Matter of New York City Asbestos Litig.

2017 NY Slip Op 01523 [148 AD3d 233]

February 28, 2017

Saxe, J.

Appellate Division, First Department

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In the Matter of New York City Asbestos Litigation.

Mary Juni, Individually and as Administratrix of the Estate of Arthur H. Juni, Deceased, Appellant, v A.O. Smith Water Products Co. et al., Defendants, and Ford Motor Company, Respondent.

First Department, February 28, 2017

Matter of New York City Asbestos Litig., 48 Misc 3d 460, affirmed.

APPEARANCES OF COUNSEL

Weitz & Luxenberg, P.C., New York City (*Alani Golanski* of counsel), for appellant.

McGuire Woods LLP, New York City (*J. Tracy Walker, IV*, of the Virginia bar, admitted pro hac vice, and *Tennille J. Checkovich* of counsel), and *Aaronson Rappaport Feinstein & Deutsch, LLP*, New York City (*Nancy L. Pennie* and *Oded Burger* of counsel), for respondent.

[*2]*Malaby & Bradley, LLC*, New York City (*Robert C. Malaby* and *Maryellen Connor* of counsel), for amici curiae.

Linda Popejoy, Washington, D.C., and *William L. Anderson*, Washington, D.C., for the Coalition for Litigation Justice, Inc., amicus curiae.

{**148 AD3d at 235} OPINION OF THE COURT

Saxe, J.

This appeal requires us to address whether a plaintiff who seeks damages for contracting mesothelioma based on exposure to a defendant's asbestos-containing products must satisfy the standards expressed in *Parker v Mobil Oil Corp.* (7 NY3d 434 [2006]) and *Cornell v 360 W. 51st St. Realty, LLC* (22 NY3d 762 [2014]) by offering evidence that, if it does not provide an exact mathematical quantification of that exposure, at least provides some "scientific expression" (*Parker* at 449) of the level of exposure to toxins in defendant's products that was sufficient to have caused the disease.

Plantiff's decedent, Arthur Juni, commenced this personal injury action due to his mesothelioma allegedly caused by claimed exposure to asbestos-containing products while he worked as an auto mechanic. Juni died on March 16, 2014, after which his widow, Mary Juni, who also has a loss of consortium claim, was substituted as administratrix for Juni's estate. This appeal concerns only the trial of claims against defendant Ford Motor Company, based on Juni's exposure to asbestos over the years he worked on the brakes, clutches, and manifold gaskets of Ford vehicles, during which work, plaintiff says, asbestos dust was released.

After a trial in which a jury returned a verdict in plaintiff's favor, the trial court granted defendant Ford Motor Company's motion to set aside the verdict (48 Misc 3d 460 [2015]; CPLR 4404 [a]). We affirm that determination.

As the trial court recognized, under CPLR 4404 (a) the court may set aside a verdict or judgment entered after trial, and direct that judgment be entered in favor of a party entitled to judgment as a matter of law, if the verdict was not supported by legally sufficient evidence, since under those circumstances there is "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*, 45 NY2d 493, 499 [1978]).

[1] As the trial court pointed out, plaintiff was obliged to prove not only that Juni's mesothelioma was caused by exposure to asbestos, but that he was exposed to sufficient{**148 AD3d at 236} levels of the toxin from his work on brakes, clutches, or gaskets, sold or distributed by defendant, to have caused his illness. We agree with the trial court that the standards enunciated by *Parker* and *Cornell* are applicable here, that they are not altered by *Lustenring v AC&S, Inc.* (13 AD3d 69 [1st Dept 2004], *lv denied* 4 NY3d 708 [2005]) or other asbestos cases, and that plaintiff's evidence failed to satisfy that standard.

The Court of Appeals recently succinctly reiterated the standard in *Sean R. v BMW of N. Am., LLC* (26 NY3d 801 [2016]):

"In toxic tort cases, an expert opinion on causation must set forth (1) a plaintiff's exposure to a toxin, (2) that the toxin is capable of causing the particular injuries plaintiff suffered (general causation) and (3) that the plaintiff was exposed to sufficient levels of the toxin to cause such injuries (specific causation) (*see Parker v Mobil Oil Corp.*, 7 NY3d 434, 448 [2006]). Although it is 'not always necessary for a plaintiff to quantify exposure levels precisely' (*id.*), we have never 'dispensed with a plaintiff's burden to establish sufficient exposure to a substance to cause the claimed adverse health effect' (*Cornell v 360 W. 51st St. Realty, LLC*, 22 NY3d 762, 784 [2014]). 'At a minimum, . . . there must be [*3]evidence from which the factfinder can conclude that the plaintiff was exposed to levels of th[e] agent that are known to cause the kind of harm that

the plaintiff claims to have suffered' (*id.*, quoting *Wright v Willamette Indus., Inc.*, 91 F3d 1105, 1107 [8th Cir 1996])" (26 NY3d at 808-809).

