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*)

REPLY BRIEF FOR PLAINTIFF-APPELLANT

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December 20, 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.

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Certification Pursuant to 22 NYCRR § 500.13(c)(1)

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A. INTRODUCTION

The sole issue in this appeal is whether, on the issue of causation, there was any "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). The jury's finding that Mr. Juni's "expos[ures] to asbestos from brakes, clutches or gaskets sold or distributed by defendant Ford" [A-2388] were a concurrent cause in the development of Mr. Juni's mesothelioma [A-2389] was clearly not "utterly irrational." *Id.* As emphasized by the dissenting opinion below, the trial court and the Appellate Division majority "misapplied the standard of review for legal sufficiency, and misapplied the law concerning general and specific causation in asbestos cases" [A-20].

During the 2014 trial, counsel conscientiously hewed to this Court's guidance in *Parker v. Mobil Oil Corp.*, 7 NY3d 434 (2006), as well as the Court's then-recently issued opinion in *Cornell v. 360 West 541st Street Realty*, LLC, 22 NY3d 762, 783 (2014). In its present Brief ("D/B"), defendant Ford self-servingly misstates plaintiff's position as being "that a scientific expression of exposure is unnecessary in asbestos cases" [D/B, at 47]. That is the *very opposite* of plaintiff's position; nor was any such position the import of the dissenting Justice's view. Rather, for all of the reasons stated by the dissenting opinion [A-39 to 43], the evidence below fully comported

with the *Parker* standard, and with well-settled case law recognizing the overwhelmingly accepted manner in which general and specific causation is scientifically expressed by the medical and scientific community in such cases.

B. THE PROOFS CONSTITUTED A SCIENTIFIC EXPRESSION CAUSALLY LINKING MR. JUNI'S OCCUPATIONAL EXPOSURES TO ASBESTOS DUSTS RELEASED FROM FORD'S PRODUCTS TO HIS MESOTHELIOMA

As a backdrop to assessment of the legal causation issue, it is important to note that it was undisputed at trial, and in Ford's instant Brief, that "mesothelioma is a rare and deadly "signal" or "signature" disease, caused almost exclusively by respirable asbestos" [A-23, 63, 87, 120]. The trial court also fully accepted this threshold fact [A-11812 to 11813].

The evidence further overwhelmingly established that exposure to "chrysotile asbestos causes mesothelioma" [A-24, 90-97, 108-09]. Ford in its instant Brief effectively concedes this as well [D/B, at 28 (calling amphibole asbestos "highly-potent" and chrysotile "less-potent"; 29 (apparently acknowledging that "chrysotile in general can cause mesothelioma")]. Chrysotile asbestos has comprised approximately 95 percent of all the asbestos used commercially in the United States [A-82], and all of the mainstream scientific and health entities addressing the matter share the view that chrysotile asbestos is a causative agent in the development of mesothelioma [A-1098 to 1099; A-108 to 110, 1094].

Nevertheless, while Ford's epidemiological witness at trial, 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," she indicated that she withholds that conclusion solely because the asbestos fiber at issue is chrysotile [A-1268 to 1269]. The jury clearly did not credit Dr. Teta's cryptic hesitation on this issue [A-11689 to 11690].

Accordingly, as Ford itself states albeit at a slightly different angle [D/B, at 10, 19-20, 29], the issue is whether the mere fact that the chrysotile asbestos fibers emanated from automotive products – namely, brakes, clutches and gaskets (only the first being a "friction" product) – disqualified those fibers from having an ultra-carcinogenic general capability, and from having been, at the least, a concurrent cause in the specific development of Mr. Juni's mesothelioma.

In this vein, the Appellate Division majority, like Ford in its instant submission, erroneously concluded that, just because the asbestos emanated from vehicle-related products, the causation proofs "were therefore legally insufficient to establish that Juni's exposure to asbestos from brakes, clutches, or gaskets sold or distributed by defendant constituted a significant contributing factor in causing Juni's mesothelioma" [A-12]. Indeed, the majority panel paradoxically voiced continuing

approval of prior rulings involving proofs that were far less quantitatively exacting than in the present case, but were nevertheless deemed to establish the general and specific causation elements in *non-vehicle*-related asbestos gasket and other contexts [A-10 (approving of prior decisions in *Lustenring v. AC&S, Inc.*, 13 AD3d 69 (1st Dept 2004) (dust from asbestos gaskets), *Penn v. Amchem Prods.*, 85 AD3d 475 (1st Dept 2011) (dust from asbestos-containing dental liners)].

