To Be Argued By:
Tennille J. Checkovich
Time Requested: 30 Minutes

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Court of Appeals

STATE OF NEW YORK

MARY JUNI, as Administratrix for the Estate of
ARTHUR H. JUNI, JR. and MARY JUNI, Individually,

Plaintiffs-Appellants,

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 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

(Caption Continued on the Reverse and Following Page)

BRIEF FOR DEFENDANT-RESPONDENT

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Date Completed: December 6, 2017

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,

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.

TABLE OF CONTENTS

TABLE OF AUTHORITIES

Page(s)

Cases

Other Authorities

PRELIMINARY STATEMENT

This Court has articulated the operative standards governing the admissibility and sufficiency of causation testimony required of expert witnesses in toxic tort cases on no less than three separate occasions. *See, e.g.*, *Sean R. v. BMW of N. Am., LLC*, 26 N.Y.3d 801 (2016); *Cornell v. 360 W. 51st St. Realty, LLC*, 22 N.Y.3d 762 (2014); *Parker v. Mobil Oil Corp.*, 7 N.Y.3d 434 (2006). Such testimony must establish that: (1) the toxin to which a plaintiff alleges exposure is capable of causing the plaintiff's particular illness (general causation), and (2) the plaintiff was exposed to sufficient levels of that toxin to cause the illness (specific causation). *See, e.g.*, *Parker*, 7 N.Y.3d at 449. The Junis acknowledge that *Parker* and its progeny govern here, but they contend that their experts' causation testimony satisfies *Parker*'s requirements. They are wrong.

With respect to general causation, the overwhelming majority (21 of 22) of the applicable epidemiological studies show that vehicle mechanics do not face an increased risk of contracting mesothelioma. Rather than point to any contrary evidence (which, as the plaintiffs in this case, they were required to do), the Junis spend the vast majority of their brief nit-picking this science. Even if their criticisms were correct (and they are not), that would not warrant reversal—parsing of scientific evidence by counsel on appeal is no substitute for scientific expert testimony at trial. To the extent the Junis actually attempt to identify evidence

supporting their claims as to general causation, they rely on an admittedly flawed study that is incapable of illuminating the causation inquiry and they contort the testimony of Ford's experts. This approach plainly fails to satisfy the requirements set forth in *Parker* and its progeny with respect to the foundation and sufficiency of expert general causation testimony, as the Supreme Court properly held.

With respect to specific causation, this Court held in *Parker* that expert testimony must provide some scientific expression of a plaintiff's exposure to a particular defendant's product and establish that such exposure was sufficient to cause the plaintiff's illness. *Parker*, 7 N.Y.3d at 448. The Junis' specific causation expert ignored this clear requirement, however, opting instead to rely on two unscientific "shortcuts" to specific causation. Neither shortcut satisfies New York law. First, testimony concerning the presence of "visible dust" from friction products in Mr. Juni's workplace does not establish that he was exposed to sufficient quantities of chrysotile asbestos to cause mesothelioma because the contents of that dust were unknown. Second, testimony that Mr. Juni's mesothelioma was the result of his "cumulative exposure" to asbestos fails to satisfy *Parker* because it ignores the frequency and duration of his exposure and glosses over the significant differences in toxicity among the various asbestos fiber types to which Mr. Juni was exposed. Thus, the Junis' expert testimony fails to

satisfy New York law regarding the foundation and sufficiency of expert testimony on specific causation.

Sound public policy supports application of *Parker* and its progeny in this case. New York's prevailing standards concerning the foundation and sufficiency of expert causation testimony are critical to ensuring that only valid scientific evidence is presented at trial. Additionally, the causation standards ensure that toxic tort defendants are not held liable for potentially *de minimis* contributions, if any, to a particular plaintiff's injury, which is particularly important in the context of asbestos cases against the manufacturers of friction products given the comparatively low levels of exposure attributable to those products and the lesser toxicity of the chrysotile asbestos embedded in them.

In sum, the Junis have failed to provide any justification for disturbing the well-reasoned opinions of the courts below. Therefore, this Court should affirm the Appellate Division's decision affirming the Supreme Court's entry of judgment notwithstanding the verdict in favor of Ford.

QUESTIONS PRESENTED

1. Is Ford entitled to judgment notwithstanding the verdict on the ground that the Junis failed to prove general causation?

2. Is Ford entitled to judgment notwithstanding the verdict on the ground that the Junis failed to prove specific causation?

3. Was the trial court within its discretion to hold that the Junis' causation experts lacked foundation for their opinions, which left the Junis without sufficient proof of either general or specific causation?

STATEMENT OF FACTS

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

The Junis characterize Mr. Juni's exposure to asbestos in Ford friction products as "substantial" and his exposure to asbestos from other sources as "slight," "fleeting," and "short-lived." (Appellant's Br. 2, 17.) The record demonstrates the opposite is true. While Mr. Juni recalled servicing Ford vehicles throughout his career, he did not recall how frequently, if ever, he encountered asbestos-containing products sold or distributed by Ford. (A407-10, 427, 433-37, 563, 568-69, 580.) By comparison, Mr. Juni was able to describe extensive exposure to asbestos-containing products from other sources in great detail. (A392, 401-02.)

Mr. Juni was first exposed to asbestos when working during college as a driver at Orange & Rockland Utilities in 1961, 1962, and 1963. (A390.) Every morning, after reporting to work at the Hillburn Generating Station, he walked through the powerhouse to the machine shop to pick up his truck. (A391-93.) In the dusty environment of the powerhouse, asbestos was "all over the place." (A392-93.) Mr. Juni saw pieces of asbestos-containing pipe covering lying on the floor and testified that he breathed the dust emanating from that material as he

walked through the plant. (A393-96.) Mr. Juni walked through the power plant again at the end of every day. (A396.)

From 1963 to 1964, Mr. Juni was a full-time courier for Orange & Rockland. (A399-400) During that time, he delivered packages and mail to various power plants operated by the utility, including the Hillburn power plant. (A398-400.) Mr. Juni testified that he was exposed to and breathed asbestos at the Hillburn power plant and that asbestos was "all over the place" at that location. (A401-02.)

From 1964 to 1966, Mr. Juni worked as a third- and then second-class mechanic for Orange & Rockland at the Nyack garage, where he was responsible for pumping gas, changing oil, and changing tires. (A405-07.) In their brief, the Junis assert, without citation to the record, that Mr. Juni was exposed to asbestos on a "daily" basis while working at the Nyack garage. (Appellants' Br. 12-13.) The record is otherwise. Mr. Juni testified that he "[did] not know" if he was exposed to asbestos while working at the Nyack garage. (A407.) Indeed, none of his job responsibilities involved working with asbestos-containing products. (A405-07.) Mr. Juni did not personally perform any brake jobs at the Nyack garage and does not know how many brake jobs he may have observed because he generally "minded [his] own business." (A408.) The Junis attempt to quantify Mr. Juni's indirect exposure during that time by stating that "the mechanics would be

working on '[a]bout five' trucks at a time." (Appellants' Br. 13.) But, the quote to which the Junis refer says only that five trucks *could* be worked on at a time, not that five trucks were always receiving brake jobs, or for that matter being serviced in any way, at any given time. (A411.)

While Mr. Juni recalled that the trucks being serviced during his time at the Nyack Garage were manufactured by McKay Powers and Ford, he did not know who manufactured *any* of the friction products that were removed from or installed in those vehicles. (A408-10.) Mr. Juni believed that Ford manufactured the replacement brake pads that were installed on some service vans, but he could not estimate how many of these Ford replacement parts were used. (A563.) He also did not know who manufactured the brake pads that were removed from the vehicles. (A410.) When asked if he could recall the percentage of time that the first-class mechanics would use original equipment, such as Ford brake shoes or pads, versus replacement parts made by other manufacturers, Mr. Juni testified, "No, I can't." (A568.)

Mr. Juni also recalled that, while he was working at the Nyack garage, the first-class mechanics performed clutch work on certain Ford trucks and vans. (A414-15.) Mr. Juni did not perform any of this work himself, and he did not know how often the mechanics were performing this work. (A414-18.)

The Junis note in passing that Mr. Juni "assisted" in performing clutch work at the Nyack garage (Appellants' Br. 13 (citing A412, 415-17, 420).) As Mr. Juni explained, however, his role in this process was very limited, and did not involve working with friction material:

> Q. You told me that the first-class mechanics also did clutches at the Nyack garage. Did you ever do clutch work or was that the work of a first-class mechanic?
>
> A. Well, no, that was the work of the first-class man, but you went underneath when he was going to put the transmission back in, you assisted him to line up the transmission with the pilot bearing, with the throw-out bearing and in through the clutch.

(A414.)

