

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)

REPLY 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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INTRODUCTION

Amici curiae respectfully move for leave to file this reply brief due to the unusual nature and extent of argument contained in the Plaintiff-Appellant's response to the initial *Amici* brief (the "Response Brief") in support of Appellee Ford Motor Company. A number of the arguments made in the Response Brief are factually inaccurate, and others misrepresent *Amici's* initial arguments or the Plaintiff-Appellant's experts' actual positions. Fundamentally, the Response brief does not rebut or refute the most critical flaws in the testimony of Drs. Markowitz and Moline, nor does it deflect the clear applicability of *Parker v. Mobil Oil*¹ to this case. *Amici* stand by their statements of both the science and the law as previously set forth and addressed further herein and urge the Court to uphold Judge Jaffe's well-reasoned opinion rejecting the expert testimony.²

¹ *Parker v. Mobil Oil Corp.*, 7 N.Y.3d 434 (2006).

² The Response Brief is written with a significant amount of *ad hominem* criticism of the *Amici* themselves for filing a brief that Plaintiff-Appellant apparently views as improper. Rather than respond to those criticisms, *Amici* note that similar briefs written and filed by the Coalition for Litigation Justice and others have been accepted by appellate courts, without criticism, and in some cases cited or quoted, in Pennsylvania, Texas, California, Georgia, Illinois, Michigan, Washington, Virginia, the Ninth Circuit Court of Appeals, and other appellate jurisdictions. The New York Court of Appeals accepted a very similar amicus brief from the Coalition for Litigation Justice in the *Parker* case itself.

ARGUMENT

I. **The Response Brief Largely Ignores the Controlling Requirements of *Parker*.**

Plaintiff-Appellant barely mentions the most important and controlling statement of New York law on this type of expert testimony – the *Parker* case – until the very end of the Response Brief. And yet the *Parker* opinion is controlling law for this appeal. The *Parker* Court unequivocally rejected testimony founded on no attempt to assess or quantify the dose.³ Plaintiff-Appellee’s *lawyers* have gone to great lengths in the Response Brief (pp. 5-13) to save the opinions of Drs. Markowitz and Moline by arguing exposure evidence. But the experts themselves did not identify any need for or rely on the *extent* of Mr. Juni’s exposures for their opinions. The foundation of their opinions is the simple formula of exposure equals causation – Mr. Juni worked with asbestos-containing brakes, therefore that work is the cause of his disease. This is classic error under *Parker*.

Nor does the Response Brief deal with these experts’ fundamental error of wrongly defining the exposure at issue to begin with. The *Parker* court established as a first principle that the experts in question must rely on the type of studies relevant to the actual exposure. In *Parker* the experts jumped from studies documenting disease from heavy exposures to benzene in its pure form in factory

³ *Parker*, 7 N.Y.3d at 449-50; *see Juni v. A.O.Smith Water Prods.*, 11 N.Y.S.3d 416, 426-27 (N.Y. 2015).

settings, to the much different world of gasoline exposures (containing only small amounts of benzene). *Parker*, 7 N.Y.3d at 432-33, 445, 450. In the process, the *Parker* experts discounted and ignored several studies showing no association between exposure to gasoline itself and the disease at issue. *Id.* at 442, 443-44, 450. The *Parker* court noted repeatedly the error in that approach.

The Response Brief does not refute the experts' similar error here – that Drs. Markowitz and Moline focused on studies of exposures to asbestos generally (typically from insulation work and products) as a cause of mesothelioma to opine that *mechanic* work with bonded, chrysotile-only brakes and friction products must also be a cause of disease. *See Juni*, 11 N.Y.S.3d at 421. Rather than deal with this major flaw, the Response brief doubles down on using the wrong exposure/disease analysis.⁴ The brief also fails to cite to any series of compelling studies demonstrating excess mesothelioma in *mechanics* (there is no such series) as opposed to differently exposed occupations.⁵ The failure of these experts to

