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Court of Appeals
of the
State of New York

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

(Caption continued on inside cover)

BRIEF OF DEFENDANT-RESPONDENT FORD MOTOR COMPANY IN RESPONSE TO THE BRIEF OF *AMICUS CURIAE* MILITARY-VETERANS ADVOCACY, INC. IN SUPPORT OF PLAINTIFF-APPELLANT

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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, FMC CORPORATION, on behalf of its former CHICAGO PUMP & NORTHERN PUMP BUSINESSES,

Defendants,

—and—

FORD MOTOR COMPANY,

Defendant-Respondent,

—and—

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

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

Defendants.

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INTRODUCTION

Amicus Curiae Military-Veterans Advocacy, Inc. ("Veterans Advocacy") misperceives the key issue before this Court and it urges a flawed reading of the now well-settled causation standard for toxic tort cases set out in *Parker*.

Like the Junis in their opening brief, Veterans Advocacy frames the issue on appeal as whether a jury, on the evidence before it, could have rationally concluded that Mr. Juni's exposure to gaskets and friction products sold or distributed by Ford caused his mesothelioma. But the issue is not what the jury could have concluded from the evidence presented, it is whether, under *Parker* and its progeny, (a) the Junis' expert witnesses had an adequate foundation for their causation opinions, and (b) whether the Junis met the legal standard for causation in a toxic court case. *See Parker v. Mobil Oil Corp.*, 7 N.Y.3d 434, 448-50 (2006). Absent the causation testimony of Moline and Markowitz, there was no basis, rational or otherwise, for the jury to render a verdict against Ford.

Veterans Advocacy argues that the *Parker* causation standard is "versatile" and that its application depends upon "the particular toxin" and "the contemporary development of the science." (Veteran Advocacy Br. at 2, 13-14.) While *Parker* is certainly flexible in terms of the evidence that can be used to establish causation, the clear standard it articulates for proving causation in toxic tort cases does not vary based upon the toxin at issue in a particular case. Under *Parker*, it was

incumbent on Markowitz and Moline to present evidence establishing (1) that chrysotile asbestos, as incorporated into products sold or distributed by Ford Motor Company, is capable of causing mesothelioma, and (2) that Mr. Juni was exposed to sufficient levels of chrysotile asbestos, as incorporated into products sold or distributed by Ford, to cause his mesothelioma. *See Parker*, 7 N.Y.3d at 448-49. As to the latter, *Parker* requires some "scientific expression" of the plaintiff's exposure. *Id.* at 448.

As set forth in prior briefing and below, Markowitz and Moline failed to present testimony or evidence sufficient to comply with the *Parker* causation standard. Between them, they could not demonstrate the exposure level at which asbestos in Ford's products presented any risk of mesothelioma, nor could they demonstrate that Mr. Juni's exposure to products sold or distributed by Ford exposed him to any increased risk of mesothelioma, much less that it caused it.

Veterans Advocacy argues that affirmation of the decisions below will all but put a lock on the courthouse door for all individuals that are currently ill, or may later become ill, from asbestos disease. (Veterans Advocacy Br. at 2.) Ford never sought, and neither the trial court nor the appellate division rendered, such sweeping relief. This case turns narrowly on its unique facts. Under the clear and straightforward standard articulated in *Parker*, neither Markowitz nor Moline

posited a proper foundation for their causation opinions, and thus the trial court and the appellate division properly rejected their testimony.

ARGUMENT

I. Markowitz's and Moline's Concessions are not "Minor Discrepancies."

Veterans Advocacy describes the various concessions made by Markowitz and Moline as mere "minor discrepancies" that go to the weight of their testimony, not its admissibility. (Veterans Advocacy Br. at 5.) But the concessions were not minor. Granting all inferences in the Junis' favor, Markowitz and Moline failed to establish either general or specific causation and their conclusory causation opinions lacked any proper, scientific basis.

A. Markowitz and Moline failed to identify the exposure level at which chrysotile asbestos residue in brake and clutch dust causes mesothelioma or that Mr. Juni's exposure exceeded any such threshold.

Parker requires a plaintiff in a toxic tort case to establish that she "was exposed to sufficient levels of the toxin to cause the illness." *Parker*, 7 N.Y.3d at 448. As explained further by this Court,

> "[i]t is therefore not enough for a plaintiff to show that a certain … agent sometimes causes the kind of harm that he or she is complaining of. 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."

