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Court of Appeals

of the

State of New York

In re New York City Asbestos Litigation:

MARY JUNI, as Administratrix for the Estate of ARTHUR H. JUNI, JR., and MARY JUNI, Individually,

Plaintiff-Appellant,

– against –

A.O. SMITH WATER PRODUCTS CO., *et al.*,

Defendants,

and

FORD MOTOR COMPANY

Defendants-Respondent.

BRIEF OF CONCERNED PHYSICIANS AND SCIENTISTS REGARDING CAUSATION OF ASBESTOS-RELATED DISEASE, AS *AMICI CURIAE* IN SUPPORT OF APPELLANT

John Ned Lipsitz, Esq.
LIPSITZ & PONTERIO, LLC
Attorneys for proposed Amicus Curiae for Dr. Philip J. Landrigan and other concerned physicians and scientists
424 Main Street, Suite 1500
Buffalo, New York 14202
Tel.: (716) 849-0701
Fax: (716) 849-0708

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STATEMENT OF INTEREST

We write to express our understanding of what counts as a "scientific expression" of significant-contributing-factor causation that is "generally accepted as reliable in the scientific community," in the context of a vehicle mechanic's occupational exposures to dusts from asbestos-containing vehicle-related products such as brakes, clutches and gaskets.

The signers of this paper collectively possess hundreds of years of experience researching, diagnosing, and treating asbestos-related diseases in workers and their families, and/or are well versed in these issues.[1] Many of us have published extensively in this field for more than 40 years and have conducted dozens of epidemiological and other studies into the issues of asbestos and disease, and have also testified before legislative and regulatory bodies, and in court proceedings, regarding asbestos and disease, including mesothelioma.

As mainstream scientists who have studied asbestos outside the context of litigation, we are knowledgeable concerning the causal relationship between exposure to asbestos fibers and mesothelioma and other asbestos-related diseases. We deem it irrefutable that repeated exposures to asbestos-containing dust from automotive brakes, clutches and gaskets, over a period of years, would contribute to

[1] This paper is being signed by physicians and scientists who reviewed the document, support the contents and asked to have their name listed. The signers have received no compensation for their participation in this paper.

a person's total dose and to that person's risk or probability of developing mesothelioma and other cancers. This is scientific fact.

We assume for purposes of these comments that Mr. Juni was diagnosed with mesothelioma about 2012, that he was occupationally exposed to asbestos from new and used brakes, clutches and gaskets over a period of approximately 25 years as a mechanic maintaining a fleet of approximately 500 Ford vehicles, and that those exposures began in approximately 1964, about 48 years prior to his diagnosis.[2] Assuming this to be the case, these exposures would have been many orders of magnitude above the miniscule amount of exposure Mr. Juni may have received from "ambient" or "background" asbestos.

Accordingly, given our understanding of the disease mesothelioma and the asbestos exposures Mr. Juni experienced, we are convinced that, outside the context of litigation, any objective scientist would deem Mr. Juni's exposures to such asbestos-containing dusts to have increased his risk of contracting mesothelioma.

Courts should view scientific issues in a way that is consistent with how the mainstream scientific community approaches these issues, and not in the light of rhetorical frameworks fabricated by lawyers. The argument made by some lawyers that, unless there is a specific, statistically significant, epidemiological study of a

[2] We take no position regarding the accuracy of the evidence regarding Mr. Juni's exposures.

particular job title, or of *a particular asbestos-containing product*, then a scientist cannot reach scientifically sound conclusions regarding the consequences of exposure to asbestos in a particular individual, is incorrect and not accepted by the mainstream, non-litigation scientific community.

Moreover, in the context of decades of occupational asbestos exposures from working with automotive brakes, clutches and gaskets, reliance on certain "negative" general epidemiologic "studies of mesothelioma" and industry-financed meta-analyses of some of those studies to claim that these types of asbestos exposure are harmless, leads to scientifically insupportable assertions, particularly when those studies are irrelevant to the exposures at issue and statistically insignificant.[3]

There is also no scientific merit to the claim that a precise mathematical dose of exposure to a particular product must be calculated as a requisite to a scientist's valid causation opinion. Nor do scientific assessments about the cumulative effect of 25 years of repeated occupational exposures equate to an opinion that each such

[3] We place "negative" in quotations because the industry characterization of many of these studies is opportunistic and ignores the overriding limitations of the studies as well as data in the studies that is inconsistent with the characterization. A concise description of the flaws of the approach of the asbestos-defendants to this question in the context of asbestos exposures sustained by mechanics is set forth by Kay Teschke – an author of one of the papers most heavily relied upon by the asbestos companies – in a peer-reviewed letter to the editor in *Annals of Occupational Hygiene* (Teschke, 2016). As noted by Dr. Teschke, with respect to the asbestos defendants' myopic focus on "job-title" specific studies and their limitations when examining jobs – like mechanics – where job duties, and therefore exposures, vary greatly between individuals: "[t]he question is whether the exposure caused the disease. A job cannot cause disease, its exposures may."

exposure or "every fiber" constitutes a "substantial contributing factor" in causing a mesothelioma.[4]

When scientists making causal determinations take into consideration the diagnosis, the latency period, the medical and occupational history, biological plausibility, and relevant case reports, industrial hygiene studies, individual risks and susceptibilities, case series, and statistical epidemiological studies, they have followed the appropriate methodology used by mainstream scientists outside of litigation. To require more would defy long standing generally accepted scientific principles of etiology.

SUMMARY OF ARGUMENT

In the case of a "signature" and sentinel asbestos-caused disease, such as mesothelioma, the mainstream scientific community views causation as a function of cumulative exposure. The cumulative exposure approach looks at the entirety of an individual's exposures, including their qualities (potential range of magnitude, regularity and frequency), over the course of that individual's exposure period. This mainstream scientific approach does not, under any circumstances, mean that "any exposure, no matter how slight," is deemed a significant causative factor. The frequency and regularity of an individual's exposures to an asbestos-containing

[4] It is important to note that one gram (approximately 1/28th of an ounce) of chrysotile asbestos contains from 10 to 100 *trillion* fibers. Therefore, the entire notion of a "single fiber" of exposure is not based in reality.

product or products, along with the latency period between exposure and manifestation of disease, are critical, and a causal assessment is based on the "cumulative exposure" to the particular products "viewed as a whole" considering, quantitatively, semi-quantitatively, or qualitatively, all the exposures that a worker might have had.

