APL-2017-00114
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# Court of Appeals

**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., AERCO INTERNATIONAL, INC., AGCO CORPORATION f/k/a and as Successor in interest to MASSEY-FERGUSON, INC., AIR & LIQUID SYSTEMS CORPORATION, as successor-
*(Caption continued on inside cover)*

## *BRIEF AMICUS CURIAE* OF DR. JOHN HENDERSON DUFFUS, RONALD E. GOTS, M.D., PROFESSOR ARTHUR M. LANGER,, PROFESSOR ROBERT NOLAN, DR. GORDON L. NORD, ALAN JOHN ROGERS and PROFESSOR EMANUEL RUBIN IN SUPPORT OF RESPONDENT FORD MOTOR COMPANY

Martin S. Kaufman (NY Bar No. 1704824)
Atlantic Legal Foundation
500 Mamaroneck Avenue, Suite 320
Harrison, NY 10528
(914) 834-3322
mskaufman@atlanticlegal.org

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by-merger to BUFFALO PUMPS, INC., AMCHEM PRODUCTS, INC., n/k/a RHONE POULENC AG COMPANY, n/k/a BAYER CROP SCIENCE INC., ARVINMERITOR, INC., Individually and as successor-in-interest to ROCKWELL AUTOMOTIVE, BMCE, INC. f/k/a UNITED CENTRIFUGAL PUMP, BOISE CASCADE CORPORATION, BORG-WARNER CORPORATION, by its successor-in-interest, BORG-WARNER MORSE TEC, INC., BW/IP, INC. and its wholly owned subsidiaries, CARLISLE CORPORATION, CATERPILLAR, INC., CBS CORPORATION, f/k/a VIACOM INC., successor by merger to CBS CORPORATION, f/k/a WESTINGHOUSE ELECTRIC CORPORATION, COURTER & COMPANY INCORPORATED, CRANE CO., CUMMINS ENGINE COMPANY, INC., DANA COMPANIES, LLC, DEERE & CO., DENTSPLY INTERNATIONAL, INC., Individually and as Successor to DENTSPLY AUSTENAL and DENTSPLY CERAMCO, EATON CORPORATION, as successor-in-interest to CUTLER HAMMER, INC., EMPIRE-ACE INSULATION MFG. CORP., FEDERAL-MOGUL ASBESTOS PERSONAL INJURY TRUST, as successor to FELT PRODUCTS MFG., CO., FEDERAL-MOGUL ASBESTOS PERSONAL INJURY TRUST, as successor to the former VELLUMOID INC., division of FEDERAL-MOGUL CORPORATION, FLOWSERVE CORPORATION, Individually and Solely as Successor to Durco, Durion; BW/IP, Anchor Darling, Superior Group, Pacific Pumps, Sier-Bath Pumps, Edward Vogt, Vogt Valves, Nordstrom Valves and Edward Valve, Inc.; FLOWSERVE US, INC., Solely as Successor to Rockwell Manufacturing Company, Edward Valve Inc., Nordstrom Valves, Inc., Edward Vogt Valve Company and Vogt Valve Company, FMC CORPORATION, on behalf of its former CHICAGO PUMP & NORTHERN PUMP BUSINESSES,

*Defendants,*

*—and—*

FORD MOTOR COMPANY,

*Defendant-Respondent,*

*—and—*

FOSTER WHEELER, L.L.C., GENERAL ELECTRIC COMPANY, GOULDS PUMPS, INC., HARLEY-DAVIDSON, INC., HONEYWELL INTERNATIONAL, INC., f/k/a ALLIED SIGNAL, INC./BENDIX, IMO INDUSTRIES, INC., INGERSOLL-RAND COMPANY, INTERNATIONAL TRUCK AND ENGINE CORPORATION, ITT CORPORATION, ITT INDUSTRIES, INC., Individually and as successor to BELL & GOSSETT COMPANY and as successor to KENNEDY VALVE MANUFACTURING CO., INC. and as successor to GRINNELL VALVE CO., INC., KELSEY HAYES COMPANY d/b/a TRW, KENNEDY VALVE MANUFACTURING CO., INC.,

KENTILE FLOORS, INC., KERR CORPORATION d/b/a KERR DENTAL CORPORATION, Individually and as successor by merger to KERR MANUFACTURING COMPANY, KORODY-COLYER CORPORATION, LIPE-AUTOMATION CORP., MACK TRUCKS, INC., MAREMOUNT CORP., MCCORD CORPORATION, Individually and as successor in interest to A. E. CLEVITE, INC. and J.P. INDUSTRIES, INC., MOTION CONTROL INDUSTRIES, INC., as predecessor in interest to CARLISLE CORPORATION, O'CONNOR CONSTRUCTORS, INC., f/k/a THOMAS O'CONNOR & CONNOR & CO., INC., OWENS-ILLINOIS, INC., PACCAR, INC., Individually and through its division, PETERBILT MOTORS CO., PARKER-HANNIFIN CORPORATION, PEERLESS INDUSTRIES, INC., PERKINS ENGINES, INC., PFIZER, INC. (PFIZER), PNEUMO ABEX, LLC, successor in interest to ABEX CORPORATION (ABEX), RAPID-AMERICAN CORPORATION, RESEARCH-COTTRELL, INC., ROGERS CORPORATION, SEQUOIA VENTURES, INC., f/k/a BECHTEL CORPORATION, SPIRAX SARCO, INC. Individually and as successor to SARCO COMPANY, STANDARD MOTOR PRODUCTS, INC., THE FAIRBANKS COMPANY, THE J.M. NEY COMPANY, TRANE U.S. INC., f/k/a AMERICAN STANDARD, INC., TREADWELL CORPORATION, TYCO INTERNATIONAL (US) INC., Individually and as Successor to Hancock Valves and Lonergan Valves and Yarway Corporation and Grinnell Corporation, U.S. RUBBER COMPANY (UNIROYAL), UNION CARBIDE CORPORATION, UNITED CONVEYOR CORPORATION, WARREN PUMPS, LLC, WEIL-MCLAIN, a division of The Marley-Wylain Company, a wholly owned subsidiary of The Marley Company, LLC, WESTINGHOUSE AIR BRAKES COMPANY, f/k/a UNION SWITCH & SIGNAL CO, WHIP MIX CORPORATION, YARWAY CORPORATION, YUBA HEAT TRANSFER, LLC.,

Defendants.