⁴ Plaintiff-Appellant's reliance on general asbestos versus the specifics of a mechanic's work is documented by the following Response Brief claim at p. 8: "Mr. Juni's exposures to visible dusts comprised of asbestos fibers released upon sanding new brakes, and asbestos dusts released from clutch and gasket work, were *no different from any asbestos victim's exposure's (sic)* to visible asbestos dusts from *any other asbestos-laden product*" (emphasis added). This statement equates the minimal chrysotile release from brake, gasket, and clutch work to, for instance, the far more excessive releases of amphibole fibers from extended work with insulation, in shipyards, and in factories. The *Parker* experts made the same error.

⁵ *Juni*, 11 N.Y.S.3d at 421. The Response Brief makes a fundamental scientific error by asserting that "a valid epidemiological study must begin with an exposed group" and then criticizing the vehicle mechanic studies because they do not do so. This statement refers to (Continued...)

utilize the relevant studies (the vehicle mechanic studies) is fundamentally the same error as that of the experts in *Parker*.

In addition, the entire methodology used by the experts in *Juni* is on all fours with the rejected methodology in *Parker*. In relying on their *cumulative* or *any exposure* testimony, these experts opined that Mr. Juni's brake exposures, without any attempt to quantify or measure their degree or extent, were sufficient to cause disease. Like the *Parker* benzene experts, these asbestos experts did not identify any guiding principle that would distinguish between causative brake exposures (assuming there are any) and those that are not. Dr. Markowitz, as Plaintiff-Appellant helpfully repeats in the Response Brief, relies on a methodology with no foundation – merely “adding up all of the individual exposures” (Response Brief p. 17).⁶ Thus, according to Dr. Markowitz, all work activity exposures must be counted as significant, excessive, and causative, and he needs no further information on dose or extent of exposure. This is the exact approach of the

“cohort” studies, one major type of epidemiological study. But it ignores “case control” studies – the other major category. Case control studies begin with persons who have the disease and look backward to determine whether exposures in their past are excessively associated with the disease group. See Federal Judicial Center, Michael Green et al., *Reference Guide on Epidemiology*, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 3d at 559 (2011). Most of the vehicle mechanic studies are of this type. Only three of the 22 vehicle mechanic studies were funded by automotive companies – begging the question why the Response Brief refers to the entire set as studies “funded by Ford and other automobile companies” (Response Brief at 33-34).

⁶ Dr. Markowitz actually never “added up” anything in a quantitative sense because he did not estimate or rely on any “total” fiber dose level. All he means by this phrase is that he includes any form of asbestos work activity exposure as causative.

benzene testimony in *Parker* – no dose assessment, relying on a series of anecdotal exposures, and substitution of irrelevant epidemiology studies for those on point.

Plaintiff-Appellant makes an effort at the end of the brief to distinguish *Parker* on the ground that the appellate court would allow “particularized issues” in another case to suffice. But there is no citation to the *Parker* case for this proposition. The *Parker* Court required a competent dose assessment by a scientific method – nothing less will suffice, in any toxic tort case. The specifics provided in the Response Brief are either indistinguishable from *Parker* or minor issues that do not salvage these experts’ fundamental failure to assess the dose.⁷

II. Both the *Cumulative Exposure* Theory and the *Any Exposure* Theory Are Inappropriate Bases for Testimony Under *Parker*, as *Amici* Made Clear in Their Opening Brief.

