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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)

AMICI CURIAE BRIEF OF COALITION FOR LITIGATION JUSTICE, INC., BUSINESS COUNCIL OF NEW YORK STATE, LAWSUIT REFORM ALLIANCE OF NEW YORK, NEW YORK INSURANCE ASSOCIATION, INC., NORTHEAST RETAIL LUMBER ASSOCIATION, NATIONAL ASSOCIATION OF MANUFACTURERS, CHAMBER OF COMMERCE OF THE UNITED STATES OF AMERICA, AMERICAN TORT REFORM ASSOCIATION, AMERICAN INSURANCE ASSOCIATION, AND NFIB SMALL BUSINESS LEGAL CENTER SUPPORTING DEFENDANT-RESPONDENT FORD MOTOR COMPANY

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January 24, 2018

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.

DISCLOSURE STATEMENT

Pursuant to 22 NYCRR 500.1(f), the associations represented on this brief have no parents, subsidiaries, or affiliates.

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INTEREST OF *AMICI CURIAE*

Amici are organizations whose members have an interest in ensuring that the legal obligations in toxic tort cases are applied consistently and in conformity with sound science and public policy. *Amici* file this brief to provide background on the science of asbestos and to explain why the causation theories espoused by Plaintiff-Appellant's experts do not conform to the standards set forth in *Parker v. Mobil Oil Corp.*, 7 N.Y.3d 434 (2006), and its progeny.

STATEMENT OF THE CASE

Amici adopt Ford Motor Company's Statement of the Case as relevant to our argument.

INTRODUCTION AND SUMMARY OF ARGUMENT

The *Parker* decision addressed the standard for causation testimony in toxic tort cases: plaintiff experts must present a scientific expression of exposure coupled with scientific studies showing that those exposures are sufficient to cause disease. There is nothing novel or unique about this standard. The Court reaffirmed those requirements in *Cornell v. 360 West 51st St. Realty*, 22 N.Y.3d 762 (2014), and *Sean R. v. BMW of N. Am., LLC*, 26 N.Y.3d 801 (2016).

New York's lower courts in asbestos cases have nevertheless often permitted plaintiff expert testimony that does comply with *Parker*, as if there were an "asbestos exception" to the accepted causation standard for toxic tort cases.

Experts for asbestos plaintiffs have often testified, with court permission, to causation based on various versions of the *each and every exposure* theory. Plaintiff experts simply opine that every workplace exposure to dust from an asbestos-containing product is a cumulative part of the overall dose and therefore causative. The theory does not utilize any kind of dose assessment to show that the exposure was large enough to cause anything.[1] This approach violates *Parker*.

In this case, the trial court and First Department faithfully applied *Parker* to asbestos expert causation testimony. The courts looked behind the self-serving statements of plaintiff's experts and reached the logical conclusion – expert testimony that relies on claimed dust and "no safe dose" speculation in lieu of a valid dose and causation assessment must be stricken and cannot support a verdict.

The opinions below provided a timely course correction to a litigation that, scientifically and logically, has largely gone off the rails. Asbestos litigation in recent years, due to the declining population of pre-1970 "dusty trades" workers with causative levels of exposure, has shifted focus to a population of much more speculative and low-level plaintiff exposures. Many of today's plaintiffs are not insulation workers or asbestos factory workers; they consist of individuals who

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[1] *See* Mark A. Behrens & William L. Anderson, *The "Any Exposure" Theory: An Unsound Basis for Asbestos Causation and Expert Testimony*, 37 Sw. U. L. Rev. 479 (2008); William L. Anderson, et al., *The "Any Exposure" Theory Round II: Court Review of Minimal Exposure Expert Testimony in Asbestos and Toxic Tort Litigation Since 2008*, 22 Kan. J.L. & Pub. Pol'y 1 (2012).

removed a few gaskets, worked with dental tape, took insulation off wires, or were merely present in a building containing asbestos insulation. Because there is no epidemiology documenting disease from such low-exposure activities, the only way these cases survive is through the *every exposure* and similar theories that ignore dose and assume all exposures to be causative.

This brief will provide the Court the background and history of these causation theories, set against the fundamentals of asbestos medicine and science. The proponents of these theories present them exclusively in litigation – none of these litigation experts has ever published and obtained peer review in a reputable, independent journal of the notion that all workplace exposures, regardless of quantity, cumulative or not, are actual causes of disease.

Amici request that the Court affirm the decisions below and use clear language to ensure that asbestos litigation conforms to the widely accepted scientific and tort principles repeatedly set forth by this Court.

