To Be Argued By:
ALANI GOLANSKI
Time Requested: 30 Minutes

APL-2017-00114
New York County Clerk's Index No. 190315/12

Court of Appeals

STATE OF NEW YORK

IN RE: NEW YORK CITY ASBESTOS LITIGATION.

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

Plaintiff-Appellant,

—against—

A.O. SMITH WATER PRODUCTS CO., AERCO INTERNATIONAL, INC., AGCO CORPORATION f/k/a and as Successor in interest to MASSEY-FERGUSON, INC., AIR & LIQUID SYSTEMS CORPORATION, as successor-by-merger to BUFFALO PUMPS, INC., AMCHEM PRODUCTS, INC., n/k/a RHONE POULENC AG COMPANY, n/k/a BAYER CROP SCIENCE INC., ARVINMERITOR, INC., Individually and as successor-in-interest to ROCKWELL AUTOMOTIVE, BMCE, INC. f/k/a UNITED CENTRIFUGAL PUMP, BOISE CASCADE CORPORATION, BORG-WARNER CORPORATION, by its successor-in-interest, BORG-WARNER MORSE TEC, INC., BW/IP, INC. and its wholly owned subsidiaries, CARLISLE CORPORATION, CATERPILLAR, INC., CBS CORPORATION, f/k/a VIACOM INC., successor by merger to CBS CORPORATION, f/k/a WESTINGHOUSE ELECTRIC CORPORATION, COURTER & COMPANY INCORPORATED, CRANE CO., CUMMINS ENGINE COMPANY, INC., DANA COMPANIES, LLC, DEERE & CO., DENTSPLY INTERNATIONAL, INC., Individually

(*Caption continued on inside cover*)

BRIEF FOR PLAINTIFF-APPELLANT

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September 28, 2017

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,

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, KENENDY 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.

STATEMENT PURSUANT TO RULE 500.13(a)

As of the date of the completion of this Brief, there is no related litigation pending before any court.

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Dates: New York, New York
September 28, 2017

WEITZ & LUXENBERG, P.C.

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Alani Golanski

STATEMENT OF JURISDICTION

This Court has jurisdiction to entertain this appeal by authority of CPLR 5602(a). This action originated in the Supreme Court, County of New York. The majority Decision and Order of the Appellate Division, First Department, affirmed a final judgment entered by the IAS court (Hon. Barbara Jaffe) upon the court's post-trial Decision and Order setting aside the jury's verdict and dismissing plaintiff's claims. The questions raised in this appeal are questions of law and are reviewable pursuant to CPLR 5501(b).

The judgment appealed from was entered June 3, 2015 [A-11827],[1] and plaintiff timely served and filed her Notice of Appeal on June 23, 2015 [A-11835]. The Appellate Division dismissed plaintiff's earlier appeal filed May 14, 2015 [A-11834], from the trial court's ruling as subsumed in the appeal from the judgment [A-13].

The Appellate Division's Decision and Order was entered February 28, 2017 [A-2]. Plaintiff, on March 29, 2017, filed her motion seeking reargument or, in the alternative, leave to appeal to this Court. By Order entered May 30, 2017, the Appellate Division denied the motion for reargument and granted leave to appeal to this Court [A-45-46].

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[1] Numbers in brackets following "A-" refer to pages in the Appendix for Plaintiff-Appellant.

STATEMENT OF ISSUES PRESENTED

1. In this personal injury and products liability action arising from Mr. Juni's tragic affliction with mesothelioma caused by his ultimately fatal exposures to carcinogenic asbestos-containing components supplied and/or installed by defendant Ford Motor Company ("Ford") on its vehicles, where plaintiff's causation experts' testimony and methodology comported in all respects with mainstream science and this Court's precedents, and where the evidence in this two-month trial established, *inter alia*, that:

– Mr. Juni, a mechanic who serviced a fleet of 500 Ford vehicles for twenty-five consecutive years, was daily exposed to substantial quantities of asbestos-laden dust released from new and used high-asbestos content brakes, clutches, and manifold and engine gaskets, including asbestos dust blown "all over the place" using "compressed air" blow-out methods Ford's own scientists deemed ultrahazardous [A-501, 657.4 to 657.5];

– Mr. Juni sustained slight asbestos exposures to any source other than the Ford vehicles [*see* A-21];

– mesothelioma is a "signature" disease "caused only by exposure to asbestos," as summarized by the trial court [A-11812 to 11813];

– as found in numerous quantitative scientific studies discussed by plaintiff's experts and in voluminous internal Ford communications, asbestos fiber levels

produced during the very sort of work engaged in by Mr. Juni were several hundred times above the OSHA regulatory exposure limit, representing "significant exposure" at levels "that we know" cause mesothelioma (general causation) [A-1010, 1095, 1208 to 1213, 1853, 2332];

– the *only* epidemiological study that patterned Mr. Juni's occupational experiences (as defendant's epidemiological expert conceded) showed a statistically significant greater-than-doubling of the relative risk of mesothelioma for vehicle mechanics occupationally exposed to asbestos [A-1447]; and

– plaintiff's esteemed causation expert testified that, to a reasonable degree of medical certainty, Mr. Juni's occupational exposures to visible asbestos dust from Ford brakes, clutches and gaskets, "viewed as a whole," substantially contributed to his mesothelioma (specific causation),

did the Appellate Division majority misconstrue this Court's precedents in concluding that the evidence was insufficient as a matter of law to support the jury's exposure and causation findings [A-7, 11819]?

2. In this latent injury case arising from plaintiff's affliction with mesothelioma manifesting decades following his injurious exposures to asbestos dust released from defendant's vehicles, and where:

– the trial court erroneously disregarded the quantitative testimony underlying

the experts' scientific recognition of Mr. Juni's "regular," "frequent," "excessive," and "extensive" exposure to Ford's dusty, high asbestos-content brakes, clutches and gaskets [A-11813, 11819]; and

– the Appellate Division majority erroneously ascribed dispositive weight to certain studies cited by defendant – including automotive industry-funded for-litigation meta-analyses – that Ford's own epidemiological expert admitted (a) were *all* entirely irrelevant to Mr. Juni's occupational experience, (b) *all* reported statistically insignificant results [A-1528], and (c) "were not designed" or "powered" to look at the causal link between exposure to asbestos-containing vehicle components and mesothelioma [A-1441 to 1442, 1489, 1505],

did the trial court and Appellate Division majority erroneously conclude that, notwithstanding the jury's careful review of the abundant quantitative as well as qualitative proof at trial, the verdict should be set aside?

PRELIMINARY STATEMENT

The principal issue in this appeal is whether the First Department majority erred in concluding that, notwithstanding all of the evidence fully establishing that Mr. Juni's continuous exposures to carcinogenic asbestos dust released from his work with a variety of asbestos-containing products in defendant Ford's vehicles over a full

twenty-five (25) year period substantially contributed to his mesothelioma, the jury's verdict was nevertheless utterly irrational in finding a causal link between those exposures and Mr. Juni's mesothelioma.

In undertaking its CPLR 4404(a) post-trial sufficiency assessment, the Appellate Division acknowledged with approval its prior decisions upholding the causation proofs in asbestos-related cases such as *Lustenring v. AC&S, Inc.*, 13 AD3d 69 (1st Dept 2004), *Penn v. Amchem Prods.*, 85 AD3d 475 (1st Dept 2011), and *In re New York City Asbestos Litig.: Marshall v. John Crane, Inc. ("Marshall")*, 28 AD3d 255 (1st Dept 2006), deeming them to have been noncontroversial rulings based on sufficient "evidence linking visible dust to the use of the particular defendant's product" [A-10 to 11].[2]

Yet the expert testimony and methodology applied in the present case was even stronger in certain ways, and more quantitatively exacting, than in those cases. Plaintiff's eminent general causation expert and public health scientist, Dr. Steven Markowitz, began by describing the method by which scientists trained in occupational medicine assess the cause of a worker's disease against the backdrop of that worker's lifetime of occupational hazards encountered [A-128 to 129]. His explanation tracked the methodology well-accepted in the scientific community and

[2] Like the present case, *Lustenring* arose from the plaintiff's affliction with mesothelioma caused by his exposure to fibers released from asbestos-containing gaskets. 13 AD3d at 70.

articulated in the *Reference Manual on Scientific Evidence*,[3] cited by this Court as the authoritative guide to mainstream science in toxic tort causation contexts.[4]

Upon this foundation, Dr. Markowitz testified to a series of studies conducted by the world's then-leading asbestos research facility, Mount Sinai, finding that asbestos fiber levels in the air from brake blowout work such as Mr. Juni's exceeded "30 fibers per cc" [cubic centimeter], being 300 times above the Occupational Safety and Health Administration ("OSHA") regulatory exposure limit [A-288].[5] He also testified about numerous additional studies, such as those performed by the National Institute for Occupational Safety and Health ("NIOSH") in 1980 focusing on the repair of asbestos-containing brake equipment at garages operated by the City of New York City Department of Sanitation, the Department of Transportation, and the New York Police Department, demonstrating time-weighted averages of asbestos-containing fibrous dusts several times above the OSHA level [A-354].[6]

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[3] *See* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 512-13, 587 (3d ed. 2011).

[4] *See Sean R. v. BMW of North America, LLC*, 26 NY3d 801, 812 (2016) (relying upon the *Reference Manual* as the source for discerning "generally accepted methodologies" for determination causation in toxic tort cases); *Cornell v. 360 West 541st Street Realty, LLC*, 22 NY3d 762, 783 (2014) (same).

[5] *Cf. Parker v. Mobil Oil Corp.*, 7 NY3d 434, 443 (2006) (crediting defense expert's citation to a NIOSH study of benzene exposure for service station employees showing the greatest level of exposure as 0.19 parts per million ("ppm") time-weighted average, "which is less than the 1 ppm occupational standard set by [OSHA]").

[6] A time-weighted average is used to calculate a worker's daily exposure to a hazardous (continued...)

Hence, the dissenting opinion below emphasized that Dr. Markowitz's testimony was:

> based on the "firmly established" and accepted knowledge that chrysotile asbestos causes mesothelioma, industrial hygiene studies that measured chrysotile asbestos among workers using friction products, case series of mesothelioma occurring among mechanics who work with friction products, evidence that persons who work with friction products in vehicle repairs develop nonmalignant asbestos-related disease, peer-reviewed studies examining the pertinent literature, and materials from various health and safety agencies and organizations.

[A-24 to 25].