This Court's jurisprudence has understandably not set forth a strictly enumerated list of precisely what would or would not qualify, in any particular setting, as a scientific expression. As the *Parker* opinion instructs, in addition to "establish[ing] the dosage at which a substance is toxic and the amount of exposure a plaintiff actually experienced[, t]here could be several other ways an expert might demonstrate causation." 7 NY3d at 448-49. The opinion notes the possibility, for instance, "that the intensity of exposure to benzene may be more important than a cumulative dose," and that exposure could be estimated through "[c]omparison to the exposure levels of subjects of other studies" if sufficiently comparable to plaintiff's own exposure experience. *Id.* at 449. Moreover, continued the *Parker* Court, "[i]t is also possible that more qualitative means could be used to express a plaintiff's exposure . . . if they were found to be generally accepted as reliable in the scientific community." *Id.*

Although this Court has thus not strictly delineated the sort of proffer that may

be deemed to constitute a scientific expression of causation, its rulings are instructive on this point, both explicitly and by example. On that basis, as shown in plaintiff's main Brief and in the following sections, plaintiff has demonstrated that the quantitative and qualitative evidence in the present case – arising from the signature disease referenced above and from plaintiff's exposures to the very substance known to cause that disease – constituted a scientific expression in numerous respects.

1. Testimony About Quantitative Studies of Exposures Exceeding Regulatory Limits Is an Accepted Scientific Expression

In *Parker*, this Court credited defendant's expert's reliance, in part, on a National Institute for Occupational Safety and Health (NIOSH) study of benzene exposure for service station employees establishing that "the maximum concentration of benzene in gasoline was 2% with the greatest level of exposure 0.19 ppm TWA, which is less than the 1 ppm occupational standard set by the Occupational Safety and Health Administration [OSHA]." 7 NY3d at 443.

In the present case, the scientist experts similarly relied, in part, on critically relevant NIOSH and other studies involving exposures comparable to those of Mr. Juni.[1] Although regulatory *standards* alone will not establish causation in a tort case,

[1] NIOSH is a scientific research entity dedicated to investigating occupational health issues, "including surveillance, research and technology transfer." www.cdc.gov/niosh.

reliance upon regulatory *studies*, reports, and findings is well-accepted in the scientific community as part of the scientist's causation and risk analysis.[2]

In the present case, 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 – precisely the work engaged in by Mr. Juni – exceeded "30 fibers per cc," this being 300 times above the OSHA regulatory exposure limit [A-288]. He also testified about numerous additional studies, such as those performed by NIOSH in 1980 specifically and pointedly focusing on the repair of asbestos-containing brake equipment – again, precisely the source of Mr. Juni's exposures – 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].[3]

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[2] *See Allen v. Pennsylvania Engineering Corp.*, 102 F.3d 194, 197 (5th Cir. 1996) (deeming a NIOSH study probative); *Ellis v. International Playtex, Inc.*, 745 F.2d 292, 301 (4th Cir. 1984) (ruling that considerations of reliability "argue[] strongly for admission of the contested studies . . . carried out by public offices"); *Jowers v. BOC Group, Inc.*, 608 F. Supp. 2d 724, 738 (S.D. Miss. 2009) (OSHA measurements showed that 30% of welders experienced manganese fumes at higher than allowable concentrations); Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology, in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 633, 650 (3d ed. 2011) (crediting "[r]isk assessment as practiced by government agencies involved in regulating exposure to environmental chemicals").

[3] Highly misleadingly, Ford notes one response given by Dr. Markowitz on cross-examination, at which point the witness knew that studies in the 1970s had shown a time-weighted average of asbestos fiber exposures at two fibers/cc, but did not recall the citations to specific

(continued...)

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].