ARGUMENT

I. THE LOWER COURT'S MUCH-NEEDED COURSE CORRECTION FOR ASBESTOS LITIGATION IS CONSISTENT WITH *PARKER*

A. Asbestos, Like Benzene and Many Other Toxins, Does Not Cause Disease from "Every" Exposure

Claims involving low levels of asbestos exposure are similar to the benzene claims addressed in *Parker*, the mold exposures addressed in *Cornell*, and the gasoline vapor addressed in *Sean R* – none of which was proven to be legally

causative. The human body experiences exposures from all kinds of potentially hazardous substances in our atmosphere, homes, and workplaces. Most of those exposures are too low or from substances not potent enough to pose health risks. Disease causation occurs when an exposure overwhelms the body's defenses.

Thus, as discussed in *Parker*, the benzene epidemiology studies demonstrate that heavy exposures in factory settings may induce acute myelogenous leukemia (AML), but minor exposures from small amounts of benzene in gasoline do not. Common examples abound, such as aspirin, alcohol, and sunlight. Even known "poisons" like arsenic are poisonous only if the dose is high enough.

In toxicology, this principle is known as the "dose makes the poison." This fundamental requirement is set forth in the Federal Judicial Center's *Reference Manual on Scientific Evidence: Reference Guide on Toxicology*,[2] and even more concretely in David L. Eaton, *Scientific Judgment and Toxic Torts – A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & Pol'y 5 (2003), one of the best medical descriptions of the application of toxicology to litigation.[3] As Professor Eaton explains: "Dose is the single most important factor to consider in evaluating

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[2] *See* Bernard D. Goldstein & Mary Sue Henifen, *Reference Manual on Scientific Evidence: Reference Guide on Toxicology* 633, 636 (Fed. Jud. Ctr. 3d ed. 2011).

[3] Many courts have looked to the Eaton article to apply the dose principle and reject various forms of the *any exposure* theory. *See, e.g.*, *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1242 (11th Cir. 2005); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 770 (Tex. 2007); *Adams v. Cooper Indus., Inc.*, 2012 WL 2339741, at *1 (E.D. Ky. 2012*); Henrickson v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1156 (E.D. Wash. 2009).

whether an alleged exposure caused a specific adverse effect." *Id.* at 11. This dose principle holds true for asbestos just as it does for any other toxin:

> 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).

The human body manages exposures to asbestos fibers much as it does benzene molecules, smoke, sunlight, or other potential toxins. Many inhaled fibers are expelled, destroyed, or rendered impotent by the body's clearance and defense processes. The fibers that remain are often controlled (or the cells they impact are controlled) by defenses that prevent cancer. The body has DNA repair mechanisms that must be overwhelmed before carcinogenesis can result. Further, chrysotile fibers, the type at issue in this case, are the least harmful because the body breaks them down. The half-life of a dose of chrysotile is only a few months, meaning the fibers dissipate relatively quickly.[4]

Plaintiffs' experts acknowledge this fundamental truth. They agree that background exposures to asbestos from the ambient air (which virtually all humans experience) do not cause disease, even though background exposures are also

[4] *See* Clare Gilham, et al., *Pleural Mesothelioma and Lung Cancer Risks in Relation to Occupational History and Asbestos Lung Burden*, 73 Occupational Envtl. Med. 290 (2016).

"cumulative" in the lung and can add up over a lifetime to a greater exposure than many workplace encounters.[5] Dose is the critical factor distinguishing causative exposures from those that are not harmful, including in the workplace.

B. Asbestos Epidemiology Demonstrates that Not All Asbestos Exposures Are a Source of Mesotheliomas

Epidemiology studies of asbestos exposures have documented the following principles, found in common toxicology textbooks and many articles:

- Not all instances of mesothelioma are due to asbestos exposure. Epidemiology studies regularly document between 20-50% of non-asbestos related cases in populations with mesothelioma.[6]
- Many lower-level exposures to asbestos have never been shown to cause disease. The brake worker and mechanic epidemiology discussed in Ford's brief is only one such example.[7]

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5 *See e.g.*, *Betz v. Pneumo-Abex LLC*, 44 A.3d 27, 54 (Pa. 2012).

6 The medical literature documents the existence of spontaneous (non-asbestos) cases, for all cancers and for mesothelioma specifically. *See* Stanley Venitt, *Mechanisms of Spontaneous Human Cancers* 104 Envtl. Health Perspective 633, 633, 635 (1996); Cristian Tomasetti & Bert Vogelstein, *Variation in Cancer Risk Among Tissues Can Be Explained by the Number of Stem Cell Divisions*, 347 Science 78 (2015); Brooke T. Mossman, et al., *Asbestos: Scientific Developments and Implications for Public Policy*, 247 Science 294 (1990) ("approximately 20 to 30% of mesotheliomas occur in the general population in adults not exposed occupationally to asbestos"); *see also Butler v. Union Carbide Corp.*, 712 S.E.2d 537 (Ga. App. 2011), (acknowledging role of spontaneous mesotheliomas).

