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New York Supreme Court

Appellate Division—First Department

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-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

(For Continuation of Caption See Inside Cover)

BRIEF FOR *AMICI CURIAE* THE COALITION FOR LITIGATION JUSTICE, INC., THE CHAMBER OF COMMERCE OF THE UNITED STATES OF AMERICA, THE BUSINESS COUNCIL OF NEW YORK STATE AND THE NATIONAL ASSOCIATION OF MANUFACTURERS

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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, and 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
and YUBA HEAT TRANSFER, LLC.,

Defendants.

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STATEMENT OF INTEREST

Amici are organizations whose members have an interest in ensuring that the rules and legal obligations applied in asbestos and other toxic tort litigation are consistently applied in conformity with sound science and public policy.[1] *Amici* regularly file briefs before state and federal appellate courts to address legal and scientific issues in asbestos and toxic tort litigation. *Amici* file this brief to provide the Court with background on the science of asbestos and why the causation theories espoused by the Plaintiff-Appellant experts in this case do not comport with good science, as the *Juni* trial court held.

INTRODUCTION

The causation theory employed by the experts in this case[2] – a variation on the *any exposure* theory often asserted by plaintiffs' experts in asbestos litigation – disdains any need even to estimate the actual dose of a plaintiff in a toxic tort case. The theory is driven by reliance on the assumption of "no safe dose" for a carcinogen and on a plaintiff's allegation of mere exposure – *any* exposure, in fact – as sufficient to prove the case. Plaintiff-Appellant's brief is filled with

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[1] The Coalition for Litigation Justice consists of its members Century Indemnity Company; San Francisco Reinsurance Company; Fireman's Fund Insurance Company; Great American Insurance Company; Nationwide Indemnity Company; Resolute Management, Inc. a third-party administrator for numerous insurers; and TIG Insurance Company.

[2] *In re New York City Asbestos Litig., Juni v. A.O. Smith Water Prod.*, 48 Misc.3d 460, 11 N.Y.S.3d 415 (N.Y. Sup. Ct. 2015).

references to how many times Mr. Juni was near or involved in brake, clutch, and gasket work and whether his exposures exceeded a governmental standard. But neither of Plaintiff-Appellant's experts relied on any of this. Both experts believed that Mr. Juni's actual dose, including any attempt to estimate or quantify that dose, was irrelevant and thus a scientific expression of the quantity of Mr. Juni's dose formed no part of their opinions.

The correct scientific methodology for connecting latent diseases to distant exposures, as is the case with asbestos and mesothelioma, must account for dose, not ignore it. The beginning point is the existing set of epidemiology studies, from which the expert must determine (not assume) whether a particular individual had sufficient exposure to the right type of substance to fall into the class of workers that show actual disease. Here, that set of epidemiology is very clear – 21 out of 22 studies have found no link between mechanic work and mesothelioma. Drs. Markowitz and Moline have steadfastly refused to acknowledge this literature or to assess Mr. Juni's dose, largely because if they did so, the dose would be well within the class of chrysotile-exposed workers who have no identified link with mesothelioma. They would thus have no support for a causation opinion.

Causation theories that fail to assess the dose have been thoroughly discredited and rejected by dozens of courts as an unscientific foundation for expert causation testimony in toxic tort litigation. The New York Court of Appeals

has joined that chorus, not once but three times – in the *Parker, Cornell,* and *Sean R.* opinions that control the outcome here.[3] Asbestos litigation over the years has developed asserted causation approaches out of step with science, and the *cumulative* or *any exposure* theory used by these two experts and many others is sitting at the top of that list. Today, the litigation is increasingly dominated by cases claiming minimal and often second-hand exposure to this ubiquitous and naturally-occurring material, many of which are supported by no credible scientific evidence that such low exposures would cause disease. The decisions rejecting *any exposure* and similar theories are returning asbestos litigation to a more scientific and rational basis, and in fact to the same standards that apply in other toxic tort contexts.

Amici urge this Court to affirm the trial court's decision. State appellate courts need to assist the trial courts in drawing logical and scientific lines around non-causative workplace exposures by refusing to allow experts to declare or speculate that all exposures are a substantial causative factor. The carcinogenic properties of asbestos are similar to those of most other carcinogens. But the experts here are attempting to do in asbestos cases what they clearly could not do under New York law in a benzene, mold, or any other type of toxic tort case.

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[3] *Parker v. Mobil Oil Corp.,* 7 N.Y.3d 434 (2006); *Cornell v. 360 W. 51st St. Realty, LLC*, 22 N.Y.3d 762, 784 (2014); *Sean R. v. BMW of North America, LLC,* 2016 WL 527107 (N.Y. Ct. App., Feb. 11, 2016).

Because the experts failed to satisfy the criteria of *Parker* and its progeny, the trial court correctly rejected that testimony and the sufficiency of the Plaintiff-Appellant's evidence.

ARGUMENT

I. Dose Is a Critical Component for Toxicology and Causation Assessments.

A. The *Any* or *Cumulative Exposure* Theory Does Not Conform to the Dose Principle.

Under the clear standards for assessing toxic tort causation set forth in three recent Court of Appeals' decisions, the *cumulative exposure* theory relied on by Plaintiff-Appellant's experts is not a sufficient basis for a jury verdict. The *any exposure* and *cumulative exposure* experts believe that all workplace or hobby (but not background) exposures to asbestos, regardless of dose, must be considered part of the cause of the disease. This is a form of the *any exposure* theory, even though Dr. Moline tried very hard to avoid using that phrase. Her alternative, the *cumulative* exposure theory, is based on the same underlying principle – since she contends that there is "no safe dose" of asbestos, then every exposure is assumed to be causative because all exposures are cumulative.[4] The trial court saw through the

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[4] Dr. Moline's reliance on the *cumulative exposure* theory is just the latest label that asbestos plaintiffs' experts are applying to the widely discredited *any exposure* or *each and every exposure* theory. This switch in label is a transparent attempt to dodge the many rulings (Continued...)

sophistry: "Many of those courts [addressing sufficiency of the expert evidence] require specific proof of exposure and have rejected the so-called cumulative exposure theory and its variant, the "each and every" exposure theory." *Juni*, 11 N.Y.S.3d at 437 (citations omitted).