As Dr. Markowitz explained, the NIOSH studies of occupational conditions precisely parallel to Mr. Juni's during his twenty-five year period of exposure to Ford-related asbestos products:

> 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 elevated levels. [O]ther 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

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[3](...continued)
studies [D/B, at 13; A-288]. Ford then blatantly misconstrues this response to mean that "[h]e was unable to identify any specific study that showed that the time-weighted averages were in violation of then-existing standards" [D/B, at 13], and hence that the studies upon which Dr. Markowitz relied "cannot possibly support his opinions regarding general causation" [D/B, at 14]. Ford thereby wholly ignores Dr. Markowitz's testimony on redirect examination, referencing numerous supportive and relevant NIOSH studies that "spanned almost two decades" and that the witness was now able to identify with particularity as demonstrating ultrahazardously high "*time-weighted averages*" that "will cause lung cancer and mesothelioma in a certain percentage of workers who work with it at that level for a long period of time" [A-350, 353-54 (emphasis added)].

those diseases.

[A-355-56].

Dr. Moline 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 quantified to "16 fibers per cubic centimeter," and hence "at levels capable of causing disease," a "hundred thousand times greater [than] background levels of exposure to asbestos," and far higher "than is even required for mesothelioma" [A-1208 to 1209].

In this regard as well, Dr. Moline explained that the foundation for her testimony included "[m]y knowledge of [such] industrial hygiene studies" [A-1093], hence deemed by Dr. Moline to involve study conditions comparable to Mr. Juni's occupational experiences. In this regard, Dr. Moline explained that the scientific position that "[c]hrysotile causes mesothelioma [has] been shown in animal studies, in human studies, in groups of workers that have worked with chrysotile," and that Mr. Juni's "exposures were to [Ford's] products containing chrysotile asbestos, and not dissimilar since chrysotile was the main fiber used in the United States" [A-1097].

2. Sensory Threshold Testimony Is An Accepted Proxy for Injurious Exposure

In *Sean R. v. BMW of North America, LLC*, 26 NY3d 801 (2016), this Court

approved of a further method for proffering a scientific expression of exposure at causally injurious levels. The Court explained that sensory – in *Sean R.*, odor – thresholds "can be particularly helpful in occupational exposure cases, where the odor threshold of a substance exceeds permissible workplace safety standards." 26 NY3d at 811. The *Sean R.* Court noted that the odor threshold of the gasoline constituent at issue was "far below toxicity." *Id.* Yet plaintiff, alleging *in utero* exposures, sought an inference based on his mother's experience of symptoms. This Court concluded that "[p]laintiff has not shown that such a 'symptom-threshold' methodology, unlike the odor threshold methodology admitted in other cases, has been generally accepted in the scientific community." *Id.*

In the present case, dusts from the asbestos-containing products at issue here were shown to contain asbestos fiber counts many times above regulatory thresholds. Dr. Markowitz testified, for instance, that even 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)]. Ford's toxicology witness Brent Finley acknowledged that Ford's *new* brake linings were comprised of fifty-percent (50%) asbestos by weight, with "billions, with a B, of fibers" in each brake [A-1797 to 1798]. Mr. Juni breathed these fibers in when he "had to scuff it up with sandpaper" [A-657.6 to 657.7].

Indeed, Mr. Juni helped service a fleet of 500 Ford vehicles for twenty-five

consecutive years, was continually 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]. His exposures at issue in this case arose, in effect, from six asbestos-filled product categories for which Ford is responsible: new brakes (sanded and beveled), used brakes (compressed air blow-out), new clutches (scuffing [A-657.7], used clutches (compressed air blow-out), new manifold and engine gaskets and used manifold and engine gaskets (scraping, drilling, compressed air blow-out [A-501, 657.4]).

In line with *Sean R.*, Dr. Moline 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; *see also* A-108 ("chrysotile in friction products, if it becomes airborne and inhaled, can cause malignant mesothelioma")].