> Asbestos is a variety of natural fibrous, incombustible silicate minerals whi[redacted] are linked to asbestosis, a progressive lung-scarring disease, and to cancer of the lungs and other organs. The hazard of asbestos is linked to the inhalation of many small airborne fibers of this chemically stable mineral which cannot be metabolized. The health hazard increases as number of inhaled fibers increases (concentration of fibers in inhaled air x length of exposures x number of times exposures have occurred = inhaled dose; i.e., increased dose = increased risk). [5]

The assertion that an expert's sound testimony about medically significant, real world exposures is nothing more than the theory that any exposure/single fiber is a significant contributing factor seriously misrepresents the cumulative exposure principle, and the reality of how workers are exposed to asbestos in real life. Biologically, an individual's diagnosed asbestos disease is the result of the cumulative effect of his lifetime dose of exposures to asbestos. Even 1986 and 1995

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[5] Appendix X (excerpt from Ford Motor Company Industrial Relations Bulletin No. 4 (October 14, 1986) (FAFD0011183). Ford's own industrial hygiene studies published in the early 1970s show that a mechanic cleaning truck brakes would be exposed to over 7 million asbestos fibers in his breathing zone. D.E. Hickish & K.L. Knight, *Exposure to Asbestos During Brake Maintenance*, 113 ANN. OCCUP. HYG. 17-21 (1970). Indeed, Hickish & Knight found the time-weighted average exposure of the truck mechanic to be 1.75 fibers per cubic centimeter of air, or 1,750,000 asbestos fibers in the cubic meter of air in the mechanic's breathing zone. This level is over 17 times the current OSHA exposure limit and would result in millions of fibers of asbestos being breathed by the mechanic every day.

Ford Motor Company internal policy documents acknowledged that cumulative exposure is a cornerstone of medicine and science.

ARGUMENT

I. ONGOING EXPOSURES TO ASBESTOS DUSTS, INCLUDING DUSTS RELEASED FROM AUTOMOTIVE BRAKES, CLUTCHES AND/OR GASKETS, CONTRIBUTE TO AN INDIVIDUAL'S TOTAL DOSE OF ASBESTOS

It is a scientific fact that an individual's ongoing significant occupational, domestic and/or environmental exposures to asbestos contribute to a person's total dose.[6] It is also well established that, as cumulative exposure to asbestos increases, the risk of mesothelioma (and other asbestos diseases) increases. The exposure-response relationship is unquestionable.[7]

Studies, including those performed by NIOSH and others, and those in the peer-reviewed literature, have shown that work on asbestos-containing brakes released significant levels of airborne asbestos. For example, one study performed by researchers at Mt. Sinai School of Medicine included the photograph of a mechanic in New York City using compressed air to blow out automotive brake dust, clearly illustrating the substantial exposures some mechanics experienced:

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[6] The exposure categories of "significant occupational, domestic or environmental" exposure derives from the Consensus Report, *Asbestos, Asbestosis, and Cancer, the Helsinki criteria for diagnosis and attribution 2014*, 41 SCAN. J. WORK ENVIRON. HEALTH 5, 6 (2015).

[7] The mainstream scientific community is in consensus that all forms of asbestos can and do cause mesothelioma. Therefore, the issue of the relative potency of the fiber types is not germane in considering an individual mesothelioma patient's case.

A table collecting data from many of these studies and published in the *International Journal of Occupational and Environmental Health* illustrates this point:

TABLE 1
Studies Showing High Asbestos Exposures During Brake Work

Author	Year	Exposure Type	Exposures Reported
Lee [Published]	1970	Blow out	3-5 f/cc
Boillat & Lob [Published]	1973	Brake work undefined	0.3-29.2 f/cc

TABLE 1
Studies Showing High Asbestos Exposures During Brake Work

Author	Year	Exposure Type	Exposures Reported
Castleman & Ziem [citation omitted]	1985	Damp rag Squirt bottle Stoddard Solvent Dry rag Brake washer	High: 2.6 f/cc; TWA: 0.28 f/cc High: 0.54 f/cc; TWA: 0.21 f/cc High: 0.68 f/cc; TWA: <0.1 f/cc High: 0.81 f/cc; TWA: 0.2 f/cc High: 1.1 f/cc
Hatch [citation omitted]	1970	Compressed Air	Fibers >5 um: 2.1-8.2; 10 minute avg: 0.8
K. Redelsperger [Published]	1986	Passenger car (various operations) Truck (various operations)	Mean: 3.8-4.7 f/cc Mean: 4.4-9.9 f/cc
Kauppien & Korhonen [Published]	1987	Truck (various operations) Grinding	<0.1-125 f/cc; TWA: 0.1-0.2 f/cc 7 f/cc
Hickish[Ford study, unpublished]	1968	Auto blow out	Peak exposure: 7.09 f/cc
Hickish [Ford study, unpublished]	1968	Auto brake work, various	TWA: 1.57-2.55 f/cc
Clark[citation omitted]	1976	Auto disc brake change	0.2-1.9 f/cc
Hatfield & Longo[unpublished]	1998	Bendix Chrysler (filling and cleaning)	8.53-14.57 f/cc
Hatfield & Longo[unpublished]	n.d.	Bendix Ford (filling and cleaning)	5.47-12.67 f/cc
Hatfield & Longo[unpublished]	2000	Sweeping and cleaning brake shop	Personal Samples: 7.5-8.8 f/cc

TABLE 1
Studies Showing High Asbestos Exposures During Brake Work

Author	Year	Exposure Type	Exposures Reported
			Area Samples: 2.0-2.4 f/cc
Hatfield, Longo & Newton [unpublished]	2000	Grinding	4.83-12.51 f/cc
Hatfield, Longo & Newton [unpublished]	2000	Hand grinding	12.57-21.43 f/cc
Hatfield, Newton & Longo [unpublished]	2001	Hand sanding	0.5-0.96 f/cc
Rohl et al. [Published]	1977	Blowing dust Beveling	6.6-29.4 f/cc 23.7-72.0 f/cc
Osborn[citation omitted]	1934	Grinding	17 mppcf
Roberts & Zumwalde[NIOSH, unpublished]	1982	Compressed air	0.14-15.0 f/cc
Lloyd [citation omitted]	1975	Servicing brakes	3.75-37.3 f/cc
Longo, Mount & Hatfield [unpublished]	2004	Hand sanding and grinding and other operations	19.7-35.7 f/cc [8]