Plaintiff-Appellant attacks the *Amici* brief for falsely claiming that the experts testified in reliance on the *any exposure* theory. That attack is wrong on two counts – (1) Plaintiff-Appellant’s admission that these experts relied on the *cumulative exposure* theory is in fact an admission that they relied on the *any exposure* theory, because they are in essence the same thing; and (2) *Amici*

⁷ Plaintiff-Appellant’s approach to the *Sean R.* case is also not availing. The principal holding in *Sean R.* is that experts cannot rely on anecdotal evidence of exposure in lieu of competent exposure assessments – the same holding as *Parker*. *Sean R. v. BMW of North America, LLC*, 26 N.Y.3d 801 (2016). Plaintiff-Appellant argues that the “odor threshold” approach justifies her reliance on “dust” as an exposure assessment, but dust is a very common material in almost any setting. Reliance on seeing “dust” in a workplace is hardly a substitute for a dose assessment that the Court of Appeals would approve.

explicitly contended that *both* versions of this testimony (assuming they are even different) are invalid in any event.

The *any exposure* and *cumulative exposure* theories are indistinguishable in substance – the sophistry of changing the name does not change the essence of the theory. The *any exposure* experts claim that “each and every exposure” above background is a cause of mesothelioma, regardless of dose. The *cumulative exposure* experts, as Dr. Markowitz so stated, contend that “all individual exposures are cumulative,” regardless of dose, and all of them, individually and together, are therefore causative. Both theories include virtually all workplace exposures as part of the cause, without evidence those exposures either individually or as a whole rise to a level sufficient to cause disease. Neither theory provides any guidance for determining when an exposure is high enough – being in the presence of “dust” is good enough. See Response Brief at 8, 38; *Juni*, 11 N.Y.S.3d at 422 (Moline testified that “visible dust” is a surrogate for significant exposure). Neither theory has any need for industrial hygiene testimony as to the actual levels or degree of exposure. Neither theory carves out any reasonable type or degree of workplace exposure as non-causative. Both theories are based on the same fundamentals – the assumption that there is no safe dose of asbestos, the “increased risk” of being exposed to any amount of asbestos, the use of theoretical risk government standards, and the lack of epidemiology specific to the exposure at

issue to support the causation opinions. And the end result of both theories is exactly the same – under either theory, the experts include virtually all workplace exposures, regardless of degree, duration, or intensity, as a cause of disease.

What the *cumulative exposure* experts in fact do is mere sophistry. They claim that their testimony is now different (they used to testify to the *any exposure* theory until courts started excluding it) because they opine that *this plaintiff's exposures* are cumulatively sufficient, not that each and every exposure of this plaintiff is sufficient. There is no meaningful difference in these two propositions – despite forty pages of Response brief, Plaintiff-Appellant never identifies any principled basis for carving out some workplace exposures as causative under either theory.

If the Court does detect some difference in the two theories, it does not matter for purposes of the *Amici* brief because the brief, like the *Juni* opinion itself, explicitly addressed both. In recent cases, Plaintiffs' experts have increasingly attempted to use the purportedly new *cumulative* approach to avoid exclusion. Well aware of this trend, *Amici* addressed *both* versions repeatedly in its opening brief (because they both fail to assess the dose) to prevent the very argument Plaintiff-Appellant now makes.⁸ This entire section of the Response Brief ignores

⁸ See, e.g., *Amici* Brief at 1, 3-4 (“[T]he cumulative exposure theory relied on by Plaintiff-Appellant’s experts is not a sufficient basis for a jury verdict.”); *Id.* at 5 (“Many of those courts (Continued...)”)

the actual *Amici* argument and is therefore moot and irrelevant. The flaws are the same in both theories, as the *Juni* trial opinion so clearly articulates. *Juni*, 11 N.Y.S.3d at 436-39. *See also Yates v. Ford Motor Co.*, 113 F.Supp.3d 841, 849-63 (E.D.N.C. 2015) (distinguishing between *any exposure* theory and Dr. Mark's "special exposure" approach but finding the latter – based on cumulative exposure – also flawed scientifically).

III. Mr. Juni's Claimed Actual Exposures Were Not Excessive, Not in Excess of OSHA Standards, and Are Irrelevant to the Experts' Opinions.