1. Dose Assessment Is Critical for all Toxins Including Carcinogens.

The most critical flaw in this approach – whatever name these experts use – is that the experts are blatantly ignoring the most important principle of toxicology: "the dose makes the poison." Or put another way, no substance is poisonous unless the dose is sufficient.[5] This fundamental requirement is set forth in the Federal Judicial Center's *Reference Manual on Scientific Evidence, Reference Guide on Toxicology* 403 (2d ed. 2000), and even more concretely in one of the best medical descriptions of the application of toxicology to litigation, by Dr. David Eaton of the University of Washington. As Professor Eaton's article explains: "Dose is the single most important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect."[6]

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excluding *any exposure* testimony. But whatever label is used, the theories rest on the same flawed foundation, as the *Juni* court determined.

[5] Federal Judicial Center, *Reference Manual on Scientific Evidence Third Edition* at 403 (National Academies Press, 2011) (the "fundamental tenet" of toxicology).

[6] David L. Eaton, *Scientific Judgment and Toxic Torts – A Primer In Toxicology For Judges and Lawyers*, 12 J.L. & Pol'y 5, 11 (2003). Many courts have looked to the Eaton article in recent years to apply the dose principle and reject various forms of the *any exposure* (Continued...)

Asbestos, like any toxin, requires some level of overall dose to produce disease. The human body is capable of defending itself against a whole array of small, daily exposures to known toxins. Disease results when those exposures reach a level that overwhelms our defenses, called the "threshold" point. Aspirin, alcohol, sunlight, even known poisons like arsenic are only poisonous if the dose is high enough to make them so. At lower doses, they are either harmless or beneficial.

As Professor Eaton notes, this dose principle holds true for carcinogens like asbestos just as much 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). Airplane passengers receive doses of radiation above background at high elevations, but scientists do not ascribe cancer to those flights.[7] Foods often contain low levels of natural carcinogens not known to cause any

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theory. *See, e.g., McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1242 (11th Cir. 2005); *Borg-Warner Corp.*, 232 S.W.3d 765, 770 (Tex. 2007); *Adams v. Cooper Indus., Inc.*, 2012 WL 2339741, *1 (E.D. Ky. 2012); *Henrickson v. ConocoPhillips Co.*, 605 F.Supp.2d 1142, 1156 (E.D. Wash. 2009).

[7] *See* Health Physics Soc'y, *Radiation Exposure During Commercial Airline Flights* (2014), *at* http://www.hps.org/publicinformation/ate/faqs/commercialflights.html; Health Physics Soc'y, *Airport Screening Fact Sheet* (2011), *at* http://hps.org/documents/airport_screening_fact_sheet.pdf (compiling studies).

harm. Science has cleared these "exposures" through the use of epidemiology studies that have found no link between such low-level exposures and cancer, even when the substance is without question a carcinogen at high doses.[8] Car owners experience small amounts of benzene vapor when they fill their cars. To claim that every such exposure is "cumulative" of an overall lifetime exposure and therefore a cause of disease is nonsensical and completely contrary to *Parker*. A bucket of water does not contribute to the ocean, not in any meaningful sense. Nor, as *Parker* illustrates, can an expert claim that minor exposures to a substance (*e.g.*, benzene) are a cause of disease merely because much higher levels produce that disease. In all of these settings, the human body defends against lower level exposures.

2. Asbestos Is Governed by the Same Rules of Toxicology as Other Carcinogens.

Asbestos is no different. The principles that apply to other carcinogens apply to asbestos as well. Mesothelioma and other asbestos diseases, for instance,

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[8] Epidemiology is universally recognized as the "most desirable evidence" for assessing causation in the science of toxicology. Michael Green, *Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of the Agent Orange and Bendectin Litigation*, 86 NW. U. L. REV. 643, 646 (1992); *see also id.* at 648. *See* Bert Black, *Epidemiologic Proof in Toxic Tort Litigation*, 52 FORDHAM L. REV. 732, 736 (1984) ("[E]pidemiology is the only generally accepted scientific discipline . . . to identify and establish the causes of human diseases."); Mary Andrues, *Proof of Cancer Causation in Toxic Waste Litigation: The Case of Determinacy Versus Indeterminacy*, 61 S. CAL. L. REV. 2075, 2088 (1988) ("The only valid way to identify human carcinogens and establish medical causation is to observe differences in the incidence of cancer between humans exposed to toxic wastes and those who are not.").

are dose-dependent – the higher the dose, the greater the disease in the exposed population. The same is true of benzene, radiation, and other carcinogens.[9] Asbestos, like other carcinogens, likely causes cancer either by an inflammatory process or by causing mutational changes in the cell's chromosomes.[10] But the human body has many mechanisms for defending against such process, both for asbestos and for other carcinogens.[11] Thus, humans can be exposed to naturally-occurring asbestos, like other carcinogenic agents, or lower levels of occupational exposures without incurring disease. Many carcinogens have long latency periods, as does asbestos – that is not a distinguishing factor.[12] And other carcinogens produce "signature" cancers much like asbestos does with mesothelioma – benzene, for instance, is most strongly related to AML, radon exposures produce lung cancers, and a mold known as "aflotoxin" produces liver cancer.[13]

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[9] *See* Eaton, *supra* n.6 at 13 ("Most chemicals that have been identified to have 'cancer-causing' potential (carcinogens) do so only following long-term, repeated exposure for many years."). The *Parker* opinion describes the role of dose in benzene's association with AML.

[10] *See* Eaton, *supra* n.6 at 30 (discussing mechanisms of carcinogenesis and explaining that even for mutagenic agents "the dose response for mutagenesis is critically important to consider."). *See generally,* Tim D. Oury, Thomas A. Spron, Victor L. Roggli, *Pathology of Asbestos-Associated Diseases,* at Chapter 10 (3d. ed., Springer (2014)) (describing asbestos carcinogenic mechanisms).