Ford's 1968 study showing peak exposures of 7.09 f/cc (fibers per cubic centimeter) means that in the cubic meter around the mechanic blowing out a brake drum, there were over seven million (7,000,000) asbestos fibers. Consistently, the U.S. EPA determined that merely hitting a brake drum with a hammer or wiping a

[8] Egilman, *Fiber Types, Asbestos Potency, and Environmental Causation, A Peer Review of Published Work and Legal and Regulatory Scientific Testimony*, IJOEH 15:202-28 (Table 1) (2009) (internal citations omitted).

brake drum with a dry rag or brush can release millions of asbestos fibers in the garage environment.[9] Exposures from automotive work do not involve a "single" fiber; a gram of brake dust contains billions or trillions of fibers.[10]

II. SCIENTISTS EMPLOY A MULTIFACETED DIAGNOSTIC APPROACH TO ASSIGN CAUSATION OF ASBESTOS-RELATED DISEASE, BUT NEVER REQUIRE A PRECISE QUANTIFICATION OF AN INDIVIDUAL'S EXPOSURE LEVEL

Consideration of exposure history, latency period, individual risks and susceptibilities, biological plausibility, relevant case reports and case series, industrial hygiene studies, and statistical epidemiological studies, is a widely accepted scientific methodology for ascribing causation. Applying this methodology to the facts of a particular individual with a demonstrated mesothelioma is deemed sufficient evidence of causation in the non-litigation scientific world.

1. Occupational history

Using work histories to identify significant harmful exposures has ancient roots in occupational medicine.[11] Collecting and evaluating the patient's qualitative history of exposure in determining the cause of the patient's disease has been the

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9 U.S. Environmental Protection Agency, *Guidance for Preventing Asbestos Disease Among Auto Mechanics*, EPA-560-OPTS-86-002 (June 1986).

10 Richard A. Lemen, *Asbestos in Brakes: Exposure and Risk of Disease*, 45 AM. J. IND. MED. 229, 230 (2004).

11 *See* Bernardino Ramazzini, De Morbis Artificum Diatriba (1713), Trans. by W.C. Wright IN A.L. BIRMINGHAM, CLASSICS OF MEDICINE LIBRARY (1983), in MEDICINE IN QUOTATIONS: VIEWS OF HEALTH AND DISEASE THROUGH THE AGES 276 (Edward J. Huth & T.J. Murray, eds., 2nd ed. 2006).

driving force behind the discovery and development of knowledge regarding the dangers of asbestos and countless other substances.

In the seminal 1960 Wagner study,[12] thirty-three (33) individual cases of mesotheliomas were reported from a South African asbestos mining town. Thirty-two (32) of those cases had a known qualitative history of exposure to asbestos. There were no exposure measurements nor was there any statistical epidemiological analysis. Nearly half of the mesothelioma cases were from environmental and household exposures, rather than from working at the mine. For nearly 50 years, the mainstream scientific community has widely considered the Wagner study to have cemented the causal relationship between asbestos and the very rare disease mesothelioma.[13]

Similarly, in his seminal 1965 paper on determining "general" causation, Sir Austin Bradford Hill emphasized the importance of exposure history used in

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12 J.C. Wagner, *et al., Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province*, 17 BRIT. J. INDUS. MED. 260 (1960).

13 The first epidemiological cohort study on mesothelioma was published in 1963. This landmark study provided to epidemiologists and other public health professionals proof of a statistical association. Thomas F. Mancuso, *et al., Methodology in Industrial Health Studies: The Cohort Approach, with Special Reference to an Asbestos Company*, 6 ARCH. ENV. HEALTH 210 (1963).

conjunction with existing literature to determine a causal link, and cautioned that no one type of scientific evidence overrides the others.[14]

Internationally respected asbestos disease specialists reached a scientific and medical consensus, originally in 1997[15] and reaffirmed in 2014,[16] regarding the methodology for experts to attribute a given mesothelioma to asbestos exposure. One of the stated purposes of the Helsinki Criteria meeting was to create a standardized methodology for causal attribution of asbestos related diseases (including mesothelioma) in published studies to facilitate comparisons of international asbestos disease literature. The original 1997 committee, with over 1,000 scientific articles published between them, agreed in the "Helsinki Consensus" with the settled approach in occupational medicine that "a history of significant occupational,

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[14] Sir Austin Bradford Hill, *The Environment and Disease: Association or Causation?* Proceedings of the Royal Society of Medicine 295 (1965) ("There are, of course, instances in which we can reasonably answer those questions from the general body of medical knowledge . . . a particular chemical is known to be toxic to man and therefore suspect on the factory floor"); *see* David H. Kaye & David A. Freedman, *Reference Guide on Statistics*, *in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 211, 221 n.24 (3d ed. 2011) (citing Hill, *The Environment and Disease*). Sir Austin Bradford Hill's credentials and his appreciation of the strengths and weaknesses of statistical epidemiology cannot be questioned. He also co-authored the seminal statistical epidemiological paper regarding smoking and lung cancer. Richard Doll & A. Bradford Hill, *Smoking and Carcinoma of the Lung: Preliminary Report*, 2 BRIT. MED. J. 739-48 (1950).

[15] A. Tossavainen, *et al., Consensus Report: Asbestos, Asbestosis, and Cancer: The Helsinki Criteria for Diagnosis and Attribution*, 23 SCAND. J. WORK ENVIRON. HEALTH 311 (1997).

[16] Henrik Wolff, *et al., Consensus Report: Asbestos, Asbestosis, and cancer, the Helsinki Criteria for Diagnosis and Attribution 2014: Recommendations,* 41 SCAND. J. WORK ENVIRON. HEALTH 5 (2015).

domestic or environmental exposure will suffice for attribution" in an individual with mesothelioma.

From the medical and scientific perspective, to unnecessarily require a near-impossible quantification of a precise individual dose of asbestos would be a public health travesty, all the more so where the patient had repeated, significant exposures to the signature causal agent for mesothelioma.

2. Individual Susceptibility

Another important factor is individual susceptibility. 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.[17] On the other hand, comparatively low level exposures, including those to chrysotile asbestos, in the absence of occupational exposures, has been shown to induce high incidences of mesotheliomas in family groups that also have a germ-line BAP-1 mutation.[18]

A diagnosis of mesothelioma, combined with a significant occupational, domestic or environmental exposure history consistent with the Helsinki Consensus,

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[17] J. 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).