Cornell v. 360 W. 51st St. Realty, 22 N.Y.3d 762, 784 (2014) (quoting *Wright v. Willamette Indus.*, 91 F.3d 1105, 1107 (8th Cir. 1996) (emphasis added).

In this case, Markowitz and Moline each failed to identify an exposure level at which either chrysotile asbestos generally, or friction product residue specifically, is capable of causing mesothelioma. It follows that neither had any basis to testify that Mr. Juni's exposure to asbestos from Ford products was sufficient to cause his mesothelioma.

Markowitz explained that we all may be breathing some level of asbestos, and that studies show individuals with millions of asbestos fibers in their lungs from the ambient air with no symptoms of disease. (A182-83.) Moline agreed that there is asbestos in the air that we breathe. (A1189.) She testified that the lung fiber burden from background exposures is in the "millions of fibers." (A1190.) Neither witness identified any risk of mesothelioma from background exposures, acknowledging that there is some threshold below which chrysotile asbestos does not cause mesothelioma. *See Bostic v. Georgia Pacific*, 439 S.W.3d 332, 339 (Texas 2014) ("If any exposure at all were sufficient to cause mesothelioma, everyone would suffer from it or at least be at risk of contracting the disease.").

Despite their effective concession that low level exposures to asbestos are not known to cause mesothelioma, neither Markowitz nor Moline identified the

threshold level at which some risk of disease exists. Acknowledging his failure to do so, Markowitz testified that the threshold exposure level that creates a risk of mesothelioma is not knowable. (A101 ("No level has been identified that separates out increased risk from no risk.").) But if the threshold is unknown, then it is logically impossible to demonstrate that a particular exposure exceeded the threshold. Put another way, "[j]ust because we cannot rule anything out does not mean we can rule everything in." *Bostic*, 439 S.W.3d at 341 (quoting *Smith v. Ford Motor Co.*, 2013 WL 214378, No. 2:08-cv-630, at *2-3 (D. Utah Jan. 18, 2013); *see also Yates v. Ford*, 113 F. Supp.3d 841, 847 (E.D.N.C. 2015) ("the failure to identify a threshold level of exposure is different from showing that a given exposure is hazardous") (citation omitted).

Absent any evidence from Markowitz establishing the threshold level at which chrysotile asbestos generally, or as incorporated into the Ford products at issue specifically, causes mesothelioma, Moline had no basis to testify that Mr. Juni's exposure to asbestos from Ford products even increased his risk of mesothelioma, much less caused it. *See Yates*, 113 F. Supp.3d at 857-58 ("[T]he mere proposition that [some] studies support that chrysotile asbestos may cause mesothelioma does not provide any quantitative or qualitative measure to establish any particular level at which chrysotile asbestos becomes hazardous.").

In the absence of a known threshold at which exposure to asbestos from gaskets and friction products might increase the risk of mesothelioma, the Junis could have offered evidence that Mr. Juni's exposure was at some higher level known to cause mesothelioma, but neither Markowitz nor Moline did that either. While Markowitz pointed to a variety of NIOSH studies conducted in various garage settings in the 1970s and 1980s that measured asbestos exposures among workers using friction products (*see* A245-248), he offered no testimony that those workers had an increased risk of mesothelioma at those exposure levels. *See Yates*, 113 F. Supp.3d at 857-58 ("[T]he mere proposition that these studies support that chrysotile asbestos may cause mesothelioma does not provide any quantitative or qualitative measure to establish any particular level at which chrysotile asbestos becomes hazardous."). Moreover, even if the referenced garage studies were probative as to the threshold exposure issue, neither Markowitz nor Moline made any meaningful comparison of Mr. Juni's exposure to that of the workers involved in the NIOSH studies, a comparison that would be necessary to draw any causation inferences, especially in light of Markowitz's agreement that exposure concentrations are affected by the work practices utilized and the existing environmental conditions and controls at each facility. (A357.)

Markowitz also relied upon studies of factory workers engaged in the *manufacture* of friction products. Unlike in an automobile garage, those workers

were exposed to raw asbestos used in the manufacturing process. (A190-91.) Markowitz agreed that the McDonald study, as an example, involved an exposure that was "very, very different" than Mr. Juni's exposure. (A191.) These studies shed no light on the exposure threshold for friction products and gaskets necessary to cause mesothelioma, much less that Mr. Juni's exposure reached such a level.

