Asbestos, An Extrinsic Factor in the Pathogenesis of Bronchogenic Carcinoma and Mesothelioma*, Cancer 19: 1143 – 1148 (1966).
[201] Godwin et al., *Asbestos and Mesothelioma* (Letter), JAMA 204:11 151 (1968).
[202] Hillerdal, *Mesothelioma: cases associated with non-occupational and low dose exposures*, Occup. Environ. Med. 56: 505-513 (1999).
[203] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012) (discussing Welch et al., *Asbestos and peritoneal mesothelioma among college-educated men*, Int. J. Occup. Environ. Health 11: 254–258 (2005)).
[204] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).

138. Henley et al. studies mesothelioma incidence from the U.S. SEER Program and the National Program for Cancer Registries for the years 2003-2008. Of the total of 19,011 mesotheliomas, 15,615 were pleural (82.1%) and 1754 (09.2%) peritoneal. The authors conducted a correlational analysis by site by location and found a Pearson correlation of 0.70, $p<0.0001$ for males and 0.78 $p<0.0001$ for females which supported the hypothesis that pleural and peritoneal mesothelioma share a common cause, exposure to asbestos. This correlation is similar to those reported in Italy and other places internationally.[205]

139. Dement et al. noted that there were several death certificates that mentioned "cancer of the abdomen," but no autopsies or other data were available to establish whether these cases were mesotheliomas. A follow-up of this South Carolina chrysotile asbestos textile worker cohort found three cases of mesothelioma including one female case of mesothelioma of the peritoneum.[206]

140. In Spirtas et al. (1994), for men with peritoneal cancer, the attributable risk asbestos exposure was 58% and 23% for women with both mesothelioma of the pleura and peritoneum combined. The authors identified possible explanations for the discrepancy between men and women, was greater misclassifications of exposure in women, a lower background incidence rate, or lower asbestos exposures.[207]

141. Burdorf et al. explored peritoneal mesothelioma from the Swedish and Netherlands Cancer Registers (1989-2003) aiming to investigate the role of asbestos. One clear finding was a downward shift in rates, especially in Sweden, around the 1999-2000 period, probably due to a change in the International Classification of Diseases (ICD) code to version 10. It is presumed that previous to the year 2000 many peritoneal tumors in females were misclassified as ovarian and vice versa.[208]

142. Mesothelioma of the peritoneum is an exceptionally rare cancer, making up less than 20% of the reported cases of mesothelioma. This fact, along with the poor historical recognition of mesothelioma of the peritoneum and potential for confusing it with other cancers such as ovarian cancer, makes much of the epidemiology literature unhelpful in evaluating asbestos as a cause of mesothelioma. For example, as Boffetta (2006) observed, of 34 studies reviewed, only two had sufficient mortality to provide "reasonable power to detect a risk."[209]

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[205] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).
[206] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).
[207] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).
[208] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).
[209] Boffetta, *Epidemiology of peritoneal mesothelioma: a review.* Annals of Oncology, doi:10.1093/annonc/mdl345, 1-6 (2006).

143. Some industry-aligned authors have hypothesized – erroneously – that chrysotile asbestos does not cause mesothelioma of the peritoneum. For example, Price and Ware (2004) reached this false conclusion because they assumed women did not have potential occupational exposures to asbestos and even excluded all female peritoneal mesotheliomas from their analysis.[210] As Kanarek (2016) observed, Price & Ware's "projection of a possible decline in female mesothelioma rates in the future due to female asbestos exposures falling below a 'threshold' of exposure is not sufficiently justified in the paper."[211]

144. Kradin recently reported on a series of 61 cases of mesothelioma of the peritoneum.[212] Exposure to chrysotile alone was found in 25% of his cases (15% of the cases had occupational exposure and 10% had paraoccupational exposures).

145. The available scientific literature is exceptionally limited when it comes to evaluating the risk of mesothelioma in women. In addition to the general lack of statistical power in the existing epidemiologic studies and the problems caused by historical misdiagnosis of mesothelioma of the peritoneum as ovarian cancer, most studies likely suffer from exposure misclassification (erroneously identifying women as unexposed to asbestos without investigating the potential for take-home exposure from a family member or cohabitant) which would mask any real effects (bias toward the null).

146. A number of studies documented transport of asbestos fibers from the lungs to the human peritoneum. For example, Dodson et al. (2000) found asbestos in extrapulmonary sites of 17 of 20 cases and with chrysotile in the peritoneum of 25% of the cases[213] and an analysis of twenty mesothelioma patients with occupational exposure to asbestos. Dodson et al. (2000) commented that "[l]ong fibers of chrysotile reached the omentum in several cases, which indicates that chrysotile is also translocated and could be potentially important in the pathogenesis of peritoneal mesothelioma."

147. Dodson et al. (2001) reported on asbestos bodies found in mesentery and omentum among 20 individuals in whom mesothelioma was diagnosed.[214] Heller et al. (1999) found asbestos fiber burdens ranging from 56,738 to 1,963,250 fibers per gram wet

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[210] Price et al., *Mesothelioma trends in the United States: an update based on Surveillance, Epidemiology, and End Results Program data for 1973 through 2003*, Am. J. Epidemiol. 159: 107-112 (2004).

[211] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).

[212] Kradin, *Peritoneal Mesothelioma*, Presented at Current Concepts and Controversies in Asbestos-Related Disease: 2016, Massachusetts General Hospital (November 12, 2016).

[213] Dodson et al., *Asbestos in Extrapulmonary Sites – Omentum and Mesentery*, Chest 117: 486 – 493 (2000).

[214] Dodson et al., *Asbestos content of omentum and mesentery in nonoccupationally exposed individuals*, Toxicol. Indust. Health 17: 138-143 (2001).

weight tissue in the tumor tissue among six of seven women with peritoneal mesothelioma.[215]

148. Aust, et al. (2011) reviewed some of the evidence that asbestos is translocated from the lung to the rest of the body through various mechanisms, including the lymphatic system and blood.[216] Reviewing work by Oberdorster (1988)[217], Aust, et al. (2011) explained

> Data showed that there was a fast translocation of fibers from the airspaces of the lung to the lymph nodes and even into postnodal lymph. The findings indicated that the "structures of the peripheral lung and lymph node itself act like size selective filters, permitting only the fine fiber fraction to penetrate." This conclusion is consistent with the findings of the actual fiber burden in the lung and lymph nodes from exposed humans.[218]
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> . . .
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> Monchaux and associates (1982) evaluated the translocation of REMP via the respiratory system following intrapleural injection of either UICC chrysotile A, UICC crocidolite, or JM 104 glass fibers into the pleural cavity of rats. "Ninety days after their intrapleural injection, fibres were found in all organs analyzed: mediastinal lymph nodes, lung parenchyma, spleen, liver, kidneys and brain. The resultant fibre concentrations were in the same range for all organs except in thoracic lymph nodes where they were considerably higher (10–100 times)."[219]

149. Suzuki et al. (1991) and Kohyama et al. (1991) looked for asbestos fibers and asbestos bodies (iron coated asbestos fibers) in the lung (examine both parenchyma and lung tumor), pleural plaques, and pleural and peritoneal mesothelioma tissues from thirteen North American insulation workers; fibers were found in extrapulmonary sites.[220]

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[215] Heller et al., *Presence of asbestos in peritoneal malignant mesotheliomas in women*, Int. J. Gynecol. Cancer 9: 452-455 (1999).

[216] Aust, et al., *Morphological and Chemical Mechanisms of Elongated Mineral Particle Toxicities*, J. Toxcol. Environ. Health, Part B 14:40 – 75 (2011).

[217] Oberdorster, et al., *Size dependent lymphatic short-term clearance of amosite fibers in the lung*, *Ann. Occup. Hygiene* 32(suppl. 1):149–156 (1988).

[218] Aust, et al., *Morphological and Chemical Mechanisms of Elongated Mineral Particle Toxicities*, J. Toxcol. Environ. Health, Part B 14:40 – 75 (2011) (citing Dodson, et al., *Characteristics of the asbestos concentration in the lung as compared to asbestos concentration in various levels of lymph nodes that collect drainage from the lung*, *Ultrastruct. Pathol* 31:95–133 (2007)).

[219] Aust, et al., *Morphological and Chemical Mechanisms of Elongated Mineral Particle Toxicities*, J. Toxcol. Environ. Health, Part B 14:40 – 75 (2011) (citing Monchaux, et al., Translocation of mineral fibres though the respiratory system after injection into the pleural cavity of rats. *Ann. Occup. Hyg.* 26:309–318 (1982).

[220] Kohyama et al., *Analysis of asbestos fibers in lung parenchyma, pleural plaques, and*

Asbestos fibers have been found in mesenteric lymph nodes in autopsies of individuals with asbestos exposure, supporting the hypothesis that lymph drainage is an important translocation mechanism for asbestos in the human body. Kurimoto et al. (2009) found in cases of peritoneal mesothelioma asbestos fibers at concentrations >10,000 fibers/g dry tissue, which were found in all samples of intra-abdominal tissue examined (except in the small intestine).[221]

150. Heller et al. (1996) reported that "[s]ignificant asbestos fiber burdens were detected in 9 out of 13 women with household asbestos exposure (69.2%), and in 6 out of 17 women who gave no exposure history."[222] Three women had more than 1,500,000 chrysotile asbestos fibers/gram wet weight and no commercial amphibole. Significantly, one of the women who had chrysotile alone lived with her father who was an "asbestos and insulation worker" and another had a husband who "worked as carpenter and with concrete." In addition, one other woman with chrysotile along found in her ovarian tissue had a household member who worked in a shipyard. This shows that insulation and shipyard exposures are not, as is often implied, synonymous with commercial amphibole exposure. The "great majority of the fibers were greater than 3 microns with a minimum aspect ration of 10" and much of the asbestos found in the ovaries of these three women with no commercial amphibole was longer than 10 microns. The fact that 35% (6/17) of women with no reported history of asbestos exposure had elevated asbestos detected in their ovarian tissue underscores my belief that many people are unwittingly exposed to asbestos.

151. Heller et al. (1999) examined the tissues of seven peritoneal malignant mesotheliomas in women, with no recorded asbestos exposure history, by transmission electron microscopy (TEM) energy-dispersive spectroscopy, and electron diffraction. [223] Asbestos fiber burdens were found in 86% (6 of 7) of the cases with no known asbestos exposure. Two showed chrysotile alone, and one showed chrysotile and tremolite. These findings strongly support the need for experienced investigators to take thorough exposure histories and underscore how false conclusions could be drawn about whether asbestos causes mesothelioma of the peritoneum in women. Furthermore, recent data indicates that millions of women were unwittingly exposed to asbestos from cosmetic talc products, exposures that were likely missed in many women counted as unexposed in historic medical literature.

152. There are many cases of peritoneal mesothelioma in cohorts of chrysotile-exposed populations with a significant excess of mesothelioma, and peritoneal mesotheliomas

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mesothelioma tissues of North American insulation workers, Ann. N. Y. Acad. Sci. 643: 27-52 (1991).

[221] Kurimoto et al., *Malignant peritoneal mesothelioma: Quantitative analysis of asbestos burden*, Pathology Intern. 59: 823-827 (2009).

[222] Heller et al., *Asbestos Exposure and Ovarian Fiber Burden*, Am. J. Indust. Med. 29: 435 – 439 (1996).

[223] Heller et al., *Presence of asbestos in peritoneal malignant mesotheliomas in women*, Int. J. Gynecol. Cancer 9: 452-455 (1999).

have been well documented in chrysotile exposed populations. Borow et al. described 72 cases of mesothelioma from a hospital in New Jersey near an asbestos mill. Twenty one of the cases (29%) were peritoneal. Chrysotile was the main asbestos used in the mill, with some crocidolite having been present in some of the processes. Only chrysotile was used in the textile division as far as can be determined.[224]

153. Egilman et al. (2010) reported an important "black swan" case of peritoneal mesothelioma in a 62 year old who worked for many years in a Canadian chrysotile mine claimed to lack trace tremolite.[225] Egilman et al.'s "black swan" puts the theory that tremolite-free chrysotile can't cause mesothelioma of the peritoneum to rest.

154. Additional cases further support my opinion that relatively low doses of asbestos, including chrysotile asbestos, causes mesothelioma of the peritoneum. Loggie et al. (2001) reported two cases of peritoneal mesothelioma in "mechanics with occupational exposure to asbestos" from brake linings.[226] Similarly, Ruiz et al. (2011) reported a case of mesothelioma of the peritoneum in a patient living with an auto brake mechanic.[227] Mirabelli et al. (2008) reported on a woman with mesothelioma of the peritoneum with environmental exposure to chrysotile (without tremolite) due to living and working close to a chrysotile mine.[228]

155. Experimental data in animals shows that peritoneal injection of asbestos causes mesothelioma. Intra-pleural and intra-peritoneal inoculation of asbestos into a variety of animals, especially rats and hamsters, has been the most frequently used technique for assessment of mesothelioma induction demonstrating that if asbestos reaches the peritoneal surface it is capable of inducing mesothelioma.[229] My own work, Frank et al. (1998), showed that the UICC Reference chrysotile used in these experiments was free of tremolite to exceptionally low levels. Therefore, chrysotile asbestos itself appeared to be the responsible carcinogen.[230]

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[224] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).
[225] Egilman et al., *A case of occupational peritoneal mesothelioma from exposure to tremolite-free chrysotile in Quebec, Canada: A black swan case*. Am. J. Ind. Med. 54:153–156 (2010).
[226] Loggie et al., *Prospective Trial for the Treatment of Malignant Peritoneal Mesothelioma*, The Am. Surgeon 67:999-1003 (2001).
[227] Ruiz et al., *Mesotelioma Peritoneal Maligno – Informe de un Caso y Revision de la Lituratura*. Rev. Med. Inst. Mex. Seguro Soc. 49(1):79-84 (2011).
[228] Mirabelli et al., *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy*. Occup. Environ. Med. 65:815–819 (2008) (referring to Case 22).
[229] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).
[230] Frank et al., *Carcinogenic implications of the lack of tremolite in UICC reference chrysotile*. Am J Ind Med 34: 314-317. (1998).

156. Browne and Smither (1983), had six cases of peritoneal mesothelioma with six months or fewer of exposure, and three cases with two or fewer months.[231]

157. Camargo et al. (2011) reviewed occupational exposure to asbestos and ovarian cancer and supported a causative association.[232] These authors discussed the difficulties in differentiating between ovarian cancer and peritoneal mesothelioma in females and found asbestos causative for ovarian cancer.

158. Kanarek (2016) concluded:

> It is clear from the epidemiology studies that many types of occupational exposures to all types of asbestos fibers, in many different exposures settings, including mining, making materials out of asbestos and working with asbestos in place, have contributed to peritoneal mesothelioma causation. In addition, asbestos neighborhood exposures have been documented to be causally associated. It is clear that both amphibole and chrysotile asbestos exposed people of both sexes in many countries of the world have died from peritoneal mesothelioma.[233]
>
> ....
>
> Welch et al. conducted a case-control study of 40 cases of primary peritoneal mesothelioma cases compared to controls with appendicular cancer. Twelve of the cases had asbestos exposure as compared to eight of the controls and eight had done brake lining work as compared to six controls.[234]

[231] Browne et al., *Asbestos-related mesothelioma: factors discriminating between pleural and peritoneal sites*, Br. J. Indus. Med. 40: 145-152 (1983).
[232] Camargo et al., *Occupational exposure to asbestos and ovarian cancer: a meta-analysis*, Environ. Health Perspect. 119: 1211-1217 (2011).
[233] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2.- 7 (2016).
[234] Kanarek et al., *Peritoneal Mesothelioma and Asbestos: Clarifying the Relationship by Epidemiology*, Epidemiology 6:2 2 - 7 (2016).

V. Substantial Epidemiological Data Supports the Consensus that All Types of Asbestos Can Cause Mesothelioma in Humans.

159. "There is a broad consensus that chrysotile asbestos causes human malignant mesothelioma. . . . The scientific basis for the mesothelial carcinogenicity of chrysotile is an established body of published epidemiological studies, animal carcinogen assays, and pleural fiber burden studies."[235]

160. In addition to these consensus documents from national and international agencies, numerous peer-reviewed epidemiological studies, meta-analyses, reviews and reports also conclude that chrysotile asbestos causes mesothelioma.[236] Lemen provides an excellent summary of some of the most important epidemiological evidence regarding asbestos.[237]

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[235] Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015).

[236] Kanarek, *Mesothelioma from Chrysotile Asbestos: Update*. AEP Vol. 21, No. 9, pp. 688-97 (2011); Henley et al., *Mesothelioma incidence in 50 states and the District of Columbia, United States, 2003 – 2008*. Int. J. Occup. Environ Health Vol. 19; 1 – 10 (2013); Elliott et al., *Lung cancer mortality in North Carolina and South Carolina chrysotile asbestos textile workers*. Occup. Environ. Med. 10.1136 (2012); Li et al., *Cohort studies on cancer mortality among workers exposed only to chrysotile asbestos: a meta-analysis*. Biomed. Environ. Sci. 17(4):459-468 (2004); Loomis et al., *Lung cancer mortality and fibre exposures among North Carolina asbestos textile workers*. Occup. Environ. Med. 66:535-542 (2009); Hein et al., *Follow-up study of chrysotile textile workers: Cohort mortality and exposure-response*. Occup. Environ. Med. 64:616–625 (2007); Silverstein et al., *Developments in asbestos cancer risk assessment*. Am. J. Ind. Med. 52:850–858 (2009); Finkelstein et al., *Malignant Mesothelioma among employees of a Connecticut factory that manufactured friction materials using chrysotile asbestos*. Ann. Occup. Hyg. 54(6):692–696 (2010); Egilman et al., *A case of occupational peritoneal mesothelioma from exposure to tremolite-free chrysotile in Quebec, Canada: A black swan case*. Am. J. Ind. Med. 54:153–156 (2010); Pira et al., *Mortality from cancer and other causes in the Balangero cohort of chrysotile asbestos miners*. Occup. Environ. Med. 66:805–809 (2009); Mirabelli et al., *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy*. Occup. Environ. Med. 65:815–819 (2008); Turci et al., *Role of associated mineral fibres in chrysotile asbestos health effects: The case of Balangeroite*. Ann. Occup. Hyg.; 53:491–497 (2009); Everatt et al., *Occupational asbestos exposure among respiratory cancer patients in Lithuania*. Am. J. Ind. Med. 50:455–463 (2007); Madkour et al., *Environmental exposure to asbestos-response relationship with mesothelioma*. Eastern Mediterranean Health J. 15:25–38 (2009); Yano et al., *Mesothelioma in a worker who spun chrysotile asbestos at home during childhood*. Am. J. Ind. Med.;52: 282–287 (2009); Baumann et al., *Pleural mesothelioma in New Caledonia: An acute environmental concern*. Cancer Detect Prev. 31:70–76 (2007); Baumann et al., *Pleural mesothelioma in New Caledonia: Associations with environmental risk factors*. Environ. Health Perspect. 119:695–700 (2011); Nishikawa et al., *Recent mortality from mesothelioma, historical patterns of asbestos use, and adoption of bans: A global assessment*. Environ. Health Perspect. 116:1675–1680 (2008); Welch et al., *Asbestos and peritoneal mesothelioma among college-educated men*. Int. J. Occup. Environ. Health., 11: 254–258 (2005); Lemen, *Asbestos in brakes: exposure and risk of disease*. Am. J. Ind. Med. 2004; 45(3):229-237 (2004); Frank et al.,

161. The epidemiological evidence that all forms of asbestos cause human malignant mesothelioma is so convincing that a consortium of epidemiological and public health groups recently came together to issue the following position statement:

> A rigorous review of the epidemiologic evidence confirms that all types of asbestos fibre are causally implicated in the development of various diseases and premature death. Numerous well-respected international and national scientific organizations, through an impartial and rigorous process of deliberation and evaluation, have concluded that all forms of asbestos are capable of inducing mesothelioma, lung cancer, asbestosis and other diseases. These conclusions are based on the full body of evidence, including the epidemiology, toxicology, industrial hygiene, biology, pathology, and other related literature published to the time of the respective evaluations. . . .
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> [A]n Italian chrysotile mining cohort in Balangero, Italy, has been followed up over the years (Piolatto, 1990; Mirabelli, 2008) and has demonstrated a statistically significant four-fold excess (6 cases vs. 1.5 expected) of pleural mesothelioma among blue-collar workers, and also among other classes of workers as well as among allied workers (Mirabelli, 2008). The chrysotile mined at Balangero was reported to be free of tremolite and other amphiboles.[238]

This position was endorsed by at least eight mainstream professional organizations, including the American College of Epidemiology, the American Public Health Association and the Canadian Society for Epidemiology and Biostatistics.

162. A recent peer reviewed article by Markowitz cited similar evidence for the ability of chrysotile asbestos to cause mesothelioma:

Carcinogenic implications of the lack of tremolite in UICC reference chrysotile. Am. J. Ind. Med. 34(4):314-317 (1998); Smith et al., *Chrysotile asbestos is the main cause of pleural mesothelioma.* Am. J. Ind. Med. 30:252-266 (1996); Cullen, *Chrysotile asbestos: enough is enough.* Lancet. 351(9113):1377-1378 (1998); Landrigan et al., *The hazards of chrysotile asbestos: a critical review.* Ind. Health 37(3):271-280 (1999); Landrigan et al., *Collegium Ramazzini call for an international ban on asbestos.* Am. J. Ind. Med. 47(6):471-474 (2005); Stayner et al., *Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis.* Am. J. Public Health 86:179-186(1996).

[237] Lemen, Asbestos: Risk Assessment, Epidemiology, and Health Effects. 2d Ed., Chapter 5, *Epidemiology of Asbestos-Related Diseases and the Knowledge that Led to What is Known Today*, Boca Raton: Taylor and Francis (2011) at pages 131 – 267.

[238] Position Statement on Asbestos from the Joint Policy Committee of the Societies of Epidemiology (JPC-SE) June 4, 2012.

> Epidemiological studies have shown that malignant mesothelioma has developed among a variety of workers whose exclusive or near-exclusive exposure to asbestos was to chrysotile asbestos. Examples include the Quebec chrysotile miners and millers (33 cases of malignant mesothelioma); workers who were active at the chrysotile mines in Balangero, Italy, or used the mine products (17 cases of malignant mesothelioma); textile workers at North Carolina textile plants (8 cases of mesothelioma and pleural cancer); textile workers at a South Carolina plant (3 malignant mesothelioma); railroad machinists (14 malignant mesotheliomas); and workers in a friction products plant (6 cases of malignant mesothelioma). In addition, malignant mesothelioma has been associated in neighborhood or household settings with levels of exposure to chrysotile that are generally less than those seen in companion occupational environments, including seven cases of malignant mesothelioma among Quebec residents in mining areas and five cases following household or environmental exposure to chrysotile in relation to the Balangero mine.[239]

163. Prior risk assessments looking at the potency of the various fiber types used unreliable and incomplete data about exposures and thus yielded unreliable conclusions. A recent meta-analysis, using only epidemiological studies with more reliable data, yielded results which show that chrysotile is much more potent for causing mesothelioma than previously believed.[240] These authors recognize that "[a]sbestos is a well-known

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[239] Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015) (citing Camus et al., *Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer*, N. Engl. J. Med. 338(22):1565–1571 (1998)); Mirabelli, *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy,* Occup Environ Med 65(12):815–819 (2008); McDonald et al., *Mesothelioma in Quebec chrysotile miners and millers: epidemiology and aetiology,* Ann. Occup. Hyg. 41(6):707–719 (1997); Loomis et al., *Lung cancer mortality and fibre exposures among North Carolina asbestos textile workers*, Occup. Environ. Med. 66(8):535–542 (2009); Hein et al., *Follow-up study of chrysotile textile workers: cohort mortality and exposure-response*, Occup. Environ. Med. 64(9):616–625 (2007); Mancuso, *Relative risk of mesothelioma among railroad machinists exposed to chrysotile*, Am. J. Ind. Med. 13(6):639–657 (1988); Teta et al., *Mesothelioma in Connecticut, 1955-1977. Occupational and geographic associations*, J. Occup. Med., 25(10):749–756 (1983); Finkelstein et al., *Malignant mesothelioma among employees of a Connecticut factory that manufactured friction materials using chrysotile asbestos*, Ann. Occup. Hyg. 54(6):692–696 (2010).

[240] Burdorf et al., *Applying Quality Criteria to Exposure in Asbestos Epidemiology Increases the Estimated Risk.* Ann. Occup. Hyg., Vol. 55, No. 6, pp. 565–568 (2011) (discussing Gezondheidsraad, Asbestos—risks of environmental and occupational exposure, The Hague, the Netherlands: Health Council of the Netherlands, report 2010/10E (2010). Available at

carcinogen responsible for cancer of the pleura and peritoneum (mesothelioma) and lung cancer. The profound consequence of historical exposure to asbestos is well documented in many countries. *Id.* (citing Lin et al., 2007).[241] Based on the more accurate measurements, the authors of this risk assessment recommended lowering the exposure limit to 0.002 f/cc or 2% of the current PEL (0.1 f/cc) set by OSHA in the United States.

164. IARC's recent update on the carcinogenicity of asbestos points out the weaknesses, limitations and incomplete nature of two risk assessments, (Berman & Crump (2003 and 2008) and Hodgson & Darnton (2000)), that suggested large potency differences between amphibole forms of asbestos and chrysotile.[242] IARC pointed out that neither Berman et al. (2003 and 2008) nor Hodgson et al., (2000) considered the important data on chrysotile potency data from Loomis et al. (2009) and Mirabelli et al. (2008). Egilman et al. (2005)[243] and Finkelstein et al. (2010)[244] reported additional cases of mesothelioma from a cohort exposed primarily to chrysotile, yet neither Berman et al. (2008) or Hodgson et al. (2009) included this data, leading to the erroneous implication that chrysotile was less potent than the evidence suggests. Using the new data first reported by Egilman (2005) and reasonable assumptions, Finkelstein (2011) concluded as follows:

> The oft-repeated statement that there were no cases of mesothelioma from the Connecticut friction materials plant studied by McDonald et al. (1984) is not correct. We have described five cases of mesothelioma from the files of a Connecticut law firm and mentioned two cases previously identified by Teta et al. (1983). Calculations suggest that mesothelioma rates at this plant were similar to those observed in the South Carolina textile factory and in Quebec mining and milling. These observations have implication for the risk assessment of chrysotile asbestos.

165. IARC also noted that "there is a high degree of uncertainty concerning the accuracy of the relative potency estimates derived from the Hodgson & Darnton and Berman & Crump analyses because of the severe potential for exposure misclassification in these

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www.gezondheidsraad.nl/en/publications/asbestos-risks-environmental-and-occupational-exposure. Accessed March 28, 2012).

[241] Lin et al., *Ecological association between asbestos-related diseases and historical asbestos consumption: an international analysis.* Lancet. 369: 844–9 (2007).

[242] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012) (discussing Berman et al., *Update of potency factors for asbestos-related lung cancer and mesothelioma.* Crit. Rev. Toxicol. 38: Suppl 11–47 (2008) and Hodgson et al., *The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure*, Ann. Occup. Hyg., 44: 565–601 (2000)).

[243] Egilman et al., *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability.* Int. J. Occup. Environ. Health 11:360-371 (2005).

[244] Finkelstein et al, *Malignant Mesothelioma Among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos*, Ann. Occup. Hyg., 54(6) 692–696 (2010).

studies." Significantly, IARC found that the "Berman & Crump meta-analyses provided weak evidence that fibre length is a determinant of the potency of asbestos." Others believe that the disparity in results and methods renders quantitative risk assessments like these unreliable.[245] The lack of reliable exposure data for most of the historic cohorts of asbestos exposed workers was a fundamental reason why the EPA abandoned its attempt to develop a "bin-specific" model for quantifying the danger of various types and sizes of asbestos fibers.[246] The weight of the evidence supports the conclusion and it is my opinion that all forms of asbestos cause mesothelioma, that the fibers of all lengths can contribute to the risk of disease and that the existing data is insufficient to *quantify* any differences in the relative potency of the types of asbestos for causing disease.[247] Indeed, one of the authors of Hodgson & Darnton (2000) more recently wrote "All forms of asbestos, serpentine (chrysotile) and amphiboles (crocidolite, amosite, tremolite) are carcinogenic to humans, although *the potency of chrysotile might be lower than that of other types*."[248]

166. Soeberg, et al. (2016) observed that "[c]hrysotile-only exposures were noted in 4% of cases [in Australia] where the main type of exposure through occupational exposure in automotive mechanic work, as well as individuals exposed to asbestos products know to only contain chrysotile and person involved in production of chrysotile-only products."[249]

167. The commonly discussed potency estimates from the published, peer reviewed literature are outdated and based on erroneous and incomplete information. Risk assessments used

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[245] Elliott et al., *Lung cancer mortality in North Carolina and South Carolina chrysotile asbestos textile workers*. Occup. Environ. Med. 69:6 385-390 (2012) (citing Greenland, Meta-analysis. In: Rothman KJ, Greenland S, Lash TL. Modern Epidemiology. 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 652e82 (2008) for proposition that "the strong heterogeneity between cohorts underscores the potential differences between them and suggests a single estimate of effect may not be possible."). Berman himself has admitted to agreeing with eight criticisms of his methodology and only addressed two of them in the 2008 publication. *See* Berman, Wayne, Letter to Vivian Turner, EPA Science Advisory Board (1400F) re Comments on the Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos (July 3, 2008).

[246] Silverstein et al., *Developments in asbestos cancer risk assessment*. Am. J. Ind. Med. 52:850–858 (2009); Johnson S. Letter from Stephen L. Johnson, EPA Administrator to Dr. Agnes Kane, Chair of Science Advisory Board Asbestos Committee. 12/29/2009.

[247] *Id.*

[248] Brown et al., *Occupational cancer in Britain Respiratory cancer sites: larynx, lung and mesothelioma*, British J. of Cancer 107, S56 – S70 (2012) (citing IARC. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42, Vol. 7. International Agency for Research on Cancer: Lyon (1987); World Health Organization. Environmental Health Criteria 203: Chrysotile Asbestos. Geneva: World Health Organization; (1998)).

[249] Soeberg, et al., *Malignant mesothelioma in Australia 2015: Current incidence and asbestos exposure trends*, J. Tox. Environ. Health, Part B, 19:5-6, 173-179 (2016).

to form potency estimates must include accurate, up-to-date information about the incidence of disease. For example, Hodgson & Darnton (2000) and Berman & Crump (2003; 2008) do not include up-to-date mesothelioma data for the Balangero chrysotile cohort[250], the Connecticut chrysotile cohort[251] and fail to include data from Union Carbide's Bound Brook, N.J., plant[252] (which had large numbers of mesotheliomas and was recently recognized as a plant that used only short-fiber chrysotile asbestos). The calculations by Hodgson & Darnton (2000) and Berman & Crump (2003; 2008) include a small fraction of the 27 cases reported by Mirabelli et al. (2008). Furthermore, since 2008 these Italian researchers have identified additional mesotheliomas attributable to exposure to Balangero chrysotile [amphibole-free], calculating "an estimated Relative Risk of Mesothelioma in the Balangero Miners cohort of 5.6 (95% CI 2.6 to 10.7, with reference to the Piedmont Region population."[253] Hodgson & Darnton (2010) did further calculations including new data from the North Carolina cohort[254] of asbestos textile workers, leading to an increase in the relative potency of chrysotile by a factor of 10, showing the sensitivity of the methods. [255] Thus, Hodgson & Darnton (2000) and Berman & Crump (2003; 2008) are hopelessly inaccurate and grossly underestimate the mesothelioma risk posed by chrysotile asbestos.

168. As of 1996, according to Stayner et al. (1996), "[t]wo cases of mesothelioma have been reported among chrysotile asbestos miners and millers in Zimbabwe, where the chrysotile ores are believed to be free of tremolite contamination."[256]

169. Outside of the litigation context, makers and sellers of asbestos products have long admitted what they deny in court: asbestos of all types causes mesothelioma of the pleura and peritoneum.[257]

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[250] Mirabelli, *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy,* Occup Environ Med 65(12):815–819 (2008).

[251] Finkelstein et al, *Malignant Mesothelioma Among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos*, Ann. Occup. Hyg., 54(6) 692–696 (2010).

[252] Egilman et al., *Short Fiber Tremolite Free Chrysotile Mesothelioma Cohort Revealed*, Am. J. Ind. Med. (2016).

[253] Magnani et al., Malignant mesothelioma after exposure to chrysotile in Balangero, Italy: an update. 12th International Mesothelioma Interest Group Conference, 21 - 24 October 2014, Cape Town, South Africa.

[254] Loomis et al,. *Lung cancer mortality and fibre exposures among North Carolina asbestos textile workers*, J. Occup. Environ. Med. 66(8):535–542 (2009).

[255] Hodgson et al., *Mesothelioma risk from chrysotile*, Occup. Environ. Med. 67(6):432 (2009).

[256] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis,* Am. J. Public Health 86(2) (1996).

[257] Ford Motor Company, *Industrial Relations Bulletin 4 Industrial Hygiene - Asbestos*, (October 14, 1986); Ford Motor Company, *Industrial Relations Bulletin 4 Industrial Hygiene - Asbestos*, (February 22, 1995)(stating "Bronchogenic carcinoma (lung cancer) and mesothelioma of the peritoneum and pleura (cancer of membranous lining of abdominal and chest cavities) are also associated with asbestos inhalation."); Tawiah, Jacob, *Health-Hazards of Asbestos – Review*

VI. All Types of Asbestos Cause Lung Cancer, Asbestosis and Pleural Disease.

170. The Agency for Toxic Substances and Disease Registries (ATSDR) "assess[ed] all relevant toxicological testing and information that has been peer reviewed" and concluded in its 2001 Toxicological Profile on Asbestos that "[a]vailable evidence indicates that all asbestos fiber types are fibrogenic".[258] The American Thoracic Society (ATS) also concluded in its 2004 statement *Diagnosis and Initial Management of Non-Malignant Disease Related to Asbestos* that all fiber types can cause lung fibrosis (asbestosis).[259] Recently, Loomis et al. (2010) reported on four textile plants using chrysotile asbestos that have shown an increased risk of both asbestosis and lung cancer, where the incidence of both diseases increased with increasing dose of asbestos.[260] Loomis et al. also measured excess incidence of mesotheliomas among the various plants, including when plants that did not use commercial amphibole were excluded from the analysis.

171. Both the previously discussed toxicological data as well as the extensive human epidemiology prove that all forms of asbestos cause both lung cancer and asbestosis. ATSDR has concluded "[t]here is little doubt that all types of asbestos can cause lung cancer. For example, statistically significant increases in lung cancer mortality have been reported in workers exposed primarily to chrysotile".[261] The International Agency for Research on Cancer (IARC) has also concluded that chrysotile asbestos causes lung cancer in humans.[262] A recent meta-analysis by Li reaches the same conclusion.[263] Analysis of a chrysotile cohort in China also confirmed "that exposure to chrysotile asbestos is associated with an increased risk of death from lung cancer and asbestosis,

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of the Medical Literature, The Bendix Corporation Corporate Engineering Staff Southfield, Michigan (December 11, 1975) (stating "[p]resent medical knowledge associates asbestos with three primary diseases: Asbestosis; Bronchogenic (lung) Cancer; and Mesothelioma, a rare form of cancer. All three diseases affect the lungs and in the case of mesothelioma, the abdominal cavity may also be affected. Asbestosis can cause death but it is not always fatal, bronchogenic cancer. . . . All commercial forms of asbestos cause asbestosis, bronchogenic cancer and mesothelioma.").

[258] U.S. Public Health Service, U.S. Department of Health & Human Services. Toxicological profile for asbestos. Atlanta: Agency for Toxic Substances and Disease Registry; 2001.

[259] American Thoracic Society. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am. J. Respir. Crit. Care Med.;170(6):691-715 (Sep 15 2004).

[260] Loomis et al., *Asbestos fibre dimensions and lung cancer mortality among workers exposed to chrysotile*. Occup. Environ. Med. 67(9):580-4 (2010).

[261] U.S. Public Health Service, U.S. Department of Health & Human Services. Toxicological profile for asbestos. Atlanta: Agency for Toxic Substances and Disease Registry; 2001.

[262] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012).; IARC. Asbestos: Monograph on the Evaluation of Carcinogenic Risk to Man. Lyon: International Agency for Research on Cancer; (1988).

[263] Li et al., *Cohort studies on cancer mortality among workers exposed only to chrysotile asbestos: a meta-analysis*. Biomed. Environ. Sci. 2004 Dec;17(4):459-68.

and shows a clear exposure response relationship."[264] Asbestos may be more potent for causing lung cancer than some previously thought.[265] The evidence shows that even low level exposures to asbestos causes a substantial number of lung cancers in occupationally exposed workers.[266]

172. Markowitz recently laid out some of the evidence for asbestos as a cause of lung cancer:

> Asbestos is universally recognized as a human lung carcinogen. Like malignant mesothelioma, all of the major asbestos fiber types—chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite—are established as causing lung cancer, based on the cumulative scientific evidence provided by animal experiments, epidemiologic and pathology studies, and mechanism-based research.
>
> The International Agency for Research on Cancer and others have recently reviewed over 60 epidemiologic studies that examined the relation between occupational exposure to asbestos and lung cancer. Most studies showed excess risk of lung cancer from asbestos exposure. The considerable differences in the level of excess risk of lung cancer among different epidemiological studies vary according to a large number factors, including study methods, data quality, reference populations, age structure of study population, country, occupation, industry, industrial process, job tasks, calendar years, exposure intensity and duration, fiber type, fiber dimensions, smoking information, the presence of other lung carcinogens, and others. The best studied contrast in lung cancer risk in different exposure settings is between the Quebec mines and mills, where excess lung cancer risk is modest, and the textile factory in South Carolina, where the risk of lung cancer is much higher. Notably, in both settings, Canadian chrysotile asbestos was used.[267]

173. Clearly, lung cancer risk increases with cumulative exposure to asbestos.[268]

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[264] Deng et al., *Exposure-response relationship between chrysotile exposure and mortality from lung cancer and asbestosis*, Occup. Environ. Med. (2011).

[265] Gustavsson, *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. Epidemiol. 155 (11) (2002).

[266] De Matteis et al., *Impact of occupational carcinogens on lung cancer risk in a general population*. Int. J. Epidemiol. Advance Access (published March 31, 2012).

[267] Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015) (citations omitted).

[268] Henderson et al., *After Helsinki: a multidisciplinary review of the relationship between asbestos exposure and lung cancer, with emphasis on studies published during 1997-2004.* Pathology 36(6): 517–550 (2004); Consensus Report: *Asbestos, asbestosis, and cancer: the*

174. Older studies have confirmed the same: Seidman et al. studied amosite asbestos manufacturing workers in New Jersey and found a 3-fold increase in lung cancer deaths among workers who worked less than 1 year in the plant, which rose to 5.6-fold for workers who worked between 1 and 2 years, and 6.5-fold for workers who worked over 2 years.[269] Gustavsson et al. performed a population-based case–control study of 1,038 lung cancer cases in Stockholm, controlling for cigarette smoking, and found an OR of 1.90 (95% CI: 1.32–2.74) for cumulative exposure to asbestos of 4 fiber/mL-year of exposure to asbestos, and the risk on the lower end of the range of exposure was greater than that predicted by a linear dose–response relationship.[270]

175. Recent studies have confirmed and extended knowledge that lower levels of occupational exposure to all types of asbestos, including chrysotile, cause lung cancer. For example, Loomis et al. (2009)[271] identified mortality patterns among workers at four asbestos textile plants in North Carolina that used chrysotile almost exclusively and found an overall Standardized Mortality Ratio (SMR) for lung cancer of 1.96 (95% CI: 1.73–2.20). Among workers who worked for less than one year (< 1 year) and from 1 to 5 years, the SMR was 1.82 (95% CI: 1.50–2.19) and 1.86 (95% CI: 1.45–2.34), showing substantial risk with relatively brief exposures. Pira et al. followed up 1,973 Italian textile workers exposed to mixed fiber types from 1946 to 1996 and found an overall SMR of 282 (95% CI: 222–354) for lung cancer. Lung cancer risk rose with duration of employment from SMR 139.1 among workers who worked < 1 year at the plant to a SMR 250.8 among workers who worked 1 to <5 years at the plant, to a SMR 530.9 among workers of > 10 years work at the plant.[272] Hein et al. (2007) evaluated mortality among 3,072 workers at a South Carolina textile factory that used chrysotile asbestos and found an elevated SMR (1.54, 95% CI: 1.07–2.15) for workers with < 1.5 fiber/mL-year cumulative exposure;

Helsinki criteria for diagnosis and attribution, Scand. J. Work Environ. Health, 23:311-6 (1997); Lenters et al., *A meta-analysis of asbestos and lung cancer: is better quality exposure assessment associated with steeper slopes of the exposure-response relationships?*, Environ Health Perspect 119(11):1547–1555 (2011); Nielsen et al., *Occupational asbestos exposure and lung cancer—a systematic review of the literature,* Arch Environ Occup Health 69(4):191–206(2014); van der Bij et al., *Lung cancer risk at low cumulative asbestos exposure: meta-regression of the exposure-response relationship,* Cancer Causes Control 24(1):1–12 (2013); Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015).

[269] Seidman et al., *Short-term asbestos work exposure and long-term observation,* Ann. N. Y. Acad. Sci. 330:61–89 (1979).

[270] Gustavsson et al,. *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. Epidemiol. 155(11):1016–1022 (2002).

[271] Loomis et al,. *Lung cancer mortality and fibre exposures among North Carolina asbestos textile workers*, J. Occup. Environ. Med. 66(8):535–542 (2009) (reporting 277 workers died from cancer of the lung or trachea).