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## INTEREST OF *AMICI CURIAE*

*Amici* are scientists who have studied the role that scientific issues play in public affairs and in particular the way in which they can illuminate disputes between different persons or elements of society in courts of law. *Amici* include physicians, chemists, geologists, epidemiologists and toxicologists.[1]

*Amici* also believe that the decisions of trial court and the Appellate Division correctly evaluated the testimony of plaintiff's causation experts as against scientific principles and apprehends the current state of scientific knowledge and scientific methodology.

None of the *amici* is employed by, is receiving funding from, or has testified within the prior 10 years as an expert for, any of the parties in this case.

*Amici curiae* affirm that no counsel for any party authored this brief in whole or in part, no person other than *amici curiae* or their counsel participated in the preparation or submission of this brief, and no party or counsel made a monetary contribution to fund the preparation or submission of this brief.

*Amici* submit this brief to explain why the causation theories espoused by Plaintiff's experts do not conform to the standards accepted in the scientific community and do not satisfy the criteria for proving causation in toxic tort cases set forth in *Parker v. Mobil Oil Corp.,* 7 N.Y.3d 434 (2006), *Cornell v. 360 W.*

[1] The credentials of *amici* are set forth in the Biographical Appendix hereto.

*51st St. Realty, LLC*, 22 N.Y.3d 762 (2014), and *Sean R. v. BMW of N. Am., LLC*, 26 N.Y.3d 801 (N.Y. 2016).

## STATEMENT OF THE CASE

*Amici* adopt Ford's statement of the case, but highlight the salient facts below.

**Mr. Juni's Exposure to Asbestos-Containing Products**

Mr. Juni recalled servicing Ford vehicles for much of his working life. However, he did not recall how frequently he worked with asbestos-containing products made, sold or distributed by Ford. (See, *e.g.*, A407-10, 427, 433-37, 563, 568-69, 580.) Mr. Juni described extensive exposure to asbestos-containing products from other sources. (See, *e.g.*, A390-402, 405-407.) He remembered that he worked with replacement parts made by many suppliers besides Ford. (A458-59.) As time went on, much of Mr. Juni's work was supervisory, and not directly with brake and clutch parts. See, *e.g.*, A446.

**Testimony of Plaintiff's General Causation Expert**

Plaintiff's general causation expert Dr. Steven Markowitz testified that exposure to asbestos in automotive friction products increases a person's risk of contracting mesothelioma. (A294-95.)

Dr. Markowitz conceded that the available epidemiological studies addressing asbestos exposure in vehicle mechanics "do not show much evidence

in support of a relationship between mesothelioma and exposure to friction products" (A114), he relied instead on several industrial hygiene studies as the basis for his opinion. Markowitz relied heavily on the presence of dust in Mr. Juni's workplaces as evidence of the amount of asbestos to which Mr. Juni was exposed. But Markowitz could not establish that the "dust" actually contained active asbestos fibers capable of causing mesothelioma, the volume of dust, or the actual number or volume of asbestos fibers in the "dust," let alone the number or volume of asbestos fibers from Ford products. (A251.)

**Testimony of Plaintiff's Specific Causation Expert**

Dr. Jacqueline Moline, Plaintiff's expert on specific causation, testified that Mr. Juni's mesothelioma was caused by exposure to asbestos in Ford products based on her assumption that: (1) during his working years from 1964 to 1988, Mr. Juni "personally and regularly" assisted in performing brake and clutch work, including on Ford brakes and clutches; (2) Mr. Juni assisted in removing original Ford brakes and clutches and replacing them with new Ford brakes and clutches; and (3) Mr. Juni's work created and exposed him to visible asbestos dust.[2]

[2] The evidence at trial did not support the assumption that Mr. Juni "personally and regularly" performed brake or clutch work that generated dust, let alone dust containing respirable asbestos fibers. Most of Mr. Juni's brake and clutch work consisted of assisting or supervising other mechanics. (A426-27, 449.)

Dr. Moline she did not quantify or not attempt to offer "a scientific expression" of Mr. Juni's actual exposure to asbestos from Ford products. She conceded that she had no information on the issue. (A1157.) She also did not know whether any of the asbestos fibers from friction products to which Mr. Juni was allegedly exposed were still biologically active (that is, whether they had biologic potential to cause mesothelioma). (A1199.)

Dr. Moline testified that friction products in general "contained virtually all chrysotile," that "[c]hrysotile causes mesothelioma" (A1095-98) and that because Mr. Juni performed work on friction products installed in Ford vehicles, Ford, along with every other friction product manufacturer Mr. Juni recalled in his deposition, is responsible for Mr. Juni's mesothelioma. (A1102) ("There's no way to say it's not my company's product or one[] company's product and it's everyone else's. It all goes to the cumulative exposure that one has that causes the disease.").

Dr. Moline assumed that whenever Mr. Juni was exposed to "visible dust," the dust contained asbestos (which she further assumed constituted a "significant exposure." (A1095.) Her opinion was not based on any scientific inquiry into frequency, intensity, or proximity of exposure. Dr. Moline's opinion did not distinguish between the differences in toxicity among the various fiber types to

which Mr. Juni was exposed; instead, she treated all of Mr. Juni's alleged exposures as having similar potency. (A1095-97.)