B. The toxin at issue is chrysotile asbestos as incorporated into Ford friction products.

Veterans Advocacy is critical of Ford for purportedly "reformulating" the case to be about chrysotile asbestos as incorporated into friction products rather than asbestos generally. (Veterans Advocacy Br. at 10.) Yet that is squarely what the case is about, and analyzing the evidence in that fashion is squarely consistent with the approach in *Parker* of considering benzene in gasoline rather than benzene alone.

Importantly, and not addressed by Veterans Advocacy, Markowitz testified to significant differences among the various forms of asbestos, including their chemical makeup, physical properties, biopersistence, and potency. (A179, 192-93, 240-41.) He agreed that these differences can all be "important properties that relate to a fiber's biological activity." (A273.) He agreed that elevated temperatures (which occur in braking and in the clutch), convert chrysotile

asbestos to non-toxic forsterite.[1] (A274-76.) He agreed that studies have found that the "vast majority" of asbestos in brake wear debris has been converted to non-toxic substances, and that most studies find less than one percent asbestos in the debris. (A250.) Not surprisingly, Markowitz agreed that this case is not about just chrysotile asbestos. (A285.)

C. Moline conceded she had no basis to conclude that Mr. Juni's exposure to Ford products caused his mesothelioma.

Moline acknowledged, but took no account of, the many differences among the fiber types identified by Markowitz. Nor did she account for the difference between raw chrysotile fibers and the fiber residue present in brake and clutch dust debris. When asked, she explained that she could not differentiate between the different fiber types because "there were no measurements of what Mr. Juni was exposed to." (A1090.) She likewise did not account for the transformation to the fibers that results from high temperatures. When asked whether the fibers Mr. Juni was exposed to were still biologically active, she responded: "No one knows." (A1199 ("No one did any measurements of the fibers, so no one knows.").) Moline conceded that she had no basis to say whether the fibers Mr. Juni was exposed to had the potential to cause mesothelioma. (*Id.*)

[1] Finley, Ford's expert, explained that the heat and pressure generated in the operation of the brakes and the clutch would modify the embedded asbestos fibers even prior to their transformation to forsterite. (A1696, 1707-08, 1716.)

This concession was no mere discrepancy. Under *Parker*, the Junis had to establish that Mr. Juni's exposure to asbestos from products sold or distributed by Ford was sufficient to cause his mesothelioma. In the absence of any knowledge of whether the fibers Mr. Juni was exposed to were still biologically active, i.e., that they could cause mesothelioma, it was logically and scientifically impossible for Moline to conclude that they could. Her testimony on this point alone highlights the fundamental and fatal failure of any causation proof at trial.

D. Markowitz admitted that epidemiological studies show no increased risk of mesothelioma from exposure to friction products and failed to identify any relevant scientific support for his contrary opinion.

On direct examination, Markowitz testified without caveat that the numerous epidemiological studies that have addressed the risk of mesothelioma among garage mechanics, including brake workers, "do not show much evidence in support of a relationship between mesothelioma and exposure to friction products." (A114.) Markowitz identified twenty-two such studies, and he agreed that the first twenty-one show "no excess risk" of mesothelioma for friction product workers. (A310-15.) He agreed further that he was unaware of any epidemiological cohort study that demonstrates an increased risk of mesothelioma from exposure to automobile brakes, clutches, or gaskets.[2] (A172-173.)

[2] Markowitz also referenced, but did not appear to reply upon, the Roeloff's study, which the Junis say shows an increased risk of mesothelioma for garage

Veterans Advocacy attacks the epidemiological studies that show no increased risk of mesothelioma as "specious," and it accuses the authors of "manipulating the meta-data to produce skewed results." (Veterans Advocacy Br. at 11-12.) There is no basis for such an attack, whether in the record or outside it.

Markowitz identified challenges in conducting epidemiological studies to determine the risk of mesothelioma in garage mechanics. (A116-119.) He testified, for example, that it is "hard" to get data and that the characterization of the workers is not well-defined. (A116, 117.) He did not, however, attack the existing studies as invalid, and, in fact, conceded that at least some of the studies are on point. (A116 ("[W]hen we get to the brake literature or the friction product literature, the epidemiology, we don't actually find that kind of study or, if we have, *it's been very few of them* ...") (emphasis added).)