7 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); *see also* Julian Peto, et al., *Occupational, Domestic and Environmental Mesothelioma Risks in Britain: A Case-Control Study*, 73 UK Health & Safety Exec. 1145 (2009); Christine Rake, et al., *Occupational, Domestic and Environmental Mesothelioma Risks in the British Population: A Case Control Study*, 100 Brit. J. Cancer 1175, 1182 (2009). *See also* David Rees, *Case Control Study of Mesothelioma in South Africa*, 35 Am. J. Indus. Med. 213, 220 (1999) (South African chrysotile miners experienced no instances of mesothelioma despite years of heavy exposure).

- Whether an exposure causes disease is dependent on factors used by industrial hygienists to determine hazardous exposures – i.e., the duration, extent, and frequency of the exposure and the potency of the fiber type.
- Populations most prone to disease are those subject to exposures not seen since before OSHA imposed asbestos standards in 1972 – the dusty trades of another era such as shipyard workers, insulation workers, asbestos factory workers, and crocidolite miners.[8]
- No chrysotile-only cohorts have demonstrated a statistically significant increase in mesothelioma from the limited brake exposures or doses comparable to those likely incurred by Mr. Juni.[9]

These fundamentals of asbestos medicine mean that it is imperative for testifying experts to distinguish between levels of exposure that are causative and those that are not. This includes workplace exposures, which often do not rise to the level of producing disease in similar populations – brake mechanics being the principal example. The approach used by plaintiff experts relies on assumptions about low exposures that are not supported by dose assessments or science.

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[8] *See* Emily Goswami, et al., *Domestic Asbestos Exposure: A Review of Epidemiologic and Exposure Data*, 10 Int'l J. Envtl. Res. & Pub. Health 5629 (2013); Ellen Donovan, et al., *Evaluation of Take Home (Para-Occupational) Exposure to Asbestos and Disease: A Review of the Literature*, 42 Critical Rev. in Toxicology 703 (2012).

[9] *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) (review article summarizing cohorts with chrysotile exposures that did not produce mesotheliomas and identifying a minimum-required exposure level well above those of brake mechanics).

C. The Correct Process for Determining Asbestos Causation Follows the Process Set Forth in *Parker* and Its Progeny

Asbestos causation experts who correctly apply scientific principles should begin by identifying the nature of the exposure, just as the *Parker* Court required the experts to assess the potential of *gasoline*, not just pure benzene, to cause AML. The product at issue here is brakes – not insulator or shipyard exposures to highly friable amphibole insulation or crocidolite exposures in mining operations. Bromides such as "all asbestos types cause mesothelioma" or "even a few days of exposure can cause mesothelioma" are smoke screens that plaintiffs' experts use to cover up the lack of evidence of causation in the circumstances at hand. The experts turn to these types of pronouncements to avoid the exercise required by *Parker* – identifying the product at issue and rendering an opinion on its propensity to cause disease. Here, as both lower courts determined, that product is brakes, not generalized asbestos.

Second, experts in an asbestos case should develop a competent dose assessment for each alleged exposure or product in order to distinguish exposures that have not been shown to cause mesothelioma from those that have. *Parker* requires such an assessment, yet plaintiff experts are refusing to do so. In cases like this one, plaintiffs' counsel assert that a perfect quantification is not possible. There is, however, a large gap between a perfect dose quantification and none at all. There are many resources available to assess a possible range of overall dose

for an asbestos-exposed individual, typically from published and unpublished studies of other workers conducting similar activities.[10]

For example, Mr. Juni's exposures can be roughly estimated from competent studies of similarly limited brake work, based on OSHA's methodology of computing the eight-hour time-weighted average exposure over the frequency of the work. The lifetime dose, articulated in fiber per cubic centimeter (cc)/years, for Mr. Juni – if actually estimated by these experts – would likely place his exposures *well below* the levels of career brake workers and below today's OSHA accepted level of exposure.[11]

The third step in a competent causation analysis requires an expert to compare the estimated dose with credible epidemiology to determine whether those exposures are capable of causing mesothelioma. This Court reviewed that evidence in *Parker* and noted that epidemiology studies for *gasoline* exposure in

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[10] In many of today's low-dose cases, for instance, defense industrial hygienists often assess the frequency and duration of exposure events by relying on comparable studies to develop a lifetime dose assessment. The results are often expressed as below OSHA permissible levels or consistent with background exposures. The refusal of plaintiff experts to attempt a dose assessment reflects a litigation-driven agenda – to avoid documenting how low the exposures actually were.

[11] As an illustration only, brake workers likely experienced exposures in the range of 0.04 fibers(f)/cc 8-hr TWA, based on the most comprehensive review of the studies involving such work. *See* Dennis Paustenbach, *An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust*, 18 Applied Occupational Envtl. Hygiene 786 (2003). Four years of such work would produce a lifetime f/cc year level of only 0.16 f/cc years (4 years x 0.04 f/cc), compared to OSHA's current asbestos exposure standard of 0.1 f//cc over 45 years, or 4.5 f/cc years. Thus, Mr. Juni's four-year exposure, even under a rough estimate such as this, falls well within the OSHA requirements for today's workplace.

low-exposure settings documented no increased risk of AML. The cohorts for much higher exposed benzene factory workers were not sufficiently relevant to support the opinions.