[18] Joseph R. *Testa, et al., Germline BAP1 Mutations Predispose to Malignant Mesothelioma,* 43 NATURE GENETICS (2011). This is *not* to suggest that the BAP-1 mutation is necessary for an individual to develop mesothelioma after comparatively low-level cumulative exposure.

is certainly proof of the body's defenses being overwhelmed and defeated. The capacity of the body's defense is influenced heavily if not defined by individual susceptibility.

3. Biological Plausibility

The emerging consensus among physicians and scientists is that most forms of cancer develop in a multistage process. This process typically involves a number of genetic mutations, and these mutations must involve particular genes and/or follow in some particular sequence for the cell(s) in question to become a fully cancerous cell which develops into a tumor: "[It is] . . . now recognized that asbestos fibers themselves are carcinogenic, mainly by indirect mechanisms, and that malignant transformation is a multi-stage process."[19]

Scientists have identified "established mechanistic events" in the development of asbestos-induced mesothelioma. These are initiated and promoted by the combined effects of repeated exposures and the ongoing effects of past exposures, all of which combine over time to cause the ultimate cancer. Put simply, the consensus position of the non-litigation scientific community is that the ultimate cancer results from the accumulated damage caused by the cumulative exposures sustained over a lifetime by the individual with cancer.

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[19] *See generally* Samuel P. Hammar, *et al., Neoplasms of the Pleura, in* 2 DAIL AND HAMMAR'S PULMONARY PATHOLOGY VOLUME II: NEOPLASTIC LUNG DISEASE (Joseph F. Tomashefski, Jr., *et al.*, eds., 3rd ed. 2008), at 587-599.

In this respect, cumulative exposure is merely a corollary of the ancient concept of dose-response.[20] "Toxicologists generally posit two main dose-response curves: those that have a 'threshold' and those that do not . . . The second general type of a dose-response curve is one that is considered to have no threshold. The most important example for toxic torts is that of cancer. The underlying cause of many cancers is a persistent genetic mutation allowing the unbridled growth of a cell which then results in a clone of cancer cells."[21,22]

A substantial body of evidence demonstrates that inhaled chrysotile asbestos (such as that found in brakes, clutches and gaskets), reaches the extra pulmonary sites where mesothelioma develops.[23] Elevated levels of asbestos, free of any commercial amphibole asbestos, have been found in the lungs of mechanics. See

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[20] Bernard D. Goldstein, *Toxic Torts: The Devil is in the Dose*, 16 J.L. & POL'Y 551 (2008). Bernard D. Goldstein is Professor of Environmental and Occupational Health and former Dean of the University of Pittsburgh Graduate School of Public Health. Professor Goldstein is also the lead author of the *Reference Guide on Toxicology*, *in* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 669-670 (Federal Judicial Center, 3d ed. 2011).

[21] *Id.* at 554-55.

[22] *See also* Goldstein & Heniftn, *supra*, at 669-670 (explaining that threshold exposure analysis "*is not applied to substances that exert toxicity by causing mutations leading to cancer*") (emphasis added).

[23] *See* Yasunosuke Suzuki & Steven R. Yuen, *Asbestos Tissue Burden Study on Human Malignant Mesothelioma*, 39 INDUS. HEALTH 150 (2001) ("The majority of asbestos types seen in the mesothelial tissues were chrysotile alone"); Ronald F. Dodson, *et al., Analysis of Asbestos Fiber Burden in Lung Tissue from Mesothelioma Patients*, 21 ULTRASTRUCTURAL PATHOLOGY 321 (1997); R.E. Gordon & S. Dikman, Asbestos Fiber Burden Analysis of Lung and Lymph Nodes in 100 Cases of Mesothelioma, 179 AM. J. RESPIRATORY & CRITICAL CARE MED. A5892 (Apr. 2009).

Finkelstein (2008); Gordon & Dikman (2009); Sanyal, *et al.* (2017).[24] While fiber burden studies are not necessary to demonstrate causation, the weight of reliable evidence shows that vehicle mechanics with mesothelioma can have elevated tissue burdens of asbestos from chrysotile products (chrysotile and/or tremolite/actinilite) with no commercial amphibole asbestos.

4. Case Reports, Case Series, and Sentinel Events/Signature Diseases

As noted by Hill more than 50 years ago, no one form of scientific evidence overrides any or all of the others. There are numerous examples of observational epidemiological studies providing significant insight into and/or proof of a causal connection between an agent and a disease.[25] Such case reports and case series also support the attribution position taken in the Helsinki Consensus.

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[24] Soma Sanyal, *et al., Mesothelioma with No Evidence of Commercial Amphibole Asbestos Exposure -- 35 Cases with Chrysotile, Non-Commercial Amphibole or Asbestiform Talc by Lung Fiber Burden Analysis*, 195 AM. J. RESPIRATORY & CRITICAL CARE MED. A3863 (2017).

[25] Select examples include: (A) the first observation of an occupational cancer, cancer of the scrotum caused by work as a chimney sweep, was based case reports. Percivall Pott, Chirurgical Observations: Observations on the Cancer of the Scrotum (London: Hawes, Clark, and Collins, 1775); (B) the fact that contaminated water caused cholera was determined by a case series. On the communication of cholera by impure Thames water, Snow, J. 9 Med. Times and Gazette 365-66 (Oct. 7, 1854); (C) the fact that a particular bacillus could cause cholera was determined from an autopsy case report. Osservazioni microscopiche e deduzioni patologiche sul cholera asiatico (Microscopic observations and pathological deductions on Asiatic cholera, F. Pacini, Gazzetta Medica Italiana: Toscana, 2nd Series), 4(50) : 397-401 ; 4(51): 405-412 (1854); and (D) the fact that asbestos exposure could cause pulmonary fibrosis was determined from an autopsy case report. W.E. Cooke, *Pulmonary Asbestosis*, 2 BRIT. MED. J. 1024-25 (1927).

Mesothelioma is a signature malignancy for asbestos exposure. This has long been generally accepted in the scientific community.[26] Mesothelioma was listed as a sentinel health event of occupational origin in the U.S. over thirty years ago.[27] The agent responsible was identified as asbestos and the industry/occupation of concern was "[a]sbestos industries and utilizers."[28] In the United States, there are no other proven substantial causes of malignant mesothelioma.