Plaintiff-Appellant spends a large portion of the Response Brief arguing that Mr. Juni had significant exposures well above OSHA standards. There are multiple problems with those arguments.⁹

Most fundamentally, the experts did not rely on this evidence or need any testimony from an industrial hygienist laying out all the information provided in

[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" (citing *Juni*, 11 N.Y.S.3d at 437); *Id.* at 7 (criticizing claim that every exposure is "cumulative").

⁹ For reasons that are not clear, Plaintiff-Appellant chose to open her argument with an attack on a single statement on page 23 in the *Amici* brief referencing trial evidence of changes to chrysotile fibers in the manufacturing process and during braking. The Response Brief challenges that statement as not supported by the record. The discussion of those changes is contained in the court's opinion, *Juni*, 11 N.Y.S.3d, at 421 (with citations to the record), and at 435 – as admitted by Dr. Markowitz, the fibers are bound in resins and made not respirable during manufacturing, and they are converted by heat to non-asbestos material during braking. The point is a minor one in any event, and Plaintiff-Appellant's attack does not alleviate her experts' refusal to assess the dose.

the Response Brief. As noted above, the *cumulative exposure* theory simply includes “all individual exposures” as causative. Dr. Moline may have relied on a hypothetical about Mr. Juni’s exposures (Response Brief p. 20), but her opinion was in no way dependent on the *degree* of exposure in that hypothetical as opposed to the mere *fact* of exposure. Her response to the hypothetical told the jury nothing about whether the extent was even important – she gave no indication or principled basis that her causation testimony would be any different if Mr. Juni had performed a different number of brake jobs. The Response Brief cannot save the opinions by citing to evidence that made no difference to the experts.

Second, the Response Brief improperly criticizes the *Amici* brief for not understanding OSHA’s fiber measurement process and the impact of smaller fibers (pp. 12-14). This portion of the argument is complicated and relies on esoteric fiber measurement science and OSHA technical requirements, and thus *Amici* provide some background to assist in the Court’s understanding.

OSHA regulates the safety of the workplace primarily not based on instantaneous or short-term exposures to asbestos, but based on what is called an “eight-hour time weighted average” or TWA.¹⁰ The TWA averages out an

¹⁰ The OSHA regulations have also included a short-term or “ceiling” limit for shorter exposures (usually 15 to 30 minutes) that is much higher than the TWA cited by Plaintiff-Appellant in the brief and that is considered a “good housekeeping” measure rather than a health-based standard. *See id.* § 1910.1001(c)(2).

employee's exposures over a full eight hours of work.¹¹ Thus, if the standard were 1.0 fibers per cc of air over an eight-hour TWA, the worker could work in an environment that over the full course of a day did not exceed that average level of exposure. If the worker was exposed to a setting involving 0.8 fibers/cc for an hour, and then had no further exposure, his average for the day would be 0.8 divided by 8 hours or 0.1 fibers/cc –today's OSHA standard.

Many of the studies and exposure numbers for brake work cited by Plaintiff-Appellant in the Response Brief are actually only short term exposure measurements that neither the studies nor Plaintiff-Appellant converted to OSHA's eight-hour TWA standard.¹² Short term measurements cannot be used to compare

¹¹ See 29 C.F.R. § 1910(c)(1) ("Time-weighted average limit (TWA). The employer shall ensure that no employee is exposed to an airborne concentration of asbestos in excess of 0.1 fiber per cubic centimeter of air as an eight (8)-hour time-weighted average (TWA) as determined by the method prescribed in Appendix A to this section, or by an equivalent method."). See also *id.* at § 1910(d)(1)(a) ("Representative 8-hour TWA employee exposures shall be determined on the basis of one or more samples representing full-shift exposures for each shift for each employee in each job classification in each work area."). The OSHA regulations require the sampling to reflect a full eight-hour shift, not a short, task-specific sample that could be misleading.