For her part, it is not clear that Moline even considered all of the studies. She testified on direct that "[t]here are a number of studies of brake workers or actually, in general, it's garage workers or auto mechanics that don't show an

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mechanics. (A175.) As set forth in more detail in Ford's Brief of Defendant-Respondent Ford Motor Company, the authors of the Roelofs' study admitted in the study report that they could not determine whether the mesothelioma cases identified in their study resulted from exposure to asbestos during the reported occupation (e.g., garage mechanic) or during a prior or other occupation not reported as their usual occupation. Markowitz called Roelofs "the weakest kind of study" based on its PIR methodology. (A316-317.)

elevated risk, but I have looked *at many of these studies* and have found that there are discrepancies in the studies." (A1104-1105 (emphasis added).)

Veterans Advocacy points out, accurately, that epidemiological studies have not been conducted for all products that contain asbestos. (Veterans Advocacy Br. at 11-12.) But that is neither here nor there. There are such studies for garage mechanics, including brake workers, and they show almost conclusively that Mr. Juni's exposure to Ford products was not the cause of his mesothelioma. Those same studies, and others, show that a more likely cause of the mesothelioma was Mr. Juni's exposure to asbestos at the Hillburn power plant where he was admittedly exposed to a more potent form of asbestos.

In its consideration of the epidemiological studies, the trial court was not weighing the evidence. To the contrary, it was determining, in light of the studies, whether the testimony from Markowitz and Moline was sufficient to establish causation. As explained by the United States Court of Appeals for the Tenth Circuit:

> [E]pidemiology is the best evidence of general causation in a toxic tort case. … While the presence of epidemiology does not necessarily end the inquiry, where epidemiology is available, it cannot be ignored. As the best evidence of general causation, it must be addressed.

Norris v. Baxter Healthcare, Corp., 397 F.3d 878, 882 (10th Cir. 2005); *see also Cano v. Everest Minerals Corp.*, 362 F. Supp.2d 814, 820 (W.D. Texas 2005)

("[i]n toxic tort cases, 'the most useful and conclusive type of evidence . . . is epidemiological studies.'") (citation omitted). Moreover, the testimony regarding the epidemiological studies that were unfavorable to the Junis' case was elicited on direct examination by the Junis.

Markowitz pointed out general concerns he had with *some* of the epidemiology studies, but he did not contend that any single study was invalid, much less the studies as a group. He acknowledged that at least some of the studies assess the risk of mesothelioma for brake workers, and he volunteered that the studies "do not show much evidence in support of a relationship between mesothelioma and exposure to friction products." (A114.) With the "best evidence" against him, Markowitz simply had no basis for his general causation opinion.

E. Markowitz and Moline relied upon a disfavored and unscientific "every exposure" theory that was at odds with their own explanations of the risk of disease arising from asbestos exposure.

In the absence of any evidence of Mr. Juni's actual exposure to asbestos from Ford products, or even any rough estimate of it, both Markowitz and Moline resorted to an "every exposure" or "cumulative exposure" theory that numerous courts have found to be unscientific.

According to Markowitz, "if and when they develop mesothelioma … then we look back and then say, yeah, that exposure viewed as a whole contributed,

caused their malignant mesothelioma … and *every part* of that exposure that they had acted as a contributed factor." (A128 (emphasis added).) Although Markowitz testified that he only considers exposures that are "substantial," he conceded that in his vernacular, "there's no magic number above which there's a substantial factor and below which there's not." (A228-229, 236.)

Moline was even more pointed in her reliance on an every exposure theory. She explained that Mr. Juni's mesothelioma was caused by "[t]he cumulative exposure he had. So his cumulative exposure to asbestos caused his mesothelioma. It's not possible to separate out one or exclude one. It's the cumulative exposure." (A1088-89.) She concluded, without any consideration of relative dose or the magnitude of any exposure that "[a]ll of his occupational exposures were substantial factors." (A1090.)

The "every exposure" or "cumulative exposure" theory upon which Markowitz and Moline rely has been widely discredited. *See, e.g., Krik v. Exxon Mobil Corp.*, 870 F.3d 669 (7th Cir. 2017). As set forth in *Krik*, the theory ignores fundamental principles of toxicology and improperly shifts the burden of proof to a defendant to demonstrate that exposure to its product was not a cause of disease. *Id.* at 677. Not only is the "every exposure" theory discredited, it is also flatly inconsistent with Markowitz's and Moline's own explanation of the medical science behind causation.