As Dr. Markowitz explained, single asbestos fibers are only "seen in clusters, it's seen as part of dust," whereas "individual fibers" are not visible [A-80 to 81]. The "industrial hygiene studies that show that brake workers and other vehicle-repair

workers have significant exposure to asbestos, going back in time when those studies were done, in the '60s, '70s and '80s" [A-112 to 113], *were* a significant part of the "foundation" underlying the experts' scientific expressions rooted in the sensory threshold approach.[4]

Ford seeks to defend the rulings below by claiming that there can be no "shortcuts" to proving specific causation based on the ongoing breathing of "visible dust" released from its products [D/B, at 2]. Ford recites that the trial court below "rejected both experts' statements that they could use evidence of visible dust as a proxy for establishing a scientific expression of exposure in this case" [D/B, at 21; A-l1819 to 20]. As Ford further notes, the concurring opinion in the Appellate Division "observed that reliance on the existence of visible dust as proxy for exposure would carve 'a gaping hole in the *Parker* standard of proof on causation'" [D/B, at 23; A-17].

Sean R. establishes, however, that such rulings erred as a matter of law. The trial court in its post-trial decision drew insupportable factual inferences from the testimony, and otherwise improperly viewed the evidence in the light most favorable to the movant/losing party, opining that the evidence "is insufficient to prove that

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[4] *See also, e.g.*, A-2078 (1986 EPA publication attesting that the "consequences of inadequate prevention" of exposure to asbestos-containing dusts released from "brake linings and clutch facings" include mesothelioma, a "cancer [that] occurred among brake mechanics, their wives, and their children") (citing numerous studies [A-2089 to 2090])).

the dust to which Juni was exposed contained any asbestos" at all [A-11819].[5] The Appellate Division concurrence misapprehended the legal significance of *Sean R.* in the context of *Parker* [A-17]. Indeed, the *Sean R.* proxy method appropriately highlights one further type of evidence that provides a scientific expression of exposure and causation under the standard enunciated in *Parker*.

In this regard as well, the Appellate Division's majority decision conflicts with subsequent appellate decisions that adhere to long-settled law set forth in this State's toxic tort litigation arising from tragic asbestos-related disease. Hence, in *Dominick v. Charles Millar & Son Co.*, 149 AD3d 1554 (4th Dept 2017), the Fourth Department credited plaintiff's expert's opinion "that, if a worker sees asbestos dust, that is a 'massive exposure . . . capable of causing disease'[, which] was sufficient to establish specific causation." 149 AD3d at 1555-56 (citing *Parker*, 7 NY3d at 448; *Cornell*, 22 NY3d at 784; *Sean R.*, 26 NY3d at 808, as well as numerous asbestos-related decisions). By Order entered December 14, 2017, this Court denied defendant's motion for leave to appeal in *Dominick* (Motion № 2017-878).

Even more recently, in *In re New York City Asbestos Litig.: Miller v. BMW of North Am., LLC.*, 154 AD3d 441 (1st Dept 2017), a case involving dusts released from

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[5] In light of the industrial hygiene and other studies establishing the actual release, during precisely the sort of occupational work engaged in by Mr. Juni, of ultra-carcinogenic asbestos fibers at levels well exceeding 100 times the regulatory standard [A-264 to 266, 288, 1208-09], the issue of the transformation of the asbestos in high-asbestos content brakes – just one of the six product categories – during extended high-heat use, was a red herring.

asbestos-containing brake products, a different First Department panel now cited to *Sean R.*, and ruled that, given the "asbestos-laden dust" released, "plaintiff's expert testimony was sufficient to establish that plaintiff's use of that grinder on automobile brake linings caused his exposure to asbestos dust in sufficient quantities to cause his mesothelioma." 154 AD3d at 441.

3. The Scientific Expression of Specific Causation Rested on an Established and Proper Foundation

Dr. Moline additionally attested that:

> There were studies done in the 1970s that measured the exposure of automobile mechanics in New York and they found that . . . 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. [This] 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.

[A-1208 to -09].

Dr. Moline further testified with particularity that "[c]hrysotile tends to move more to the pleura, which is where Mr. Juni's tumor was, not the lung. . . . [C]hrysotile doesn't persist as long in the lung, and then it moves to different parts of the body, including the pleura, which is where the tumor arose and where his mesothelioma was that killed him" [A-1210].

Counsel presented Dr. Moline with fact-specific hypotheticals reflecting Mr. Juni's exposures to equipment and products installed in Ford's vehicles. During his twenty-five-year tenure servicing Ford vehicles "Mr. Juni personally assisted with brake work and clutch work on a regular basis including on Ford brakes and Ford clutches associated with various Ford vehicles and was also present at times when others in his vicinity performed the same work" [A-1091].