[272] Pira et al., *Cancer mortality in a cohort of asbestos textile workers,* Br. J. Cancer 92(3):580–586 (2005).

there was a clear trend of increasing lung cancer risk with increasing exposure.[273] An Italian case-control study by De Matteis et al. reviewed 1,537 lung cancer cases from the general population and found a lung cancer OR of 1.76 (95% CI: 1.42–2.18) among those deemed to have low exposure to asbestos.[274] In a Chinese study, Wang et al. reviewed the history of more than one thousand chrysotile asbestos manufacturing workers and found that the group with "low level" exposure to asbestos had a hazard ratio of 1.94 (95% CI: 0.84–4.46) with a statistically significant trend of lung cancer risk with increasing exposure.[275]

176. Markowitz recently addressed the issue of threshold risks for asbestos-induced cancers:

> Studies of asbestos-exposed cohorts provide evidence that a threshold for cancer risk has not been established for asbestos. Stayner et al. evaluated alternative exposure–response models using data from a South Carolina textile factory. [citation omitted] The study of asbestos-related diseases at this South Carolina textile factory was judged to employ one of the highest quality exposure assessments available in the published literature on asbestos-related disease [citation omitted] They found that the best model for lung cancer was linear on a multiplicative scale with the best data fit obtained when the threshold was set at zero. Referring to asbestos exposure, the authors concluded that "there was absolutely no significant evidence for a threshold in ...lung cancer." In a recent meta-analysis, van der Bij et al. used a variety of statistical models to examine the issue of lung cancer risk associated with relatively low exposure to asbestos and found that a natural spline model best fit the data. They note that no threshold for lung cancer risk due to asbestos exposure has been identified. In a recent systematic review of asbestos and lung cancer, Nielsen and colleagues report that most relevant meta-analyses have been predicated on linear dose–response relationship models, suggesting no exposure threshold for lung cancer risk. Nielsen et al. concluded that there "is no evidence for a no observed effect level concerning asbestos-related lung cancer."[276]

177. Pleural plaques are often considered to be markers for significant asbestos exposure. Recently, researchers in France found a statistically significant association between

[273] Hein et al,. *Follow-up study of chrysotile textile workers: cohort mortality and exposure-response*, J. Occup. Environ. Med. 2007;64(9):616–625 (2007).

[274] De Matteis et al., *Impact of occupational carcinogens on lung cancer risk in a general population*, Int. J. Epidemiol. 41(3):711–721 (2012).

[275] Wang et al., *A 37-year observation of mortality in Chinese chrysotile asbestos workers*, Thorax 67(2):106–110 (2012).

[276] Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015) (citations omitted).

pleural plaques and mesothelioma.[277] The unadjusted hazard ratio (HR) = 8.9, 95% confidence interval [CI] = 3.0 to 26.5 led Pairon et al. to conclude that "[t]he presence of pleural plaques may be an independent risk factor for pleural mesothelioma."[278] Welch, et al. (2015) found that mesothelioma risk increased in workers with pleural plaques.[279] Specifically, Welch, et al. observed that "[w]hile mortality risk for mesothelioma . . . was significantly elevated among workers without pleural changes [SMR = 6.08, 95% CI = 4.55 to 7.95], risk was further increased among those with pleural changes [SMR = 11.19, 95% CI = 7.65 to 15.79]." This generally accepted fact is important because pleural plaques are a far more common disease than mesothelioma and are easier, in general, to detect in epidemiological studies. Since populations with increased incidence of plaques are at increased risk of mesothelioma, epidemiological studies of workers such as brake workers that demonstrate increased incidence of plaques also demonstrate an increased risk of mesothelioma. It is universally acknowledged that pleural plaques require greater exposures to asbestos than mesothelioma. These unequivocal findings regarding plaques supplement the largely equivocal studies of "mechanics" that look for mesothelioma and demonstrate that this group has medically significant asbestos exposure in excess of that needed to cause mesothelioma.[280]

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[277] Ameille et al., *Asbestos-Related Diseases in Auto Mechanics*. Ann. Occup. Hyg. 56(1) 55 – 60 (2012).

[278] Pairon et al., *Pleural Plaques and the Risk of Pleural Mesothelioma*. JNCI (Advance Access January 25, 2013).

[279] Welch, et al., *Mortality Among Sheet Metal Workers Participating in a Respiratory Screening Program*, Am. J. Indust. Med. 58:378-391 (2015).

[280] The term "mechanics" is used in quotations here because the various studies frequently cited in litigation by automotive defendants as relating to "brake workers" generally use broad, non-specific job classifications like "mechanic", "garage" or "automobile repair and related services."

VII. Other Medical and Scientific Evidence that All Types of Asbestos Cause Mesothelioma.

178. In addition to the extensive reliable epidemiological evidence that all types of asbestos cause mesothelioma in humans, there is substantial other evidence from animal studies that supports my opinion that all types of asbestos cause mesothelioma in humans. Lung cancer and mesothelioma have been found in rats in inhalation studies. Although the results vary, at least one study, Wagner et al. (1974), found chrysotile caused as many cancers as crocidolite.[281] Markowitz recently observed the following in concluding laboratory science supports a finding that chrysotile asbestos causes mesothelioma:

> Rat inhalation and intrapleural injection studies have compared mesothelioma tumor production in response to exposure to the different types of asbestos. Rat inhalation studies show relatively low percentages of animals developing mesotheliomas, but the highest percentage of mesotheliomas was found in animals exposed to chrysotile. Wagner found 2.9% of chrysotile-exposed animals developed mesotheliomas versus 0.7 to 2.8% among the animals exposed to various amphiboles. Davis et al. also found mesotheliomas among rats exposed to chrysotile in inhalation studies, though fiber length confounded the findings. Intrapleural and intraperitoneal injection studies in different strains of rats show high proportions of tested animals developing mesotheliomas in response to various types of asbestos, including chrysotile.[282]

179. According to Stayner et al. (1996),

> Rats exposed to asbestos by inhalation also develop mesotheliomas, albeit at a low incidence. Wagner et al. exposed rats to 10 mg/m^3 of Union International Contre le Cancer reference asbestos for period of 1 day to 2 years; the mesothelioma yields were Amosite, 0.7%; anthophyllite, 1.4%; crocidolite, 2.8%; and Canadian chrysotile, 2.9%. No mesotheliomas were observed in control animals or animals exposed to chrysotile from Zimbabwe. Similarly, Davis et al. and Davis and Jones reported small numbers of mesotheliomas in response to 1-year inhalation exposures to Amosite, crocidolite, Canadian chrysotile, and Zimbabwe chrysotile. The highest mesothelioma incidence in these studies, 7.5%, was produced by exposure to long-fiber chrysotile. Although the low incidence rates and small numbers of animals make quantitative comparisons uncertain, it cannot be said that

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[281] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012) (discussing Wagner et al., *The effects of the inhalation of asbestos in rats.* Br. J. Cancer, 29: 252–269 (1974)).

[282] Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015) (citation omitted).

these studies provide convincing support for the amphibole hypothesis.[283]

. . .

Canadian chrysotile, and Zimbabwe chrysotile all produced mesotheliomas in rats after intrapleural inoculation.[284]

. . . .

Overall, the implantation studies suggest that chrysotile asbestos does have the potential to induce mesothelioma, but these studies do not resolve the question of whether or not chrysotile is less potent in this regard than the amphibole forms.[285]

180. Stayner et al. (1996) summarized the animal testing: "consistent dose-response relationship was observed in these experiments, but (summing across all dose groups) chrysotile asbestos produced mesotheliomas in 9.5% of the animals vs 5.1% for crocidolite."[286]

181. Proper scientific inquiry requires consideration of all forms of animal studies regarding asbestos exposure, including inhalation, instillation and injection studies. While each of these types of studies has limitations, they also have strengths and must be considered. This is no different than the strengths and limitations of various types of epidemiological studies or, for that matter, all types of scientific evidence.

182. Numerous animal studies using both intrapleural and intraperitoneal injection have demonstrated all forms of asbestos cause mesothelioma.[287] Studies exposing animals via intratracheal administration have shown that asbestos fibers induced lung tumors in rats, and lung tumors and mesotheliomas in hamsters.[288]

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[283] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis,* Am. J. Public Health 86(2) (1996).

[284] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis,* Am. J. Public Health 86(2) (1996).

[285] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis,* Am. J. Public Health 86(2) (1996).

[286] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis,* Am. J. Public Health 86(2) (1996).

[287] Wagner, *Experimental production of mesothelium tumours of the pleura by implantation of dusts in laboratory animals.* Nature, 196: 180–181 (1962); Wagner et al., *Mesotheliomas in rats following inoculation with asbestos.* Br. J. Cancer, 23: 567–581 (1969); Pott et al., *Relevance of non-physiologic exposure routes for carcinogenicity studies of solid particles. In: Toxic and Carcinogenic Effects of Solid Particles in the Respiratory Tract.* 4th International Inhalation Symposium Hanover 1 - 5 March, 1993. Mohr U, editor. Washington, D.C: ILSI-Press, pp. 109–125 (1993); Stanton et al., *Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals.* J. Natl. Cancer Inst. 67: 965–975 (1981).

[288] Pott et al., *Carcinogenicity studies on fibres, metal compounds, and some other dusts in rats.* Exp. Pathol. 32: 129–152 (1987); Smith et al., *Long-term health effects in hamsters and rats exposed chronically to man-made vitreous fibres.* Ann. Occup. Hyg., 31: 4B731–754 (1987);

183. At least one animal study, Kogan et al. (1987), demonstrated peritoneal mesothelioma in rats exposed to high doses of chrysotile asbestos via intragastric administration.[289] Tumors were seen in 18 of 75 exposed rats, between 18–30 months after the beginning of the experiment, including two peritoneal mesotheliomas, eight gastric adenomas, two gastric adenocarcinomas, one gastric carcinoma, one cancer of the forestomach, one small intestine adenocarcinoma, and three abdominal lymphoreticular sarcomas. No tumors were observed in 75 control animals.

184. Studies of asbestos-exposed pets have also confirmed a relationship between environmental exposure to asbestos and mesothelioma. A case control study showed an eight fold (statistically significant) increased risk of mesothelioma in dogs with asbestos exposures as compared to those without asbestos exposure.[290]

185. This conclusion is further supported by experimental data showing that chrysotile is transported to the pleural and peritoneum, and animal experiments showing development of lung fibrosis and lung cancer. Suzuki (2001) demonstrated that chrysotile is preferentially transported to mesothelial tissues, like the pleura, while amosite is more likely to be retained in the lung itself.[291] Fiber studies also show that asbestos, including chrysotile, is also transported to the peritoneum. Fibrosis has been produced in animals by inhalation or by intratracheal exposure to chrysotile.[292] In addition, studies in animals have reported increased incidence of lung cancer following chronic inhalation exposure to chrysotile.[293] Exposure to chrysotile fibers less than 5 microns in length (short fibers)

Pott et al., *Lung carcinomas and mesotheliomas following intratracheal instillation of glass fibres and asbestos*. In: Proceedings of the VIth International Pneumoconiosis Conference 20–23 September 1983. Bochum, Germany: International Labour Office, pp. 746–756 (1984).

[289] Kogan et al., *Possibility of inducing glandular cancer of the stomach in rats exposed to asbestos*. Br. J. Ind. Med. 44: 682–686 (1987). Given the shorter lifespan of rats as compared to humans, high doses of potential carcinogens are often used to evaluate the carcinogenic potential of a substance.

[290] Glickman et al., *Mesothelioma in pet dogs associated with exposure of their owners to asbestos*. Environ. Res. 32: 305–313 (1983).

[291] Suzuki. *Asbestos tissue burden study on human malignant mesothelioma*. Ind. Health. 39(2):150-60 (Apr 2001).

[292] O'Neil et al., *Lung Volume Changes in Rats Exposed to Chrysotile Asbestos*. Am. Rev. Respiratory Disease 123 (4) 146 (1981) ("Interstitial fibrosis was seen histologically in all exposed animals after one year and increased in severity during the year in air"). Purportedly tremolite-free Union Carbide brand asbestos produced similar results with less than half the dose.

[293] IARC. Asbestos: Monograph on the Evaluation of Carcinogenic Risk to Man. Lyon: International Agency for Research on Cancer; (1988); World Health Organization. Environmental Health Criteria 203: Chrysotile Asbestos. Geneva: World Health Organization; (1998).

is reported to increase the incidence of lung cancer, with a dose-response relationship.[294] The animal data strongly suggest that chrysotile asbestos fibers themselves, rather than amphibole contamination alone, play a role in causing mesothelioma.[295] The data do not support a claim that fibers less than 5 microns are inert or non-potent, nor was the adoption of the 5 micron length cut-off for NIOSH/OSHA measurements based upon any conclusion that fibers less than 5 microns in length are harmless.[296] Indeed, NIOSH made clear the 5 micron counting protocol was a method of convenience because it used a readily available microscope and that it was "*only an index of total fiber exposure and does not imply that shorter fibers do not pose a health hazard*".[297]

186. Other relevant data on the ability of asbestos to cause human cancer include toxicokinetics, routes of exposure, deposition, clearance, and translocation in humans, molecular pathogenesis, and mechanisms of carcinogenesis.

187. Recent experiments with cell cultures demonstrated that "continuous exposure to low doses of asbestos fibers" for more than a year "indicated that asbestos exposure induced the reduction of anti-tumor immunity."[298]

Asbestos Tissue Burden Studies

188. Lung asbestos burden studies are often discussed in the medical and scientific literature. Their usefulness may vary. According to Stayner et al. (1996), "studies of fiber counts in extrapulmonary sites raise serious questions about the validity of using lung burden studies for assessing mesothelioma risk. Several investigators reported cases in which short chrysotile fibers were the predominant fiber found in the pleura, pleural plaques, or pleural fibrotic tissue when amphiboles were the predominant fiber found in the lung. These results suggest that chrysotile may be preferentially translocated to the pleura and that the fiber counts found in the lung may not accurately reflect the concentrations found at the site for mesothelioma induction."[299]

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[294] Stayner et al., *An epidemiological study of the role of chrysotile asbestos fibre dimensions in determining respiratory disease risk in exposed workers*. Occup. Environ. Med. 65(9):613-9 (Sep 2008).

[295] Frank et al., *Carcinogenic Implications of the Lack of Tremolite in UICC Reference Chrysotile*. Am. J. Ind. Med. 34:314-317 (1998).

[296] Lemen, *Asbestos in brakes: exposure and risk of disease*. Am. J. Ind. Med. 2004; 45(3):229-237 (2004); Dodson et al., *Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review*. Am. J. Ind. Med 44:291–297 (2003).

[297] NIOSH, Revised Recommended Asbestos Standard, DWEW (NIOSH) Publication No. 77-169 (December 1976) (emphasis added).

[298] Ying et al., *Enhancement of regulatory T cell-like suppressive function in MT-2 by long-term and low-dose exposure to asbestos*, Toxicology 338:86-94. doi: 10.1016/j.tox.2015.10.005. (Epub October 23, 2015).

[299] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis*, Am. J. Public Health 86(2) (1996).

189. Because all mesothelial tissue essentially is the same, the ability of asbestos to cause mesothelioma in the body probably depends on the ability of asbestos to get to the site of the mesothelioma. The relative frequency of mesothelioma in different sites of the body is probably related to the lower doses of asbestos reaching the less common sites for mesothelioma. The most common site for mesothelioma is in the pleura, followed by the peritoneum, the pericardium and the tunica vaginalis.

190. A recent article, D'Antonio et al. (2015), reported a case of mesothelioma of the spermatic cord in a railroad worker with asbestos exposure.[300]

191. Studies of extrapulmonary human tissues demonstrate that inhaled or ingested asbestos can reach most parts of the human body. For example, Auerbach et al. (1980) found asbestos in human kidney, heart, liver, spleen, adrenal, pancreas, brain, prostate and thyroid tissues.[301] Walls et al. (2003), discussed a case of pericardial thickening and calcification where a pericardial biopsy "report [showed] the presence of 'numerous ferruginous (asbestos) bodies, some of which have the finely beaded appearance suggestive of asbestos.'"[302] I discuss additional findings of asbestos in extrapulmonary tissues below.

192. Several research organizations, including ATSDR[303] and IARC[304] have performed excellent reviews of the *in vivo* and *in vitro* evidence that supports a finding that all types of asbestos cause all the asbestos-related diseases, including mesothelioma (pleural, peritoneal, pericardial and tunica vaginalis). The *in vivo* and *in vitro* evidence overwhelmingly demonstrates that when asbestos comes in contact with mesothelial tissues, it causes the changes that can lead to mesothelioma.[305] Chrysotile asbestos, like all other types of asbestos, can and does cause various "mechanistic" events that are associated with mesothelioma, including the following: "impaired fibre clearance leading to macrophage activation, inflammation, generation of reactive oxygen and nitrogen

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[300] D'Antionio et al., *Malignant Mesothelioma of Spermatic Cord in an Elderly Man with a History of Asbestos Exposure*; Urology pii:S0090-4295(15)00920-6. Doi: 10.1016/j.urology.2015.09.020 (Oct 1 2015).

[301] Auerbach et al., *Presence of Asbestos Bodies in Organs Other than the Lung*. Chest 77:2 pp. 133 – 137 (February, 1980).

[302] Walls et al., *An uncommon clinical presentation of asbestos-related disease*. New Zealand Med. J. Vol. 116 No. 1171 (2003).

[303] U.S. Public Health Service, U.S. Department of Health & Human Services. Toxicological Profile for Asbestos. Atlanta: Agency for Toxic Substances and Disease Registry; 2001 at Chapter 3 and 4. The profile was peer reviewed and "reflects the ATSDR's assessment of all relevant toxicologic information that has been peer reviewed."

[304] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012), sections 3 (Cancer in Experimental Animals) and 4 (Other Relevant Data).

[305] Straif et al., *A review of human carcinogens--part C: metals, arsenic, dusts, and fibres*. Lancet Oncol.; 10(5):453-4 (May 2009).

species, tissue injury, genotoxicity, aneuploidy and polyploidy, epigenetic alteration, activation of signaling pathways, [and] resistance to apoptosis."[306]

193. Substantial evidence shows that asbestos fibers of all types can be inhaled deeply into the lung due to their aerodynamic qualities. Once in the lung, asbestos fibers of all types may also interact with lung epithelial cells, penetrate into the interstitium, and translocate to the pleura and peritoneum or more distant sites. Fibers that are not efficiently cleared or altered by physicochemical process (e.g. breakage, splitting, or chemical modification) are termed bio-persistent in the tissue where they are found. Many animal studies have looked at bio-persistence of asbestos in various tissues. As discussed above, while animal studies are important scientific evidence, it is important to be cautious when interpreting the data from such studies, due to methodological issues and differences between species.[307] As a recent review observed, "[t]he relevance of bio-persistence to [malignant mesothelioma] in humans has also been questioned. Due to the prolonged latency associated with mesothelioma, the absence of fibres at autopsy, some 40 years after first exposure, is hardly surprising".[308] These authors also cautioned regarding potential biases that can be interjected into animal models by preparation of samples. For example, Bernstein et al., were faulted for aggressively treating the asbestos fiber in a manner that would "markedly shorten the bio-persistence of fibres."[309]

194. While many investigators have looked at asbestos content in the lungs, lung asbestos content is less relevant to questions of mesothelioma causation than it is to questions of asbestosis and lung cancer causation; mesothelioma occurs in the mesothelial tissues around the lungs (pleura), the abdomen (peritoneum), heart (pericardium) and sex organs (tunica vaginalis).[310] Numerous investigators have looked at tissue beyond the lungs and

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[306] *Id.*

[307] IARC Man-made vitreous fibres, IARC Monograph Eval Carcinogens Risks Humans, 81: 1–381 (2002).

[308] Linton et al., *The ticking time-bomb of asbestos: Its insidious role in the development of malignant mesothelioma.* Critical Reviews in Oncology/Hematology 84:2 200 - 212 (September 2012).

[309] *Id.*

[310] *See*, e.g., Warnock et al., *Asbestos Burden and the Pathology of Lung Cancer*. Chest; 89:20-26 (1986) ("the pulmonary asbestos burden is probably not an accurate indicator of the degree of asbestos exposure"); Sebastien et al., *Asbestos Retention in Human Respiratory Tissues: Comparative Measurements in Lung Parenchyma and in Parietal Pleura,* in Biological Effects of Mineral Fibre, VOL. 1 Wagner, J.C., ed. (1980) (a lung asbestos count "is not a good indicator of pleural retention"); Suzuki et al., *Asbestos fibers and human malignant mesothelioma,* in *Advances in the Prevention of Occupational Respiratory Diseases*, Chiyotani, Hosoda, Aizawa, eds. (1998) (arguing that "asbestos fibers in the lung do not fully represent a total picture of asbestos exposure because translocated asbestos fibers are not retained in the lung."); Suzuki et al., *Asbestos Tissue Burden Study on Human Malignant Mesothelioma.* Ind. Health 2001, 39, 150-60 (questioning the adequacy of approach of "researchers have been focusing almost exclusively on asbestos fibers in the lung tissue"); Dodson et al., *A Technical Comparison of Evaluating Asbestos Concentration by Phase-Contrast Microscopy (PCM), Scanning Electron*

found asbestos, predominantly chrysotile, in people with mesothelioma. Studies confirm that asbestos fibers are biopersistent and accumulate in lung tissue as well as in lymph nodes.[311] Asbestos fibers have also been identified in the pleura following autopsy[312] and in the parietal pleura in samples collected during thoracoscopy.[313] Tissue asbestos measurements consistently show that chrysotile asbestos is related to human mesothelioma; there have been numerous reports of mesotheliomas in people where

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Microscopy (SEM), and Analytical Transmission Electron Microscopy (ATEM) as Illustrated From Data Generated From a Case Report, Inhalation Toxicology, 20:723-732, (2008) (questioning the approach of looking at the lung when pleural or peritoneal cancer is at issue because "[w]ith respect to cancer, the concentration of asbestos at the site where the tumor starts is thought to be the most important factor in determining causation. It is impossible to know how much asbestos it takes to produce an asbestos-induced disease."); Finkelstein, *Asbestos Fibre Concentrations in the Lungs of Brake Workers: Another Look.* Ann. Occup. Hyg. 52(6):455-461 (2008) (explaining that "[s]ince chrysotile is cleared from the lungs of brake workers, tremolite is arguably a better marker of exposure to Quebec chrysotile than is chrysotile itself"); Kohyama et al., *Analysis of Asbestos Fibers in Lung Parenchyma, Pleural Plaques, and Mesothelioma Tissues of North American Insulation Workers.* 643 Ann. N.Y. Acad. Sci. 27 (1991) (stating that "[d]espite the absence of high concentrations of chrysotile fibers in the lung, significant accumulation of chrysotile fibers in pleural and peritoneal tissues should be considered a potentially important factor in the induction of human malignant mesothelioma"); Baker, *Limitations in Drawing Etiologic Inferences Based on Measurement of Asbestos Fibers from Lung Tissue.* 643 Ann. N.Y. Acad. Sci. 61 (1991) (discussing the many problems with fiber burden analysis and criticizing fiber burden analysis as inadequate to estimate past exposures); McDonald et al., *The epidemiology of mesothelioma in historical context.* Eur. Respir. J. 9, 1932-1942, 1938 (1996) (discussing the potential problems and "substantial questions" of fiber burden analyses); Dufresne et al., *Fibers in Lung Tissues of Mesothelioma Cases Among Miners and Millers of the Township of Asbestos, Quebec.* Am. J. Ind. Med. 27:581-592, 587 (1995) (explaining that "[b]ecause of the relatively low durability of chrysotile asbestos in lung tissues, it is difficult if not impossible to relate chrysotile lung content to asbestos-related diseases in humans"); Frank et al., *Carcinogenic Implications of the Lack of Tremolite in UICC Reference Chrysotile.* Am. J. Ind. Med. 34:314-317 (1998); Adib et al., *Short, Fine and WHO Asbestos Fibers in the Lungs of Quebec Workers With an Asbestos-Related Disease.* Am. J. Industr. Med. (Online Accepted 4 February 2013).

[311] Dodson et al., *Asbestos content of lung tissue, lymph nodes, and pleural plaques from former shipyard workers.* Am. Rev. Respir. Dis. 142: 843–847 (1990); Dodson et al., *Measurements of asbestos burden in tissues.* Ann. N.Y. Acad. Sci. 1076: 281–291 (2006).

[312] E.g., Dodson et al., *Asbestos content of lung tissue, lymph nodes, and pleural plaques from former shipyard workers.* Am. Rev. Respir. Dis. 142: 843–847 (1990); Gibbs et al., *Fibre distribution in the lungs and pleura of subjects with asbestos related diffuse pleural fibrosis.* Br. J. Ind. Med., 48: 762–770 (1991); Suzuki et al., *Asbestos tissue burden study on human malignant mesothelioma.* Ind. Health, 39: 150–160 (2001).

[313] Boutin et al., *Black spots concentrate oncogenic asbestos fibres in the parietal pleura. Thoracoscopic and mineralogic study.* Am. J. Respir. Crit. Care Med. 153: 444–449 (1996).

chrysotile is the only or the vast majority of the fiber present.[314] Fundamentally, it is well recognized and generally accepted that lung tissue fiber burden does not provide an accurate index of prior exposures to chrysotile asbestos.

195. Because mesothelioma occurs outside the lung, in mesothelial tissue around the lungs, abdomen, heart and testes, the tissue burden analysis of greatest interest is that of tissue outside the lung. Auerbach et al., (1980), investigated the translocation of asbestos throughout the body and found asbestos bodies in nine different organs including the

[314] Godwin, *Letter to the Editor: Asbestos and Mesothelioma.* 204 JAMA 151 (1968) (finding that "[a]nalysis of tissue by x-ray diffraction indicated that chrysotile was the only form of asbestos present."); Rogers et al., *Relationship Between Lung Asbestos Fiber Type and Concentration and Relative Risk of Mesothelioma: A Case-Control Study.* 67 Cancer 1912 (1991) (reporting two cases of peritoneal mesothelioma with only chrysotile in their lungs, and two cases of mesothelioma with only chrysotile in their lungs with a history of exposure only to chrysotile); Roggli et al., *Asbestos Fiber Type in Malignant Mesothelioma: An Analytical Scanning Electron Microscopic Study of 94 Cases.* Am. J. Ind. Med. 23:605-614 (1993) (concluding that "chrysotile along with its contaminant, tremolite – are capable of producing mesotheliomas in humans and experimental animals."); Dufresne et al., *Fibers in Lung Tissues of Mesothelioma Cases Among Miners and Millers of the Township of Asbestos, Quebec.* Am. J. Ind. Med. 27:581-592 (1995) (concluding that chrysotile (and its contaminant tremolite) were likely the cause of several cases of mesothelioma among this population."); Dodson et al., *Asbestos in Extrapulmonary Sites: Omentum and Mesentery.* Chest. 117;486-493 (2000) (reporting that "[l]ong fibers of chrysotile reached the momentum in several cases, which indicates that chrysotile is also translocated and could be potentially important in the pathogenesis of peritoneal mesothelioma."); Kohyama et al., *Analysis of Asbestos Fibers in Lung Parenchyma, Pleural Plaques, and Mesothelioma Tissues of North American Insulation Workers.* 643 Ann. N.Y. Acad. Sci. 27 (1991) (finding "fibrotic pleura and/or hyaline plaques of these workers were found to contain mainly chrysotile, the converse was true for the lung parenchyma. . . . [L]arge numbers of chrysotile fibers were detected in the extrapulmonary sites, such as in the pleural plaques and in pleural and peritoneal mesotheliomatous tissues."); Suzuki et al., *Translocation of Inhaled Asbestos Fibers From the Lung to Other Tissues.* Am. J. Ind. Med. 19:701-704, 702 (1991) (reporting "asbestos fibers detected in [a type of peritoneal fibrosis] were overwhelmingly chrysotile"); Suzuki et al., *Asbestos fibers and human malignant mesothelioma, in Advances in the Prevention of Occupational Respiratory Diseases*, K. Chiyotani et al., eds. (1998) (indicating that "[t]he asbestos type seen in the mesothelial tissue was chrysotile alone in the majority (68/86; 79.0%)."); Suzuki et al., *Asbestos Tissue Burden Study on Human Malignant Mesothelioma.* Ind. Health 39, 150-160 (2001) (on review of lung and mesothelial tissue, the authors reported "[c]hrysotile was the most common asbestos type detected in the mesothelial tissues. It was present in 62 of the 64 cases (96.9%); chrysotile was exclusively detected in 48 of the 62 cases (77.4%)."); Suzuki et al., *Asbestos Fibers Contributing to the Induction of Human Malignant Mesothelioma.* Ann. N.Y. Acad. Sci. 982:160-176 (2002) (finding six cases of mesothelioma (including one case of peritoneal mesothelioma) with solely chrysotile present in the lung tissue.).

heart.[315] Huang et al. (1988), found asbestos in many different extrapulmonary sites.[316] In a series of papers, Suzuki et al. (2001;[317] 2002; [318] and 2005[319]) compared asbestos content of the lung tissue of mesothelioma victims and extra-pulmonary tissues. Asbestos is even found in stillborn infants where the likely route exposure was through the bloodstream of the mother.[320] Dodson et al. (2006), also discussed this evidence and additional findings.[321] Suzuki et al. (2002), found that

> In mesothelial tissues, chrysotile fibers were 30.3 times more common than amphiboles. . . . In some mesothelioma cases, the only asbestos fibers detected in either lung or mesothelial tissue were chrysotile fibers. . . . The average number of asbestos fibers in both lung and mesothelial tissues was two orders of magnitude greater than the number found in the general population. . . . The majority of asbestos fibers in lung and mesothelial tissues were shorter than 5 μm in length.[322]

Chrysotile asbestos "was exclusively detected in 55 of the 74 cases (74.3%)" of Suzuki's cases.[323] Based on those findings, Suzuki concluded:

> 1) Fiber analysis of both lung and mesothelial tissues must be done to determine the types of asbestos fibers associated with the induction of human malignant mesothelioma; 2) short, thin asbestos fibers should be included in the list of fiber types contributing to the induction of human malignant mesothelioma; 3)

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[315] Auerbach et al., *Presence of Asbestos Bodies in Organs other then the Lung*, Chest 77: 2 (February 1980). The authors found asbestos bodies in the kidney, heart, liver, spleen, adrenals, pancreas, brain, prostate, and thyroid.

[316] Huang et al., *Asbestos Fibers in Human Pulmonary and Extrapulmonary Tissues*, Am. J. Indust. Med. 14: 331 – 339 (1988).

[317] Suzuki et al., *Asbestos tissue burden study on human malignant mesothelioma.* Ind. Health. 39: 150–160 (2001).

[318] Suzuki et al., *Asbestos Fibers Contributing to the Induction of Human Malignant Mesothelioma.* Ann. N.Y. Acad. Sci. 982: 160-176 (2002).

[319] Suzuki et al., *Short, thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence.* Int. J. Hyg. Environ.-Health 208: 201–210 (2005).

[320] Haque et al., *Assessment of Asbestos Burden in the Placenta and Tissue Digests of Stillborn Infants in South Texas*, Arch. Environ. Contam. Toxicol. 35, 532 – 538 (1998).

[321] Dodson et al., *Measurements of asbestos burden in tissues.* Ann. N. Y. Acad. Sci., 1076: 281–291 (2006).

[322] Suzuki et al., *Asbestos Fibers Contributing to the Induction of Human Malignant Mesothelioma.* Ann. N.Y. Acad. Sci. 982: 160–176 (2002).

[323] Suzuki *et al., Asbestos Fibers Contributing to the Induction of Human Malignant Mesothelioma.* Ann. N.Y. Acad. Sci. *982:160-176 (2002)*

Results support the induction of human malignant mesothelioma by chrysotile.[324]

196. According to Suzuki et al. (2005), their data demonstrates that among asbestos types detected in the lung and mesothelial tissues, "chrysotile was the most common asbestos type to be categorized as short, thin asbestos fibers. . . . Compared with digestion technique of the bulk tissue, ashing technique of the tissue section was more effective to detect short, thin fibers. We conclude that contrary to the Stanton hypothesis, short, thin, asbestos fibers appear to contribute to the causation of human malignant mesothelioma." In Suzuki's lab, "such fibers were the predominant fiber type detected in lung and mesothelial tissues from human mesothelioma patients."[325]

197. Gordon et al. (2009) examined the lymph node and lung tissues of 100 cases and found chrysotile-only or chrysotile and tremolite alone in several cases and concluded that "mesotheliomas cannot be considered idiopathic or unrelated to chrysotile exposure when it is possible to identify the causative agent in the lungs and/or regional lymph nodes, regardless of the amount."[326] "These findings suggest that it is not prudent to take the position that short asbestos fibers convey little risk of disease."[327] Sebastien et al. (1980),[328] Dodson et al. (2000a; 2000b; and 2003),[329] and Adib et al. (2013)[330] further support a cautious approach to ignoring the effects of short, thin chrysotile fibers."[331]

198. The above-referenced evidence led one group of reviewers to comment that "[a]sbestos fibers are found in all organs of subjects either occupationally exposed or not exposed to asbestos."[332] Asbestos fibers translocated into the mesothelial tissue play an important

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[324] *Id.*

[325] Suzuki et al., *Short, thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence.* Int. J. Hyg. Environ.-Health. 208: 201–210 (2005).

[326] Gordon et al., *Abstract, Asbestos Fiber Burden Analysis of Lung and Lymph Nodes in 100 Cases of Mesothelioma.* Am. J. Respir. Crit. Care Med. 179; A5892 (2009).

[327] Suzuki et al., *Short, thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence.* Int. J. Hyg. Environ.-Health. 208: 201–210 (2005).

[328] Sebastien et al., *Asbestos retention in human respiratory tissues: Comparative measurements in lung parenchyma and in parietal pleura.* In: Wagner, J.C., editor. Biological effects of mineral fibers. Lyon: IARC. p 237–246 (1980).

[329] Dodson et al., *Asbestos content in the lymph nodes of nonoccupationally exposed individuals.* Am. J. Ind. Med. 37: 169–174 (2000a); Dodson et al., *Asbestos in extrapulmonary sites—Omentum and mesentery.* Chest. 117:486–493 (2000b); Dodson et al., *Asbestos fiber length as related to potential pathogenicity: A critical review.* Am. J. Indus. Med. 44:291–297 (2003).

[330] Adib et al., *Short, Fine and WHO Asbestos Fibers in the Lungs of Quebec Workers With an Asbestos-Related Disease.* Am. J. Ind. Med. (Online Accepted 4 February 2013).

[331] Gordon et al., *Abstract, Asbestos Fiber Burden Analysis of Lung and Lymph Nodes in 100 Cases of Mesothelioma.* Am. J. Respir. Crit. Care Med. 179; A5892 (2009).

[332] Miserocchi et al., *Translocation pathways for inhaled asbestos fibers*, Environ. Health 7:4; 1 – 8 (2008).

role for the induction of asbestos related serosal disease, such as pleural and peritoneal fibrosis, as well as malignant pleural and/or peritoneal mesothelioma.[333]

199. In 2014, a thorough review of published evidence on the pathogenicity of asbestos fibers shorter than 5 microns, concluded:

> In view of the experimental and epidemiological studies, the toxicity of SAF cannot be dismissed. The potential toxicity of SAF remains widely debated in the scientific community. The lower effect of SAF in comparison with LAF is mostly founded on experimental studies as few epidemiological studies took short fibers into consideration. Additional data are needed as recent epidemiological studies suggest a risk for short fibers. Based on literature data determining the role of fiber size in biological effects of asbestos fibers and on our present knowledge on their mechanism of action, it appears that the measurement of airborne asbestos concentrations limited to fibers with a length >5 μm leaves out other types of fibers that may also have health adverse effects.[334]

200. Gordon et al. (2009) presents interesting data on asbestos tissue burden for several different types of exposure including drywall joint compound and asbestos floor tile products. **Table X** presents interesting information showing that construction workers were often exposed primarily to chrysotile asbestos (and contaminant tremolite). In this study, 17 of 17 (100%) mesothelioma cases had only chrysotile or chrysotile and tremolite in both lung tissue and lymph nodes. This supports my opinion that chrysotile asbestos from any source can and does cause mesothelioma.

[333] Miserocchi et al., *Translocation pathways for inhaled asbestos fibers*, Environ. Health 7:4; 1 – 8 (2008) (citing Suzuki et al., *Short, thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence*. Int. J. Hyg. Environ.-Health. 208: 201–210 (2005)).

[334] Boulanger G. et al., *Quantification of short and long asbestos fibers to assess asbestos exposure: a review of fiber size toxicity*. Environmental Health 2014, 13:59; 1-18 http://www.ehjournal.net/content/13/1/59

TABLE X – Asbestos Lung and Lymph Node Burden in Patients Exposed to Construction Materials

CASE INFO.[335]		LUNGS				LYMPH NODES				PATIENT INFO		
Case No.	Asbestos History Occupation	Asbestos Concentration	Fiber Type	Ratio	AB	Asbestos Concentration	Fiber Type	Ratio	AB	Age First Exposed	Years Exposed	Age at Diagnosis
8	Laborer (Construction)	7,650	Chrysotile, Tremolite	1:1	0 LM	22,465	Chrysotile, Tremolite	3:1	0 LM	24	35	72
9	Carpenter	6,750	Chrysotile, Tremolite	1:1	0 LM	15,675	Chrysotile, Tremolite	2:1	0 LM	26	34	69
10	Flooring Installation	16,585	Chrysotile, Tremolite	2:1	0 LM	45,876	Chrysotile, Tremolite	4:1	0 LM	33	12	69
15	Carpenter	4,600	Chrysotile, Tremolite	1:2	0 LM	15,460	Chrysotile, Tremolite	3:1	0 LM	28	32	68
26	Maintenance (Floor)	12,564	Chrysotile		0 LM	26,510	Chrysotile		0 LM	21	16	58
27	Floor Layer	14,600	Chrysotile, Tremolite	1:2	10 LM	35,500	Chrysotile, Tremolite	2:1	0 LM	22	12	64
30	Taper	14,500	Chrysotile, Tremolite	1:3	12 LM	65,200	Chrysotile, Tremolite	1:1	20 LM	31	31	76
31	Worked with Sheetrock	8,600	Chrysotile		0 LM	22,000	Chrysotile		0 LM	27	8	63
38	Carpenter	65,450	Chrysotile, Tremolite	5:4	24 LM	196,125	Chrysotile, Tremolite	5:2	36 LM	28	30	68
64	Laborer	23,650	Chrysotile		0 LM	64,200	Chrysotile		0 LM	24	10	66
66	Installed Flooring	22,760	Chrysotile		0 LM	31,120	Chrysotile		0 LM	25	10	63
67	Housewife-Husband Carpenter	19,650	Chrysotile		0 LM	28,310	Chrysotile		0 LM	21	30	72
72	House Repair	52,800	Chrysotile		12 LM	46,000	Chrysotile		16 LM	30	20	56
86	Carpenter	43,220	Chrysotile		18 LM	16,420	Chrysotile		0 LM	22	40	76
90	Lather	45,165	Chrysotile, Tremolite	4:1	0 LM	18,120	Chrysotile		0 LM	24	28	65
92	Roofing and Siding	14,800	Chrysotile, Tremolite	1:2	26 LM	54,650	Chrysotile, Tremolite	3:2	26 LM	19	40	69
99	Construction	58,250	Chrysotile		0 LM	142,450	Chrysotile		0 LM	23	32	67

[335] Gordon et al., *Abstract, Asbestos Fiber Burden Analysis of Lung and Lymph Nodes in 100 Cases of Mesothelioma*. Am. J. Respir. Crit. Care Med. 179; A5892 (2009).

201. While recognizing that fiber burden has its limitations, Gordon et al. (2009) offers some helpful information relating to sorts of exposures experienced by mechanics working on cars, trucks and heavy equipment. As the **Table Y** below, containing data excerpted from Gordon et al. (2009), shows, chrysotile asbestos was found in the lungs and lymph nodes of 13 of 13 (100%) of the vehicle mechanics sampled and in the lungs and lymph nodes of a parts distributor.[336] Gordon et al. (2009) did not find any commercial amphibole asbestos in the fourteen automotive service workers (0/14) analyzed. It should also be noted that, as expected in people with primarily exposure to chrysotile asbestos, no asbestos bodies were found amongst the vehicle mechanics analyzed. As Baker (1991) explains, "absence of fibers [in lung tissue] cannot be used to rule out exposure. Light microscopy and counting asbestos bodies cannot be used to estimate exposure to asbestos, especially for chrysotile."[337]

202. A new study of asbestos lung burden, Merler, et al. (2016) reinforces my view on the importance of lung fiber burden in people exposed to mainly or primarily to chrysotile asbestos, such as vehicle mechanics: "The lung burden among participants with MPM working in auto mechanics exposed to asbestos during brake repair, who exhibited the lowest fibre burden and the lowest percentage of amphibole fibres, is in line with the knowledge that chrysotile was the component of brakes and suggests caution on the opinion of absence of risk for MPM among auto mechanics."[338]

[336] Gordon et al., *Abstract, Asbestos Fiber Burden Analysis of Lung and Lymph Nodes in 100 Cases of Mesothelioma*. Am. J. Respir. Crit. Care Med. 179; A5892 (2009).

[337] Baker, *Limitations in Drawing Etiologic Inferences Based on Measurement of Asbestos Fibers from Lung Tissue*. 643 Ann. N.Y. Acad. Sci. 61, 69 (1991).

[338] Merler, et al., *Residual fibre lung burden among patients with pleural mesothelioma who have been occupationally exposed to asbestos*, Occup. Environ. Med. doi:10.1136/oemed-2015-103382 [E-pub ahead of print] (2016) (questioning the conclusions of Finley, et al., *Malignant pleural mesothelioma in US automotive mechanics: reported vs expected number of cases from 1975 to 2007*, Regul. Toxicol. Pharmacol. 6:104–16 (2012) and Roggli VL, Sharma A. *Analysis of tissue mineral fiber content*, In: Oury TD, Sporn TA, Roggli VL, eds. Pathology of asbestos-associated diseases. 3rd edn. New York, NY: Springer, 53–92 (2014)).