**The Decision Below**

The Appellate Division understood that *Parker* and its progeny require plaintiff "to prove not only that Mr. Juni's mesothelioma was caused by exposure to asbestos, but that he was exposed to sufficient levels of the toxin from his work on brakes, clutches, or gaskets, *sold or distributed by defendant*, to have caused his illness." (A5-6, emphasis supplied.)

The Appellate Division concluded that the Junis' evidence was insufficient because both Markowitz and Moline "effectively testified only in terms of an increased risk and association between asbestos and mesothelioma" and "failed to either quantify the decedent's exposure levels or otherwise provide any scientific expression of his exposure level with respect to Ford's products." (A7.)

The Appellate Division rejected the Junis' argument that application of the *Parker* standard to asbestos cases would make recovery impossible, noting correctly that "[e]ven if it is not possible to quantify a plaintiff's exposure, causation from exposure to toxins in a defendant's product must be established through some scientific method, such as mathematical modeling based on a plaintiff's work history, or comparing the plaintiff's exposure with that of subjects of reported studies." (A6-7.) The Appellate Division also rejected the

argument that asbestos toxic tort cases should be treated differently from toxic tort cases involving exposures to other substances because "there is no valid distinction to be made between the difficulty of establishing exposure to, say, benzene in gasoline and exposure to asbestos. In each type of matter, a foundation must be made to support an expert's conclusion regarding causation." A10.

## SUMMARY OF ARGUMENT

*Parker* addressed the standard for causation testimony in toxic tort cases: plaintiff's experts must present a scientific expression of exposure coupled with scientific studies showing that such exposures are sufficient to cause disease. This Court reaffirmed those requirements in *Cornell* and *Sean R*.

Those criteria are consistent with the approach taken by most scientists in assessing disease causation. The testimony of plaintiff's causation experts did not satisfy professional standards and accepted scientific techniques and reasoning, nor did it meet criteria set forth in *Parker* and its progeny.

Plaintiff's experts' testimony that every workplace exposure to dust from an asbestos-containing product is a cumulative part of the overall dose and therefore each exposure is causative does not enable a court to determine whether any particular defendant is responsible for plaintiff's injury.

In this case, the trial court and the First Department correctly applied *Parker* to asbestos causation testimony.

This Court should affirm the decision of the Appellate Division, First Department.

## ARGUMENT

### I. PLAINTIFFS' EXPERTS' METHODOLOGY AND CONCLUSIONS ARE INCONSISTENT WITH ACCEPTED SCIENTIFIC METHOD

Dr. Moline's methodology is unacceptable in the relevant scientific community of experts in the fields of toxicology and epidemiology of asbestos and the diseases caused by asbestos for numerous reasons.

First, Dr. Moline did not consider the physical, chemical and toxicological differences among various types of asbestos. She ignored the overwhelming evidence that chrysotile asbestos, the type used in automobile brakes and the type to which Mr. Juni was exposed has far less, and maybe nil, potential to cause mesothelioma, compared with other types of asbestos.

Second, Dr. Moline ignored the large body of toxicological studies by official government and other disinterested investigators that shows that chrysotile asbestos has a very small potential for causing mesothelioma, and that in its use in automotive brakes and clutches it undergoes physical and chemical changes that render it even less potent as a cancer-causing agent.

Third, Dr. Moline ignored the generally accepted distinction between general causation and specific causation. Her methodology does not even establish general causation for chrysotile asbestos, and the scientific consensus is that a finding of general causation for chrysotile asbestos is doubtful. Where there is no proof of general causation, there is no basis for finding specific causation.

Fourth, Dr. Moline did not consider the dose or level of exposure of Mr. Juni. Determining the minimum threshold of exposure levels is critical to any consideration of medical causation.

Empirical testing is the hallmark of scientific methodology. The idea that even small amounts of certain types of asbestos fibers contribute to mesothelioma is not new. However, it is merely an hypothesis that is incapable of direct verification.[3] To the extent that epidemiologists and toxicologists have empirically tested that conjecture, they have found it to be unverified.

Each asbestos fiber in a person's lungs *does not* lead to mesothelioma. If it did, we would all die from mesothelioma because all of us, even without occupational exposures, have millions of asbestos fibers in our lungs, even in

[3] Scientific methodology is based on generating hypotheses and testing them to see if they can be falsified. "[T]he criterion of the scientific status of a theory is its falsifiability, or refutability, or testability." K. Popper, *Conjectures and Refutations: The Growth of Scientific Knowledge* 37 (5th ed. 1989). *See also* C. Hempel, *Philosophy of Natural Science* 49 (1966) ("[T]he statements constituting a scientific explanation must be capable of empirical test.").

early childhood, yet only a very small percentage of people develop mesothelioma.[4] Dr. Moline's approach and assumptions are absurd *unless* a minimum threshold of fiber levels for causing harm is specified. In this case that was not even attempted.[5]

The risk for mesothelioma causation is very different for different types of asbestos; the risk is different even among chrysotile asbestos from different sources. An understanding of these differences must inform a scientist's attribution of causation. This, too, was ignored by Dr. Moline.

An implication of Dr. Moline's testimony is that even small amounts of asbestos can cause mesothelioma. This is sometimes referred to as the "single fiber" hypothesis: every cancer, or other adverse medical condition, starts with the inhalation of "one fiber."[6] This remains an unproven hypothesis and

[4] Asbestos is found in the lungs of most people everywhere. In the United States, chrysotile is commonly found in the lung parenchyma of the general population, (A. M. Langer, R. P. Nolan, *Chrysotile Biopersistence in the Lungs of Persons in the General Population and Exposed Workers*, 102 Environmental Health Perspectives, Supplement 5 at 235 (1994)) and in the ambient air, ®. J. Thompson, *Ambient Air Monitoring for Chrysotile in the United States*, National Bureau of Standards, Special Publication 506, Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods 355 (1978)).