In 1966, Mr. Juni transferred to the Spring Valley garage at Orange & Rockland, where he worked until his retirement in 2009, twenty-one years after his alleged exposure ended.[1] (A423.) Mr. Juni started at Spring Valley as a second class mechanic. (A424.) He believes that it is "possible" that he was exposed to asbestos while working as a second-class mechanic at Spring Valley, though he did not personally perform many brake jobs. (A426-27 ("I didn't do it as much, but other guys did it.").) Shortly after arriving at the Spring Valley garage, Mr. Juni was promoted to first-class mechanic, where he worked the night shift. (A424-25.) While serving as a first-class mechanic, Mr. Juni testified that he spent only

[1] Mr. Juni's employer issued respirators in the fall of 1988, after which he no longer alleges exposure. (A447.)

twenty-five percent of his time performing vehicle repair work. (A530.) He spent the rest of his time welding, which did not involve exposure to friction products. (*Id.*)

Mr. Juni was promoted to working foreman in 1970, and he held that job until he retired. (A425, 446.) As a working foreman, Mr. Juni had the administrative responsibilities of "organiz[ing]" the other mechanics and "keep[ing] them busy." (A446.) As a working foreman, Mr. Juni's repair work was limited to assisting other mechanics. (A449.)

Mr. Juni could not estimate how frequently original equipment was used in comparison to aftermarket brakes at Spring Valley. (A580.) Mr. Juni testified that Raybestos and Bendix manufactured the brake parts that were installed on the Ford vans and light trucks during this time period, but he did not know who manufactured the brakes that were removed. (A428-29, 433-38.) Although he "imagine[d]" that "original manufacturer" brakes may have been removed for first-time brake jobs on some of the Ford vehicles, Mr. Juni did not state how often that occurred. (A452.) Likewise, Mr. Juni testified that replacement clutches were manufactured by Lipe, Spicer, Borg Warner, and the original equipment manufacturer, but just as with the brakes, he did not state how frequently he encountered each product. (A458-59.) Mr. Juni also installed replacement gaskets manufactured by Fel-Pro, Victor, and the original equipment manufacturer.

(A496-97.) In sum, while Mr. Juni identified a number of manufacturers of friction products to which he was exposed, he did not provide any meaningful estimate of how many of those products were Ford products or how often he was exposed to Ford products.

Throughout their brief, the Junis make much of the fact that Mr. Juni testified that he would, on occasion, sand new brakes before installing them. (*See, e.g.*, Appellants' Br. 14.) Mr. Juni's own testimony on this point was very limited, however. Mr. Juni testified that, when installing a new brake, "*[s]ometimes* you had to scuff it up with sandpaper." (A657.6-657.7 (emphasis added).) He acknowledged that he "didn't need to [sand] it every time he performed a brake job," and he did not quantify how often this sanding work occurred. (A657.15-657.16.) He also acknowledged that, when sanding was required, it was usually "a quick process." (A657.15.) Mr. Juni did not provide any testimony concerning how often he sanded Ford friction products as compared to those of other manufacturers.

The Junis also note several times that Mr. Juni occasionally used compressed air to "blow" dust out of certain brake drums. (Appellants' Br. 6, 17, 23.) Mr. Juni acknowledged, however, that he did not have to use compressed air every time he did a brake job, and he admitted that he could not quantify how often that occurred, or how often he did so with respect to Ford products. (A657.16.)

Indeed, Mr. Juni admitted that, before using compressed air to blow a brake drum, he would occasionally wipe the dust with a wet rag first (*id.*), which would reduce the amount of dust that might become airborne with the use of compressed air. He did not, in any way, describe how often that would occur.

II. Chrysotile in Friction Products Lacks the Toxicity of Amphibole Asbestos Found in Insulation Products.

Up until the late 1980s, automotive friction products contained chrysotile asbestos. (A1533-36.) Unlike the asbestos contained in insulation products—which comes from a family of minerals known as "amphiboles"—chrysotile has short, curly fibers that are easily swept away by the body's natural defense mechanisms. (A241.) There is no dispute that amphibole asbestos is significantly more potent than chrysotile in terms of increasing the risk of mesothelioma—even the Junis' experts agree on this point. (A238-41, 273, 1191-92, 1197.)

The evidence introduced at trial also established that, whatever the toxicity of raw chrysotile asbestos, processed chrysotile found in automotive friction products does not have the same toxicity. As Ford's expert Brent Finley explained, the surface charge and chemistry of the asbestos in friction products are fundamentally modified during the brake manufacturing process:

> Q. How does the brake begin? What's the process of making a brake that creates the friction product?
>
> A. So it's made in a mold basically and you have a mold that's either shaped like a disc or a drum and into that they pour -- the

> manufacturers pour this goo that has a lot of resin and the fiber is already mixed in there and binders and a lot of inert stuff just to hold everything together, and then they bake it. They bake this goo at high temperature, it's called curing it, and then what you get is this hard piece, it's like a rock, basically. If you ever held a brake in your hand, you know, it is like a rock and then that's what goes on the car.

(A1708-09; *see also id.* at 219.) Even the Junis' general causation expert Steven Markowitz acknowledged that, when asbestos fibers are "embedded in the phenolic resin, they would not be respirable."[2] (A219.) Additionally, both Markowitz and the Junis' specific causation expert Jacqueline Moline acknowledged that the heat generated during the braking process causes the chrysotile in friction parts to undergo a fundamental transformation, converting most of it to a biologically inert substance known as forsterite. (A220, 244-51 (Markowitz) and A1198 (Moline).) Markowitz admitted that most studies show that any resulting dust is composed of less than one percent asbestos. (A250-51.)

Given all this, it is unsurprising that 21 of the 22 epidemiological studies of vehicle mechanics performed worldwide found *no increased risk* of mesothelioma among vehicle mechanics. (A1309-11.) The only epidemiological study

[2] The Junis appear to dispute whether the brakes to which Mr. Juni was exposed were made with phenolic resin. (Appellants' Br. 41.) In support, they point to testimony from Markowitz in which he noted, in passing, that at some undefined point "in history" phenolic resins were not used in brake manufacturing. (A219-20.) There is no indication, however, that the brakes on the Ford vehicles to which Mr. Juni was exposed were manufactured using these "historic" practices. The Junis cannot establish otherwise by pointing to a generic passing comment made by one of their experts, and they failed to establish any such evidence through their questioning of any of Ford's experts.

purportedly to the contrary, by Roelofs, *et al*., acknowledges on its face that it fails to account for additional exposures experienced by mechanic participants in other or prior jobs. As a result, the authors of the Roelofs study could not determine whether the cases of mesothelioma under study "resulted from exposure to asbestos experience[d] during employment in the reported usual occupation, or if these cases were exposed to asbestos in a prior occupation such as shipbuilding or in the military or in another occupation or exposure context not reported as their usual occupation." (A1572-73.)

This omission completely vitiates the study's ultimate conclusion. It is well established that individuals performing work around boilers, insulation, and power plants have a much higher risk of contracting mesothelioma compared to the general population, so the study's failure to account for other occupational exposures means that it ignored the actual cause of mesothelioma in many of the study's participants. (A1311-12.) Had Mr. Juni participated in the Roelofs study, the study would have failed to account for Mr. Juni's exposure to highly potent amphibole asbestos while working for Orange & Rockland at the Hillburn power plant. (A391-92, 401.) Thus, as Ford epidemiology expert Mary Jane Teta explained, this study is "not representative[,]" and the court should not "really put confidence in what they came up with." (A1571.)

The fact that the weight of existing science does not support a finding that brake mechanics face an increased risk of contracting mesothelioma is fatal to the Junis' ability to prove both general and specific causation, as explained in more detail below.

III. The Junis' Causation Experts

A. General Causation Expert Steven Markowitz

The Junis' general causation expert Steven Markowitz testified that exposure to asbestos in automotive friction products increases a person's risk of contracting mesothelioma. (A294-95.) Because, by his own admission, the available epidemiological studies addressing asbestos exposure in vehicle mechanics "do not show much evidence in support of a relationship between mesothelioma and exposure to friction products" (A114), he relied instead on a smattering of inapposite industrial hygiene studies as the basis for his opinion.

For example, Markowitz purported to rely on a series of studies conducted by NIOSH from the 1970s analyzing asbestos fiber counts in garages in New York City. (A245-46.) Markowitz conceded on cross-examination, however, that these studies dealt only with short-term or peak exposures. (A288.) He was unable to identify any specific study that showed that the time-weighted averages were in violation of then-existing standards.[3] (*Id*.) Critically, Markowitz was also unable

[3] As Ford's expert Brent Finley explained, evidence of allegedly high short-term exposure levels is misleading because the level of asbestos in the air in a garage varies considerably throughout

to point to any scientific evidence indicating that the exposures discussed in those studies were sufficient to cause mesothelioma. (A288-89.) As a result, they cannot possibly support his opinions regarding general causation.

Additionally, Markowitz purported to rely on other "industrial hygiene studies that have measured chrysotile asbestos among workers who are using friction products." (A286.) On cross-examination, however, he conceded that the subjects of those studies were factory workers who mass-produced friction products using *raw asbestos*, not garage workers working with or assisting others who were working with finished friction products. (A190-91) He also conceded that exposure to raw asbestos in the factory setting differs significantly from a mechanic's exposure to asbestos in a vehicle repair garage. (A191.)