TABLE Y – Asbestos Lung and Lymph Node Burden in Patients Exposed to Auto Parts

CASE INFO[339]		LUNGS				LYMPH NODES				PATIENT INFO		
Case No.	Asbestos. History Occupation	Asbestos Concentratio0n	Fiber Type	Ratio	AB	Asbestos Concentration	Fiber Type	Ratio	AB	Age First Exposed	Years Exposed	Age at Diagnosis
6	Mechanic (Auto)	3,450	Chrysotile, Tremolite	4:1	10 LM	13,450	Chrysotile, Tremolite	3:1	0 LM	26	25	62
7	Mechanic (Auto)	5,875	Chrysotile, Tremolite	2:1	0 LM	16,750	Chrysotile, Tremolite	3:1	0 LM	29	30	64
16	Mechanic (Auto)	5,600	Chrysotile, Tremolite	1:2	0 LM	12,760	Chrysotile, Tremolite	1:1	0 LM	33	25	62
17	Mechanic (Auto, Tractor)	6,750	Chrysotile, Tremolite	1:1	0 LM	18,650	Chrysotile, Tremolite	3:1	0 LM	24	10	69
28	Auto Mechanic	7,800	Chrysotile		0 LM	16,200	Chrysotile		0 LM	28	25	71
35	Mechanic-Heavy Equipment	9,460	Chrysotile, Tremolite	1:3	0 LM	52,400	Chrysotile, Tremolite	3:1	0 LM	37	8	71
43	Mechanic	32,100	Chrysotile		0 LM	96,125	Chrysotile, Tremolite	2:1	0 LM	24	28	79
57	Auto Mechanic	12,450	Chrysotile		0 LM	24,240	Chrysotile		0 LM	22	30	70
63	Brakes-Mechanic	5,300	Chrysotile		0 LM	9,345	Chrysotile		0 LM	28	14	68
78	Heavy Equipment Mechanic	56,125	Chrysotile		0 LM	42,200	Chrysotile		0 LM	30	20	58
79	Housewife-Husband Mechanic	6,400	Chrysotile		0 LM	22,160	Chrysotile		0 LM	18	35	76
87	Auto Mechanic	18,900	Chrysotile		0 LM	6,500	Chrysotile		0 LM	26	35	79
95	Mechanic	15,450	Chrysotile		0 LM	31,800	Chrysotile		0 LM	24	30	74
97	GM-Parts Distribution	5,425	Chrysotile, Tremolite	1:2	0 LM	18,500	Chrysotile, Tremolite	3:1	0 LM	35	20	61

[339] Gordon et al., *Abstract, Asbestos Fiber Burden Analysis of Lung and Lymph Nodes in 100 Cases of Mesothelioma.* Am. J. Respir. Crit. Care Med. 179; A5892 (2009).

203. Although Butnor et al. (2003)[340] and Marsh et al. (2011)[341] claim that asbestos fiber burden studies of workers whose only exposure to asbestos was through mechanic work did not show increased levels of chrysotile in the absence of commercial amphibole fibers, Finkelstein (2008)[342] exposed some of the methodological errors and erroneous conclusions reached by Butnor et al. (2003). In a follow up letter to the editor, Finkelstein further explained the extent of the errors in Butnor et al. (2003):

> [Butnor et al. (2003)] demonstrated poor data analysis in my opinion. Their statistical analysis involved discarding 70% of the data and performing a qualitative overview of the remaining 30%. BSR failed to perform such basic analyses as plotting their data or analyzing correlation.
>
> . . .
>
> The final paragraph of the [Butnor et al. (2003)] paper states 'Lung burden analyses in automotive brake repair workers with MM in our series reflect tissue asbestos within the normal range or elevated commercial amphiboles'. This conclusion is incorrect. As shown in my paper (Finkelstein, 2008), using three different methods of statistical analysis, their lung burden analyses reflect not only elevated levels of commercial amphiboles but also elevated levels of Quebec asbestos. As shown here, levels of commercial amphiboles are not predictive of the levels of Quebec asbestos. The final sentence of the conclusion in BSR states: 'Other cases have tissue asbestos content indistinguishable from background controls and may be considered to be spontaneous or idiopathic'. This is not true. If one ignores the three cases in their series with detectable levels of chrysotile, the mean level of tremolite among the remaining seven cases is substantially higher (mu = 1120) than among the 19 controls (mu = 640). So, in fact the other cases have, on average, higher levels of Quebec asbestos in their lungs than do the controls.[343]

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[340] Butnor et al, *Exposure to Brake Dust and Malignant Mesothelioma: A Study of 10 Cases with Mineral Fiber Analyses*, Ann. Occup. Hyg. 47:4 325-330 (2003).

[341] Marsh et al, *Asbestos fiber concentrations in the lungs of brake repair workers: commercial amphiboles levels are predictive of chrysotile levels*, Inhalation Toxicology 23(12): 681-688 (2011). Dr. Roggli, a co-author of Marsh et al. (2011), has testified he became a co-author of this paper when informed by a lawyer for Honeywell, Inc. (an asbestos brake manufacturer),

[342] Finkelstein, *Asbestos Fibre Concentrations in the Lungs of Brake Workers: Another Look*, Ann. Occup. Hyg. 52(6):455-461 (2008).

[343] Finkelstein, *Asbestos Fibre Concentrations in the Lungs of Brake Workers: Reply to Roggli et al.*, Ann. Occup. Hyg. (2009) (references omitted).

204. The fact that Butnor et al. (2003) and Marsh et al. (2011) use erroneous data renders the conclusions in the papers unreliable.[344] Finkelstein (2012) points out additional discrepancies between the methods described in the publications and the actual methods used by Roggli's lab:

> It can thus be seen that there are discrepancies between the counts of both commercial amphiboles and tremolite recorded in the laboratory counting sheets and the concentrations of fibres reported and analysed by Marsh and colleagues. I have recently learned from Dr. Roggli that the reported concentrations of commercial amphiboles may be the *sum* of fibres 5μ or greater in length *and* asbestos bodies. This is an unusual procedure and is nowhere described in the published paper.
>
> . . .

Perhaps more troubling is Finkelstein's observation that "[t]he data Marsh and colleagues used in their regression analyses do not, in many cases, correspond with the fiber count data recorded by the electron microscopist."[345]

205. In addition to being base on values not found in the underlying data (per Finkelstein (2012), Butnor et al. (2003) and Marsh et al. (2011) both include manufacturing "arbitrary values for nondetects", a practice that Ogden (2010) referred to as including "a guess, for what [the value] might be."[346] Finkelstein's statistical approach was commented upon favorably by Helsel (2010):

> Finkelstein (2008) presents a strong case for performing hypothesis tests with methods drawn from survival analysis to compare control versus test groups, rather than only the heuristic comparison of group medians for censored data. He used MLE procedures— nonparametric methods for censored data are also available. The message of his paper is entirely consistent with 'Nondetects And Data Analysis' (Helsel, 2005), ignoring methods that incorporate-censored data lead to wrong decisions both economically and for human or ecosystem health. In my 2005 book, I used the flawed decision to launch the Challenger shuttle as the example. Finkelstein's example of [Butnor et al. (2003)'s] missing the effects of asbestos in the lungs of brake mechanics is equally compelling.[347]

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[344] Finkelstein, *Letter re Marsh et al*, Inhalation Toxicology 24(2): 139-140 (2012) (discussing discrepancies in 35% (7 of 20) data points among the Butnor (2003) cases and the laboratory data Butnor (2003) was purportedly based upon).
[345] Finkelstein, *Letter re Marsh et al*, Inhalation Toxicology 24(2): 139-140 (2012)
[346] Ogden, *Handling results below the level of detection*, Ann. Occup. Hyg. 54:3 255-256 (2010).
[347] Helsel, *Much Ado About Next to Nothing: Incorporating Nondetects in Science*, Ann Occup. Hyg. 54(3): 257-262 (2010).

As pointed out by Finkelstein (2012), the calculations in Marsh et al. (2011) "lack validity" because the underlying data reported by Roggli and Butnor "do not, in many cases, correspond to the fiber counts reported by Dr. Roggli on his laboratory counting sheets."[348] Based on the use of faulty data, Finkelstein recommended the calculations by Marsh et al. (2011) be re-done with correct data. That has not been done, to the best of my knowledge.

206. In 2013, Finkelstein performed another analysis of all the available mechanic fiber burden data from Roggli's lab (including the data from Butnor et al. (2003), Marsh et al. (2011), and Egilman (2012)), using statistical methods avoiding the disfavored use of fabricated data for non-detects (a/k/a values below the limit of detection in the analytical method).[349] Analyzing all Roggli's lung fiber burden data for mechanics available at the time, using statistical methods that avoid "the substitution of arbitrary values for nondetects," Finkelstein found the "concentrations of Quebec asbestos fibers in the lungs of brake mechanics were significantly higher than in the control population."[350]

207. Another recent publication by Finkelstein further undermines Butnor et al.'s hypothesis that people working with asbestos brakes are not exposed to levels of asbestos above those who are unexposed to asbestos.[351]

208. Finkelstein (2015) reported on a case of mesothelioma with no exposure to asbestos other than working in a garage where asbestos containing brakes linings (primarily Bendix brand) were drilled, old linings were peeled off, and the new lining was ground. In addition, the patient was exposed to high levels of asbestos from brakes from blowout of brake drums. Using proper statistical methods, Dr. Finkelstein compared this patient's lung burden of tremolite to the controls used by Butnor et al. (2003) and reported that the patient's "tremolite fibre burden was 6.6-fold higher (95% confidence interval = 1.5 – 23) than the mean tremolite fibre burden among control subjects." I strongly agree with Finkelstein (2015)'s conclusion that "[i]nhalation and retention of asbestos fibres places mechanics working with friction products at increased risk of asbestos-related diseases."

209. According the National Toxicology Program, "[t]remolite is a common contaminate in chrysotile and talc deposits."[352]

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[348] Finkelstein, *Letter re Marsh et al*, Inhalation Toxicology 24(2): 139-140 (2012) (discussing discrepancies in 35% (7 of 20) data points among the Butnor cases).

[349] Finkelstein, *The Analysis of Asbestos Count Data With "Nondetects": The Example of Asbestos Fiber Concentrations in the Lungs of Brake Workers*, Am. J. Indus. Med. 56:1482-1489 (2013).

[350] Finkelstein, *The Analysis of Asbestos Count Data With "Nondetects": The Example of Asbestos Fiber Concentrations in the Lungs of Brake Workers*, Am. J. Indus. Med. 56:1482-1489 (2013).

[351] Finkelstein, *Asbestos Fibres in the Lungs of an American Mechanic Who Drilled, Riveted, and Ground Brake Linings: A Case Report and Discussion*, Ann. Occup. Hyg. 59:4 525-527 (2015).

210. Despite my own research that calls into question the importance of tremolite contamination in chrysotile asbestos,[353] various observers have used sensitive analytical methods to determine whether brake shoes made from chrysotile asbestos contained tremolite. As shown by the **Table Z,** every sample of asbestos brakes tested was shown to contain trace levels of tremolite. Based on this evidence, it seems highly likely that the elevated lung burdens of tremolite and chrysotile discussed in Finkelstein (2008), Finkelstein (2013) and Finkelstein (2014) came from the chrysotile asbestos used in brake shoes. This data also explains the findings, by Gordon et al. (2009), of tremolite in lung tissue and lymph nodes in mesothelioma patients whose sole exposure to asbestos was from working with asbestos brakes.

TABLE Z

STUDY	PRODUCT	% BY VOLUME CHRYSOTILE (PLM)	% WEIGHT TREMOLITE (TEM)	AMPHIBOLE COUNT Fibers/gram of brake lining
Millette MVA7040	Ford Brake, MVA Sample # 1810	50%	0.019%	Not Calculated
Millette MVA7040	Brake Delco Drum, MVA Sample # 4792	50%	0.0004%	Not Calculated
Compton, Millette MVA8219	Bendix Brakes	30-50%	0.01%	Not Calculated
Compton, Millette MVA8219	Sears Roadhandler Brakes, MVA Sample # V1757B	25-45%	0.02%	Not Calculated
Compton, Millette MVA8219	Grizzly Relined Brake Shoes	30-50%	0.004%	Not Calculated
Longo, Mount - MAS	Ford Brake Shoes, Sample M59671-01A	25%	0.0072%	51,000,000
Longo, Mount - MAS	Ford Brake Shoes, Sample M59671-01B	25%	0.003%	31,000,000
Longo, Mount - MAS	Ford Brake Shoes, Sample M59671-01C	25%	0.019%	27,000,000

[352] Department of Health and Human Services. National Toxicology Program. Report on Carcinogens, Thirteenth Edition. Asbestos CAS No. 1332-21-4; pp. 1-3

[353] Frank et al., *Carcinogenic implications of the lack of tremolite in UICC reference chrysotile.* Am. J. Ind. Med. 34(4):314-317 (1998).

STUDY	PRODUCT	% BY VOLUME CHRYSOTILE (PLM)	% WEIGHT TREMOLITE (TEM)	AMPHIBOLE COUNT Fibers/gram of brake lining
Longo, Mount - MAS	Ford Brake Shoes, Sample M59671-01D	25%	0.002%	17,000,000
Compton, Millette MVA10494	Ford Brake Linings	25-35%	0.0005%	2,200,000
Compton, Millette MVA10494	Ford Brake Linings	25-35%	0.0006%	51,000,000
Millette MVA7380	Ford Brake Shoes, MVA Sample # T0193a	45%	0.01%	Not Calculated
Millette MVA7380	Ford Brake Shoes, MVA Sample # T0193b	40%	0.04%	Not Calculated
Millette MVA7621	Bendix Brakes, MVA Sample # T3145A	30%	0.003%	Not Calculated
Millette MVA7621	Bendix Brakes, MVA Sample # T3145B	30%	0.009%	Not Calculated
Millette MVA8005	Ford Brakes, MVA Sample # X0014A	20-40%	0.01%	32,000,000
Millette MVA8005	Ford Brakes, MVA Sample # X0014B	20-40%	0.01%	255,000,000
Millette MVA8005	Ford Brakes, MVA Sample # X0015A	20-40%	0.002%	132,000,000
Millette MVA8005	Ford Brakes, MVA Sample # X0015B	20-40%	0.01%	278,000,000
Compton, Millette MVA8680	VW Road Baron Brake Pads, MVA Sample # V2260	30-50%	0.01%	Not Calculated
Compton, Millette MVA8680	VW Replacement Brake Pad Set, MVA Sample # V2261-A	30-50%	0.03%	Not Calculated
Compton, Millette MVA8680	VW Replacement Brake Pad Set, MVA Sample # V2261-B	20-40%	NAD	Not Calculated

STUDY	PRODUCT	% BY VOLUME CHRYSOTILE (PLM)	% WEIGHT TREMOLITE (TEM)	AMPHIBOLE COUNT Fibers/gram of brake lining
Compton, Millette MVA8680	Grand Pro Disc Brake Pads	40-60%	0.03%	Not Calculated
Compton, Millette MVA8706	Pronto Remanufactured Brake Shoes	20-40%	0.0007%	Not Calculated
Millette MVA7275	Honda Brake Pad Kit	20%	0.0001%	Not Calculated
Millette MVA5744	Friction box materials, brake parts	35%		Not Calculated
Turner, Millette MVA7293	Western Auto Brake Kits and Brake Parts - Stop Rite Lining Set (Chrysler, Dodge, Plymouth, 1935-1941), MVA Sample # S1538	40%	0.0007%	Not Calculated
Turner, Millette MVA7293	Western Auto Brake Kits and Brake Parts - Stop Rite Lining Set (Chrysler, Dodge, Plymouth, 1935-1941), MVA Sample # S1539	40%	0.001%	Not Calculated
Turner, Millette MVA7293	Western Auto Brake Kits and Brake Parts - Stop Rite Lining Set (Chrysler, Dodge, Plymouth, 1935-1941), MVA Sample # S1540	40%	0.0005%	Not Calculated
Turner, Millette MVA7293	Western Auto Brake Kits and Brake Parts - Wizard 30 Brake Linings), MVA Sample # S1541	30%	NAD	Not Calculated

STUDY	PRODUCT	% BY VOLUME CHRYSOTILE (PLM)	% WEIGHT TREMOLITE (TEM)	AMPHIBOLE COUNT Fibers/gram of brake lining
Turner, Millette MVA7293	Western Auto Brake Kits and Brake Parts - Wizard 30 Brake Linings), MVA Sample # S1542	30%	0.0001%	Not Calculated
Turner, Millette MVA7385	VW Brake Pads (Western Auto Tough One, MVA Sample # S0229	10-20%	0.0001%	Not Calculated
Turner, Millette MVA7385	VW Brake Pads (Western Auto Tough One, MVA Sample # S0230	10-20%	0.0008%	Not Calculated
Millette MVA7275	Honda Brake Pad Kit, MVA Sample # T0142	15-20%	0.004%	Not Calculated
Millette MVA7275	Honda Brake Pad Kit, MVA Sample # T0143	15-20%	0.007%	Not Calculated
Millette MVA7380	Chrysler Brake Shoes	45%	0.004%	Not Calculated
Millette MVA7380	General Motors Brake Shoes	25%	0.04%	Not Calculated
Turner, Millette MVA7620	Girling Brake Pads	20%	0.0003%	Not Calculated
Turner, Millette MVA7651	Girling Brake Pads, MVA Sample # T3303	30%	0.05%	Not Calculated
Turner, Millette MVA7651	Girling Brake Pads, MVA Sample # T3327	20%	0.0006%	Not Calculated
Turner, Millette MVA7746	Abex Heavy Duty Brake Shoes	20%	0.00005%	Not Calculated
Millette MVA 8041	Western Auto Wizard 30 Brakes, MVA Sample #U1475	25-35%	NAD; 0.02%	Not Calculated

STUDY	PRODUCT	% BY VOLUME CHRYSOTILE (PLM)	% WEIGHT TREMOLITE (TEM)	AMPHIBOLE COUNT Fibers/gram of brake lining
Millette MVA 8041	Western Auto Wizard 30 Brakes, MVA Sample #U1483	25-35%	0.004%; 0.002%	Not Calculated
Compton, Millette MVA10529	Fruehauf Heavy Duty Brake Linings, MVA Sample # Z1541-A	20-40%	0.002%	Not Calculated
Compton, Millette MVA10529	Fruehauf Heavy Duty Brake Linings, MVA Sample # Z1541-E	20-40%		Not Calculated
Turner, Millette MVA7277	Friction Product - 6.5" disc, MVA Sample # S1436	50%	0.00002%	Not Calculated
Turner, Millette MVA7277	Friction Product - 6.5" disc, MVA Sample # S1437	50%	0.0012%	Not Calculated
Turner, Millette MVA7277	Friction Product - 6.5" disc, MVA Sample # S1438	50%	0.0003%	Not Calculated
Turner, Millette MVA7277	Friction Product - 6.5" disc, MVA Sample # S1439	50%	0.003%	Not Calculated
Turner, Millette MVA7277	Dust from box interior	1%	Not calculated	Not Calculated

211. Given the evidence of exposure to chrysotile and tremolite by mechanics working with asbestos brakes, I agree with the 2008 survey of passenger car and heavy truck auto shops in Iran by Kakooei et al. (2011) that concluded that "it is to be expected that the auto mechanics will suffer negative health effects due to exposure to the serpentine and amphibole asbestos fibres."[354]

212. Accordingly, it is generally recognized that a reliable occupational history – and not lung fiber burden studies – is the best indicator of past exposures to chrysotile[355] and that the

[354] Kakooei et al., *Evaluation of asbestos exposure during brake repair and replacement.* Ind Health; 49:374-80 (2011).
[355] Roggli et al., *Tremolite and Mesothelioma.* Ann. Occup. Hyg. 5:447-53 (2002).

absence of chrysotile on digestion, particularly at low magnification, does not provide a basis for concluding that an individual did not have a biologically significant exposure to chrysotile in the past.[356] In response to Baker's discussion of the limitations on fiber burden analysis, Dr. Selikoff remarked that

> Dr. Baker has reminded us that the tissue burden game is rigged. But it's the only game in town, and I think we have to learn how to use the information as best we can, and he has advised us how to do it.[357]

"The best indicator of past asbestos exposure (the gold standard) remains the detailed past work history."[358] Ten years before Begin et al. (2001) addressed the issue, Baker concluded "[f]iber burden studies cannot be used to estimate past exposure to chrysotile fibers or relative exposure to different types of asbestos because of the differential persistence and translocation of fibers in lung tissue."[359] In NIOSH's Division of Respiratory Disease Studies, *Occupational Respiratory Diseases*, DHHS (NIOSH) Publication No. 86-102 (September 1986) Dement et al. explained a reasonable approach for a clinician to determine whether a disease is asbestos-related:

> Clinical evaluation of the asbestos-exposed worker should include a full occupational and environmental history, full medical history, chest radiographs, and spirometry. Evaluation of the occupational and environmental history is especially important. The patient may have had only a few weeks of employment in construction or a shipyard as a summer job years before; yet, it is well documented that such brief exposures may manifest in asbestos related diseases 20 to 30 years later. It is important to assess other occupational exposures, such as coal or hard rock mining, which may produce rounded opacities on radiographic evaluation. Family history is also important. Asbestos insulation workers, as in many trades, tend to work in that trade from generation to generation. Therefore, the possibility of asbestos exposure in the home as a child should not be overlooked.

213. More recently, the Collegium Ramazzini published its concerns about over-reliance on fiber burden studies in causal attribution:

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[356] Baker, *Limitations in Drawing Etiologic Inferences Based on Measurement of Asbestos Fibers from Lung Tissue*. 643 Ann. N.Y. Acad. Sci. 61 (1991).
[357] Discussion of Baker, *Limitations in Drawing Etiologic Inferences Based on Measurement of Asbestos Fibers from Lung Tissue*. 643 Ann. N.Y. Acad. Sci. 61, 73 (1991).
[358] Begin et al., *Detailed Occupational History – The Cornerstone in Diagnosis of Asbestos-related Lung Disease*, Am. J. Resp. Critical Care Med. 163 (3 pt 1 598-99 (2001).
[359] Baker, *Limitations in Drawing Etiologic Inferences Based on Measurement of Asbestos Fibers from Lung Tissue*. 643 Ann. N.Y. Acad. Sci. 61, 69 (1991).

> The Collegium Ramazzini emphasizes that a carefully obtained history of occupational exposure to asbestos is the cornerstone of an accurate diagnosis of the diseases caused by asbestos (Bauer, Abraham et al. 2015. An occupational history taken by an experienced clinician and supplemented as necessary by an exposure assessment conducted by an experienced industrial hygienist is a far more sensitive and specific indicator of lung cancer risk from chrysotile asbestos than asbestos body counting or lung fiber burden analysis . . .. (Begin and Christman 2001, WHO 2009).[360]

Human Case Series and Case Reports Support the Conclusion that All Types of Asbestos Cause Human Mesothelioma and That Brief, Indirect and Low Level Exposures are Sufficient to Cause Mesothelioma

214. Case series – such as Wagner (1960) – are particularly informative in situations where there are identified occurrences of very rare conditions for which there are few, if any, established causal factors. In fact, recognition of even a small number of cases of the "sentinel" diseases – such as liver angiosarcoma related to vinyl chloride and malignant mesothelioma which is strongly related to asbestos exposure – establishes causation.[361]

215. Even industry-aligned scientists recognize that case series, in conjunction with case reports, can establish a causal relationship:

> Wagner et al. (1960) first reported an increased risk of MM in persons working and living in the vicinity of crocidolite mines in Northern Cape Provinces, South Africa. (Chapter 1). *This finding established MM as a definite disease and crocidolite as the cause.*[362]

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[360] Collegium Ramazzini. *Comments on the 2014 Helsinki Consensus Report on Asbestos.* (2014); pp 1-7 (citing Baur X, Abraham JL, Budnik T, Egilman DS, Frank AL, Hammar SP, Lemen RA, Soskolne CL (2015). *A critique of the pathology sections of the Consensus report Asbestos, Asbestosis, and Cancer, the Helsinki Criteria for Diagnosis and Distribution 2014: Recommendations*, and that of its complete FIOH website version and a call for revisions." in preparation; Begin R, Christman JW (2001). *Detailed occupational history: the cornerstone in diagnosis of asbestos-related lung disease.* Am J Respir Crit Care Med 163(3 Pt 1): 598-599 and World Health Organization, *Global health risks: mortality and burden of disease attributable to selected major risks,* (2009)).

[361] Checkoway et al., Research Methods in Occupational Epidemiology. 2nd ed. London: Oxford University Press; (2004).

[362] Gibbs et al., *Epidemiology and Risk Assessment,* In Craighead JE, & Gibbs AR, editors. Asbestos and Its Diseases. New York: Oxford University Press (2008) at p. 97 (emphasis added). Drs. Craighead and Gibbs testify for defendants in asbestos litigation. In a book review of the Craighead and Gibbs text in the New England Journal of Medicine, the reviewer discussed the biases of the authors' penchant for presenting "strong convictions" as facts when discussing

216. While outside the courtroom there is no doubt about the ability of chrysotile asbestos to cause mesothelioma, a recent Chinese case series, Gao et al. (2015)[363], reported on twenty-eight (28) confirmed mesotheliomas cases (nineteen (19) mesotheliomas of the peritoneum and nine (9) mesotheliomas of the pleura) in women with occupational and domestic exposure to chrysotile asbestos. This study provides evidence that is strikingly similar to Wagner et al. (1960) and is strong, additional evidence that chrysotile asbestos causes mesothelioma in the peritoneum and pleura.

217. The scientific community has concluded that, for sentinel diseases such as mesothelioma, case series reports can be sufficient by themselves to allow reliable conclusions to be drawn regarding causation. Again, as noted by Checkoway:

> "Case series reports can be virtually conclusive in their own right when the health outcome is a very rare disease or an uncommon manifestation of a relatively common condition."[364]

218. In an article by Gemba et al. (2012),[365] a significant number of mesotheliomas, both pleural and peritoneal, were found in the automobile manufacturing industry. As the article points out, friction materials and other automobile products contain predominantly chrysotile. This further supports the conclusion that chrysotile exposure gives rise to multiple types of mesothelioma.

219. Statistical epidemiology is not required to reach conclusions regarding causation. For example, one tongue-in-cheek publication pointed out that medical science does not require an epidemiological study to know that the use of parachutes reduces the risk of death for people jumping out of airplanes.[366] It is also important to recognize that occupational and environmental epidemiology is a blunt instrument and is not, in most cases, well suited to examining precise dose-response relationships.

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issues central to asbestos litigation (such as the ability of chrysotile asbestos to cause mesothelioma). Burdorf, Book Review of *Asbestos and its Diseases*, N. Engl. J. Med. 359;5, 544-45 (2008). It is because of the well-known biases of these authors – in favor of asbestos defendants – that it is notable that we agree on the importance of case series in the context of mesothelioma caused by asbestos.

[363] Gao et al. *Asbestos Textile Production Linked to Malignant Peritoneal and Pleural Mesothelioma in Women: Analysis of 28 Cases in Southeast China*, Am. J. Ind. Med., 58 (10): 1040-49 (2015).

[364] *Id.*

[365] Gemba et al., *National survey of malignant mesothelioma and asbestos exposure in Japan*. Cancer Sci. 103 (3):483 – 90 (2012).

[366] Smith et al., *Parachute use to prevent death and major trauma related to gravitational challenge: systematic review of randomised controlled trials*. Brit. Med. J. Vol. 327 (Dec. 23 – 27, 2003).

220. Because the consensus of the mainstream medical and scientific community is that, in North America and elsewhere, mesothelioma is a "signal tumor" or "signature tumor" with essentially one cause – asbestos – the scientific community has long considered individual cases of mesothelioma to be sentinel events. A sentinel event is a case of disease that, when it appears, signals the need for action. In 1983 Rutstein developed a list of Sentinel Health Events ("SHE-O") that are occupationally related.[367] Mesothelioma was included as a sentinel disease for asbestos exposure on the initial list of SHE-O, and has been included in all subsequent revisions. In fact, most asbestos scientists agree that the worldwide acceptance of mesothelioma as an asbestos-related cancer began with the case series published by Wagner in 1960.[368] When examining the question of causation of sentinel diseases like mesothelioma the scientific community recognizes case reports and case series reports are useful and valid tools.

221. Moreover, unlike many other cancers for which there are multiple, well-documented causal factors, mesothelioma is overwhelmingly caused by asbestos. "Mesothelioma is a rare cancer with one major etiologic exposure, therefore surveillance using each case as a sentinel event might seem more reasonable for this disease than for cancers with multi-factorial causation."[369] This so well-established, authors treat high rates of mesothelioma as proof that a group has elevated asbestos exposure, For example, Järvholm et al. (2014) stated "[t]he incidence of pleural malignant mesothelioma in the occupational groups was used as a marker of exposure to asbestos, that is, a high incidence of mesothelioma indicates a high exposure to asbestos."[370]

222. In the case of asbestos and mesothelioma, while case reports and case series support the overwhelming mass of scientific evidence discussed above on the issue of general causation – whether the substance causes the disease – they are more instructive as evidence supporting the scientific consensus that brief, indirect and low level exposures to asbestos are sufficient to cause mesothelioma. Proper scientific inquiry cannot ignore the implications of this wealth of published data. This applies to animal data as well.

223. The medical literature contains numerous case reports of mesotheliomas caused by as little as a few months, weeks, or even days of asbestos exposure.[371] Over the past several

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[367] Rutstein et al., *Sentinel health events (occupational): a basis for physician recognition and public health surveillance.* Am. J. Pub. Health. 73:1054-61 (1983).

[368] Wagner et al., *Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province.* Br. J. Ind. Med. [17], 260-271. (1960).

[369] Teschke et al., *Mesothelioma surveillance to locate sources of exposure to asbestos.* Can. J. Public Health; 88(3):163-8 (May 1997).

[370] Järvholm et al., *The Risk of Lung Cancer After Cessation of Asbestos Exposure in Construction Workers Using Pleural Malignant Mesothelioma as a Marker of Exposure,* JOEM 56(12) (December 2014).

[371] *See*, e.g., Skammeritz et al., *Asbestos Exposure and Survival in Malignant Mesothelioma: A Description of 122 Consecutive Cases at an Occupational Clinic.* 2(4) J. Occup. & Environ. Med. 228, 228-29 (Oct. 2011); Browne et al., *Asbestos-related Mesothelioma: Factors Discriminating between Pleural and Peritoneal Sites.* 40 Br. J. Ind. Med. 145, 147 (1983) (in a

decades many case series of mesothelioma have been published, and some of these reports provide detailed exposure histories for the mesothelioma cases. Many of these cases of mesothelioma have had limited exposure to asbestos, either because the exposure was of a short duration or because it occurred in a scenario unlikely to generate high levels of airborne fibers. For example, Browne and Smither,[372] in a study of 143 cases of mesothelioma, reported that 32 cases were exposed for less than one year, of whom 21 had no more than six months and 9 had no more than three months asbestos exposure. Greenberg and Davies,[373] reporting on cases of documented mesothelioma from a mesothelioma register between 1967-68, found several with short duration of exposure: one case had only 1 day of exposure to sawing asbestos cement sheets (an activity known to cause exceptionally high concentrations of airborne asbestos dust); another case had limited household exposure through her husband who worked in an asbestos factory for only two years, and a third had intermittent exposure to asbestos through her brother's work over a 3 year period. Newhouse and Thompson[374] reported 2 cases of mesothelioma with 2 months or less exposure to asbestos in a case series from London. In 1973, Borow[375] reviewed 72 cases, which included 2 mesotheliomas in stock clerks who worked in areas "not heavily contaminated with asbestos" for 10 months and 18 months respectively. In 2001, Neumann described the characteristics of 1,600 mesothelioma cases from the German mesothelioma registry from 1987-1999, and

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study of 143 cases of mesothelioma, 32 cases were exposed for under one year, of whom 21 had no more than six months of exposure and 9 had no more than three months); Greenberg et al., *Mesothelioma Register 1967-68*, 31 Br. J. Ind. Med. 91, 96, 103 (1974) (documenting mesothelioma following an asbestos exposure of 3 weeks in one case and 1 day in another); 1965 Newhouse and Thompson at 267 (documenting 2 cases of mesothelioma with 2 months or less exposure to asbestos); Borow et al., *Critical Review, Mesothelioma following Exposure to Asbestos: A review of 72 Cases*. 64(5) Chest 641, 642 (1973) (documenting mesotheliomas in stock clerks who worked in areas "not heavily contaminated with asbestos" for 10 months and 18 months respectively). *See* also National Institute for Occupational Safety & Health ("NIOSH"), U.S. Department of Health & Human Services, Workplace Exposure to Asbestos, Review and Recommendations, Publication No. 81-103, 3 (1980) ("[A]ll levels of asbestos exposure studied to date have demonstrated asbestos-related disease, and a linear relationship appears to best describe the shape of the dose-response curve. These considerations led the committee to conclude that there is no level of exposure below which clinical effects do not occur. Third, the absence of a threshold is further indicated by the dramatic evidence of asbestos-related disease in members of asbestos-worker households and in persons living near asbestos-contaminated areas. These household and community contacts involved low level and/or intermittent casual exposure to asbestos. Studies of duration of exposure suggest that even at very short exposure periods (1 day to 3 months) significant disease can occur.").

[372] Browne et al., *Asbestos-related mesothelioma: factors discriminating between pleural and peritoneal sites*. Br. J. Ind. Med.;40: 145-52 (1983).

[373] Greenberg et al., *Mesothelioma register 1967-68*. Br. J. Ind. Med. 31(2):91-104 (Apr 1974).

[374] Newhouse et al., *Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area*. Br. J. Ind. Med. 1993; 50(9):769-78 (Sep 1965).

[375] Borow et al., *Mesothelioma following exposure to asbestos: a review of 72 cases*. Chest. 64(5):641-6 (Nov 1973).

reported exposure as short as 1 month in one case.[376] Leigh et al., have described the characteristics of mesothelioma cases from 1945-2002 from the Australian Mesothelioma Surveillance Program, and report 3% of cases had exposures shorter than 3 months; the shortest duration of exposure for one case of mesothelioma was 16 hours of loading asbestos fiber on ships (an activity known to cause exceptionally high concentrations of airborne asbestos dust).[377] Miller[378] reviewed the details of 32 mesothelioma cases attributed to household exposure; one case lived in the same boarding house as several shipyard workers for eight years, another was a wife exposed to asbestos on her husband's clothes for only a year, and 3 others had household exposure less than 3 years. Hansen[379] described the pattern of asbestos-related disease in residents of Wittenoom Township in Australia, the location of a large asbestos mine. Twenty-four residents who had never worked in the mine developed mesothelioma by the time of this report; two had lived in Wittenoom for a very short time (6 weeks and 3 months) and had no other identified exposure to asbestos. Ascoli[380] described a series of 79 cases, including two cases with exposure to asbestos solely through living or working in a building with asbestos insulation or roofing. Schneider[381] reported a case of mesothelioma with asbestos exposure documented through a fiber burden analysis of the lung; her only exposure to asbestos was working for three years in an office where asbestos had been sprayed onto steel beams exposed in the ceiling. Chen[382] reported a similar case of mesothelioma in a man whose only exposure to asbestos was on visits to building sites in his role as executive of a building materials firm; asbestos exposure was documented through lung fiber analysis.

224. Lemen (2004) reported, based on a review of the published literature relating to brakes, more than two hundred cases of mesothelioma in people exposed to chrysotile asbestos from brakes.[383] Consistent with these findings, Welch et al. (2005), published a small case-control study of college-educated men with peritoneal mesothelioma and limited past exposures to asbestos, a finding contrary to the claim that asbestos-related peritoneal

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[376] Neumann, et al., *Malignant mesothelioma – German mesothelioma register 1987 – 1999*, Int. Archives Occup. Environ. Health 74: 383-395 (2001) (reporting that 84.8% of all the mesothelioma cases had an elevated asbestos fiber burden.).

[377] Leigh et al., *Malignant mesothelioma in Australia. 1945-2002.* Int. J. Occup. Environ. Health. 9(3):206-17 (Jul 2003).

[378] Miller, *Mesothelioma in Household Members of Asbestos-Exposed Workers: 32 United States cases since 1990.* Am. J. Ind. Med.; 47:458-62 (2005).

[379] Hansen et al., *Environmental exposure to crocidolite and mesothelioma: exposure-response relationships*. Am. J. Respir. Crit. Care Med.; 157(1):69-75 (Jan 1998).

[380] Ascoli et al., *Malignant mesothelioma in Rome, Italy 1980-1995. A retrospective study of 79 patients*. Tumori; 82(6):526-32 (Nov 1996).

[381] Schneider et al., *Pleural Mesothelioma Associated with Indoor Pollution of Asbestos*. J. Cancer Res. Clin. Oncol. 127(2):123-7 (2001).

[382] Chen et al., *Malignant mesothelioma with minimal asbestos exposure*. Hum. Pathol.; 9(3):253-8 (May 1978).

[383] Lemen, *Asbestos in brakes: exposure and risk of disease*. Am. J. Ind. Med. 45(3):229-237 (2004).

mesotheliomas only occur after high dose exposures to asbestos.[384] These cases demonstrate that very limited exposure to asbestos is found in many case series of mesothelioma. Case reports and case series show that mesothelioma occurs in people with exclusively chrysotile exposure or with mostly chrysotile exposure.[385] A recent registry study identified large numbers of pleural and peritoneal mesotheliomas with low-level chrysotile exposure in the Japanese automobile manufacturing industry.[386]

[384] Welch et al., *Asbestos and peritoneal mesothelioma among college-educated men*, Int. J. Occup. Environ. Health 11: 254–258 (2005).

[385] Enticknap et al., *Peritoneal Tumours in Asbestosis*. Br. J. Ind. Med. 21, 20 (1964); Godwin et al., *Letter to the Editor: Asbestos and Mesothelioma*. 204 JAMA 151 (1968); Champion, *Two Cases of Malignant Mesothelioma after Exposure to Asbestos*. 103 Am. Rev. Resp. Disease 821 (1971); Borow et al., *Mesothelioma following Exposure to Asbestos: A Review of 72 Cases*. Chest. 64;641-646 (1973) (finding all 72 cases of mesothelioma occurred since the mill had been using predominantly chrysotile and that "it has been shown clinically and experimentally that Chrysotile is a factor in the development of mesothelioma."); Acheson et al., *Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40-year follow-up*. Br. J. Ind. Med. 39;344-348 (1982) (reporting a case of mesothelioma among employees at a factory that used only chrysotile); Cullen et al., *Chrysotile Asbestos and Health in Zimbabwe: I. Analysis of Miners and Millers Compensated for Asbestos-Related Diseases Since Independence*. (1980), Am. J. Ind. Med. 19:161-169 (1991) (finding two cases of mesothelioma among Zimbabwe chrysotile miners; one confirmed at autopsy and a second probable case); Egilman et al., *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability*. Int. J. Occup. Environ. Health 11:360-371 (2005) (reporting new cases of mesothelioma out of a plant using predominantly chrysotile); Egilman et al., *A Case of Occupational Peritoneal Mesothelioma From Exposure to Tremolite-Free Chrysotile in Quebec, Canada: A Black Swan Case*. Am. J. Ind. Med. (2010) (reporting a peritoneal mesothelioma from exposure to asbestos in a chrysotile mine which may not be contaminated with tremolite).

[386] Gemba et al., *National survey of malignant mesothelioma and asbestos exposure in Japan*. Cancer Sci. 103 (3):483 – 90 (2012).

VIII. Asbestos Causes Numerous Other Cancers Including Ovarian Cancer, Laryngeal Cancer, As Well As A Variety Of Gastrointestinal Tract, Oro-Pharyngeal, And Kidney Cancers.

225. In addition to mesothelioma and lung cancer, the malignant diseases related to asbestos include, ovarian cancer, laryngeal cancer, as well as a variety of gastrointestinal tract, oro-pharyngeal, and kidney cancers.[387] I will discuss the evidence for each in greater depth, below.

226. IARC noted that a causal association between exposure to asbestos and cancer of the larynx was clearly established based on:

> fairly consistent findings of both the occupational cohort studies as well as the case-control studies, plus the evidence for positive exposure–response relationships between cumulative asbestos exposure and laryngeal cancer, cancer of the larynx reported in several of the well-conducted cohort studies. This conclusion was further supported by the meta-analyses of 29 cohort studies encompassing 35 populations and of 15 case-control studies of asbestos exposure and laryngeal cancer, cancer of the larynx undertaken by the [Institute of Medicine] (2006).[388]

227. IARC[389] concluded that a causal association between exposure to asbestos and cancer of the ovary was clearly established, based on "strongly positive cohort mortality studies of women with heavy occupational exposure to asbestos" including Acheson et al. (1982)[390]; Wignall et al. (1982)[391]; Germani et al. (1999)[392]; Berry et al. (2000)[393]; Magnani et al. (2008).[394] Additional support for the causal relationship between asbestos and ovarian cancer can be found in studies showing that women and girls with

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[387] Frank et al., *The Global Spread of Asbestos*, Ann. Global Health 80(4): 257 - 62 (2014).
[388] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012) (citing IOM, Asbestos: Selected Cancers. Institute of Medicine of the National Academy of Science [http://books.nap.edu/catalog/11665.html] (2006)).
[389] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012).
[390] Acheson et al., *Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40-year follow-up*, Br. J. Indust. Med. 39: 344–348 (1982).
[391] Wignall et al., *Mortality of female gas mask assemblers*, Br. J. Indust. Med. 39: 34–38 (1982).
[392] Germani et al., *Cohort mortality study of women compensated for asbestosis in Italy*, Am. J. Indust. Med. 36: 129–134 (1999).
[393] Berry et al., *Mortality from all cancers of asbestos factory workers in East London, 1933–80*, Occup. Environ. Med. 57: 782–785 (2000).
[394] Magnani et al., *Cancer risk after cessation of asbestos exposure: a cohort study of Italian asbestos cement workers*, Occup. Environ. Med. 65: 164–170 (2008).

environmental exposure to asbestos including Ferrante et al. (2007)[395], Reid et al. (2008)[396], and Reid et al. (2009).[397] According to IARC, two case-control studies, though not statistically significant, provided modest support for the link between asbestos and ovarian cancer.[398]

228. Laboratory findings documenting that asbestos can accumulate in the ovaries of women with household exposure to asbestos (Heller et al., 1996[399]) or with occupational exposure to asbestos (Langseth et al., 2007[400]) are consistent with the other evidence that asbestos causes ovarian cancer. For example, Heller et al. (1996) [401] was a histopathological study of ovaries from 13 women who had household contact with men who had documented exposure to asbestos, and of 17 women who gave no history of potential for asbestos exposure. Heller et al. (1996) found "significant asbestos fibre burdens" in the ovaries of nine (60.2%) of the exposed women and in only six (35%) of the unexposed women. Three of the exposed women had asbestos fibre counts in ovarian tissue of over 1 million fibres per gram (wet weight). By contrast, only one of the 17 women without household exposure had counts in that range.