The *correct* approach for Mr. Juni would point *not* to the irrelevant asbestos factory work, insulation, shipyard, and mining studies often cited by plaintiff experts, but to the twenty-one out of twenty-two epidemiology studies demonstrating that even full-time mechanic work is not associated with mesothelioma. Those and other, similar studies were conducted and published by several government research bodies and over 100 individual publishing authors, all across populations in ten countries and under a wide variety of sponsorship, methodology, and mechanic populations.[12] That set of studies provides a robust and credible set of data with no increased incidence of disease.[13] There is no

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[12] Most of the studies are discussed and summarized in the three published meta-analyses of brake mechanics and mesothelioma – none of which also found any link between mechanic work and mesothelioma. *See* David Garabrant, et al., *Response to Kay Teschke. Re: Mesothelioma Among Motor Vehicle Mechanics: An Updated Review and Meta-analysis,* 60 Annals of Occupational Hygiene 1036 (2016); Michael Goodman, et al., *Mesothelioma and Lung Cancer Among Motor Vehicle Mechanics: A Meta-analysis,* 48 Annals of Occupational Hygiene 309 (2004); Otto Wong, *Malignant Mesothelioma and Asbestos Exposure Among Auto Mechanics: Appraisal of Scientific Method,* 34 Regulatory Toxicology Pharmacology 170 (2001); *see also* Francine Laden, et al., *Lung Cancer and Mesothelioma Among Male Automobile Mechanics: A Review,* 19 Rev. on Envtl. Health 39 (2004) (review article finding no basis for linking mechanic work to mesothelioma).

[13] Many of these 100 authors have submitted their findings to publication and peer review, whereas the 38 signers of the Concerned Scientists' Brief have never published their contrary views in peer-reviewed journals. Many of them are testifying plaintiff experts whose opinions on *every exposure* have been excluded by other courts (e.g., Welch, Frank, Castleman, Egilman, Maddox). One of the signers is the *very expert excluded* by this Court in *Parker* (Landrigan). (Continued...)

credible, alternative set of epidemiology studies repeatedly documenting an increased incidence of disease from brake work.

Plaintiffs contend that the *Parker* holding would deprive deserving asbestos plaintiffs of their day in court. That would be true only if the limited brake-related exposures of someone like Mr. Juni actually caused the disease. The result here should be much the same as the plaintiff in *Parker*, who was not able to meet his burden of producing scientific evidence to support his claim.

The scientific methodology should govern courtroom testimony to avoid rank speculation offered by asbestos plaintiff experts. Applying *Parker* and its progeny to asbestos litigation in New York will curb the prevalence of unsupported cases.

II. *EVERY EXPOSURE* EXPERTS IGNORE SCIENTIFIC FUNDAMENTALS IN FAVOR OF THE UNSUPPORTED ASSUMPTION THAT ALL EXPOSURES ARE CAUSATIVE

If experts such as plaintiff's experts Drs. Moline and Markowitz applied the *Parker* principles they would be forced to abandon their causation opinions in low-exposure asbestos cases. Instead, they resort to the *every* or *cumulative* exposure

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Plaintiff's brief focuses heavily on only one study – the Roelofs article. Excluding 21 competent studies in favor of a single study is an exercise in improper *cherry-picking*. *See e.g.*, *In re Bextra & Celebrex*, 2008 N.Y. Misc. LEXIS 720, at *20 (2008) (New York's *Frye* rule requires expert to "look at the totality of the evidence and not ignore contrary data."); *see also id*. at *36 (selective review of studies by expert "smacks of 'cherry-picking'" and "contradicts the accepted method for an expert's analysis of epidemiological data."); *In re Zoloft (Sertraline Hydrochloride) Prods. Liab. Litig.*, 858 F.3d 787, 798 (3d Cir. 2017) (selectively used meta-analysis made expert's testimony unreliable).

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. The *Cumulative* Exposure Testimony Does Not Comport with Basic Scientific Principles or Logic

For Drs. Moline and Markowitz, the duration, extent and frequency of exposure – all critical elements of industrial hygiene and medical causation – are not part of their analysis. These experts merely *recite* the frequency and extent of exposures, as if recitation is all that is required, and claim that such exposures are enough. They never provide any scientific principle why that particular exposure would be sufficient and less frequent or intense workplace exposures would not. This is the fundamental flaw of the *every exposure* theory – it allows the experts to capture even the smallest amounts of workplace exposures, i.e., those that are too small own their own (or cumulatively) to cause disease. They jettison dose and epidemiology, and instead base causation on the regulatory "linear no-threshold" approach used as a conservative assumption in rulemakings by some regulators. That approach *assumes* there is no safe dose of asbestos rather than requiring evidence proving that assumption.[14] The "linear no-threshold" model is a

[14] Regulatory bodies often state that there is no known safe dose to justify setting protective limits far below the levels of proven disease occurrence as documented in epidemiology studies. Courts have recognized the fallacy of relying on these assumptions to prove causation in a courtroom setting. *See, e.g., Betz v. Pneumo-Abex,* 44A.3d 27, 49 n.25 (Pa. 2012) (citing cases (Continued...)

precautionary approach. It ignores DNA repair and is wrong for a fact-based analysis of causation in tort law.