[11] *See* Eaton, *supra* n.6. at 32 (describing some of the body's protective mechanisms).

[12] *See id.* (latency for most cancers is 20 to 40 years, similar to asbestos).

[13] http://www.cancer.gov/about-cancer/causes-prevention/risk/substances/radon/radon-fact-sheet; *see* Eaton, *supra* n.6 at 25. Claims of "no threshold" or "no known safe dose" are also commonly asserted for other toxic substances besides asbestos and are frequently rejected by other courts. A simple search for no safe dose on

(Continued...)

In addition, millions of people – in fact, virtually everyone alive today – have experienced low levels of "background" or ambient exposure to asbestos because the fibers are ubiquitous in the environment and found as a naturally occurring substance in many areas of the country. And like asbestos, other carcinogens frequently accumulate in the body and thus build up over time, but still may not cause cancer if the levels are not high enough.[14] Much like background, naturally occurring levels of radon, dioxins, radiation, and other carcinogenic materials, these "background" levels of asbestos have never been shown to cause mesothelioma, even though over a lifetime they easily exceed by many times the "millions" of fibers that Plaintiff-Appellant tries to ascribes to a single brake exposure.[15]

Google will retrieve dozens of websites claiming there is "no safe dose" for a host of materials, including radiation, alkylating agents, alcohol, and aspartame. *See also Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1165-66 (E.D. Wash. 2009) ("Other courts have similarly rejected expert opinions that are based on the "no-threshold" model. As one court explained in excluding the plaintiffs' experts using the same no threshold theory, "[t]he linear non-threshold model cannot be falsified, nor can it be validated."); *Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671, 679 (6th Cir. 2011) (expert claimed that Mrs. Pluck "probably had an injurious exposure to benzene and other organic solvents considerably above background"; and that '[t]here is no safe level for benzene in terms of causing cancer.' We find this analysis unpersuasive, particularly because the levels of benzene in the Plucks' wells never exceeded the maximum permissible contaminant level of 5 ppb designated by the EPA."); *Newkirk v. ConAgra Foods, Inc.*, 727 F. Supp. 2d 10006, 1015 (E.D. Wash. 2010) ("As to general causation, Dr. Egilman forwards the following opinions: "There is no known safe level of diacetyl exposure.")

[14] *See, e.g.,* Eaton, *supra* n.6 at 29 (discussing accumulation of dioxin in the human body).

[15] Plaintiff-Appellant's reference to the "millions" of fibers in brake dust is highly misleading because the fibers, like particles of dust, are incredibly small and the reference to a (Continued...)

Further, the industrial hygiene principles that apply to other carcinogens also apply to asbestos. Asbestos is measured via the number of fibers of a certain length captured by a monitoring device in the worker's breathing zone.[16] A similar process is used for other dust, fiber, and fume based workplace materials. And estimating a worker's dose of asbestos is not particularly different than estimating that of a long-time service station attendant's occasional exposures to benzene in gasoline (as in *Parker*).

3. Zero Exposure Is Not Necessary to Protect Against Asbestos Diseases.

Plaintiff-Appellant's theory of causation would require a "zero exposure" approach to asbestos, but "zero" is not consistent with the findings of science or the goal of health standards. Many asbestos workers have received minor or low-level asbestos exposures with no apparent harm, especially from chrysotile. Some worker populations have not shown any increased asbestos disease despite working with asbestos their entire careers. The vehicle mechanic studies discussed in *Juni*

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pure fiber count has no meaning from a health standpoint. Industrial hygienists instead use the concept of a fiber/cc longer than five microns to compare exposures to health standards. Claiming that an exposure involves "millions" of fibers is no more meaningful that pointing out that household air has "millions" of dust particles in it or background exposures involve millions of asbestos fibers.

[16] *See* OSHA Detailed Procedures for Asbestos Sampling and Analysis (App. B. to 29 CFR § 1910.1001).

are a good but only one such example.[17] South African chrysotile miners likewise have not demonstrated a single case of mesothelioma despite decades of heavy mining exposures.[18] Chrysotile is the same fiber type found in the brake linings, clutches and gaskets at issue in this case.

Demonstrating that the goal for health purposes has always included an expected level of inconsequential exposure, OSHA's asbestos standard today is *not* zero – it is 0.1 f/cc on an 8-hour time-weighted basis. According to OSHA's regulations, this level is an "acceptable exposure" for a 45-year work life – an overall life dose of 45 fibers/cc year. The U.S. Environmental Protection Agency allows school children back into an asbestos-remediated school building if exposures are below 0.01 f/cc – again, not zero.[19]

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[17] The studies are summarized and discussed in David Garabrant, et al., *Mesothelioma among Motor Vehicle Mechanics: An Updated Review and Meta-analysis*, ANN. OCCUP. HYG. 1-19 (2015) (prepublication version available at http://annhyg.oxfordjournals.org/). *See also* Julian Peto *et al.*, *Occupational, Domestic and Environmental Mesothelioma Risks in Britain: A Case-Control Study*, UK HEALTH & SAFETY EXEC., at x (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).

[18] *See* David Rees, *Case Control Study of Mesothelioma in South Africa*, 35 AM. J. INDUS. MED. 213, 220 (1999).

[19] Asbestos Hazard Emergency Response Act (AHERA), 40 CFR Pt. 763, §763.90(i)(5).