[5] As emphasized by Sir Austin Bradford Hill many years ago,, an "association" is not the same as "causation." Association is but one element, usually the first element, in establishing causation. Epidemiology recognizes that certain considerations must be assessed in order for an expert to make a valid determination of causation. If an association exists scientists consider the criteria such as Bradford Hill's guidelines to evaluate whether an epidemiologic association is consistent with a causal association in a particular individual. Those considerations are: (1) consistency; (2) strength of association; (3) dose response; (4) biological plausibility; (5) coherence; (6) temporality; (7) specificity; (8) analogy; and (9) experimentation. See A.B. Hill, *The Environment and Diseases: Association and Causation*, 58 Proc. Royal Soc. Med., Sec. Occup. Med. 295 (1965).

[6] The *amicus* brief of Concerned Physicians and Scientists in Support of Plaintiff-Appellant

(continued...)

speculative. This hypothesis has not advanced to the level of a generally accepted theory. Moreover, it overlooks the important questions of cumulative exposures and asbestos type and instead focuses on which fiber out of the totality of the exposure is responsible for the plaintiff's disease.[7]

Courts do not and should not accept unproven, and untestable, theory as evidence. Plaintiff contends otherwise, but that position is, in the opinion of *amici,* incorrect.

### A. General Causation and Specific Causation

General causation addresses the question of whether exposure to the agent of concern has ever caused the disease in question. This is usually discussed by showing that a group of people with high levels of exposure have developed the adverse outcome, significantly more frequently than among a otherwise similar unexposed group. If general causation cannot be proven, then it is superfluous to ask the specific causation question.

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[6](...continued)
quarrels with the phrase "single fiber theory," pointing out that a small volume of air can contain millions of asbestos fibers. Be that as it may, plaintiff's theory in this case, as in many others, fails to quantify, even approximately, the number of fibers in the pleura of the injured person, what type of fibers were found, the approximate number to which the plaintiff was exposed over time, the minimum number or volume of fibers traceable to a particular defendant, and the threshold number of fibers needed to cause the disease.

[7] Many scientists would argue that each exposure adds, not to the disease, but to the probability of the disease. The risk or probability of developing a disease is proportional to the extent of the exposure or cumulative exposure. Therefore, scientists have emphasized a difference between a practical threshold below which it is unknown whether a risk exists and a theoretical threshold which is generally used in a regulatory setting. No epidemiologic studies exist which have the ability to assess the risk due to extremely low levels of exposure, and therefore there is no practical threshold.

If general causation is established then specific causation can be addressed for the exposure history specific the individual and to the case. Specific causation asks whether a particular individual developed a disease as a result of exposure to the agent. It is obvious that this requires knowledge of the individual's exposure level. Dr. Moline was not familiar with Mr. Juni's asbestos exposure history; indeed, her description of Mr. Juni's exposure was not consistent with Mr. Juni's testimony.

The cumulative exposure theory implies a complete rejection of the generally accepted distinction between general and specific causation. Proponents of that theory contend that because "asbestos" can cause mesothelioma, anyone with any exposure to any asbestos type developed his mesothelioma as a result of that exposure. This is circular reasoning. Dr. Moline has conflated a known association between certain exposures to certain types of asbestos and Mr. Juni's the disease, with no evidence of a causal relationship in the specific individual who may or may not have experienced a substantial exposure to the toxin.

**B. Exposure to Which Asbestos Type?**

Dr. Moline did not discuss the important steps in establishing whether the type of asbestos associated with brake materials is accepted as a cause of

mesothelioma. In the opinion of *amici*, her testimony is insufficient to prove general causation.

Even if one were to accept the "cumulative exposure" hypothesis, we first must consider "Cumulative exposure to what?" It has long been known, and has become ever clearer in the last 10 to 15 years, that we must distinguish between different minerals called "asbestos." Asbestos fibers crystallize in a way that makes them more biologically activity than fragments of the bulk mineral. But these asbestiform fibers behave very differently as between different types of asbestos.

Two major groupings are important, "amphibole asbestos" (actinolite, amosite, anthophyllite, crocidolite, and tremolite) and "serpentine asbestos" (chrysotile). Chrysotile asbestos has historically been the dominant type of asbestos used commercially and is the only asbestos type still in commerce worldwide. All of the amphibole asbestos minerals have been out of world commerce since 1997. Moreover, the dominant form of asbestos used in brakes has been chrysotile asbestos. Therefore Mr. Juni almost certainly was exposed only to chrysotile asbestos from Ford products.

The differences in carcinogenic potency for mesothelioma causation between the major commercial asbestos types has been known and generally accepted since at least 1965. In his lecture on asbestos-related disease, Dr. John

C. Gilson found that the largest number of mesothelioma deaths occurred among workers exposed to crocidolite. Among workers with high exposure to chrysotile, small numbers, or zero, mesothelioma cases were reported. J. C. Gilson, Wyers Memorial Lecture 1965, *Health Hazards of Asbestos, Recent Studies on its Biological Effects*, 16 Trans. Soc. Occup. Med. 62 (1966).

Hodgson and Darnton estimated the mesothelioma causing potency of chrysotile, amosite, and crocidolite as 1:100:500, meaning amosite and crocidolite are, respectively, 100 and 500 times more potent in causing mesothelioma than chrysotile. J. T. Hodgson and A. Darnton, *The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure*, 44 Ann. Occup. Hyg. 565 (2000) ("Hodgson and Darnton 2000"). In a later paper, Hodgson, *et al.* show that the amphibole asbestos types (amosite and crocidolite) explain the mesothelioma distribution and that chrysotile has zero weight, indicating that chrysotile is unlikely to be responsible for any of the mesothelioma cases diagnosed in Great Britain. J. T. Hodgson, D. M. McElvenny, A. J. Darnton, M. J. Price and J. Peto, *The Expected Burden of Mesothelioma Mortality in Great Britain from 2002 to 2050*. 92 Brit. J. Cancer 587, at 590, Fig 5A (2005).