The trial court was correct to reject this testimony under *Parker*, and the First Department was correct to affirm that ruling, consistent with the majority of courts. The Court in *Parker* did *not* excuse experts from developing any dose assessment at all, or from using a scientifically acceptable means of distinguishing causative doses from non-causative exposures. As the Court reiterated in *Sean R.:*

> 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 reliable, scientifically accepted method) for assessing Mr. Juni's dose from his brake and clutch work. The experts did not model anything; they did not quantify anything; they did not 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 a series of epidemiologic studies showing that exposures like

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rejecting regulatory linear no-threshold approach to support causation); *Allen v. Pennsylvania Eng'g Corp.,* 102 F.3d 194, 198 (5th Cir. 1996); *Henricksen v. ConocoPhillips Co*., 605 F. Supp. 2d 1142, 1166 (E.D. Wash. 2009); *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).

Mr. Juni's would cause disease. The contrast with the requirements of *Parker*, *Cornell*, and *Sean R.* could not be more dramatic.

A comparison between the testimony here and that rejected in *Parker* shows that the expert testimony in the two cases is indistinguishable:

- The *Parker* experts conflated exposure to benzene with exposure to gasoline to avoid the epidemiology studies documenting that gasoline exposures do not cause AML. Here, the experts conflated the idea that "asbestos has been proven to cause mesothelioma" with the far different proposition that working with brakes, clutches, and gaskets causes mesothelioma – also disproven by the epidemiology.
- The *Parker* experts relied on the anecdotal exposures to gasoline of the plaintiff gas station attendant and assumed, without actually estimating any dose, that there was sufficient exposure from those experiences. Drs. Markowitz and Moline likewise relied here on nothing more than Mr. Juni's reported dust from brake-related work that he mostly did not even participate in himself.
- The *Parker* experts ignored several epidemiology studies finding no link between service station work and AML. Here, Drs. Markowitz and Moline ignore a far larger set of epidemiology studies showing no link between mechanic work and mesothelioma.
- The *Parker* experts relied on conclusory statements ("excessive," "extensive") in lieu of a dose assessment. Here, Drs. Moline and Markowitz did not even take this minimal step, instead relying on the notion that even small exposures are causative and offering neither quantitative nor qualitative assessments of Mr. Juni's dose.
- The *Parker* experts relied on the notion that there is no safe level of exposure to benzene and "the theory that there is no threshold of exposure under which there will be no negative effects to health." This is the *every exposure* theory, rejected by the *Parker* court. Markowitz and Moline used the same or very similar flawed approach as to asbestos.

The Court in *Cornell* rejected the expert's testimony in a mold case because the expert "made no effort to quantify [plaintiff's] level of exposure" to mold. 22 N.Y.3d at 784. The Court in *Sean R.* rejected the experts' "backwards" calculation of dose from "reported symptoms to divine an otherwise unknown concentration of gasoline vapor." 26 N.Y.3d at 802. Much like the *Sean R.* experts, Drs. Markowitz and Moline essentially engage in circular logic by concluding that since Mr. Juni alleges exposure to asbestos and has mesothelioma, he must have been exposed to enough asbestos to cause his mesothelioma.[15] The trial court and First Department opinions thus applied well-established New York law to asbestos litigation and appropriately rejected these experts' opinions.

B. Experts in Asbestos Cases Should Not Be Relieved of Causation Proof Because "Dust" Was Present

Both the Plaintiff's Brief and the allied *amicus* brief filed by "Concerned Scientists" include multiple references to brake exposure studies that purportedly show high levels of fiber/cc exposures. There is a fundamental problem with Plaintiff's reliance on this information – his experts neither relied on this data nor

[15] *See Butler v. Union Carbide*, 712 S.E.2d 537, 550-551 (Ga. App. 2011) (it is circular reasoning for an expert to presume that a plaintiff "must have somehow been exposed to a high enough dose to exceed the threshold (necessary to cause the illness), thereby justifying his initial diagnosis.") (citations omitted); *Young v. Burton*, 567 F. Supp. 2d 121, 137 (D.D.C. 2008), *aff'd*, 354 F. App'x 432 (D.C. Cir. 2009) (improper for expert to engage in circular reasoning to work backwards from diagnosis to proof of exposure).

needed it for their *every exposure* opinions. The level of exposure is not relevant to the causation inquiry for experts who testify that every exposure is causative.