¹² On p. 5-6, the Response Brief refers to testimony of exposures from brake work "several hundred times above the OSHA permissible exposure limit." This is the Lorimer study, William v. Lorimer, et al., *Asbestos Exposures of Brake Repair Workers in the United States*, 43 Mt. Sinai J. Indus. Med 207 (1978), which measured *short term* exposures that cannot be compared to the OSHA PEL at all. Dr. Moline admitted on cross that these were only short-term measurements (R. 3527). Similarly, the Response Brief cites to testimony of pre-OSHA measurements being "160 times above the OSHA standard, just for *used brake work alone*" (emphasis in original) without noting that those measurements also were not eight-hour TWA measurements. The Rohl study quoted on p. 10 describes the measurements as "peak exposures," not eight-hour TWAs. The 1980 NIOSH Report discussed on p. 11 as showing "five times the OSHA standard" and "more than twenty-six times the OSHA standard" did not include any such finding – both of these numbers (0.54 f/cc and 2.62 f/cc) were peak samples only, as reflected in the chart on p. 14 (Continued...)

to any OSHA health standard – they fail to reflect the averaging out over the day required for an OSHA measurement. Presenting these numbers as proof of a violation of OSHA is completely contrary to OSHA’s own dictates. Only by cherry-picking the studies can Plaintiff-Appellant identify any eight-hour TWA brake-related exposures that would exceed even today’s standard. In reality, the studies taken together document that the mean or average exposure of vehicle mechanics was in the range of 0.04 fibers/cc 8-hr TWA, less than half the OSHA current standard.¹³

The Response Brief also focuses on its experts’ reliance on counting small fibers by transmission electron microscopy (TEM), which *Amici* criticized as creating a false sense of a high exposure. This Response Brief argument is also misleading and mischaracterizes OSHA’s actual approach. OSHA long ago determined that it would use a marker of fiber exposure rather than require employers to count every individual fiber of any size in the sample.¹⁴ OSHA did

of the report. The NIOSH study documented levels that were only modestly above today’s OSHA TWA of 0.1 f/cc (in the 0.2 to 0.3 range) and well below the 2 f/cc standard in place in 1980.

¹³ Dennis J. Paustenbach, et al., *An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust*, 18 *Applied Occup. & Environ. Hygiene* 786 (2003).

¹⁴ The methodology is based on NIOSH 7400 and reflected in the following link, where OSHA directs: “[C]ount only fibers equal to or longer than 5 micrometers.” See 29 C.F.R. § 1926.1101 App. A located at https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=108

this by using the best available technique in the early 1970s, one relying on light microscopy (phase-contrast microscopy or PCM) to count only fibers longer than five micrometers. PCM techniques cannot distinguish asbestos fibers from non-asbestos fibers such as cellulose, so this process often results in over counting for asbestos. Later, when TEM became widely available, OSHA added a feature whereby employers could use TEM to identify *whether the fibers seen were asbestos or something else*.¹⁵ Under mandatory OSHA procedures, the percent of asbestos fibers identified by TEM is used to *reduce* the fiber count by light microscopy to reflect the actual presence of asbestos as opposed to other fibers that light microscopy cannot distinguish from asbestos. As OSHA plainly states: “Transmission Electron Microscopy (TEM) methods may be used to identify fibers, but may not be used to quantify air concentrations for occupational exposure.”¹⁶ TEM technique has thus *never* been used by OSHA to *count* shorter fibers, and anyone doing so today is not counting per an approved OSHA methodology.

The five micrometer limitation on counting still holds today because it is the best index of whether an exposure is hazardous or not. This is why *Amici* in the opening brief criticized the Plaintiff-Appellant experts and Response Brief for

¹⁵ See OSHA, *Safety and Health Topics, Asbestos, Evaluating and Controlling Exposures*, located at <https://www.osha.gov/SLTC/asbestos/evaluation.html>.

¹⁶ *Id.*

using hyperbolic language about “millions of fibers” in asbestos dust not counted by OSHA. An opinion relying on TEM counting to opine as to the health risk is not grounded on any foundation or scientific evidence, including OSHA procedures.