The United States Environmental Protection Agency commissioned a study by Berman and Crump which reached a similar conclusion. D. W. Berman, K. S.

Crump, *Update of Potency Factors for Asbestos-Related Lung Cancer and Mesothelioma*, 38 Crit. Rev. Tox. (supp 1) 1 (2008); D. W. Berman and K. S. Crump, *A Meta-Analysis of Asbestos-Related Cancer Risk That Addresses Fiber Size and Mineral type*. 38 Crit. Rev. Tox. (supp 1) 49 (2008).

Other investigators have concluded that chrysotile exposure does not cause mesothelioma to any appreciable extent. J. S. Pierce, M. A. McKinley, *et al.*, in *An Evaluation of Reported No-Effect Chrysotile Asbestos Exposures for Lung Cancer and Mesothelioma*, 38 Crit. Rev. Tox. 191 (2008) summarized the cumulative exposure-response data for predominantly chrysotile-exposed cohorts in the published literature and found that the predominance of the cumulative "no-effects" exposure levels for mesothelioma fall in the range of approximately 15-500 fiber per milliliter x years. This number far exceeds one fiber in a lung, which is implied by Dr. Moline's claim that any exposure contributes substantially to mesothelioma. These studies would seem to be free of the general selection bias exhibited by reports of expert witnesses for a party to litigation.[8, 9]

[8] Peer-reviewed, published, scientific literature not prepared for purposes of litigation is an accepted source of evidence. See *Daubert v. Merrell Dow Pharmaceuticals*, 509 U.S. 579, 593 (1993); *see also* J. Ziman, *Reliable Knowledge: An Exploration of the Grounds for Belief in Science* 130 (1978); A. S. Relman and M. Angell, *How Good Is Peer Review?*, 321 New Eng. J. Med. 827 (1989). Studies by academic researchers are generally less susceptible to selection or other biases that reports prepared for litigation.

[9] Dr. Moline did not take into account differences in mesothelioma-causing potency of different fiber types with respect to Mr. Juni's chrysotile-only exposure from Ford brake products in rendering her causation opinion. This was a significant deviation from accepted methodology.

### C. General Causation for a Group

It is not generally accepted that one can usefully discuss whether a specific worker with a specific occupational exposure can get a specific disease before first having a clear discussion about whether workers in that specific occupation with that exposure have a significant excess incidence of that specific disease. An examination of the epidemiological studies finds no reliable evidence that working in the automotive repair with friction products causes mesothelioma. P.A. Hessel, *et al.*, in *Mesothelioma Among Brake Mechanics: An Expanded Analysis of a Case-control Study*, 24 Risk Analysis 547 (2004) studied brake mechanics over their lifetime work in trades with the potential for other asbestos exposures and when these alternative employments with a potential for asbestos exposure are controlled for found that brake work presents no increased risk and the risk did not increase with increasing duration of brake work. Those authors note that "The results are consistent with the existing literature indicating that brake work does not increase the risk of mesothelioma." *Id*.

The studies of motor vehicle mechanics are discussed in David Garabrant, *et al.*, *Mesothelioma Among Motor Vehicle Mechanics: An Updated Review and Meta-Analysis*, 60 Annals of Occupational Hygiene 8 (2015). As the courts below pointed out, 21 of 22 studies showed no increased incidence of mesothelioma among the cohort examined.

### D. Consistency of Epidemiological and Toxicological Findings For Automotive Friction Products

Chrysotile asbestos dust from friction products has a much reduced (possibly zero) biological potential due to the effects of heat. The degradation of the fibers and their loss of activity begins at ~550° C and occurs around 810-820° C. One interpretation of this is that environmental measurements (fibers per cubic centimeter of air) overstate the hazard. See Arthur M. Langer, *Reduction of the Biological Potential of Chrysotile Asbestos Arising from Conditions of Service on Brake Pads*, 38 Regul. Toxicol. Pharmacol. 71 (2003). Data on brake temperatures predict that automotive brake pad surface temperatures routinely exceed 500° C, with asperity temperatures reaching more than 1000° C. Chrysotile heated for 30 days is destroyed between 475 and 500° C, whereas chrysotile heated to 800° C survives for only minutes. *See* P.A. Candela, C.D. Crummett, D.J. Earnest, M.R. Frank, A.G. Wylie, *Low-pressure Decomposition of Chrysotile as a Function of Time and Temperature*, 92 American Mineralogist 1704 (2007).[10]

The experimental toxicological data are consistent with and explain the epidemiological studies showing that there is a large difference between the different asbestos types in producing mesothelioma. The toxicology studies also

[10] On cross-examination, Dr. Moline agreed that 99% of the asbestos in brakes is transformed by the heat from the friction of the braking process into forsterite, and that there is no evidence that forsterite causes mesothelioma. [E. 897-898].

show how the physical-chemical properties of chrysotile are altered when that fiber type is used in automotive brakes and clutches. The reduction in the biological activity of chrysotile by thermal effects explains why exposure to the chrysotile asbestos in brake dust is not associated with an increased risk of asbestos-related disease as compared with the occurrence of such disease in other cohorts with different occupational exposure to chrysotile.

Directly pertinent to after-service friction products such as those alleged in this case to have caused Mr. Juni's mesothelioma, the chrysotile fibers which survive the mechanical and thermal forces to which they are subjected are so profoundly altered that their biological potential is reduced to nil. *See* A.M. Langer, *Reduction of the Biological Potential of Chrysotile Asbestos Arising from Conditions of Service on Brake Pads*, 38 J. Reg. Toxicol. and Pharmacol. 71 (2003).