The real foundation for the plaintiff experts' view that the exposures were meaningful is not the exposure studies, but the mere testimony that dust was generated by brake-related work. In lieu of a scientific assessment of the dose, the experts have substituted the highly imprecise measure of "visible dust." Because dust is ubiquitous in workplaces, and an allegation of dust is so easy to procure from plaintiff and co-worker testimony, the experts' reliance on testimony about dust has become a key to the *every* or *cumulative* exposure testimony in modern asbestos litigation. That reliance cannot survive even minimal scrutiny under *Parker, Cornell,* and *Sean R.*

Testimony that visible dust was present at a workplace can never substitute for an actual dose assessment. Workplaces experience all kinds of dusts, including the ordinary kind that invades homes, businesses, and vehicles. Lay witnesses cannot distinguish between ordinary dust and asbestos-containing dust. Even dust from an asbestos-related activity can contain a wide variation in quantity or type of asbestos, which means one type of dust could be harmless and another potentially dangerous. A great deal of dust from asbestos-related work activity is often not even respirable and/or would not make it into the worker's breathing zone.

Causation based on nothing more than visible dust being present would constitute a form of absolute liability for any company utilizing an asbestos product.

Claims of dust exposure are similar to the rejected notion in *Parker* and *Sean R.* that breathing of fumes or detecting an odor suffices for causation. Likewise, the presence of mold in *Cornell* was not enough without a measured exposure. Dr. Moline acknowledged that the amount, duration, and frequency of exposure are critical factors, but she then relied on just dust in the environment and proceeded to ignore all of those factors in rendering her case-specific opinion.

The opinions below in this case are not alone in rejecting the notion that dust testimony is an acceptable substitute for a competent industrial hygiene exposure assessment. A Pennsylvania trial court found plaintiff's testimony that he saw dust insufficient with no proof that the dust contained asbestos, there were multiple potential other sources of dust in industrial facility, and there was no testimony as to plaintiff's distance from dust or degree of exposure. *See Sterling v. P&H Mining Equip.,* 113 A.3d 1277 (Pa. Super. 2015). A North Carolina federal court rejected an asbestos *cumulative exposure* expert's reliance on visible dust as a substitute for an asbestos exposure assessment. *See Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841 (E.D.N.C. 2015), *reconsideration denied*, 143 F. Supp. 3d 386 (E.D.N.C. 2015). The Texas Supreme Court also rejected reliance on testimony of clouds of visible dust as a substitute for the "approximate quantum" of actual

asbestos exposure. *See Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 774 (Tex. 2007).

The Court should confirm that it is no more acceptable to refer to dust as an assessment of exposure to asbestos than it is to resort to smells, fumes, visible mold, and similar non-scientific forms of exposure assessment.

III. COURTS HAVE REJECTED ATTEMPTS BY PLAINTIFF EXPERTS TO "RE-BRAND" *EVERY EXPOSURE* TESTIMONY AS *CUMULATIVE EXPOSURE*

The history of how the *every exposure* theory came to be rejected all over the country illustrates a point critical for this case. Plaintiff experts have changed the descriptor for this type of testimony several times to avoid exclusion rulings – today, many plaintiff experts like Dr. Moline call it *cumulative exposure* testimony. But the fundamentals never change. As a result, courts have seen through this semantic tactic, rejecting testimony that does not properly assess the dose and prove causation.

More than a decade ago, when asbestos cases began to include more tangential and minimal exposures, the *every exposure* approach took on critical importance. In response, defendants began to file motions attacking the *every exposure* theory – or the *single fiber* theory as these same experts often called it at that time ("a single fiber of asbestos can cause mesothelioma"). The Sixth Circuit Court of Appeals rejected such testimony in a matter involving the minimal

exposure scenario of removing chrysotile-containing gaskets.[16] The same year, a Pennsylvania trial judge – in what later became *Betz v. Pneumo Abex LLC*, 44 A.3d 27 (Pa. 2012) – issued a thorough opinion identifying the logical and scientific fallacies in "each and every exposure" testimony in a case involving brake mechanics with years of exposures.[17] Over the next few years, courts including the Supreme Courts of Pennsylvania and Texas excluded or criticized a number of plaintiff experts who failed to assess the dose.[18]

After these early rejections, plaintiffs' experts learned not to testify based on the patently extreme *single fiber* theory. Instead, they began to testify that "each and every exposure above background is a substantial factor in causing mesothelioma." The shift in language –intended to dodge the previous court rulings – did not change the actual causation opinion. The two approaches are identically grounded in a failure to assess the dose; every workplace exposure no matter how medically inconsequential still counted under their theory. As a result, courts continued to exclude this testimony under the *each and every* exposure

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[16] *See Bartel v. John Crane, Inc*., 316 F. Supp. 2d 603 (N.D. Ohio 2004), *aff'd sub nom. Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488 (6th Cir. 2005).