Testimony proffered by plaintiff's other esteemed causation expert, Jacqueline Moline, an occupational medicine specialist, established that she adhered to the methodology described by Dr. Markowitz and set forth in the *Reference Manual*. She attested that "the relevant considerations" informing her methodology for assessing specific causation were "the amount of the exposure, the duration of an exposure, and the frequency of the exposure" [A-1150 to 1151]. She similarly testified to studies performed in the 1970s finding that the exposures to asbestos for workers engaged in work substantially similar to that of Mr. Juni "were at levels capable of causing disease," a "hundred thousand times greater [than] background

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[6](...continued)
substance (such as chemicals, dusts, fumes, mists, gases, or vapors, as well as asbestos fibers) or agent (such as occupational noise), averaged to an eight-hour workday, taking into account the average levels of the substance or agent and the time spent in the area. "This measurement can also be expressed in ppm-years." *Parker*, 7 NY3d at 443 n.1.

levels of exposure to asbestos," and far higher "than is even required for mesothelioma" [A-1208 to 1209].

Not only did the testimony of Drs. Markowitz and Moline establish general and specific causation, but Ford's own experts conceded the irrelevance and statistical insignificance of the studies upon which Ford relied in its attempt to create doubt with regard to the scientific causation proofs.[7] The proofs also established that, through the period of Mr. Juni's daily exposures, Ford's sophisticated industrial hygiene personnel well knew that work precisely such as that performed by Mr. Juni on new and used high asbestos-content brakes, clutches and gaskets resulted in heavy exposures to asbestos-laden dust.

Ford admitted, for example, in its internal documents and health bulletins directed only at its own employees, that brake repair work exactly like Mr. Juni's resulted in the heavy presence of "typical chrysotile asbestos fibers" [A-2333], that "[o]verexposure" to ultra-carcinogenic asbestos fibers "occurred when brakes and brake drums were cleaned using compressed air blowoff" [A-905 to 906, 2246, 2330], and that, "*[u]nder no circumstances shall compressed air or dry brushing be used for cleaning*" [A-

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[7] *E.g.*, A-1442 (Ford's epidemiological witness conceding that she was "unaware of whether any of the studies [relied upon by Ford] did a power calculation to detect a risk of mesothelioma in people exposed to asbestos brakes and clutches"); A-1493 (same witness admitting, with regard to her own study heavily relied upon by Ford, "It's possible that I had no vehicle mechanics at all in this study," and hence no study or control subjects whatsoever who "actually worked with asbestos brakes," clutches or gaskets "at all").

2104 (Ford's emphasis)]. Tragically, while conveying this cautionary information to its own employees, Ford recklessly failed to warn workers such as Mr. Juni, as the jury found [A-11789].

In this regard, admitted in evidence to show Ford's notice of its products' hazards during Mr. Juni's exposure period was the Environmental Protection Agency's ("EPA") 1986 publication *Guidance for Preventing Asbestos Disease Among Auto Mechanics* [A-1903, 2077]. Citing studies by numerous researchers, including the NIOSH-OSHA Asbestos Work Group and the National Research Council [A-2089], the EPA therein reported that:

> [m]illions of asbestos fibers can be released during brake and clutch servicing. Grinding and beveling friction products can cause even higher exposures. . . . When you see a dust cloud during brake work, you are seeing clumps containing thousands of fibers. Most of the smaller fibers will not show up with the methods commonly used for measuring asbestos levels in the air . . . . Asbestos released into the air lingers around a garage long after a brake job is done . . . . Mesothelioma is a type of fatal cancer of the lining of the chest or abdominal cavity. It can be caused by very low exposures to asbestos. This cancer has occurred among brake mechanics, their wives, and their children.

[A-2078 to 2079 (omitting study citations)].

In his own testimony, Mr. Juni repeatedly recalled his exposures to dust released from Ford's asbestos-filled brakes, clutches and gaskets [*e.g.*, A-413, 419, 426-27, 442-43, 491-92, 657.4 to 657.7]. Desperate to avoid the legal significance of this testimony, Ford persisted in claiming, in its post-trial and appellate briefing, that

the proofs should be deemed insufficient as a matter of law unless Mr. Juni were able to account *precisely* for each brake, clutch and gasket "he encountered" [Ford's App Div. Brief (filed Apr. 20, 2016) ("D/B"), at 1 ("how many"), 5 ("how many"), 7 ("how often"), 8 ("how often")].

Ford similarly attempted to fault plaintiff's causation experts for not supplying the precise numerical quantifications that Mr. Juni naturally could not provide, and hence that were not available to the experts some forty years later [D/B, at 10, 36]. At the same time, Ford asked the Appellate Division to ignore the compelling quantification evidence that *is* included in the record, introduced via expert testimony and via Ford's trove of internal scientific documents [A-2077 to 2360].

Unfortunately, the majority panel erred in overlooking all of this evidence in its Rule 4404(a) sufficiency exercise. The majority misunderstood well-accepted, mainstream science when it incorrectly concluded that "concessions made by both of plaintiff's experts so undermined their assertions of causation as to render those assertions groundless or unsupported" [A-7]. This conclusion critically misapprehended the proofs, and improperly viewed the testimony in the light most favorable to the losing party at trial by virtue of ignoring the experts' testimony in full. The Decision overlooked the fact that, in reality and in context, plaintiff's experts did not make any such concessions.

The majority panel also gravely erred in misconstruing plaintiff's experts'

testimony to "assert[] that even a single exposure to asbestos can be treated as contributing to causing an asbestos-related disease" [A-11]. *The testimony was exactly the opposite.* Indeed, when Ford attempted to foist the straw-man "single exposure" misinterpretation upon plaintiff's experts during cross-examination, they flatly rejected the suggestion: Dr. Markowitz explained that the mainstream scientific approach he applies does not, under any circumstances, mean that "any exposure" is deemed a causative factor [A-285]; Dr. Moline similarly *flatly rejected* counsel's notion that she somehow "belie[ved] each and every one of those [exposures] would have constituted an independent significant cause of [Mr. Juni's] disease" [A-1154]. They explained that, quite the contrary, the "period of time" of an individual's exposures to a defendant's asbestos-containing products is critical, and a causal assessment is based on "adding up all the individual exposures . . . viewed as a whole" [A-127 to 128, 1154].[8]

Accordingly, the dissenting opinion explained that the majority "misapplied the standard for review for legal sufficiency, and misapplied the law concerning general and specific causation in asbestos cases" [A-20]. All of the evidence abundantly established causation, the experts' methodology adhered to this Court's guidance in *Parker* and *Cornell* and comported with *Sean R.*, and this Court should reject Ford's

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[8] *E.g.*, Hirotaka Nagai, *et al., Asbestos surface provides a niche for oxidative modification*, 102 CANCER SCI. 2118, 2118 (2011) ("both mesothelial cell injury and persistent macrophage activation are thought to be essential, if not sufficient, for mesotheliomagenesis").

effort to depart from those guiding precedents.

STATEMENT OF THE CASE

A. MR. JUNI'S EXPOSURES

During three summers while in his early twenties, Arthur Juni did outdoor work clearing high tension lines of vegetation [A-390 to 391]. To retrieve his truck, he would walk through a powerhouse machine shop – which was as short in length as the office in which he was being deposed – and observed asbestos materials on the floor and on pipes [A-392 to 394]. Mr. Juni next worked as a courier for a year, traveling from depot to depot and only occasionally entering a power plant [A-398 to 401]. He explained that, at those moments, he did not walk "through the powerhouse"; he "just went to the offices, that's it" [A-402].

Mr. Juni began working as an auto mechanic for Orange & Rockland Utilities in 1964, first at the utility's Nyack garage and then, beginning in 1966, at its Spring Valley garage [A-404 to 405, 423]. From 1964 until the Safety Department issued respirators in 1988 – hence for a twenty-five (25) year period [A- 447] – Mr. Juni worked with high asbestos-content products in Ford vehicles, and was daily exposed to dusts emanating from new and used asbestos-containing brakes, new and used asbestos-containing clutches, and new and used asbestos gaskets. These asbestos components were installed in the hundreds upon hundreds of Ford vehicles Mr. Juni

serviced [A-409 and *passim*; *see* A-11791]. As he noted, "we were a Ford fleet, so most of the vehicles were all Ford" [A-509].

Mr. Juni began his tenure at the Nyack garage as a third-class mechanic [A-405]. First-class mechanics performed brake jobs beside him on Ford trucks and Ford service vans, and installed new Ford brake shoes, all of which Mr. Juni identified as a source of his asbestos exposure [A-407 to 410]. Assisting them, Mr. Juni would "access the [brake] drums" [A-411]. "[Y[ou took that drum off, and . . . there's brake dust in there [that] went on the ground [that he] swept up later on" [A-413, 559]. Working on "[a]bout five" trucks at a time, the mechanics would then *grind and contour* the new Ford brake shoes "to the brake drum so you had a better fit" [A-410 to 413].

Mr. Juni was further exposed when he "assisted" the first-class mechanics in performing clutch work on a variety of Ford trucks [A-412, 415-417, 420]. After lowering the transmission, "[o]nce you got the bell housing off, there was always clutch dust" [A-419].

Orange & Rockland transferred Mr. Juni, now a second-class mechanic, to its Spring Valley garage in 1966 [A-423 to 424]. This was a larger facility, also "a Ford shop," with nine bays, and plaintiff helped service a fleet of 500 vehicles [A-423, 428, 433]. He was daily exposed to asbestos dust there, including when "[y]ou took the brake dust inside the drums and you just dumped the drums on the floor and that

brake dust . . . got cleaned up, with a broom" [A-427]. Plaintiff performed this work "every week, because the amount of trucks we had. . ., [e]verything right from the vans up to the F-350s" [A-428], which was a large Ford "super duty" pick-up truck.

When Mr. Juni became a first-class mechanic in the late 1960s, and then a foreman about 1970, he continued to work nearly exclusively on Ford vehicles [A-434, 445, 450]. This work also exposed him to asbestos-containing dusts from high asbestos-content brakes, clutches and gaskets. "[L]ike I said before," for instance, "you took the brake drums off; . . . you dumped it on the ground" [A-432]. Mr. Juni also installed new Ford asbestos-containing brakes at Spring Valley, as well as removing the used, worn and dusty Ford brakes [A-451 to 452]. He testified that he "breathed" asbestos dust both upon opening the packages of "new asbestos brake[s]" and when he "had to scuff it up with sandpaper. . . . I put it in the air [and] breathe[d] that" [A-657.6 to 657.7].