Based on the evidence, Dr. Moline assumed, for example, that Mr. Juni or nearby workers:

– "dump[ed] brake drums on the floor which created visible asbestos dust";

– swept this dust "with a broom";

– "would sometimes use compressed air to clean brake drums and sweep up after, which caused visible asbestos dust to be pushed into the air";

– "when working with a new asbestos line brake, Mr. Juni at times would scuff the lining with sand paper which would create visible asbestos dust";

– "Mr. Juni would also be exposed to visible asbestos dust from opening up new brake packages," all such dust being "released into Mr. Juni's breathing zone";

– clutch jobs Mr. Juni assisted with included taking off the bell housing which created asbestos dust which would fly off the fly wheel and the clutch disk to the exterior of the housing";

– "Mr. Juni would use compressed air to clean out a clutch and that this also caused

asbestos dust to be released";

– "all of this dust would be released into Mr. Juni's breathing zone" [A-1091 to -92].

Dr. Moline then opined, to a reasonable degree of medical certainty, that Mr. Juni's exposures thus described "were a substantial factor in contributing to his mesothelioma" [A-1093]. The foundation for Dr. Moline's testimony included "my clinical experience, interviewing and evaluating folks who have been working with brakes and clutches and their descriptions of the exposures which were in line of with Mr. Juni's exposures" [A-1093]. *See Kulak v. Nationwide Mut. Ins. Co.*, 40 NY2d 140, 146 (1976)*; People v. Hood*, 47 AD2d 971, 971 (3d Dept 1975).

In its Brief, Ford responds that "the hypothetical should have been stricken. . . . Ford raised this point in its post-trial briefing, but the Supreme Court did not reach this issue, holding instead that, even assuming the facts contained in this hypothetical did have support in the record, testimony based on these facts was not sufficient to establish specific causation" [D/B, at 16]. In reality, however, the trial court expressly declined to reach Ford's wholly waived and solely post-trial claim concerning the "hypothetical facts," and stated that, "[r]ather, for purposes of determining specific causation only, I assume that the facts posed in the hypothetical are based on the trial evidence" [A-11817 to -18].

This Court has long held that, "[i]f the facts in the hypothetical question are fairly inferable from the evidence, the expert may state his opinion without further

foundation. The extent to which he elaborates or fails to elaborate on the technical basis supporting the opinion affects only the weight of the expert testimony." *Tarlowe v. Metropolitan Ski Slopes, Inc.*, 28 NY2d 410, 414 (1971); *see Dominick*, 149 AD3d at 1555 ("Contrary to the contention of the Millar defendants, the evidence is sufficient to establish that asbestos in products they supplied was a substantial factor in causing or contributing to plaintiff's injuries . . . . Plaintiff testified that he was exposed to asbestos dust from asbestos boards and cement supplied by the Millar defendants that were used in the heat treat area of a pneumatic-tool making plant. The hypothetical question that plaintiff asked his expert was based on plaintiff's testimony or was otherwise 'fairly inferable from the evidence'").

Hence, because the questions posed to Dr. Moline concerning Mr. Juni's exposures to vehicle-related asbestos products were clearly "fairly inferable from the evidence," and indeed were taken as so by the trial court below, this expert's responses formed a portion of her scientific expression of the cause of Mr. Juni's signature mesothelioma disease. Ford, in its instant Brief, is wrong is claiming that, "[u]nlike in *Rost [v. Ford Motor Co.*, 151 A.3d 1032 (Pa. 2016)], neither Markowitz nor Moline's testimony was tied to the circumstances of Mr. Juni's exposure; rather, they merely assumed that all of Mr. Juni's exposures were causative without regard to the frequency or regularity of those exposures" [D/B, at 49].

Dr. Moline's specific causation testimony was indeed in all respects "tied to the

circumstances of Mr. Juni's exposure." Under CPLR 4515, for example, Dr. Moline was entitled to rely on the hypothetical questions posed to her that specified those circumstances; and hence Dr. Moline accurately 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].