[17] *See In re Toxic Substances Cases*, 2006 WL 2404008 (Pa. Com. Pl. Allegheny Cnty. Aug. 17, 2006), *aff'd sub nom. Betz v. Pneumo Abex, LLC*, 44 A.3d 27 (Pa. 2012).

[18] *See Gregg v. V-J Auto Parts Co.*, 943 A.2d 216 (Pa. 2007); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765 (Tex. 2007); *Georgia-Pacific Corp. v. Stephens*, 239 S.W.3d 304 (Tex. App.-Hous. 2007); Transcript of Record at 144-45, *Anderson v. Asbestos Corp*., No. 05-2-04551-5SEA (Wash. King Cnty. Super. Ct. Oct. 31, 2006); *In re W.R. Grace & Co*., 355 B.R. 462 (Bankr. D. Del. 2006).

verbiage. Between 2008 and 2010, the Sixth Circuit Court of Appeals rejected *every exposure* testimony, joined by another Pennsylvania trial court, a Washington state court, and a Texas appellate court.[19]

By the end of 2013, over twenty courts had issued opinions criticizing and rejecting *every exposure* and other dose-less causation theories as the basis for expert testimony or as insufficient evidence in asbestos litigation. The courts adding their opinions included the Supreme Court Virginia; a Georgia appellate court, a District of Columbia federal court, more Texas appellate courts, the Sixth Circuit again, and federal judges in Utah.[20]

The string of opinions did not end there. In the last three years, at least *sixteen more courts* have rejected *any exposure* and similar forms of testimony, including the Georgia Supreme Court, the Seventh and Ninth Circuit Courts of Appeal, the Texas Supreme Court (for the second time), an Ohio appellate court,

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[19] *See Martin v. Cincinnati Gas & Elec. Co*., 561 F.3d 439 (6th Cir. 2009); *Free v. Ametek,* 2008 WL 728387 (Wash. Super. Ct. King Cnty. Feb. 28, 2008); *In re Asbestos Litig. (Certain Asbestos Friction Cases Involving Chrysler LLC),* 2008 WL 4600385 (Pa. Com. Pl. Phila. Cnty. Sept. 24, 2008); *Smith v. Kelly-Moore Paint Co., Inc.,* 307 S.W.3d 829 (Tex. Ct. App. 2010).

[20] *See Betz v. Pneumo Abex LLC,* 44 A.3d 27 (Pa. 2012); *Butler v. Union Carbide Corp.*, 712 S.E.2d 537 (Ga. App. 2011); *Ford Motor Co. v. Boomer*, 736 S.E.2d 724 (Va. 2013); *Wannall v. Honeywell Int'l, Inc*., 292 F.R.D. 26 (D.D.C. 2013), *aff'd*, 775 F.3d 425 (D.C. Cir. 2014); *Moeller v. Garlock Sealing Tech., LLC*, 660 F.3d 950 (6th Cir. 2011); *Smith v. Ford Motor Co.*, 2013 WL 214378 (D. Utah Jan. 18, 2013); *Anderson v. Ford Motor Co*., 2013 WL 3179497 (D. Utah June 24, 2013).

seven federal court decisions from five different states, and the trial court and First Department in this matter.[21]

Several of these opinions rejected yet another shift in plaintiffs' experts' terminology – the shift to "cumulative" exposure that the experts here employed. This new phrasing is in all relevant aspects the same as *every exposure* testimony – all of the plaintiff's workplace exposures constitute, cumulatively, the cause of his disease, no matter how small or inconsequential any of those exposures from one site or product might have been. The trial court's analysis and rejection of "cumulative" exposure testimony is one of the most thorough and carefully reasoned such opinions to date. Federal district courts in North and South Carolina have also rejected *cumulative* exposure testimony on the same grounds as *every exposure* testimony. *See Yates,* 113 F. Supp. 3d at 856-57; *Haskins,* 2017 WL 3118017, at *6-*8 (D.S.C. July 21, 2017) (cumulative exposure testimony violates the substantial factor causation standard). The most recent court to reject