Mr. Juni was further exposed to asbestos while "perform[ing] clutch jobs as a first-class mechanic" [A-438 to 439, 447]. He recalled doing these clutch jobs on Ford's "C-8000" trucks as "a daily occurrence" [A-454 to 455, 516], and "almost weekly" would do this clutch work on "1979 C-8000 Ford cabovers. . . . They were bucket trucks" [A-441 to 442]. In this four-hour long procedure, "you would take off the bell housing, which contained the asbestos, because it would fly off the flywheel and the clutch disc to the exterior of the housing" [A-442 to 443]. "All [of the]

clutches contained asbestos" [A-652], and were manufactured by Ford [A-444]. Mr. Juni was exposed to asbestos-containing brake and clutch dust while performing these jobs "two to three times a week" [A-448].

Mr. Juni's exposure testimony included the following:

> Question: Did you ever have to do anything to the clutches before you installed them?
>
> Answer: Well, yeah, sometimes you had to scuff them.
>
> Question: What, if anything, happened to the air when you did that?
>
> Answer: I got it in the air.
>
> Question: Did you breathe that dust, too?
>
> Answer: I breathe it.
>
> Question: What about in the box from the clutch, was there any dust associated with that?
>
> Answer: Yes.
>
> Question: Did you breathe that dust, too?
>
> Answer: Yes.

[A-657.7].

Mr. Juni also described his occasional work replacing clutches on "an International backhoe" as being similar to, but on a smaller scale than, his regular work on the Ford clutches [A-490 to 491]. In this work, "you would take off the bell housing which usually contains asbestos fibers from the clutch spinning . . . It was

like a dirty dust" [A-491 to 492].

Additionally, Mr. Juni assisted his crew in replacing Ford's manifold gaskets [A-449, 453]. These jobs would take "[a]n hour and a half, two hours" [A-452]. He personally replaced gaskets in "Ford C8000 cab-over bucket truck[s]" [A-496], as follows:

> when you took [the Ford engine] apart, there was your gasket. There was an adhesive on it. It kept it to the metal parts. You had to scrape it, okay. . . and you put them in an electric drill and then you spun it. Oh, it was like a Brillo pad, a little Brillo pad . . . . You spun it up and you would go along, clean it. Then when you found the stuff all over the place, you took your air gun and (making noises) or you went and got the vacuum.

[A-501].

Moreover, when "replacing the intake manifold gasket itself, . . . the majority of that job involved removing the old gasket and cleaning up the surface" [A-618]. Mr. Juni emphasized that, "whatever the gasket was, there was asbestos involved in it" [A-621].

Wholly disregarded by the Appellate Division majority, but noted in the dissent [A-21], Mr. Juni and those working near him "use[d] compressed air to . . . clean off gasket material" and also to clean out clutches [A-657.4]. At the end of the day, "you generally used a broom. . . . You pushed the stuff up into the air" [A-657.5]. His testimony continued as follows:

Question: What stuff was pushed up into the air?

Answer: The asbestos on the brakes, the clutches and the gaskets.

Question: Did you breathe this material, this dust?

Answer: Yes.

[A-657.5].

In sum, Mr. Juni was injuriously exposed to ultra-carcinogenic dusts and debris released from Ford's new and used asbestos brakes, new and used asbestos clutch housings, new and ripped-out engine and manifold asbestos gaskets, along with dusts created by his constant use of compressed air blow-out of asbestos dusts from the Ford components, as well as his unprotected clean-up of asbestos dusts released during the work day from all the Ford components. Asbestos exposure was the only possible cause of his mesothelioma, and compared to Mr. Juni's twenty-five years of Ford-related exposures, his exposures to non-Ford asbestos-containing products were fleeting and short-lived.

B. FORD'S DOCUMENTARY ADMISSIONS

Tragically, Ford possessed conclusive scientific data that mechanics working with asbestos-containing products, precisely as Mr. Juni did, were being "overexposed" to ultra-carcinogenic asbestos fibers released from Ford's high asbestos-content brakes, clutches and gaskets, yet Ford failed to warn those workers. The jury received numerous internal documents wherein Ford admitted that, based

on the scientific, medical and industrial hygiene materials known to it during Mr. Juni's exposure period, Ford associated vehicle-related work during which the "recognized human carcinogen" asbestos is "handled in any way which causes dust to become airborne" [A-2098], with extreme risks of cancer and mesothelioma.

Ford Trial Exhibit 1, for example, was an internal Ford memorandum dated November 8, 1973, from Ford's "Industrial Hygienist Section" to its Chassis Division Safety Engineer [A-705, 2094]. The memorandum discussed an internal study focusing solely upon brake repair work. Disregarding all of the asbestos fibers less than five microns in length,[9] the study nevertheless quantified airborne fiber concentrations many times greater than the current OSHA regulatory limit (0.1 fiber per cc), including from brake sanding operations (1.3 fibers/cc, or 13 times greater than the current OSHA limit) [A-2096]. Given the asbestos dust found in the air and known to accumulate on the floor for later cleaning, Ford's Industrial Hygienist recommended that Ford's own brake operations be enclosed to ensure its workers' safety [A-711, 2095], a caution not communicated by Ford to vehicle mechanics such

[9] As Dr. Markowitz explained, pursuant to OSHA regulatory requirements, asbestos fiber counts begin at five microns as "a practical matter" based on the capabilities of the light microscope, this being "the affordable, accessible way of measuring asbestos fibers"; accordingly, "[f]ibers that are smaller than 5 microns in length . . . will be in the air, the workers will breathe those fibers in, but they won't be counted under OSHA rules because the light microscope can't detect them" [A-123 to 124]. Such ultrahazardous asbestos fibers have, however, been detected by use of the more expensive and more exacting electron microscopy used in certain studies. *See, e.g., infra* text accompanying note 10.

as Mr. Juni.

Ford's designated corporate representative, Matthew Fyie [A-658 to 659], admitted that Ford specified sanding "the surface of the brake lining to get rid of the glazing," and that Ford knew "that the asbestos dust from the brakes is not cleaned up after each sanding operation. [Rather, t]he dust is allowed to accumulate for about one week" [A-709 to 711]. When Mr. Juni took new brakes out of their dusty boxes, "you had to take sand paper and rub them down a little bit to get the glaze off" [A-561].

In July 1973, Paul Toth, the Supervisor of Ford's Industrial Hygiene Section (Ford Trial Exhibit 7 – [A-2246]), attended the "Occupational Exposures to Asbestos Dust From Brake Linings Meeting" sponsored by NIOSH and the Department of Health, Education, and Welfare, the Center for Disease Control ("CDC"). The minutes of the July 21, 1973 meeting (Ford Trial Exhibit 31 [R. 899, 2333]) demonstrate that, upon learning that brake repair workers were vulnerable to developing mesothelioma, Dr. Irving J. Selikoff, the world's leading researcher into asbestos-related disease, had initiated studies *in consultation with defendant Ford* and others "to determine if brake lining workers were exposed to significant levels of asbestos" [A-2333].

The minutes reflect the outcome of such studies, particularly the heavy presence of "typical chrysotile asbestos fibers" [A-2333]. The minutes continue that,

based on:

> studies of asbestos dust exposure in automobile repair shops in New York City. . ., [f]iber counts, using the OSHA method, were from 6 to 30/ml at 3-5 feet from brake drums cleaned with compressed air. At 5-10 feet, counts ranged from 2 to 4 ml; at 10-20 feet 0.4 to 4.8 ml. . . . Eighty to 99% of the fibers as observed by electron microscopy were less than 0.4 microns in length, indicating a much higher exposure to asbestos fibers than indicated by optical microscopy.

[A-2334, 907].[10] Hence, studies undertaken in consultation with defendant Ford during the early portions of Mr. Juni's exposure period quantified fiber counts at *300 times* the OSHA standard for precisely the sort of work Mr. Juni described.

By its internal memorandum dated August 3, 1973, authored by Mr. Toth, Ford further admitted that "[r]ecent industrial hygiene studies have demonstrated *overexposure to asbestos fiber* in air during certain vehicle brake rebuilding and inspection operations. Overexposure occurred when brakes and brake drums were cleaned using compressed air blowoff" [A-2246 (emphasis added), 905-906].[11] The memorandum recommends warnings and protective measures, which Ford conveyed internally to its staff, but not to plaintiff [A-2247; *accord* A-2325 (Ford Trial Exhibit 24); A-2330 (Ford Trial Exhibit 28)].

Shortly after Mr. Toth issued his internal memorandum, Ford's Industrial

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[10] One fiber per millileter ("ml") is equivalent to 1 fiber per cubic centimeter [A-266].

[11] *See also* Ford Trial Exhibit 28 (internal memorandum "Controlling Asbestos Exposure" (Nov. 16, 1973)) ("Cleaning or 'dusting' of brakes or brake components by means of compressed air is *to be discontinued and prohibited as soon as possible*") [A-2330 (emphasis added), 888].

Hygiene Section internally issued, on August 20, 1973, the results of its asbestos air sampling study [A-2328 (Ford Trial Exhibit 27)]. Noting the then-existing 5.0 fiber/cc OSHA regulatory limit – which is fifty (50) times higher than the current OSHA standard – Ford's internal study found that even that limit was exceeded during the "cutting and filing" of brake material [A-2328 to 2329].

In a further internal memorandum by Mr. Toth, dated April 23, 1975, concerning "Exposure of Garage Mechanics to Brake Dust" (Ford Trial Exhibit 29 [A-2332]), Ford admits that its own studies of "the exposure of Company employees to brake dust during brake relining operations in our service garages" revealed "exposures in excess of limits established in the OSHA standards on asbestos dusts" [A-2332, 898]. Again, the OSHA regulatory limit in 1975 was 5.0 fibers/cc, fifty times (50) what it would later become.[12]

Importantly as well, in April 1977, Ford internally distributed a "Key Issue Report" (Ford Trial Exhibit 21 [A-2301]), noting that OSHA was then expected to pass an asbestos regulatory limit value of 0.5 fibers/cc (five times the current limit) [A-2301]. Critically, Ford therein admitted that its own suppliers of asbestos-containing automotive brake, clutch and gasket components "have already expressed

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[12] *See generally Asbestos Information Ass'n/North America v. Occupational Safety and Health Admin.*, 727 F.2d 415, 418-19 (5th Cir. 1984) ("OSHA bases its conclusion that a grave danger exists on quantitative risk assessments, which are mathematical extrapolations, of the likelihood of contracting an asbestos-related disease at various levels of exposure to asbestos particles").

an inability to meet the proposed OSHA limit of 0.5 fibers/cc" [A-2301, 856-857], thereby acknowledging that such limit was being exceeded in the field.