Finally, Markowitz relied on case reports purportedly describing individual vehicle mechanics who had contracted mesothelioma. (A303.) However, as Ford's epidemiology expert Mary Jane Teta explained, case reports merely document individual cases and, on occasion, posit a hypothesis regarding the cause of a disease. (A1268, 1271.) The only way to draw a meaningful conclusion from

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the day. (A1721 ("So you got peaks and valleys throughout the day.").) As a result, the more relevant metric to use in assessing an individual's exposure – and the one that is incorporated in the federal Occupational Health and Safety Administration's ("OSHA's") workplace standards – is an individual's average exposure measured over the course of an entire day. (A1720-23.) This is known as a "time-weighted average." (A1720-21.) As Finley further explained, "when you look at all the brake job data, the measurements were always below the workplace standard that was in place at the time." (A1723-24.)

a case report would be to follow up with a "full-blown [epidemiological] study". (A1268.)

Given the absence of meaningful scientific support for his opinions, Markowitz was forced to concoct a "shortcut" for establishing general causation. (A130.) According to Markowitz, "when [dust] becomes visible we know then that product has been disturbed," and an exposure to that dust is sufficient to have substantially contributed to causing mesothelioma. (A130; 127-28.) But Markowitz failed to establish that the "dust" Mr. Juni encountered at the Orange & Rockland garage actually contained active asbestos fibers capable of causing mesothelioma, much less asbestos fibers from Ford products. (A251.) This is significant, because, as explained above, not all dust from friction products is asbestos fibers. To the contrary, the vast bulk of it is an inert substance, forsterite. (A250-51 (Markowitz) and A1198 (Moline).) Thus, Markowitz simply had no way of knowing whether the dust to which Mr. Juni was allegedly exposed was actually capable of causing mesothelioma, and any testimony regarding the toxicity of such dust is, at best, speculation.

B. Specific Causation Expert Dr. Jacqueline Moline

Dr. Jaqueline Moline, the Junis' expert on specific causation, testified that Mr. Juni's mesothelioma was caused by exposure to asbestos in Ford products based on the following assumptions: (1) from 1964 to 1988, Mr. Juni "personally

and regularly" assisted in performing brake and clutch work, including on Ford brakes and clutches; (2) Mr. Juni assisted in removing original Ford brakes and clutches and replacing them with new Ford brakes and clutches; and (3) Mr. Juni's work created and exposed him to visible asbestos dust.[4]

At no point in her lengthy testimony did Moline attempt to offer a scientific expression of Mr. Juni's actual exposure to asbestos from Ford products. Indeed, she conceded that she *had no information* on the issue:

> Q. How often was he exposed to Ford products? Can you give me once a week? Once every other week? Once a day? Once a month? Can you tell us?
>
> A. I don't know if he was asked specifically some of those questions in his deposition. So I don't know if it's possible to fully answer that question.
>
> Q. So the answer is that you do not at this moment in time possess the information to answer that question. Whether it's a recall issue or the data didn't get presented to you, you do not possess it at this moment?
>
> A. Correct.

[4] As is apparent from the facts stated above, the evidence presented at trial did not support the assumption that Mr. Juni "personally and regularly" performed brake or clutch work. Indeed, the vast majority of Mr. Juni's brake and clutch work occurred while he was assisting other mechanics. (A426-27, 449.) As a result, the hypothetical should have been stricken. *Espinosa v. A & S Welding & Boiler Repair, Inc.*, 120 A.D.2d 435, 437 (1st Dep't 1986) (affirming Supreme Court's exclusion of expert testimony in response to hypothetical question where "there was no factual basis for the question"). Ford raised this point in its post-trial briefing, but the Supreme Court did not reach this issue, holding instead that, even assuming the facts contained in this hypothetical did have support in the record, testimony based on these facts was not sufficient to establish specific causation. (A11817-25.)

Q. And am I correct that you did not perform any dose reconstructions or fiber dose assessments to attempt to assess the amount of exposure that Mr. Juni had to Ford friction products, that you did not do that?

A. There was no data. If I had even wanted to, I did not.

(A1157.) She also admitted that she did not know whether *any* of the chrysotile asbestos fibers from friction products to which Mr. Juni was allegedly exposed were still biologically active (that is, whether they maintained their biologic potential to cause mesothelioma). (A1199.)

Given this, Moline defaulted to a catch-all opinion that is neither scientifically sound nor product- or manufacturer-specific. Like Markowitz, Moline testified that friction products in general "contained virtually all chrysotile" and that "[c]hrysotile causes mesothelioma." (A1095-98.) Therefore, because Mr. Juni performed work on friction products installed in Ford vehicles, it was Moline's position that Ford, along with every other product manufacturer Mr. Juni recalled in his deposition, is responsible for causing Mr. Juni's mesothelioma. (A1102 ("There's no way to say it's not my company's product or one[] company's product and it's everyone else's. It all goes to the cumulative exposure that one has that causes the disease.").)

This opinion did not require any scientific inquiry into frequency, intensity, or proximity of exposure, because those factors were all conveniently presumed. Moline simply assumed that whenever Mr. Juni testified that he was exposed to

"visible dust," that dust contained asbestos (which she further assumed, without citing specific evidence, emanated from a Ford product) and constituted a "significant exposure." (A1095.) This opinion also failed to account for the significant differences in toxicity among the various fiber types to which Mr. Juni was exposed; instead, it treated all of Mr. Juni's alleged exposures as having similar toxicities, even though science says otherwise. (A1095-97.)

Moline denied that, under her theory, each and every one of Mr. Juni's exposure to asbestos would be deemed causative. (A1154.) Her denial is a smoke screen. Moline's only basis for distinguishing her theory from the theory that each and every exposures to asbestos is causative of mesothelioma is that her theory takes into account the "regularity" of exposure. (*Id.*) But that cannot possibly be true, as Moline freely admitted that no evidence existed regarding the regularity with which Mr. Juni worked with Ford products. (A1154-57.) Because Moline lacked any evidence or other foundation from which she could have determined that Mr. Juni was exposed to asbestos from Ford products "with regularity," her testimony collapsed into the tautology that "any" exposure Mr. Juni had to Ford products necessarily caused his disease.

In an effort to paper over the failings of Dr. Moline's specific causation testimony, the Junis highlight her testimony concerning studies that looked at short-term exposure to asbestos among workers in New York City garages during

the 1970s. (Appellants' Br. 7-8, 19-20, 32 (citing A1208-09).) Moline did not, however, compare the exposure of the workers involved in those studies with Mr. Juni's exposure. (A1208-13.) Indeed, absent some scientific expression of Mr. Juni's exposure, such a comparison is impossible. Thus, even if these studies did show that the mechanics under study faced an increased risk of contracting mesothelioma (which they do not), they cannot possibly provide foundation for Moline's specific causation testimony here because she had no way of knowing whether the exposure levels in the studies are comparable to Mr. Juni's exposure levels.

IV. The Decisions Below

A. Supreme Court

The Supreme Court held that neither of the Junis' causation experts had a sufficient foundation for their opinions under the governing standard set forth in *Parker*. (A11817-23) Absent such foundation, the court determined that the Junis failed to meet their burden to prove general and specific causation and entered judgment as a matter of law in favor of Ford. (A11825.)

The Supreme Court began its inquiry by addressing the general causation testimony of Markowitz. It held that Markowitz's reliance on generalized statements that exposure to chrysotile asbestos can cause mesothelioma was not sufficient to support his opinions here, because the issue in this case is "whether

chrysotile asbestos, *as contained within friction products*, causes mesothelioma." (A11814 (emphasis added).) Similarly, the industrial hygiene studies purportedly showing elevated levels of asbestos in garages actually involved factory workers who were exposed to raw chrysotile asbestos, and Markowitz conceded that exposure in such settings "was significantly greater than that occurring in a vehicle repair garage." (*Id.*) The Supreme Court also pointed out that those studies indicated only elevated levels of asbestos, which does not support Markowitz's theories because "proof of a risk, even an increased risk, does not constitute proof of causation." (*Id.*) Finally, the court noted Markowitz's acknowledgment that 21 of the 22 epidemiological studies involving vehicle repair workers showed that those workers were *not* at an increased risk of contracting mesothelioma. (A11817.) Based on all of this, the Supreme Court found that Markowitz's testimony failed to establish general causation. (*Id.*)

The Supreme Court then addressed Moline's specific causation testimony. The court noted that, because Moline had no knowledge of the amount, duration, or frequency of Mr. Juni's exposure to asbestos from Ford products, she could not "even minimally quantify" his exposures. (A11818.) The court also noted that Moline failed to "use any other method identified by the Court in *Parker* and *Cornell* to express Juni's exposure scientifically." (*Id.*) Thus, the Court found that Moline had failed to establish specific causation. (*Id.*)

The court rejected both experts' statements that they could use evidence of visible dust as a proxy for establishing a scientific expression of exposure in this case. (A11819-20.) The court pointed out, correctly, that Mr. Juni "never testified that he saw clouds of dust, or that he worked all day for long periods in clouds of dust[,]" and "neither Moline nor Markowitz knew whether the dust at issue contained enough asbestos to cause mesothelioma." (A11820.) As a result, the court found that, even if New York law did allow a jury to infer causation from evidence of visible dust alone, which it does not, the limited evidence concerning visible dust in this case was not sufficient to make such a showing. (*Id.*)

Finally, the court categorically rejected both experts' assumptions that each and every exposure to asbestos is sufficient to cause mesothelioma. According to the court, this opinion "is irreconcilable with the well-recognized scientific requirement, acknowledged by Moline, that the amount, duration, and frequency of exposure be considered in assessing the sufficiency of an exposure in increasing the risk of developing a disease." (A11821.) The court reiterated that New York law, specifically *Parker*, requires some "scientific expression" of the plaintiff's exposure to the allegedly harmful substance. (A11822, A11825.) Because the assumption that each and every exposure to asbestos is causative fails to address this fundamental requirement, the court held that it did not supply an adequate foundation for the testimony of Markowitz and Moline here. (A11825.)