As Markowitz explained, an individual's cumulative exposure to a toxin is the sum of all the individual exposures the individual may have had to the toxin. (A127-128.) He testified that as the cumulative exposure increases, the individual has an increased *risk* of disease. (A127-128, 199-201.) But a showing of increased *risk*, without more, is insufficient to establish *causation*. *Cornell*, 22 N.Y.3d at 783 (reports that "speak in terms of 'risk' and 'linkage' and 'association' do not establish causation).

Markowitz effectively acknowledged this very point. He explained that the concept of cumulative dose is a "probabilistic" concept. (A201.) Put another way, it is akin to walking across a busy street - the more times you walk across the street, the more likely you will be hit by a car. (*Id.*) But as Markowitz's own explanation makes clear, increased risk does not equal causation. A pedestrian who repeatedly crosses a busy street has a greater risk of harm than one who crosses just once, but if either are struck by a car on one of the crossings, it is just the one car that has caused the harm. There is no basis in the law to hold the drivers of the other cars liable for the harm.

The same holds true for asbestos and mesothelioma. Markowitz could not point to any study showing that a DNA defect leading to mesothelioma is caused by the total amount of asbestos in one's body as opposed to a single exposure. (A200.) He testified that the greater the exposure, the greater the risk, but he

offered no scientific basis for the notion that every exposure an individual has contributes to cause mesothelioma.

For her part, Moline testified that the amount, duration, and frequency of an exposure are "critical" in assessing whether there is sufficient exposure in order for someone to have an increased risk of disease from the exposure. (A1150-1151.) But Moline made no assessment at all of Mr. Juni's exposure to products sold or distributed by Ford or, for that matter, any other manufacturer. She did not consider amount. She did not consider duration. She did not consider frequency. Directly contrary to her own testimony (and contrary to what is required by *Parker* and its progeny), she declared that every exposure Mr. Juni had caused his mesothelioma. (A1190, 1101-1102.)

Markowitz and Moline took pains to avoid using the term "each and every exposure," but substantively, there is no distinction between their testimony and the discredited "each and every exposure" theory. For the many reasons set forth in the well-reasoned decisions condemning the theory, the courts below properly rejected this testimony. *See, e.g.*, *Betz v. Pneumo Abex LLC*, 44 A.3d 27 (Pa. 2012); *Butler v. Union Carbide Corp.*, 712 S.E.2d 537 (Ga. App. 2011); *Ford Motor Co. v. Boomer*, 736 S.E.2d 724 (Va. 2013); *Moeller v. Garlock Sealing Tech., LLC*, 660 F.3d 950 (6th Cir. 2011).

II. "Visible Dust" is not a "Scientific Expression" of Exposure.

Ignoring *Parker* and its progeny, Markowitz and Moline made no effort to describe, qualitatively or quantitatively, Mr. Juni's exposure to asbestos from products sold or distributed by Ford. Instead, they used what Markowitz called a "shortcut," and what Moline called a "surrogate." (A130 (presence of visible dust is "really a shortcut" for the proposition that material became airborne); A1095).) Put most simply, a "shortcut" is not a scientific expression of exposure.

Moline testified that visible dust "is a surrogate for telling us that [Juni] was exposed to asbestos dust at levels that are above the level that we know asbestos is capable of causing disease at." (A1095.) Putting aside her fatal concession, described above, that she does not know whether the dust from Ford products was capable of causing mesothelioma, her testimony does not provide any scientific, qualitative or quantitative assessment of Mr. Juni's exposure, nor could it.

"Visible dust," as a descriptor, provides no meaningful information regarding the total amount of dust at issue, the content of the dust, the location of the dust relative to the viewer, or the amount of dust, if any, ingested by the viewer. Indeed, there is no indication in the phrase itself that the visible dust is airborne.

"Visible dust" is not a scientific term and it is marginally descriptive at best.[3] While both Markowitz and Moline mention studies that have measured the asbestos content of varying types of dust (with different results), neither points to any scientific study or literature adopting "visible dust" as a scientific term. The notion of "visible dust" is highly and unreliably imprecise. It could range from a "smidgeon" of dust detected by a white glove cleanliness inspector at a fancy hotel or restaurant to the clouds of talc dust described in *Westberry* that were so thick that the workers left footprints in the dust on the floor. *See Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 264 (4th Cir. 1999).