Nor can Ford now plausibly claim that the hypotheticals interpreting the evidence to demonstrate Mr. Juni's personal exposures to visible asbestos-containing dusts emanating from components in Ford's vehicles over a prolonged twenty-five-year period "should have been stricken" [D/B, at 16]. Ford lodged absolutely no objection at trial to the testimony of which it now complains. It is well-established that a litigant who fails to object at trial has waived any objection regarding the proffer of testimony, including by way of expert hypotheticals. *Tarlowe*, 28 NY2d at 414; *Robillard v. Robbins*, 78 NY2d 1105, 1106 (1991); *Mroz v. 3M Co.*, 151 AD3d 1606, 1607 (2017) ("3M failed to preserve at trial its contention that there was no evidentiary foundation for the expert's testimony").

As a further matter, Dr. Moline explained that the foundation for her specific causation opinion included studies "that have found elevated levels of dust from the manipulation of brakes and, in fact, have seen asbestosis in brake mechanics showing that there was exposure to asbestos from the manipulation. It's based on animal

studies that have shown association between asbestos and the type of asbestos used in brakes. It's [based] on human studies that show an association between asbestos and mesothelioma" [A-1093 to -94].

Moreover, "[t]he National Institute for Occupational Safety and Health is a research organization. The International Agency for Research on Cancer is a research organization. Those helped form the basis of my opinion" as a foundational matter [A-1094], as did scientific data and/or conclusions published by "the World Health Organization, and the World Trade Commission, the Consumer Product Safety Commission, the EPA, the Occupational Safety and Health Administration," as well as "the American Thoracic Society, the American College of Occupational and Environmental Medicine, all of whom say that all forms of asbestos, including chrysotile, cause mesothelioma" [A-1098 to -99]. *See Kulak,* 40 NY2d at 146 ("a proper foundation is laid [when] – the witness called to the stand is familiar with the practices of [other relevant practitioners]. Such knowledge may have been acquired in consequence of direct representation . . ., or may have come from broader experience in the field"); *In re Wendy P.*, 2017 WL 5575061, at *1 (N.Y. App. Div., 1st Dept, Nov. 21, 2017) ("a proper foundation does not require general acceptance in the scientific community, but may be properly laid by the expert based on her 'personal knowledge acquired through professional experience'").

Contrary to Ford's incorrect assumption [D/B, at 16-17 ("Moline . . . *had no*

information [regarding] Mr. Juni's actual exposure to asbestos from Ford products") (Ford's emphasis)], this is not the sort of "information" required of an expert witness. As such a witness, Dr. Moline need not have had direct personal knowledge of Mr. Juni's daily exposures, but rather personal knowledge "acquired through professional experience" of the methodologies for rendering a causation opinion.

Also attesting to the scientific nature of the expert causation proof at trial is the well-considered analysis of scientific causation in asbestos-related cases published in the *Reference Manual on Scientific Evidence* (Federal Judicial Center, 3d ed. 2011). This Court has cited to the *Reference Manual* as an authoritative guide to mainstream science in toxic tort causation contexts. *See Sean R.*, 26 NY3d at 812 (relying upon the *Reference Manual* as the source for discerning "generally accepted methodologies" for determination causation in toxic tort cases); *Cornell*, 22 NY3d at 783 (same); *see also Nonnon v. City of New York*, 32 AD3d 91, 104-23 (1st Dept 2006).

Dr. Moline's testimony adhered to the methodology described by Dr. Markowitz and set forth in the *Reference Manual*, which 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 exposure and need not invariably provide the basis for an expert's opinion on causation." Michael D. Green, *et al., Reference Guide on Epidemiology*,

REFERENCE MANUAL, *supra*, at 549, 586-87; *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." REFERENCE MANUAL, *supra*, 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. By the time disease occurs, documentary proof of exposure and 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; *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).

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].

Next, with regard to the experts' testimony concerning Mr. Juni's cumulative lifetime exposures to asbestos fibers released from products for which Ford is responsible, it is an established and universally-applied principle in the scientific community and in the medical field that, biologically, an individual's diagnosed asbestos disease is the result of the cumulative effect of his lifetime dose of exposures to asbestos. Hence, 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].

Accordingly, for instance, in its recent decision in *Rost*, the Supreme Court of Pennsylvania emphasized 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.