B. Appellate Division

The Appellate Division, First Department, affirmed. Like the Supreme Court, the Appellate Division pointed out that *Parker* and its progeny obligate the Junis "to prove not only that Mr. Juni's mesothelioma was caused by exposure to asbestos, but that he was exposed to sufficient levels of the toxin from his work on brakes, clutches, or gaskets, sold or distributed by defendant, to have caused his illness." (A5-6.) The Appellate Division concluded that the Junis' evidence was insufficient under this standard because both Markowitz and Moline "effectively testified only in terms of an increased risk and association between asbestos and mesothelioma" and "failed to either quantify the decedent's exposure levels or otherwise provide any scientific expression of his exposure level with respect to Ford's products." (A7.)

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

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

One judge filed a dissenting opinion in which he embraced the Junis' argument that "visible dust released from an asbestos product contains high levels of fibers of asbestos capable of producing disease" and that such evidence is sufficient to establish causation under existing New York law. (A36.) In a concurring opinion, however, another judge observed that reliance on the existence of visible dust as proxy for exposure would carve "a gaping hole in the *Parker* standard of proof on causation," "eviscerate[] its fundamental evidentiary requirements," and "effectively overrule the Court of Appeals' holding in *Cornell*." (A17.) Thus, the concurring judge agreed with the panel majority that the Junis failed to establish causation as a matter of New York law. (A18.)

STANDARD OF REVIEW

The Junis are correct that the Supreme Court's entry of judgment as a matter of law is reviewed *de novo*. *Cohen v. Hallmark Cards, Inc.*, 45 N.Y.2d 493, 499

(1978). However, rulings on the admissibility of expert testimony, including the Supreme Court's ruling that Markowitz and Moline lacked adequate foundation for their opinions, are "addressed to the discretion of the trial court" and should not be disturbed on appeal absent an abuse of discretion. *De Long v. Cty. of Erie*, 60 N.Y.2d 296, 307 (1983).

ARGUMENT

I. The Appellate Division Properly Affirmed Judgment as a Matter of Law in Favor of Ford.

The courts below properly held that the Junis failed to meet their burden to prove that asbestos from Ford friction products caused Mr. Juni's mesothelioma. In order to establish causation in a toxic tort case under New York law, a plaintiff must offer scientifically reliable expert testimony demonstrating that: (1) the toxin to which plaintiff alleges exposure is capable of causing the plaintiff's particular illness (general causation), and (2) the plaintiff was exposed to sufficient levels of that toxin to cause the illness (specific causation). *Parker*, 7 N.Y.3d at 448. Regardless of whether the Junis can establish that Mr. Juni was exposed to asbestos attributable to Ford products (which, as noted above, is uncertain at best), they have failed to establish in this case that asbestos from friction products is capable of causing mesothelioma or that Mr. Juni was exposed to sufficient levels of asbestos from Ford products to cause his mesothelioma. As a result, the

Appellate Division correctly affirmed judgment notwithstanding the verdict in favor of Ford.

A. The Junis failed to prove general causation.

1. New York law requires a plaintiff to prove, through valid scientific evidence, that a toxin is capable of causing a particular disease in order to establish general causation.

In order to prove general causation, a plaintiff must offer "proof that the toxin in question can in fact cause the illness, and the amount of exposure required to cause the illness (the dose-response relationship)." *Sean R.*, 26 N.Y.3d at 808-09; *Cornell*, 22 N.Y.3d at 784; *Parker*, 7 N.Y.3d at 445-46 & n.2. Epidemiology, "the study of disease patterns in human populations" is the branch of science that bears most directly on these questions. *Nonnon v. City of New York*, 88 A.D.3d 384, 394 (1st Dep't 2011). Epidemiology studies "observe the effect of exposure to a single factor upon the incidence of disease in two otherwise identical populations" in an effort to determine whether unusual patterns of disease are associated with environmental or biological risk factors. *Id.* (citation omitted).

In cases where, as here, the great weight of the epidemiology does *not* find an association between exposure to a particular substance and a disease, a toxic tort plaintiff seeking to prove such an association must address those studies and provide some valid, scientific evidence that supports a contrary conclusion. *See, e.g.*, *Parker*, 7 N.Y.3d at 450 (finding expert general causation testimony

insufficient based on the fact that "[p]laintiff's experts were unable to identify a single epidemiologic study finding an increased risk of AML as a result of exposure to gasoline"); *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 882 (10th Cir. 2005) ("We are not holding that epidemiological studies are always necessary in a toxic tort case. We are simply holding that where there is a large body of contrary epidemiological evidence, it is necessary to at least address it with evidence that is based on medically reliable and scientifically valid methodology.").

Parker and *Cornell* are the leading New York cases on the issue of general causation. *Parker* involved a claim by a plaintiff who alleged that exposure to benzene over the course of a seventeen-year career working as a gas station attendant caused his acute myeloid leukemia ("AML"). *Parker*, 7 N.Y.3d at 442. His expert witness on general causation "concentrate[d] on the relationship between exposure to benzene and the risk of developing AML[,] an association that [was] not in dispute." *Id.* at 449. This Court found that such testimony was insufficient to establish general causation and that, instead, the appropriate comparison was "the relationship, if any, between exposure to *gasoline* containing benzene as a component and AML." *Id.* at 449-50. Because the expert failed to make that specific causal connection, this Court found that the plaintiff had failed to establish general causation. *Id.* at 450 ("[Plaintiff's expert] fails to make [the

connection between gasoline and AML] perhaps because, as defendants claim, no significant association has been found between gasoline exposure and AML. Plaintiff's experts were unable to identify a single epidemiologic study finding an increased risk of AML as a result of exposure to gasoline.").

This Court took a similar approach in *Cornell*. *Cornell* involved a tenant's claim that mold in her apartment caused her to suffer various physical and mental injuries. *Cornell*, 22 N.Y.3d at 766. Her expert witness on general causation opined that "it is generally accepted within the relevant community of scientists . . . that exposure to mold causes human disease." *Id.* at 781. In support, the expert cited (1) a variety of government reports from public health agencies that "issued guidelines and recommended precautions to safeguard against the risk of harm from indoor mold exposure" and (2) various studies that purported to establish an "association" between mold exposure and Cornell's ailments. *Id.* at 782-83.

This Court found that the testimony was insufficient to establish general causation. As this Court held, "studies that show an *association* between a damp and moldy indoor environment and [a plaintiff's alleged injuries] do not establish that the relevant scientific community generally accepts that molds *cause* these adverse health effects." *Cornell*, 22 N.Y.3d at 783. Additionally, the fact that public health agencies had issued reports discussing the risks posed by mold exposure was "irrelevant since 'standards promulgated by regulatory agencies as

protective measures are inadequate to demonstrate legal causation.'" *Id.* at 782 (quoting *Parker*, 7 N.Y.3d at 450). Thus, this Court held that the general causation testimony lacked adequate foundation and entered judgment as a matter of law in favor of the defendant.

2. The Junis' experts failed to base their general causation testimony on proper scientific foundation.

Like the expert witnesses in *Parker* and *Cornell*, the Junis' witnesses lacked sufficient foundation for their general causation opinions. As noted above, the Junis identified only one epidemiological study that purports to find that vehicle mechanics are at an increased risk of contracting mesothelioma: the Roelofs study. (Appellants' Br. 46.) However, the Roelofs study acknowledges on its face that it fails to account for additional exposures experienced by the mechanic participants in other or prior jobs. (A1570, 1572-73.) This is a critical flaw that renders any conclusions regarding the actual cause of the subjects' mesothelioma meaningless. (A1573.) By the authors' own admission, it is impossible to determine whether the study subjects' mesothelioma was caused by their work as mechanics or by their exposure to more potent amphibole asbestos from other employment. (*Id.*) Thus, much like Markowitz and Moline's testimony here (which obscures the significant differences between Mr. Juni's exposure to highly-potent amphibole asbestos at the power stations and his exposure to less-potent chrysotile asbestos in friction products) Roelofs' failure to account for prior occupations "mask[ed] the

true source of the asbestos exposure" and probably caused the authors to incorrectly attribute the subjects' mesothelioma to their vehicle mechanic work when "it's really another job that's causing the problem." (A1572 (Ford expert Mary Jane Teta explaining the shortcomings of the Roelofs study).)