By way of Ford Trial Exhibit 5 [A-2150, 751], the jury received Ford's 1980 admission that "[a]sbestos is found in a variety of automotive components, and thus many occupational exposures are possible. *The most critical exposure occurs during brake and clutch repair*" [A-2163, 792-793 (emphasis added)].

In August 1983, a Ford Employee Health Services bulletin (Ford Trial Exhibit 3 [A-2100, 725]) warned that Ford's own employees were at risk of sustaining fatal asbestos-related injuries, including cancer and mesothelioma, from such operations as clutch and brake work, and admitted that "the fibers are nearly indestructible, and a potential health risk arises *whenever asbestos fibers are set free*, resulting in airborne asbestos dust" [A-2100, 728 (emphasis added)].[13]

A second 1983 Ford bulletin (Ford Trial Exhibit 4 [A-2104, 742]) instructed that certain dust avoidance measures should be strictly followed to protect Ford employees by "minimiz[ing] asbestos dust exposures during brake and clutch

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[13] Unfortunately, in its underlying decision setting aside the jury's verdict, the trial court misapprehended the evidence to mean that, once initially embedded in a resin during the manufacturing process, asbestos fibers are forever thereafter rendered innocuous [A-11794]. The court wrongly inferred, contrary to the rest of the evidence, that the fibers would permanently be nonrespirable, regardless of handling or the generation of dust. As Dr. Markowitz testified, "[t]he chrysotile in friction products, when it's manipulated, becomes airborne and the worker breathes in that asbestos dust, th[ey] develop lung cancer as a result of that exposure" [A-112; *see also* A-2098 (Ford admitting the same)].

servicing of trucks, tractors, trailers, automobiles" and other vehicles [A-2104, 744]. The bulletin instructed that, "*[u]nder no circumstances shall compressed air or dry brushing be used for cleaning*" [A-2104 (Ford's emphasis)]. This warning was not communicated to workers such as Mr. Juni, however. Consequently, Mr. Juni and his co-workers naively used compressed air to blow out the asbestos dust formed in and around brakes, clutches and gaskets [A-657.3 to 657.4].

C. EXPERT TESTIMONY AT TRIAL

1. General Causation

The scientists' medical concerns and findings, and Ford's admissions about fiber release and worker exposure, were further borne out at trial through the testimony of both parties' expert witnesses. The testimony demonstrated that asbestos fibers released from compressed air blow-out, new and used gaskets, new and used clutches, sanding and beveling of new high asbestos-content brake linings and used brake blow-out and repair, are as carcinogenic and toxic as the asbestos fibers released from any other asbestos-containing products.

Dr. Markowitz testified to his decades of treating and studying thousands of "patients with asbestos-related diseases or exposures" [A-63].[14] He first described

[14] Dr. Markowitz is an authoritative spokesperson for the mainstream scientific approach to causation. His active memberships include the American Public Health Association (continued...)

lung function and the respiratory system to the jury in great detail, including the differences between healthy and diseased pleural tissue enveloping the lungs [A-71 to 76]. Ordinarily safeguarding the body from intrusion by foreign particles are the body's defense mechanisms, including macrophage cells, which "defeat or engulf these foreign particles, dust, bacteria, viruses and the like, when they get actually into the lung tissue itself" [A-77 to 79].

Asbestos fibers "can stay in the air for quite some period of time" [A-83], are not individually detectable but are "seen in clusters . . . as part of dust," and have "the ability to bypass the defense mechanisms" [A-81 to 82]. The fibers "get into the lung tissue, provoke inflammation, and eventually that inflammation develops into, for some people, into scars" [A-84]. Moreover, "malignant mesothelioma is a cancer that starts in the pleura when it occurs in the chest. So there's pleural tissue, this normally very thin Saran wrap layer of tissue that can have scarring produced by asbestos, it can also separately develop cancer due to asbestos, and that cancer, when it starts in the pleura, is a mesothelioma" [A-87].

Approximately 95 percent of all the asbestos used commercially in the United

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[14](...continued)
and the Collegium Ramazzini, an authoritative international entity consisting of merely 180 invitation-only members, whose scientific and medical occupational health findings are deemed authoritative by governments and regulatory entities worldwide [A-67 to 68]. *See In re Methyl Tertiary Butyl Ether (MTBE) Products Liability Litig.: Tonneson v. Sunoco, Inc.*, № 03 Civ. 8284, 2008 WL 2607852, at *2 (S.D.N.Y. July 1, 2008) ("the Collegium Ramazzini [is] an organization comprised of scientists and physicians who are highly regarded in their field").

States has been the chrysotile type of asbestos [A-82].[15] Dr. Markowitz explained that, based on numerous human, animal, epidemiological and other types of studies – including pathology showing chrysotile fibers in the pleural tissue of mesothelioma victims – it is clear that "chrysotile causes mesothelioma" [A-90 to 95].[16] Unanimously providing the foundation for his conclusion – "firmly established and accepted" in mainstream science [A-108 to 109] – that chrysotile exposures cause mesothelioma, are the findings of such national and international medical and scientific organizations as "[t]he World Health Organization, the International Agency for Research on Cancer, the National Cancer Institute, the Environmental Protection Agency, the Occupational Safety and Health Administration, the Agency for Toxic Substances and Disease Registry, the World Trade Organization" [A-96 to 100]. Ford's epidemiological witness, Mary Jane Teta, conceded the same [A-1430].

Dr. Markowitz next turned his attention to studies establishing that vehicle mechanics working with asbestos-containing brakes, clutches and gaskets were

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[15] The other type is called "amphibole" [*see* A-89].

[16] *See* Dep't of Labor: Occupational Safety and Health Admin., *Occupational Exposure to Asbestos* (29 CFR Parts 1910, 1915, & 1926), 59 Fed. Reg. 40964, 40979 (Aug. 10, 1994) ("OSHA believes that the evidence in the record supports similar potency for chrysotile and amphiboles"), *referenced in Sec'y of Labor v. ConocoPhillips Bayway Refinery*, 654 F.3d 472, 474 (3d Cir. 2011). *See generally Berger v. Amchem Prods.*, 13 Misc.3d 335, 346 (Sup. Ct., NY County, 2006) (Hon. Helen E. Freedman) ("Chrysotile asbestos fibers have been found embedded in the pleural tissue of brake mechanics who died of mesothelioma"); *Relyea v. Borg Warner Corp.*, № 12 cv 3564 (DLC), 2015 WL 5567034, at *2 (S.D.N.Y. Sept. 22, 2015) (referencing "peer-reviewed scientific literature linking the alleged cancer-causing properties of asbestos or chrysotile to the alleged cancer-causing properties of brake dust").

exposed to ultra-carcinogenic levels of asbestos fibers. He explained that "there have been industrial hygiene studies that have measured chrysotile asbestos among workers who are using friction products and I rely on those studies," as well as on "individual reports and also group reports – called 'case series' -- of malignant mesothelioma occurring among garage mechanics or people who work with friction products in the vehicle-repair setting" [A-109]. Dr. Markowitz also relied on studies establishing the occurrence of asbestos-related lung tissue scarring among workers "repairing brakes, removing engine gaskets, working with clutches," as well as peer-reviewed studies and agency findings linking such work to asbestos-related disease [A-110].

Dr. Markowitz further testified to his reliance upon the epidemiological study lead-authored by Dr. Roelofs in 2013 "that showed an increased risk of mesothelioma in garage mechanics or auto repair workers or the like" [A-175]. Indeed, Ford's own epidemiological witness conceded that the Roelofs study was a "case-control study that was statistically significant" [A-1447], "look[ed] at the question of specifically brake mechanics and mesothelioma" [A-1551], and found that "the relative risk is greater than two, to a 95 percent confidence level" – which, as Dr. Teta admitted, means that "chance was ruled out as the explanation" [A-1447, 1558-1559]. Dr. Teta further conceded that "the range of exposures . . . in the Roelof study is closer to Mr. Juni's actual situation when he was exposed to asbestos brakes, clutches, and engine gaskets than" the other, prior studies relied upon by Ford at trial,

including defense-funded meta-analyses [A- 1554; *see* A-1251 ("funded by the automobile companies"), 1364].[17]

Additionally, Dr. Markowitz discussed his reliance upon a finding reported by Dr. Ladin in an article titled, "Lung Cancer and Mesothelioma among Male Automobile Mechanics," to the effect that, in the 1970s, "episodic peak concentrations of up to 16 fibers per milliliter were reported during blowing off of loose dust" during brake repair work [A-264 to 266]. He further testified that "a series of studies out of Mount Sinai in the mid-1970s clearly demonstrated that there were very high levels of asbestos in the air" resulting from brake work. "When they did blowout, for instance, there was a range that went up to over 30 fibers per cc," being *300 times* above the current OSHA exposure limit [A-288]. Dr. Markowitz also described the OSHA "short-term exposure" limit ("STEL"), being "1 fiber per cc over a 30-minute period," and explained that a number of studies found very high short-term exposures sixteen times over that limit for asbestos-related clutch and brake repair work, signifying an "excess or increased risk of lung cancer and

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[17] *See generally* David S. Egilman, *et al., Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability*, 11 INT'L J. OCCUP. ENVIRON. HEALTH 360, 360 (2005) ("To support their arguments, the automobile manufacturers have hired consultants to reanalyze previously published hygiene and epidemiologic studies. Some manufacturers of asbestos-lined brakes – including GM, Ford, Daimler-Chrysler, and Bendix – . . . have spent millions of dollars to generate these epidemiologic studies in order to refute claims of causation and thereby avoid compensation payments to victims and their families. [Such] papers demonstrate the use of two practices associated with the industry's involvement in scientific research: the redefinition of scientific criteria for the determination of cause-effect relationships and the manipulation of scientific data").

mesothelioma" [A-347 to 349].[18]

Dr. Markowitz further discussed a series of NIOSH studies performed in 1980 that focused on the repair of asbestos-containing brake equipment at garages operated by the City of New York City Department of Sanitation, the Department of Transportation, and the New York Police Department [A-349 to 351]. For the Sanitation Department study, "the fiber dust concentration found on the sample during active brake work was .33 fibers per cc," and a time-weighted average "of .21 fibers per cc was found for the total sampling time of the brake mechanic" [A-353]. For the Transportation Department study, "the time-weighted average of fibrous dust concentrations for personal samples . . . were .23, .28, and .24 fibers per cc" [A-354]. And the corresponding time-weighted exposure concentrations in the Police Department study "were .20, .19 and .30 fibers per cc," all such outcomes being "two to three times the current OSHA level" [A-354 to 355].