### E. Plaintiff's Causation Expert Ignored Basic Principles of Toxicology

Dr. Moline ignored the central tenets of toxicology:

(1) "The dose makes the poison." This implies that all chemical agents are intrinsically hazardous – whether they cause harm is only a question of dose. Even water, if consumed in large quantities, can be toxic. *See* Casarett and Doull's *Toxicology: The Basic Science of Poisons* 13 C. D. Klaassen (ed.), 5th ed. 1996); see also E. K. Silbergeld, *The Role of Toxicology in Causation: A*

*Scientific Perspective*, 1 Cts. Health Sci. & L. 374, 378 (1991). As David L. Eaton, *Scientific Judgment and Toxic Torts – A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & Pol'y 5 (2003) explains "Dose is the single most important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect." *Id.* at 11. The dose principle holds true for asbestos:

> Most chemicals that have been identified to have "cancer-causing" potential (carcinogens) do so only *following long-term, repeated exposure for many years. Single exposures or even repeated exposures for relatively short periods of time (e.g., weeks or months) generally have little effect* on the risk of cancer, unless the exposure was remarkably high and associated with other toxic effects.

*Id.* at 9 (emphasis added).

(2) "Evidence of exposure is essential in determining the effects of harmful substances." Bernard D. Goldstein & Mary Sue Henifen, *Reference Manual on Scientific Evidence: Reference Guide on Toxicology* 633, 666 (Fed. Jud. Ctr. 3d ed. 2011). "The more time spent in contact with a toxic substance, or the higher the dose, the greater the organism's response" Toxicology Guide at 681. See also Eaton, *supra*, at 9.

(3) Determining the dose-response relationship is "essential in evaluating a causal connection between an alleged exposure and a particular disease." Eaton, "Scientific Judgment," *supra*, at 18. In order for an opinion on causation to be

reliable, it must be premised on three criteria, each of which depends on a dose-response relationship:

> First, the expert should analyze whether the disease can be related to chemical exposure by a biologically plausible theory. Second, the expert should examine if the plaintiff was exposed to the chemical in a manner that can lead to absorption into the body. Third, the expert should offer an opinion as to whether the dose to which the plaintiff was exposed is sufficient to cause the disease.

Toxicology Guide at 661.

Causation "is based on an assessment of the individual's exposure, including the amount, the temporal relationship between the exposure and disease, and other disease-causing factors. This information is then compared with scientific data on the relationship between exposure and disease" (Toxicology Guide at 665) and "[w]hen an exposure to a chemical is less than that known to produce a toxic response, scientific data cannot, as a rule, support a claim of a causal connection." (The relevant question is "Is any *meaningful* amount [of a toxin] present?" R. E. Gots, Toxic Risks: Science, Regulation, & Perception, at 108, 163 (CRC Press 1993)(emphasis supplied). Many lower-level exposures to asbestos have never been shown to cause disease. The brake worker and mechanic epidemiology is discussed in Defendant-Respondent Ford's brief.[11]

No chrysotile-only cohorts have shown a statistically significant increase in

[11] The mechanic studies are summarized and discussed in David Garabrant, et al., *Mesothelioma Among Motor Vehicle Mechanics: An Updated Review and Meta-Analysis*, 60 Annals of Occupational Hygiene 8 (2015).

mesothelioma from the limited brake exposures or doses comparable to those probably experienced by Mr. Juni.[12]

A "methodology" by which an expert replaces the testing of the expert's hypothesis by epidemiological studies with questionably relevant animal studies, case reports, and unscientific assumptions is at odds with the very essence of the scientific method, by which an hypothesis is "formulated" *and then empirically tested*.

General causation of pleural mesothelioma for the group "brake mechanics" by exposure to chrysotile asbestos from friction products cannot be said to be generally accepted in the relevant scientific community.

**F. Plaintiff Lacks Evidence of Specific Causation**

Plaintiff's evidence of specific causation is even more problematic. When studying specific causation, it is necessary to have some idea of the exposure of the specific individual and the relationship of that exposure to the exposure of the group for which general causation has been established. No such evidence was ever presented by the plaintiff and none was mentioned by Dr. Moline.

Dr. Moline offered her causation opinion without even estimating (1) Mr. Juni's overall asbestos exposure from any product,(2) Mr. Juni's overall asbestos

[12] *See, e.g.,* Jennifer Pierce, et al., *An Evaluation of Reported No-effect Chrysotile Asbestos Exposure for Lung Cancer and Mesothelioma*, 38 Critical Rev. in Toxicology 191 (2008) (summarizing cohorts with chrysotile exposures that did not produce mesotheliomas and identifying a necessary minimum exposure level that is well above exposures of brake mechanics).

exposure from all defendants' products collectively, (3) Mr. Juni's overall asbestos exposure from any product of any one defendant, (4) Mr. Juni's exposure from Ford products, or (5) whether Mr. Juni's cumulative exposure to asbestos from the use of Ford's products exceeded his overall exposure to asbestos from the ambient air.

Dr. Moline never estimated, let alone calculated, how much asbestos Mr. Juni, was exposed to from Ford's products. Dr. Moline's opinions are not based on empirical data, and therefore cannot be considered "scientific." Dr. Moline could have made reasonable estimates of Mr. Juni's exposure based on his work history as an automobile mechanic, but the record is devoid of data regarding his exposure

to friction products. *Parker* demands that an admissible expert opinion be based on such data.

## II. PLAINTIFF'S CAUSATION EXPERTS IGNORED SCIENTIFIC PRINCIPLES AND INSTEAD RELIED ON UNSUPPORTED AND UNTESTED HYPOTHESES

If plaintiff's experts Drs. Moline and Markowitz had applied the principles set out in *Parker* and its progeny, they would be compelled to abandon their causation opinions in low-exposure asbestos cases. Instead, they resort to the *every* exposure or *cumulative* exposure approach. As many courts have found,

and the courts below concluded, neither the *every exposure* approach nor the *cumulative exposure* version used here should be admissible.