B. The *Juni* Court Correctly Rejected the Experts' Failure to Assess Dose as Part of a Causation Opinion.

The trial court understood that the experts' failure to assess dose is fatal to their opinions. A proper analysis of causation in any toxic tort case must begin with an assessment, or at least a reasonable estimate, of the likely dose received by the plaintiff.[20] The expert should then compare that dose to the dose known to cause the subject disease, typically through epidemiology studies.[21] If the scientific evidence includes a series of epidemiology studies in which some highly exposed populations have an increased level of disease, and other populations of much lower exposed workers shows no increased disease, the experts need to prove that the plaintiff's exposures match those of the higher exposed group. And the testimony needs to concentrate on the exposures really at issue – here, to the

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[20] It does not matter that a particular individual was not himself or herself subjected to air monitoring during the relevant exposures and time periods. Epidemiologists and other researchers have long engaged in the widely recognized practice of "dose reconstruction" for a group of workers who may not have been monitored – they do it by estimating the duration, frequency, and intensity of the worker's exposure, compared to studies performed on similar work activities or populations. For Mr. Juni, there are dozens of studies of the amounts of exposure from activities similar to his work environment (Plaintiff-Appellant cites to just a few of them), and they can be used to estimate the range of dose he likely received. This task is not impossible, and is not even difficult if the worker can recount his exposures with a modest degree of detail.

[21] *Parker*, 7 N.Y.3d at 449-450 (rejecting expert testimony that failed to include any "scientific expression of [plaintiff's] exposure level" and also failed to specify how any such exposure exceeded that found sufficient to cause disease in relevant epidemiological studies). *See also* Federal Judicial Center, *Reference Manual on Scientific Evidence, Reference Guide on Epidemiology* 338 (3d ed. 2011).

much less potent chrysotile form of asbestos as used in resin-bonded brake pads, not to asbestos in general.

The decision by the trial court in *Juni* correctly applied this fundamental dose principle, consistent with the vast majority of courts who have looked closely at this issue.[22] The *Parker* court, while acknowledging that modeling or other approaches may be appropriate means of estimating the dose (*Parker,* 7 N.Y.3d at 449), does not excuse experts from even trying or from using a scientifically acceptable means of distinguishing causative doses from non-causative exposures. "These, along with others, could be potentially acceptable ways to demonstrate causation *if they were found to be generally accepted as reliable in the scientific community.*" *Id. (emphasis added).* As reiterated by the Court of Appeals 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.

2016 WL 527107, at *5.

In this case, Plaintiff-Appellant's experts failed to use any method at all (let alone a reliable, scientifically accepted method) for assessing Mr. Juni's dose from

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[22] See *infra*, Section III.

his brake and clutch work. They 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 epidemiology studies showing that exposures like Mr. Juni's would cause disease. As described more in Section II below, the contrast with the requirements of *Parker*, *Cornell,* and *Sean R.* could not be more stark.

Indeed, the experts apparently did not even try, as the experts in *Parker* did, to use insufficient qualitative terms like "excessive" or "extensive" to describe Mr. Juni's exposures.[23] Dr. Moline "did not know if Juni had worked with friction brakes, clutches, or gaskets sold or distributed by defendant . . . or how often he had been exposed to such products, nor did she attempt any dose reconstructions or assessments to quantify his exposure." *Juni,* 48 Misc.3d at 467. The trial court here correctly refused to allow this attempted end-run around well-established causation principles, and its decision should be upheld.

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[23] The closest adjective they seem to have come up with is "regular." *See Juni*, 11 N.Y.S.3d at 435.

II. Plaintiff-Appellant's Experts Did Not Meet the Clear Requirements of the New York Trio of Causation Evidence Cases – *Parker, Cornell* and *Sean R.*

The seminal Court of Appeals *Parker* case set the ground rules in New York for sufficient causation evidence in a toxic tort case – rules that these two experts failed entirely to follow. One of the *Amici* joining this brief (the Coalition for Litigation Justice) also participated in the Court of Appeals *Parker* briefing, for the very purpose of assisting that Court in understanding how dose works in toxicology and why a clear assessment of dose is critical to proving causation in a litigation context. *Parker* is not only the applicable law in New York, the case has been cited favorably elsewhere as a model for how dose assessment should work in a toxic tort case.[24]

The experts in *Parker* undertook the same or very similar flawed and unscientific approach as the one used by Drs. Markowitz and Moline here:

- The *Parker* experts conflated exposure to benzene with exposure to gasoline to avoid the epidemiology studies documenting that gasoline exposures do not cause AML. *Parker,* 7 N.Y.3d at 445, 450. The experts here 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 – and for the same purpose, to avoid dealing with the many epidemiology studies finding no link between mechanic work and mesothelioma.

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[24] *See, e.g., Adams v. Cooper Indus., Inc.,* 2007 WL 2219212, *6, n.4 (E.D. Ky. 2007); *Henrickson,* 605 F.Supp.2d at 1176.

- The *Parker* experts relied on the anecdotal exposures to gasoline of the plaintiff gas station attendant (*id.* at 442) 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 activities around and with brakes, gaskets, and clutches and without any attempt to assess the dose.

- The *Parker* experts cited to studies showing that benzene causes AML, but those studies involved highly exposed factory workers and raw benzene and were not probative of whether the small amounts of benzene in gasoline would do the same.[25] Drs. Markowitz and Moline likewise rely on high dose, mostly amphibole asbestos studies, to claim that much lower exposures to chrysotile asbestos in brakes cause mesothelioma.

- The *Parker* experts ignored several epidemiology studies finding no link between service station work and AML (*id.* at 443-44, 450). Both Drs. Markowitz and Moline here ignore a far larger set of epidemiology studies showing no link between mechanic work and mesothelioma (21 out of 22 studies showing no association).

- The *Parker* experts relied on conclusory statements ("excessive," "extensive") in lieu of a dose assessment. 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." *Id.* at 446-47. This is the *any exposure* theory, and Drs. Markowitz and Moline used the same or very similar flawed approach as to asbestos.