229. Numerous studies show a positive association between exposure to asbestos and cancer of the pharynx, based on the positive findings in a series of well-conducted cohort studies of populations occupationally exposed to asbestos.[402] The positive findings of three case–

[395] Ferrante et al., *Cancer mortality and incidence of mesothelioma in a cohort of wives of asbestos workers in Casale Monferrato, Italy*, Environ. Health Perspectives, 115: 1401–1405 (2007).

[396] Reid et al., *The mortality of women exposed environmentally and domestically to blue asbestos at Wittenoom, Western Australia*, Occup. Environ. Med. 65: 743–749 (2008).

[397] Reid et al., *Gynecologic and breast cancers in women after exposure to blue asbestos at Wittenoom*, Cancer Epidemiol. Biomarkers Prev. 18: 140–147 (2009).

[398] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012) (citing Vasama-Neuvonen et al., *Ovarian cancer and occupational exposures in Finland*, Am. J. Indus. Med. 36: 83–89 (1999); Langseth & Kjærheim, *Ovarian cancer and occupational exposure among pulp and paper employees in Norway*, Scand. J. Work Environ. Health, 30: 356–361 (2004).

[399] Heller et al., *Asbestos exposure and ovarian fibre burden*, Am. J. Indus. Med., 29: 435–439 (1996).

[400] Langseth et al. *Asbestos fibres in ovarian tissue from Norwegian pulp and paper workers*, Int. J. Gynecol. Cancer 17: 44–49 (2007).

[401] Heller et al., *Asbestos exposure and ovarian fibre burden*, Am. J. Indus. Med., 29: 435–439 (1996).

[402] Selikoff et al., Asbestos-associated deaths among insulation workers in the United States and Canada, 1967–1987. Ann N Y Acad Sci, 643: 1 Third Wave 1–14 (1991); Sluis-Cremer et al., *The mortality of amphibole miners in South Africa, 1946–80*, Br. J. Indust. Med. 49: 566–575 (1992); Reid et al. *Aerodigestive and gastrointestinal tract cancers and exposure to crocidolite (blue asbestos): incidence and mortality among former crocidolite workers*, Int. J. Cancer 111: 757–761 (2004); Pira et al., *Cancer mortality in a cohort of asbestos textile workers*, Br. J. Cancer, 92: 580–586 (2005).

control studies, Zheng et al. (1992)[403], Marchand et al. (2000)[404] and Berrino et al. (2003)[405] provide additional support for the role of asbestos in cancer of the pharynx.

230. A meta-analysis conducted by the IOM provided additional support for the causal relationship between asbestos and cancer of the pharynx.[406]

231. IARC found a positive association between exposure to asbestos and cancer of the colorectum, based on the "fairly consistent findings of the occupational cohort studies, plus the evidence for positive exposure–response relationships between cumulative asbestos exposure and cancer of the colorectum consistently reported in the more detailed cohort studies."[407]

232. Four large, well-performed meta-analyses, Frumkin & Berlin (1988)[408]; Homa et al. (1994); IOM (2006)[409]; and some data in Gamble (2008)[410], support my opinion that asbestos cause cancer of the colorectum. I am aware of the non-positive findings of some case–control studies but find those results unpersuasive, as does IARC, because:

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[403] Zheng et al., *Risk factors for oral and pharyngeal cancer in Shanghai, with emphasis on diet,* Cancer Epidemiol. Biomarkers Prev. 1: 441–448 (1992).

[404] Marchand et al., *Laryngeal and hypopharyngeal cancer and occupational exposure to asbestos and man-made vitreous fibres: results of a case–control study,* Am. J. Indust. Med. 37: 581–589 (2000).

[405] Berrino et al., *Occupation and larynx and hypopharynx cancer: a job-exposure matrix approach in an international case–control study in France, Italy, Spain and Switzerland.* Cancer Causes Control, 14: 213–223 (2003).

[406] IOM, Asbestos: Selected Cancers. Institute of Medicine of the National Academy of Science [http:// books.nap.edu/catalog/11665.html] (2006).

[407] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012) (citing McDonald et al. *Dust exposure and mortality in chrysotile mining, 1910–75,* Br. J. Indus. Med. 37: 11–24 (1980); Albin et al., *Asbestos and cancer: An overview of current trends in Europe,* Environ. Health Perspectives 107: Suppl 2289–298 (1999); Berry, *Mortality from all cancers of asbestos factory workers in East London 1933–80,* Occup. Environ. Med. 57: 782–785 (2000); Aliyu et al., *Evidence for excess cancer of the colorectum incidence among asbestos-exposed men in the Beta-Carotene and Retinol Efficacy Trial,* Am. J. Epidemiol.162: 868–878 (2005).

[408] Frumkin et al., *Asbestos exposure and gastrointestinal malignancy review and meta-analysis,* Am. J. Indust. Med.14: 79–95 (1988).

[409] IOM, Asbestos: Selected Cancers. Institute of Medicine of the National Academy of Science [http:// books.nap.edu/catalog/11665.html] (2006).

[410] Gamble, *Risk of gastrointestinal cancers from inhalation and ingestion of asbestos* Regul. Toxicol. Pharmacol., 52: SupplS124–S153 (2008).

> [t]he majority of these case–control studies incorporated relatively little information on levels of asbestos exposure; indeed, most of them considered exposure as simply a dichotomous yes/no variable. Some of the case–control studies also may be compromised by inadequate duration of follow-up. Thus, [Garabrant et al. (1992)] may be subject to the criticism, offered by Gerhardsson de Verdier et al. (1992) that "the highest duration of exposure…was 'at least 15 years,' a period that may be too short to detect an elevated risk."[411]

233. The weight of the evidence supports my opinion that asbestos causes kidney cancer. Smith et al. (1989) provided persuasive evidence:

> The role of asbestos in the etiology of lung cancer and of mesothelioma of the pleura and peritoneum has been well documented. The evidence for a causal association between asbestos and other human cancers is not as extensive but suggests that asbestos may be carcinogenic at several different sites. This paper is concerned specifically with a possible causal association between asbestos and human kidney cancer. A review of the evidence to date indicates that only three human studies have sufficient statistical power to detect an excess mortality from kidney cancer among workers exposed to asbestos. All three were occupational cohort studies, and two of these gave strong direct evidence for such an excess; a study of U.S. insulators (kidney cancer SMR = 2.22, 90% CI 1.44-3.30), and a study of U.S. asbestos products company workers (kidney cancer SMR = 2.76, 90% CI 1.29-5.18). The third study, of Italian shipyard workers, reported excess mortality from "cancers of the kidney, urinary bladder, and other urinary organs" (SMR = 1.98, 90% CI 1.42-2.70). Further support for a causal association includes studies finding asbestos fibers in human kidneys and urine, as well as reports of kidney tumors in two animal bioassays. It is concluded that asbestos should be regarded as a probable cause of human kidney cancer.[412]

[411] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012) (citing Garabrant et al., *Asbestos and colon cancer: lack of association in a large case–control study*. Am J Epidemiol, 135: 843–853 (1992) and de Verdier et al., *Occupational exposures and cancer of the colon and rectum*, Am. J. Ind. Med. 22: 291–303 (1992)).

[412] Smith et al., *Asbestos and kidney cancer: the evidence supports a causal association*, Am. J. Ind. Med. 16(2):159-66 (1989).

234. According to Pesch et al. (2000), asbestos products workers have shown consistently elevated risk for renal cell carcinoma.[413] Enterline, et al. (1987) found a statistically significant excess risk of kidney cancer (7 cases observed and 2.54 expected, yielding an SMR=275.8).[414] McLaughlin et al. (1996) "considered the association of asbestos and [Renal Cell Carcinoma] development as the most consistently observed occupational link."[415]

[413] Pesch et al., *Occupational risk factors for renal cell carcinoma: agent-specific results from a case-control study in Germany*, Intern. J. of Epidem. 29: 1014-1024 (2000) (citing Enterline et al., *Asbestos and cancer: a cohort followup to death*, Am. J. Indus. Med. 44:396-401 (1987)).

[414] Enterline, et al., *Asbestos and cancer: a cohort followed up to death*, Brit. J. Indust. Med. 44: 396-401 (1987).

[415] Pesch et al., *Occupational risk factors for renal cell carcinoma: agent-specific results from a case-control study in Germany*, Intern. J. of Epidem. 29: 1014-1024 (2000) (citing McLaughlin et al., *Renal Cancer*, In Schottenfeld, D Fraumeni, JF (eds) Cancer Epidemiology and Prevention, New York: Oxford University Press pp. 1142-55 (1996).

IX. There is No Safe Exposure to Any Type of Asbestos: Exposures Above the Miniscule "Ambient" Levels of Asbestos Can Cause Mesothelioma in Humans.

235. Although different investigators have identified different ranges of asbestos in ambient air in different environments, I believe there is likely more asbestos in the air in urban environments than in rural environments. ATSDR has published ranges of asbestos in ambient air from 0.0001 f/cc (urban)[416] to 0.000000167 f/cc (rural)[417]. Any estimates of background in excess of 0.0001 f/cc (urban) are not true ambient background exposures and probably represent environmental exposures from a point source, such as a shipyard, construction site, or factory using asbestos. According to ATSDR, the cumulative lifetime exposure (in f/cc/years) from an outdoor ambient level for the general population at a level of 0.000002 f/cc, assuming people inhale 20 cubic meters of air per day and lives to age 70, spending 10% of time outdoors, equals 0.000014 f/cc/years. [418] Assuming occupational exposure of 0.1 f/cc (the current PEL) for 40 years, breathing 8 cubic meters per day, working 5 days per week, 49 weeks per year, equals 1.1 f/cc/years. The ATSDR's table with assumptions follows:

[416] U.S. Public Health Service, U.S. Department of Health & Human Services. *Public Health Statement Asbestos CAS#: 1332-21-4.* Atlanta: Agency for Toxic Substances and Disease Registry (September 2001).

[417] U.S. Department of Health & Human Services. Health Consultation, Evaluation of Community-Wide Asbestos Exposures EL DORADO HILLS NATURALLY OCCURRING ASBESTOS SITE EL DORADO HILLS BOULEVARD, EL DORADO HILLS, CALIFORNIA EPA FACILITY ID: CAN000906083 AUGUST 16, 2011. Atlanta, GA: Agency for Toxic Substances and Disease Registry (2011).

[418] U.S. Public Health Service, U.S. Department of Health & Human Services. *Public Health Statement Asbestos CAS#: 1332-21-4.* Atlanta: Agency for Toxic Substances and Disease Registry (September 2001)(Table 6-4 at page 169).

Table 6-4. Summary of Typical General Population and Occupational Exposures

Exposed population	Exposure medium	Typical concentration	Assumed exposure	Cumulative exposure level (f-yr/mL)	Estimated dose to gastrointestinal tract[a] (MF/day)
General population	Ambient (outdoor) air	$2x10^{-6}$ PCM f/mL	20 m^3/day, 70 years (10% of time outdoors)[e]	0.000014[b]	0.0000012[c]
	Indoor air[e]	$3x10^{-6}$ PCM f/mL	20 m^3/day, 70 years (90% of time indoors)[b]	0.00019[f]	0.000016[g]
	Drinking water	0.017 MFL[e]	2 L/day	–	0.034
Asbestos worker	Workplace air	0.1 PCM f/mL[h]	40 years, 8 m^3/day, 5 days/week, 49 weeks/year[i]	1.1[j]	0.16[k]

[a]Assumes 30% of inhaled fibers are transferred to stomach (NAS 1983)
[b]Approximate value based on EPA 1989e
[c]Cumulative exposure level (values in []): Typical concentration [$2x10^{-6}$ f/mL] x Life span [70 years] x Fraction of time outdoors [0.1]
[d]Dose to gastrointestinal tract:(values in []): Typical concentration [$2x10^{-6}$ f/mL] x Volume inhaled/day [20 m^3] x Fraction of time outdoors [0.1] x Fraction of inhaled fibers transferred to gastrointestinal tract [0.3] x 10^6 mL/m^3 x 10^{-6} MF/f
[e]Millette et al. 1980; concentration converted from TEM basis to PCM basis using 1 TEM f=1/60 PCM f (NRC 1984).
[f]Cumulative exposure level (values in []): Typical concentration [$3x10^{-6}$ f/mL] x Life span [70 years] x Fraction of time indoors [0.9]
[g]Dose to gastrointestinal tract:(values in []): Typical concentration [$3x10^{-6}$ f/mL] x Volume inhaled/day [20 m^3] x Fraction of time indoors [0.9] x Fraction of inhaled fibers transferred to gastrointestinal tract [0.3]x10^6 mL/m^3 x 10^{-6} MF/f
[h]Time-weighted average (TWA) Permissible Exposure Limit (PEL) (OSHA 1998c)
[i]NAS 1983
[j]Cumulative exposure level (values in []): Typical concentration [0.1 f/mL] x Working life span [40 years] x Fraction of air breathed in workplace [8 m^3/day/20 m^3/day x 5 days/7 days x 49 weeks/52 weeks]
[k]Dose to gastrointestinal tract:(values in []): Typical concentration [0.1 f/mL] x Volume inhaled/workday [8 m^3 x 5 days/7 days x 49 weeks/52 weeks] x Fraction of time outdoors [0.1] x Fraction of inhaled fibers transferred to gastrointestinal tract [0.3]x10^6 mL/m^3 x 10^{-6} MF/f

f/mL = fibers per milliliter; MF = million fibers; MFL = million fibers per liter; PCM = phase contrast microscopy; TEM = transmission electron microscopy

236. There is no safe level of exposure to any type of asbestos fiber. [419] Asbestos is genotoxic.[420] Genotoxic agents are recognized as having no safe level or threshold of

[419] Soeberg, et al., *Malignant mesothelioma in Australia 2015: Current incidence and asbestos exposure trends*, J. Tox. Environ. Health, Part B, 19:5-6, 173-179 (2016) (stating "The dose-response relationship between asbestos exposure and mesothelioma has no threshold, and exposures as short as 1 day were found.").

exposure for carcinogenic effects.[421] "Genotoxic damage can occur through direct interaction of retained asbestos fibres and mesothelial cells around the stomata of the parietal pleura."[422] "Although the exact mechanisms of asbestos carcinogenesis are not known at present, asbestos fibers, including chrysotile, are genotoxic and are, therefore, 'DNA reactive.'"[423]

237. In a 2016 study of asbestos lung burden in patients with mesothelioma of the pleura, the authors recognized that "[m]esotheliomas may occur at relatively low exposures to asbestos, as it is highlighted in the fraction of MPMs with low fibre counts or even below the [detection limits using electron microscopy].[424]

238. This is not a new or novel opinion in the medical and scientific community; rather the literature is replete with physicians and scientists reaching that opinion. In 1956, one asbestos company scientist published his opinion that "it is prudent to set the standard for cancerigenic [sic] substances substantially at zero . . . and no considerations can justify allowing inhalation of any concentration which is avoidable."[425]

239. In 1964, at a major medical conference on asbestos-related disease, another asbestos industry medical officer expressed the opinion clearly and concisely:

> Our own conclusion, as we began seeing what was happening in our own process, was that *the only safe amount of asbestos dust exposure was zero* and that the efforts in terms of achieving that lay basically in engineering, and, secondly, in education. But as far as a safe level of asbestos dust is concerned, our own conclusion in

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[420] Straif K et al., *A review of human carcinogens--part C: metals, arsenic, dusts, and fibres.* Lancet Oncol.; 10(5):453-4 (May 2009) (Table: Metals, arsenic, dusts and fibers assessed by the IARC Monograph Working Group).

[421] Patty's Industrial Hygiene (5th Ed.) Vol. 3 CH. 40 page 1872.

[422] Røe et al., *Malignant pleural mesothelioma: History, Controversy and Future of a Manmade Epidemic,* Eur Respir Rev 24:115-131 (2015).

[423] Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues,* Semin. Respir. Care Med. 36:334-346 (2015)(citing International Agency for Research on Cancer. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 100c: Arsenic, Metals, Fibres and Dusts. WHO Press (2012); Nymark et al,. *Molecular and genetic changes in asbestos-related lung cancer,* Cancer Lett 265(1):1–15 (2008); Jaurand et al., *Mesothelioma: Do asbestos and carbon nanotubes pose the same health risk?,* Particle Fibre Toxicol 6:16 (2009)).

[424] Merler, et al., *Residual fibre lung burden among patients with pleural mesothelioma who have been occupationally exposed to asbestos,* Occup. Environ. Med. doi:10.1136/oemed-2015-103382 [E-pub ahead of print] (2016).

[425] Smyth, *Improved Communication – Hygienic Standards for Daily Inhalation.* Ind. Hyg. Quarterly. 17(2) (1956) (Dr. Smyth was an employee of Union Carbide, which at the time was a major manufacturer of asbestos containing phenolics and which later became a major miner and distributor of asbestos).

Hogansville, Ga., is that *there is no safe level. The safe level is nil and anything above the safe level represents certain risk.*"[426]

This echoes the work of Merewether et al., of several decades earlier.[427]

240. Experienced medical and scientific experts continue to agree that there is no safe level of exposure to asbestos. For example, in 2011, the Inspector General for the United States Environmental Protection Agency wrote that "[a]sbestos is a human carcinogen with no safe level of exposure."[428]

241. Studies show an excess of mesothelioma with non-occupational exposures, which are generally understood to be at lower doses than occupational exposures. This data, in combination with the data that 90% or more of mesotheliomas have either a demonstrated history of exposure or substantial asbestos in lung tissue, again suggest that the potential of causation by asbestos should be considered for every mesothelioma. Brown et al. (2012) reported, based on analysis of three studies, that from 96% - 98% of mesotheliomas in men were attributable to asbestos exposure and from 75% - 90% of mesotheliomas in women were attributable to asbestos exposure.[429] According to Brown et al. (2012), 100% (1,937 of 1,937) of the cases were caused by asbestos.

242. More recently, the Health & Safety Executive in Great Britain reported that all cases of mesothelioma (2,538/2,538) in Great Britain were "caused by past exposure to asbestos."[430]

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[426] Wells, Ann. N.Y. Acad. Sci. 132 (1)1-766 (1965) (reporting discussion at page 336) (emphasis added).

[427] Merewether et al., *Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry*. Her Majesty's Stationery Office (1930).

[428] Environmental Protection Agency ("EPA"). Office of Inspector General, Early Warning Report: Use of Unapproved Asbestos Demolition Methods May Threaten Public Health, Report No. 12-P-0125 (December 14, 2011).

[429] Brown et al., *Occupational cancer in Britain Respiratory cancer sites: larynx, lung and mesothelioma*, Br. J. Cancer 107: 556 – 570 (2012) (analyzing data from Howel et al, *Routes of asbestos exposure and the development of mesothelioma in an English region*, Occup. Environ. Med. 54: 403–409 (1997); Yates et al, *Malignant mesothelioma in South East England: clinicopathological experience of 272 cases*. Thorax 52: 507–512 (1997); Rake, et al, *Occupational, domestic and environmental mesothelioma risks in the British population: a case-control study*, Br. J. Cancer 100(7): 1175–1183 (2009) for the male rates and Spirtas et al, *Malignant mesothelioma: attributable risk of asbestos exposure*, Occup. Environ. Med. 51: 804–811(1994) and Goldberg et al., *The French National Mesothelioma Surveillance Program*, Occup. Environ. Med. 63: 390–395 (2006) for the female rates).

[430] Health & Safety Executive, Health and Safety Statistics Annual Report for Great Britain 2014/15 (accessed at http://www.hse.gov.uk/statistics/overall/hssh1415.pdf).

X. Accurate, Reliable Individual "Dose-Reconstruction" Cannot Be Performed In the Absence Of Individual Exposure Measurements From the Worker's Jobsites Over Time.

243. Only rarely are humans exposed to chemicals in a manner that permits a quantitative determination of adverse outcomes ... Human exposures occurs most frequently in occupational settings where workers are exposed to industrial chemicals like lead or asbestos; however, even under these circumstances, it is usually difficult, if not impossible, to quantify the amount of exposure.[431]

244. This is not a new concept. For example, Marr (1964) explained that "[i]t is impossible to determine the exposure of the employee without spending hours in observation and sampling."[432] A few years later, Balzer and Cooper (1968) recognized that [r]econstruction of past exposures is impossible."[433]

245. Nicholson, et al. (1971) recognized that "correlation between disease and levels of past exposure, estimated from current measurements, is necessarily approximate. Such estimates of past exposures are, of course, fraught with obvious difficulties, However, as limited as is our knowledge of past exposure levels measured by optical microscopy, the correlation between disease and exposure is further complicated by the fact that light microscopy measures only a small fraction of the total number of fibres present. This would be acceptable if the fraction measured were a constant one. However, such is not the case."[434] Nicholson, et al. (1982) confirmed that estimates of exposure, at the cohort level, were "necessarily uncertain."[435]

246. In 2002, at an American Industrial Hygiene Association (AIHA) sponsored conference devoted to the topic of "retrospective exposure assessment," Dr. Morton Corn, CIH, explained litigation-based dose-reconstruction as follows:

> Retrospective exposure assessment had its roots in epidemiologic studies. . . . We had anchor points for exposure with the time in the same facilities, and the challenge was to fill in the missing years and the missing exposures which could be correlated with technological change usually, and that became known as the job exposure matrix and retrospective exposure assessment.
> . . .

[431] Reference Manual on Scientific Evidence, Federal Judicial Center (1994). Reference Guide on Toxicology, Goldstein B., Henifen, M. at p 187.
[432] Marr, *Asbestos Exposure During Naval Vessel Overhaul*, Am. Indust. Hygiene Assoc. J. 25: 264 (1964).
[433] Balzar and Cooper, *The Work Environment of Insulating Workers*, Am. Indust. Hygiene Assoc. J., May-June, 222 - 227 (1968)
[434] Nicholson, et al., *Direct and Indirect Occupational Exposure to Insulation Dusts in United States Shipyards*, Symposium on Safety and Health in Shipbuilding and Ship Repairing (1971).
[435] Nicholson, et al., *Occupational Exposure to Asbestos: Population at Risk and Projected Mortality – 1980 – 2003*, Am. J. Indust. Med. 3:259, 270 (1982).

> We are using data for individuals not in those places where they worked. We all know the variability of the workplace. We have heard some point estimates which I think we just cannot give. The variability is essential to the data we assume, and it is even greater when you realize that data is not for the establishments for the individuals involved.
>
> . . .
>
> The bottom line for what I am saying is, we are going to be faced in the courtroom with one of us on one side and one of us on the other side presenting these arguments one to the pleasure of the Plaintiff and one to the pleasure of the defense, and the stakes are not only individual credibility but the credibility of our field. Do not oversell these methods ... But as the practitioners, *we should recognize just how soft much of it is* and that we are in an arena to persuade. *This isn't science.* Thank you." (Applause)[436]

247. One of the presenters of the dose-reconstruction methods, Doug Fowler, agreed with Dr. Corn's comments, stating that "there is always grave uncertainty" in such dose-reconstructions and that such calculations were really "guesses, rather than estimates." [437] Another presenter, Jim Rasmuson, agreed that these methods are highly uncertain, stating "[a]ll models are wrong. . . . I think, as I indicated in the talk, recognizing the limitations of the methodology is just as important as recognizing the strengths of the methodology."[438] One attendee, Allen Rogers, pointed out the problems with the limited availability of data to make dose estimates, concluding that "[t]he data that you plug into these models and the calculations is so limited that it makes the final extrapolation near impossible to determine its accuracy." [439]

248. While it can be appropriate for epidemiologists to make exposure estimates amongst populations studied, one cannot – given the vast differences in workplace conditions – take exposure estimates from an epidemiological study and simply conclude that an individual's exposures were accurately represented by the group. While it may be tempting to generalize exposure levels based on a job title, there are wide variations in exposure levels for people in a particular industry. For example, Nicholson, et al. (1972) reported that shipboard insulators' exposures ranged from "less than 0.1 fibres/cm^3 to more than 100 fibres/cm^3."[440] Specific information about the products used, the composition of asbestos products, the

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[436] Transcript of American Industrial Hygiene Conference & Expo 2002, *Asbestos Exposure Dose Reconstruction Forum 244*, pages 76 – 80.
[437] Transcript of American Industrial Hygiene Conference & Expo 2002, *Asbestos Exposure Dose Reconstruction Forum 244*, pages 80 – 81.
[438] Transcript of American Industrial Hygiene Conference & Expo 2002, *Asbestos Exposure Dose Reconstruction Forum 244*, page 82.
[439] Transcript of American Industrial Hygiene Conference & Expo 2002, *Asbestos Exposure Dose Reconstruction Forum 244*, pages 83 – 84.
[440] Nicholson, et al., *Occupational Exposure to Asbestos: Population at Risk and Projected Mortality – 1980 – 2003*, Am. J. Indust. Med. 3:259, 270 (1982).

manner in which the products were used, and other information would be needed to make a potentially valid comparison.

249. Furthermore, one must use exposure estimates from epidemiological studies, particularly those performed before the advent of modern air monitoring techniques, with reservation because of the multiple problems inherent in using small sets of historical data collected from different instruments before standardized methodologies were widely used. As will be discussed below, data collected using old technology to estimate exposures in millions of particles per cubic foot (MPPCF) cannot be compared to exposures measured in fibers per cubic centimeter.

250. For example, three meta-analyses, Hodgson & Darnton (2000), Berman & Crump (2008) and EPA's proposed "Brattin & Crump" methodology are unreliable because they are based on speculative dose estimates that, in the case of the Canadian miner studies funded by the Quebec Asbestos Mining Association ("QAMA") and run by McGill University researchers, and South Carolina textile studies, have been proven to be inaccurate. Egilman (2009)[441] accurately summarized the problems in analyzing the dose-response curve based on inaccurate historical exposure data:

> Some experts have used meta-analyses of asbestos cohorts to claim that exposure to chrysotile asbestos must exceed some "background" threshold to cause mesothelioma.[442] Recently, an EPA-appointed Science Advisory Board (SAB) focusing on asbestos concluded that the available historical exposure data was too scant to reliably differentiate any potential potency differences by fiber type as attempted by Berman and Crump.[443] Finkelstein commented, "In essence all of the input data would consist of

[441] Egilman, *Fiber Types, Asbestos Potency, and Environmental Causation – A Peer Review of Published Work and Legal and Regulatory Scientific Testimony*, Int'l. J. Occup. Environ. Health 15:2 202-227 (2009).

[442] Berman, et al., *A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type*. Critical Reviews Toxicol. 38 Suppl 1:49-73 (2008); Berman, et al., *Update of potency factors for asbestos related lung cancer and mesothelioma*, Critical Reviews Toxicol. 38 Suppl 1:1-47 (2008); Kane A. (Chair, SAB Asbestos Committee). Letter to Johnson S. (EPA Administrator), *SAB Consultation on EPA's Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos Cancer Potency Factors for Inhalation Exposure to Asbestos*, EPA-SAB-09-004 (2008).

[443] Berman, et al., *A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type*. Critical Reviews Toxicol. 38 Suppl 1:49-73 (2008); Kane A. (Chair, SAB Asbestos Committee). Letter to Johnson S. (EPA Administrator), *SAB Consultation on EPA's Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos Cancer Potency Factors for Inhalation Exposure to Asbestos*, EPA-SAB-09-004 (2008).

guesses and the output of the model would not be credible."[444] As the EPA's SAB concluded, impinger data (which measured total particles and did not distinguish dust from fibers) "cannot" be "used to generate PCM comparisons."[445] There is some evidence that the asbestos-mesothelioma relationship may follow more than one dose-response curve. There are many case reports of mesothelioma in individuals with brief or "low dose" environmental or home exposure (see Table 1).[446] On the other hand, "only" 10% of even the most heavily exposed cohorts develop mesothelioma.[447] Clearly, genetic factors and other exposures interact to produce mesothelioma in some, but not all, people with similar exposures.

Hodgson and Darnton attempted to evaluate the relative potency of asbestos types using some of the same studies used by Berman and Crump.[448] Rogers and Major, referring to Australian exposure data used by Hodgson and Darnton, noted that, "the[se] exposure values . . . should be recognized as 'guesstimates', made by people who have not been trained in occupational hygiene and who have no experience in asbestos dust monitoring."[449] In addition to using the 'guesstimates' of the Australian exposures, there was no exposure data for other crocidolite cohorts in their study, and the authors simply assumed an exposure level. Hodgson and Darnton then compared the crocidolite exposure guesstimates to the inaccurate exposure data from Canadian miner and miller cohorts. These McGill University studies funded by the Quebec Asbestos Mining Association found a slight inverse relationship between the particle

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[444] Kane A. (Chair, SAB Asbestos Committee). Letter to Johnson S. (EPA Administrator), *SAB Consultation on EPA's Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos Cancer Potency Factors for Inhalation Exposure to Asbestos*, EPA-SAB-09-004 (2008).

[445] Kane A. (Chair, SAB Asbestos Committee). Letter to Johnson S. (EPA Administrator), *SAB Consultation on EPA's Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos Cancer Potency Factors for Inhalation Exposure to Asbestos*, EPA-SAB-09-004 (2008).

[446] Miller, *Mesothelioma in household members of asbestos exposed workers: 32 United States cases since 1990*, Am. J. Indust. Med. 47:458-62 (2005); Greenberg, et al., *Mesothelioma register 1967-68*, Brit. J. Indust. Med. 31:91-104 (1974).

[447] Selikoff, et al., *Asbestos-associated deaths among insulation workers in the United States and Canada, 1967-1987*, Ann. N.Y. Acad. Sci. 643:1-14 (1991).

[448] Hodgson, et al., *The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure*, Ann. Occup. Hygiene 44:565-601 (2000).

[449] Rogers A, Major G., *The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure: The Wittenoom data.* Ann. Occup. Hygiene 46:127-8; author reply 8-9 (2002).

counts they used and fiber counts.[450] Their dose estimates were slightly better than random guesses.[451] McGill researchers were aware of this problem and ignored it. In 1969, during a discussion on asbestos counting methods at an international conference on pneumoconiosis in Johannesburg, South Africa, McGill's Corbett McDonald asked, "Can an inaccurate instrument like the midget impinger (MI), give an accurate result?"[452] He was informed that it could not. Just as a stopped watch, which is correct twice a day, should not be used to tell time, unreliable exposure estimates should not be used to devise inevitably unreliable estimates of relative fiber potency. Hodgson and Darnton's comparison of dose-response relationships between these two large cohorts is as reliable as the square of the "guesstimate." Hodgson and Darnton were aware of these problems as well, and wrote, "Certainly these estimates are much less soundly based than one would wish." Unfortunately, they pressed on stating, "Some view does however need to be taken . . . ."[453] A wrong view based on inadequate data can be worse than no view at all; it can and has encouraged the continued use of chrysotile and been used to persuade juries that chrysotile products are harmless. Another weakness of the Hodgson-Darnton review is that it dealt with 17 cohorts representing special industries. It did not include any case-referent studies for end-use exposures, which represent the most common pattern for asbestos associated mesotheliomas.[454] Despite these failings and contrary to the positions taken by Price and Ware and Teta et al., Hodgson and Darnton (whose model inherently adopts a no-threshold assumption) rely on these "guestimates" to calculate relative potency for crocidolite, amosite, and chrysotile for mesothelioma induction of 500:100:1.[455] Leigh and Robinson demonstrated the arbitrariness of these estimates.[456] They recalculated them and accounted for clearance of amphibole and found potency ratios to be 26:14:1 which represents a twenty fold

[450] Gibbs, et al., *Dust-fiber relationships in the Quebec chrysotile industry. Arch Environ Health,* 28:69-71 (1974).

[451] Gibbs, et al., *Dust-fiber relationships in the Quebec chrysotile industry. Arch Environ Health,* 28:69-71 (1974).

[452] Shapiro HA. Pneumoconiosis; Proceedings of the International Conference, Johannesburg, 1969. Cape Town, New York,: Oxford University Press; 1970.

[453] Hodgson J, Darnton A. Letter. Annals Occup. Hygiene 45:336-8 (2001).

[454] Hodgson, et al., *The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure,* Ann. Occup. Hygiene 44:565-601 (2000).

[455] Hodgson, et al., *The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure,* Ann. Occup. Hygiene 44:565-601 (2000).

[456] Leigh J, Robinson B., *The history of mesothelioma in Australia 1945-2001,* In: Robinson B, Chahinian A, eds. Mesothelioma. London: Martin Dunitz; 55-86 (2000).

difference for crocidolite.[457] An often-cited set of potency ratios in the literature is 30:15:1.[458] Most other cohorts are too small to evaluate the effects of even moderate levels of exposure. Even fiber PCM counts may be misleading.[459] Hein, et al. found that "Current PCM-based methods may underestimate asbestos exposures to the thinnest fibers, which were the strongest predictor of lung cancer or asbestosis mortality."[460] It is possible that amphiboles are more potent than amphibole-contaminated chrysotile, but existing epidemiology cannot support or rebut this theory no matter how often it is repeated. Peto et al. titled their recent discussion of the issue of chrysotile causation "Speculations on the Contribution of Chrysotile," and with respect to ecological epidemiology, speculation it is.[461]

251. Egilman, et al. (2003) explained many of the problems with using historical exposure data as follows:

> Gibbs and Hui (1971) used available dose measurements from 1949 to 1966, which were measures of "total" particles collected by midget impinger.[462] This method cannot distinguish fibers from other dust particles, such as silica and other "non-toxic" dusts.[463] Only *fibers*, which may or may not be captured in the total particle measurements, cause disease. In reality, it is difficult to make accurate estimates of the actual exposure of the Canadian miners and millers during that period. However, the estimates that have been made indicate that the miners were, in all likelihood, exposed to fewer fibers than were the South Carolina textile workers--rather than the other way around, as the QAMA-McGill researchers suggest. The dose estimates from the QAMA-McGill mine studies are wholly inaccurate. Based on the comparative mine/textile risk ratio, it seems clear that they have systematically overestimated the actual exposures.

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[457] Leigh J, Robinson B., *The history of mesothelioma in Australia 1945-2001*, In: Robinson B, Chahinian A, eds. Mesothelioma. London: Martin Dunitz; 55-86 (2000).
[458] Henderson DW. Supplementary Report, On Causation, for Mr. Frank Gregory Lansley. Adelaide, South Australia; 2006 February 27.
[459] Costas, et al., *Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer*, New England J. Med. 339:1000; author reply 1-2 (1998).
[460] Hein, et al., *Follow-up study of chrysotile textile workers: cohort mortality and exposure response*, Occup. Environ. Med. 64:616-25 (2007).
[461] Peto, et al., *Occupational, domestic and environmental mesothelioma risks in Britain: A case-control study*, Health and Safety Executive Research Report No.: 696 (2009).
[462] Gibbs, *The organic content of Canadian chrysotile*, Am. Indust. Hygiene Assoc. J, 32:519-528 (1971).
[463] Egilman, et al., *The asbestos TLV: early evidence of inadequacy*, Am. J. Indust. Med. 30:369-370 (1996).

As early as 1951, QAMA researchers realized that the accurate calculation of dose estimates for Quebec miners was impossible. While the problem of dose conversion exists in all studies that are based on historical particle count data, this problem was exacerbated in the mine studies due to the large number of locations and jobs involved. As Vorwald, a consultant to the QAMA, wrote to Cartier, the director of the QAMA industrial disease clinic in Thetford mines:

> Last week, while in Washington, I had the opportunity to discuss our program concerning the epidemiology of pulmonary cancer in subjects exposed to asbestos dust and to present the problem which you posed regarding job classification. I agree with your views. Certainly it is an *impossible* task to tabulate the various jobs on comparable scientific data, since such data does not exist. Therefore the code suggested by both you and Ken [Smith, medical director of Johns-Manville] should be used [Emphasis added].[464]

Cartier later served as a consultant to the QAMA-McGill researchers as well.

What was clearly "impossible" in 1951 became the dose reconstruction of 1971.[465] Dr. McDonald was aware of this problem by no later than April 23, 1969. During the discussion period following his chairing of a session on asbestos measurement techniques, which was highly critical of the midget impinger method, he asked, "Can an *inaccurate* instrument like the midget impinger (MI), give an accurate result?" Rendall responded, "I have not had enough experience with the MI but it is the wrong instrument on which to base standards" [466] Despite the MI's drawbacks, the QAMA researchers have continued to use MI data to estimate exposures through the 1990's, a quarter of a century after the mines converted to fiber counting. Dose-response relationships based on a completely inaccurate, but *apparently* large set of exposure data, provide a false sense of statistical security to these results.

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[464] Vorwald A., Letter to Paul Cartier (July 19, 1951).

[465] Gibbs, *The organic content of Canadian chrysotile,* Am. Indust. Hygiene Assoc. J, 32:519-528 (1971).

[466] Shapiro, H. A. 1970. 1969 Pneumoconiosis: Proceedings of the international conference, Johannesburg. Cape Town, S. Africa, Oxford University Press.

252. Egilman, et al. (2003)[467] explained several of the problems in relying on exposure data from the Canadian miner/miller studies performed by the Quebec Asbestos Mining Association ("QAMA"):

> Particle-fiber Conversion Issues
>
> QAMA has long known both the impossibility of estimating asbestos fiber exposures for every job class, and the irrelevance of particle counts in determining toxicity. In 1953, the QAMA executive board meeting minutes noted, "The industrial hygiene surveys that have been made in the past, and in which only dust particles were measured, are practically without value".[468]
>
> QAMA waited twenty years to change to membrane filter measurement after obtaining this information. The QAMA-McGill researchers were left with only particle counts, but if the particle counts could not be correlated with fiber counts or were inversely related to fiber levels, then the particle counts were useless as indices of exposure to determine asbestos toxicity. Gibbs and Lachance tested this hypothesis by performing 87 matched pairs of tests utilizing light microscopy and membrane filters to count fibers and comparing these with midget impinge particle counts.[469] They found that, overall, the relationship between particle counts and fiber counts were thirteen percent better than *random* number generation. Incredibly, for low fiber count exposures, the particle counts were *inversely* related to fiber exposures. This inverse relationship occurred in more than one third of the samples (31/87). Therefore, for at least one third of the particle counts, it was determined that the higher the count, the lower the workers' exposure to asbestos. Gibbs and Lachance note:
>
> For thirty-one samples with less than one fiber per field, the linear correlation was very close to zero, - 0.03, and the correlation of log rhythmically transformed data was 0.25. However, these correlations suggest that for all mines the regression lines are unsatisfactory for the prediction of fiber counts from impinger counts, as the improvement and prediction for the best correlation, 0.45, is only 13% better than a conversion obtained at random.

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[467] Egilman, et al., *Exposing the "Myth" of ABC, "Anything But Chrysotile": A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies*, Am. J. Indust. Med. 44: 540 – 557 (2003).

[468] Jackson, J. H. 1953. June 10. Asbestos Textile Institute, Hygiene committee meeting minutes.

[469] Gibbs, et al., *Dust-fiber relationships in the Quebec chrysotile industry. Arch Environ Health*, 28:69-71 (1974).

> Thus the conversion of dust disease relationships to fiber disease relationships does not seem possible.[470]

Gibbs and Lachance acknowledge the poor correlation of side-by-side midget impinger samples and recommend

> that safety standards, at least in this industry, should continue to be based on dust counts for which there is considerable epidemiologic support rather on fiber counts, for which there is no direct evidence.[471]

They concluded, "The conversion of dust disease relationships for the Quebec Mining and Milling Industry to fiber disease relationships *does not seem possible* at the present time." . . .

The lack of scientific validity of these dose estimates did not stop the QAMA-McGill research team. They selected a single conversion factor for all processes and henceforth all subsequent publications have relied on this single value (although minor adjustments to the value have been made from time to time). McDonald states in a 1973 IARC conference publication that dust-sampling methods, in addition to unreliable particle-fiber conversions, produced data too variable to be considered a reliable basis for estimating exposure.[472]

253. Egilman, et al. (2003)[473] also discusses the effects of using a single, inaccurate conversion factor to translate particle counts to fiber counts:

> *Conversion Factors*
>
> First Gibbs and Lachance (1974)[474] and later Liddell, et al. (1984, 1998)[475] evaluated the merits of converting particle counts to fiber

[470] Gibbs, et al., *Dust-fiber relationships in the Quebec chrysotile industry. Arch Environ Health*, 28:69-71 (1974).
[471] Gibbs, et al., *Dust-fiber relationships in the Quebec chrysotile industry. Arch Environ Health*, 28:69-71 (1974).
[472] McDonald, *Asbestos in chrysotile mines and mills*, In: Bogosvki P, Gilson JC, Timbrell V, Wagner JC. Biological Effects of Asbestos. Lyon: International Agency for Research on Cancer, IARC Sci Pub No. 8. (1973).
[473] Egilman, et al., *Exposing the "Myth" of ABC, "Anything But Chrysotile": A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies*, Am. J. Indust. Med. 44: 540 – 557 (2003).
[474] Gibbs, et al., *Dust-fiber relationships in the Quebec chrysotile industry. Arch Environ Health*, 28:69-71 (1974).
[475] Liddell, et al., *Fibre exposure and mortality from pneumoconiosis, respiratory and abdominal malignancies in chrysotile production in Quebec, 1926-75*, Annals of the Academy of Medicine, Singapore 13:340-344 (1984); Liddell, et al., *Dust exposure and lung cancer in Quebec chrysotile miners and millers*, Ann. Occup. Hygiene 42:7-20 (1998).