Regarding the specific scientific validity of consideration of case reports in looking at asbestos related cancer, Phillip Enterline, M.D., in a report funded by and generated for the industry group Asbestos Information Association, stated:

> the clinicians and pathologists who contributed to the early literature . . . showed remarkable insight into the meaning of the observations . . . Even a single case report is a kind of epidemiological observation, since the basis of such reports is usually a feeling, unexpressed, that the case is somehow aberrant for a human population.[29]

Regulatory bodies such as the Department of Labor's Occupational Safety & Health Agency (OSHA), after conducting a comprehensive literature review, have

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[26] Wagner, *supra*; Irving J. Selikoff, *Opening Remarks*, 132 ANN. N.Y. ACAD. SCI. 7 (1965); Margaret R. Becklake, *Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice*, 114 AM. REV. RESP. DISEASE 187 (1976); World Health Organization (WHO), International Agency for Research on Cancer (IARC), IARC Monographs on the Evaluation of the Carcinogenic Risks for Humans, Chemical and Industrial Processes Associated with Cancer in Humans: IARC Monographs, Volumes I to 20, Supp. 1 (1979); 100C World Health Organization, International Agency for Research on Cancer, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, A Review of Human Carcinogens, Part C: Arsenic, Metals, Fibres, and Dusts 1-40, 219-309 (2012).

[27] David D. Rutstein, *et al., Sentinel Health Events (Occupational): A Basis for Physician Recognition and Public Health Surveillance*, 73 AM. J. PUBLIC HEALTH 1054 (1983).

[28] *Id.* at 1056.

[29] PHILIP E. ENTERLINE, ASBESTOS AND CANCER: THE FIRST THIRTY YEARS (1978).

found and reported case reports in scientific literature showing very short exposures being capable of causing mesothelioma: "Asbestos exposures as short in duration as a few days have caused mesothelioma in humans."[30]

5. Statistical Epidemiological Studies

There is overwhelming consensus in the medical and scientific communities that exposure to all forms of commercial asbestos can induce mesotheliomas based, in part, on peer-reviewed, published statistical epidemiological studies.[31] Mesothelioma is caused by asbestos, 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 statistical epidemiology study be available for an opinion regarding causal attribution to be well-founded.

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[30] 27 United States Department of Labor, Occupational Safety & Health Administration (OSHA), Safety and Health Topics, Asbestos, https://www.osha.gov/SLTC/asbestos/ (accessed March 12, 2015) (citing E. Skammeritz, *et al.*, *Asbestos Exposure and Survival in Malignant Mesothelioma: A Description of 122 Consecutive Cases at an Occupational Clinic*, 2 INT. J. OCCUP. ENVIRON. MED. 224 (2011)); Morris Greenberg & T.A. Lloyd Davies, *Mesothelioma Register 1967-68*, 31 Brit. J. Ind. Med. 91 (1974); World Health Organization (WHO), International Agency for Research on Cancer (IARC), IARC Monographs on the Evaluation of the Carcinogenic Risks to Humans, Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42, Supplement 7 106-116 (1987); and John T. Hodgson & Andrew Damton, *The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure*, 44 ANN. OCCUP. HYG. 565 (2000).

[31] 31 IARC 2009, *supra*.; IARC 2012, *supra*.; Joint Policy Committee of the Societies of Epidemiology, Position Statement on Asbestos from the Joint Policy Committee of the Societies of Epidemiology (JPC-SE) (2012).

It is well-accepted in the scientific and epidemiological community that, "when there are multiple asbestos exposures, each contributed to cumulative exposure and hence to the risk and causation of M[alignant] M[esothelioma], within an appropriate latency interval."[32] While researchers have found some other factors important in analyzing the asbestos mesothelioma dose-response curve, such as time since first exposure, total cumulative dose consistently is the best indicator of risk: "In this study…the dose-response seemed to be described best by Cumulative Exposure Index."[33]

Human dose-response studies show statistically significant increased risk of mesothelioma at very low levels of cumulative exposure. These studies have found statistically significant increased risk of 269% to 790% based on exposures well below the cumulative working lifetime exposures under the current OSHA regulatory exposure limit.[34] *See e.g.* 2014 LaCourt, *et al.*, ≤ 0.1 f/cc/years, Odds

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[32] Hammar, *supra*. This is consistent with the multi-stage development of mesothelioma at a cellular and genetic level discussed above.

[33] Jean Bignon, *et al.*, HISTORY AND EXPERIENCE OF MESOTHELIOMA IN EUROPE, IN MESOTHELIOMA 29¬53 (Bruce W.S. Robinson & A. Philippe Chahinan, eds., 2002).

[34] While the OSHA "permissible" level is a useful bookend for looking at comparative exposure levels and risk, the "permissible" exposure levels under the OSHA regulatory exposure limit have long been acknowledged to carry a substantial increased risk of cancer. *See generally* United States Dept of Labor – OSHA, at https://www.osha.gov/SLTC/asbestos/#4 (visited Aug. 18, 2017) ("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") (omitting numerous citations). These more recent studies demonstrate that a massive increased risk exists well below the levels "permitted" by OSHA.

Ratio (OR) 4.0 (99% Confidence interval (CI) 1.9-3.3)[35]; 2001 Rodelsberger, *et al*., ≤ 0.15 f/cc/years, OR 7.9 (95% CI 2.1-30.0)[36]; 2014 Offermans, *et al*., ≤ 0.2 f/cc/years, Hazard Ratio (HR) 2.69 (95% CI 1.60 o4.53)[37]; 2017 Jiang, *et al.*, 0 – 0.5 f/cc/years, OR 28 (95% CI 6 – 137)[38]; and the 1998 Iwatsubo, *et al.*, 0.5 - 0.99 f/cc/years, OR 4.0 (95% CIf 2.0-8.8).[39,40]

Statistical epidemiological studies provide additional support for a weight-of-the-evidence conclusion that asbestos from brakes – and certainly from clutches and asbestos gaskets – can and does cause mesothelioma. Epidemiological studies and case reports document mesothelioma in persons whose primary exposure to

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35 A. LaCourt, *et al., Occupational and Non-Occupational Attributable Risk of Asbestos Exposure for Malignant Pleural Mesothelioma*, THORAX 1 (2014).

36 Klaus Redelsperger, *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. INDUS. MED. 262 (2001).

37 Nadine S.M. Offermans, *et al., Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherlands Cohort Study*, 56 J. OCCUP. ENVIRON. MED. 6 (2014).

38 Zhaoqiang Jiang, *et al., Hand-spinning chrysotile exposure and risk of malignant mesothelioma: a case-control study in Southeastern China*, Accepted Article, INT. J. CANCER doi 10.1002/ijc.31077 (Sept. 26, 2017).