Further attesting to the significance of those findings, Dr. Markowitz explained that:

> they clearly represent significant exposures to asbestos, both the peak level, meaning the short-term level when a particular task is done, but also when they averaged it out a certain amount of time, those are

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[18] "STEL" is a term used in occupational health, industrial hygiene and toxicology. Some of the PELs (permissible exposure limits) are listed as ceiling values – concentrations above which a worker should never be exposed, or STELs – average concentrations which should not be exceeded over a 15 minute time period. *See* https://www.osha.gov/dsg/annotated-pels/tablez-2.html.

> elevated levels. There have been – other studies have shown higher levels than that, but even these levels in the 1980 showed appreciable levels of asbestos, levels that are not safe and levels that certainly can contribute to cancer, lung cancer and mesothelioma, if that brake mechanic eventually develops one of those diseases.

[A-355 to 356].

The evidence further showed that, during the process of braking, asbestos brake linings create dust and debris that accumulate within the brake drums. The process of braking generates tremendous heat within those brake drums (with temperatures exceeding 600° centigrade (Celsius), or over 1,100° Fahrenheit [A-274]). This extremely high heat, which occurs only during the actual process of braking, converts a percentage of the asbestos in the brake linings to another mineral known as forsterite. [A-359]. The conversion in brake linings from asbestos to forsterite is limited solely and exclusively to the dust generated during the braking process, and thus has no relevance to the dust emanating from *new brakes*. Nor is there any dispute that the asbestos content of the *clutch plates* and *gaskets* installed in the Ford vehicles, and released into the air upon installation, repair, and removal, was not converted in *any* amount into forsterite, but instead remained fully intact.

Just with regard to the used brakes, mechanics are exposed to this "reduced asbestos" brake dust within brake drums during the process of removing the old, worn out brakes. No other manipulation of, or process involving, asbestos brakes (or any other asbestos component) engenders the creation of forsterite. Accordingly, as

just stated, the use or manufacture of asbestos clutches and asbestos gaskets, and of new asbestos brakes, has nothing to do with the creation of forsterite. Therefore, all of Mr. Juni's additional exposures to asbestos from his work with Ford asbestos products – *i.e.*, upon sanding, filing or grinding new asbestos brakes, upon removing and replacing asbestos clutches, and scraping and replacing asbestos gaskets – had nothing whatsoever to do with a "conversion" to forsterite, and there was absolutely no evidence to the contrary.[19]

Yet, the evidence established that even exposures to dusts released from used asbestos-containing brakes remained fully carcinogenic and ultrahazardous. In this regard, Dr. Markowitz discussed a 1982 NIOSH garage study concerning used brake dust that employed a "Transmission Electron Microscope ["TEM"], [a] much more sensitive way of looking at fibers and you can see much thinner fibers and much smaller fibers with an electron microscope" [A-358 to 359].[20] Dr. Markowitz explained that, when samples were analyzed most precisely by use of TEM, "they're finding 30 percent of the fibers as chrysotile . . ., with the remaining fibers being categorized as forsterite (20 percent) or unknown (50 percent)" [A-359]. Hence, as noted above, Dr. Markowitz elaborated by discussing studies demonstrating that, in

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[19] Unfortunately, the trial court misapprehended all of this testimony, and erroneously leaped to an insupportable inference that asbestos contained within every other type of asbestos-containing vehicle-related product "becomes degraded in the manufacturing process" [A-11813].

[20] *See supra* note 9.

relation to the current OSHA exposure limit of .1 fiber per milliliter, episodic peak concentrations a full *160 times above this level* "were reported during blowing off of loose dust" during brake repair work [A-264 to 266], precisely the activity in which Mr. Juni engaged while working on the Ford fleets.

With further regard to his methodology, Dr. Markowitz explained that, in mainstream medical practice, when a patient presents with mesothelioma, "as an occupational medicine physician, we go back and say, 'Okay. Did you have this exposure? Where did you have this exposure? Over what period of time?' . . . because it's the cumulative exposure that matters" [A-127 to 128].[21]

Ford's counsel nevertheless attempted to discredit Dr. Markowitz's mainstream and well-accepted scientific view that causation is a function of cumulative exposures, by misleadingly asking, "So do I understand your opinion that any exposure to any asbestos can increase the risk of disease?" [A-285]. Overlooked by the Appellate Division majority, Dr. Markowitz *flatly rejected* this suggestion, explaining that the mainstream scientific approach he applies does not, under any

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[21] As stated throughout, in her *specific* causation testimony, Dr. Moline also considered the quantitative exposure and fiber release studies in vehicle maintenance settings comparable to Mr. Juni's work experience [A-1208 to 1209], including his ultra-carcinogenic Ford-related exposure levels to asbestos fibers at "one hundred thousand times greater" than ambient air [A-1209], the amount, duration, and frequency of his exposures to Ford's asbestos components over the course of his work life [A-1150], Mr. Juni's medical records, the particularized characteristics of the chrysotile asbestos and the human lung by virtue of which "[c]hrysotile tends to move more to the pleura" whereat mesothelioma tumors develop [A-1210], and so forth.

circumstances, mean that "any exposure" is deemed a causative factor [A-285]. The "period of time" of a plaintiff's exposures to a defendant's asbestos-containing product is critical, and a causal assessment is based on the "cumulative exposure" to the particular defendant's products "viewed as a whole" and "adding up all the individual exposures that a worker might have had" [A-127 to 128].

2. Specific Causation

Dr. Moline similarly testified to "studies done in the 1970s [finding] that the exposures for the folks working with the brakes were 16 fibers per cubic centimeter [from] several feet away[, which] means that the exposures were at levels capable of causing disease and . . . higher . . . than is even required for mesothelioma" [A-1208 to 1209].

Dr. Moline explained:

> [t]here were studies done in the 1970s that measured the exposure of automobile mechanics in New York and they found . . . that the exposures for the folks working with the brakes were 16 fibers per cubic centimeter and that you could have measurable – there were measurements several feet away and it persisted. So there have been studies of the exposure from manipulation of the brake products. . . . It means that the exposures were at levels capable of causing disease and, in fact, there were additional studies that showed folks who worked with brakes developed asbestosis, which is the disease that's found with higher levels of exposure than is even required for mesothelioma. . . . Those studies [concerned] friction products and friction materials.

[A-1208 to 1209].

Given Mr. Juni's intense and regular occupational exposures while performing brake and clutch repair work and blow-out, and engine gasket rip-out and replacement, from 1964 to 1988 without warning or protection, concluded Dr. Moline, and given the visible asbestos dusts created by that work, such exposures, "with a reasonable degree of medical certainty," "were a substantial factor in contributing to his mesothelioma" [A-1089 to 1094].[22] Dr. Moline agreed that, "[f]rom a dose response perspective, . . . the relevant considerations are the amount of the exposure, the duration of an exposure, and the frequency of the exposure," and that "those factors are critical in assessing whether there is sufficient exposure in order for someone to have an increased risk of disease from the exposure" [A-1150 to 1151].

In addition to her familiarity with Mr. Juni's records establishing his "[a]sbestos contact having worked for many years with motor vehicle brakes," as well as new and used clutches, and new and used gaskets, while servicing the fleets of Ford vehicles [A-1086, 1108, 1151], and in addition to her knowledge of the studies referenced above showing ultra-carcinogenic exposure levels for vehicle mechanics against which

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[22] As stated by the Committee on Pattern Jury Instructions, "[a]n act or omission is regarded as a cause of an injury . . . if it was a substantial factor in bringing about the injury . . . . There may be more than one cause of an injury . . ., but to be substantial, it cannot be slight or trivial. You may, however, decide that a cause is substantial even if you assign a relatively small percentage to it." NYPJI § 2:70; *Turturro v. City of New York*, 28 NY3d 469, 483-84 (2016); *see In re New York City Asbestos Litig.: Dummitt v. A.W. Chesterton*, 27 NY3d 765, 786 (2016). The trial court itself so instructed [A-9174, 9187].

she clearly compared Mr. Juni's exposures [A-1093, 1097-1098], Dr. Moline further explained:

> The fact that he was exposed to visible dust tells us the magnitude of the exposure in a qualitative setting because no one was actually measuring his actual exposure. The use of visible dust is a surrogate for telling us that he was exposed to asbestos dust at levels that are above the level that we know asbestos is capable of causing disease at[,] far higher than what's considered a minimal exposure – a level capable of causing disease.

[A-1095].[23]

Dr. Moline, former Vice-Chair of Mount Sinai's Department of Preventive Medicine [A-1075], based her medical and scientific assessments on her "training and experience and [her] review of all the records in this case" [A-1088], including her experience and prominent role within NIOSH, which is a CDC research and education agency, and her knowledge of the NIOSH treatises addressing the effects of exposure to asbestos [A-1100 to 1101]. She further relied upon her "clinical experience, interviewing and evaluating folks who have been working with brakes and clutches and their descriptions of the exposures which were in line with Mr. Juni's

[23] *See Sean R.,* 26 NY3d at 811 (instructing, in an analogous context, that sensory, here "[o]dor thresholds can be particularly helpful in occupational exposure cases, where the odor threshold of a substance exceeds permissible workplace safety standards"); *see also Dominick v. Charles Millar & Son Co.*, 149 AD3d 1554, 1555-56 (4th Dept 2017) ("Here, plaintiff's expert opined that, if a worker sees asbestos dust, that is a 'massive exposure . . . capable of causing disease[,' which] was sufficient to establish specific causation").

exposures" [A-1093].[24]

3. Ford's Experts Conceded the Hazard

With regard to the issue of exposures to debris from brake wear – being just one of several different types of exposures Mr. Juni daily sustained while servicing Ford vehicles during his twenty-five year exposure period – Ford's expert toxicology witness Brent Finley admitted that, notwithstanding the conversion to forsterite of some percentage of the asbestos fibers in brakes while in use, "*millions of asbestos fibers are still released* when one blows out a brake drum" [A-1796 to 1797 (emphasis added)]. Dr. Finley agreed to "talk about the numbers" on cross-examination, and conceded that, even for used brakes involving the conversion of some of the asbestos fibers to forsterite, the time-weighted eight-hour average found by Ford in its own testing of brake work on passenger cars was .68 asbestos fibers/cc, and for brake work on