The two more recent opinions of the Court of Appeals in *Cornell* and *Sean R. v. BMW* applied and extended *Parker* to make the same points even more

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[25] *Parker*, 7 N.Y.3d at 444 ("Landrigan cited several studies that linked benzene exposure to leukemia," including an Ohio rubber plant).

forcefully, and in other toxic tort contexts. The *Cornell* court rejected the expert's testimony in a mold case because the expert "made no effort to quantify [plaintiff's] level of exposure" to mold. *Cornell*, 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." *Sean R.*, 2016 WL 527107 at 4. Much like the *Sean R.* experts, Drs. Markowitz and Moline essentially engage in circular logic by concluding that since asbestos exposure causes mesothelioma, Mr. Juni must have been exposed to enough asbestos to cause his mesothelioma. The circular logic is even more egregious where these and other plaintiff experts acknowledge that a fair percentage of mesotheliomas in epidemiology studies are not known to be associated with asbestos at all.[26] And these experts' reliance on "visible dust" as proof of sufficient exposure also duplicates the *Sean R.* experts' attempt to rely on smell and symptoms to prove a causative level of gasoline in the car – an approach the

[26] An increasing proportion of these cases are likely spontaneous, produced by errors in the human body's transcription of DNA billions of times in reproducing cells. The medical literature fully documents the existing of spontaneous cases, for all cancers and for mesothelioma specifically. *See* Stanley Venitt, *Mechanisms of Spontaneous Human Cancers* 104 ENVIRON. HEALTH PERSP. 633, 633, 635 (1996), article available at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1469658/; Christian Tomasetti, and Bert Vogelstein, *Variation in Cancer Risk Among Tissues Can Be Explained by the Number of Stem Cell Divisions*, 347 SCIENCE 78 (Jan. 2015) ; B.T. Mossman *et al., Asbestos: Scientific Developments and Implications for Public Policy*, 247 SCIENCE at 294 (1990) ("approximately 20 to 30% of mesotheliomas occur in the general population in adults not exposed occupationally to asbestos"). *See, e.g., Butler v. Union Carbide Corp.*, 712 S.E. 2d 537 (Ga. App. 2011), (acknowledging role of spontaneous mesotheliomas).

Court rejected. The *Juni* opinion simply applies well-established New York law to asbestos litigation.

III. The *Juni* Decision Is Supported by Many Other Decisions Around the Country Rejecting Cumulative and Any Exposure Testimony.

Juni is hardly the first opinion in the country to reject the testimony of experts who attempt to opine as to causation while eschewing any assessment of the dose. In fact, dozens of courts have already rejected the *any exposure* theory (along with other failures to include dose) as applied in both asbestos and other contexts.[27] In addition to the Court of Appeals in *Parker, Cornell,* and *Sean R.,* the courts rejecting or at least challenging the *any exposure* theory and its kin include several state supreme courts, multiple state intermediate courts of appeals, two federal circuit courts, and many federal district and state trial courts in Texas, Pennsylvania, Nevada, Virginia, Florida, Delaware, Ohio, Louisiana, Mississippi, Utah, California, Washington, North Carolina, and Pennsylvania. Most of the opinions are in the asbestos context, but many involve other alleged exposures, thus demonstrating that asbestos litigation is just another toxic tort and needs to follow the same rules. Some highlights of those rulings include the following:

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[27] For a survey of *any exposure* opinions and issues, *see* Mark Behrens & William Anderson, *The "Any Exposure" Theory: An Unsound Basis for Asbestos Causation and Expert Testimony*, 37 SW. U. L. REV. 479 (2008); William Anderson, Lynn Levitan & Kieran Tuckley, *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. POLICY 1 (2012).

- The Supreme Court of Pennsylvania has soundly rejected *any exposure* testimony three times in asbestos litigation, calling the theory a "fiction" and requiring experts to prove a causative dose.[28]

- The Virginia Supreme Court held that experts "must opine as to what level of exposure is sufficient to cause mesothelioma, and whether the levels of exposure at issue . . . were sufficient."[29]

- The Texas Supreme Court (twice) and two Texas intermediate courts have considered multiple aspects of the *any exposure* theory and plaintiff arguments for it, and have rejected all of them.[30]

- The federal Sixth Circuit Court of Appeals has rejected *any exposure* testimony four different times, both in asbestos cases and otherwise.[31]

- The Ninth Circuit Court of Appeals remanded an $11 million trial verdict because the district judge did not sufficiently test, among other things, the *any exposure* theory of the plaintiff's experts.[32]

- Multiple federal district courts have rejected *any exposure* testimony in carefully reasoned and well-documented diseases, much like the *Juni*

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[28] *See Betz v. Pneumo Abex LLC*, 44 A.3d 27 (Pa. 2012). *See also Gregg v. V-J Auto Parts Co.*, 943 A.2d 216 (Pa. 2007); *Howard ex rel. Estate of Ravert v. A.W. Chesterton, Inc.*, 78 A.3d 605 (2013). A third case, *Rost v. Ford Motor Co.*, No. 56 EAP 2014, is presently on appeal and awaiting oral argument.

[29] *Ford Motor Co. v. Boomer*, 736 S.E.2d 724, 733 (Va. 2013). *See also Wannall v. Honeywell Int'l, Inc.*, 292 F.R.D. 26 (D.D.C. 2013) (applying *Boomer*), *aff'd*, 775 F.3d 425 (D.C. Cir. 2014).

[30] *See Bostic v. Georgia-Pacific Corp.*, 439 S.W.3d 332 (Tex. 2014); *Flores v. Borg-Warner Corp.*, 232 S.W.3d 765, 772 (Tex. 2007); *Georgia-Pacific Corp. v. Stephens*, 239 S.W.3d 304 (Tex. App.-Houston 2007); *Smith v. Kelly-Moore Paint Co., Inc.*, 307 S.W.3d 829 (Tex. App. 2010).

[31] *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); *Moeller v. Garlock Sealing Tech., LLC*, 660 F.3d 950 (6th Cir. 2011); *Martin v. Cincinnati Gas & Elec. Co.*, 561 F.3d 439 (6th Cir. 2009); *Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671 (6th Cir. 2011) (benzene).

[32] *Estate of Barabin v. AstenJohnson, Inc.*, 740 F.3d 457, 464-65 (9th Cir.), *cert. denied*, 135 S. Ct. 55 (2014).

opinion.[33] The *Juni* court cited to an extended discussion of one of these decisions, *Comardelle v. PennGen Ins. Co. Juni,* 11 N.Y.S.3d at 437.