Many of the cases Veterans Advocacy points to that purportedly support the use of "visible dust" as a qualitative quantification of exposure actually have other descriptive terms that provide at least some basis for understanding the amount of dust at issue. In *Westberry*, for example, there was testimony that the plaintiff was exposed to very high levels of talc "so thick that one could see footprints in it on the floor." 178 F.3d at 264 ("He worked in clouds of talc and it covered him and

[3] In *Sean R. v. BMW of N. Am.*, 26 N.Y.3d 801, 809-12 (2016), this Court references the potential use of "odor" thresholds as a means to estimate exposures to toxic substances. An "odor threshold" is the lowest concentration of a specific compound that can be perceived by the human sense of smell. Odor thresholds are the subject of substantial scientific inquiry. There is no such science built up around visual thresholds. Moreover, testimony from a plaintiff that he or she smelled something necessarily means that whatever the plaintiff has ingested the substance into their nasal cavity at a minimum threshold level. No scientific analogy can be drawn between odor thresholds and visible dust.

his clothes."). Indeed, later cases citing *Westberry* point out its unique facts when reiterating that a toxic tort plaintiff "must demonstrate the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure." *Zellers v. NexTech*, 533 Fed. Appx. 192, 196, 196n. 6 (4th Cir. 2013) (noting that quantification was not required in *Westberry* "because the record in that case clearly established that the plaintiff had been substantially exposed to the allegedly harmful substance in such a way that specific evidence was not necessary.")

While Mr. Juni provided a description akin to the one provided in *Westberry* for his exposure to asbestos at the Hillburn power plant, there was no such evidence relating to his work at the Nyack or Spring Valley garages. Mr. Juni testified that he could see dust, but there was no description of the volume of dust or, to the extent it was airborne, its density. There was no reference to "clouds" of dust. There was no testimony that the dust got on Mr. Juni's clothing. Putting aside that the dust Mr. Juni saw may have been comprised of road dirt and other items unrelated to any friction product, and putting aside the remarkable concession by Moline that she did not know if any asbestos fibers in the dust were biologically active, there was simply no evidence of any substantial asbestos exposure and there was no qualitative or quantitative measurement or estimation of the exposure.

As Justice Kahn noted in her concurring opinion below, *Parker* rejected exposure evidence that is general, subjective, and conclusory and lacking in specific relation to the plaintiff's alleged exposures. (A16-17 (noting that, in *Parker*, the Court of Appeals "acknowledg[ed] the plaintiff's exposure to the carcinogenic substance[]" but "rejected the plaintiff's expert evidence as too general, subjective and conclusory, and lacking in specific relation to the plaintiff's exposures, to satisfy its announced standard") (internal citations and quotations marks omitted). She explained that sanctioning the use of "visible dust" as a scientific expression of Mr. Juni's exposure would "carve such a gaping hole in the *Parker* standard of proof on causation [so as to] eviscerate[e] its fundamental evidentiary requirements" and would effectively overrule *Cornell.* (A17.) Other courts have reached the same conclusion. *See, e.g.*, *Bostic*, 439 S.W.3d at 339 (explaining that the adoption of "a less demanding standard for mesothelioma cases" would result in "absolute liability against any company whose asbestos-containing product crossed paths with the plaintiff throughout his entire lifetime").

III. The Junis' Hypothetical Question did not Render Moline's Testimony Admissible.

Veterans Advocacy suggests that all is cured by the Junis use of a hypothetical question to elicit Moline's specific causation opinion. (Veterans Advocacy Br. at 4, 17-23.) But not only was the hypothetical question inconsistent with the factual evidence adduced at trial (and an objection was timely made

(A1093)), it did not provide the requisite foundation to allow a proper causation opinion compliant with the *Parker* standard.

The hypothetical question posited that Mr. Juni assisted with brake and clutch work on a regular basis from 1964 to 1988, that during that time he engaged in a variety of repair activities, some of which involved friction products sold or distributed by Ford,[4] that the activities caused visible asbestos dust, and "that all of this dust would be released into Mr. Juni's breathing zone." (A1091-93.) As set forth in detail in Ford's Brief of Defendant-Respondent Ford Motor Company, granting all inferences in the Junis favor, the facts posited in the question are not fairly supported by the evidence at trial.