Additionally, like the *Parker* expert witness discussed above, Markowitz and Moline relied on studies involving the wrong substance. By their own admission, Markowitz and Moline relied on a number of studies involving raw asbestos exposure among factory workers. (A190-91 (Markowitz) A1095 (Moline).) However, similar to *Parker*, the relevant question here is not whether exposure to chrysotile in general can cause mesothelioma, but, rather, whether exposure to chrysotile asbestos contained in Ford friction products can cause mesothelioma. As a result, like the witness in *Parker*, the general causation testimony of the Junis' witnesses fails as a matter of New York law.

In their brief, the Junis argue repeatedly that the Roelofs study is "closer" to Mr. Juni's actual exposures than the remaining 21 studies and, as a result, it is the only epidemiological study that is "relevant" to the question before the Court in this case. (Appellants' Br. 26-27.) Even if this were true (and the Junis fail to explain why they think that it is), it would be immaterial. As Teta explained, "epi[demiological] studies aren't perfect" and, therefore, one "can't draw a final conclusion from a single study." (A1274, 1264.) Instead, in order to determine

whether a certain population is at an increased risk for contracting a disease, epidemiologists look for "consistency"—that is, a pattern of similar results across a series of different studies. (A1274.)

The Junis devote several pages of their brief to nit-picking the extensive body of epidemiological studies relied on by Ford's experts. (Appellants' Br. 43-44.) Their attempts are unavailing. As an initial matter, such arguments misapprehend the burden of proof. It is the Junis, not Ford, who must establish the reliability of their experts' causation opinions. As the courts below held, the fact that 21 of the 22 epidemiological studies find no increased risk for garage workers weighs heavily in favor of finding that Markwotz's opinions are inadequate as a matter of New York law. Moreover, as explained above, courts evaluating epidemiological evidence to determine whether general causation exists should employ a meta-analysis and look for consistency across studies, not rely on a single, critically flawed study whose results are undermined by its admitted failure to account for alternative causes. (A1274, 1264.) As Teta explained, minor shortcomings in any one study are insufficient to undermine a consensus view across a series of different studies. (A1274, 1264.)

The Junis' inability to identify any reliable epidemiological evidence demonstrating that vehicle mechanics are at an increased risk of contracting mesothelioma is fatal to their experts' general causation opinions. While it is true,

in some cases, that a plaintiff can prove general causation even absent epidemiological support, that is not the case where, as here, there is a large body of epidemiology that undermines a suggested causal relationship. In such cases, a plaintiff cannot meet his burden to prove general causation without evidence countering the existing epidemiology. *See, e.g.*, *Parker*, 7 N.Y.3d at 450; *Norris*, 397 F.3d at 882.

In the absence of any valid epidemiological studies supporting their position, the Junis try to fill the gaps in their expert witness testimony by pointing to a mishmash of inapposite evidence. For example, Moline and Markowitz sought to rely on a variety of industrial hygiene studies involving brake workers, but they admitted that those studies addressed the risk of cancer arising from exposure to unprocessed asbestos in a factory setting. (A190-91 (Markowitz), A1163-64 (Moline).) Just like the studies relied upon by the witness excluded in *Parker*, these studies do not address the relevant putative causal relationship—that is, the alleged relationship between chrysotile in friction products and mesothelioma. *Parker*, 7 N.Y.3d at 446. As a result, this evidence fails to supply a sufficient foundation for the Junis' causation experts' testimony.

The Junis also note that both Markowitz and Moline rely heavily on a variety of government documents warning of the potential risks of asbestos-containing friction products and recommending measures to prevent those risks.

(Appellants' Br. 9.) But prophylactic regulations, grounded on risk, can never prove causation. The fact that the medical and scientific community is not aware of a *known* safe level of exposure does not justify an inference, in a court of law, that all exposures are *causative* of a disease. *Cano v. Everest Mineral Corp*., 362 F. Supp. 2d 814, 849 (W.D. Tex. 2005). Regulatory standards, which are precautionary in nature and intended to protect the public of potential future adverse uses of toxic substances, employ a lower threshold of proof than that which is necessary to prove causation in a court of law. *Yates v. Ford Motor Co.*, 113 F. Supp. 3d 841, 847 (E.D.N.C. 2015) (explaining that "courts have recognized a distinction between evaluations made by regulatory agencies and the standard of causation necessary to show tort liability") (citations omitted); David L. Eaton, *Scientific Judgment and Toxic Torts—A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & POL'Y 5, 36 (2003) ("[R]egulatory levels . . . are of limited value in judging whether a particular exposure was a substantial contributing factor to a particular individual's disease or illness.") These considerations drove this Court to hold—on at least two occasions—that "standards promulgated by regulatory agencies as protective measures are inadequate to demonstrate legal causation" *Cornell*, 22 N.Y.3d at 782 (quoting *Parker*, 7 N.Y.3d at 450). Thus, whatever weight these documents might have carried in establishing Ford's alleged

duty to warn (which is not at issue here), they are simply irrelevant on the question of general causation.

Because the Junis' witnesses lacked any meaningful scientific support for their general causation opinions, the Appellate Division properly affirmed the Supreme Court's grant of judgment as a matter of law in favor of Ford. The judgment below should be affirmed on this basis alone.

3. Ford has not "admitted" that chrysotile in friction products is capable of causing mesothelioma.

In an effort to buttress their experts' insufficient general causation testimony, the Junis argue at length that some of the internal Ford documents introduced at trial contain "admissions" that vehicle mechanics are at an increased risk of developing mesothelioma. (Appellants' Br. 17-23.) These attempts fail. As an initial matter, neither of the Junis' expert witnesses relied on any of these alleged admissions, so they cannot possibly supply the necessary foundation for their opinions. Moreover, the Junis' characterization of this evidence is simply inaccurate. As set forth below, none of the documents identified by the Junis amounts to an admission that vehicle mechanics are at an increased risk of contracting mesothelioma.

Internal Ford Memos Discussing Recommended Safety Practices. The Junis cite a number of memos and bulletins written by Ford employees detailing recommended safety measures designed to minimize exposure to brake dust in

Ford garages. (Appellants' Br. 18-19, 20, 22-23.) For example, a 1973 memorandum from Ford Industrial Hygienist Norman Brusk reported the results of various OSHA compliance tests and recommended that Ford adopt safety measures to confine brake dust to certain areas of the work space. (A2094.) Another 1973 memorandum by Paul Toth recommended certain methods for safe disposal of brake dust. (A2246.) The Junis also cite a 1983 bulletin that makes similar recommendations. (A2100.)

None of the documents constitutes an admission that vehicle mechanics are at an increased risk of developing mesothelioma. There is no discussion in the documents at all regarding whether brake dust is causally related to mesothelioma or any other asbestos-related disease. (A2094, 2100, 2246.) The mere fact that Ford employees recommended the adoption of preventative safety practices is not in any way an admission that vehicle mechanics are at an increased risk of contracting an asbestos related disease. As Ford's state of the art and toxicology expert Brent Finley explained at trial, precautionary measures were often recommended before there was scientific proof of a risk. (A1768.) Thus, a jury could not reasonably infer, based on these recommendations, that vehicle mechanics face an increased risk of contracting mesothelioma.

Meeting Minutes. The Junis also cite minutes from a 1973 meeting co-sponsored by several federal agencies. (Appellants' Br. 19-20 (citing A2333).)

Two Ford representatives were in attendance. (A2338.) The stated purpose of the meeting was "(1) to consider preliminary data on asbestos exposure among brake lining workers and (2) to discuss a future course of action based on this preliminary information." (A2333.) Among the items discussed at the meeting were the 1970s era studies of asbestos in New York City garages relied upon by the Junis' witnesses. (A2334.) The data extracted from those studies was admittedly preliminary, and neither the studies themselves nor the attendees at the meeting attempted to draw any conclusions regarding the causal connection between asbestos in brake dust and any disease. (*Id*.)

Even if such a connection had been discussed at the meeting, the mere fact that Ford employees were present does not give rise to an inference that Ford agreed with any statements made by other attendees. In order to qualify as an admission by Ford, the Junis would have to establish that Ford "acknowledge[d] and assent[ed]" to a statement such that it was "effectively [Ford's] own admission." *People v. Campney*, 94 N.Y.2d 307, 311 (1999) (citation omitted). Thus, even if the Junis could point to any reference in the meeting minutes tending to show that mesothelioma was caused by exposure to asbestos in brake parts, the Junis failed to point to any evidence tending to show that Ford assented to any statements discussed in this document. As a result, the Junis cannot establish that these minutes contain any admissions by Ford.