Many of these opinions are under *Daubert* or *Frye* standards, but they all turn on the lack of logical, scientific foundation for the speculative opinions of experts who testify essentially as Drs. Markowitz and Moline. Thus, the reasoning of these cases applies equally to the post-trial rulings in *Juni* directed to the foundation of the experts' testimony. As all of these other courts have held, *cumulative* or *any exposure* testimony (1) is illogical because it ignores these experts' own admission that background exposures also accumulate in the lungs, in the millions of fibers, but are *not* causative; (2) assumes improperly that disease caused at high levels of exposure would also occur at much lower doses with no evidence that it does; (3) disregards the difference in fiber potency by treating chrysotile exposures (*e.g.,* brake and clutch exposures) the same as amphibole

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[33] *See, e.g., Yates v. Ford Motor Co.,* 113 F.Supp.3d 841, (E.D.N.C. 2015); *Smith v. Ford Motor Co.*, No. 2:08-CV-630, 2013 WL 214378 (D. Utah Jan. 18, 2013); *Sclafani v. Air & Liquid Sys. Corp.*, No. 2:12-CV-3013, 2013 WL 2477077 (C.D. Cal. May 9, 2013); *In re W.R. Grace & Co.*, 355 B.R. 462 (Bankr. D. Del. 2006), *appeal denied*, 2007 WL 1074094 (D. Del. Mar. 26, 2007); *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142 (E.D. Wash. 2009) (benzene); *Comardelle v. Penn Gen. Ins. Co.,* No. 13-6555, 2015 WL 64279 (E.D. La., Jan. 5, 2015). *See also Barabin,* 740 F.3d at 464-65 (reversing trial verdict on the grounds that the trial judge did not perform a sufficiently rigorous *Daubert* review of expert testimony, including the *any exposure* approach). Although the federal courts, and some state courts, rely on the *Daubert* standard, the *Parker* court found *Daubert opinions* instructive in their discussion of reliability. 7 N.Y.3d at 448, n.4. Indeed, as the New York Court of Appeals explained, even when the general acceptance test is resolved under *Frye*, there is a separate and distinct admissibility inquiry that must be made as to the "specific reliability of the procedures followed to generate the evidence proffered and whether they establish a foundation for the reception of the evidence at trial." *Id.* at 447 (quoting *People v. Wesley*, 83 N.Y.2d 416, 429 (1994)).

exposures such as insulation; and (4) has no epidemiology studies to support the notion that even the lowest levels of exposure are causative.

The *any exposure* theory also eliminates plaintiff's ordinary "substantial factor" burden of proof, which requires distinguishing "substantial" from "insubstantial" exposures based on credible studies or other evidence. According to Drs. Markowitz and Moline, plaintiff need only claim breathing "dust," and then defendants must prove those exposures non-causative. In fact, as other courts have noted, none of these experts has ever published a study supporting the notion that any amount of workplace exposure, or the mere breathing of dust, must be considered causative – they only express these opinions in court. New York law requires more.[34]

IV. The Key Elements of Plaintiffs' Causation Theory Are Illogical and Unscientific.

The *Juni* opinion is one of the most thorough and articulate in the country in examining the underpinnings of the plaintiffs' experts' theory and then dissecting the lack of logic and scientific validity in each of them. It is an opinion that cannot

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[34] *See Cornell,* 22 N.Y.3d at 784, 986 N.Y.S.2d 389, 9 N.E.3d 884 ("At a minimum, ... there must be evidence from which the factfinder can conclude that the plaintiff was exposed to levels of that agent that are known to cause the kind of harm that the plaintiff claims to have suffered.") *quoting Wright v. Willamette Ind., Inc.,* 91 F.3d 1105 (8th Cir.1996)). *See also* David E. Bernstein, *Getting to Causation in Toxic Tort Cases,* 74 BROOK. L. REV. 51, 59 (2008) (recent any exposure opinions acknowledge that *de minimis* exposure to asbestos should not suffice for causation).

easily be discounted. Plaintiff-Appellant's attempts to nit-pick the decision do not do justice to the forcefulness of the opinion's primary findings and the careful examination the court undertook. The trial court looked beyond the self-serving statements of the experts and discovered the many holes and inconsistencies behind the Plaintiff-Appellant's causation theories. This is an essential role for a gatekeeping trial court or one that, in this case, tested the sufficiency of the evidence following a full trial presentation by the experts. The key elements of the opinion, and why the trial judge got it right, are addressed briefly below.

A. The *Juni* Court Framed the Issue Correctly – These Experts Cannot Rely on the General Proposition that Asbestos Causes Mesothelioma.

The *any exposure* experts engage in a sleight of hand when asked to produce epidemiology studies demonstrating that exposure to low levels of chrysotile asbestos, like those a vehicle mechanic would receive, causes mesothelioma. In response, they repeatedly resort to the mantra that "all forms of asbestos are known to cause mesothelioma." The *Juni* trial judge recognized this diversion and required the experts to demonstrate that *mechanic* work with specific products (brakes, gaskets, clutches) was a cause of disease, not just asbestos generally, because of the many differences between raw asbestos exposure scenarios (*e.g.*, insulator or shipyard work) and the much more limited exposures to chrysotile

asbestos from mechanic work. In doing so, the court faithfully applied the lesson of *Parker* to frame the issue properly and insist on proof of the exposure at issue.

The trial court's required proof makes, if anything, even more sense for asbestos than for the benzene/gasoline situation in *Parker*. In *Parker,* the gasoline contained the same substance (benzene) as the industrial AML studies, just a great deal less of it. Here, the brake work does not even contain the same type of asbestos fiber as most of the higher exposure epidemiology studies relied on by Plaintiff-Appellant's experts. As courts have noted, not all forms of asbestos are the same.[35] Ford also introduced considerable evidence of the many changes to the chrysotile fibers occurring either through the manufacturing process or via the intense heat of braking, and neither expert took any of that information into consideration. The correct causation principle these experts needed to establish was that mechanic work causes mesothelioma, not that asbestos or even chrysotile asbestos does.