> counts. In addition to their realization that worker interviews indicated that the original dose estimates were even less accurate than previously assumed, they again recognized that particle/fiber ratios were "virtually independent of the level of exposure."[476] Furthermore, it was clear that if a conversion factor were to be used, it needed to be specific for each job category.[477] Disregarding their own findings, they used *a single particle/fiber ratio standard for all years in all job categories.* They based this standard on the worker histories, which they then proceeded to ignore in calculating the actual particle counts because they postulated that the histories would introduce a "systemic bias" into their analysis.

Liddell (1998) again recognized the inadequacy of their dose estimates:

> *the classification of jobs by dust category would not be a reliable classification by fibre count.*[478]

They also understood why the dose estimates were so inaccurate. Fiber/dust ratios necessarily differed by orders of magnitude for different types of work. In addition, fiber/dust ratios differed by orders of magnitude for the same work process at different points in time. Liddell et al. noted that, "The two important reports by Gibbs and Lachance 1972; and Gibbs and Lachance 1974, give some indication of the inherent complexity; a simple example is that work on the tailings dump in 1968 was extremely dusty but, as most of the fibre would have been extracted the fibre:dust ratio must have been quite low."[479] The exposure data was so inaccurate that, "taken at face value," exposures even appeared *protective* for the workers. In other words, un-manipulated, the exposure data indicated that chrysotile exposure actually *prevented* workers from developing pneumoconiosis, lung cancer or mesothelioma.[480] Their original findings would be plausible if one considered an alternative hypothesis in which workers who had the highest exposures died from nonmalignant disease before the latent period for the induction of cancer had been attained. Instead, since they believed an inverse exposure relationship was ludicrous, they manipulated the exposure estimates until the dose-response curve fit their *a priori* understanding of the proper form for the dose-response relationship. The researchers discarded all of the exposure levels that were inversely

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[476] Liddell, et al., *Fibre exposure and mortality from pneumoconiosis, respiratory and abdominal malignancies in chrysotile production in Quebec, 1926-75*, Annals of the Academy of Medicine, Singapore 13:340-344 (1984)

[477] Gibbs, *The assessment of exposure in terms of fibres,* Ann. Occup. Hygiene 38:477-10 (1994).

[478] Liddell, et al., *Dust exposure and lung cancer in Quebec chrysotile miners and millers*, Ann. Occup. Hygiene 42:7-20 (1998).

[479] Liddell, et al., *Dust exposure and lung cancer in Quebec chrysotile miners and millers*, Ann. Occup. Hygiene 42:7-20 (1998).

[480] Liddell, et al., *Dust exposure and lung cancer in Quebec chrysotile miners and millers*, Ann. Occup. Hygiene 42:7-20 (1998).

related to disease. They described this manipulation in an appendix titled "Elimination of negative regression coefficients," and proceeded to "revise" the exposure data to make it appear credible and to create a result that would allow them to argue that chrysotile exposure was "innocuous."[481]

> Apparently, the QAMA-McGill researchers understood what effects were "significant" before they began the studies and fitted and manipulated the data in order to make it comport to these pre-determined effects.

Historical Efforts to Quantify Exposure Are Unreliable Because Of Well-Recognized Limitations with Light Microscopy.

254. The measurement of fibers by light microscopy and any conversion from particle to fiber count rests on the assumption that the visible fibers measured constitute some fraction of the total number of fibers present in the air. This is because most fibers present in the air are not visible under light microscopy. In addition, by convention, light microscopy does not measure fibers that were less than five microns in length.[482] Therefore, for QAMA exposure estimates to be considered valid, two requirements must be met. First, there must have been a consistent proportional relationship between visible fibers and total fibers. This also necessitates a consistent relationship between visible fibers and fibers less than five microns in length in various processes (i.e., mining, milling, maintenance). These relationships needed to be maintained over a 60-year time period during which many processes changed dramatically. There is no evidence that this was the case.

255. Asbestos fiber-counting is a "tip of the iceberg" phenomenon because fibers are counted by light microscopy. Since chrysotile fibers split longitudinally, some of the fibers are too narrow to be seen and are not counted. The first steps of the textile process are specifically designed to split fiber bundles; therefore, textile exposures involve a higher percentage of

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[481] Liddell, et al. (1998) wrote:

> In all the conditional regression analyses of the full model, i.e. with 13 exposure measures, there was at least one negative regression coefficient, which taken at face value would imply a protective effect of exposure. Years in the highest relevant dust category were pooled with those in the adjacent category and the analysis was repeated. This process was iterated until either all coefficients had become positive, when it was terminated, or until the only negative coefficient was for category 1; in that circumstance, category 1 was eliminated from the model, which was the equivalent of setting the coefficient to zero and the odds ratio to unity . . . Admittedly, *there was a degree of arbitrariness in some of the pooling carried* out but every effort was made to retain any 'significant effects."

Egilman, et al. (2003), Note 12 citing Liddell, et al., *Dust exposure and lung cancer in Quebec chrysotile miners and millers*, Ann. Occup. Hygiene 42:7-20 (1998) (emphasis added).

[482] Sebastien, et al., *Respiratory cancer in chrysotile textile and mining industries: exposure inferences from lung analysis*. British Journal of Industrial Medicine 46:180-187 (1989).

thin (invisible) fibers than mill or mine exposures.[483] As a result of increased fiber splitting in the textile process, each fiber counted represents many more uncounted fibers than those in the mining and milling process. Nicolson reviewed this issue as well:

> Moreover, as with length distribution, diameter distribution varies with activity and fiber types. As a result, the fraction of fibers longer than 5 um visible by light microscopy varies from about 22 percent in chrysotile and crocidolite mining and amosite/chrysotile insulation manufacturing to 53 percent in amosite mining. Intermediate values of 40 percent are measured in chrysotile brake lining manufacturing and 33 percent in amosite mill operations. Thus, even perfect measurement of workplace air, with accurate enumeration of fibers according to currently accepted methods, would be expected to lead to different exposure-response relationships for any specific asbestos disease when different work environments are studied."[484]

256. Fiber length is not the only consideration relevant to fiber counting. Nicholson notes that as many as half of the fibers may have been missed using optical microscopy that cannot measure fibers of the smallest diameters:

> Using electron microscopy, Rendall and Skikne (1980) measured the percentage of fibers with a diameter less than 0.4 um (the approximate limit of resolution of an optical microscope) in various asbestos dust samples. In general, they found that more than 50 percent of the 5 um or longer fibers are less than 0.4 um in diameter and, thus, are not visible using a standard phase contrast optical microscope.[485]

[483] Dement, et al., *Estimates of pulmonary and gastrointestinal deposition for occupational fiber exposures,* DHEW Publication No. (NIOSH) 79-135 (1979).
[484] Nicholson WJ., Airborne Asbestos Health Assessment Update. EPA/600/8-84/003F. 42-43 (1986).
[485] Nicholson WJ., Airborne Asbestos Health Assessment Update. EPA/600/8-84/003F. 42-43 (1986).

XI. Qualitative Analysis of Lifetime Cumulative Exposure to Asbestos is a Generally Accepted Scientific Method for Ascribing Causation.

257. Evaluation of lifetime cumulative exposure, from an occupational history, is the best method for ascribing causation of asbestos-related disease. In December 2015, I joined sixty-six (66) other scientists in explaining the generally accepted, scientific approach to ascribing mesothelioma to asbestos exposure:

> The analysis of cumulative asbestos exposure in order to ascribe causation has been performed by scientists in hundreds of peer-reviewed, published epidemiological studies, case series and case reports. And these peer-reviewed articles reinforce the scientific consensus that each occupational and para-occupational exposure to asbestos contributes to the cumulative lifetime asbestos exposure and increases a person's risk of developing mesothelioma.
>
> The general scientific acceptance of this fundamental, reliable scientific principle was reiterated by the recent statement of the Collegium Ramazzini, an internationally recognized "scientific society that examines critical issues in occupational and environmental medicine with a view towards action to prevent disease and promote health." In response to an asbestos-industry expert's hypothesis that only the earliest exposures contribute to the risk of mesothelioma the Collegium Ramazzini concluded that proper analysis of the body of scientific evidence confirms that "the risk of malignant mesothelioma is related to cumulative exposure to asbestos in which all exposures – early as well as late – contribute to the totality of risk." Collegium Ramazzini, *Comments On The Causation Of Malignant Mesothelioma: Rebutting The False Concept That Recent Exposures To Asbestos Do Not Contribute To Causation Of Mesothelioma*, http://www.collegiumramazzini.org/ download/Causation _Malignant_ Mesothelioma_Comments(2015).pdf (accessed November 17, 2015).
>
> It is the generally accepted scientific consensus that the cumulative effect of occupational and para-occupational exposures is the ultimate cause of mesothelioma in individuals with mesothelioma and that all such exposures increase the risk of mesothelioma. We reject the suggestion that these established scientific conclusions are equivalent to the opinion that a single fiber of asbestos (e.g. "any exposure no matter how small") can cause mesothelioma.[486]

[486] 67 Scientists at pages 1 - 2.

258. A recent French case-control study, Lacourt et al., (2014) found significant excess cancer at levels lower than the amount permitted under the current OSHA standards.[487] Lacourt found elevated risk as follows: ≤ 0.1 f/cc/years, Odds Ratio (OR) 4.0 (99% Confidence interval (CI) 1.9 – 8.3).

259. Rödelsperger et al. (2001) concluded there was a distinct dose-response relationship, even at extremely low levels of asbestos exposure, with exposures from >0 to <0.15 f/cc-yrs showing a significantly increased risk of mesothelioma.[488]

260. A recent Dutch cohort study, Offermans et al. (2014) found excess risk as follows: ≤ 0.2 f/cc/years, Hazard Ratio (HR) 2.69 (95% CI 1.60 - 4.53).[489]

261. A large case-control study by Iwatsubo et al., found an excess of pleural mesothelioma in the lowest exposure group with an estimated total exposure between 0.001 and 0.49 f/ml-yrs.[490]

262. A recent Danish article, Petersen et al. (2015), linked mesothelioma cases to asbestos exposure because the exposures for the cases were greater than the levels found to increase risk in Rödelsperger et al. (2001).[491] This same approach was also recently advocated by Markowitz (2015) and by the 58 Scientists Brief in support of my testimony:

> Moreover, human dose-response studies show significant increased risk at very low levels of exposure. These studies have found increased risk of 2.69 to 7.9-fold based on low-level exposure. *See e.g.* 2014 Lacourt et al., ≤ 0.1 f/cc/years, Odds Ratio (OR) 4.0 (99% Confidence interval (CI) 1.9 – 8.3); 2001 Rödelsperger et al., ≤ 0.15 f/cc/years, OR 7.9 (95% CI 2.1-30.0); 2014 Offermans et al., ≤0.2 f/cc/years, Hazard Ratio (HR) 2.69 (95% CI 1.60 – 4.53) and the 1998 Iwatsubo et al., 0.5 - 0.99 f/cc/years, OR 4.0 (95% CI 2.0 –8.8).

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[487] Lacourt et al., *Occupational and Non-Occupational Attributable Risk of Asbestos Exposure for Malignant Pleural Mesothelioma*, Thorax 1 (2014).

[488] Rödelsperger et al., *Asbestos and man-made vitreous fibers as risk factors for diffuse malignant mesothelioma: Results from a German hospital-based case-control study.* Am. J. Ind. Med. 39:262-275 (2001).

[489] Offermans et al., *Occupational asbestos exposure and risk of pleural mesothelioma, lung cancer, and laryngeal cancer in the prospective Netherlands cohort study*, J. Occup. Environ. Med. 56(1):6–19 (2014)

[490] Iwatsubo et al., *Pleural mesothelioma: Dose response relation at low levels of asbestos exposure in a French population-based case-control study.* Am. J. of Epidemiol. 148:133-142 (1998).

[491] Petersen et al., *Non-occupational pleural mesothelioma*, Ugeskr Læger 177:V09140480 (2015) (translated from Danish).

> And epidemiological studies provide additional support for a weight of the evidence conclusion that asbestos from brakes can and does cause mesothelioma. There are numerous epidemiological studies and case reports of mesothelioma in persons whose primary exposure to asbestos is from asbestos-containing brake materials. From an industrial hygiene perspective, it does not take long, at levels known to exist in garage environments without asbestos dust control, for a person to receive appreciable exposures.[492]

Since the 58 Scientists weighed in, yet another cohort study has found highly elevated risk of mesothelioma at low levels of cumulative exposure. Ferrante et al. (2015)[493] reported increased risk of mesothelioma at all levels of exposure studied: Less than or equal to 0.1 to less than 1.0 fiber/cc/years, Odds Ratio (OR) 4.4 (95% CI 1.7-11.3).

263. Ferrante et al. (2015) observed "[o]ur study clearly shows a relationship between PMM risk and cumulative exposure also after non-occupational exposures. This is a novel result that confirms with quantitative data our previous results from studies in the area[494] and underlines the need to carefully control all sources of asbestos exposure."

264. Stayner explained the significance of Ferrante et al. (2015) as follows:

> This study provides strong evidence of an association between pleural mesothelioma and non-occupational exposures to asbestos. An approximately twofold increase in risk was observed for having lived with a family member who worked in the Eternit asbestos cement plant (OR=2.4, 95% CI1.3 to 4.4), or having been exposed from domestic or environmental sources (OR=2.0, 95% CI 1.2 to 3.2).
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> . . .
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> What is new is that this study considers the exposure–response relationship between pleural mesothelioma and cumulative exposure to asbestos from domestic, environmental and occupational. Not surprisingly, the study found strong evidence of an exposure–response relationship between higher cumulative exposure to asbestos from all sources and increased risk of pleural mesothelioma. Of particular concern is their finding of an

[492] 58 Scientists pp. 17-18 (footnotes omitted).
[493] Ferrante et al., *Cancer Mortality and Incidence of Mesothelioma in a Cohort of Wives of Asbestos Workers in Casale Monferrato, Italy*, Environ. Health Perspectives 115:10; 1401-1405 (2015).
[494] Citing Maule et al., *Modeling mesothelioma risk associated with environmental asbestos exposure,* Environ Health Perspect 115:1066-71 (2007).

approximately fourfold (OR=4.4, 95% CI 1.7 to 11.3) increased risk of pleural mesothelioma at relatively low levels of asbestos.[495]

265. In 2000, Bourdès et al. reviewed the literature and performed a meta-analysis of the risk of pleural mesothelioma from environmental exposure to asbestos.[496] Bourdès et al. identified eight relevant studies on the risk of pleural mesothelioma from household or neighborhood exposures to asbestos. These studies did not include the case-control studies outlined below. These authors found that the relative risks of pleural mesothelioma for household exposure ranged between 4.0 and 23.7. They also found a summary risk estimate of 8.1 (95% CI, 5.3–12). For neighborhood exposures, the relative risks reported ranged between 5.1 and 9.3 and the summary estimate was 7.0 (95% CI, 4.7–11). This analysis appears to be in agreement with the studies by Magnani et al.[497] and Rödelsperger et al. (see above). Bourdès et al. commented that their data were insufficient to estimate the magnitude of excess risk at the levels of environmental exposure commonly experienced by the general population in industrial countries (in other words, from the general environment).

266. Pan et al. (2005) performed a population-based study on the distribution of mesothelioma in California.[498] After attempted allowance for occupational exposures, these researchers reported an apparent direct correlation between the risk of mesothelioma and proximity of residence according to the distribution of asbestos-containing rocks in the general environment (mainly chrysotile, with some other forms of asbestos including tremolite). These authors found about a 6% reduction in the odds of mesothelioma for residence for every 10 km further away from the asbestos-containing rocks.

267. In the article by Hodgson et al. (2000), there is extrapolation information with regards to crocidolite that at cumulative exposure levels of only 0.01 f/ml-yrs, there are 20 deaths per 100,000 exposed with the highest arguable estimate 100 and the lowest 2 cases. Even at the lowest estimate of 2 cases per 100,000 exposed, this would be in excess of 20 times the figure commonly used as an *assumed* level of background or spontaneous mesothelioma development, which is approximately 1-2 cases per million people per year.[499] With respect

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[495] Stayner, *Para-occupational exposures to asbestos: lessons learned from Casale Monferrato, Italy* Occup Environ Med Published Online First: 10 November 2015 doi:10.1136/oemed-2015-103233.

[496] Bourdès et al., *Environmental exposure to asbestos and risk of pleural mesothelioma: review and meta-analysis*. Eur. J. Epidemiol. 16:411–417 (2000).

[497] Magnani et al., *Multicentric study on malignant pleural mesothelioma and non-occupational exposure to asbestos*. Br. J. Cancer. 83: 104–111 (2000).

[498] Pan et al., *Residential proximity to naturally occurring asbestos and mesothelioma in California*. Am. J. Respir. Crit. Care Med. 172:1019– 1025 (2005).

[499] One should note that this is an assumed level used to allow a standard for comparison. While it is commonly used as a comparison point, as discussed above, several significant papers have failed to find evidence to support any measurable "background" incidence of mesothelioma. Strauchen, *Rarity of Malignant Mesothelioma Prior to the Widespread Commercial Introduction of Asbestos: The Mount Sinai Autopsy Experience 1883 – 1910*, Am. J. Industr. Med. 1-3 (2011);

to amosite, at a level of cumulative exposure of 0.01 f/ml-yrs, the estimate is 3 deaths per 100,000 exposed, with the highest arguable estimate 20 and the lowest insignificant. For chrysotile, the risk for development of mesothelioma at 0.01 f/ml-yrs was stated to be probably insignificant, although the highest arguable estimate was 1 death per 100,000 exposed, which would still be 10 times that of the *assumed* background rate of 1 case per 1,000,000. The authors stated: "Taking this evidence together, we do not believe there is a good case for assuming any threshold for mesothelioma risk."[500]

268. The consensus is that medical science has yet to identify a threshold or minimum amount of asbestos exposure required to cause mesothelioma.[501] Frontario et al. (2015) summarized the mainstream view:

> Cumulative asbestos exposure, either directly or indirectly, remains the leading cause of mesothelioma. It has been previously determined that cumulative asbestos exposure leads to a proportional increase in mesothelioma risk. Mesothelioma can result from non-industrial environmental contact with asbestos fibers, and para-occupational exposure occurs; for example, women who have laundered their husband's work-related clothing. Cumulative asbestos exposure, either directly or para-occupational, remains the most common factor related to the development of mesothelioma.[502]

269. An update by Hodgson and Darnton in 2009 concerning mesothelioma risk from chrysotile asbestos stated that, when information from a number of recent, well-conducted studies was incorporated into their mathematical model, the risk of mesothelioma caused by chrysotile derived from these data increased by a factor of 10 over the estimate from their earlier meta-analysis. The authors stated these new results strengthened the case for the proposition that the per fibre risk of mesothelioma from chrysotile textile plants was greater than it was in the mines. Whether this applied to other settings of chrysotile was stated to not be clear.[503] What is abundantly clear is that these estimates are susceptible to wide swings in magnitude depending upon the data included. Significantly, Hodgson & Darnton's recalculation likely

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Mark et al., *Absence of Evidence for a Significant Background Incidence of Diffuse Malignant Mesothelioma Apart from Asbestos Exposure.* Ann. N.Y. Acad. Sci. 643:196 – 204 (1991).

[500] Hodgson et al., *The quantitative risk of mesothelioma and lung cancer in relation to asbestos exposure.* Ann. Occup. Hyg.; 44:565-601; specifically, Table 11, page 585 (2000).

[501] Wiggins, Statement on malignant mesothelioma in the United Kingdom. 56 Thorax 250,252 (2001) ("[t]here is no evidence for a threshold dose of asbestos below which there is no risk" of mesothelioma); World Health Organization, Environmental Health Criteria 203: Chrysotile Asbestos, 144 (1998) ("No threshold has been identified for carcinogenic risks."). In fact, attempts to deduce such a threshold for mesothelioma have been dismissed as "logical nonsense." *See* Hodgson et al., *The Qualitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure.* 44 Ann. Occup. Hyg. 565, 583 (2000).

[502] Frontario et al., *Primary Peritoneal Mesothelioma Resulting in Small Bowel Obstruction: A Case Report and Review of Literature*, Am J Case Rep 16:496-500 (2015).

[503] Hodgson et al., *Mesothelioma risk from chrysotile.* Occup. Environ. Med. (2009).

still underestimates the potency of chrysotile because they failed to include numerous chrysotile-induced mesotheliomas from the Connecticut chrysotile cohorts.[504] Inclusion of these cases of chrysotile-induced mesotheliomas in Hodgson & Darnton's model would obviously diminish the magnitude of potency differences substantially.

270. Markowitz (2015)[505] provides an excellent discussion of the problems with the reliability of relative potency estimates based on mathematical models:

> [R]ecent studies[506] have raised doubts and led to revisions in the estimates developed by Hodgson and Darnton. A new study of mortality at three North Carolina textile plants[507] caused Hodgson and Darnton to revise their fiber potency ratios for malignant mesothelioma downwards sevenfold to 70:14:1 for crocidolite:amosite:chrysotile.[508] Additional studies have updated the mortality experience of two of the chrysotile asbestos cohorts originally studied by Hodgson and Darnton but have not yet been addressed in a revised meta-analysis.[509] Both of these studies have described additional mesothelioma deaths in their respective cohorts. These include (1) an update of the chrysotile miner cohort in Balangero, Italy, where[510] workers active in mine operations and an additional[511] other exposed individuals have developed malignant mesothelioma,[512] and (2) an update of mesothelioma

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[504] Finkelstein et al., *Malignant Mesothelioma among employees of a Connecticut factory that manufactured friction materials using chrysotile asbestos*. Ann. Occup. Hyg. 54(6):692–696 (2010) (reporting on several mesothelioma cases originally published by Egilman).

[505] Markowitz, *Asbestos-Related Lung Cancer and Malignant Mesothelioma of the Pleura: Selected Current Issues*, Semin. Respir. Care Med. 36:334-346 (2015) (citation in original).

[506] Hodgson et al., *Mesothelioma risk from chrysotile*, Occup. Environ. Med. 67(6):432 (2009); Loomis et al., *Lung cancer mortality and fiber exposures among North Carolina asbestos textile workers*. Occup. Environ. Med. 66:535–542 (2009).

[507] Loomis et al., *Lung cancer mortality and fiber exposures among North Carolina asbestos textile workers*. Occup. Environ. Med. 66:535–542 (2009).

[508] Hodgson et al., *Mesothelioma risk from chrysotile*, Occup. Environ. Med. 67(6):432 (2009).

[509] Mirabelli et al., *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy*. Occup. Environ. Med. 65:815–819 (2008); Finkelstein et al., *Malignant Mesothelioma among employees of a Connecticut factory that manufactured friction materials using chrysotile asbestos*. Ann. Occup. Hyg. 54(6):692–696 (2010).

[510] Marinaccio et al., *Incidence of extrapleural malignant mesothelioma and asbestos exposure, from the Italian national register*, Occup. Environ. Med. 67(11): 760–765 (2010).

[511] Leigh et al., *Malignant mesothelioma in Australia, 1945- 2002*, Int. J. Occup. Environ. Health 9(3):206–217 (2003).

[512] Mirabelli et al., *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy*. Occup. Environ. Med. 65:815–819 (2008).

cases in the Connecticut friction product plant, where additional cases of malignant mesothelioma have been documented.[513]

> Two outstanding considerations—one scientific and the other practical—color the reliability and significance of putative differences in fiber-specific potency in causing malignant mesothelioma. First, the difficulties in ascertaining mesothelioma deaths over the relevant decades and the highly variable quality in exposure assessments in different study settings temper the validity of study results to form the basis of risk estimates. Many of the relevant individual study exposure assessments have been found to be lacking for numerous reasons: (1) restriction of fibers counted by phase contrast microscopy to fibers >5 μ min length and >0.25 μm in width, thereby failing to count the majority of chrysotile fibers; (2) variation in counts by different phase contrast microscopes; (3) variation in counting methods between laboratories and over time; (4) use of area samples to characterize personal exposures; (5) absence of, or a limited number of, measurements from selected periods in the history of the study site; (6) incomplete work histories; and (7) uncertain and variable conversion of fiber counts from one method (e.g., midget impinger-based dust particle counts in millions of particles per cubic foot) to another (fiber counts by phase contrast microscopy).[514] Such limitations formed an important basis for the U.S. Environmental Protection Agency (EPA) to reject a proposed update of asbestos risk assessment in 2008.[515] The International Agency for Research on Cancer recently reviewed this issue and concluded that "there is a high degree of uncertainty concerning the accuracy of the relative potency estimates ... because of the severe potential for exposure misclassification in these studies" (p. 239).

Markowitz is hardly the first scientist to question estimates of relative potency of asbestos types due to inadequate and unreliable potency data. For example, Stayner et al.

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[513] Finkelstein et al., *Malignant Mesothelioma among employees of a Connecticut factory that manufactured friction materials using chrysotile asbestos.* Ann. Occup. Hyg. 54(6):692–696 (2010).

[514] Henderson et al., *After Helsinki: a multidisciplinary review of the relationship between asbestos exposure and lung cancer, with emphasis on studies published during 1997-2004,* Pathology 36(6): 517–550 (2004); Silverstein et al., *Developments in asbestos cancer risk assessment,* Am. J. Ind. Med, 52(11):850–858 (2009); Lenters et al., *A meta-analysis of asbestos and lung cancer: is better quality exposure assessment associated with steeper slopes of the exposure-response relationships?*, Environ. Health Perspect. 119(11):1547–1555 (2011).

[515] Johnson S. Letter from Stephen L. Johnson, EPA Administrator to Dr. Agnes Kane, Chair of Science Advisory Board Asbestos Committee (2008).

(1996) observed that "[h]istoric exposures in most of the epidemiologic investigations were based on impinger samples that assessed the number of fibers, and conversion factors were applied to estimate the number of fibers longer than 5 μm. Concerns have been raised about the accuracy of these conversion factors and the potential impact of associated errors on the assessment of risk."[516]

271. Regardless of the relative potency of various types of asbestos, chrysotile made up approximately 95% of the asbestos used in the United States. However, as Terracini et al. (2016) recently observed,

> the number of cancer cases produced by any environmental agent depends on the extent of opportunities for human beings to be exposed to it. Any (hypothesized) relatively 'low' carcinogenicity of chrysotile is largely balanced by the fact that, nowadays worldwide, over 2 million metric tons of this material per year are used in industrializing countries and that the majority of human beings are not protected by any strategy intended to reduce or eliminate the presence of asbestos in the environment.[517]

272. In an abstract presented at the International Mesothelioma Interest Group Meeting, Rolland et al. (2006) evaluated the risk of pleural mesothelioma in a French population-based case-control study between 1998 and 2002. The authors studied 19 French districts within the National Mesothelioma Surveillance Program covering 25% of the French population. The report was based on 467 confirmed cases (80% males, 41-93 years old) and 868 controls matched for sex, age and district. The authors found that among men, the highest risk was observed for the occupations of plumbers, pipefitters and sheet metal workers, and for the industries of ship repair, asbestos products, metal products and construction. The authors stated a significant dose-response relationship was found between cumulative occupational asbestos exposure and pleural mesothelioma, even for the lowest category (greater than 0-0.07 fibers/ml year; odds ratio 2.8, 95%; CI 1.7-4.7).[518]

273. Indeed, the scientific *consensus* is that there is no safe level of exposure to asbestos and that all levels of exposure carry with them some risk of cancer. Markowitz (2015) observed:

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[516] Stayner et al., *Occupational Exposure to Chrysotile Asbestos and Cancer Risk: A Review of the Amphibole Hypothesis,* Am. J. Public Health 86(2) (1996).
[517] Terracini et al., *Asbestos and product defence science*, Int'l J. Epidem. Advance Access pp. 1-5 (2016).
[518] Rolland et al., *Risk of pleural mesothelioma: A French population-based case control study [1998-2002].* Cancer. 54: Suppl 1S9, abstract 35 (2006).

> The current consensus is that there is no known safe level of exposure to asbestos.[519] Stated otherwise, no threshold has been demonstrated below which there is no identified risk of cancer related to asbestos. This view is based on several lines of evidence, specific and nonspecific to asbestos. Current risk assessment guidelines in the United States support linear extrapolation from the known dose–response curve to lower levels of exposure for DNA-reactive agents.[520] Although the exact mechanisms of asbestos carcinogenesis are not known at present, asbestos fibers, including chrysotile, are genotoxic and are, therefore, "DNA reactive."[521]

Although the foregoing discussion mainly evaluates the risk of lung cancer at low doses, conventional wisdom is that it takes greater exposures to cause increased risk of lung cancer than for mesothelioma. After discussing Lacourt (2014), Rödelsperger (2001), Offermans (2014) and Iwatsubo (1998), Markowitz (2015) observed that "finding excess cancer risk at low levels of occupational exposure to asbestos supports the notion that there is no safe level of exposure to asbestos". None of the major scientific bodies that have studied asbestos and mesothelioma have been able to identify a level of asbestos exposure below which mesothelioma will not occur. *See* World Health Organization: "No threshold has been identified for the carcinogenic risk of chrysotile";[522] National Cancer Institute Fact Sheet, *Asbestos Exposure and Cancer Risk* ("the overall evidence suggests there is no safe level of asbestos exposure") [523]; the British Thoracic Society concludes that "a history of occupational asbestos exposure can be obtained in about 90% of cases in the U.K." and there is "no evidence for a threshold dose of asbestos below

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[519] World Health Organization. Elimination of asbestos-related diseases. Geneva, Switzerland: World Health Organization; Report No.: WHO/SDE/OEH/6.03 (2006); National Institute of Occupational Safety and Health-Occupational Safety ("NIOSH") and Health Administration Work Group. Workplace Exposure to Asbestos: Review and Recommendations. 81–103: DHHS (NIOSH); 1980; Nielsen et al., *Occupational asbestos exposure and lung cancer—a systematic review of the literature*. Arch. Environ. Occup. Health 69(4):191–206 (2014); van der Bij et al., *Lung cancer risk at low cumulative asbestos exposure: meta-regression of the exposure response relationship,* Cancer Causes Control 24(1):1–12 (2013).

[520] Environmental Protection Agency ("EPA"). Risk Assessment Forum. *Guidelines for Carcinogen Risk Assessment*. 630/P-03/001F. Washington, D.C. (2005).

[521] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012); Nymark et al., *Molecular and genetic changes in asbestos-related lung cancer,* Cancer Lett. 265(1):1–15 (2008); Jaurand et al., *Mesothelioma: Do asbestos and carbon nanotubes pose the same health risk?*, Part. Fibre Toxicol. 6:16 (2009).

[522] World Health Organization. Elimination of asbestos-related diseases. Geneva, Switzerland: World Health Organization; Report No.: WHO/SDE/OEH/6.03 (2006).

[523] National Cancer Institute. Factsheet - Asbestos: Questions and Answers. Bethesda MD, National Institutes of Health. Ref Type: Pamphlet (2003).

which there is no risk."[524] A recent study examining the relationship between historical asbestos use and disease rates further supports the conclusion that a linear dose-response relationship exists between exposure to asbestos and disease, even at low doses.[525] In fact, the Occupational Health and Safety Administration (OSHA) determined that even at the lowest level of asbestos exposure at which OSHA found it feasible to set a standard in the workplace, 0.1 f/cc, there is significant risk of mesothelioma.[526]

274. Several agencies have commented that there is no safe level of exposure to asbestos:

a. NIOSH, 1976 (page 92): "excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or a 'safe' level of asbestos exposure."
b. NIOSH, 1980 (page 3): "All levels of asbestos exposure studied to date have demonstrated asbestos related disease...there is no level of exposure below which clinical effects do not occur."
c. USPHS, 1980: "It is important to point out that when a permissible level for exposure (PEL) to a certain carcinogen is set by OSHA, there is no implication that such a level is safe. To the contrary, it is the agency's policy that any occupational exposure to a carcinogen carries with it some risk of disease, even if it cannot be easily or precisely measured."[527]
d. NIOSH, 1986 (page 319): "a linear, no threshold, dose-response relationship ... Any asbestos exposure carries with it some increased risk of asbestos related disease."
e. OSHA, 1994 (page 40978): "reducing exposure to 0.1 f/cc would further reduce, but not eliminate, significant risk. The 0.1 f/cc level leaves a remaining significant risk."

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[524] British Thoracic Society. Statement on malignant mesothelioma in the United Kingdom. Thorax. 56(4): 250-65 (2001).

[525] Lin et al., *Ecological association between asbestos-related diseases and historical asbestos consumption: an international analysis*. Lancet. 369(9564): 844-9 (Mar. 10, 2007)

[526] Occupational Safety and Health Administration. Occupational exposure to asbestos; final rule. Federal Register; 59:40964-1162 (1994).

[527] In WORKPLACE EXPOSURE TO ASBESTOS Review and Recommendations DHHS (NIOSH) Publication No. 81-103 NIOSH-OSHA Asbestos Work Group (April 1980) states:

> Where asbestos exposures cannot be eliminated, they must be controlled to the lowest level possible. A significant consideration in establishing a permissible exposure limit should be the lowest level of exposure detectable using currently available analytical techniques. At present this level would be 100,000 fibers greater than 5 microns in length per cubic meter averaged over an 8-hour workday. Regardless of the choice of a permissible exposure limit, the best engineering controls and work practices should be instituted, and protective clothing and hygiene facilities should be provided and their use required of all workers exposed to asbestos. Respirators are not a suitable substitute for these control measures. *The committee also reiterates its judgment that even where exposure is controlled to levels below 100,000 fibers, there is no scientific basis for concluding that all asbestos-related cancers would be prevented.*

f. WHO, 1998 (page 144): "Exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner. No threshold has been identified for carcinogenic risks."

g. WTO, 2000: "the experts confirm the position of the European Communities according to which it has not been possible to identify any threshold below which exposure to chrysotile would have no effect. The experts also agree that the linear relationship model, which does not identify any minimum exposure threshold, is appropriate for assessing the existence of a risk. We find therefore that no minimum threshold level of exposure or duration of exposure has been identified with regard to the risk of pathologies associated with chrysotile, except for asbestosis."

h. OSHA, (2016): "There is no "safe" level of asbestos exposure for any type of asbestos fiber. Asbestos exposures as short in duration as a few days have caused mesothelioma in humans. Every occupational exposure to asbestos can cause injury of disease; every occupational exposure to asbestos contributes to the risk of getting an asbestos related disease. Where there is exposure, employers are required to further protect workers by establishing regulated areas, controlling certain work practices and instituting engineering controls to reduce the airborne levels. The employer is required to ensure exposure is reduced by using administrative controls and provide for the wearing of personal protective equipment. Medical monitoring of workers is also required when legal limits and exposure times are exceeded."[528]

275. The introduction of *any* source of asbestos above the trace background amounts into the environment has been shown to create a significant increase in the risk of mesothelioma. For example, a recent article examining the incidence of mesothelioma in six Egyptian neighborhoods surrounding a plant that used chrysotile asbestos found 83 cases representing a "26-fold excess risk of pleural mesothelioma due to environmental exposure."[529] The levels of asbestos in these neighborhoods were very low - 17 of the mesothelioma cases occurred in a neighborhood a half a mile away from the plant where airborne asbestos the dust was measured at 0.04 f/cc. An additional 27 mesothelioma cases occurred in neighborhoods between 1 and 2.5 kilometers away with a dust measurement of 0.025 f/cc or less. Other studies have shown similar risks. Azuma (2009)[530] compared mesothelioma rates and environmental exposure levels at different periods in Japan and predicted that the "cumulative number of deaths from mesothelioma due to environmental asbestos exposure would be around 13,000-30,000 by 2039"; a study by Pan supported the hypothesis that residential proximity to naturally occurring deposits of asbestos in California is significantly associated with an increased risk of

[528] Occupational Safety & Health Administration ("OSHA"), *Safety and Health Topics – Asbestos*, https://www.osha.gov/SLTC/asbestos/ (accessed September 9, 2016) (citations omitted).

[529] Madkour et al., *Environmental exposure to asbestos and the exposure-response relationship with mesothelioma*, East Mediterr. Health J. 15(1): 25-38 (Jan 2009).

[530] Azuma et al., *Mesothelioma risk and environmental exposure to asbestos: past and future trends in Japan*. Int. J. Occup. Environ. Health.; 15(2): 166-72 (Apr 2009).

mesothelioma.[531] The United States Environmental Protection Agency has noted that, because of the nature of asbestos and its interaction with the human body, each exposure increases the likelihood of developing an asbestos-related disease.[532]

276. Attempts to postulate thresholds or safe levels for exposure to asbestos have been dismissed as "logical nonsense."[533] The lack of a defined "safe" level for exposure to asbestos is supported by research, including both epidemiology and medical journal reports. For example, a large French study recently concluded that substantial excess mortality occurs at exposure levels below current regulatory levels.[534]

277. "Malignant pleural mesothelioma is a global, manmade cancer problem with increasing death tolls due to sustained mining and use of asbestos. Preventive measures including a global ban on asbestos should be mandatory."[535] In 2013, the International Commission on Occupational Health (http://www.icohweb.org/site/about-icoh.asp), issued its statement on a *Global Asbestos Ban and the Elimination of Asbestos Diseases*[536], stating:

> There is sufficient evidence in humans for the carcinogenicity of all forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite and anthophyllite). Malignant asbestos-related diseases include lung cancer, mesothelioma and cancers of the ovary and larynx.[537] Non-malignant asbestos-related diseases include

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[531] Pan et al., *Residential Proximity to Naturally Occurring Asbestos and Mesothelioma Risk in California.* Am. J. Respir. Crit. Care Med. (2004); 172:1019-25 (2005).

[532] Environmental Protection Agency ("EPA"). A Guide for Ship Scrappers: Tips for Regulatory Compliance. Environmental Protection Agency; Report No.: 315-B-.00-001 (2000).

[533] Hodgson et al., *The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure.* Ann. Occup. Hyg. 44(8):565-601 (Dec 2000).

[534] Iwatsubo et al., *Pleural mesothelioma: dose-response relation at low levels of asbestos exposure in a French population-based case-control study.* Am. J. Epidem. 148(2):133-42 (Jul 15 1998). Indeed, Iwatsubo et al., (1998) found that attempts to quantify the minimum dose of asbestos that will result in mesothelioma through epidemiology have demonstrated that "[a] significant excess of mesothelioma was observed far below the limits adopted in most industrial countries during the 1980s."; Rödelsperger et al., *Asbestos and man-made vitreous fibers as risk factors for diffuse malignant mesothelioma: results from a German hospital-based case-control study.* Am. J. Ind. Med.; 39(3):262-75 (Mar 2001).

[535] Røe et al., *Malignant pleural mesothelioma: History, Controversy and Future of a Manmade Epidemic,* Eur Respir Rev 24:115-131 (2015).

[536] International Commission on Occupational Health. ICOH Statement: Global Asbestos Ban and the Elimination of Asbestos-Related Diseases. pp 1-3 (Oct 2013).

[537] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite). IARC Monographs, Volume 100C, 2012. http://monographs.iarc.fr/ENG/Monographs/vol100C/mono100C-11.pdf

asbestosis and pleural abnormalities such as pleural thickening, pleural calcification and pleural effusion.[538]

International consensus has recommended that a total ban on production and use of all forms of asbestos is the best way to eliminate the occurrence of asbestos-related diseases. In 2006 WHO stated that the efficient way to eliminate asbestos-related diseases is to stop using all types of asbestos.[539]

As there is sufficient evidence by the International Agency on Research on Cancer (IARC 2012) of the carcinogenicity in humans for all forms of asbestos, amphibole-only bans are inadequate; asbestos bans need to include chrysotile as well.

Even after a total ban on production and use of asbestos is achieved, occupational exposure to asbestos will persist due to the continued presence of asbestos from prior use in building materials and durable machinery/equipment. Workers who carry out maintenance, demolition and removal of asbestos-containing materials will thus continue to be at risk. Therefore a set of protective measures must be implemented to optimize effective prevention.

278. There is no exposure level below which asbestos-related disease risk can be totally eliminated. A total global ban represents the best form of primary prevention.

279. The National Research Council Committee on Non-Occupational Health Risks of Asbestiform Fibers found background environmental exposure of 0.0004 f/cc over a 73 year lifetime (which results in a cumulative dose of 0.03 f/cc-y) was associated with 9 cases of mesothelioma per million. A "higher" exposure of 0.002 fibers/cc (which results in a cumulative dose of 0.146 f/cc-y) was associated with 46 cases of mesothelioma per million – a five-fold risk[540].

[538] American Thoracic Society. *Diagnosis and initial management of nonmalignant diseases related to asbestos*. Am J Respir Crit Care Med 2004; 170:691-715.

[539] World Health Organization. Elimination of asbestos-related diseases. WHO/SDE/OEH/06.03. September 2006. http://www.who.int/occupational_health/publications/asbestosrelateddiseases.pdf

[540] Asbestiform Fibers Nonoccupational Health Risks, Committee on Non-Occupational Health Risks of Asbestiform Fibers, Board on Toxicology and Environmental Health Hazards, Commission on Life Sciences, National Research Council, National Academy Press, Washington, D.C. (1984).

280. A recent study examining the relationship between historical asbestos use and disease rates further supports the conclusion that a linear dose-response relationship exists between exposure to asbestos and disease and that no "safe" level of exposure exists.[541]

281. The consensus of the mainstream scientific community is that studies have shown that any identified occupational, domestic, or environmental exposure to asbestos increases the risk of mesothelioma.[542] While scientists working for the asbestos industry and defendants in asbestos product liability lawsuits contend that one can extrapolate a "no adverse effect level" from the existing data and/or that massive potency differences between hypothetical identical fibers of different types of asbestos, those opinions are outside of the scientific mainstream and have been considered and rejected by independent panels of scientific experts with no bias or agenda, such as IARC, ATSDR and NIOSH.[543]

282. In determining cause and effect, physicians and scientific researchers typically look at two distinct issues, general causation and specific causation. General causation focuses on the issue of whether a particular substance is capable of causing a particular injury or condition in the general population. Specific causation, on the other hand, addresses the issue of whether an exposure to a substance or substances has caused or contributed to the development of a particular individual's injury or disease. To determine general causation, researchers evaluate a variety of data sets including animal studies, toxicology studies, human cellular toxicology studies, molecular studies, case reports, epidemiologic case-control and cohort studies and general biologic principles. If a review of these data

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[541] Lin et al., *Ecological association between asbestos-related diseases and historical asbestos consumption: an international analysis.* Lancet. 369(9564):844-9 (2007).