39 Y. Iwatsubo, *et al., Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study*, 148 AM. J. EPIDEMIOL. 133 (1998).

40 Again, the limitations of the available historic exposure measurements preclude any non-speculative attempt to quantify the alleged potency differences between the various types of asbestos. Silverstein *et. al., Developments in asbestos cancer risk assessment*, 15 AM J IND MED 850–858 (2009). Chrysotile compromised the overwhelming majority of asbestos used historically, and the consensus position of the non-litigation scientific community is that all forms of asbestos can and do cause mesothelioma, and that there is a lack of an identifiable threshold for mutagenic carcinogens.

asbestos is from asbestos-containing brake materials.[41] From an industrial hygiene perspective, it does not take long, at levels known to exist in garages using asbestos brakes, clutches and gaskets without asbestos dust control, for a person to receive significant exposures.

These and other asbestos exposure and disease studies in brake workers were reviewed by Dr. Richard Lemen, Assistant Surgeon General (Ret.) in 2004. Dr. Lemen concluded that "[e]ven the so-called 'controlled' use of asbestos-containing brakes poses a health risk to workers, users, and their families."[42] Recently, a cancer registry-based study in Massachusetts reported a greater than two-fold excess of mesothelioma in auto mechanics.[43] For another epidemiological example, an analysis of mortality in bus drivers and bus maintenance workers in Genoa, Italy, demonstrated significant excess deaths (SMR 3.67) from pleural mesothelioma

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41 D. McDonald, *et al.*, *Epidemiology of Primary Malignant Mesothelial Tumors in Canada*, 26 CANCER 914 (1970); A. E. Anderson, et al., Asbestos Emissions from Brake Dynamometer Tests, SAE Technical Paper 730549 (1973); K. Redelsperger, *supra*; M. Huncharek, *et al., Pleural Mesothelioma in a Brake Mechanic*, 46 BRIT. J. IND. MED. 69 (1989); Iwatsubo, *supra*; Kay Teschke, *et al., Mesothelioma Surveillance to Locate Sources of Exposure to Asbestos*, 88 CANADIAN J. PUB. HEALTH 163 (1997).

42 Lemen, *Asbestos in Brakes*, *supra*.

43 Cora R. Roelofs, *et al., Mesothelioma and Employment in Massachusetts: Analysis of Cancer Registry Data 1988-2003*, 56 AM. J. IND. MED. 985 (2013).

when compared to Italian males.[44] A recent review of the Australian Mesothelioma Registry found that vehicle mechanics had elevated lifetime risk of mesothelioma and that "[c]hrysotile-only exposures were noted in 4% of the cases" where one of the main types of exposure was through occupational exposure in automotive mechanic work.[45]

Moreover, in their 1999 study "Work-related cancer in the Nordic countries,"[46] Aage Andersen, *et al.*, found a statistically significant increased risk of pleural cancer (SIR = 149 (95% CI 114 –191) in engine and motor operators. These scientists reported that "[t]he elevated risk of pleural cancer can be considered an indication of asbestos exposure, most importantly from brake linings."[47]

Brake manufacturers and their consultants invent their own "scientific rule" that a collection of approximately 18-22 "negative" friction studies and industry-

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[44] Domenico Franco 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). The choice of comparison population can influence the findings regarding mesothelioma in an urban or working population. There was not an increased risk in the maintenance workers when compared to the population in the immediate surrounding area, because the surrounding region contained several shipyards that historically used asbestos and, accordingly, had a high rate of mesothelioma. This illustrates the need for thoughtful analysis when interpreting study findings or individual exposure histories.

[45] Soeberg, M, *et al., Malignant Mesothelioma in Australia 2015: Current incidence and asbestos exposure trends*, J. TOXICOL. ENVIRON. HEALTH, Part B, 19, 5-6, 173 – 189 (2016).

[46] A. Anderson, *et al., Work-related cancer in the Nordic Countries*, 25 SCAN. J. WORK ENVIRON HEALTH 1 (supp. 2, 1999).

[47] *Id.* at 73.

financed meta-analyses thereof trumps all other evidence.[48] But for a study or studies to be truly "negative" in the sense claimed 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.[49] Moreover, it is highly preferable that a study be specifically designed to detect an increase in risk for the disease in question. The "negative" friction studies in question fail on this count as well.

IARC's Criteria for Causality also succinctly explains why these so-called "negative" studies, when properly interpreted, cannot reliably exclude the existence of a risk of mesothelioma from automotive-related asbestos exposure:

> Such a judgment requires first that . . . 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, . . . 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

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[48] Importantly, the interpretation of these "negative" studies as negative despite the limitations of the studies is often contrary to the interpretation of the authors of the very studies and at times the studies themselves. *See, e.g.,* Teschke (2016), *supra* (rejecting industry consultant reliance upon her 1997 study as "negative" – discussed further below); H.J. Woitowitz & K. Redelsperger, *Mesothelioma Among Car Mechanics*, 38 ANN. OCCUP. HYG. 635 (1994) (study expressly notes its lack of power to detect a small but real risk); E.S. Hansen, *Mortality of Auto Mechanics – A Ten-Year Follow-up*, 15 SCAN. J. WORK ENVIRON. HEALTH 43 (1989) (expressly stating increased risk).

[49] Median latency since first exposure for mesothelioma is 38.4 years. *See* A. Reid, *et al., Mesothelioma Risk after 40 Years since First Exposure to Asbestos: a Pooled Analysis*, 69 THORAX 843 (2014).

periods substantially shorter than 30 years cannot provide evidence for lack of carcinogenicity.[50]

Most of the studies relied on by industry were not designed to evaluate the risks of working with asbestos-containing auto parts, suffer from serious and well-documented problems with exposure misclassification, are seriously under-powered to detect elevated risk (due to small size and the rarity of mesothelioma), and do not adequately address latency,[51] and ironically lack information on the dose levels for the subjects in the study.[52]

The National Cancer Institute cogently summarized these issues, noting: "[s]tudies evaluating the cancer risk experienced by automobile mechanics exposed to asbestos through brake repair are limited, but the overall evidence suggests there is no safe level of asbestos exposure."[53]

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

[51] Median latency since first exposure for mesothelioma is 38.4 years. *See* Reid, *supra*.