### A. Cumulative Exposure Testimony Does Not Comport With Basic Scientific Principles

For Drs. Moline and Markowitz the duration, extent and frequency of exposure – crucial elements of medical causation – are not part of their analysis. This is the fundamental flaw of the *cumulative exposure* theory – it allows the experts to attribute causative potential to minute workplace exposures, i.e., those that have not been shown to cause disease.

They abandon dose, and instead base causation on the regulatory "linear no-threshold" approach used as a conservative assumption by some regulators. That approach *assumes* there is no safe dose of asbestos, rather than requiring evidence proving that assumption. The "linear no-threshold" model is a precautionary approach. It is not a valid basis for assigning liability in tort law.[13]

The trial court was correct in rejecting this testimony under *Parker*, and the First Department was correct in affirming that ruling. As this Court reiterated in *Sean R.:*

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[13] Regulatory agencies often state that there is no known safe dose to justify setting protective limits far below the levels of proven disease occurrence documented in epidemiology studies. Courts have recognized the fallacy of relying on these assumptions to prove causation in a litigation. *See, e.g., Betz v. Pneumo-Abex,* 44A.3d 27, 49 n.25 (Pa. 2012) (citing cases rejecting regulatory linear no-threshold approach to support causation); *see also Sutera v. Perrier Group of Am., Inc*., 986 F. Supp. 655, 666 (D. Mass. 1997) ("there is no scientific evidence that the linear no-safe threshold analysis is an acceptable scientific technique" to determine causation.).

> Although it is sometimes difficult, if not impossible, to quantify a plaintiff's past exposure to a substance, *we have not dispensed with the requirement that a causation expert in a toxic tort case show, through generally accepted methodologies, that a plaintiff was exposed to a sufficient amount of a toxin* to have caused his injuries.

26 N.Y.3d 801, at 812 (emphasis added).

In this case, Plaintiff's experts failed to use any method at all (let alone a scientifically accepted method) for assessing Mr. Juni's dose from his work with friction products. Juni's experts did not quantify or estimate any level of exposure; they did not establish the threshold level below which Mr. Juni's exposures would be inconsequential; and they did not present epidemiology studies showing that exposures similar to Mr. Juni's would cause disease.

### B. The Presence of "Dust" Is Not Probative of Dose

Plaintiffs and their "Concerned Scientists" *amici* refer to brake mechanic exposure studies that purportedly show high levels of fiber/cc exposures. These studies were neither relied on by Plaintiff's experts nor the basis of their causation opinions.

The actual foundation for the plaintiff experts' conclusion that Mr. Juni's exposures were the cause of his disease was testimony that Juni was exposed to dust in his workplaces."Visible dust" became a proxy for "significant dose."

Testimony that visible dust was present at a workplace is no substitute for an actual dose assessment. Such testimony provides no information about how long the dust persisted, whether it was tested and how large and representative a sample of dust was tested, what the constituents of the dust were, how many absestos fibers were present in a given volume of dust, what type(s) of asbestos were in the dust, whether the dust was airborne and respirable. Testimony that a witness observed dust visually does not distinguish between ordinary dust and asbestos-containing dust. Even dust from an asbestos-related activity can contain a wide variety of types and condition of asbestos fibers present (if any). One type of dust could be harmless and another potentially dangerous. A great deal of dust from asbestos-related work activity is often not respirable.

Dr. Moline acknowledged that the amount, duration, and frequency of exposure are critical factors, but she then ignored all of those factors in rendering her opinion, relying just on dust in the room to establish sufficient exposure.

## CONCLUSION

For these reasons, *amici* urge the Court to affirm the rulings below.

Respectfully submitted,

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Martin S. Kaufman (NY Bar No. 1704824
Atlantic Legal Foundation
500 Mamaroneck Avenue, Suite 320
Harrison, NY 105238
(914) 834-3322
mskaufman@atlanticlegal.org
Attorneys for *Amici Curiae*,
John Henderson Duffus, *et al.*

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# BIOGRAPHICAL APPENDIX

**JOHN HENDERSON DUFFUS, B.Sc., Ph.D., D.Sc., C.Sci., C.Chem., F.R.S.C., C.Biol., M.S.B.** is the Director of the Edinburgh Centre for Toxicology and the author of more than 200 publications including books, book chapters, and research papers in refereed journals. He has also helped to organize postgraduate courses on toxicology in the United Kingdom and many other countries, sponsored by international organizations such as the International Programme on Chemical Safety (World Health Organization, United Nations Environmental Programme, and International Labour Organization) and prepared distance learning material and defined terminology in toxicology for the International Union of Pure and Applied Chemistry (IUPAC), and for the United States Society of Toxicology, and for TOXNET, the toxicology internet portal of the United States National Library of Medicine. He has acted as a consultant on toxicology to the World Health Organization and to the European Commission, as well as to the United Kingdom government and to private individuals and international companies. He has never been connected with the asbestos industry in any way although his main area of expertise relates to the carcinogenicity and toxicity of inorganic substances including the various forms of asbestos. He is currently the Chair of the IUPAC Chemistry and Human Health Division Subcommittee on Toxicology and Risk Assessment.