[21] *See Krik v. Exxon Mobil Corp.,* 870 F.3d 669 (7th Cir. 2017); *McIndoe v. Huntington Ingalls Inc.,* 817 F.3d 1170 (9th Cir. 2016); *Estate of Barabin v. AstenJohnson, Inc.,* 740 F.3d 457 (9th Cir.), *cert. denied*, 135 S. Ct. 55 (2014); *Stallings v. Georgia-Pacific Corp.,* 675 F. App'x 548 (6th Cir. 2017); *Scapa Dryer Fabrics, Inc. v. Knight*, 788 S.E.2d 421 (Ga. 2016); *Georgia-Pacific Corp. v. Bostic*, 439 S.W.3d 332 (Tex. 2014); *Watkins v. Affinia Group,* 54 N.E.3d 174 (Ohio Ct. App. 2016); *Haskins v. 3M Co.*, 2017 WL 3118017 (D.S.C. July 21, 2017); *Comardelle v. Pennsylvania Gen. Ins. Co.,* 76 F. Supp. 3d 628 (E.D. La. 2015); *Sclafani v. Air & Liquid Sys. Corp.*, 2013 WL 2477077 (C.D. Cal. May 9, 2013); *Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841 (E.D.N.C. 2015), *reconsideration denied*, 143 F. Supp. 3d 386 (E.D.N.C. 2015); *Vedros v. Northrup Grumman Shipbuilding, Inc.*, 119 F. Supp. 3d 556 (E.D. La. 2015); *Davidson v. Georgia Pacific LLC*, 2014 WL 3510268 (W.D. La. July 14, 2014), *vacated on other grounds*, 819 F.3d 758 (5th Cir. 2016); *Suoja v. Owens-Illinois, Inc.,* 211 F. Supp. 3d 1196 (W.D. Wis. 2016).

cumulative exposure testimony, the Seventh Circuit Court of Appeals, held simply: "[The district judge] readily and correctly concluded that the cumulative exposure theory was no different from the 'each and every exposure' theory…." *Krik v. Exxon Mobil Corp.*, 870 F.3d 669, 676 (7th Cir. 2017).

Today, based on the rulings above, *every* or *cumulative exposure* testimony utilizing the same foundations, methodology, and conclusions as Drs. Moline and Markowitz is insufficient or inadmissible in the Seventh Circuit, the Sixth Circuit (under Ohio and Kentucky law); the Ninth Circuit; the District of Columbia federal courts; the Supreme Courts of Virginia, Texas, New York (under *Parker*), and Georgia;[22] and in many state and federal courts in Washington, Illinois, Utah, Nevada, California, Mississippi, Louisiana, Ohio, Wisconsin, North Carolina, South Carolina, among others.[23] Neither *cumulative* nor *every exposure* testimony

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[22] Pennsylvania and California are in flux. Pennsylvania's Supreme Court has rejected *every exposure* exposure testimony, broadly and in clear terms, at least three times. *See Betz v. Pneumo Abex LLC*, 44 A.3d 27 (Pa. 2012); *Gregg v. V-J Auto Parts Co.*, 943 A.2d 216 (Pa. 2007) (the theory is a "fiction"); *Howard ex rel. Estate of Ravert v. A.W. Chesterton, Inc.*, 78 A.3d 605 (Pa. 2013). After a dramatic shift in the makeup of the court in the last election, the court approved a version of *every exposure* testimony in *Rost v. Ford Motor Co.*, 151 A.3d 1032 (Pa. 2016), in apparent disregard of that state's *stare decisis* rule, and in an attempt to narrow the *Betz* holding to a point of uselessness. California courts have issued mixed opinions at both the state and federal level, even though the Ninth Circuit has rejected the theory and the California Supreme Court has not issued a determination on it.

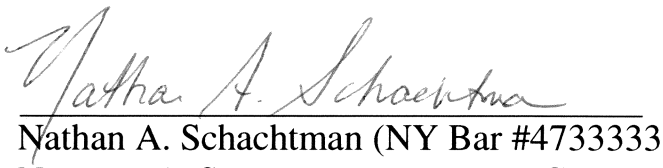
[23] In the last few years, some courts have chosen to allow any exposure testimony. Some of those opinions involve causation law not applicable in New York or this case. *See, e.g., Payne v. CSX Transp., Inc.*, 467 S.W.3d 413 (Tenn. 2015) (ruling under FELA's more generous causation standard). Others have failed to conduct any inquiry into the support for the expert's testimony, electing instead simply to cite to the expert's own self-serving statements. *See, e.g., Neureuther v. Atlas Copco Compressors, L.L.C.*, 2015 WL 4978448 (S.D. Ill. Aug. 20, 2015) (citing only to (Continued...)

comports with the extensive rulings in these courts, or with this Court's own determinations in *Parker, Cornell,* and *Sean R.* The trial court and First Department were correct to apply *Parker* and strike the testimony of these experts.

CONCLUSION

Parker should be applied to asbestos litigation to ensure that expert testimony in such cases conforms with standard causation and scientific principles. For these reasons, *amici* urge the Court to affirm the rulings below.

Respectfully submitted,



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expert's own claims). These are not persuasive opinions, and more critically, none of these courts were under the instruction of the *Parker* case.

CERTIFICATION PURSUANT TO NYCRR § 500.13(c)(1)

Pursuant to NYCRR § 500.13(c)(1), I certify that the brief was prepared with Microsoft Word 2010 using Times New Roman proportionally spaced typeface in 14-point font (12-point font for the footnotes). The total number of words in the portions of the brief that must be included in the word count is 6,313 words according to the word processing system used to prepare the brief.

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Dated: January 24, 2018