This does not mean – and was not intended or stated to mean by Drs. Markowitz or Moline in the present case – that every exposure is a substantial contributing factor. Rather, the question was whether the plaintiff's cumulative exposure to the defendant's particularized products were a substantial contributing factor in the development of his mesothelioma. As Dr. Markowitz stressed, 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].

4. The Epidemiological Testimony Also Contributed to the Experts' Scientific Expressions of General and Specific Causation

Dr. Markowitz noted abundant, well-accepted epidemiological literature establishing that chrysotile asbestos causes mesothelioma [A-115, 1163]. The trial court recognized that "epidemiological studies specific to a profession, or even epidemiological studies in general, are not necessary to prove causation" [A-11816]. Occupational groups lending themselves to epidemiological study include insulators

and textile workers, and miners, because tens of thousands of workers can be studied at a time, "[t]hey're sizable numbers [and] they're doing similar things at the workplace" [A-115 to 116]. By contrast, "people who work in the garages, service stations and the like work in relatively small numbers, small groups, and it's very difficult to assemble a very large group of them" [A-116; *see also* A-27].

There was one such task-specific epidemiological study discussed at trial, being the *only* epidemiological study that patterned Mr. Juni's occupational experiences (as defendant's epidemiological expert conceded). Ignored by both the trial court and the Appellate Division majority, this study showed a statistically significant greater-than-doubling of the relative risk of mesothelioma for vehicle mechanics occupationally exposed to asbestos [A-1447].

Unlike the jury, which carefully attended to the detailed testimony, the decisions appealed from viewed Ford's claims in the light most favorable to the post-trial movant. Ford's own epidemiological expert admitted, however, that it relied upon automotive industry-funded for-litigation meta-analyses, as well as other studies that (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].

Attempting to evade the irrelevancy of the studies at issue, Ford in its Brief

now disingenuously toggles between (nearly always) improperly *mislabeling* the inapposite studies "vehicle mechanic" studies [D/B, at 1, 11, 13, 20] and (just once!) correctly labeling them "garage worker" studies [D/B, at 30]. *None* of the epidemiological studies deemed by the courts below as "undermining" the experts' causation views concerned vehicle mechanics working on asbestos-containing brakes, clutches and gaskets. As Ford's epidemiological witness admitted regarding all such studies, there was no reason to suppose that they concerned any occupational groups other than "garage workers" who only pumped gasoline, only changed motor oil, only performed body work, only worked in a parts department, did muffler work or tow trucks tasks; and did not include a single worker who actually did vehicle brake, clutch or gasket work [A-1509 to 1514].[6]

In epidemiological research, the first task must always be to define the exposed group, either for purposes of supplying the study subjects (cohort study) or control group (case-control study). Over-simplifying, cohort studies follow exposed individuals to determine their incidence rate of disease, whereas in case-control studies, which identify the single disease of interest, it is critical to define exposure populations within the source population control group in order to supply the

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[6] *See also* 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").

statistical denominators. Hence, for example, albeit totally ignored by Ford and the decisions below, the leading text *Modern Epidemiology* explains:

> Case-control studies are best understood and conducted by defining a source population at the outset, which represents a hypothetical study population in which a cohort study might have been conducted, and by identifying a single disease of interest. . . . In a case-control study, these same cases are identified *and their exposure status is determined just as in a cohort study* . . . . The purpose of this control group is to *determine the relative size of the exposed and unexposed denominators* with the source population.

Kenneth J. Rothman, *et al., Types of Epidemiological Studies, in* MODERN EPIDEMIOLOGY 87, 95 (Rothman *et al.*, eds, Wolters Kluwer, 2008) (emphasis added).

Only one epidemiological study discussed at trial was demonstrated to have been relevant to the instant litigation. Ford is entirely incorrect in claiming that plaintiff did not "identify any reliable epidemiological evidence demonstrating that vehicle mechanics are at an increased risk of contracting mesothelioma" [D/B, at 30]. In this regard, Dr. Markowitz 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 (emphasis added)], 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].

In its instant Brief, however, Ford attempts to negate the significance of the Roelofs study by highlighting its expert's recitation of the study's routine caveat that it is not theoretically fully possible to rule out the possibility that certain of the mesothelioma-afflicted vehicle mechanics "were exposed to asbestos in a prior occupation such as shipbuilding . . . or in another occupation or exposure context not reported as *their usual occupation*" [A-1573 (emphasis added); D/B, at 12, 28].