B. Testimony About Increased "Risk" Is Not a Substitute for Competent Causation Testimony.

Dr. Markowitz also attempted an end run around causation testimony by resorting to "increased the risk" testimony. *See, e.g., Juni* at 464 ("no level has

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[35] *See, e.g., Yates v. Ford Motor Co.*, 113 F.Supp.3d at 853-54; *Butler*, 712 S.E.2d at 542.

been identified that separates out increased risk from no risk"); 466 (Markowitz "not aware of any epidemiological cohort studies supporting his opinion that there is an increased risk of contracting mesothelioma from exposure to auto brakes, clutches, or gaskets"). What he means by his "increased risk" testimony is that breathing a few fibers must present a greater risk that not breathing a few fibers, since additional fibers are added to the lungs. He then makes the great leap to the conclusion that "increased risk" must therefore also equate to causation. And he does this with no studies to support his claim.

This mixing of the concepts of risk and causation is a common diversionary tactic of these experts that is intended to hide the lack of causation studies to support their claims. The "risk" approach is completely speculative, is at most an unproven hypothesis, and has no scientific support. Dr. Markowitz does not even attempt to quantify the increase in risk to show that it is a meaningful number in the context of health consequences. The "risk" testimony also completely fails the standard of proof in New York, which is not based on risk but on causation.[36]

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[36] "Proof of a risk, even an increased risk, does not constitute proof of causation." *Juni,* 11 N.Y.S.3d at 433; *Cornell,* 22 N.Y.3d at 782–783, 986 N.Y.S.2d 389, 9 N.E.3d 884 (reports and studies using terms like risk, link, or association do not establish general causation). *See also Butler,* 712 S.E.2d at 552 (risk assessment is distinct from causation assessment).

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

The *Juni* court correctly rejected Plaintiff-Appellant's attempt to substitute the *Lustenring* series of cases for *Parker* and *Cornell* (the Court of Appeals *Sean R.* opinion had not issued yet). *See Juni,* 11 N.Y.S.3d at 429-31. *Lustenring* pre-dated *Parker,* and none of the cases citing to *Lustenring* are Court of Appeals opinions. Whatever *Lustenring* stands for, it does not appear to be good law following *Parker, Cornell,* and *Sean R.*

What Plaintiff-Appellant is actually seeking, however – as noted by the *Juni* court – is to be relieved of any obligation of proving a causative dose in an asbestos case. *See id.* at 432 ("plaintiffs suggest that they should be relieved of the burden of establishing some quantifiable level of exposure"). But Plaintiff-Appellant has provided no meaningful scientific basis to treat asbestos as if it were not a dose-based toxin. *Parker* applies, and the only methods to causation are through a competent dose assessment or something close to that (*e.g.*, modeling).

In particular, Plaintiff-Appellant's insistence that mere "dust" suffices was properly rejected by the trial court. *Juni*, 11 N.Y.S.3d at 435-36. The reasons mere "dust" testimony can never substitute for an actual dose assessment are many, including:

- Workplaces experience all kinds of dust, including the ordinary kind that invades homes, businesses, cars and every other human place of activity. Witnesses typically cannot distinguish between ordinary dust and asbestos-containing dust.

- Even dust from an asbestos-related activity can contain a wide variation in quantity of asbestos, which means one type of dust could be harmless and another potentially dangerous. That is why a dose assessment of the exposure, based on competent industrial hygiene studies, is critical.

- A great deal of "dust" from an asbestos-related work activity is often not even respirable and/or would not make it into the worker's breathing zone – yet another reason why industrial hygiene exposure studies from similar work activities are essential, not just sightings of dust

- In the heated environment of litigation, witnesses will invariably identify "dust" being present, which under these experts' approach would constitute a form of absolute liability for any company utilizing an asbestos product.

Claims of "dust" exposure are very similar to the rejected notion in *Parker* and *Sean R.* that breathing of fumes or detecting an odor suffices for causation. *Parker,* 7 N.Y.3d at 449-50 (expert's opinion that plaintiff was "frequently" exposed to "excessive" amount of benzene, without foundation, "cannot be characterized as a scientific expression of Parker's exposure level"). Likewise, the presence of mold in *Cornell* was not enough without a measured exposure. 22 N.Y.3d at 784. 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. Other courts have rejected reliance on speculative testimony of "dusty" conditions. *See, e.g., Sterling*

v. P&H Mining Equip., No. 1006 EDA, 2015 WL 1743156 at *4 (Pa. Super. Apr. 17, 2015), at 8 (plaintiff testimony that he "saw dust" insufficient with no proof that dust contained asbestos, multiple potential other sources of dust in industrial facility, no testimony as to distance from dust, etc.); *Yates,* 2015 WL 3948303 at *8-*9 (critiquing and rejecting expert's reliance on "visible dust" as a basis for causation finding); *Borg-Warner*, 232 S.W.3d at 774 (testimony re "clouds" of dust insufficient because "we do not know the contents of that dust, including the approximate quantum of fibers to which [plaintiff] was exposed").

The Court can use this opportunity to confirm that in asbestos litigation – as in all other types of toxic tort litigation – a plaintiff's experts must assess and establish a causative dose to prevail at trial, even if the plaintiff can claim to have breathed "dust" or seen asbestos-containing materials in some number of workplace occasions.

D. The Experts Cannot Rely on Statements in Governmental Publications About "No-Safe Dose" of Asbestos in Lieu of Actual Proof of Causation.

The trial court here also correctly rejected Plaintiff's experts' reliance on the notion that there is "no safe dose" of asbestos. That argument is derived not from any epidemiological or other study, but from government publications adopting conservative positions to protect public health broadly. These agencies are not

required to, and do not conform to, the causation standards of a court of law.[37] As the *Juni* court held, the assumption that there is "no safe dose" of a toxin is not a substitute for the required quantification (or estimation) of an individual plaintiff's exposure, and "the reports and findings of governmental agencies [declaring there to be no safe dose of asbestos] are irrelevant as they constitute insufficient proof of causation." *Juni*, 11 N.Y.S.3d at 432, 433. Both the *Cornell* and *Parker* courts rejected such reliance, so there is no room whatsoever for Plaintiff-Appellant to travel down this route in an asbestos case. *Cornell,* 22 N.Y.3d at 782 (standards promulgated by regulatory agencies as protective measures inadequate to establish legal causation); *Parker*, 7 N.Y.3d at 450 (same).