[542] Pan et al., *Residential Proximity to Naturally Occurring Asbestos and Mesothelioma Risk in California.* Am. J. Respir. Crit. Care Med. (2004); 172:1019-25 (2005); Iwatsubo et al., *Pleural mesothelioma: dose-response relation at low levels of asbestos exposure in a French population-based case-control study.* Am. J. Epidem. 148(2):133-42 (Jul. 15, 1998); Rödelsperger et al., *Asbestos and man-made vitreous fibers as risk factors for diffuse malignant mesothelioma: results from a German hospital-based case-control study.* Am. J. Ind. Med.; 39(3):262-75 (Mar 2001); Skammeritz et al., *Asbestos Exposure and Survival in Malignant Mesothelioma: A Description of 122 Consecutive Cases at an Occupational Clinic.* 2(4) J. Occup. & Environ. Med. 228, 228-29 (Oct. 2011) (noting that for some patients the total asbestos exposure was "a few days"); Newhouse et al., *Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area.* 22(4) Br. J. Ind. Med. 261, 261-66 (1965) (two cases with 2 months or less exposure to asbestos); Borow et al., *Critical Review, Mesothelioma following Exposure to Asbestos: A review of 72 Cases.* 64(5) Chest J. 641 (1973); Greenberg & Davies, supra note 2; Workplace Exposure to Asbestos: Review and Recommendations (DHHS (NIOSH) pub. no. 81-103, Apr. 1980) ("Studies of duration of exposure suggest that even at very short exposure periods (1 day to 3 months) significant disease can occur.").

[543] While I am aware that asbestos defendants and their lawyers often claim that the evaluations and conclusions of OSHA and EPA and other agencies with regulatory duties are "based upon the precautionary principle and not science" or other similar claims, no such claim can be levied at IARC, ATSDR or NIOSH whose duties are to evaluate the science, not set policy.

sets establishes that there is a general cause and effect relationship, physicians can then determine specific causation by ascertaining whether an exposure caused or contributed to a particular individual's disease, full assessing all relevant factors such as frequency of exposure, proximity, qualitative dose, latency and more.

283. As an overall model for determining causality, the considerations espoused by Sir Austin Bradford Hill are well accepted and have been widely used by epidemiologists and scientists of other disciplines.[544] They are: temporality, biologic gradient (dose-response), consistency, biologic plausibility, strength of association, analogy, experimental evidence, coherence and specificity. The scope of medical evidence that substantiate these considerations is both comprehensive and widely inclusive of all the available data. The empirical support for the considerations over such a large epistemological landscape represents, in itself, the ultimate merit of the considerations. The Hill Criteria are generally accepted but Hill himself recognized that no one factor was dispositive and that all the evidence matters, nor did all need to be met.

284. In his classic piece on causality, Hill himself recognized at the outset that epidemiological studies were not always necessary to reach a conclusion about the cause of a particular disease:

> There are, of course, instances in which we can reasonably answer these questions from the general body of medical knowledge. A particular, and perhaps extreme, physical environment cannot fail to be harmful; a particular chemical is known to be toxic to man and therefore suspect on the factory floor.[545]

285. The fact that any one consideration or piece of scientific evidence can always be subject to criticism reinforces the need for consideration of all forms of scientific evidence. As Hill noted, "None of my nine view points can bring indisputable evidence for or against the cause-and-effect hypothesis, and none can be required as a *sine qua non*." Before applying this framework to the issue of whether exposure to chrysotile asbestos causes or contributes to cause mesothelioma, it is important to reflect upon the relative significance of each of these considerations in making such a determination. None of Hill's considerations require statistical epidemiologic data in the sense that that term is used to describe statistical analysis to the exclusion of observational epidemiology.

286. Lemen (2004), using the Bradford Hill considerations, reviewed the evidence for chrysotile's ability to cause mesothelioma and concluded that chrysotile exposure increased the risk of mesothelioma in humans.[546] IARC's most recent review also

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[544] Hill, *The Environment and disease: association or causation?* 58(5) Proc. Royal Soc. Med. 295, 299 (1965).

[545] Hill, *The Environment and disease: association or causation?* 58(5) Proc. Royal Soc. Med. 295, 295 (1965).

[546] Lemen, *Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model.* Int. J. Occup. Environ. Health. 10:233–239 (2004).

concludes, using the Bradford Hill considerations, that chrysotile is a cause of pleural and peritoneal mesothelioma, lung cancer and asbestosis, among other diseases.[547] When it comes to looking at cause and effect through epidemiology, IARC noted important limitations:

> When several epidemiological studies show little or no indication of an association between an exposure and cancer, a judgment may be made that, in the aggregate, they show evidence of lack of carcinogenicity. Such a judgment requires first that the studies meet, to a sufficient degree, the standards of design and analysis described above. Specifically, the possibility that bias, confounding or misclassification of exposure or outcome could explain the observed results should be considered and excluded with reasonable certainty. In addition, all studies that are judged to be methodologically sound should (a) be consistent with an estimate of effect of unity for any observed level of exposure, (b) when considered together, provide a pooled estimate of relative risk that is at or near to unity, and (c) have a narrow confidence interval, due to sufficient population size. Moreover, no individual study nor the pooled results of all the studies should show any consistent tendency that the relative risk of cancer increases with increasing level of exposure. It is important to note that evidence of lack of carcinogenicity obtained from several epidemiological studies can apply only to the type(s) of cancer studied, to the dose levels reported, and to the intervals between first exposure and disease onset observed in these studies. Experience with human cancer indicates that the period from first exposure to the development of clinical cancer is sometimes longer than 20 years; latent periods substantially shorter than 30 years cannot provide evidence for lack of carcinogenicity.[548]

287. The IARC criteria for causality – studies that demonstrate of lack of elevated risk obtained "from several epidemiological studies can apply only to the type(s) of cancer studied, to the dose levels reported, and to the intervals between first exposure and disease onset observed in these studies" – explain why, given the body of literature regarding chrysotile asbestos, I believe the medical and scientific evidence that asbestos from brakes causes mesothelioma. Most of the studies including people who worked with asbestos brakes lack important information about exposure levels, duration, and latency.

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[547] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012).

[548] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012) at page 22.

288. "Strength of association" is a reflection of the power of a study. Human epidemiologic studies are not the only source of information type of data available to access this consideration. This consideration can be determined from human, animal or microbiologic studies. The relevance of this consideration is limited by the prevalence of co-factors that may interfere with the measurement of the factor that is being studied. Strength of association is not a measure of the importance of a particular factor in causation.[549] It is a gauge of potential errors due to confounding or bias. Studies with large rate ratios are less likely to contain errors attributable to bias or confounding. Causal factors with "relatively low rate ratios" may be equally or more important than strong associations from a public health perspective. In addition, a rate ratio of two is not required to establish that a factor contributed to a disease in a particular individual (specific causation). For example, chronic smoking of less than a pack a day induces less than a two fold increase in the risk of heart disease. Nonetheless, it is a universal opinion among physicians that smoking contributes to a smoker's heart disease if he/she smoked at this rate. In fact smoking is a contributing cause of death for about 400,000 people annually but "only" contributes to fewer than 100,000 cases of lung cancer each year. The same is true of second-hand or environmental tobacco smoke. The consensus of the medical community is that second-hand smoke causes cancer and other diseases notwithstanding the fact that the pooled risk estimate of the risk of lung cancer caused by second hand smoke is approximately 1.3.[550] Most elevations of blood cholesterol that require medical treatment do not double the risk of heart disease. Furthermore, physicians, when treating a patient for a heart attack, will indicate that previous smoking of a half pack of cigarettes per day for 30 years, family history of heart disease (non-genetic), history of elevated cholesterol of 250 mg/dl are all contributing causes of their patient's heart attack. Considered by themselves, none of these factors have an elevated rate ratio greater than two. Epidemiological studies can, when evaluated together, provide more confidence in an association even in the absence of a "statistically significant" finding from any individual study. Greenland states,

> ...lack of 'statistical significance' is not evidence of a lack of hazard... a claim by an expert that 'statistical significance' or 'nonsignificance' demonstrates presence or absence of causation should serve as a warning to the court that said expert is incompetent in the use of statistics for causal inference.[551]

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[549] Rothman, K. J. *Causal Inference* --- Lanes, S. F.: Error and uncertainty in causal inference. In Causal Inference, pp. 182-183.

[550] U.S. Department of Health and Human Services. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, (2006) (Surgeon General finding that a 20-30% increase in risk was sufficient to infer causation of lung cancer from secondhand tobacco smoke at p. 445).

[551] *See* Declaration of Professor Sander Greenland, taken on June 11, 2001.

289. Recent epidemiologic studies have showed strong associations between chrysotile asbestos and mesothelioma.[552]

290. After delineating each of his nine points, Hill's final emphasis placed responsibility on scientists for making causal judgments without blind (in fact without any) reliance on "statistical tests."

291. Hill explained his consideration as follows:

> *What they* [Hill's nine points] *can do, with greater or less strength, is to help us to make up our minds on the fundamental question- is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?* ***No formal tests of significance can answer those questions.*** *Such tests can, and should, remind us of the effects that the play of chance can create, and they will instruct us in the likely magnitude of these effects. Beyond that they contribute nothing to the 'proof' of our hypothesis.... The question that I had to answer, by the use of the National Health Insurance records of that time* [1930], *was this: Do the workers in the cardroom of the spinning mill, who tend the machines that clean the raw cotton, have a sickness experience in any way different from that of other operatives in the same mills who are relatively unexposed to the dust and fibre that were features of the cardroom? The answer was an unqualified 'Yes.' From age 30 to age 60 the cardroom workers suffered over three times as much from respiratory causes of illness whereas from non-respiratory causes their experience was not different from that of the other workers. This pronounced difference with the respiratory causes was derived not from abnormally long periods of sickness but rather from an excessive number of repeated absences from work of the cardroom workers.*
>
> *All this has rightly passed into the limbo of forgotten things. What interests me today is this: My results were set out for men and women separately and for half a dozen age groups in 36 tables. So there were plenty of sums. Yet I cannot find that anywhere I*

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[552] *E.g.*, Elliott et al., *Lung cancer mortality in North Carolina and South Carolina chrysotile asbestos textile workers.* Occup. Environ. Med. doi:10.1136 (2012); Loomis et al., *Lung cancer mortality and fiber exposures among North Carolina asbestos textile workers.* Occup. Environ. Med. 66:535–542 (2009); Mirabelli et al., *Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy.* Occup. Environ. Med. 65:815–819 (2008); Mamo et al., *Mortality experience in an historical cohort of chrysotile asbestos textile workers.* WS-E-03. Paper presented at the Global Asbestos Congress, Waseda University, Tokyo, Japan, November 19–21, (2004).

> *thought it necessary to use a test of significance. The evidence was so clear-cut, the differences between the groups were mainly so large, the contrast between respiratory and non-respiratory causes of illness so specific, that no formal tests could really contribute anything of value to the argument. So why use them?*
>
> *...some editors of journals will return an article because tests of significance have not been applied. Yet there are innumerable situations in which they are totally unnecessary- because the difference is grotesquely obvious. ...*
>
> *Of course I exaggerate. Yet too often I suspect we waste a deal of time, we grasp the shadow and lose the substance. We weaken our capacity to interpret data and to take reasonable decisions whatever the value of P. And far too often we deduce 'no difference' from 'no significant difference.' Like fire, the chi square test is an excellent servant and a bad master.*[553]

292. Hill recognized that decisions have to be made in the absence of perfect data: "All scientific work is incomplete--whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time."[554]

293. Not every question of causation needs an epidemiological study. This concept was recently endorsed by the 58 Scientists Brief:

> Mesothelioma is caused by asbestos-containing dust, not by a job classification or product type — the issue is simply one of inhalation of respirable asbestos fibers. Just as there is no scientific "rule" that worker exposures be quantified in order for attribution, there is no "rule" that a product specific positive epidemiology study be available for attribution.[555]

294. It is in this context that the mainstream scientific community has reviewed the literature on asbestos and concluded that asbestos from any source is a cause of mesothelioma in someone with cumulative exposure beyond that of the background exposures sustained by all.[556] "No exposure to asbestos is without risk."[557] Cumulative dose best explains the

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[553] Hill, *The Environment and disease: association or causation?* 58(5) Proc. Royal Soc'y Med. 295, 299 - 300 (1965) (bold and italics added).

[554] Hill, *The Environment and disease: association or causation?* 58(5) Proc. Royal Soc'y Med. 295, 299 - 300 (1965) at page 12.

[555] 58 Scientists, p. 16.

[556] In this regard, all identifiable exposures of an individual are beyond "background" as a matter of simple logic as they are in excess of the "background" exposures of that individual. It is a

increased risk of mesothelioma in the population.[558] Numerous authors from all over the world recognize that cumulative exposure to asbestos is the best measure of risk and critical for evaluating causality in an individual.[559]

295. The greater the asbestos exposure a patient receives, the more that exposure contributes to the patient's risk of mesothelioma. The reasons for this may lie in the origins of cancer at the cellular level:

> Several authors have studied the mechanism of fibre deposition and retention in the lungs. Once deposited in the lung, asbestos fibres may be translocated into different organs and tissues, including the pleura. This was demonstrated in animals following inhalation or intratracheal deposition, and in humans by investigation of fibre retention in different body compartments including the pleura. A recent paper discusses the translocation pathways of asbestos fibres to the pleura.[560]

"Fibre deposition in the lung is followed by the recruitment of inflammatory cells, which produce several factors: ROS (reactive oxygen species), RNS (reactive nitrogen species), clastogenic factors and cytokines that may stimulate and/or damage neighbouring mesothelial cells. Fibres also may produce ROS. Moreover, mesothelial cells respond by

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logical impossibility to measure the risk created by "background" exposures sustained by all individuals as there is no unexposed comparison group against which to measure the rate of disease. Put another way, it is fallacious to say that "background" exposures to asbestos are free from danger – the risk of such exposures simply cannot be measured on a population basis.

[557] LaDou et al., *The Case for a Global Ban on* Asbestos. Environ. Health Perspectives 118:7 (July, 2010).

[558] Hammar et al., Dail and Hammar's Pulmonary Pathology Vol II, Neoplastic Lung Disease 3rd Ed Chapter 43 Neoplasms of the Pleura 587 (2008) ("[W]hen there are multiple asbestos exposures, each contributes to cumulative exposure and, hence, to the risk and causation of MM [malignant mesothelioma]."); Bignon et al., *History and Experience of Mesothelioma in Europe*. Mesothelioma 36 (Bruce W. Robinson & Phillippe Chahinian eds., 2002).

[559] *E.g.* Frontario et al., *Primary Peritoneal Mesothelioma Resulting in Small Bowel Obstruction: A Case Report and Review of Literature,* Am J Case Rep, 16:496-500 (2015) (stating "[i]t has been previously determined that cumulative asbestos exposure leads to a proportional increase in mesothelioma risk. Mesothelioma can result from non-industrial environmental contact with asbestos fibers, and para-occupational exposure occurs; for example, women who have laundered their husband's work-related clothing."); Nemo et al., *Mesothelioma in a Wine Cellar Man: Detailed Description of Working Procedures and Past Asbestos Exposure Estimation,* Ann. Occup. Hyg., Vol 58, No. 8, 1168-1174 (2014) (discussing mesothelioma in wine maker with cumulative exposure to chrysotile asbestos in the range of 0.5 f/cc/years to 3.0 f/cc/years).

[560] Jaurand et al., *Mesothelioma: Do asbestos and carbon nanotubes pose the same health risk?*, Particle and Fibre Toxicity 6:16 (2009) (citations omitted).

fibre internalisation according to a phagocytic process associated with oxidative reactions."[561] Significantly, increased exposure can lead to increased genotoxic events:

> Many investigations have focused on DNA damage provoked by asbestos fibres in mesothelial cells. Several studies have demonstrated different types of DNA damage (DNA breakage, base oxidation), and perturbation of the mitotic process, showing that base oxidation and DNA breakage (single strand and double strand breaks) were detected in asbestos-treated mesothelial cells. These may be due to ROS/RNS production and to the mesothelial cells' ability to phagocytise asbestos fibres. . . . Asbestos fibres produce structural chromosome alterations; significant enhancement of aneuploid cells, abnormal anaphases and telophases. Induction of micronuclei by all types of asbestos in primary cultures of human mesothelial cells has been reported by Poser *et al.*[562] Other studies have shown genomic alterations in asbestos-treated human mesothelial cells. Loss of heterozygosity was detected as asbestos- induced mutations in a human mesothelioma cell line.[563]

Although these scientific observations are not conclusive proof, this type of evidence helps explain why mesothelioma is an exposure-response disease.

296. Based on evidence like that above, I joined the 67 Scientists in concluding that it "is the generally accepted scientific consensus that the cumulative effect of occupational and para-occupational exposures is the ultimate cause of mesothelioma in individuals with mesothelioma and that all such exposures increase the risk of mesothelioma."[564] We rejected the claim that that recognizing the dose-response relationship was the equivalent of stating that a single fiber of asbestos is a substantial cause of a patient's mesothelioma, pointing out that "[o]ccupational and para-occupational exposures to asbestos constitute exposures to millions, billions, trillions, or more asbestos fibers in a single day."[565]

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[561] Jaurand et al., *Mesothelioma: Do asbestos and carbon nanotubes pose the same health risk?*, Particle and Fibre Toxicity 6:16 (2009) (citations omitted).

[562] Poser, et al., *Modulation of genotoxic effects in asbestos exposed primary human mesothelial cells by radical scavengers, metal chelators and a glutathione precursor*, Mutation Res 559:19-27 (2004).

[563] Jaurand et al., *Mesothelioma: Do asbestos and carbon nanotubes pose the same health risk?*, Particle and Fibre Toxicity 6:16 (2009) (citations omitted).

[564] 67 Scientists at page 2.

[565] 67 Scientists at page 3 (citing Abraham, J, *Letter to the Editor, re: When Science Crosses Politics: The Case of Naturally Occurring Asbestos*, J. Environ. Health 67:3 40-41 (2004) for the fact that "[a] simple calculation based on the size and mass of an average asbestos fiber will tell you that in a single gram of pure (100%) asbestos, there will be on the order of one trillion fibers (1,000,000,000,000 fibers/gram in pure asbestos)."

297. I am aware of a substantial body of data indicating the high levels of exposure to asbestos that can occur depending upon individual circumstances. I am familiar with historical measurements from industry, measurements from government investigators and measurements taken by consultants. I believe it is appropriate to consider all of these measurements when considering the implications of particular sources of asbestos exposure.

XII. Asbestos Dust from Brakes and Clutches Causes Mesothelioma.

298. From the beginning of my medical and scientific training, I learned that it was exposure to as substance, rather than the job title, that causes disease. During my career at Mount Sinai, I participated in a clinical study of 90 (84 working and 6 retired due to age) union vehicle maintenance workers in New York City.[566] 61 workers did more than one brake job a week over the 20 – 36 years working as mechanics. The authors of the published study, among the first studies of vehicle mechanics, reported the prevalence both of chest x-ray changes and restrictive function results was significantly higher after 20 years of exposure than before, a result expected after occupational exposure to asbestos.

> Measurements were made by optical microscopy of asbestos levels during brake repair and maintenance work in New York City. Both time-weighted averages and peak levels showed significant asbestos exposure. . . . Over one-quarter of a group of experienced vehicular maintenance workers examined had evidence of x-ray abnormalities consistent with asbestosis; one-quarter also had restrictive pulmonary function test findings. While this preliminary study was limited in scope and was restricted to volunteers, and its results cannot therefore readily be generalized to all brake maintenance workers in the United States, the findings suggest that asbestos disease will be present among such workers and that appropriate control measures should be urgently instituted.[567]

299. In NIOSH's Division of Respiratory Disease Studies, *Occupational Respiratory Diseases*, DHHS (NIOSH) Publication No. 86-102 (September 1986), Dement et al.'s chapter on Asbestosis, identified the population at risk of asbestosis:

> By far the largest number of workers with potential asbestos exposures may be found in industries which utilize asbestos products such as the construction industry, the automobile servicing industry (including remanufacturing of asbestos containing parts), and the shipbuilding and repair industry. In the construction industry, including those doing demolition and repair, an estimated 180,000 to 408,000 workers are potentially exposed to asbestos. The automobile servicing industry includes brake and clutch servicing garages, rebuilding and refacing friction components, and repackaging of friction products. Within this sector, 2 million workers are potentially exposed to asbestos (17). Approximately 3,800 workers are potentially exposed to asbestos in shipbuilding and repair.

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[566] Lorimer et al., *Asbestos exposure of brake repair workers in the United States*. 43(3) Mt. Sinai J. Med. 207, 207-18 (1976). This study was partially funded by Ford Motor Company.
[567] Lorimer et al., *Asbestos exposure of brake repair workers in the United States*. 43(3) Mt. Sinai J. Med. 207, 207-18 (1976).

If automotive workers are at risk of asbestosis, as indicated by Lorimer et al. (1976), Nicholson et al. (1984)[568] and Dement's chapter in the NIOSH text, such workers are at a greatly increased risk of mesothelioma.

300. NIOSH's Division of Respiratory Disease Studies, *Occupational Respiratory Diseases*, DHHS (NIOSH) Publication No. 86-102 (September 1986), Lilis' chapter on Mesothelioma identified some of the people at risk of mesothelioma from asbestos exposure:

> **LIST OF OCCUPATIONS AND INDUSTRIES INVOLVED**
>
> Occupations and industries at risk to mesothelioma include all of those listed for asbestosis. [Dement et al. listed "the construction industry, the automobile servicing industry (including remanufacturing of asbestos containing parts), and the shipbuilding and repair industry. . . . The automobile servicing industry includes brake and clutch servicing garages, rebuilding and re-facing friction components, and repackaging of friction products."]
>
> All available information indicates that mesothelioma may be the result of low levels and/or relatively short (of the order of several weeks to several months) asbestos exposure. The dose-response relationship for mesothelioma is therefore different than that for asbestosis (which develops with higher exposure levels over longer time periods) or bronchial carcinoma associated with asbestos exposure (which increases in incidence even after short periods of high asbestos exposure levels, but shows a marked increase in incidence with duration of exposure). Since low asbestos exposure levels carry a significant risk of mesothelioma, occupations and industries characterized by relatively low asbestos levels (auto mechanics and brake repair, tapers in dry wall construction, handling of finished asbestos products including asbestos cement), while at relatively low risk for the development of parenchymal interstitial fibrosis (asbestosis), are nevertheless at high risk for mesothelioma.
>
> Equally important is the fact that numerous workers in the various trades which do not simply [involve] direct asbestos exposure, such as electricians, painters, welders, carpenters, etc., in shipbuilding or ship repair, in construction, in maintenance work at chemical plants, and even automobile salesmen supervising repair work, are frequently exposed to asbestos due to their mere presence in work areas where asbestos is being handled. This

[568] Nicholson et al., *Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos*, NIOSH Contract 210-77-0119 (1984).

> "bystander" exposure has been repeatedly documented to be responsible for numerous cases of mesothelioma. It is therefore important to establish the principle that such indirect exposure carries a significant risk of mesothelioma.[569]

Lilis also pointed out exposure to asbestos was they key to risk, indicating the following populations are at risk:

> • all occupations with direct contact and handling of asbestos.
>
> • employees with other occupations (electricians, welders, painters, carpenters, etc.) who work or have worked--even for short periods- in areas where asbestos has been handled by others.
>
> • family members (household contacts) of asbestos workers who have been exposed to asbestos fibers brought into the household by the worker. Household contamination has been found to result in asbestos exposure of family members of asbestos workers, sufficient in magnitude to induce mesothelioma [citations omitted].
>
> • individuals who have resided in the vicinity (one mile) of an asbestos plant, shipyard, or other source of asbestos contamination.

301. Based on my work evaluating the health of vehicle mechanics, my clinical practice and my experiences, I published my belief that auto mechanics were at risk of asbestos-related diseases, including mesothelioma, in 1995.[570] I reached this conclusion with full knowledge of the epidemiological and other scientific evidence on asbestos and with the understanding of the difficulties of studying small workforces engaged in varied activities in diverse work environments. As I explained in 1995, when one sees evidence of asbestosis in a workforce, such as in mechanics, such workers are at increased risk of cancer, including mesothelioma:

> a multitude of factors lead to the development or non-development of malignancies in any individual, including genetics, host factors, and immunologic status. Nevertheless, the basic principle is that even small amounts of exposure carry some risk, although the risk is clearly related to how much exposure takes place. The classic dose-response principle, which applies to so much of biology, clearly applies here as well.
>
> Having recognized that the risk for cancer occurs at levels below which one would not expect to see evidence of asbestosis has led to the realization that in virtually every occupational population in

[569] NIOSH's Division of Respiratory Disease Studies, *Occupational Respiratory Diseases*, DHHS (NIOSH) Publication No. 86-102 (September 1986) (citations omitted).
[570] Frank, *Medical and Public Health Approaches to Asbestos Disease*, Mount Sinai J. Med. 62(5) (1995).

which asbestos disease has been looked for, it has been found (Table 3)[reproduced below].[571]

TABLE 3
Selected Occupations Shown to Be Associated with Asbestos-Related Diesease

Miners and millers	Railroad workers
Insulators	Shipyard workers
Textile workers	Custodial workers
Construction workers	Telephone installers
Plumbers/pipefitters	Steelworkers
Electricians	Cement workers
Sheetmetal	Maintenance workers
Painters	Seamen
Elevator installers	Jewelers
Others	Powerhouse workers
Auto mechanics	Refinery workers

Significant environmental exposure occurs in those living near mines, factories, and shipyards and in family members of workers.

302. It is precisely because scientists and physicians understand the limitations of epidemiology and how certain factors can bias studies toward a lack of statistical significance or finding of a point estimate of no increased risk, that we look at the epidemiology of a substance along with the other scientific data described above. Each epidemiological study must be evaluated for its strengths and weaknesses, and decisions about cause and effect should only be made on reliable data.

303. A 1990 bulletin produced by the Ohio State University and NIOSH provided accurate information about the hazards of asbestos from brakes, explaining:

> We can't see it, smell it, taste it, or feel it, but we know that asbestos can cause debilitating and often fatal diseases. We also know that these diseases take as long as 20 years to develop. What we *don't* know is how *much—or how little—exposure* to asbestos can cause them.
>
> There is *no known safe level of exposure.* Anyone who works with any quantity of asbestos for any length of time risks developing serious disease later in life. And that person puts *others* at risk. Invisible asbestos fibers cling to clothes, hair, and skin. When they become airborne, as they do in natural movement, anyone near that person *can* inhale them.
>
> Since asbestos is used in clutches and brakes, you may be exposing yourself and your loved ones to that risk.[572]

[571] Frank, *Medical and Public Health Approaches to Asbestos Disease*, Mount Sinai J. Med. 62(5) (1995).

After pointing out that "asbestos can cause fatal diseases years after exposure" and that "[a]sbestos is used in clutches and brakes," the bulletin explains the risks from working with asbestos brakes:

> You can't see these fibers in the air – some asbestos particles are so small that nearly 200 of them would have to be bundled together to equal the diameter of one human hair.
>
> These invisible particles are in brake dust and you can inhale them right through unapproved protective dust masks. . . .
>
> How dangerous is asbestos exposure? Asbestos fibers are often found at tumor sites in the lung, and exposure to asbestos causes deadly lung diseases. Among these are:
>
> ·Lung Cancer
>
> ·Mesothelioma: This deadly cancer is *100% fatal,* usually within one year of diagnosis - and asbestos is the major cause of this disease. No one knows how little it takes. Even *indirect* exposure is deadly. Wives, children, and pets of people who work with asbestos have died just from exposure to the clothes of the worker. This disease damages the lining of the chest and abdominal cavities.
>
> ·Other cancers . . .
>
> ·Asbestosis . . .[573]

304. Particularly telling about hazards of asbestos from brakes is what industry says outside of court. For example, Ford Motor Company ("Ford") and Bendix have consistently told their employees that asbestos from brakes poses a cancer risk. For example, Egilman (2009), commended Ford for several of its internal training programs, especially the visual communication via videotape. The following images were identified by Egilman (2009) as particularly helpful:

[572] DHHS (NIOSH) Publication 97-162, pages 179-80, *DANGER asbestos – Working on brakes? Think about this . . .* (1990).
[573] DHHS (NIOSH) Publication 97-162, pages 179-80, *DANGER asbestos – Working on brakes? Think about this . . .* (1990).

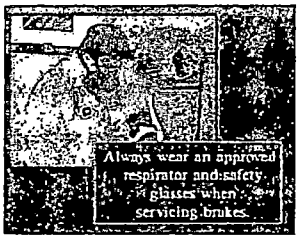

Figure 1—Ford's training video visually instructs workers to wear personal protective equipment while servicing brakes.

Figure 2—Ford's training video textually instructs workers to wear personal protective equipment while servicing brakes.

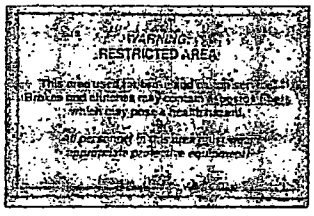

Figure 4—Ford's training video provides an example of a workplace warning of asbestos risk and requiring the use of personal protective equipment.

305. Despite having funded numerous Doubt Science publications downplaying the risks of asbestos from brakes discussed above, outside the litigation context,[574] Ford has clearly and repeatedly acknowledged that asbestos from brakes and clutches can cause mesothelioma and other cancers. For example, a 1994 Ford training materials for dealership mechanics included a book and videotaped presentation discussing the risk of inhaling asbestos from brakes, recommended wearing respirators even for inspecting brakes and stated:

> This friction material may also contain asbestos. While asbestos works well on brake shoes, it does not work well with the human body. Always wear an approved respirator and safety glasses when servicing brakes. Never blow brake dust with compressed air and always follow the safety procedures as described in the vehicle service manual.[575]

Egilman (2009) also details a more recent program adopted by Ford, General Motors and Chrysler, among others, known at the Coordinating Committee for Automotive Repair (CCAR) "Safety and Pollution Prevention" (SP/2) training program. The SP/2 program contained the following statements:

[574] Egilman, *Ford, General Motors, Chrysler, Asbestos and "Sane Appreciation of the Risks"*, Int. J. Occup. Environ. Health 15(1): 109-110 (2009).

[575] Egilman, *Ford, General Motors, Chrysler, Asbestos and "Sane Appreciation of the Risks"*, Int. J. Occup. Environ. Health 15(1): 109-110 (2009) (quoting DVD Transcription. Ford General Brakes Theory & Operational Video.).

Asbestos Dust and Fibers are Dangerous

Although asbestos is a very *useful* material and *inexpensive* to produce, there is a *dangerous downside*. If asbestos, and *particularly the fine dust* from wear, is not handled correctly during repair, the tiny asbestos fibers can *become airborne and can be inhaled*. Inhalation of too much dust and fibers can cause a disease called *asbestosis*. This disease can lead to other lung diseases, including mesothelioma and cancer.

. . .

Servicing Allows Asbestos to Float Free

During brake and clutch servicing, asbestos fibers are worn to sizes so small that they can easily float in the air. If you see a dust cloud during brake work with asbestos, *you are seeing clumps containing thousands of asbestos fibers.* Wiping with a wet rag or brush does little to prevent the scattering of asbestos. When the rag dries out, the fibers become free to float into the area again. . . . The mere act of banging a brake drum with a hammer can release quantities of brake dust floating into the air.

These statements are similar to those discussed by EPA in its videotape asbestos hazard training called "*Don't Blow It! Protecting Yourself from Asbestos in Brake Servicing*", in the EPA's Gold Book and NIOSH's recent guidance to mechanics. Not surprisingly, in 1987 in an internal memorandum, Ford recommended EPA's "*Don't Blow It!*" videotape for training brake mechanics regarding asbestos hazards, stating *Don't Blow It!* "covers the potential health hazards from asbestos exposure in brake dust and how to effectively control brake dust."[576]

306. Various manufacturers of asbestos containing brake systems reviewed the script for "*Don't Blow It*" and a final draft of the EPA's *Guidance for Preventing Asbestos Disease Among Auto Mechanics* (colloquially known as the "Gold Book") before they were released.[577] I am familiar with the videotape entitled "*Don't Blow It*" and believe it is an excellent training tool that provides an effective warning to mechanics about the well-studied hazards of working with asbestos brakes. "*Don't Blow It*" provides specific warnings about the diseases associated with asbestos brakes, including asbestosis, lung cancer and mesothelioma of the pleura and peritoneum. "*Don't Blow It*" also warns of the risks of mesothelioma to the families of mechanics. The videotaped warning in "*Don't Blow It*" is the type of visual and audio warning that catches the attention of the viewer and properly explains the gravity of the risk.

[576] Wabeke, Supervisor, Industrial Hygiene Section, Environmental Health Affairs, Employee Health Services, Ford Motor Company Memorandum re *Exposure of Brake Mechanics to Asbestos Fibers*, (February 20, 1987).

[577] Environmental Protection Agency ("EPA"), *Guidance for Preventing Asbestos Disease Among Auto Mechanics*, EPA-560-OPTS-86-002 (June 1986).

307. EPA's Gold Book also provides accurate, plain language information to mechanics about the hazards of asbestos. For example, it tells mechanics that "[f]riction materials, such as brake linings and clutches, often contain asbestos [and] [m]illions of asbestos fibers can be released during brake and clutch servicing." The Gold Book also points out that "[a]sbestos released into the air lingers around the garage long after a brake job is done and can be breathed in by everyone inside a garage, including customers [and that] [w]hile lowering exposure lowers the risk, there is no known safe level of exposure to asbestos below which health effects do not occur." [578] Importantly, the Gold Book points out that "[m]esothelioma is a type of fatal cancer of the lining of the chest or abdominal cavity. It can be caused by very low exposure asbestos. This cancer has occurred among brake mechanics, their wives and their children." [579] In addition, the Gold Book explains the important concept of latency:

> It usually takes 15 to 30 years or more for cancer or asbestos lung scarring to show up after exposure. (Scientists call this the latency period.) Until then, the victim often feels fine. This gives a false sense of security. For example, if one touches a hot stove, one gets burned right away. With asbestos, the damage isn't obvious until many years later. This false sense of security can easily lead the worker and/or supervisor to follow work practices which can cause harmful exposures, since they are not aware that disease may develop later.

Finally, the Gold Book explains work practices that can cause high level exposures, including compressed air blowout ("up to 16 million asbestos fibers in the cubic meter around the mechanic's face), liquid spray methods ("over a million fibers can be release near a mechanic's face") and cleanup with normal shop vacuum (about as much as compressed air blowout [16 million fiber/m^3]), light grinding of new brakes (4.8 million fibers) and beveling new linings (72 million fibers). According to EPA, use of a non-HEPA filtered vacuum can result in exposure to people 75 feet away from the mechanic.[580]

308. The National Toxicology Program, in the *Report on Carcinogens, Thirteenth Edition. Asbestos* CAS No. 1332-21-4 identified "brake repair and maintenance workers" as being among people with ongoing potential asbestos exposure in the United States.[581]

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[578] Environmental Protection Agency("EPA"), *Guidance for Preventing Asbestos Disease Among Auto Mechanics*, EPA-560-OPTS-86-002 (June 1986).
[579] Environmental Protection Agency("EPA"), *Guidance for Preventing Asbestos Disease Among Auto Mechanics*, EPA-560-OPTS-86-002 (June 1986) (citations omitted).
[580] Environmental Protection Agency ("EPA"), *Guidance for Preventing Asbestos Disease Among Auto Mechanics*, EPA-560-OPTS-86-002 (June 1986) (citations omitted).
[581] Department of Health and Human Services. National Toxicology Program. Report on Carcinogens, Thirteenth Edition. Asbestos CAS No. 1332-21-4; pp. 1-3.

309. Specifically, because the epidemiological and all of the other scientific evidence that chrysotile asbestos causes mesothelioma is so strong that the mainstream scientific community has concluded, and I am able to conclude, that a mesothelioma in a patient exposed to dust from chrysotile asbestos brakes or clutches (or any other source of chrysotile asbestos) was caused, in whole or in part, by that dust.

310. Until recently, there were no epidemiological studies of people exposed primarily to asbestos from brakes reporting a statistically significant increased risk for mesothelioma, but that was likely a reflection of the fact that few studies were designed to detect such a risk. Over the years, various experts hired by companies defending asbestos product liability cases arising from exposure to asbestos from automobile parts collected a long list of equivocal studies (often just a line-item of data extracted from a study that was not designed to test this hypothesis) and then claimed that these equivocal studies proved asbestos from brakes is somehow without risk for mesothelioma.[582] As explained by Freeman et al. (2012), the industry scientists violated "the maxim that lack of evidence of causation is not the same as evidence of lack of causation."[583] Freeman critically analyzed the industry position and explained the fallacy of relying upon poorly designed, under-powered analyses to reject causation:

> From this analysis we determined that a minimum sample of 845 cases (assuming one non-frequency matched control for each case) would be required to identify an OR=2.0 with 1–b=0.80 (power) and a=0.05. Even if all of the cases in the Goodman et al. review could be pooled, this would only account for 9.3% of the necessary cases for an adequate study of the influence of occupation in the entire automotive repair industry on the risk of mesothelioma. It is important to note that in relying on the data produced by Paustenbach et al. in their industry sponsored publications, we likely relied on an underestimated number of study subjects needed to accomplish the study goals set forth by Goodman et al. The methods used to detect fiber levels in the air underlying the data reported by Paustenbach et al. systematically miss the shorter (<5 microns in length) fibers that are commonly found in chrysotile containing brake dust samples, and thus underreport the sampled chrysotile fiber levels.
>
> It is reasonable to conclude that the "net" of evidence supporting a causal nexus between brake dust exposure and mesothelioma favors causation, and that the weak "strands" indicated by industry scientists as evidence to the contrary either do not exist or are greatly outweighed by the evidence to the contrary.

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[582] Goodman et al., *Mesothelioma and lung cancer among motor vehicle mechanics: a meta-analysis*. Ann. Occup. Hyg. 48(4): 309 – 26 (2004).

[583] Freeman et al., *Assessing specific causation of mesothelioma following exposure to chrysotile asbestos containing brake dust*. Int. J. Occup. Envir. Health 18:4 (2012).

Thus, the industry scientists ignore the mainstream requirement, as discussed by IARC[584] and others,[585] that "negative" epidemiology should only be relied upon where the studies were adequately powered to detect elevated risk. As Pearce et al. (2015)[586] recently explained,

> False negatives are more difficult to address, and perhaps they occur more frequently than false positives because of low statistical power, nondifferential misclassification of exposure and/or outcome, and incomplete follow-up, which tends to reduce the observed difference in risk between the exposed and nonexposed populations.[587]

Many of the problems with negative epidemiological studies discussed above render the studies cited by brake manufacturers unreliable for answering the question of whether asbestos from brakes poses a risk of mesothelioma. Pearce et al. (2015) also observed:

> Moreover, apparently conflicting results from epidemiologic studies do not necessarily indicate that some are false positive or false negative. This might, for example, reflect differences in levels of exposure or susceptibility to the effects of exposure (effect modification).

The 58 Scientists Brief eloquently described the problems of the asbestos brake industry model:

> Brake manufacturers and their consultants invent their own "scientific rule" demanding that a collection of approximately 18-22 "negative" friction studies and industry-financed meta-analyses thereof trumps all other evidence. But for a study or studies to be

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[584] IARC. Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012).

[585] Welch et al., *Asbestos exposure causes mesothelioma, but not this asbestos exposure: an amicus brief to the Michigan Supreme Court.* Int. J. Occup. Environ. Health. 13:318–327 (2007).

[586] Pearce et al., *IARC Monographs: 40 Years of Evaluating Carcinogenic Hazards to Humans*, Environ. Health Perspectives 123: 6 (June 2015). This article is authored by one hundred (100) scientists and researchers who support the approach to causation used by IARC. Many of these same scientists are among the 108 scientists who have expressed views on asbestos similar to mine.

[587] Citing Ahlbom, et al., *Interpretation of "negative studies" in occupational epidemiology*, Scand. J. Work Environ. Health 16:153–157 (1990); Blair, et al., *Epidemiology, public health, and the rhetoric of false positives*, Environ. Health Perspect. 117:1809–1813 (2009); Grandjean, *Non-precautionary aspects of toxicology*. Toxicol. Applied Pharmacology 207 (2 suppl):652–657 (2005); Rothman KJ, Greenland S, Lash TL. 2008. Modern Epidemiology. 3rd ed. Philadelphia: Lippincott Williams & Wilkins. (2008).

> truly "negative" in the sense hoped for by industry advocates, the study must be large enough to have sufficient "power" to detect an increase in risk, have reliable work histories to establish exposure or lack thereof, and be followed for a sufficiently long time to account for latency.[588] It is also highly preferable that the study be specifically designed to detect an increase in risk for the disease in question. The studies in questions fail on all of the above counts.[589]

Ironically, while in courtrooms the Product Defense "Doubt Scientists" often cite the "18 – 22 studies" as proof that there is no risk from asbestos in brakes, while in their published paper they deemed four of the studies to be "unreliable."[590] Furthermore, none of the authors of the epidemiologic studies relied upon by the product defense consultants actually conclude that there is no risk from asbestos exposure from brakes. Rather, the scientists who studied asbestos and brakes outside the courtroom (myself included), properly interpret these limited studies as not detecting an increased risk. However, it is significant that numerous scientists who have actually studied the risks of asbestos for people working in the automotive field have routinely reached the conclusion that "asbestos from brakes can and does cause mesothelioma." The product defense scientists routinely cite studies by Teschke et al. (1997) and Woitowitz et al. (1994) as supporting their belief that asbestos from brakes doesn't increase the risk of mesothelioma in people exposed.[591] But Dr. Hans Joachim-Woitowitz and Dr. Kay Teschke, have expressed the

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[588] Median latency since first exposure for mesothelioma is 38.4 years. *See* Reid et al., *Mesothelioma Risk after 40 Years since First Exposure to Asbestos: a Pooled Analysis,* 0 Thorax, 1 (2014).