[52] We note the lack of information concerning individual dose levels in such studies *not* because such information is necessary to ascribe causation – as we have discussed above, it is not. Rather, we note this to highlight the fact that the studies asbestos industry advocates claim to rely upon lack exposure quantification they otherwise claim to be necessary.

[53] National Cancer Institute, Asbestos Exposure and Cancer Risk (2009), at https://www.cancer.gov/about-cancer/causes-prevention/risk/substances/asbestos/asbestos-fact-sheet (visited June 14, 2017). A comprehensive survey of the brake literature is not the intent of this brief. However, Amici incorporate by reference: Lemen, *Asbestos in Brakes, supra*; David S. Egilman & Marion A. Billings, *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability*, 11 INT. J. OCCUP. ENVIRON. HEALTH 360 (2005); and Laura S. 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).

It is our understanding the one of the "negative" friction studies relied upon by Ford or its experts in this case was a 1997 study by Kay Teschke *et al*. In her letter to the editor of the *Annals of Occupational Hygiene*, Vol. 60 (2016), at pages 528-530, Dr. Teschke wrote to express her concern that her 1997 study "had been used regularly in litigation related to mesothelioma in people who worked as vehicle mechanics or in brake repair" (p. 528). She carefully explained that "the jobs or tasks [that were] being used as surrogates of chrysotile exposure" are not, in fact, "good surrogates," hence creating "problems in interpretation of epidemiological evidence such as ours in these cases" (*id*.).

Specifically, Dr. Teschke explained that, whereas "[s]ome occupations are synonymous with extensive exposure to certain agents" (*e.g.*, "it would be a rare wood furniture maker who did not have daily high exposure to hard wood dust"), no such exposure relationship holds "[i]n the case of vehicle mechanics, [for whom] brake work is not consistently performed" (pp. 528-529). Indeed, most of the studies referenced by friction defendants such as Ford "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 had chrysotile exposures similar to background levels in the population" (p. 529). Hence, continued Dr. Teschke, industry use of the results of the studies and in the meta-analyses "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" (p. 529).

Additionally, a recent editorial by the editor of *Annals of Occupational Hygiene* asked the proper rhetorical question in the context of an asbestos-industry expert study of the risks of cancer from asbestos in brakes:

> 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 exclude that there is no effect of a known carcinogen within that occupation*.[54]

Vermeulen explained the correct approach to causality when it comes to asbestos exposure:

> So how should we view these analyses on job titles with known carcinogenic exposures? Clearly, such analyses cannot be used to inform on the carcinogenicity of known carcinogenic exposures. In other words, 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.[55]

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[54] R. Vermeulen, , *When are Risk Analyses on Job Titles Informative?,* 60 ANN. OCCUP. HYG. 913–915 (2016) (emphasis added).

[55] *Id.*

A person exposed to a known carcinogen, such as asbestos used in brakes, is at increased risk of mesothelioma, just as is the insulator or pipe fitter exposed to asbestos from products used in their trades. It is the exposure, not the job title or trade, that causes mesothelioma. As the father of modern asbestos medicine in the United States, Irving Selikoff, noted in 1964: "Asbestos exposure in industry will not be limited to the particular craft that utilizes the material. The floating fibers do not respect job classifications."[56]

In sum, we emphasize that: (1) strong scientific and medical evidence supports our opinion that asbestos from asbestos-containing brakes, clutches and gaskets can and does cause mesothelioma; (2) patient-specific dose information is not necessary to reliably attribute a mesothelioma to exposure to asbestos from brakes, clutches and gaskets; and (3) an individual's history of significant occupational, domestic, and/or environmental, above-background exposure to asbestos with sufficient latency is sufficient to reliably attribute that person's mesothelioma to such exposures. Based on the assumptions about the patient history outlined above, given the years of exposure to high levels of asbestos from brakes, clutches and gaskets, it is scientifically reasonable to attribute that person's mesothelioma to the asbestos exposure from that work.

[56] Irving J. Selikoff, *et al., Asbestos Exposure and Neoplasia*, 188 J. AM. MED. ASSOC. 142, 146 (1964).

CONCLUSION

It is axiomatic that, while any particular isolated exposure to asbestos will not necessarily be a significant contributing factor, ongoing exposures to asbestos contribute to the cumulative total dose. An expert's concurrence with this scientific fact should not "infect" his testimony such that his causation opinion is negated, especially where, as here, the experts employed the generally accepted method of a multi-faceted review of the diagnosis, medical and occupational history, individual susceptibility, biological plausibility, and relevant case and epidemiological studies. Nor should the expert's mere recognition that non-relevant, statistically insignificant, and industry-funded studies or meta-analyses "exist" be deemed to undermine his or her testimony concerning causation.

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Respectfully submitted,

John Ned Lipsitz, Esq.
LIPSITZ & PONTERIO, LLC
Appearing for proposed Amicus Curiae for Dr. Philip J. Landrigan and other concerned physicians and scientists
424 Main Street, Suite 1500
Buffalo, New York 14202
(716) 849-0701

EXHIBIT "A"

AMICI

Abby Lippman, PhD
Professor Emerita
Department of Epidemiology, Biostatistics, and Occupational Health
McGill University
Montreal, Quebec, Canada

Annie Thebaud Mony
Emerite Director of Research, National Institute for Health and Medical Research (INSERM)
Interdisciplinary Research Group on Occupational Cancer (GISCOP 93)
Paris-13 University
Bobigny, France

Arthur L. Frank MD, PhD
Drexel University School of Public Health
Philadelphia, Pennsylvania, USA

Barry Castleman, ScD
Environmental Consultant
Garrett Park, Maryland, USA

Bruce P. Lanphear, MD, MPH
Professor, Faculty of Health Sciences
Simon Fraser University
Vancouver, British Columbia, Canada

Celeste Monforton, DrPH, MPH
Professorial Lecturer, Department of Environmental & Occupational Health
Milken Institute School of Public Health, George Washington University, and
Lecturer, Department of Health & Human Performance
Texas State University
San Marcos, Texas, USA

Colin L. Soskolne, Ph.D.
Professor Emeritus, University of Alberta, Edmonton, Canada
Adjunct Professor, Health Research Institute, University of Canberra, Australia
Fellow, American College of Epidemiology
Fellow, Collegium Ramazzini

Daniel Thau Teitelbaum, MD
Adjunct Professor
Environmental and Occupational Health
Colorado School of Public Health
Aurora, Colorado, USA

Dario Consonni, MD, PhD
Epidemiology Unit, Fondazione IRCCS Ca' Granda –
Ospedale Maggiore Policlinico
Milan, Italy