Even apart from the approved OSHA approach, there is a well-documented health reason for not counting these shorter fibers. Although the subject of some dispute, many reviews of asbestos health risks have concluded that short fibers are *not* a likely cause of asbestos disease.¹⁷ The Court need not resolve this dispute, but the literature undercuts Plaintiff-Appellant’s contention that *Amici* were somehow engaged in scientific misrepresentation by discrediting the experts’ reliance on fibers that OSHA does not count or take into effect.

Apart from Plaintiff-Appellant’s misuse of OSHA requirements, the entire Response Brief argument on exposures hinges on the contention that an exposure exceeding today’s OSHA standard is proof that the exposures would have been causative. But OSHA’s standard is set under the typical government conservative

¹⁷ The United States Environmental Protection Agency commissioned a review of asbestos studies by an independent panel in 2003, and one of that panel’s primary conclusions was that fibers shorter than 5 micrometers were unlikely to be involved in asbestos disease. That review references the extensive literature supporting this consensus conclusion. Eastern Research Group, Inc., *Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk*, at viii (“The Panelists also agreed that the available data suggest that the risk for fibers less than 5 μm is very low and could be zero.”). A similar panel convened by ATSDR reached the same conclusion. Eastern Research Group, Inc., *Report on the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous fibers: The Influence of Fiber Length*, at vi (2003).

approach designed to overprotect the public, in this case employees. Like many other government standards, and as pointed out in *Amici's* opening brief (pp. 27-29), OSHA's asbestos TWA is *not* a causation standard, it is *not* the standard used in a court of law, and a mere exceedance of an OSHA standard cannot form the foundation of expert causation testimony. And, to reiterate – these experts did *not* opine that Mr. Juni's exposures exceeded the OSHA standard and are therefore causative, whereas exposures under the standard are not. They willingly include all such work activity as causative regardless of OSHA compliance. The argument in the brief is, once again, lawyer argument and does not reflect the experts' actual positions.

IV. Scientifically-Based Causation Testimony Does Not Rely on Circular Logic or Assume Causation from Simple Exposure, Even for “Signature” Diseases.

Most of the remaining Response Brief arguments focus on these experts' reliance on the “signature” nature of asbestos and mesothelioma to allow them to avoid conducting a *Parker*-mandated dose assessment. Here again, much of this argument is not consistent with standard scientific practice or logic. The experts are engaged in a form of guesswork based on their belief (not evidence) that essentially every mesothelioma is the result of asbestos exposure and asbestos exposures of any form or quantity are thus always the cause.

Experts in any toxic tort case must demonstrate that the extent of exposure was sufficient to cause the disease at issue. *See Parker*, 7 N.Y.3d at 448 (“It is well-established that an opinion on causation should set forth a plaintiff’s exposure to a toxin, that the toxin is capable of causing the particular illness (general causation) and that plaintiff was exposed to sufficient levels of the toxin to cause the illness (specific causation). As the Response Brief documents, these experts, in contrast, want to avoid that requirement, on the ground that mesothelioma is a “signature” disease to be credited to even the smallest amount of asbestos exposure.¹⁸ There is no scientific basis for this position. These experts have an obligation to explain why any individual plaintiff’s disease was caused by asbestos, as opposed to representing an instance of the hundreds of mesotheliomas every year that are not asbestos related even though there is some modicum of asbestos exposure in that person’s lungs from background or minimal workplace exposures.¹⁹

¹⁸ Plaintiff-Appellant criticizes *Amici*’s use of the term “signature” (p. 15), but her own use of the term is vague – mesothelioma is associated with radiation therapy, erionite, and spontaneous generation, as well as asbestos, and asbestos at high exposures causes other diseases as well. Thus, asbestos is not particularly dissimilar from the role of benzene in causing AML as in *Parker*, the role of different types of mold in causing various pulmonary diseases as in *Cornell*, or the dominant role of smoking in causing lung cancer. *See Cornell v. 360 W. 51st St. Realty, LLC*, 22 N.Y.3d 762, 784 (2014).