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[24] The trial court sustained Ford's objection to Dr. Moline's further testimony that, relevant to Mr. Juni's case, she also relied upon the mainstream scientific and medical information generated by such organizations as "the American Thoracic Society, the American College of Occupational and Environmental Medicine, the World Health Organization, International Agency for Research on Cancer" ("IARC") [A-1094]. However, the *Reference Manual* calls entities such as IARC "the most well-respected and prestigious scientific bodies" investigating medical causation. FEDERAL JUDICIAL CENTER, REFERENCE MANUAL (3d ed. 2011), *supra*, at 20. Indeed, the trial court had previously overruled Ford's objection to Dr. Markowitz's testimony noting that "[t]he World Health Organization, the International Agency for Research on Cancer, the National Cancer Institute, the Environmental Protection Agency, the Occupational Safety and Health Administration, the Agency for Toxic Substances and Disease Registry, the World Trade Organization . . . form the foundation of [his] opinion that exposure to chrysotile asbestos is capable of causing malignant mesothelioma" [A-96 to 97].

trucks the time-weighted average was 1.75 fibers/cc [A-1853]. Dr. Finley admitted that "those numbers are way above OSHA's current permissible exposure level" and, indeed, "*35,000 times* above background level" [A-1853 to 1854 (emphasis added); *see also* A-1209].[25]

With regard to new brake linings, Dr. Finley acknowledged that these were comprised of fifty-percent (50%) asbestos by weight [A-1797]. Accordingly, "if the truck brake lining weighs 12 pounds on a heavy truck brake, there is 6 pounds of asbestos as part of that new brake," containing "billions, with a B, of fibers" [A-1798]. Consistently, Ford's representative Fyie admitted that Ford had received medical and scientific data showing that "[w]orkers engaged in grinding operations to renew brake linings were exposed to 2 to 7 optically visible fibers per milliliter," and that workers "beveling linings had high exposures, ranging from 32 to 72 fibers per milliliter," being more than *700 times* the regulatory limit [A-908].

Dr. Finley acknowledged the position unanimously taken by OSHA, by the World Health Organization, by the American Conference of Governmental Industrial Hygienists, by the Surgeon General of the United States, and by the National Cancer Institute – all of which Dr. Finley acknowledged to be authoritative – that "there's no

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[25] Dr. Finley further conceded that, in a 1970 manuscript reporting on their study conducted in 1968 (D. E. Hickish & K. L. Knight, *Exposure to Asbestos During Brake Maintenance*, 13 ANNALS OF OCCUPATIONAL HYGIENE 17-21 (1970)), researchers "found that blowout . . . of fibrous dust in brake maintenance procedures on commercial vehicles . . . was four times higher" than the regulatory limit [A-1839 to 1842].

known safe level or there's no safe level of exposure to asbestos" [A-1785 to 1788].[26] The import of this acknowledgment is that Mr. Juni's regular, frequent and continual exposure levels over a twenty-five year period to concentrations of asbestos fibers quantified as multiple times greater than the regulatory limit strongly supported the experts' causation opinions.

Additionally, Ford's epidemiological witness, Dr. Teta, conceded that she herself had written, some ten years prior to the instant trial, that various "authors, Lorimer, 1976, regulatory agencies, EPA, 1986, and trade organizations, world trade organizations, 2000, have opined in the past that motor vehicle mechanics are likely to be at increased risk of developing asbestos-related disease, most notably mesothelioma" [A-1425]. She further conceded that, as opposed to the irrelevant epidemiological studies relied on by Ford, the NIOSH studies addressing work precisely like Mr. Juni's, as cited by Dr. Markowitz, "went in and actually looked" at the facilities and the auto mechanics, and found ultra-carcinogenic asbestos fiber levels [A-1564 to 1566]. And, as already noted, Dr. Teta acknowledged that the 2013 Roelofs' epidemiological study was relevantly focused precisely on the sort of work Mr. Juni's performed, and found that "the *relative risk is greater than two, to a 95 percent confidence level*" – meaning that this study was statistically significant [A-1447, 1558-

[26] As Dr. Markowitz similarly testified, OSHA has emphasized that "the current time-weighted average of 0.1 fiber per cc [is] not safe. They admit it will cause lung cancer and mesothelioma in a certain percentage of workers who work with it at that level" [A-353].

1559 (emphasis added)].

On top of all of this, Dr. Teta effectively conceded that, given the sound epidemiological evidence "already" "connect[ing]" asbestos to mesothelioma, "a case report of an automobile mechanic exposed to friction products that contain asbestos, [and who] developed mesothelioma," would be sufficient "to establish a causal connection" solely but for the asbestos fiber type being chrysotile [A-1268 to 1269]. In the final analysis, Dr. Teta's opinions about causation clearly appeared to rest on her purported view that chrysotile asbestos is somehow safe. Yet this qualification was untenable; mainstream science and all of the regulatory entities concerned with this issue have long established that chrysotile asbestos is a potent and ultra-carcinogenic toxin, and the evidence at trial well demonstrated the same fact [A-96 to 97, 108, 115-16].

B. THE APPELLATE DIVISION'S RULING

The Appellate Division's majority decision substantially misunderstood the scientific facts at issue, overlooked the medical and scientific testimony with which the jury had the opportunity to become fully familiar, and misapprehended this Court's precedents. The majority's ruling was profoundly unfair to the grievously stricken Junis, particularly given that Mr. Juni had sustained barely momentary exposures to any asbestos products other than those for which Ford was responsible,

being continually exposed to asbestos fibers released from Ford's vehicle components without warning.

First, the majority panel wholly discounted "Dr. Moline's testimony that the visibility of the dust itself indicates the magnitude of the exposure 'at levels that are . . . capable of causing disease'" solely because, as misapprehended by the majority, "on cross-examination she conceded that studies have shown that more than 99% of the debris from brake wear is not comprised of asbestos fibers" [A-8]. Overlooked by the panel, however, there was absolutely no evidence of any transformation of the ultra-carcinogenic asbestos fibers with respect to the new brakes Mr. Juni sanded and beveled, or the new and used clutches and new and used gaskets that released substantial asbestos-filled dusts inhaled by Mr. Juni. Decedent's daily inhalation of dusts emanating from worn asbestos-containing brakes in Ford vehicles using compressed-air blowout [R. 3622-23] was but a portion of his total asbestos exposure, including his exposures occurring during clean-up of the asbestos-containing dust [A-427, 501, 711, 2095].

But even with regard to Mr. Juni's used brake exposures, Dr. Moline simply did not make the "concession" ascribed to her by the Appellate Division majority. In this regard, Dr. Moline acknowledged solely that "some studies" say that "some percentage" of the fibers in used brake debris have been transformed [A-1203]. The Appellate Division erroneously took the whole of defense counsel's unsupported

leading question to constitute the expert's "concession."

The Court's majority opinion further pointed to Dr. Markowitz's "concession" "that the high heat generated within the brake drums when the brakes are applied converts most of the asbestos in the brake lining to another mineral known as forsterite, and that studies have shown that only 1% of the dust blown out from brake drums is comprised of asbestos" [A-9]. Again, however, Dr. Markowitz made no such 1% concession. Rather, he emphasized that, when it comes to dusts released from the used brakes, "*30 percent of the fibers observed were identified as asbestos* with the remaining fibers being categorized as forsterite (20 percent) or unknown (50 percent)" [A-359 (emphasis added)].[27]

Hence, it was only by disregarding the actual testimony, and failing to view it in the light most favorable to the nonmovant plaintiff in whose favor the jury rendered a verdict, that the Appellate Division's majority transformed Dr. Markowitz's testimony into something it was not. Additionally, the testimony regarding "debris from brake wear," as noted above, concerned only one out of many sources of exposure to Ford asbestos products suffered by Mr. Juni over his career.

In this regard, only the dissenting opinion properly credited the evidence that, "[f]or more than 25 years, [Mr. Juni] was exposed directly and indirectly to

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[27] In all events, given the numerous studies establishing an actual air contamination level at 16 fibers/cc [A-264 to 266, 1208-09], the forsterite issue was a red herring.

asbestos-laden dust released from new and used brakes, clutches and gaskets when they were cleaned with 'compressed air,' and from scraping off asbestos intake on manifold and engine gaskets" [A-21].[28]

A further aspect of the testimony that the Appellate Division appears to have viewed as conclusively undermining the causation proof as a matter of law concerned the idea that, "when asbestos fibers in braking equipment are mixed with certain resins during manufacturing, 'they would not be respirable'" [A-9]. However, this testimony involved *just one of the three* Ford components that produced substantial quantities of asbestos-filled dust upon handling, compressed-air blowout, sanding, scraping, cutting and beveling. Moreover, as the dissenting opinion noted [A-26], during cross-examination by defense counsel Dr. Markowitz explained that, "[p]rior to phenolic resins there were some other more natural materials that didn't have the qualities of phenolic resin" [R. 2456-57]. On this particularized issue, Ford should not thereby receive the benefit of any available inference that its counsel did not see fit to probe. In other words, Ford's counsel was free to ask the follow up question, whether the phenolic resin had likely been in use during Mr. Juni's exposure period or only subsequently, this was the obvious follow-up, yet counsel quite conspicuously

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[28] *Cf. Dixon v. Ford Motor Co.*, 70 A.3d 328, 332 (Md. 2013) ("In performing his brake maintenance and repairs, Mr. Dixon used compressed air and a wire brush to clean the drums and remove debris, and sand paper to remove glaze on the brake linings. If new brakes were required, he would file the edges of the new brake shoes before installing them. All of this generated asbestos-laden dust").

failed to do so [A-220].