Engineering Report. The Junis also cite, without providing any meaningful context, an internal Ford engineering report that states that "the most critical exposures [to asbestos] occur during brake and clutch repair." (Appellants' Br. 22 (citing A2163).) That report was designed to identify *potential* carcinogens in an effort to improve workplace safety and comply with potential OSHA regulations. (A2163.) It does not set forth any conclusion that exposure to chrysotile asbestos from friction products can in fact cause mesothelioma. (*Id.*)

B. The Junis failed to prove specific causation.

1. New York law requires expert testimony based on a scientific expression of the plaintiff's exposure to the alleged toxin in order to prove specific causation.

The next step in any *Parker* analysis is to establish specific causation, that plaintiff was exposed to sufficient levels of the toxin to cause the illness. *Parker*, 7 N.Y.3d at 448. In order to make such a showing, the Junis were required to introduce enough reliable scientific evidence to allow the jury to conclude that Mr. Juni was "exposed to sufficient levels" of chrysotile asbestos from Ford products to cause his mesothelioma. *Id.* While this did not require the Junis to "pinpoint exposure with complete precision," it did require some "scientific expression" of Mr. Juni's alleged exposure to asbestos from Ford products. *Id.* at 449. As this Court has held multiple times, "we have never 'dispensed with a plaintiff's burden

to establish sufficient exposure to a substance to cause the claimed adverse health effect.'" *Sean R.*, 26 N.Y.3d at 808-09 (quoting *Cornell*, 22 N.Y.3d at 784).

2. Moline failed to provide a scientific expression of exposure.

Moline's specific causation testimony failed to conform to this Court's requirements set forth in *Parker*. In *Parker*, the plaintiff proffered two causation experts in support of his claim that exposure to benzene in gasoline caused his leukemia. *Parker*, 7 N.Y.3d at 442-43. One of the witnesses relied on a series of epidemiological studies involving oil refinery workers who developed leukemia and testified that, because Parker had "far more exposure to benzene than did the refinery workers in the epidemiological studies," his exposure was sufficient to cause his disease. *Id*. at 449. Parker's other witness cited similar studies and added that Parker had been exposed to "frequent" and "excessive" amounts of benzene. *Id*. Neither witness attempted to quantify Parker's exposure in any way. *Id*. at 447-49.

This Court ruled that both witnesses' testimony lacked foundation. Even though benzene is a known carcinogen, there needed to be some "scientific expression of [the plaintiff's] exposure level" in order for the testimony to be admissible. *Parker*, 7 N.Y.3d at 449. The fact that, in some cases, an expert might be unable to "pinpoint exposure with complete precision," was immaterial. *Id.* As this Court held, in such cases, "exposure can be estimated [by the expert witness]

through the use of mathematical modeling by taking a plaintiff's work history into account to estimate the exposure to a toxin." *Id.* at 449. Alternatively, "[c]omparison[s] to the exposure levels of subjects of other studies could be helpful provided that the expert made a *specific comparison* sufficient to show how the plaintiff's exposure level related to those of the other subjects." *Id*. at 449 (emphasis added). Because the expert witnesses in *Parker* failed to take any of these steps, this Court found that their testimony lacked foundation.

Likewise, Moline failed to offer any scientific expression of Mr. Juni's alleged asbestos exposure from Ford products, if any. Even though Moline agreed that "the amount of the exposure, the duration of an exposure, and the frequency of the exposure . . . are critical in assessing whether there is sufficient exposure in order for someone to have an increased risk of disease from the exposure" (A1150-51), she could not quantitatively or qualitatively assess the level of Mr. Juni's exposure to asbestos from Ford's products in any way (A1090, 1157). She also acknowledged that she failed to perform any sort of dose-response assessment. (A1157.) Rather, her testimony was based on nothing more than vague descriptions of Mr. Juni's exposure. (A1092-93 (hypothetical question asking Moline to assume that Mr. Juni was "regularly" exposed to asbestos).) Indeed, the exposure evidence here was even less specific than that firmly rejected as inadequate in *Parker* because here, unlike in *Parker*, Moline did not even attempt

to compare Mr. Juni's exposure levels to exposure levels discussed in previous studies. (A1208-14 (Moline).) In the absence of any evidence connecting Mr. Juni's disease to any particular level, frequency, or duration of exposure to Ford asbestos-containing brake products, Moline's testimony regarding specific causation was insufficient, as the courts below properly held.

3. Moline's reliance on industrial hygiene studies is insufficient because she failed to compare Mr. Juni's exposures to the exposures of the workers under study.

The Junis assert, on multiple occasions, that Moline "quantified" Mr. Juni's exposure by referencing various industrial hygiene studies measuring the asbestos exposures of New York City mechanics in the 1970s. (Appellants' Br. 19-20.) This argument misses the mark on multiple levels. Most importantly, even if these studies could be used to supply a scientific expression of Mr. Juni's exposures (which, as explained below, they cannot), there is no indication that the exposure levels documented in these studies are sufficient to cause mesothelioma. Thus, these studies show, at most, that the mechanics under study were exposed to increased amounts of asbestos fibers. This is not sufficient to establish specific causation under New York law. *See Parker*, 7 N.Y.3d at 448 (explaining that proof of specific causation must include evidence that the plaintiff was "exposed to sufficient levels of the toxin to cause the illness").

But equally significant, the Junis offer no evidence that the exposures described in these studies are representative of Mr. Juni's actual exposures in this case. Studies seeking to quantify exposure levels among friction product workers are of no consequence on the question of specific causation in this particular case absent "a *specific comparison* sufficient to show how the plaintiff's exposure level related to those of the other subjects." *Parker*, 7 N.Y.3d at 449 (emphasis added). While the Junis assert that Moline made such a comparison (Appellants' Br. 33-34), they are wrong. There is no testimony anywhere in the record comparing the specific circumstances of Mr. Juni's exposure to the specific circumstances of exposure in reported studies. (A1093, 1097-98.) As a result, such studies cannot provide foundation for Moline's specific causation testimony here.[5]

4. Testimony regarding the presence of visible dust does not satisfy *Parker*'s foundational requirements for specific causation.

The Junis argue repeatedly that the presence of visible dust in Mr. Juni's workplace is sufficient, in and of itself, to establish that he was exposed to levels of asbestos sufficient to have caused his mesothelioma. (*See, e.g.*, Appellants' Br.

[5] The failure to make such a comparison is particularly problematic for the Junis on the facts of this case because, as explained above, much of Mr. Juni's career as a mechanic was not spent performing vehicle repair work himself; instead, he spent years as a supervisor and a welder, neither of which involved exposure to asbestos from friction products. (*See* Statement of Facts, Section I, *supra*.) Additionally, Mr. Juni acknowledged that the vehicle repair work that he actually did perform involved "assisting" other mechanics. (*Id.*) Had Moline attempted to compare Mr. Juni's exposures to the exposures described in reported studies, she would have had to confront significant differences among them.

55.) There are a number of problems with this approach. Most notably, as the concurring opinion below observed, embracing this self-described "shortcut" would create a "gaping" hole in *Parker*'s standards governing the admissibility and sufficiency of expert testimony. (A17.) The Junis fail to address this issue in their brief, likely because there is no way to square this unscientific approach with *Parker*'s explicit "scientific expression" requirement. As a result, this Court's precedents demand rejection of this argument.

Additionally, even if it were appropriate to allow the presence of visible dust to serve as a proxy for dose in some cases, that approach is not possible on the present record. As the Supreme Court observed, "neither Moline nor Markowitz knew whether the dust at issue contained enough asbestos to cause mesothelioma." (A11820.) This is significant because not all dust generated by automotive friction parts contains respirable, chemically active asbestos—as explained above, the processing of the chrysotile that occurs during the manufacturing process and the heating of that chrysotile that occurs during the braking process renders the overwhelming majority of the chrysotile embedded in brake products inert. (*See* Statement of Facts, Section I, *supra*.) Thus, even if the presence of visible dust might, in some cases, serve as a "shortcut" around *Parker*'s foundational requirements, it cannot do so here.

5. Moline's cumulative exposure theory is unscientific and incompatible with New York law.

While Moline tried to avoid using the phrase "each and every exposure," acceptance of her specific causation theory would require this Court to adopt the view that each and every one of Mr. Juni's exposures to asbestos-containing friction products, regardless of how fleeting or infrequent those exposures might have been, were causative of Mr. Juni's mesothelioma. (*See* Statement of Facts, Section III.B, *supra*.)