[589] The 58 Scientists noted that "[a] comprehensive survey of the brake literature [was] not the intent of [their] paper. However, [the 58 Scientists] incorporate[d] by reference: Lemen, *Asbestos in Brakes, supra.*; Egilman et al., *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability,* 11 Int. J. Occup. Environ. Health 360 (2005); and Welch, *Asbestos Exposure Causes Mesothelioma, But Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, 13* Int. J. Occup. Environ. Health 318 (2007)." 58 Scientists, p. 20, note 48.

[590] Goodman et al., *Mesothelioma and lung cancer among motor vehicle mechanics: a meta-analysis*. Ann. Occup. Hyg. 48(4): 309 – 26 (2004) (stating that four studies, Coggon et al., *Differences in occupational mortality from pleural cancer, peritoneal cancer, and asbestosis,* Occup Environ Med; 52: 775–7 (1995), Hodgson et al., *Mesothelioma mortality in Britain: patterns by birth cohort and occupation*, Ann Occup Hyg; 41(suppl 1): 129–33 (1997), Milham et al., *Occupational mortality in Washington State 1950–1999,* Washington State Department of Health (2001) and NIOSH, personal communication, (2002), "were considered unreliable").

[591] Goodman et al., (2004) relies upon Teschke et al., *Mesothelioma surveillance to locate sources of exposure to asbestos*, Can J Public Health 88: 163–8 (1997) and Woitowitz et al., *Mesothelioma among car mechanics?*, Ann Occup Hyg; 38: 635–8 (1994). I participated, as a clinician, in gathering data for original research on the health of auto mechanics. The results of the study were published in Lorimer et al., *Asbestos exposure of brake repair workers in the United States*. 43(3) Mt. Sinai J. Med. 207, 207-18 (1976) and updated by Nicholson et al., *Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers*

opinion that asbestos from brakes can and does cause mesothelioma in people so exposed. Dr. Woitowitz is one of the supporters of the 58 Scientists Brief and Dr. Teschke joined me and fifty (50) other scientists in signing Welch (2007).

311. In response to Garabrant, et al. (2015), Dr. Teschke explained in more detail how the industry-aligned litigation consultants improperly reframed the critical scientific question that must be answered when looking to see a particular mechanic has a mesothelioma caused by asbestos; while the proper question is whether the mechanic was exposed to asbestos from working with auto parts sufficient to cause mesothelioma, the industry's litigation scientists ask whether being a vehicle mechanic causes mesothelioma. Put succinctly, "[a] job cannot cause disease, its exposures may."[592] Dr. Teschke explained:

> The issue of interest is whether chrysotile asbestos causes mesothelioma. It may be reasonable to study jobs or tasks to investigate this question, but note that the jobs or tasks are being used as surrogates of chrysotile exposure. It is important to consider whether they are good surrogates. Some occupations are synonymous with extensive exposure to certain agents. For example, it would be a rare wood furniture maker who did not have daily high exposure to hard wood dust and an exceptional asbestos insulator without daily high exposure to amphibole asbestos. For such jobs, the association between the occupation and a disease may be virtually equivalent to the association between its predominant exposure and that disease. Many jobs have much more variable exposures. For example, some nurses are regularly exposed to anesthetic gases and others are rarely or never exposed. Examining the relationship between nursing and spontaneous abortion is not equivalent to examining exposure to anesthetic gases and that outcome. Because exposures are so variable within the occupation, any true 'exposure–response' relationship will be attenuated or not observable when examined instead via an occupation–response' analysis.
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> . . .
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> In the case of vehicle mechanics, brake repair work is not

Occupationally Exposed to Asbestos, NIOSH Contract 210-77-0119 (1984). Two of the contributors to Nicholson et al., (1984), Henry Anderson and Kenneth Rosenman, joined me in signing Welch (2007). More recently, Dr. Rosenman also joined the 58 Scientists in supporting my opinion that asbestos from brakes can and does cause mesothelioma in people exposed to asbestos from brakes. While everyone is entitled to their scientific opinion, numerous investigators who have done original research on asbestos brakes have concluded that they pose a risk of asbestos disease. The only people who express doubt about the risks of disease posed by asbestos brakes appear to be expert witnesses for asbestos defendants in litigation.

[592] Teschke, *Thinking about Occupation–Response and Exposure–Response Relationships: Vehicle Mechanics, Chrysotile, and Mesothelioma*, Ann. Occup. Hygiene 60:4 528-530 (2016).

consistently performed. Three studies (Woitowitz and Rödelsperger, 1994; Teschke *et al.*, 1997; Rake *et al.*, 2009) cited in the meta-analysis (Garabrant *et al.*, 2016) included information on subjects' brake repair work separately from vehicle mechanic occupation; at least 34% of the vehicle mechanics reported no brake repair work. Yet of the 16 studies included in the meta-analysis (Garabrant *et al.*, 2016), 12 examined only the vehicle mechanic job or even broader job categories such as garage workers, auto repair and related services, and auto engineers. It is reasonable to expect that many in these categories [only] had chrysotile exposures similar to background levels in the population.

What about brake repair work itself? Four studies (Woitowitz and Rödelsperger, 1994; Teschke *et al.*, 1997; Hessel *et al.*, 2004; Rake *et al.*, 2009) in the meta-analysis reported on brake repair. The era of potentially etiologic exposure in these studies was dominantly long prior to 1980, a period when vehicle mechanics largely worked in gas stations and brake work was one possible task of many within the job. The frequency of exposure was likely to be intermittent, though it is possible that rare subjects may have worked in specialized brake shops and done this task repeatedly every day all day for most of their careers.

...

Interpretation of the results of the studies included in the meta-analysis (Garabrant *et al.*, 2016) should acknowledge the likelihood that many vehicle mechanics had done no brake repair work and that of those who had, most would have done so infrequently as part of a broad array of activities. This means that both the vehicle mechanic occupation and the brake repair task are unlikely to be good surrogates of chrysotile exposure. Associations between the occupation or task and mesothelioma were likely attenuated by subjects with background or low exposure levels.

It may be helpful to think of this in another way. Studies of chrysotile miners and textile workers have found elevated risks of mesothelioma (International Agency for Research on Cancer, 2012), whereas studies of vehicle mechanics and brake repair workers typically have not (Garabrant *et al.*, 2016). Does this mean that vehicle mechanics and brake repair workers are somehow immune to the effects of asbestos, that they are especially resistant, superhuman? No, they simply work in a job that has very varied exposures, so detecting occupation–disease relationships is difficult. In other words, the effects of chrysotile are not properly

> assessed via the surrogate measures of exposure 'vehicle mechanic' or 'brake repair'.[593]

312. In response to Teschke (2016), the brake industry litigation consultants argued that Teschke misunderstood the goal of their publication:

> Dr. Teschke first asks whether studies of motor vehicle mechanics are the appropriate setting to examine the risks of mesothelioma from chrysotile exposure. This is not the question that our meta-analysis set out to answer. Rather, we sought to answer the question of whether motor vehicle mechanics and brake repair workers were at increased risk of mesothelioma.[594]
>
> As made clear above and below, when looking at individual causality, one must look at the *evidence of exposure to asbestos* to know whether work in a job category – be it as a "vehicle mechanic" or "insulator" caused a particular patient's disease.

313. In response to the back-and-forth letters by Teschke and Garabrant, et al., the Annals of Occupational Hygiene published an editorial essentially endorsing Teschke's approach to evaluating the risk to an individual vehicle mechanic based on the equivocal epidemiological studies looking at people who may have been exposed to asbestos as mechanics:

> However, there is a question in how one should interpret this result. As Teschke (2016) pointed out in her letter to the editor, the issue of interest is whether chrysotile asbestos exposure among mechanics causes mesothelioma. Mechanics perform a variety of tasks and not all mechanics will be involved in [brake] repair. The ones that do in turn may only do so for part of the time. Using broad categories of job titles (e.g. garage workers, auto repair and related services, and auto engineers) are therefore likely poor proxies of chrysotile asbestos exposure. As such although the conclusion of the paper is correct in that no elevated risks are found among motor vehicle mechanics as a whole this cannot be equated to mean that there is no risk of mesothelioma among mechanics that were involved in [brake] repair and that had potential chrysotile exposure. In their answer to the letter to the editor, Garabrant *et al.* (2016b) indicated that the results may indicate that motor vehicle mechanics and brake workers are perhaps not at increased risk of mesothelioma as they may not have sufficient exposure to observe an increased risk. Again, this

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[593] Teschke, *Thinking about Occupation–Response and Exposure–Response Relationships: Vehicle Mechanics, Chrysotile, and Mesothelioma*, Ann. Occup. Hygiene 60:4 528-530 (2016).
[594] Garabrant, et al., *Response to letter to the editor: mesothelioma among motor vehicle mechanics: an updated review and meta-analysis*. Ann. Occup. Hygiene. (2016)

> statement is correct at the job-title level but may not hold for specific subgroups and/or individuals that may have been higher exposed.[595]

Vermeulen recognized that exposure misclassification may have biased the studies toward the null and explained "we would not, based on the absence of an association between working as a motor vehicle mechanic and mesothelioma, conclude that there is no association between chrysotile exposure and mesothelioma." [596] Vermeulen commented on the limited usefulness of the existing epidemiological studies of vehicle mechanics:

> These analyses would suggest that motor vehicle mechanics who are exposed to chrysotile would be at increased risk for mesothelioma. Therefore, analyses on job titles that constitute known carcinogenic exposures are only useful as to investigate the overall risk as to judge the likelihood and level of exposures within the broad job-category. However, this is a rather insensitive tool, as it depends strongly on the validity and power of the epidemiological study.
>
> . . .
>
> [Exposures to brake dust,] if identifiable as asbestos, have to be assumed to carry cancer risk even in the absence of epidemiologic evidence.[597]

I agree with Vermeulen's conclusion:

> So when are risk analyses on job titles informative? Analyses on job titles can be informative in the situation where job titles entail unknown carcinogens or a mix of known and unknown carcinogens. In these situations, job-title-based analyses can provide information on the role of new or suspected occupational carcinogens. However, as exposures vary considerably within job titles depending on their job activities, they can at best be regarded as crude measures of exposure. *As such, positive results may provide new information; however, null results cannot be used to*

[595] Vermeulen, *Editorial - When are Risk Analyses on Job Titles Informative?*, Ann. Occup. Hygiene 60:8, 913 – 915 (2016)
[596] Vermeulen, *Editorial - When are Risk Analyses on Job Titles Informative?*, Ann. Occup. Hygiene 60:8, 913 – 915 (2016)
[597] Vermeulen, *Editorial - When are Risk Analyses on Job Titles Informative?*, Ann. Occup. Hygiene 60:8, 913 – 915 (2016)

exclude that there is no effect of a known carcinogen within that occupation. [598]

314. The asbestos brake industry also downplays several studies demonstrating positive correlation between doing vehicle mechanic work and elevated risk of mesothelioma. Soeberg, et al. (2016) reported that vehicle mechanics had elevated lifetime risk of mesothelioma (0.7% lifetime excess risk).[599] Roelofs et al. (2013) reported a statistically significant increased risk (Standardized Morbidity Odds Ratio (SMOR) = 2.1 (95% CI = 1.1 – 4.0)) of mesothelioma for "automobile mechanics."[600] People working in "automotive repair and related services" also had statistically significant increased risk of 2.2 (95% CI = 1.2 – 3.9). When considered with all the evidence that asbestos causes human mesothelioma, this study is simply further confirmation of the mainstream position that asbestos from any source can cause mesothelioma in those exposed. While these data are accurate, other potential sources of asbestos may be unknown. These finding are consistent with the findings of Welch, et al. (2007).

315. Despite the fact that some studies fail to show statistically significant increased for people who may have been exposed to brakes, studies show that some mechanics experience exposures that are many orders of magnitude above the miniscule amount of asbestos in the ambient air. For example, the article by Kauppinen et. al. (1987)[601] found an average exposure level of 1.5 fibers per cubic centimeter (f/cc) or 1,500,000 fibers per cubic meter when blowing out brake wear debris. Comparing the exposure data from Kauppinen et al. to ambient exposure levels of 0.00001 f/cc or 10 fibers per cubic meter in rural areas and 100 fibers per cubic meter in urban areas, it is clear that can experience exposures 15,000 – 150,000 times ambient exposure.

316. Recently the British Health and Safety Executive (HSE) supported a relatively large epidemiological study of mesothelioma risk in Great Britain. Peto et al. (2010)[602] found that motor mechanics born between 1925 and 1930 and exposed before 30 years of age had a statistically significant increased risk of mesothelioma (Odds Ratio of 26.3, 95% CI 2.1 – 259.9). These motor mechanics had the highest risk of any of the "low risk industrial" groups of that age.

317. An analysis of mortality in bus drivers and bus maintenance workers in Genoa, Italy demonstrated significant excess deaths (SMR 3.67) from pleural mesothelioma when

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[598] Vermeulen, *Editorial - When are Risk Analyses on Job Titles Informative?*, Ann. Occup. Hygiene 60:8, 913 – 915 (2016) (emphasis added).

[599] Soeberg, et al., *Malignant mesothelioma in Australia 2015: Current incidence and asbestos exposure trends*, J. Tox. Environ. Health, Part B, 19:5-6, 173-179 (2016).

[600] Roelofs et al., *Mesothelioma and Employment in Massachusetts: Analysis of Cancer Registry Data 1988 – 2003*. Am. J. Indus. Med. (Epub ahead of print 2013).

[601] Kauppinen et al., *Exposure to asbestos during brake maintenance of automotive vehicles by different methods*, Am. Ind. Hyg. Assoc. J. 48(5): 499-504 (1987).

[602] Peto et al., *Occupational, domestic and environmental mesothelioma risks in Britain -- A case-control study*. RR696 (2009).

compared to Italian males.[603] As the 58 Scientists Brief explained, in support of my opinion that asbestos in brakes can and does cause mesothelioma, "[t]here was not an increased risk when compared to the surrounding population, because the surrounding region contained several shipyards that historically used asbestos. So the choice of comparison population can influence the findings regarding mesothelioma in an urban or working population. This needs to be taken into account when interpreting study findings or individual exposure histories." This phenomenon has been long recognized in the world of asbestos epidemiology, appearing no later than 1962 as criticism of the asbestos-industry funded study by Braun and Truan in which the authors changed the comparison population from the rural area around the Quebec asbestos mines to include the urban areas of Montreal and Quebec city, which had higher rates of lung cancer, to disguise the increased incidence of lung cancer in asbestos miners.[604]

318. In an analysis for the World Trade Organization, Dr. Douglas Henderson evaluated the risk of exposure to asbestos from brakes (as compared to the estimated background rate of 1-2 mesotheliomas per million person-years) and found mechanics had approximately 10-fold risk of mesothelioma.[605]

319. In litigation, industry scientists often use a misleading approach to causation that involves redefining the scientific hypothesis to help insure the results of their patrons. For example, David Garabrant recently explained the update of the Goodman et al. (2004) meta-analysis seeks to test the hypothesis of whether "motor vehicle repair work causes pleural mesothelioma" (Garabrant Aff. at para 2) when the real issue is whether asbestos exposure causes mesothelioma. Dr. Garabrant then unscientifically asserts that the only evidence that can be considered in answering his question is human epidemiological studies that have information about people who may have been exposed to asbestos from brakes. By posing this inappropriate hypothesis, Dr. Garabrant essentially guarantees the results desired by his corporate clients because it is asbestos from brakes, not the job title "motor vehicle repair work," that increases a person's risk of mesothelioma. It is for this reason that many of the authors of the purported "vehicle mechanic" epidemiological studies, given the totality of the sort of scientific evidence discussed below, have concluded that asbestos from brakes can cause mesothelioma. As Dr. Kay Teschke, the author of one study relied upon by Dr. Garabrant, recently explained:

> Studies of chrysotile miners and textile workers have found elevated risks of mesothelioma (International Agency for Research

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[603] Merlo, et al., *A Historical Mortality Study Among Bus Drivers and Bus Maintenance Workers Exposed to Urban Air Pollutants in the City of Genoa, Italy,* 67 Occup. Environ. Med. 611 (2010).

[604] Braun et al., *An Epidemiological Study of Lung Cancer in Asbestos Miners*, Archives of Indus. Health, 17:634-53 (1958). Hueper at the National Cancer Institute dismissed this brazen manipulation as "statistical acrobatics." Hueper, *Part I. Environmental and occupational cancer hazards*, Clin. Pharmacology, 3:6 776-806 (1962).

[605] World Trade Organization. European Communities – Measures Affecting Asbestos and Asbestos – Containing Products. Report No.: WT/DS135/R page 303 (2000).

> on Cancer, 2012), whereas studies of vehicle mechanics and brake repair workers typically have not (Garabrant *et al.*, 2015). Does this mean that vehicle mechanics and brake repair workers are somehow immune to the effects of asbestos, that they are especially resistant, superhuman? No, they simply work in a job that has very varied exposures, so detecting occupation–disease relationships is difficult. In other words, the effects of chrysotile are not properly assessed via the surrogate measures of exposure 'vehicle mechanic' or 'brake repair'.[606]

320. It is important to recognize that Lorimer et al. (1976) established that only 61 of 90 automotive maintenance workers did routine brake work.[607] A later study of vehicle maintenance workers in New York, Nicholson et al. (1984), further confirmed the highly variable work activities undertaken by vehicle mechanics, finding 46 of 172 (27%) vehicle mechanics never did brake work.[608] Table 60 (page 94) from Nicholson et al. (1984) is reproduced below:

Table 60

Distribution of frequency of brakework in U. A. W. Local 259 shops from personal estimates

Number of brake jobs done	Number of men	Percentage of men
1/day or more	24	14
1-4 /week	71	41
1 -5/month	27	16
less than 1/month	4	2
no brake work (engine mechanics, parts personnel, etc.)	46	27
Totals	172	100

[606] Teschke, Letter to the Editor, *Thinking about Occupation–Response and Exposure–Response Relationships: Vehicle Mechanics, Chrysotile, and Mesothelioma*, Ann. Occup. Hyg. 60 (4): 528-530 (2016). This article directly addresses the misuse of her findings by Ford Motor Company's experts in litigation and specifically references Garabrant et al. *Mesothelioma among motor vehicle mechanics: an updated review and meta-analysis, Ann. Occup. Hyg.*; 60: 8–26 (2015).

[607] Lorimer et al., *Asbestos exposure of brake repair workers in the United States.* 43(3) Mt. Sinai J. Med. 207, 207-18 (1976).

[608] Nicholson et al., *Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos*, NIOSH Contract 210-77-0119 (1984).

Because many vehicle mechanics did not work with brakes (or did so only sporadically), as discussed by Teschke (2016), studies of vehicle mechanics likely underestimate the real risk from working with asbestos brakes.

321. Product defense consultants, working for lawyers for Ford, General Motors and Chrysler, relied on part of a set of data provided by NIOSH to support a meta-analysis (Goodman et al. (2004)) written to help in lawsuit defense. Given the fact that the publication was written to help the auto companies in asbestos litigation, it was not surprising that, Goodman et al. omitted the data showing an elevated risk for black male mechanics.

322. Because many occupations, including mechanics, are exposed to asbestos, the only practical known cause of mesothelioma, it is no wonder the mainstream scientific and medical community concludes "auto brake mechanics are known to be especially at risk of developing asbestos-related diseases."[609] This conclusion is buttressed by numerous other studies of "mechanics" that demonstrate increased incidence of other asbestos related diseases known to occur at exposure levels sufficient or well in excess of the exposure levels needed to cause mesothelioma.[610]

323. Brake mechanics are known to be especially at risk of developing asbestos-related diseases because some brake mechanics had heavy exposures to asbestos. For example, Loggie et al. (2001) reported two cases of peritoneal mesothelioma in "diesel mechanics with occupational exposure to asbestos" from brake linings.[611] Recent studies in Colombia, where current brake repair practices often lead to uncontrolled exposure to

[609] Ontario Ministry of Labour, Alert: Asbestos Hazard in Vehicle Brake Repair. (Issued April 10, 2013).

[610] Marcus et al., *Asbestos-associated lung effects in car mechanics*, Scand J Work Environ Health 13:252-254 (1987) (plaques), Ameille et al., *Asbestos-Related Diseases in Auto Mechanics*. Ann. Occup. Hyg. 56(1) 55 – 60 (2012). (plaques), Roggli et al., *Malignant Mesothelioma and Occupational Exposure to Asbestos: A Clinicopathological Correlation of 1445 Cases*, Ultrastructural Pathology, 26:55 – 65 (2002) (plaques); Järvholm et al., *Asbestos associated tumours in car mechanics*, Br J Ind Med; 45: 645–6 (1988). Leigh, *Occupations, cigarette smoking, and lung cancer in the epidemiological follow-up to the NHANES I and the California Occupational Mortality Study*, Bull N. Y. Acad. Med. 73: 370–97 (1996); Burns et al., *The occupational cancer incidence surveillance study (OCISS): risk of lung cancer by usual occupation and industry in the Detroit metropolitan area*, Am. J. Ind. Med. 19: 655–71 (1991). Goodman et al., *Mesothelioma and lung cancer among motor vehicle mechanics: a meta-analysis*. Ann. Occup. Hyg. 48(4): 309 – 26 (2004) (statistically significant increased risk of lung cancer in automotive field despite substantial potential for inclusion of unexposed workers in studies); Lorimer et al., *Asbestos exposure of brake repair workers in the United States*. 43(3) Mt. Sinai J. Med. 207, 207-18 (1976) (fibrosis), Nicholson et al., *Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos*, NIOSH Contract 210-77-0119 (1984) (statistically significant asbestos related fibrosis on x-ray when controlled for duration of work), Dalqvist (asbestos related breathing problems).

[611] Loggie et al., *Prospective Trial for the Treatment of Malignant Peritoneal Mesothelioma*, The Am. Surgeon 67:999-1003 (2001).

asbestos using methods common before the advent of the OSHA regulations, have demonstrated that heavy exposures can occur doing brake work.[612]

The following table provides a sampling of the sorts of asbestos exposures experienced by mechanics using asbestos-containing automotive parts.

[612] Cely-Garcia et al., *Personal Exposures to Asbestos Fibers During Brake Maintenance of Passenger Vehicles*, Ann. Occup. Hyg. 56(9): 985-99 (2012) (heavy exposure from uncontrolled work with automotive brakes); Cely-Garcia et al., *Personal exposure to asbestos and respiratory health of heavy vehicle brake mechanics*, J. Expo. Sci. Environ. Epidemiol. 25, 26–36 (2015) (heavy exposure from uncontrolled work with heavy vehicle brakes); Salazar et al., *Asbestos Exposure among Transmission Mechanics in Automotive Repair Shops*, Ann. Occup. Hyg., 1–15 (2014) (Heavy asbestos exposure from asbestos clutches).

Author	Year	Activity	Exposures Reported
Lee	1970	Blow out	3-5 f/cm^3
Boillat & Lob	1973	Brake work undefined	0.3-29.2 f/cc
Castleman & Ziem	1985	Damp rag	High: 2.6 f/cc; TWA 0.28 f/cc
		Squirt bottle	High: 0.54 f/cc; TWA 0.21 f/cc
		Stoddard Solvent	High 0.68 f/cc; TWA <0.1 f/cc
		Dry rag	High 0.81 f/cc; TWA 0.2 f/cc
		Brake washer	High 1.1 f/cc
Hatch	1970	Compressed Air	Fibers >5 um: 2.1-8.2; 10 min. avg: 0.8
Rödelsperger	1986	Passenger car (various operations)	Mean: 3.8-4.7 f/cc
		Truck (various operations)	Mean: 4.4-9.9 f/cc
Kauppinen & Korhonen	1987	Truck (various operations)	<0.1-125 f/cc; TWA: 0.1-0.2 f/cc
		Grinding	7 f/cc
Hickish	1968	Auto blow out	Peak Exposure: 7.09 f/cc
Hickish	1968	Auto brake work, various	TWA: 1.57-2.55 f/cc
Clark	1976	Auto disc brake change	0.2-1.9 f/cc
Hatfield & Longo	1998	Bendix Chrysler (filling and cleaning)	8.53-14.57 f/cc
Hatfield & Longo	n.d.	Bendix Ford (filling and cleaning)	5.47-12.67 f/cc
Hatfield & Longo	2000	Sweeping & cleaning brake shop	Personal samples: 7.5-8.8 f/cc; Area Samples: 2.0-2.4 f/cc
Hatfield, Longo & Newton	2000	Grinding	4.83-12.51 f/cc
Hatfield, Longo & Newton	2000	Hand grinding	12.57-21.43 f/cc
Hatfield, Newton & Longo	2001	Hand sanding	0.5-0.96 f/cc
Rohl et al.	1977	Blowing dust	6.6-29.4 f/cc
		Beveling	23.7-72.0 f/cc
Osborn	1934	Grinding	17 mppcf
Roberts & Zumwalde	1982	Compressed Air	0.14-15.0 f/cc
Lloyd	1975	Servicing brakes	3.75-37.3 f/cc
Longo, Mount & Hatfield	2004	Hand sanding and grinding and other operations	19.7-35.7 f/cc
Salazar et al.	2014	Transmission repair work	TWA: 0.006-0.193 f/cm^{-3}
		Transmission repair work	TWA 8 hr 0.011-0.093 f/cm^{-3}; 0.065-0.603 f/cm
Cely-Garcia et al.	2012	Brake replacement jobs (buses & trucks)	TWA: 0.003-0.157 f/cm^3
		Brake replacement & manipulation - riveting (buses & trucks)	TWA: 0.000-0.645 f/cm^3; TEM Exposure 0.000-2.084 f/cm^3
		All brake replacement activities	TWA: 0.007-0.114 f/cm^3 (8 hour mean); 0.091-0.324 f/cm^3 (short term mean)
Cely-Garcia et al.	2015	All day brake work	TEM: <0.002-7.630 f/cm^{-3}; PCME: 0.002-0.163 f/cm^{-3}
		Brake work short term	TEM 0.015-8.835 f/cm^{-3}; PCME 0.031-0.532 f/cm^{-3}
		Brake work	TWA: TEM 0.01-3.49 f/cm^{-3}; PCME 0.002-0.07 f/cm^{-3}
		Summary of personal short term concentrations and activities	TEM 0.279-8.835 f/cm^{-3}; PCME 0.005-0.532 f/cm^{-3}

324. Garabrant et al. (2016)[613] updates the industry-funded Goodman et al. (2004) paper, adding new studies and devising a revised scoring system for the meta-analyzed studies. The authors of several of the studies reviewed by Garabrant et al. (2015) have concluded that asbestos exposure from brakes can and does cause mesothelioma. For example, Dr. Kay Teschke, author of Teschke et al. (1997), joined me in authoring Welch et al. (2007) that concluded that asbestos from brakes can and does cause mesothelioma. Dr. Hans-Joachim Woitowitz (author of Woitowitz et al. (1994)), Dr. Dario Consonni (author of Merlo et al. (2010)), Dr. Richard Clapp (author of Roelofs et al. (2013)) were also members of the 58 Scientists who supported my opinions on the ability of asbestos from Ford Motor Company brakes to contribute to causing mesothelioma. In addition, Laura Van den Borre (author of Van den Borre et al. (2015)) has stated:

> The extensive asbestos use in the automotive industry and the strong causal relation between asbestos and mesothelioma are well-established in international literature. I believe few people would doubt a relation between working in an automotive industry and developing mesothelioma. Our results do not show significant excess in asbestos-related mortality for the 1991 automotive worker cohort within the given time frame. That does not mean automotive workers are safe from asbestos exposure.[614]

The epidemiological studies relied on by Garabrant et al. (2015) are certainly severely limited, as discussed by Egilman et al. (2005), Lemen (2004) and Welch et al. (2009).[615] As Welch et al. (2009) pointed out,

> Goodman et al. (2004) included fewer than 200 cases or controls whose occupation was specifically noted as vehicle mechanic, garage worker, automobile repair, or motor vehicle repair. . . . For the 7 studies included in the meta-analysis, 4 had nonspecific asbestos exposure. Exposure assessment in this group of studies is one of the more significant limitations that must be considered in interpretation of the results. If workers without asbestos exposure were classified as exposed, misclassification will bias toward the null.

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[613] Garabrant et al. *Mesothelioma among motor vehicle mechanics: an updated review and meta-analysis, Ann. Occup. Hyg.*; 60: 8–26 (2015).

[614] Van den Borre - Personal Communication from Laura Van den Borre to Jonathan Ruckdeschel, June 25, 2015.

[615] Egilman et al., *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability,* 11 Int. J. Occup. Environ. Health 360 (2005); Lemen, *Asbestos in brakes: exposure and risk of disease.* Am. J. Ind. Med. 45(3):229-237 (2004); Welch et al., *Research on Mesothelioma from Brake Exposure: Corporate Influence Remains a Relevant Concern,* Int. J. Occup. Environ. Health 15:2 234-238 (2009).

Thirty (30) physicians and scientists and I joined Welch, et al. (2009) in a letter to the editor to explain the weaknesses in the Goodman et al. (2004) approach:

> The occupation of "vehicle mechanic" in these studies includes individuals with no exposure to asbestos during brake work, as well as individuals with exposures to asbestos in a range of other occupations including shipyard work and insulation. The misclassification of exposure and the confounding by asbestos exposure in other occupations means this group of case control studies cannot provide useful information on the risk of mesothelioma among workers engaged in brake installation and repair.
>
> Brake mechanics, as an occupational group, are not conducive to study in the same way as construction workers or asbestos textile workers. The large asbestos disease studies of the 1960s through 1980s examined disease or mortality risk among groups of workers who were either employed in the same workplace over a number of years or belonged to the same union and performed similar jobs. Auto mechanics who perform brake repair are often employed in small non-union shops. They perform many tasks in addition to brake repair, and the amount of time spent on brake repair varies from mechanic to mechanic and from shop to shop. These variable and mixed exposures, combined with the difficulty of even identifying these workers and recruiting them for a longitudinal study, makes undertaking a cohort study of brake workers a formidable, maybe even impossible, task. There simply is no good occupation-specific epidemiology regarding this large and diverse cohort of workers.

325. Egilman et al. (2005) explains other limitations of the epidemiology relied upon by Dr. Garabrant:

> The Woitowitz and Rödelsperger study focused solely on the issue of a relationship between automobile mechanic work and mesothelioma. Part of this "study is of no value in determining the relationship between any asbestos exposure and mesothelioma because the authors used lung cancer patients as controls. Exposure status is likely to be similar among the control group and the cases, which would lead to little discernable difference in risk between the two groups, despite any real association that may exist between brake work and mesothelioma. Since asbestos exposure is a well-known risk factor for lung cancer, any mesothelioma case-control study with lung cancer patients as the control group will almost certainly produce results at or below the null. This study

demonstrates this effect well, since the researchers used "population controls" as well as lung cancer patients as controls. The odds ratio (OR) for "hospital controls" was 0.75, almost half that for population controls, 1.32. Rather than report both results, Wong presented an average OR of 0.87. Goodman et al. published both ORs but included only the lower average in their meta-analysis.

McDonald and McDonald conducted a case-control study, published in 1980, examining mesothelioma cases from Canada and the United States. Like the Woitowitz and Rödelsperger study, this study relied on an inappropriate control group; control subjects were patients in cases diagnosed by pathologists in which "pulmonary metastases were present from a non-pulmonary malignant tumor." For example, the control group may have included patients who had died from mesothelioma or other asbestos-induced malignancies such as laryngeal or colon cancer. The choice of such controls biased results towards the null. In addition, the study reviewed occupational status only ten years prior to death. Since mesothelioma has a minimum latency period of about ten years, this study did not necessarily address any subject's most relevant exposures. McDonald et al. subsequently compared lung-fiber burdens between control and mesothelioma patients and found no statistically significant difference between the amounts of chrysotile, amosite or crocidolite. This is further evidence that controls were not adequately chosen, since they were clearly exposed to as much asbestos as the mesothelioma patients. This misclassification biases the results to the null.

326. Despite the existence of numerous positive epidemiological studies suggesting that people who may be exposed to asbestos in a garage environment have elevated risk of mesothelioma, lung cancer and non-malignant asbestos-related diseases, industry-aligned scientists ignore the requirement that scientific conclusions must be drawn from a critical analysis of the data, as described by IARC:

> When several epidemiological studies show little or no indication of an association between an exposure and cancer, a judgment may be made that, in the aggregate, they show evidence of lack of carcinogenicity. Such a judgment requires first that the studies meet, to a sufficient degree, the standards of design and analysis described above. Specifically, the possibility that bias, confounding or misclassification of exposure or outcome could explain the observed results should be considered and excluded with reasonable certainty. In addition, all studies that are judged to be methodologically sound should (a) be consistent with an estimate of effect of unity for any observed level of exposure, (b) when considered together, provide a pooled estimate of relative

> risk that is at or near to unity, and (c) have a narrow confidence interval, due to sufficient population size. Moreover, no individual study nor the pooled results of all the studies should show any consistent tendency that the relative risk of cancer increases with increasing level of exposure. It is important to note that evidence of lack of carcinogenicity obtained from several epidemiological studies can apply only to the type(s) of cancer studied, to the dose levels reported, and to the intervals between first exposure and disease onset observed in these studies. Experience with human cancer indicates that the period from first exposure to the development of clinical cancer is sometimes longer than 20 years; latent periods substantially shorter than 30 years cannot provide evidence for lack of carcinogenicity.[616]

Thus, the industry-sponsored, litigation defense publication, Goodman et al. (2004) and the recent update, Garabrant et al. (2015) reach conclusions while ignoring the multiple problems in the studies they claim support their conclusions that working as an automobile mechanic does not put people exposed to asbestos brakes at risk of asbestos disease. The industry-aligned scientists and physicians ignore the lack of statistical power in the small studies, ignore exposure misclassification (e.g. Teschke et al. (1997) which compares vehicle mechanics (some unknown portion of whom might have done brake and clutch work) to possibly heavily exposed controls, practically guaranteeing the study would not find elevated risk), and ignores important design flaws in the studies such as failure to account for latency and lack of information of work practices (for example, the studies do not indicate whether cases used industrial hygiene methods to control asbestos dust and the studies likely include people who rarely, if ever, did brake work).

327. Imbernon et al. (2005) appears to have recognized that it is exposure to asbestos, not the product type, that causes mesothelioma and lung cancer in mechanics:

> The most realistic scenario hypothesizes that all automobile mechanics were exposed to asbestos, that the exposure levels ranged from 0.06 and 0.25 fibers/liter per week for the period before 1997, and between 0.01 and 0.06 fibers/liter per week afterwards until 2010.
>
> Results: According to this scenario, the number of lifelong cancer deaths (lung and pleura) induced by asbestos exposure in this population is estimated at 602 "unavoidable" cases, due to

[616] IARC. Preamble to Monograph 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite and Anthophyllite), Lyon: International Agency for Research on Cancer (2012).

> exposure experienced before 2003; 43 other cases will occur if asbestos is not removed from existing automobiles.[617]

328. Cely-Garcia (2012) found high exposures in doing automotive brake work:

> Personal asbestos concentrations based on transmission electron microscopy counts were extremely high, ranging from 0.006 to 3.493 f cm−3 for 8-h TWA and from 0.015 to 8.835 f cm−3 for 30-min samples. All asbestos fibers detected were chrysotile. Cleaning facilities and grinding linings resulted in the highest asbestos exposures based on transmission electron microscopy counts.

329. Cely-Garcia (2012) pointed out the limitations of much of the existing literature regarding exposure to asbestos from brake work:

> Although several recent studies of brake mechanics in developed countries have found asbestos concentrations in compliance with OSHA standards (Hickish and Knight, 1970; Yeung *et al.*, 1999; Weir *et al.*, 2001; Blake *et al.*, 2003, 2006; Paustenbach *et al.*, 2003, 2004), the facilities involved in those studies did not perform the extensive manipulation of brake linings observed in this study. In fact, the tasks done by the mechanics described in those studies appear to include only tasks 1–4 (Fig. 1) when brake shoes are not reused, whereas most of the brake repairs observed in this study involved reuse of brake shoes. This could explain the differences in findings between this study and other recent studies. In addition, it should be noted that studies conducted a few decades ago when brake lining manipulation was still a common practice in developed countries reported air asbestos concentrations exceeding current OSHA standards.

330. According to Cely-Garcia (2012), indirect work and bystander exposure can be very significant:

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[617] Imbernon et al., *Èvaluation Quantitative du Risque de Cancer du Poumon et de Mésothéliome Pleural Chez les Mécaniciens de Véhicules Automobiles,* Rev Epidemiol Sante Publique 53:491-500 (2005).

> The activities associated with personal short-term asbestos concentrations (Table 8) indicate that exposure to asbestos results from the direct manipulation of asbestos containing brake linings and also from secondary exposures to the asbestos fibers released from the manipulation process, which deposit on the floor and other surfaces, and then can be resuspended again during other activities. In fact, some of the highest personal short-term asbestos concentrations observed using TEM were associated with cleaning activities and not the direct manipulation of the linings.

331. Cely-Garcia (2015) addressed and rejected asbestos industry Doubt Science in the context of the risk from asbestos in brakes:

> Although some studies have suggested that chrysotile is less harmful compared with amphiboles[618], there is robust evidence showing an excess mortality risk because of lung cancer, all cancers, and nonmalignant respiratory diseases in chrysotile-exposed workers, even for those exposed to low concentrations.

332. Workers in Cely-Garcia (2015) had "8-hour time-weighted average (TWA) personal exposures ranging between 0.003 and 0.157 f/cm^3. . . . The results of this study provide preliminary evidence that workers in heavy vehicle [brake repair shops] could be at excessive risk of developing asbestos-related diseases."

333. Salazar et al. (2014), while discussing the background of auto mechanics asbestos exposures, indicated that their research team has "found that the *manipulation of asbestos containing brake products (e.g. grinding, drilling) resulted in the release of important amounts of asbestos fibers*, exposing the workers." (citing Cely-Garcia *et al.*, 2012, 2015).[619] Salazar et al. (2014) also found that "[a]s expected, days with the highest number of manipulations resulted in the highest 8-h TWA PCME personal asbestos concentrations."

334. Salazar et al. (2014) concluded that manipulating asbestos clutch facings led to "high" exposure concentrations and transmission mechanics are "at excess risk of developing asbestos-related diseases" and that co-workers "that do not manipulate clutch facings may be exposed to high asbestos concentrations in the workplace." Based on the risk of asbestos exposure to auto mechanics while doing brake and clutch work, Salazar et al. (2014) "strongly recommend that the production and use of asbestos-containing products in the country should be severely restricted or banned."

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[618] Citing Bernstein et al., *Health risk of chrysotile revisited*, Crit. Rev. Toxicol. 43:154–183 (2013). This paper is fundamentally flawed and the conflict of interest disclosure falsely claims that the research was supported by a grant when it was really supported by an hourly fee-for-service consulting agreement with an asbestos industry group.

[619] Salazar et al. (2014) (emphasis added).

335. The recent studies by Cely-Garcia et al. (2012); Cely-Garcia et al. (2014) and Salazar et al. (2014) reinforce the data from earlier studies that demonstrate that some automotive maintenance work practices can cause substantial exposure. This is not new information, but it underscores the issue, often advanced by industry-aligned scientists, that average exposures for mechanics are low. Each mechanic must be evaluated based on the work practices that mechanic used. Individual exposures, especially for specific intermittent tasks may be very high. For example, Paustenbach et al. (2003) reported estimated 8-hour TWAs for mechanics servicing automobiles and light trucks "ranged from <0.002 to 0.68 f/cc with a mean of 0.04 f/cc [and] . . . 8-hour TWAs for mechanics servicing heavy trucks and buses ranged from 0.002 to 1.75 f/cc, with a mean of 0.2 f/cc."[620] While the calculation of the mean exposure may be accurate, it is inappropriate to suggest that an individual was exposed to the mean because the mean is based on aggregate measures of exposure, including from studies using dust control methods. For surrogate exposure data to apply to an individual case, it must include similar work practices. It also is important to recognize that average exposures over 8 hours may be relatively "low", but short-term "high" levels may well be more harmful.

336. Finally, it is also important to recognize that at the mean exposure of 0.04 f/cc, it would take less than three years of work as a vehicle mechanic to experience cumulative exposures of 0.12 f/cc/years. Such cumulative exposures have been shown by epidemiological studies to produced statistically significant excess risk. For example, Lacourt et al. (2014), for the cumulative exposure estimate of more than zero but less than 0.1 f/cc/years, there was a substantially increased risk of mesothelioma (≤ 0.1 f/cc/years, Odds Ratio (OR) 4.0 (99% Confidence interval (CI) 1.9 – 8.3)).[621]. Similarly, Rödelsperger et al. (2001) concluded there was a distinct dose-response relationship, even at extremely low levels of asbestos exposure, with exposures from >0 to <0.15 f/cc/years showing a significantly increased risk of mesothelioma (Hazard Ratio (HR) 2.69 (95% CI 1.60 – 4.53)).[622] Given the dose-response relationship between asbestos exposure and risk, it is clear that there is excess risk at doses below those observed in these studies.

337. A recent case report found peritoneal mesothelioma in a family member whose husband was a brake mechanic for more than 30 years.[623] This is consistent with my experience that even household exposure to asbestos from brakes can and does cause mesothelioma in some people. I have seen, personally, a lung cancer in a brake mechanic's wife, when neither were smokers.

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[620] Paustenbach et al., *An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust*, Appl. Occup. Environ. Hyg., 18:786-804 (2003).

[621] Lacourt et al., *Occupational and Non-Occupational Attributable Risk of Asbestos Exposure for Malignant Pleural Mesothelioma*, Thorax 1 (2014).

[622] Rödelsperger et al., *Asbestos and man-made vitreous fibers as risk factors for diffuse malignant mesothelioma: Results from a German hospital-based case-control study.* Am. J. Ind. Med. 39:262-275 (2001).

[623] Ruiz-Tirado et al., *Mesotelioma peritoneal maligno – Informe de un caso y revision de la literature*, Rev. Med. Inst. Mex. Seguro Soc. 49(1): 79 – 84 (2011) (abstract in English).