Even more importantly, the majority's Decision misapprehended the significance of Dr. Markowitz's literal statement on this issue, and in so doing inadvertently altered his testimony in a material way. In this regard, the Decision paraphrases Dr. Markowitz's testimony to merely state, as quoted above, "that when asbestos fibers in braking equipment are mixed with certain resins during manufacturing, 'they would not be respirable'" [A-9]. His actual testimony, however, was that, "*when they're embedded in* the phenolic resin, they would not be respirable" [A-219 (emphasis added)]. The Court's elision of the italicized actual testimony is a critical omission because, as with any asbestos product, it is the use, cutting, blowout, sanding, grinding, beveling and other manner of handling of the product that causes the fiber content to be released, airborne and respirable. As presented to the jury, there was nothing in fact about Dr. Markowitz's testimony to indicate that asbestos-containing brakes, during actual use and handling, would not have released ultrahazardous levels of asbestos-laden dusts.[29]

Next, the Appellate Division's majority Decision appeared to accept as final and dispositive Dr. Markowitz's apparent "acknowledge[ment] that 21 of 22

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[29] *See generally Berger*, 13 Misc.3d at 347 ("where it is undisputed that defendant's products were made up of as much as 50% chrysotile, even though they were embedded in resin and most but not all were shorter than five microns, and where the plaintiffs developed mesothelioma, there is sufficient empiric evidence to allow the jury to consider causation").

epidemiological studies that addressed asbestos exposure to mechanics working on friction products found no increased risk of mesothelioma" [A-8]. However, as the jury fully heard, and as explained in the dissenting opinion, "the 21 studies were not relevant to Juni's work situation" because *not a single one of them* "specifically looked at whether garage mechanics who regularly work with brakes and other friction products develop mesothelioma at a higher rate than others" [A-27]. Dr. Markowitz continued that "[t]he reasons are varied" as to why there have not been many epidemiological studies relevant to the present type of exposures, and he set those reasons out in painstaking detail [A-27, 116-119].[30]

Upon defendant's post-trial motion to set aside the verdict, the Court was required to view all of Dr. Markowitz's testimony in its proper context and in the light most favorable to the party prevailing at trial. The majority Decision not only ignored the import of Dr. Markowitz's credible testimony about the irrelevant epidemiological studies, but wholly overlooked the truly probative concessions offered by defendant's own expert, Dr. Teta, that none of those studies were, in fact, relevant.

In this regard, Dr. Teta acknowledged that "not a single study" from among the twenty-one involved circumstances resembling Mr. Juni's "exposures to brakes

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[30] *See Dixon,* 70 A.3d at 334 ("because not all mechanics work on brakes, it was difficult to do a specific job-related epidemiological study").

and asbestos clutches and asbestos gaskets" [A-1465], and that "*none*" of the epidemiological studies upon which Ford relied, amorphously concerning garage workers, "were powered, going into the study, to look at the risk specifically of people working with asbestos brakes and clutches in terms of their risk of mesothelioma" [A-1442, 1465,[31] 1509-1513, *see also* A-1493 ("It's possible that I had no vehicle mechanics at all in this study")].[32] As explained by the dissent [A-27], this was a critical oversight rendering infirm the majority panel's conclusion that Dr. Markowitz's mere acknowledgment of *the existence* of the twenty-one studies somehow so "undermined" his causation opinion as to render it "groundless or unsupported" [A-7].

In these respects, Dr. Teta conceded that "the occupational category defined as 'garage' is insufficiently specific" in the studies, hence *wholly lacking a relevantly defined exposed group*, either for purposes of supplying the study subjects (cohort study) or control group (case-control study) [R. 3939]. Dr. Teta could not rule out the

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[31] "Q: What study do you believe Mr. Juni's exposures to brakes and asbestos clutches and asbestos gaskets most closely matches his exposure? What study or studies?
A: There is not a single study" [A-1465].

[32] *See generally* Sven Hernberg, *"Negative" results in cohort studies – How to recognize fallacies*, 7 SCAN J WORK ENVIRON HEALTH 121, 121 (1981) ("A true negative study must fulfill three criteria: (i) it must be large; (ii) it must be sensitive; and (iii) it must have well-documented exposure data"). The Appellate Division's majority Decision overlooked both Dr. Markowitz's extensive testimony, and the express admissions of Ford's own epidemiological witness Dr. Teta, both of which established to the jury that the epidemiological studies that Ford referenced in its cross-examination of Dr. Markowitz unquestionably failed to meet a single one of those criteria.

possibility that the "garage workers" or "automobile repair and related services" workers included, for example, workers who may have only pumped gasoline, may have only changed motor oil, may have only done body work, may have only worked in a parts department, done muffler work or towing trucks, and may not have included a single worker who actually did brake and clutch work [R. 3937-41].

Hence, not only did Ford's own epidemiological witness thus admit the complete irrelevance and/or statistical insignificance of her own and the additional epidemiological studies relied upon by Ford, but another author of one of those "negative" friction studies Ford cited at trial, Dr. Key Teschke [*see* A-1546], has sharply criticized its misuse by industry defendants such as Ford. In her 2016 letter to the editor published in the *Annals of Occupational Hygiene*, Dr. Teschke has expressed her deep concern that her 1997 study "had been used regularly in litigation related to mesothelioma in people who worked as vehicle mechanics or in brake repair."[33] Just as Dr. Markowitz explained to the jury, Dr. Teschke noted that "the jobs or tasks [that were] being used as surrogates of chrysotile exposure" are not, in fact, "good surrogates," hence creating "problems in interpretation" stemming from "the likelihood that many vehicle mechanics had done no brake repair work"

[33] Kay Teschke, Letter to the Editor, *Thinking about Occupation–Response and Exposure–Response Relationships: Vehicle Mechanics, Chrysotile, and Mesothelioma*, 60 ANN. OCCUP. HYG. 528, 528 (2016).

whatsoever.[34]

Also wholly missing from the majority Decision was any reference to Dr. Teta's further concession that "there is a study, *the most recent study* from 2013, the lead author is Roelofs, where *the relative risk is greater than two* and, to 95 percent confidence, *chance was ruled out* as the explanation because it was statistically significant," and that the Roelofs study was "*closer to Mr. Juni's actual situation* when he was exposed to asbestos brakes, clutches, and engine gaskets" [A-1447, 1554 (emphasis added)]. This *relevant* study, involving work comparable to Mr. Juni's experience, was sufficient and probative on the causation issue and, certainly, is a valid scientific expression of causation as conceded even by Ford's own expert.

A further critically important flaw in the majority's Decision arose from its misunderstanding of Dr. Markowitz's "acknowledg[ment] that chrysotile . . . can clear the lungs through macrophages and translocation" [A-9]. Contrary to the Court's misapprehension, this evidence *strongly supports* causation of pleural mesothelioma. Dr. Markowitz explained that some of the chrysotile fibers tend to be "transported outside of the lung" [A-192 to 193]. Misapprehended by the Court, *Dr. Markowitz's point was that this is precisely the reason that the chrysotile fibers become so deadly and conducive to the development of mesothelioma* [A-99 ("chrysotile tends to dissipate from the lung tissue[,

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[34] *Id.* at 528-29.

which is] *illuminating . . . for malignant mesothelioma*" (emphasis added)]. The pleura is the "target organ" for mesothelioma, not the lungs. As Dr. Markowitz explained to the jury [*e.g.*, A-93, 127], mainstream scientists teach that "the fibres gradually clear to the pleural space where they accumulate, thereby shifting disease from lung to pleura[, explaining] how the risk of mesothelioma can be enhanced by only low exposure to asbestos." V. Courtney Broaddus & Marie-Claude Jaurand, "Asbestos Fibres and their Interaction with Mesothelial Cells *in vitro* and *in vivo*," *in* MESOTHELIOMA 273, 273 (Bruce W.S. Robinson & A. Philippe Chahinian eds., 2002).[35]

As the trial court itself correctly noted, "the general knowledge that chrysotile asbestos causes mesothelioma [is] a proposition acknowledged by defendant" [A-11814; *see also* A-1779 to 1780 (whereat Ford's expert toxicology witness Dr. Finley admits that chrysotile is classified as *a carcinogen*, hence *a cause* of cancer, by world-leading scientific organizations), A-8925 (Ford's counsel's summation acknowledging its own expert's concession that mainstream scientific "organization[s] say chrysotile can *cause* disease")]. Moreover, as shown above, abundant additional testimony directly established that "*chrysotile causes mesothelioma*" [A-90 (emphasis

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[35] *See also Rost v. Ford Motor Co.*, 151 A.3d 1032, 1038 (Pa. 2016) ("Given their smaller size, chrysotile fibers are more likely to get into the lymphatic flow and reach the pleura (the membrane on the outside lining of the lungs), and when investigators examine the target site of mesothelioma on the lung, they typically find a predominance of chrysotile fibers").

added)]. As Dr. Moline explained, "It's been shown in animal studies, in human studies, in groups of workers that have worked *with chrysotile only* that it *causes* mesothelioma" [A-1097 (emphasis added)].

ARGUMENT

THE APPELLATE DIVISION ERRED BY MISAPPLYING THIS COURT'S CONTROLLING PRECEDENTS

A. THE MAJORITY'S RULE 4404(A) ANALYSIS WAS LEGALLY ERRONEOUS

As stated by the dissenting opinion below, the majority "misapplied the standard for review for legal sufficiency, and misapplied the law concerning general and specific causation in asbestos cases" [A-20]. This Court has emphasized that a CPLR 4404 motion may not be granted unless "there is simply no valid line of reasoning and permissible inferences which could possibly lead rational jurors to the conclusion reached by the jury on the basis of the evidence presented at trial." *Cohen v. Hallmark Cards, Inc.,* 45 NY2d 493, 499 (1978).

Upon all of the evidence marshaled above and in the record, the jury's findings that Mr. Juni was "exposed to asbestos from brakes, clutches or gaskets sold or distributed by defendant Ford" [A-2388], and that Ford's failure to warn Mr. Juni about the hazards of such exposure was "a substantial factor in causing his injury" [A-2389], were clearly not "utterly irrational." *Cohen*, 45 NY2d at 499 ("in any case in

which it can be said that the evidence is such that it would not be utterly irrational for a jury to reach the result it has determined upon, and thus a valid question of fact does exist, the court may not conclude that the verdict is as a matter of law not supported by the evidence"); *Mazella v. Beals*, 27 NY3d 694, 705 (2016).

B. THIS COURT'S PRECEDENTS REQUIRE REVERSAL

The evidence in this case established, through both quantitative and qualitative proofs, that Mr. Juni's levels of exposure to ultra-carcinogenic asbestos fibers released from Ford's new and used brakes, new and used clutches, and new and used gaskets, and from his clean-up and compressed air blow-out of the dusts containing those asbestos fibers, over an unbroken twenty-five year occupational period, substantially contributed to his later affliction with mesothelioma. Indeed, it was undisputed at trial and on appeal not only that mesothelioma is "a signature disease" [A-11812], hence "caused only by exposure to asbestos" [A-11813],[36] but that Mr. Juni's only other asbestos exposures were fleeting at best. The proofs at trial were cognizant of, and fully followed and complied with, this Court's analyses in such cases as *Parker*, and *Cornell*, and also in accord with this Court's more recent Opinion in *Sean R.* Yet the trial court and majority appellate panel erred both in failing to appreciate the

[36] *See Berger,* 13 Misc.3d at 342 ("mesothelioma is a signature disease primarily if not exclusively caused by asbestos exposure"); *Dixon*, 70 A.3d at 330 ("That the mesothelioma was caused by her exposure to asbestos is not in dispute").

quantitative proofs, and in otherwise misapprehending the quantitative and qualitative mainstream scientific expressions of causation coursing through this record.