**RONALD E. GOTS, M.D.,Ph.D., DABT**, is CEO of the International Center for Toxicology and Medicine. He is a physician and board certified toxicologist, specializing in toxicology and environmental medicine. He is a member of the Society of Toxicology and the American College of Occupational and Environmental Medicine. Dr. Gots has been Adjunct Professor of Pharmacology at Georgetown University School of Medicine. Dr. Gots has focused on the scientific methods for assessing causation of diseases allegedly associated with chemical and biological agents, to the causal analysis of chemically-induced illnesses, and to workplace exposures, worker protection and environmental risk communication. He has provided medical oversight for chemically-exposed individuals. Dr. Gots has chaired two international symposia on "Multiple Chemical Sensitivities: The State of The Science." He was a member of a United Nations committee convened by the International Programme on Chemical Safety (UNEP-ILO-WHO) to evaluate the "chemical sensitivity" issue. Dr. Gots is the author of six books, and has written chapters in six additional books and has published more than 70 articles on biochemistry and toxicology. Recent book chapters include: "Toxic Risks: Science, Regulation, and Perception;" "Risk Analysis and Communication" in *Occupational, Industrial, and Environmental Toxicology,* and "Applying Principles of Science to Daubert Motions in Toxic Tort Claims" in *2000 Wiley Expert Witness Update*

**ARTHUR M. LANGER, Ph.D.**, is a Professor in the Ph.D. Program in Earth and Environmental Sciences at the Graduate School and University Center of the City University of New York, Director of the Center for Applied Studies of the Environment, and Research Associate in the Department of Earth and Planetary

Sciences of the American Museum of Natural History, New York. He previously was Professor and Director of the Environmental Sciences Laboratory of the Institute of Applied Sciences, Brooklyn College of the City University of New York; Associate Professor at the Center for Polypeptide and Membrane Research, Mount Sinai School of Medicine, New York; Associate Professor of Mineralogy, Department of Community Medicine, Mount Sinai School of Medicine, New York; Science Administrator, Environmental Sciences Laboratory, Mount Sinai School of Medicine; Associate Director of the Environmental Sciences Laboratory, an NIEHS Center, Center for the Study of Biological Effects of Environmental Agents, Mount Sinai School of Medicine; and Head of the Physical Sciences Section, Environmental Sciences Laboratory, Mount Sinai School of Medicine. He received his doctorate in Geology (Mineralogy) from Columbia University. Dr. Langer has studied the biological activity of chrysotile asbestos in brake pads and linings, and he is the author of *Reduction of the Biological Potential of Chrysotile Asbestos Arising from Conditions of Service on Brake Pads*, 38 Reg. Tox. & Pharm. 71 (2003).

**ROBERT P. NOLAN, Ph.D.** received a doctorate in chemistry from The City University of New York in 1986. He was been awarded fellowships from the Stony-Herbert Wald Foundation, National Research Council, Fulbright and the International Union for Pure and Applied Chemistry. He is the Deputy Director of the Center for Applied Studies of the Environment and a member of the doctoral faculty in Chemistry and Earth and Environmental Sciences at The Graduate School and University Center of The City University of New York. He is the author of more than fifty scientific papers and is internationally recognized as an expert in the characterization and health hazard evaluation of asbestos and other minerals.

**GORDON L. NORD, Jr., Ph. D.** was a research mineralogist with 21 years with the U.S. Geological Survey. He received the B.S. degree in geology from the University of Wisconsin at Madison, the M.S. degree in geology from the University of Idaho, and the Ph.D. degree in geology from the University of California at Berkeley. He also held a Royal Society Guest Research Fellowship in the Earth Sciences Department, Cambridge University (England), and a Visiting Fellowship, Clare Hall, Cambridge University. He was elected a Life Fellow of Clare Hall, a College for Advanced Study in the University of Cambridge. He has published over 60 papers in peer-reviewed journals. His main scientific interests at the USGS. were the broad characterization of both natural and synthetic minerals, phase separation and ordering rates and mechanisms in silicates and oxides. Before joining the USGS he was a Research Associate on a NASA-funded project investigating shock deformation of lunar samples in the Department of Geology and Division of Metallurgy and Materials Science at Case Western Reserve University and the U.S. Steel Research Laboratory. For all of these studies the tool of choice was the transmission electron microscope. While working at Case Western and U.S. Steel he began to research the accurate identification of asbestos minerals by electron diffraction. After retiring from the USGS he continued his work on asbestos characterization by electron diffraction as a Senior Scientist with the International Environmental Research Foundation. In 1995 he

founded Nord Consultants, which is concerned with computing in science and education.

**ALAN JOHN ROGERS** is the Director of Alan Rogers Occupational Health & Safety Pty Ltd, in New South Wales, Australia. He holds a Bachelor of Science degree in Pure and Applied Chemistry from the University of New South Wales, a Master of Science degree in Occupational Hygiene from the University of London and a Master of Science degree in Environmental Chemistry from the University of New South Wales. He is a Certified Industrial Hygienist with American Board of Industrial Hygiene and a Fellow of the Australian Institute of Occupational Hygienists. He has extensive postgraduate training in toxicology, statistics, epidemiology and respiratory physiology. Mr. Rogers has conducted research projects in dust diseases, quantitative risk assessments for governments and industries. For nearly 25 years, he was employed by the Australian Government to provided occupational health advice on risk assessment, measurement and control of dusts, chemicals and asbestos. Since 1978 he has published research on the association of asbestos-exposure and asbestos-related disease in Australia. He has been a member of the research panel for the Australian Mesothelioma Surveillance Programme and the Australian Mesothelioma Register responsible for assessing the occupational histories of the 3,758 Australian mesothelioma cases in terms of the degree and source of asbestos exposure.

**EMANUEL RUBIN, M.D.** is Distinguished Professor of Pathology, Anatomy and Cell Biology at Jefferson Medical College in Philadelphia , PA and Chairman Emeritus of the department. He has also served as Chairman of the Department of Pathology at the Mount Sinai School of Medicine and Drexel University Medical School. He held the position of Adjunct Professor of Biochemistry and Biophysics at the University of Pennsylvania School of Medicine for 10 years. Dr. Rubin is the author of some 300 papers in the medical and scientific literature and has been continuously funded by NIH for over 45 years, during which time he has served as Principal Investigator on more than $100,000,000 in research grants. As editor-in-chief he founded one of the major textbooks in the field of pathology, RUBIN'S PATHOLOGY, the sixth edition of which is in press. Dr. Rubin is a highly cited expert in Environmental Pathology, and has authored numerous book chapters in that field and in Pulmonary Pathology. He is recognized as an authority on the effects of asbestos on the human body.