Courts throughout the country have routinely rejected similar testimony in similar cases. *See, e.g., Krik v. Exxon Mobil Corp.*, 870 F.3d 669, 677 (7th Cir. 2017); *McIndoe v. Huntington Ingalls Inc.*, 817 F.3d 1170, 1177 (9th Cir. 2016); *Scapa Dryer Fabrics, Inc. v. Knight*, 788 S.E.2d 421, 427 (Ga. 2016); *Vedros v. Northrop Grumman Shipbuilding, Inc.*, 119 F. Supp. 3d 556, 562-63 (E.D. La. 2015); *Yates v. Ford Motor Co.*, 113 F. Supp. 3d. 841, 845-46 (E.D.N.C. 2015); *Bostic v. Georgia-Pacific Corp.*, 439 S.W.3d 332, 356 (Tex. 2014); *Smith v. Ford Motor Co.*, No. 2:08-cv-630, 2013 U.S. Dist. LEXIS 7861, at *4-14 (D. Utah Jan. 18, 2013); *Betz v. Pneumo Abex, LLC*, 44 A.3d 27, 36-58 (Pa. 2012); *Holcomb v. Georgia Pacific, LLC*, 289 P.3d 188, 194 (Nev. 2012) (treating such testimony as a "radical approach" and a "fiction"); *Gregg v. V-J Auto Parts, Co.*, 943 A.2d 216, 222-27 (Pa. 2007); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 769-72 (Tex.

2007); *Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 542-44 (Ga. Ct. App. 2011); *Smith v. Kelly-Moore Paint Co.*, 307 S.W.3d 829, 837-39 (Tex. App. 2010).

In *Scapa*, for example, the plaintiff's expert witness, Abraham, testified that, if the plaintiff actually was exposed to asbestos while working at defendant's facility, that exposure was a cause of his mesothelioma, regardless of the extent of the exposure. *Scapa*, 788 S.E.2d at 423. Like Moline, Abraham testified that "the precise point at which cumulative exposure is sufficient to cause any particular person to develop mesothelioma is not scientifically knowable, and for that reason, when a person actually has mesothelioma, it can only be attributed to his cumulative exposure as a whole." *Id.* at 423-24. Thus, according to Abraham, "[b]ecause each and every exposure to respirable asbestos in excess of the background contributes to the cumulative exposure . . . each exposure in excess of the background is a contributing cause of the resulting mesothelioma, regardless of the extent of each exposure." *Id.* at 424.

The Georgia Supreme Court rejected Abraham's testimony, finding that this testimony did not fit the causation question presented in that case. In *Scapa*, as here, the question of whether the plaintiff was exposed to "*any* asbestos beyond background—and if so, whether that exposure was anything more than de minimis—was seriously disputed at trial." *Scapa*, 788 S.E.2d at 426. The court acknowledged that the jury was, of course, free to find that the plaintiff did in fact

incur substantial exposure to asbestos, but it also noted that proof of exposure alone was insufficient to establish causation absent expert testimony connecting that exposure to the plaintiff's disease. *Id.* The court went on to find that, because "Abraham essentially told the jury that it was unnecessary to resolve the extent of [plaintiff's exposure]" his testimony could not have been helpful to the jury. *Id.* As a result, the court entered judgment in favor of the defendant.

Yates v. Ford Motor Co., 113 F. Supp. 3d 841 (E.D.N.C. 2015) is pertinent here as well. The plaintiff in *Yates* sued numerous companies, including Ford, alleging that his mesothelioma was caused by exposure to asbestos during backyard automobile work and while working at an automobile parts warehouse. His expert witness, Mark, testified that Yates' mesothelioma was caused by his "special" exposures to mesothelioma. *Id.* at 849-63. Mark defined "special" exposures as exposures "for which there is scientific evidence that the exposure increases the risk of developing diffuse malignant mesothelioma." *Id.* at 849. He also distinguished "special exposures" from "trivial exposures," and he defined "trivial exposures" as "exposures for which there is no scientific evidence to find an increased risk of disease." *Id.* Like the Junis' experts here, however, Mark did not perform a dose assessment of Mr. Yates' exposure and had no principled means of distinguishing a "special" exposure from a "trivial" one. *Id.* at 855-56. He could not point to any peer-reviewed scientific literature to support his

"special" versus "trivial" distinction, nor could he explain how workplace exposures consistent with background levels of exposure could be "special" when background exposures themselves are not. *Id.* at 854-55.

Yates argued that Mark's "special exposure" approach was not the same thing as "each and every exposure" testimony because, as here, Mark emphasized the *cumulative* nature of Yates' exposures and because Mark at least purported to be willing to exclude trivial exposures. *Yates*, 113 F. Supp. 3d at 849. The district court acknowledged the distinction between Mark's "special exposure" theory and the much-maligned "each and every" exposure theory, but went on to find that Mark's testimony should be excluded for many of the same reasons vitiating the "each and every" exposure approach. *Id.* at 851-52. According to the court, Mark's "special exposure" theory failed to account for the differences in potency or propensity to cause disease by different fiber types. *Id.* at 854. The court also undertook a thorough examination of the literature cited by Mark and found that the studies did not support Mark's opinion that low levels of chrysotile exposure can cause disease. *Id.* at 856-60. The court criticized Mark's testimony that Yates' asbestos exposure increased his "risk" for disease on the ground that regulatory or medical *risk* is not the same as legal *causation*. *Id.* at 853. Finally, the court found Mark's secondary opinion—that exposure to "visible dust increase[s] the *risk* of diffuse malignant mesothelioma"—to lack scientific foundation. *Id.* at 853-56.

Like the expert testimony eschewed in *Scapa* and *Yates*, Moline's testimony is untethered to the particular circumstances of Mr. Juni's exposure. This is a fatal flaw, and one that cannot be rectified by resort to the legal fiction that cumulative exposure asbestos is sufficient to cause mesothelioma. Rather, in order to be relevant and reliable, expert causation testimony must be tied, in some scientific way, to the actual exposure evidence introduced in a particular case. *See Scapa*, 788 S.E.2d at 425; *Yates*, 113 F. Supp. 3d at 852. Because Moline's testimony was not based, in any meaningful way, on Mr. Juni's actual levels of exposure, it should meet the same fate as the evidence excluded in *Scapa*, *Yates*, and the other cases set forth above.

6. The authorities cited by the Junis do not support their specific causation arguments here.

The majority of the Junis' limited legal argument section is devoted to a discussion of non-binding authorities which, they contend, demonstrate that their specific causation testimony was sufficient under New York law. (Appellants' Br. 49-54.) As explained in the following paragraphs, however, none of those authorities support the Junis' arguments here.

For example, the Junis rely heavily on the *Reference Manual* in support of their argument that "precise quantification" is unnecessary to establish causation under New York law. (Appellants' Br. 51-52.) The Reference Manual, however, does not go as far as the Junis seek to stretch it. While the *Reference Manual*

does acknowledge that "[a]ssessment of past exposures is especially difficult when considering diseases with long latency periods," it also notes that "courts regularly deal with evidence reconstructing the past, and assessment of toxic exposure is another application of this common practice." *See* Federal Judicial Center, *Reference Manual on Scientific Evidence* 512 (3d ed. 2011). Thus, while the *Reference Manual* acknowledges that a number of courts have adopted different standards for proving causation in asbestos cases, it does not state that those standards obviate the need to provide *some* scientific measure of exposure in each case. *Id.*

The Junis also rely heavily on the Fourth Circuit's opinion in *Westberry v. Gislaved Gummi AB*, 178 F.3d 257 (4th Cir. 1999), for the proposition that a scientific expression of exposure is unnecessary in asbestos cases. (Appellants' Br. 50.) While the Fourth Circuit did, in fact, hold that Westberry did not need to provide any quantitative evidence of exposure, that holding was based on the fact that Westberry had provided detailed qualitative evidence showing significant exposure: "talc that settled from the air around [the plaintiff's] work area was so thick that one could see footprints in it on the floor. He further stated that he worked in clouds of talc and that it covered him and his clothes." *Westberry*, 178 F.3d at 264. The Fourth Circuit subsequently held that *Westberry* does not obviate the need to prove dose in all cases, only in cases where the evidence of substantial

exposure is indisputable. *Zellers v. NexTech Northeast, LLC*, 533 F. App'x 192, 198 n.8 (4th Cir. 2013) (distinguishing *Westberry* on the ground that "the record was replete with evidence of the plaintiff's substantial exposure to talc. . . . Here, there is no evidence of such substantial exposure. Thus, *Westberry* does not support Ms. Zellars's claim that she need not put forth specific evidence regarding her level of exposure."). Additionally, unlike here, the defendant did not contest general causation in *Westberry*. *Westberry*, 178 F.3d at 264 ("The Material Safety Data Sheet (MSDS) for talc provided by [defendant] for Dr. Isenhower's examination provided that '[i]nhalation of dust in high concentrations irritates mucous membranes,' and it is undisputed that sinuses are mucous membranes.").

Finally, the Junis seek support in the Supreme Court of Pennsylvania's recent decision in *Rost v. Ford Motor Co.*, 151 A.3d 1032 (Pa. 2016). (Appellants' Br. 51.) *Rost*, however, is distinguishable. In *Rost*, the Pennsylvania high court reaffirmed "two basic precepts" important to the resolution of expert causation issues in asbestos cases:

> First, expert testimony based upon the notion that "each and every breath" of asbestos is substantially causative of mesothelioma will not suffice to create a jury question on the issue of substantial factor causation. Second, to create a jury question, a plaintiff must adduce evidence that exposure to defendant's asbestos-containing product was sufficiently "frequent, regular, and proximate" to support a jury's finding that defendant's product was substantially causative of the disease.