¹⁹ As explained in the *Amici* opening brief, many mesotheliomas are “spontaneous,” *i.e.*, created by the human body’s own genetic errors, like all cancers. The Response Brief does not contest this point scientifically but only claims that there was no testimony about spontaneous or non-asbestos induced mesothelioma in the trial. That claim is in error. Ford’s epidemiology (Continued...)

Instead of addressing this critical distinction, the experts rely on purely circular reasoning. The question in the case is *whether* Mr. Juni's exposures were sufficient to cause mesothelioma. The experts answer with the following syllogism – the plaintiff has mesothelioma, and asbestos causes mesothelioma, so any asbestos exposure is the cause of his mesothelioma. This is an entirely circular argument that does not answer the critical *whether* question, as several courts have held. See *Amici* opening brief at 17-18. The Response Brief objects to the *Amici* characterization of this argument as circular but does not refute it. The brief notes that Dr. Moline relied on a hypothetical for her opinion, but that hypothetical did nothing more than demonstrate Mr. Juni's exposure to asbestos. From that sole premise, Dr. Moline concludes, in circular fashion, that his mesothelioma is from his brake exposure. Plaintiff-Appellant's use of a "hypothetical" of case facts

expert Dr. Teta testified at some length about the degree of cases identified in the literature not attributable to any known asbestos exposure. R.3678-79. Plaintiff-Appellant seems to be arguing that her experts can be allowed to testify contrary to well-known science – and thus provide unscientific testimony – if another expert fails to mention the issue in the case. The *Butler* case cited in the *Amici* brief makes the point using the word *idiopathic* rather than *spontaneous*: “Also, Dr. Maddox stated that there are idiopathic causes of mesothelioma. Without quantification of the dose-response and its threshold for asbestos when does one *scientifically* rule out this as a cause and not asbestos?” *Butler v. Union Carbide*, 712 S.E.2d 537, 551 (Ga. App. 2011). The Response Brief criticizes *Amici's* reference to the ubiquitous nature of asbestos in the environment as confusing – the brief is not confusing and does not equivocate. The point is simply that ambient air has over the years contained a fair amount of asbestos from both natural and human uses, and virtually all persons alive in the 1970s or earlier will have a fair amount of asbestos in their lungs from these purely background exposures. Plaintiff experts fail, inexplicably, to consider any of that exposure as causative regardless of the dose.

provides the jury no basis to determine whether she is using a scientific methodology or just assuming every exposure is causative.²⁰

CONCLUSION

In a state where *Parker* (supported by *Cornell* and *Sean R.*) establishes the standard governing toxic tort causation testimony, it is not even reasonably debatable whether these experts can testify without performing a scientifically competent dose assessment and proving actual causation. Assumptions, circular “risk” testimony, and anecdotal evidence of asbestos exposure do not suffice. The Response Brief’s attacks on the *Amici* are unwarranted, and the *Juni* trial court opinion should be affirmed.

²⁰ The Response Brief attempts to pull points from the Eaton article to support their experts’ position, but the Eaton article in no form or fashion could be read to support *any exposure* or *cumulative exposure* testimony. The entire article emphasizes the importance of dose. Several courts have cited to Eaton to *reject any exposure* or *cumulative exposure* testimony. 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, (E.D. Ky. 2012); *Henrickson v. ConocoPhillips Co.*, 605 F.Supp.2d 1142, 1156 (E.D. Wash. 2009). No court to *Amici’s* knowledge has ever cited to Eaton to approve such testimony.

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Type. A proportionally spaced typeface was used, as follows:

Name of typeface: Times New Roman
Point size: 14
Line spacing: Double

Word Count. The total number of words in this brief, inclusive of point headings and footnotes and exclusive of pages containing the table of contents, table of citations, proof of service, and this Statement is 4,809.

Dated: New York, New York
July 15, 2016

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