The majority and concurring opinions below overlooked the proffers of quantitative fiber-release testimony concerning exactly the sort of asbestos-related work in which Mr. Juni engaged [*e.g.*, A-7 (faulting "[p]laintiff's experts" for failing "to quantify the decedent's exposure levels"), A-17 (concurring opinion incorrectly suggesting that plaintiff's experts transgressed *Parker* by relying upon "unquantified cumulative exposures to 'visible dust'")].

Defendant Ford persistently sought to convince the post-trial court and the Appellate Division to disregard this Court's reasoning in *Parker*, particularly those portions in which this Court "depart[ed] from the Appellate Division," 7 NY3d at 448, and adopted the reasoning in *Westberry v. Gislaved Gummi AB*, 178 F.3d 257 (4th Cir. 1999). *Parker* expressly cited *Westberry*, 178 F.3d at 264, wherein the Fourth Circuit held out the asbestos context as paradigmatically one in which the very evidence of "substantial exposure" as proffered in the present case establishes asbestos-related causation. Yet the proofs in the present case surpassed those deemed sufficient in *Westberry*, 178 F.3d 257 at 264 (explaining that "'[h]uman exposure occurs most frequently in occupational settings where workers are exposed to industrial chemicals like lead or asbestos; however, even under these circumstances, it is usually difficult, if not impossible, to quantify the amount of

exposure'") (quoting *Reference Manual*, *supra*, at 187).[37]

Indeed, in its recent decision in *Rost*, the Supreme Court of Pennsylvania rejected this very defendant's maneuver repeated in the present case, asserting: "We must agree with the Rosts that Ford has confused or conflated the 'irrefutable scientific fact' that every exposure cumulatively contributes to the total dose (which in turn increases the likelihood of disease), with the legal question under Pennsylvania law as to whether particular exposures to asbestos are 'substantial factors' in causing the disease." 151 A.3d at 1045. The Court further noted the expert's well-accepted, mainstream scientific view that "the causative agent is 'the series of exposures' . . . and the cumulative dose causes mesothelioma." *Id.* at 1039.

Similarly, this Court has unequivocally credited the *Reference Manual* as an authoritative source for standards of valid and effective scientific expression of causation, implicitly in *Parker*, and then expressly in *Cornell*. 22 N.Y.3d at 783. The *Reference Manual* itself instructs that, "while precise information concerning the exposure necessary to cause specific harm to humans and exact details pertaining to the plaintiff's exposure are beneficial, such evidence is not always available, or necessary, to demonstrate that a substance is toxic to humans given substantial

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[37] *See Rost*, 151 A.3d at 1052 (in case arising from exposure to Ford's asbestos brake and gasket products in vehicles, flatly rejecting Ford's positions and emphasizing that, "[t]o require quantification where it is almost always impossible and unnecessary to do so, would be a public health travesty. That is, this would have the effect of creating an impossible burden of proof, and no claim would be able to meet this impossible standard").

exposure and need not invariably provide the basis for an expert's opinion on causation." Michael D. Green, *et al., Reference Guide on Epidemiology*, FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 549, 586-87 (3d ed., 2011); *see also* David L. Eaton, *Scientific Judgment and Toxic Torts – A Primer in Toxicology For Judges and Lawyers*, 12 J. L. & POL'Y 5, 9 (2003) ("emphasizing that "[t]oxic substances may take many forms," and thus "a variety of approaches and techniques are used to evaluate the toxicological characteristics of chemicals").

Immediately thereafter, the *Reference Manual* applies this principle to the particularized asbestos context, explaining that, "[i]n asbestos litigation, a number of courts have adopted a requirement that the plaintiff demonstrate (1) regular use by an employer of the defendant's asbestos-containing product, (2) the plaintiff's proximity to that product, and (3) exposure over an extended period of time." *Id.* at 587. Clearly, the evidence in the present case met this standard.

The *Reference Manual* section just discussed further explains that "[a]ssessment of past exposures is especially difficult when considering diseases with very long latency periods.[38] By the time disease occurs, documentary proof of exposure and

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[38] It is well to note, in this regard, that, in contrast to the usual circumstances in an asbestos case, Mr. Parker "worked at several full-service stations between March 1981 and August 1998," *Parker*, 7 NY3d at 442, he was diagnosed with his leukemia in September 1998, and filed his suit within one year thereafter. *Parker v. Mobile Oil Corp.*, 16 AD3d 648, 649 (2d Dept 2005). Similarly, Ms. Cornell filed her suit a mere thirteen months following her last alleged exposures to the mold. *Cornell*, 22 NY3d at 765-66. Mr. Juni's onset of symptoms did not occur until June 2012, (continued...)

magnitude may have disappeared. . . . On occasion, qualitative evidence of exposure is admitted as evidence that the magnitude was great enough to cause harm." Green, *supra*, at 512-13 (citing *Westberry*); *accord Parker*, 7 NY3d at 449 ("qualitative means could be used to express a plaintiff's exposure"); Eaton, *supra*, at 30 ("when considering the potential health significance of exposure to chemical mutagens that may act as carcinogens, it is important to keep the total or *cumulative* 'dose' in mind") (emphasis added), & 32 ("Certain cancers (mesothelioma) that arise from occupational exposure to asbestos typically are not seen for thirty or more years after first exposure").

Nevertheless, the proofs in the present case included abundant evidence of actual quantitative exposure measurements from precisely the type of work performed by Mr. Juni, as discussed above. Plaintiff's specific causation expert compared those studies to the similar types of exposures sustained by Mr. Juni [A-1093].[39]

Nor did the Appellate Division majority panel's rejection of the causation experts' visible asbestos dust testimony comport with the facts established at trial or

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[38](...continued)
some twenty-five years following his last exposure to Ford's asbestos products at the Orange & Rockland site, a typical latency period for mesothelioma [A-1106].

[39] *See generally Dominick*, 149 AD3d at 1555 (holding that expert's responses to hypothetical questions "'fairly inferable from the evidence' [were] sufficient to establish that asbestos in products they supplied was a substantial factor in causing or contributing to plaintiff's injuries") (quoting *Tarlowe v. Metropolitan Ski Slopes*, 28 NY2d 410, 414 (1971)).

with this Court's guidance [A-9 to 11]. As shown above, Dr. Moline explained that exposure to visible dust emanating from high asbestos-content products such as brake linings (50% asbestos by weight [A-1797]), clutches,[40] and gaskets,[41] "tells us the magnitude of the exposure in a qualitative setting . . . telling us that he was exposed to asbestos dust at levels that are above the level that we know asbestos is capable of causing disease at" [A-1095].

This aspect of the majority decision also contravenes this Court's more recent Opinion in *Sean R.* Plaintiff in *Sean R.* had been exposed *in utero* to chemical constituents of gasoline vapor his mother smelled in her BMW, allegedly resulting in plaintiff's birth defects. Although it was undisputed that gasoline had a low odor threshold detectable at non-toxic levels, 26 NY3d at 811, plaintiff's experts had invalidly sought to infer a causal connection between the mother's symptoms of headache and nausea and the plaintiff's birth defects. *Id.* at 809-10. Importantly, however, the *Sean R.* Court (26 NY3d at 810-11) contrasted that flawed methodology with the "fundamentally different" and "true 'odor threshold' analysis" validly applied in cases such as *Manuel v. Shell Oil Co.*, 664 So.2d 470 (La. Ct. App. 1995), and

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[40] *Rost*, 151 A.3d at 1037 (noting parties' stipulation "that Ford's brakes and clutches were forty to sixty percent chrysotile asbestos by weight").

[41] *In re New York City Asbestos Litig.: Dummitt v. A.W. Chesterton*, № 1090196, 21012 WL 3642303, at *8 (Sup. Ct., NY County, Aug. 20, 2012) (noting evidence that asbestos-containing gaskets were composed of sixty to eighty-five percent (85%) asbestos).

Magistrini v. One Hour Martinizing Dry Cleaning, *180 F. Supp.2d 584* (D.N.J. 2002), *aff'd*, 68 Fed. Appx. 356 (3d Cir. 2003).

Particularly relevant to the visible-dust testimony in the instant asbestos-related case, this Court in *Sean R.* noted approvingly that the expert in *Magistrini* had derived "a plaintiff's occupational exposure to perchloroethyline based on the chemical's odor threshold, coupled with other employment information . . . ." *Sean R.*, 26 NY3d at 810. Fully analogous to Dr. Moline's testimony in the present case establishing that "visible dust" released from asbestos-containing products signifies asbestos levels "far higher" that what is likely to cause disease [A-1095],[42] the *Magistrini* court noted that the "preferred method" would have been direct "air sampling," but in the absence of such sampling "a proxy" level of exposure can be estimated by use of the sensory threshold method. *Magistrini*, 180 F. Supp.2d at 613-14; *accord Dominick*, 149 AD3d at 1555-56 (crediting expert's testimony that visible asbestos-containing dust signifies exposure "capable of causing disease") (citing *Parker*, 7 NY3d at 448; *Sean R.*, 26 NY3d at 808).

[42] *See Sean R.*, 26 NY3d at 811 ("Odor thresholds can be particularly helpful in occupational exposure cases, where the odor threshold of a substance exceeds permissible workplace safety standards").

CONCLUSION

For all of the reasons stated, plaintiff respectfully requests that the Appellate Division's February 28, 2017 Decision and Order appealed from be reversed, the judgment vacated and the jury's verdict reinstated, and that the case be remitted to Supreme Court for consideration of any remaining issues raised by defendant in its post-trial motion but not reached by that court or by the Appellate Division.

Dated: New York, New York
September 28, 2017

Respectfully submitted,

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

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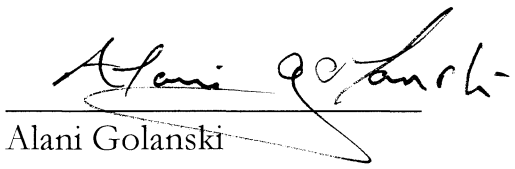

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