Rost, 151 A.3d at 1044. The court went on to distinguish the testimony offered by the plaintiff's expert, Frank, in that case from the testimony excluded in prior cases on the ground that Frank's testimony in *Rost* "confirmed [the plaintiff's] frequent, regular and proximate exposures to asbestos from Ford products while at Smith Motors" and "opined within a reasonable degree of medical certainty that the exposures at Smith Motors were sufficient, *in and of themselves*, to cause Rost's mesothelioma." *Id*. at 1048 (emphasis added).

Unlike in *Rost*, neither Markowitz nor Moline's testimony was tied to the circumstances of Mr. Juni's exposure; rather, they merely assumed that all of Mr. Juni's exposures were causative without regard to the frequency or regularity of those exposures. Thus, *Rost* cannot possibly support Plaintiffs' argument here that they have met the standards set forth by *Parker* and *Cornell*.

II. Sound Public Policy Supports Application of *Parker*'s Requirements Concerning Expert Causation Testimony Here.

For more than a decade, New York courts have applied *Parker*'s requirements governing general and specific causation testimony in toxic tort cases. Sound public policy supports the continued application of those requirements in this case.

A. ***Parker*'s requirement that a plaintiff proffer expert testimony based on a scientific expression of exposure is essential to ensuring that toxic tort defendants are not subject to liability for *de minimis* harms caused by their products.**

New York law has long recognized that, in order to justify an imposition of tort liability on a particular defendant, that defendant's tortious conduct must have been a substantial factor in bringing about a plaintiff's injury. *See* NY PJI § 2:70 (defining substantial factor causation). This requirement is designed to ensure that a defendant whose conduct has only a "trivial" impact on a plaintiff is not held liable for that plaintiff's injury. *Id.* (noting that, "to be substantial, [a cause] cannot be slight or trivial"). In toxic tort cases, this means that a plaintiff must provide reliable scientific evidence that exposure to a particular defendant's product was a substantial factor in bringing about his injury. *Sean R.*, 26 N.Y.3d at 808-09; *Cornell*, 22 N.Y.3d at 784; *Parker*, 7 N.Y.3d at 448. This Court has reaffirmed that basic principle on at least three separate occasions. *Sean R.*, 26 N.Y.3d at 808-09; *Cornell*, 22 N.Y.3d at 784; *Parker*, 7 N.Y.3d at 448.

If this Court were to relax these requirements and allow plaintiffs like the Junis to recover based on generic "cumulative exposure" testimony that fails to account for a particular plaintiff's exposure to a particular product, it would "render the substantial-factor test essentially meaningless" and "permit imposition of liability on the manufacturer of any asbestos-containing product with which a worker had the briefest of encounters on a single occasion." *Krik*, 870 F.3d at 677-

78. The result would be an imposition of absolute liability as to every company whose asbestos-containing product happened to cross paths with a future toxic tort plaintiff, regardless of how fleeting the exposure or how benign the asbestos-containing product. *Bostic*, 439 S.W.3d at 339 (noting that, if courts "were to adopt a less demanding standard for mesothelioma cases and accept that any exposure to asbestos is sufficient to establish liability, the result essentially would be not just strict liability but absolute liability against any company whose asbestos-containing product crossed paths with the plaintiff throughout his entire lifetime.").

Such an approach would be inconsistent with the universally acknowledged policy, noted in both the Restatement Second and Restatement Third of Torts, that a defendant should not be held liable for tortious conduct that has only a *de minimis* impact on a plaintiff. *See* Restatement (Second) of Torts § 433, Comment d (explaining that, in cases involving multiple causes from multiple actors, "[s]ome other event which is a contributing factor in producing the harm may have such a predominant effect in bringing it about as to make the effect of the actor's negligence insignificant and, therefore, to prevent it from being a substantial factor"); *see also* Restatement (Third) of Torts: Phys. & Emot. Harm § 36 ("When an actor's negligent conduct constitutes only a trivial contribution to a causal set … the harm is not within the scope of the actor's liability.").

Plaintiffs may argue, as they did below, that the long latency periods for asbestos-related diseases justify relaxation of causation standards in asbestos cases. This is wrong. As the First Department correctly noted, "there is no valid distinction to be made between the difficulty of establishing exposure to, say, benzene in gasoline and exposure to asbestos. In each type of matter, a foundation must be made to support an expert's conclusion regarding causation." (A10.) Both benzene and asbestos have long latency periods before symptoms of any exposure-related disease can manifest, and the Junis have failed to articulate how any difference between the latency period for asbestos-related diseases and the latency period in benzene-related diseases justifies a more lenient standard in asbestos cases. *See Eaton*, 12 J.L. & POL'Y 5 at 32 (noting that the latency for most cancers is 20 to 40 years, similar to asbestos). Indeed, no principled distinction exists.

B. Adherence to *Parker* and its progeny is particularly important in friction cases in light of the limited evidence linking exposure to friction products to asbestos-related disease.

Asbestos litigation is the longest-running mass tort in American history. *See* Lester Brickman, *Fraud and Abuse in Mesothelioma Litigation*, 88 Tulane L. Rev. 1071, 1075 (2014). Originally and for many years, asbestos litigation typically pitted a dusty trades worker against the manufacturer of an amphibole asbestos-containing insulation product. *See id.* As a result, the vast majority of the

manufacturers of asbestos-containing products in this country have gone bankrupt. *Id.* at 1075-76. Claims based on exposure to asbestos from products manufactured by these bankrupt entities are now resolved through a process administered by the bankruptcy trusts. *Id.* at 1076. This process guarantees substantial recoveries to all victims of mesothelioma who were exposed to products manufactured by these companies, and payments from these trusts have exceeded $30 billion. *Id.*

Despite this, in the search for more solvent defendants, manufacturers of chrysotile-containing gaskets or chrysotile-containing automotive friction products have become targets of asbestos filings. *See, e.g.*, *In re Garlock Sealing Technologies, LLC*, 504 B.R. 71, 81-82. Because the chrysotile contained in such products is far less potent than the amphibole asbestos contained in the insulation products manufactured by bankrupt entities, and because exposures to chrysotile in friction products generally are far less significant than exposures to asbestos from insulation products, experts like Markowitz and Moline have concocted the "cumulative exposure" theory of causation in order to ensnare any and every source of putative asbestos exposure without regard to how limited or remote exposure related to those products might be. *See* Mark A. Behrens & William L. Anderson, *The "Any Exposure" Theory: An Unsound Basis for Asbestos Causation and Expert Testimony*, 37 Sw. U. L. Rev. 479, 493 (2008) ("Through this

testimony, the any exposure experts are helping to extend the asbestos litigation to any entity that had any connection to asbestos.").

Science does not support this approach. As explained above, the overwhelming weight of the epidemiological evidence indicates that auto mechanics do not face an increased risk of mesothelioma. (*See* Statement of Facts, Section II, *supra*.) This is because the chrysotile asbestos contained in friction products is a fundamentally different substance than the amphibole asbestos contained in the products manufactured by traditional asbestos defendants. (*Id.*) The Junis' expert witnesses brush aside this distinction, treating all of Mr. Juni's alleged exposures to all types of "asbestos" generically as the same. (*See* Statement of Facts, Section III, *supra*.) This Court should not sanction that approach. The search for a solvent defendant cannot justify ignoring the available scientific evidence regarding the toxicity of chrysotile in friction products. As Judge Posner explained, "the courtroom is not the place for scientific guesswork, even of the inspired sort. Law lags science; it does not lead it." *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996) (Posner, J.).

III. Should This Court Reverse the Decisions Below, Ford Respectfully Requests Remand for Consideration of the Remaining Issues Raised in Ford's Post-Trial Brief.

Because the courts below agreed that Ford is entitled to judgment as a matter of law, they did not address the remaining arguments in Ford's post-trial

motion. (A11826.) Ford's additional arguments supported granting judgment as a matter of law or a new trial on the grounds that: (1) the recklessness instruction did not conform to New York law and the evidence was insufficient to support the jury's finding of recklessness, (2) the jury's allocation of fault was unsupportable, (3) certain evidence admitted in contravention of New York law significantly prejudiced Ford, and (4) consolidated trial of the *Juni* case with other cases was improper and prejudicial. (A11772-11783.) In the alternative, Ford argued that the jury's verdict was unreasonably excessive and not supported by the evidence such that remittitur was warranted. (A11783.)

Ford does not waive these arguments and respectfully requests that, if this Court holds that the courts below erred in granting judgment as a matter of law in favor of Ford (which, as explained above, it should not), this Court remand this case back to the Supreme Court for determination of the remaining issues raised in Ford's post-trial motion. The Junis appear to agree that this is the appropriate course of action. (*See* Appellants' Br. 56.)

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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CERTIFICATE OF COMPLIANCE

I hereby certify pursuant to 22 NYCRR § 500.13(c) that the foregoing brief was prepared on a computer.

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Dated: December 6, 2017

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