Dario Mirabelli, MD
Epidemiologist at the Cancer Epidemiology Unit
Città della Salute e della Scienza Hospital
Turin, Italy

David Egilman, MD, MPH
Clinical Professor
Department of Family Medicine, Brown University
Attleboro, Massachusetts, USA

David F. Goldsmith, MSPH, PhD
Georgetown and George Washington Universities
Washington, District of Columbia, USA

David Ozonoff, MD, MPH
Professor of Environmental Health
Boston University School of Public Health
715 Albany Street, Talbot West Room 430
Boston, Massachusetts, USA

David Rosner, MPH, PhD
Lauterstein Professor
Columbia University
Mailman School of Public Health
New York, New York, USA

Fiorella Belpoggi, MD
Director, Research Department
Cesare Maltoni Cancer Research Center
Ramazzini Institute
Bologna, Italy

James Huff, PhD, Guest Researcher
Formerly, Associate Director for Chemical Carcinogenesis
National Institute of Environmental Health Sciences
Research Triangle Park, North Carolina, USA

John Heinzow, MD
CR Emeritus
Dr. med. Birger Heinzow
formerly: State Agency for Social Services
Dept. Health Protection
Kiel, Germany

John M. Dement, Ph.D., CIH
Professor Emeritus
Division of Occupational & Environmental Medicine
Department of Community & Family Medicine
Duke University School of Medicine
Durham, North Carolina, USA

John Coulter Maddox, MD, Pathologist
Senior Partner, Peninsula Pathology Associates, Inc.
Riverside Regional Medical Center
Newport News, Virginia, USA

Karl T. Kelsey, MD, MOH
Fellow, Collegium Ramazzini

Kathleen Ruff, MA*
Honorary Fellow, Collegium Ramazzini
Ottawa, Canada

Kenneth D. Rosenman, MD, FACE, FACOEM, FACPM
Chief of the Division of Occupational and Environmental Medicine
Professor of Medicine
Michigan State University
East Lansing, Michigan, USA

L. Christine Oliver, MD, MPH, MSA
Adjunct Professor, Division of Occupational and Environmental Health
Dalla Lana School of Public Health, University of Toronto, Ontario, Canada
Associate Clinical Professor of Medicine, Harvard Medical School, Boston, Massachusetts, USA (Ret.)
President, Occupational Health Initiatives, Inc.
Brookline, Massachusetts, USA

Laura Welch, MD, FACP, FACOEM
Center for Construction Research and Training
Silver Spring, Maryland, USA

Leslie Thomas Stayner, PhD
Professor of Epidemiology
Division of Epidemiology & Biostatistics
University of Illinois at Chicago
School of Public Health (M/C 923)
Chicago, Illinois, USA

Morris Greenberg, MB, FRCP, FFOM
London, England, United Kingdom

*as corrected

Nachman Brautbar, MD
Clinical Professor Emeritus of Medicine
University of Southern California, Keck School of Medicine
Board Certified, American Board of Forensic Medicine
Board Certified, American Board of Internal Medicine
Board Certified, American Board of Nephrology
Member, Society of Toxicology
Los Angeles, California, USA

Philip J. Landrigan, MD, MSc
President, Collegium Ramazzini
Department of Environmental Medicine, Public Health
Icahn School of Medicine at Mount Sinai
New York, New York, USA

Prof. Dr. med. Xaver Baur, MD
President of European Society for Environmental and Occupational Medicine (EOM Society)
Fellow, Collegium Ramazzini
Em. University of Hamburg, School of Medicine
Former Chair of Occupational Medicine
Berlin, Germany

Prof. em. Dr. med. Hans-Joachim Woitowitz
Former head of the Institute and Outpatient Clinic of Occupational and Social Medicine of the University of Giessen, Germany
Giessen, Germany

Professor Bice Fubini
President of "G. Scansetti" Interdepartmental Center for Studies on Asbestos and other Toxic Particulates, University of Torino
Torino, Italy

Richard Kradin, MD
Massachusetts General Hospital
Associate Professor of Pathology and Medicine
Harvard Medical School
Cambridge, Massachusetts, USA

Dr. T.K. Joshi, MBBS, MS, MSc
Occupational Medicine
London, England, United Kingdom

Theresa S. Emory, MD, Pathologist
Partner, Peninsula Pathology Associates, Inc.
Riverside Regional Medical Center
Newport News, Virginia, USA

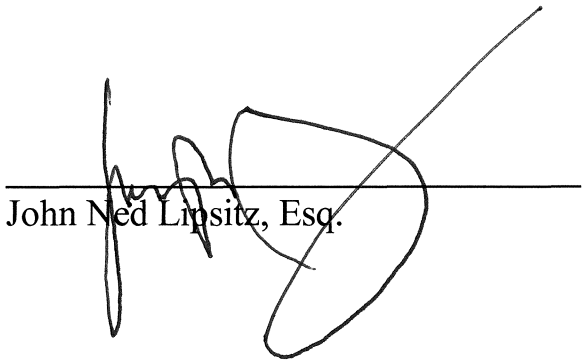
Thomas H. Gassert, MD, MSc
Assistant Professor of Medicine
University of Massachusetts Medical School
Adjunct Assistant Professor of Medicine
Dartmouth School of Medicine
Visiting Scientist and Medical Residency Advisory Committee Ombudsman
Environmental and Occupational Medicine and Epidemiology Program
Harvard T.H. Chan School of Public Health
Cambridge, Massachusetts, USA

Tony Fletcher, PhD
Associate Professor in Environmental Epidemiology,
Department of Social and Environmental Health Research,
London School of Hygiene & Tropical Medicine
London, England, United Kingdom

Yv Bonnier Viger, MD, MSc, MM, FRCPC
Médecin spécialiste en santé publique et médecine préventive,
Directeur régional de santé publique de la Gaspésie et des Îles, Québec
Professeur, Département de médecine sociale et préventive
Faculté de médecine, Université Laval
Quebec, Canada

CERTIFICATE OF COMPLIANCE

I hereby certify that the foregoing *Amicus Curiae* Brief complies with the word limitation as set out in Rule 500.13(c)(1) because it contains 6,975 words; and complies with the typeface and type-style requirements of Rule 500.1(j) because it has been prepared using Microsoft Word in a proportionally-spaced typeface with 14-point Times New Roman font.



John Ned Lipsitz, Esq.