These experts' reliance on a "no safe dose" theory fundamentally exposes their inability actually to prove causation. Having failed to produce such proof, they fall back on the premise that there is *no proof of a safe dose so therefore all*

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[37] *See Sutera v. Perrier Group of America,* 986 F. Supp. 655, 664-65 (D. Mass. 1997) (explaining that a regulator's "threshold of proof is reasonably lower than that in tort law"); *Baker v. Chevron USA, Inc.*, 680 F.Supp.2d 865, 880 (S.D. Ohio 2010) ("[R]egulatory agencies are charged with protecting public health and thus reasonably employ a lower threshold of proof in promulgating their regulations than is used in tort cases."); Eaton, *supra* note 6 at 39 ("[R]egulatory levels are of substantial value to public health agencies charged with ensuring the protection of the public health, but are of limited value in judging whether a particular exposure was a substantial contributing factor to a particular individual's disease or illness.").

doses must be unsafe. Putting aside the lack of scientific support for this theory, it is not a proper basis for a causation opinion, as other courts have held.[38]

E. Plaintiff-Appellant's Experts Cannot Credibly Discount or Ignore the Mechanic Epidemiology Studies.

The *Juni* court correctly analyzed the role that the 22 mechanic epidemiology studies should play in a case such as this, and correctly held that Plaintiff-Appellant's experts were not engaged in a scientifically reliable process by discounting that entire set of studies. *Juni,* 11 N.Y.S.3d at 484.[39] This holding is consistent with those of other courts, in circumstances where experts attempted to "reinterpret" or disregard compelling epidemiology to render opinions inconsistent with the overall findings of those studies. The original *Daubert* litigation, for instance, involved an expert who discounted the findings of a series of studies showing no birth defect link to the drug Bendectin, and instead interpreted those studies to show such a link.[40] The situation here is closely comparable to that in *Parker*, where studies linked benzene in industrial exposure settings to AML, but studies of service station workers and others exposed to *gasoline* (with small amounts of benzene in it) found no such link. Here, although

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[38] *See Butler*, 712 S.E. 2d at 552 n.37 (citing *Parker* for its correct rejection of reliance on regulatory pronouncements); *Bostic*, 439 S.W.3d at 358 (explaining that "the failure 439 S.W.3d of science to isolate a safe level of exposure does not prove specific causation").

[39] *See infra* n.11.

[40] *See Daubert v. Merrell Dow Pharma. (Daubert II),* 43 F.3d 1311 1314 (9th Cir. 1995).

studies have linked asbestos in certain high-exposure industries with mesothelioma (primarily from amphibole exposure), the studies clearly show no such link for mechanics who are exposed to low amounts of chrysotile asbestos, even over a lifetime of such work.[41]

The Plaintiff-Appellant's experts' response to this compelling set of studies consists of minor criticisms that are true of virtually any set of epidemiology studies. The litany of criticisms (not enough studies, not enough power, the authors are biased, the studies are inconclusive, "vehicle mechanics" may not have actually worked on brakes) rings hollow when matched up against the repeated, consistent finding of over sixty researchers in seven different countries that looked at thousands of persons employed in some capacity of repairing cars, trucks and other brake-containing vehicles. The *any exposure* experts have claimed for years that the mechanic studies are inconclusive and not good enough, even as more and more of them were performed each year and virtually every one of them produced the same finding: there is no evidence that mechanic work causes mesothelioma. One of the most recent is the largest case control study of mesothelioma ever

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[41] Plaintiff-Appellant's experts' biased agenda is spotlighted by their reliance on only one of the 22 studies, Roelofs, while ignoring the other 21. Epidemiology studies must be interpreted as a whole and not cherry-picked in this fashion. Roelofs itself has many flaws – the study found increased odds ratios for occupations that have nothing to do with asbestos, such as postal workers and drafting jobs, and the authors do not claim that their finding proves the link between mechanic work and mesothelioma but only justifies "further surveillance." Cora R. Roelofs, et al., *Mesothelioma and Employment in Massachusetts: Analysis of Cancer Registry Data 1988-2003*, 56 AMER. J. INDUS. MED. 985 at 7(2013).

performed in the United Kingdom, by highly respected (and not asbestos-defendant funded) researchers Julian Peto and Christine Rake. The findings were the same: "We found no evidence of increased risk associated with non-industrial workplaces or those that were classified as 'low risk', including motor mechanics and workers handling gaskets and mats that may have contained asbestos."[42]

In some states, including New York, plaintiffs in toxic tort litigation are not necessarily obligated to produce epidemiology to prove causation. But if no epidemiology exists to support their claims, presumably their burden of proof becomes *harder,* not easier, requiring some incredibly clear and strong evidence to counter the lack of epidemiology. The experts' reliance on a handful of case reports and the "no safe dose" theory hardly reaches that level. And in circumstances like this, where a large set of epidemiology studies says no, an expert who ignores these studies and instead says "yes" is not engaged in a scientific process.

CONCLUSION

If the *Parker, Cornell,* and *Sean R.* opinions mean anything, at a minimum they compel experts such as these to at least credit the need for a dose assessment by some scientific methodology. These experts have undertaken no such

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[42] Julian Peto *et al., supra,* n.17 ; Rake, C., et al, *Occupational Domestic and Environmental Mesothelioma risks in the British Population: A Case Control Study,* 100 BRITISH J. CANCER 1175, 1182 (2009).

assessment, providing this Court with the opportunity to confirm that *Parker* extends (as it must) to asbestos litigation and that no asbestos case can proceed in this state without following the *Parker*-mandated approach to causation. *Amici* urge the Court to adopt the trial court's ruling in *Juni* in its entirety and help redirect asbestos litigation in this state to one grounded in science and not speculation.

Respectfully submitted,

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