There was no evidence, for example, that Mr. Juni performed or assisted with the performance of brake and clutch jobs involving products sold or distributed by Ford on a regular basis from 1964 to 1968. As Mr. Juni explained, during most of this time period he worked the evening shift, while most of the brake work was performed during the day. (A425-26, 557.) In many, if not most, instances, the work performed did not involve component parts sold or distributed by Ford, and there was no effort to quantify or even estimate how much of the work, whether performed by Mr. Juni or others, involved component parts sold or

[4] The hypothetical question makes no reference to work involving gaskets. (A1091-92.) Because the hypothetical question forms the basis of Moline's causation opinion, Mr. Juni's alleged exposure to asbestos from gaskets is not relevant to the issues raised by the Junis on appeal.

distributed by Ford. (A409, 429, 432-35, 437-38, 451.) Perhaps most significantly, there was also no testimony supporting the proposition that all of the dust generated during the brake and clutch jobs was released into Mr. Juni's breathing zone. Mr. Juni acknowledged in bare bones fashion that he "breathed dust," but he never the described the volume or density of the dust that was generated by the garage activity nor described in any way how much he ingested. (A611, 657.5-657.6). He did *not* describe clouds of dust or mention dust getting on his clothing.

But even if the evidence had supported the hypothetical question posited to Moline, the information supplied by the hypothetical question did not supply the evidence necessary for Moline to render a causation opinion under *Parker*. Like the expert witness in *Parker* who testified that the plaintiff "'had far more exposure to benzene than did the refinery workers in the epidemiology studies,'" the "facts" posited in the hypothetical question were, at best, "general, subjective, and conclusory." *See Parker*, 7 N.Y.3d at 449. The hypothetical did not posit any quantitative or qualitative "scientific expression" of Mr. Juni's purported exposure to asbestos from Ford products, and thus could not serve as the basis for a viable causation opinion.

IV. Public Policy Favors Affirming The Result Below

Veterans Advocacy, like the Junis, relies heavily on a flawed public policy argument that all injured plaintiffs are entitled to have their cases resolved by a jury even if their causation evidence lacks some minimum level of reliability. While it is certainly good public policy to provide remedies to parties harmed by the conduct of others, there is also a strong public policy against holding persons liable for harms they did not cause. Such basic concepts as due process and burden of proof arise out of that policy.

In making its argument, Veterans Advocacy focuses on a single phrase from *Parker*, i.e., that it would be "inappropriate to set an insurmountable standard that would effectively deprive toxic tort plaintiffs of their day in court." (Veterans Advocacy Br. at 27 (quoting *Parker*, 7 N.Y.3d at 447-48). But Veterans Advocacy omits the immediate prior sentence in *Parker* in which this Court pointed out that, "[a]s with any other type of expert evidence, we recognize the danger in allowing unreliable or speculative information (or 'junk science') to go before the jury with the weight of an impressively credentialed expert behind it." *Parker*, 7 N.Y.3d at 447.

Parker balanced the interests of both plaintiffs and defendants in toxic tort cases where, indisputably, it is often difficult to marshal evidence on causation. Taking both interests into account, the Court set a well-reasoned standard that

requires plaintiffs to establish causation using methods generally accepted in the scientific community. It necessarily follows that if a plaintiff fails to meet the causation standard set by *Parker*, it would be against public policy to nevertheless impose liability on a defendant charged with causing harm to the plaintiff. And, of course, the result in *Parker* was a determination that Dr. Landrigan's testimony did not measure up. His testimony did not rely upon or provide a "scientific expression" of the plaintiff's exposure levels, and was thus properly excluded. *Parker*, 7 N.Y.3d at 449.

As set forth in *Cornell*, *Parker* "by no means" dispensed with a plaintiff's burden to establish sufficient exposure to a substance to cause the claimed adverse health effect: *Cornell*, 22 N.Y.3d at 784. As the Court noted,

> "[A]ctions in tort for damages focus on the question of whether to transfer money from one individual to another, and under common-law principles … that transfer can take place only if one individual proves, among other things, that it is more likely than not that another individual has caused him or her harm.

Id. (quoting *Wright*, 91 F.3d at 1107).

The Junis failed to establish either general or specific causation. Their experts failed to meet the now well-settled standard of proof established by *Parker* and lacked any proper foundation for their opinions. For all of these reasons, public policy would be best served by affirming the decisions below.

CONCLUSION

For the foregoing reasons, Ford respectfully requests that this Court affirm the decision of the Appellate Division affirming the trial court's entry of judgment in favor of Ford.

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I hereby certify pursuant to 22 NYCRR § 500.13(c) that the foregoing brief was prepared on a computer. A proportionally spaced typeface was used, as follows:

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September 14, 2018

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