Ford's point thereby rests on an exceedingly slim reed. The Roelofs study concluded that vehicle mechanics working with asbestos components, just as Mr. Juni did, were at greater than a double relative risk of contracting mesothelioma, to a 95 percent confidence level. The main, lifetime occupation of these study subjects was vehicle mechanic. The Roelofs study was thereby a further scientific expression supporting the experts' causation opinions. In the studies on which Ford relied on at trial, the main occupation of the study subject was not vehicle mechanic and, while useful for other purposes in the epidemiological community, the studies did not in

any way reflect the risks involved for mechanics working with asbestos-containing vehicle components.

C. FORD'S DOCUMENTARY ADMISSIONS FURTHER ATTEST TO THE SUFFICIENCY OF THE CAUSATION PROOF

In her main Brief, plaintiff showed that Ford possessed conclusive scientific data that mechanics working with high asbestos-content brakes, clutches and gaskets, precisely as Mr. Juni did, were being "overexposed" to ultra-carcinogenic asbestos fibers [A-2246, 905-06]. The jury received numerous internal documents wherein Ford admitted, based on mainstream scientific and industrial hygiene materials, that it 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 now protests that "none of the documents identified by the Junis amounts to an admission that vehicle mechanics are at an increased risk of contracting mesothelioma" [D/B, at 33]. Although the documents could properly have been admitted in evidence as substantive admissions of causation; *Cepeda v. A. C. & S., Inc.*, 2007 N.Y. Slip Op. 50584(U), 2007 WL 914540, at *2 (Sup. Ct., NY County, Mar. 5, 2007) (admitting into evidence as "admissions" Ford training materials addressing the hazards of asbestos); *People v. Campney*, 84 NY2d 307, 311-12

(1999); even if not admitted for the truth they impeached the credibility of Ford's denials of the carcinogenic properties of the asbestos-filled dusts emanating from their vehicle components. The documents further impeached the credibility of Ford's denials that such dusts contained "any asbestos" at all [A-11819].

By virtue of its internal documents, Ford "admitted" that vehicle-related work during which asbestos is "handled in any way which causes dust to become airborne" presents an extreme risks of cancer and mesothelioma, because "[a]sbestos is a recognized human carcinogen" [A-2098]. Ford's own internal statements to employees to the effect, for example, that, "[b]rake and clutch mechanisms on vehicles . . . utilize asbestos friction materials [resulting in o]verexposure to asbestos fiber . . . when compressed air is used to blow off dust" [A-2350], served to undermine the credibility of Ford's epidemiological witness suggesting that any such asbestos fibers would have been safe [A-1268 to 1269]. As just one further example, Ford's internal 1983 warning that "brake and clutch servicing of trucks," automobiles and other vehicles result in an "asbestos dust hazard [that] may cause asbestosis and cancer" [A-2104] shored up the credibility of plaintiffs' experts' critique of Ford's use of the irrelevant epidemiological studies.

Moreover, the internal Ford documents did, in fact, admit to the very sorts of research and studies upon which Dr. Markowitz relied, and hence had foundational significance [*compare* Dr. Markowitz's reliance upon Dr. Selikoff's work regarding the

use of ultrahazardous asbestos in brakes (A-237) *with* the July 1973 admissions of Ford's Industrial Hygiene supervisor (A-2246) regarding Dr. Selikoff's studies *in consultation with defendant Ford* demonstrating that "brake lining workers were exposed to significant levels of asbestos" (A-2333 to 2334, 907)].

CONCLUSION

For all of the reasons stated above and in plaintiff's main Brief, plaintiff respectfully submits that the Appellate Division's majority Decision and Order affirming the trial court's post-trial ruling should be reversed, the verdict reinstated, and either judgment entered in plaintiff's favor or the case remitted for disposition of any remaining issues raised in defendant's post-trial motion.

Dated: New York, New York
December 20, 2017

Respectfully submitted,
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CERTIFICATION
PURSUANT TO NYCRR § 500.13(C)(1)

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Dates: New York, New York
December 20, 2017

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