338. It is precisely because scientists and physicians understand the limitations of epidemiology and how certain factors can bias studies toward a lack of statistical significance or finding of a point estimate of no increased risk, that we look at the epidemiology of a substance along with the other scientific data described above. Each epidemiological study must be evaluated for its strengths and weaknesses, and decisions about cause and effect should only be made on reliable data.

339. Specifically, because the epidemiological and all of the other scientific evidence that chrysotile asbestos causes mesothelioma is so strong that the mainstream scientific community has concluded, and I am able to conclude, that a mesothelioma in a patient exposed to dust from chrysotile asbestos brakes or clutches (or any other source of chrysotile asbestos) was caused, in whole or in part, by that dust.

XIII. Asbestos in Talc Causes Mesothelioma.

340. A recent effort by Finkelstein to update the cohort study of talc miners and millers from R.T. Vanderbilt's New York talc mines supports my opinion that asbestos-containing talc causes mesothelioma.[624] Finkelstein's study updates Honda et al. (2002).[625] Talc producer, R.T. Vanderbilt, funded the Honda study but did not provide Honda et al. complete data regarding the additional deaths from mesothelioma outside the cohort definition.

341. Hull et al. (2002)[626] reported an analysis of the lung-retained particulate of ten (10) New York talc workers, including two cases of mesothelioma. Hull et al. identified fibers of anthophyllite, tremolite/actinolite, chrysotile, and talc in lung tissues. The size range of the asbestos fibers was as follows: tremolite from 1.5 to 17 microns in length, with a mean width of 0.22 microns and a mean aspect ratio of 26; anthophyllite ranged from 1.6 to 146 microns in length, with a mean width of 0.15 microns and a mean aspect ratio of 90; chrysotile from 1.9 to 36 microns in length, with a mean width of 0.05 microns and a mean aspect ratio of 93. According to Finkelstein (2012):

> These observations are compatible with the NIOSH electron-microscopic analyses of ore dust reported above and are consistent with Wylie's (2000) definition that "amphibole-asbestos fibrils range in width from about 1 to 0.01 micron" and "individual fibrils and bundles of fibrils may attain lengths of hundreds to thousands of times their widths". Many of the chrysotile and amphibole minerals in the lungs of the talc workers are thus fibers, and not cleavage fragments, and the findings of Hull et al. are consistent with the deposition of a variety of asbestos fibers in the lungs of talc miners and millers attributable to occupational exposures to mine and mill dusts.[627]

Finkelstein's update of Honda, using assumptions that would lead to an underestimate of the risk of mesothelioma (underestimating number of mesotheliomas from the cohort and overestimating number of person years at risk ("PYR")), "*found [t]here were at least five new cases of mesothelioma in the cohort and mesothelioma incidence rates were at least*

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[624] Finkelstein, *Malignant Mesothelioma Incidence Among Talc Miners and Millers in New York State*, Am. J. Ind. Med. 55(10):863-8 (Oct. 2012).

[625] Honda et al., *Mortality among workers at a talc mining and milling facility*, Ann. Occup. Hyg. 46:575-585 (2002)

[626] Hull et al, *Mesothelioma among workers in asbestiform fiber-bearing talc mines in New York State*, Ann. Occ. Hyg. 46(Suppl. 1):132-135 (2002).

[627] Citing Wylie, *The habit of asbestiform amphiboles: Implications for the analysis of bulk samples*, In: Beard ME, Rooks HL, editors. Advances in environmental measurement methods for asbestos. West Conshohocken Pennsylvania: American Society for Testing and Materials, pp. 53-69 (2000).

five (1.6-11.7) times the rate in the general population."[628] Based on this finding, my review of the literature, my experience in reviewing cases of talc induced mesothelioma, I agree with Finkelstein's conclusion that "it is prudent, on the balance of probabilities, to conclude that dusts from New York State talc ores are capable of causing mesothelioma in exposed individuals." Whether they are "cleavage fragments" or asbestos fibers, it is clear that asbestos containing talc causes mesothelioma. This is consistent with the IARC assessment of asbestos-containing talc.

342. There is substantial evidence that talcs from other areas also contain substantial amounts of asbestos (or asbestiform fibers) that can cause mesothelioma. For example, talc from Death Valley, California often contains amphibole asbestos that can cause mesothelioma. Van Gosen identified amphibole asbestos in numerous talcs from the Death Valley mines. Recently, Compton examined white talc ore from the Grantham Mine (source of ore for Sierra Talc and later owned by Johns-Manville Corp.). Using polarized light microscopy (PLM), Compton found "[t]he mineral sample was found to contain 5-15% (by volume) tremolite/actinolite as determined by PLM. The sample contains asbestiform fibers consistent with fibrous tremolite (see Figures 2 and 3) and fibrous talc."[629] Tests of other sources of talc have yielded similar results.

343. Recently, Gordon et al. (2014) reported a case of mesothelioma associated with cosmetic talc.[630]

[628] Finkelstein, *Malignant Mesothelioma Incidence Among Talc Miners and Millers in New York State*, Am. J. Ind. Med. 55(10):863-8 (Oct. 2012). Finkelstein's study updates Honda et al, *Mortality among workers at a talc mining and milling facility*, Ann. Occup. Hyg. 46:575-585 (2002)(italics in original).

[629] Compton, *Report of Results: MVA11054 Analysis of Grantham Mine Talc for asbestos*, Prepared for: Maune Raichle Hartley French & Mudd, LLC, 70 Washington St., Suite 425 Oakland, CA 94607 (July 8, 2015).

[630] Gordon et al., *Asbestos in Commercial Cosmetic Talcum Powder as a Cause of Mesothelioma in Women*, Int. J. Occup. Environ. Health 20 (4) 318-332 (2014).

XIV. Smoking Asbestos-Filtered Cigarettes (Kent) is a Risk Factor for Mesothelioma.

344. For approximately four years, from March 1952 though May 1956, Lorillard Tobacco Company made Kent cigarettes with an asbestos-containing "Micronite" filter. The filter contained approximately 7 to 25% African crocidolite asbestos by weight and 10 – 40% crocidolite by volume. In addition to the African crocidolite asbestos, the filter was comprised of crepe paper, cotton, and cellulose acetate. Each Kent cigarette smoked with the asbestos-containing filter provided heavy potential non-occupational exposures to asbestos. Scientists from MVA Scientific Consultants analyzed smoking machine tests involving Kent cigarettes using various methods and found substantial fiber release whenever whole cigarettes were smoked:

SMOKING MACHINE FILTER TEST[631]

Cigarette	Test Protocol	Crocidolite Fibers
Kent Micronite Reg	ISO (8 puffs)	10,820,923
Kent Micronite King	ISO (8 puffs)	3,950,496
Kent Micronite Reg	Intense (8 puffs)	309,169
Kent Micronite King	Intense (8 puffs)	39,000
Kent Micronite Reg	ISO (2 Puffs)	NAD >39,000
Kent Micronite Reg	Intense (2 Puffs)	NAD >39,000
Kent Micronite King	ISO (2 Puffs)	NAD >39,000
Kent Micronite King	Intense (2 Puffs)	NAD >39,000

Based on these results, it is clear that "crocidolite fiber are released from the Kent Micronite filters during smoking."[632]

345. In a published, peer reviewed study of testing of Kent cigarettes with the Micronite filter, Longo et al. (1995) concluded that "a person smoking a pack of [asbestos-containing Kent] cigarettes each day would take in more that 131 million crocidolite structures longer than 5 microns in 1 year."[633] These authors observed that their work generally confirmed work previously done by Lorillard's own scientists:

> Our data confirm the results of two series of TEM-based tests of Kent cigarette smoke performed in early 1954, one TEM series performed by Althea Revere (Life Extension Foundation), the other by Douglas Halgren and Dr. Ernest Fullam (Ernest Fullam

[631] Source: Compton, Steven and James Millette, *Report of Results: MVA8472 – Analysis of Air Filters – Kent Micronite Cigarettes,* (September 30, 2010). One test, the Kent Micronite King (intense) found only one crocidolite fiber, yielding an actual value of 38,646. Because Compton, et al. (the limit of detection was 39,000, I have presented the value as 39,000.

[632] Source: Compton, Steven and James Millette, *Report of Results: MVA8472 – Analysis of Air Filters – Kent Micronite Cigarettes,* (September 30, 2010) at page 4.

[633] Longo et al., *Crocidolite Asbestos Fibers in Smoke from Original Kent Cigarettes*, Cancer Research 55:2232-2235 (1995).

> Laboratories, Schenectady, New York). While both original reports have been lost, it is clear from other documentation that both laboratories observed asbestos structures in mainstream smoke from Kent cigarettes (12). These two studies were among the first to use electron microscopy to detect individual asbestos fibers. The present work extends these earlier studies by quantifying the amount of crocidolite released from the filter during the smoking process.[634]

346. Like the tests of the MVA Scientific Consultants, there was substantial cigarette to cigarette variability in the amount" of asbestos released."[635] The authors explained that their results probably underestimated the amount of crocidolite asbestos inhaled with each cigarette smoked:

> Our data probably underestimate the amount of crocidolite released in an actual smoking situation for 3 reasons: (a) these tests examined only smoke from the first 2 puffs, and there was still substantial release of asbestos during the second puff; (b) the numbers given, in conformance with EPA counting rules (11), reflect "structures" and not "fibers."[636]

347. The results of such testing of asbestos fiber release from the Kent Micronite filter comes as no surprise given the fact that Lorillard's own testing, before April 26, 1954, showed smoke from asbestos-containing Kent cigarettes "contained traces of mineral fiber."[637] Lorillard's Director of Research, Mr. Parmele wrote that Lorillard had "embarked upon a program of attempting to work out a method for the elimination of such fibers in the smoke."[638] The Assistant Technical Director for the manufacturer of the asbestos filters for Lorillard, H & V Specialties, wrote to Lorillard on June 4, 1954 indicating that H&V's inability to detect asbestos fibers in the smoke of Kent cigarettes was "probably due to inadequate microscopic technique."[639] This makes sense, given that a light microscope, such as the phase contrast microscope ("PCM"), cannot reliably detect asbestos fibers thinner than 0.25 microns[640] and the crocidolite fibers found by MVA Scientific Consultants were typically 0.14 microns in diameter. A later test done for

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[634] Longo et al., *Crocidolite Asbestos Fibers in Smoke from Original Kent Cigarettes*, Cancer Research 55:2232-2235 (1995).
[635] Longo et al., *Crocidolite Asbestos Fibers in Smoke from Original Kent Cigarettes*, Cancer Research 55:2232-2235 (1995).
[636] Longo et al., *Crocidolite Asbestos Fibers in Smoke from Original Kent Cigarettes*, Cancer Research 55:2232-2235 (1995).
[637] Parmele - Letter to W.J. Halley, President, P. Lorillard Co. (April 26, 1954).
[638] Parmele - Letter to W.J. Halley, President, P. Lorillard Co. (April 26, 1954).
[639] Breymeier - Letter to Dr. H.B. Parmele (June 4, 1954).
[640] National Institute for Occupational Safety and Health ("NIOSH"), Method 7400, Asbestos and Other Fibers by PCM (1994).

Lorillard found that "long, needlelike" asbestos fiber release from Kent cigarettes as well.[641]

348. On December 1, 1954, Lorillard's Dr. Parmele transmitted results of testing done for Lorillard by an electron microscopist using a transmission electron microscope ("TEM").[642] This testing showed that, using a more powerful microscope capable of resolving very thin asbestos fibers, that 100% (18 of 18) of the Kent Micronite smoke samples analyzed contained asbestos. After questioning the results of the testing, Lorillard's Parmele wrote "[w]e feel this way particularly since it is our intention to eliminate the use of asbestos in the very near future. However, if it were not for the money involved, it might be interesting to go ahead and submit the additional samples in question in order to acquire a background of information which someday in the future might be useful."[643]

349. As discussed above, there was ample evidence available in the medical and scientific literature about the hazards of asbestos when Lorillard introduced its asbestos-containing Kent Micronite filters. Despite that evidence, Lorillard marketed Kent cigarettes with the asbestos filter as a safer cigarette because of the Micronite filter, claiming in an advertisement in Life Magazine that Kent smoke had "far less irritants than any other cigarette of any kind."[644] Other cigarette brands saw Lorillard's use of asbestos, a carcinogen, as weakness to exploit in marketing. For example, Viceroy advertised that its filter "contain[ed] no dangerous powdery particles of asbestos or charcoal to come through in your smoke."[645] According to one memorandum from a consultant to Lorillard, Dr. Parmele told the consultant that the Micronite filter "used to be asbestos, but this was eliminated in good part because of fears that competition would make something of Kents "mineral" filter.[646]

350. It is also undeniable that smoking Kent cigarettes with the crocidolite asbestos Micronite filter was a dangerous activity. Numerous published, peer reviewed epidemiological studies have shown substantially increased risk of mesothelioma in populations with estimated cumulative exposures to asbestos of less than 1.0 fiber/cc/years of exposure.[647]

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[641] Armour Research Foundation of the Illinois Institute of Technology, *Progress Report No. 11 P. Lorillard Company Physical Properties of Cigarette Smoke Project No. C 593*, (August 19 – September 25, 1954).

[642] Parmele - Letter to Dr. Harold Knudson, Technical Director of Hollingsworth & Vose Co., (December 1, 1954).

[643] Parmele - Letter to Dr. Harold Knudson, Technical Director of Hollingsworth & Vose Co., (December 1, 1954).

[644] Life Magazine (June 15, 1954) at page 111.

[645] Ted Bates & Company. *Report of Conference with Client with Brown & Williamson Tobacco Corp.* (July 31, 1957).

[646] EC, *Interoffice Memorandum re New Kent* (January 14, 1958) (http://industrydocuments.library.ucsf.edu/tobacco/docs/pmmh0113).

[647] Markowitz (2015) (Table 1, citing Iwatsubo et al. (1997), Rödelsperger et al. (2001), and LaCourt et al. (2014)).

351. In an editorial discussing the dangers of environmental exposure to asbestos, Roggli made assumptions about the relative potency of crocidolite asbestos and exposure levels in various epidemiological studies and calculated that exposure to African Blue asbestos (a/k/a crocidolite) "doubles the background risk of mesothelioma at a cumulative level of 0.015 f/mL/yr. This is equivalent to approximately 2 months of exposure at the current Occupational Safety and Health Administration permissible exposure limit of 0.1 fiber/mL."[648] Assuming someone is working in an environment with an 8-hour TWA of 0.015 f/cc and assuming a working and has respiration rate of 16 breaths per minute and 500 cc's per breath, that person would breathe 14,400,000 fibers to reach 0.015 f/mL/yr cumulative dose. Using the lowest value for a smoking an entire Kent asbestos-containing cigarette from MVA Scientific Consultant's data (38,464 fibers per Kent cigarette smoked), it would take approximately 375 Kent cigarettes (less than 20 packs) to inhale 14,400,000 crocidolite fibers. Of course, many of the fibers measured by MVA Scientific Consultant's testing were not 5 microns, so this is not a perfect apples-to-apples comparison.

352. Using the data from Longo et al. (1995) that someone smoking 365 packs (smoking only two puffs) would inhale 132,000,000 crocidolite fibers longer than 5 microns and approximately 1.1 billion fibers shorter than 5 microns. Boulanger et al. (2014) reviewed the vast majority of the literature discussing the role of fiber length and concluded "In the literature, the findings that [short asbestos fibers i.e. shorter than 5 μm in length] are less pathogenic than [long asbestos fibers] are based on experiments where a cut-off of 5 μm was generally made to differentiate short from long asbestos fibers. Nevertheless, the value of 5 μm as the limit for length is not based on scientific evidence, but is a limit for comparative analyses. From this review, it is clear that the pathogenicity of [short asbestos fibers] cannot be completely ruled out, especially in high exposure situations. . . . Our findings suggest that the presence of high levels of SAF is a health concern, and alert on the degradation of ACM."[649]

353. In my medical-legal consultation, I have reviewed cases of mesothelioma for patients who smoked Kent cigarettes with the asbestos-containing micronite filter. I have also reviewed records of workers who manufactured asbestos-containing filter material who developed mesothelioma.

[648] Roggli, *Environmental Asbestos Contamination – What Are the Risks?*, Chest 131:2 336-337 (2007).
[649] Boulanger et al., *Quantification of Short and Long Asbestos Fibers to Assess Asbestos Exposure: a Review of Fiber Size Toxicity,* Environ. Health 13(59) (2014).

XV. Occupational Exposure Levels Are Much Greater Than Ambient Levels of Asbestos.

354. Due to the natural existence of and extensive and longstanding use of asbestos, the ambient air in the United States contains minute amounts of asbestos. These ambient air concentrations are also known as the "background level." Per the ATSDR,

> For example, 10 fibers are typically present in a cubic meter (fibers/m^3) of outdoor air in rural areas. (A cubic meter is about the amount of air that you breathe in 1 hour.) Health professionals often report the number of fibers in a milliliter (mL) (equivalent to a cubic centimeter [cm^3]) of air rather than in a cubic meter of air. Since there are one million cm^3 (or one million mL) in a cubic meter, there typically would be 0.00001 fibers/mL of asbestos in air in rural areas. Typical levels found in cities are about 10-fold higher.[650]

355. This "background level" of exposure to asbestos is very low when compared to occupational exposures. For example, if only OSHA fibers (*i.e.* fibers longer than 5 microns) are counted, an exposure at the current OSHA PEL of 0.1 f/cc over a single eight (8) hour workday would mean a worker would breathe 384,000 fibers as compared to the person in a rural environment who would breathe about 29 fibers every eight hours.[651] Put differently, one day at the current OSHA PEL is about 13,000 times the background over the same eight-hour workday. It bears noting that someone working with asbestos will receive whatever exposures he/she gets from the sources of asbestos in addition to whatever exposure that person receives from the local "background." Little or no data is available to know exactly what that truly low level might have been, day-by-day, in a person's lifetime. It is also important to recognize that for every OSHA fiber counted, there are many more fibers in a given air sample which are not counted under the NIOSH 7400 method.

356. In contrast to the ambient asbestos levels, one asbestos company official explained the exposures experienced at the 1985 OSHA PEL of 2.0 f/cc:

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[650] U.S. Public Health Service, U.S. Department of Health & Human Services. *Public Health Statement Asbestos CAS#: 1332-21-4*. Atlanta: Agency for Toxic Substances and Disease Registry (September 2001).

[651] The ambient air exposures were calculated based on the assumption that a person breathes 12 breaths/minute at rest, each breath is 500 cc of air, so the person breathes 6,000 cc/minute. Over eights hours, a resting person will breathe 2,880,000 cc of air (360,000 cc/hour x 8 hours). 2,880,000 x 0.00001 = 28.8 fibers for every 8 hours breathing ambient air. The OSHA PEL exposures were calculated based on the assumption that a person breathes 16 breaths/minute during moderate work, each breath is 500 cc of air, so the person breathes 8,000 cc/minute. The person would breathe 480,000 cc/hour. At 0.1 f/cc, this person would breath 800 fibers per minute or 48,000 per hour. Over eight hours, a working person will breathe cc of air (480,000 cc/hour x 8 hours) 3,840,000. 3,840,000 x 0.00001 = 384,000 fibers every 8 hours at the OSHA PEL of 0.1 f/cc.

> In physical terms 2.0 f/cc equals 2,000,000 fibers per cubic meter of air (f/m^3). Humans inhale about one cubic meter of air per hour, depending on the degree of activity. Thus, at this concentration a worker would inhale roughly 16,000,000 fiber 5 microns in length over an 8-hour day disregarding the possibility of an infinite number of shorter fibers being present under some conditions.[652]

357. With respect to background concentrations of asbestos fibers, it is my opinion that "background" is a vague term that has not been well defined. Regardless of the actual background experienced by any person, any inhalation of asbestos released from a point source would be above background by definition.

358. With respect to the "background" rate of mesothelioma, if any, that might occur in the absence of asbestos exposure, Hillerdal (1999) reported several cases of low level exposure to asbestos and the development of mesothelioma and concluded there might not be a true background rate at which mesothelioma occurs.[653] Studies have confirmed that mesothelioma is a relatively new disease and appears to correlate with the rise in usage of asbestos.[654] "The background environmental mesothelioma incidence rate and especially the true spontaneous rate is probably substantially less than one case/10^6/yr., but the true rate can only be guessed, because no significant control adult population without asbestos fibers in lung tissue can be assembled."[655]

359. I have long questioned the existence of significant numbers of idiopathic mesotheliomas because the vast majority of people have been exposed to asbestos in the environment. A recent editorial in the New England Journal of Medicine explained, in the context of cancers other than mesothelioma, that cancers without outside causes are highly unlikely to occur because they would need at least three simultaneous mutations to occur.[656]

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[652] Young, Union Carbide Internal Correspondence re *Asbestos Exposure Effects Potential of Non-Insulator Crafts* (June 28, 1985).

[653] Hillerdal, *Mesothelioma: cases associated with non-occupational and low dose exposures.* Occup. Environ. Med. 56:505-513 (1999).

[654] Mark et al., *Absence of Evidence for a Significant Background Incidence of Diffuse Malignant Mesothelioma Apart from Asbestos Exposure.* Ann. N.Y. Acad. Sci. 643:196 – 204 (1991); Strauchen, *Rarity of Malignant Mesothelioma Prior to the Widespread Commercial Introduction of Asbestos: The Mount Sinai Autopsy Experience 1883 – 1910.* Am. J. Industr. Med. 1-3 (2011).

[655] Hammar et al., Dail and Hammar's Pulmonary Pathology Vol II, Neoplastic Lung Disease 3rd Ed Chapter 43 Neoplasms of the Pleura, 558-734 (2008) (citing Hillerdal G. *Mesothelioma: cases associated with non-occupational and low dose exposures.* Occup. Environ. Med. 1999;56:505-513 ("Hillerdal (1999)").

[656] Vogelstein et al., *The Path to Cancer — Three Strikes and You're Out*, N. Engl. J. Med. 373; 20 (2015) (internal citations omitted). Vogelstein, et. al (2015) explained: "a normal adult cell cannot suddenly transform into a cancer cell. Given known mutation rates, the probability that a cell can acquire three mutations simultaneously is negligible."

360. There is much published evidence that neighborhood environmental asbestos exposures can be sufficient to cause mesothelioma. For example, a study carried out in greater Cairo, Egypt concerning asbestos and the exposure-response relationship with mesothelioma, evaluated the prevalence of malignant pleural mesothelioma due to occupational and environmental (non-occupational) exposure to asbestos among persons who worked in the asbestos manufacturing plant and in persons living in an area nearby the plant. Eighty-eight cases of mesothelioma were diagnosed, of which 87 were in the exposed group. The risk of mesothelioma was stated to be higher in the environmentally exposed group than in other groups and was higher in females than males. The prevalence of mesothelioma increased with increased cumulative exposure to asbestos.[657] Pan et al. found that residential proximity to naturally occurring asbestos showed an independent and dose-response association with mesothelioma risk.[658] Goldberg et al. stated: "there is a real burden of environmental asbestos exposure in industrialized countries that could account for approximately 20% of all mesotheliomas." However, further research was needed. Furthermore, the authors stated the high proportion of female mesothelioma cases with no identifiable asbestos exposure suggested that the burden of environmental asbestos exposure was far from negligible.[659] Therefore, based on the information available, all occupational and bystander exposures to asbestos above the background or ambient levels of asbestos within the latency period have the ability to contribute to the causation of mesothelioma.

361. It is worth noting that one group of researchers in the United Kingdom presented evidence that many mesotheliomas which previously have been termed "spontaneous" are "likely to be due to an increase in ambient asbestos exposure that coincided with the widespread occupational exposures of the 1960s and 1970s.[660]

362. Mesotheliomas from unidentified/unknown exposures are *not* the same as a "spontaneous mesotheliomas." Indeed, "[g]enome-sequencing data exclude the possibility of spontaneous tumors: a normal adult cell cannot suddenly transform into a cancer cell. Given known mutation rates, the probability that a cell can acquire three mutations simultaneously is negligible."[661] Furthermore, for a cancer to be idiopathic (or spontaneous), one must exclude exposure to known causes of the cancer. In cases with identified exposure to asbestos, it is contrary to science and medicine to pronounce the

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[657] Madkour et al., *Environmental exposure to asbestos-response relationship with mesothelioma*. Eastern Mediterranean Health J. 15:25–38 (2009).

[658] Pan et al., *Residential proximity to naturally occurring asbestos and mesothelioma risk in California*. Am. J. Respir. Crit. Care Med. 172:1019-1025 (2005).

[659] Goldberg et al., *Possible effect of environmental exposure to asbestos on geographical variation in mesothelioma rates*. Occup. Environ. Med. 67:417-421 (2010).

[660] Darnton et al., *The burden of occupational cancer in Great Britain – Mesothelioma*, Health and Safety Executive Research Report RR861 (2012) (discussing Rake et al., *Occupational, domestic and environmental mesothelioma risks in the British population: a case-control study*, Brit. J. Cancer, 1-9 (2009)).

[661] Vogelstein et al., *The Path to Cancer — Three Strikes and You're Out*, N. Engl. J. Med. 373; 20 (2015)(internal citations omitted).

cancer "idiopathic" or "spontaneous." In some ways, however, all clinical appearances of cancers in humans can be said to be "spontaneous" even when there is a known cause for that cancer. No one knows beforehand when a cancer will appear.

XVI. Product Defense Literature – Sometimes Called "Doubt Science" – Is Published to Aid in the Defense of Lawsuits and Limit Regulation.

363. Increasingly over the past several decades, industry-funded research has taken a prominent position in our society. While this phenomenon first received widespread public attention in connection with the tobacco-industry's manipulation of scientific evidence and stilted presentation of scientific data, there has been widespread analysis of the effect of industry-funding of articles in the peer-reviewed literature.

364. For example, concerns regarding the influence of industry funded science and its use in courts are so widespread in the scientific community that they led to the holding of four conferences: Coronado Conference I (2003), Coronado Conference II (2004), Coronado Conference III (2006) and Coronado Conference IV (2007) organized by the Project on Scientific Knowledge and Public Policy (SKAPP) at the George Washington University School of Public Health. The proceedings of those conferences and links to the papers published in connection with them can all be found at http://www.defendingscience.org/coronado-conference-papers. A description of the Coronado Conferences and SKAPP in general terms is contained in the published editorial *Scientific Evidence and Public Policy*, Am. J. Pub. Health, 95:S1, S5-S7 (2005) authored by David Michaels, Ph.D.

Dr. Michaels is an epidemiologist and has been the head of OSHA. Prior to assuming the position as head of OSHA, Dr. Michaels was the director of the SKAPP and a lead organizer of the Coronado Conferences. In addition to having published many articles in the peer reviewed scientific literature regarding many topics (including asbestos and disease), Dr. Michaels has published extensively regarding industry funded science and authored perhaps the preeminent text exploring the effects of industry funding and manipulation of scientific evidence – *Doubt is their Product: How Industry's Assault on Science Threatens Your Health*, Oxford Univ. Press (2008). Dr. Michaels authored or co-authored a number of articles published in connection with the Coronado Conferences.

Doubt is their Product and the papers published in connection with the Coronado Conferences regarding the need for careful scrutiny of industry-funded literature are the types of critical analysis of scientific papers reasonably relied upon by myself and others in the scientific community in weighing arguments and papers presented by scientists in the peer-reviewed literature. An excellent review of *Doubt is their Product* published in the highly regarded journal Nature.[662] Indeed, I have recommended *Doubt is their Product* to students of occupational medicine at Drexel University, where I have been employed and taught for many years. This type of analysis is critical in properly evaluating scientific papers published by authors who have financial stakes in the outcome of the research they are presenting – particularly because the methods of generating scientific arguments that are essentially guaranteed to result in publications favorable to the corporate sponsor are well-documented but not readily apparent to even occupational health students with substantial training.

[662] Taverne, *Suppressing Science*, Nature 453:857-58 (2008).

365. Products liability litigation and the costs of bringing new drugs to market have raised concern in the medical and scientific community regarding the results of industry-funded research that appears to favor the interests of the funding source. In 2013, the well-respected British Medical Journal ("BMJ") made the decision to stopped publishing "research funded by the tobacco industry for two main reasons: the research is corrupted and the companies publish their research to advance their commercial aims, oblivious of the harm they do."[663] Recently, BMJ adopted a policy of refusing to consider publishing of original research funded by entities with a financial stake in the outcome of the study.[664] While recognizing that not all industry-funded research is poorly done or biased, BMJ was so concerned about the potential for bias is industry-funded articles that it felt justified in the blanket refusal to consider such research. In my experience, such wariness of industry-funded studies is warranted. BMJ made the change because the editors "believe that the educational content we publish will have more impact if readers can trust it. We know that readers consider research papers written by authors with declared financial links to industry to be less important, relevant, rigorous, and believable . . . ."[665]

366. While I have long been concerned with scientific revisionism – by scientists and consultants involved with industry – performed to seed the medical and scientific literature with publications and data for use in litigation, this topic has become much more common in the last decade with the proliferation of litigation science. As I explained in 1995, it is my "view that much of the revisionism and inaccuracy with regard to asbestos-related disease . . . has been the result of legal activity."[666]

367. A recent editorial explained the model of "product defence" scientists:

> Product defence studies differ from other research because of the systematic and intentional introduction of bias in their design and/or lack of impartiality in the interpretation of findings: the harmlessness of any agent under investigation is claimed by raising

[663] Smith et al., *Should journals stop publishing research funded by the drug industry?*, Brit. Med. J. 348:g171 (2014) (citing Godlee et al, *Journal policy on research funded by the tobacco industry*, Brit. Med. J. 347:f5193 (2013)).

[664] Chew et al., *Medical journals and industry ties – Zero tolerance on education articles with financial links to industry*, *BMJ* 2014;349:g7197 (2014).

[665] Chew et al., *Medical journals and industry ties – Zero tolerance on education articles with financial links to industry*, *BMJ* 2014;349:g7197 (2014) (citing Schroter et al., *Does the type of competing interest affect readers' perceptions of the credibility of research?*, BMJ 328:742 (2004). Kesselheim et al., *A randomized study of how physicians interpret research funding disclosures*, N Engl J Med 367:1119-27 (2012)).

[666] Frank, *Medical and Public Health Approaches to Asbestos Disease*, Mount Sinai J. Med. 62(5) (1995) (citing the following industry-aligned publications: Mossman et al., *Asbestos-related diseases*. N Engl J Med 26:1721-1730 (1989) and Mossman et al., *Asbestos: scientific developments and implications for public policy*, Science 247:294-301 (1990).

> doubts as well as shaping and skewing the scientific literature, manufacturing and magnifying the unavoidable residual scientific uncertainty.[667]

Terracini et al. (2016) further explained the purpose of "product defence" articles:

> Raising doubts about the evidence that drastic (and feasible) measures are needed to protect people from environmental hazards is a major strategy of scientists working on behalf of polluting industries. In the case of asbestos, such industry-driven strategy spans a wide range of recurrent subjects, such as the role of individual susceptibility in asbestos carcinogenicity, the effectiveness of preventive measures, the diagnostic uncertainties of mesothelioma, the shape of the dose-response curve, the relative carcinogenic potency of the various types of asbestos, and several other subjects. Inevitable gaps in knowledge about such topics are ambiguously used to discredit the evidence of risk.[668]

368. The earliest evidence of the creation of product defense literature to aid in the defense of asbestos lawsuits – "Doubt Science" – can be traced back to the 1930s. In 1935, The Temporary Organizing Committee for the Industrial Dust Problem (which ultimately led to the formation of the Industrial Hygiene Foundation) proposed that a group of companies with asbestos and silica dust "problems" work to set up "authoritative and approved standards for the control of industrial dusts which, if complied with by industries, or by industrial companies, will act as a defense against personal injury suits."[669] This memorandum from 1935 was a discussion of the origin of what later became known as the Threshold Limit Value ("TLV").

369. By 1973, the asbestos industry was privately admitting what they knew or should have known for many years: asbestos was a killer. For example, the AIA/NA secretary, Matthew Swetonic, wrote in a speech to the Asbestos Textile Institute and circulated to the AIA/NA member companies that "there is no doubt that the inhalation of substantial amounts of asbestos can lead to increased rates of various types of lung disease, including two forms of cancer. These are facts which cannot be denied, even if they do not apply in all circumstances and under all conditions."[670]

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[667] Terracini et al., *Asbestos and product defence science*, Intern. J. Epidem. Advance Access pp. 1-5 (2016).
[668] Terracini et al., *Asbestos and product defence science*, Intern. J. Epidem. Advance Access pp. 1-5 (2016).
[669] Weidlein - Letter to Roger A. Hitchins (President, American Refractories Institute) (January 21, 1935).
[670] Swetonic, Why Asbestos – A Speech Before the Asbestos Textile Institute Arlington, Virginia, June 7, 1973, (produced by Union Carbide).

370. Swetonic's *Why Asbestos?* provides insight into the mindset of asbestos companies in the asbestos industry. Mr. Swetonic, who later served as a public relations representative for the tobacco industry, explained the industry position:

> In our original concept, the Association would limit its activities to providing accurate, unbiased information on asbestos and health to the press, to the public and to interested politicians and other government officials. . . . Fortunately -- and properly -- the Association has had the wisdom to alter its original limited concept of its proper functions, and now endeavors to assume whatever activities and responsibilities it deems necessary to protect the interests of the asbestos manufacturing industry in the United States vis-à-vis asbestos health."[671]

Swetonic's explanation of AIA/NA's role was presented to asbestos textile companies and disseminated to AIA/NA's members including Union Carbide, CertainTeed Corp. and Bendix.

371. The AIA/NA published numerous pamphlets aimed at providing some information to inform workers of the hazards, but those materials sought to downplay the hazards of asbestos and to create a false sense of security on the part of the employer that asbestos could be used safely with minimal effort. AIA/NA member companies, such as Union Carbide, relied on AIA/NA position papers to help them sell asbestos products.[672]

372. Some aspects of AIA/NA's approach to limiting public knowledge and misleading the consumer about the hazards of asbestos set forth in Swetonic's speech continue to this day. In efforts to avoid responsibility in court and to enable continued commerce in asbestos around the world, the asbestos industry has sought to cast doubt on the hazards of asbestos. Industry and their lawyers have employed a steady stream of "Doubt Scientists" who publish product defense literature in industry-friendly journals.[673, 674]

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[671] Swetonic, Why Asbestos – A Speech Before the Asbestos Textile Institute, Arlington, Virginia, June 7, 1973, (produced by Union Carbide).

[672] Rhodes – Letter to Thurber re "OSHA Regulations – Asbestos Use Warnings, May 30, 1975 (stating that "In lieu of any specific definition of our own, we have been relying on the AIA/NA information which, as I understand it, can be summarized as follows: (1) The 2 fibers/cc limit is safe for chrysotile; (2) Increased incidence of lung cancer require both asbestos exposures sufficient to cause asbestosis and smoking. The effects of these two are synergistic, however; and (3) Mesothelioma has only been clearly associated with crocidolite inhalation. There is a real doubt that it can be caused by inhalation of chrysotile fiber.").

[673] Michaels, *Doubt is their product. How industry's assault on science threatens your health.* New York: Oxford University Press (2008).

[674] Michaels, *Manufactured uncertainty: Protecting public health in the age of contested science and product defense*. Ann NY Acad Sci 1076: 149–162 (2006).

373. Using tactics that closely resemble those used by the tobacco industry, the asbestos industry "continues to generate endless debate on the relative hazards of asbestos of different fiber type and dimension. In these debates industry spokespersons argue that some forms of asbestos are less harmful than others. However, epidemiological and statistical efforts to characterize relative cancer potencies for different asbestos fiber types and for fibers of different sizes have not been able to overcome limitations of the exposure data. Nor can these analyses account for the fact that in the real world exposure is almost always to mixtures of asbestos fibers of different types and sizes."[675]

374. In an effort to defeat liability claims, the asbestos industry commissions "the publication of articles, primarily in toxicology journals, termed 'product defense' articles. These articles are frequently sponsored by asbestos interests such as the defendants in personal injury asbestos litigation. They are distinguished from other science papers in that they are written by scientific consultants and consulting firms, who are paid substantial sums for their work."[676] All together, the auto industry, including Ford Motor Company ("Ford"), General Motors Corporation ("GM"), Chrysler, Borg Warner and Honeywell, Inc., has paid tens of millions of dollars to employ expert witnesses to publish articles for use in product defense.[677]

375. Evidence recently uncovered exposed that the authors employed by the product defense firms (Exponent, Inc. and Chemrisk, Inc. (now CardnoChemrisk, Inc.)) did not have expertise in the hazards of asbestos when they were contacted by lawyers for Ford around the year 2000. For example, Dennis Paustenbach, a frequent contributor to Ford-funded product defense literature testified that he was approached in 2000 by a Ford lawyer, Darrell Grams, with whom Paustenbach has worked on breast implant litigation defense, who explained to Paustenbach that he needed help because "there had been a series of plaintiff wins or defense losses involving brake dust."[678] Paustenbach understood that Ford's lawyer wanted him to become an expert to assist Ford in the defense of asbestos brake litigation.[679] Paustenbach admits he did not seriously study asbestos until he was hired as a litigation expert by Ford.[680] Paustenbach did not attempt to become an expert in asbestos until after he met Ford's in house counsel.[681] Paustenbach had never published on asbestos and

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[675] Collegium Ramazzini, *Asbestos Is Still With Us: Repeat Call for a Universal Ban.* Am. J. Indust. Med. 54:168-173 (2011).

[676] Collegium Ramazzini, *Asbestos Is Still With Us: Repeat Call for a Universal Ban.* Am. J. Indust. Med. 54:168-173 (2011).

[677] LaDou et al., *The Case for a Global Ban on Asbestos.* Environ. Health Perspectives 118:7 (July, 2010) (indicating GM, Ford and Chrysler sponsored several paper written by product defense consultants and "paid almost $37 million between 2001 and 2008" for various services relating to the articles).

[678] Paustenbach, Dennis Dep. *In Re: All Asbestos Litigation filed by Maune Raichle Hartley French & Mudd, LLC v. 3M Company et al.*, Madison County Circuit Ct., Il Case No: 95 ASALLLIT (6/17/15) at 17:14-18:7.

[679] Paustenbach Dep. (6/17/15) at 22:17-19.

[680] Paustenbach Dep. (6/17/15) at 27:7-14.

[681] Paustenbach Dep. (6/17/15) at 27:25-28:8.

diseases before being hired as a litigation expert by Ford.[682] Project manager for the expert witness/friction litigation publications, Patrick Sheehan, also knew the research the consultants were doing was for litigation support, rather than to help Ford make or sell better cars.[683] All documents relating to the litigation support publications by Exponent for Ford "Draft, Attorney Work Product, Privileged and Confidential, By Joint Defense Agreement."[684] It does not appear that the litigation-oriented purpose for these product-defense articles was disclosed to the journals in which these publications appeared.

376. On August 28, 2014, Exponent, Inc. was deposed pursuant to a court order. Dr. Patrick Sheehan and Richard "Rick" Schlenker (Exponent's chief financial officer) testified as Exponent's corporate representatives. Dr. Sheehan testified that the "Big Three" paid for Exponent to write publications to help Ford, Chrysler and GM lawyers defend those companies in asbestos litigation. For example, Dr. Sheehan admitted that two of the manuscripts written and published by lead author Dennis Paustenbach, then an Exponent consultant, were created to support his expert testimony on behalf of Ford.[685] Dr. Sheehan admitted that the September 17, 2014 memorandum setting forth the plan to seed the literature directly led to the publications that were the subject of the subpoena.[686] Dr. Sheehan's deposition reveals the close ties between Ford attorneys and Patrick Sheehan, a co-author of these supposedly unbiased scientific articles. For example, Sheehan admitted that the memo includes tasks Exponent was proposing to do for the auto industry for litigation.[687] Particularly, Dr. Sheehan admitted that the Exponent manuscripts came out of Exponent's work on friction litigation.[688]

377. The Exponent internal documents bear the words "Attorney Work Product" because all of the work for the auto industry was "[r]elated to the attorneys' work on litigation."[689]

378. Exponent, in its internal documents, admits that it views its work as commodities, rather than scientific research, referring to its litigation generated publications and slides for litigation as "products." [690]

379. CardnoChemrisk recently admitted that it charged Ford Motor Company alone $21,045,019.00 in consulting fees between 2003 and 2015.[691] Chemrisk billed General

[682] Paustenbach Dep. (6/17/15) at 28:11-29:1.
[683] Sheehan, Patrick Dep. Steve K. Allen and Judy Allen v. 3M Company et al. Madison County Circuit Ct. IL; Case No: 14L131 Vol. 1 & Vol. 2 (August 28, 2014) at 45:1-11.
[684] Sheehan Dep. (8/28/14) Vol. 1 at 69:22-24.
[685] Sheehan Dep. (8/28/14) Vol. 1 at 154:19-155:10.
[686] Sheehan Dep. (8/28/14) Vol. 2 at 185:1-7
[687] Sheehan Dep. (8/28/14) Vol. 2 at 186:12-17.
[688] Sheehan Dep. (8/28/14) Vol. 2 at 187:4-9.
[689] Sheehan Dep. (8/28/14) Vol. 1 at 136:1-9.
[690] Sheehan Dep. (8/28/14) Vol. 1 at 137:2-9.
[691] Paustenbach Dep. (6/17/15) at 123:19-22.

Motors $1,072,131.00 and Daimler-Chrysler $820,196.00 for the same 2003 to 2015 period.[692]

380. According to Ford Motor Co., Exponent billed Ford $18,241,726.10 from 2001 to 2015.[693] The costs for the litigation defense publications are set forth in the following table:

[692] Paustenbach Dep. (6/17/15) at 123:11-18.

[693] Ford Motor Company's First Supplemental Responses to Plaintiffs' Supplemental Request for Production to Defendant Regarding Payments, *In Re: All Asbestos Litigation Filed by Maune Raichle Hartley French & Mudd, LLC v. Ford Motor Company et al.*, Cause No. 95 AS ALL LIT, In The Circuit Court, Third Judicial District Madison County, Illinois (September 9, 2015).