Therefore, the fact that asbestos, or chrysotile, has been linked to mesothelioma, is not enough for a determination of liability against a particular defendant; a causation expert must still establish that the plaintiff was exposed to sufficient levels of the toxin from the defendant's products to have caused his disease (*see Sean R.*, 26 NY3d at 809). Even if it is not possible to quantify a plaintiff's exposure, causation from exposure to toxins in a defendant's product must be established through some scientific method, such as mathematical modeling based on a plaintiff's work history, or comparing the plaintiff's exposure with that of subjects of reported studies (*Parker* at 449).

[2] The evidence presented by plaintiff here was insufficient because it failed to establish that the decedent's mesothelioma{**148 AD3d at 237} was a result of his exposure to a sufficient quantity of asbestos in friction products sold or distributed by defendant Ford Motor Company. Plaintiff's experts effectively testified only in terms of an increased risk and association between asbestos and mesothelioma (*see Cornell*, 22 NY3d at 783-784), but failed to either quantify the decedent's exposure levels or otherwise provide any scientific expression of his exposure level with respect to Ford's products (*see Sean R.*, 26 NY3d at 809; *Parker*, 7 NY3d at 449).

While both of plaintiff's experts asserted that the asbestos in Ford's friction products was a cause of Juni's mesothelioma, the concessions made by both of plaintiff's experts so undermined their assertions of causation as to render those assertions groundless or unsupported. Dr. Jacqueline Moline, plaintiff's expert in internal medicine and occupational and environmental science, asserted that Juni's "cumulative exposures to asbestos caused his mesothelioma," referring to "the sum total of [his] exposure to asbestos . . . over [his] lifetime," but she admitted that "there were no measurements of what Mr. Juni was exposed to,"

noting that "[h]e was exposed in different locations where historically there have been mixed exposures," and that "[a]ll of his occupational exposures were substantial factors" contributing to his disease. Further, 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" was undermined when 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. In addition, Dr. Moline acknowledged that most chrysotile fibers in brake pads undergo a transformation during the braking process, and she did not know whether the fibers from the brake debris to which Juni was exposed were still active.

Plaintiff's other expert witness, Dr. Steven Markowitz, an internist, occupational medicine specialist, and epidemiologist, provided opinions that, after cross-examination, were similarly lacking in support on the issue of causation by Ford products. While he asserted that "chrysotile in friction products, if it becomes airborne and inhaled, can cause malignant mesothelioma" he acknowledged that 21 of 22 epidemiological studies that addressed asbestos exposure to mechanics working on friction products found no increased risk of mesothelioma. He also acknowledged that chrysotile has a curly and flexible{**148 AD3d at 238} structure, with shorter fibers, dissolves in the lungs, to an extent, and can clear the lungs through macrophages and translocation, and that when asbestos fibers in braking equipment are mixed with certain resins during manufacturing, "they would not be respirable." Further, Dr. Markowitz conceded 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.

[*4]

The trial court was at pains to point out that unlike here, in other litigation Dr. Markowitz has offered a scientific basis for claims that visible dust emanating from a particular defendant's asbestos-containing product contained enough asbestos dust to be hazardous, citing *Caruolo v John Crane, Inc.* (226 F3d 46 [2d Cir 2000]), in which Markowitz cited studies that measured the amount of asbestos fibers released by the products there at issue, and showed that the amount was hazardous. In contrast, no such supportive reports were offered at this trial. Rather, the reports or studies of mesothelioma in garage mechanics or those who work with friction products in a vehicle repair setting showed only an association between the work and mesothelioma.

Our dissenting colleague suggests that the proof in asbestos cases need not be analyzed using the same criteria as those we use to analyze exposure in other toxic tort cases, namely, the quantification or other "scientific expression of . . . exposure" required by *Parker*. The dissent also suggests that applying the same criteria would set an insurmountable standard for asbestos claims. However, there is no valid distinction to be made between the difficulty of establishing exposure to, say, benzene in gasoline and exposure to asbestos. In each type of matter, a foundation must be made to support an expert's conclusion regarding causation.

Moreover, our decisions in *Lustenring v AC&S, Inc.* (13 AD3d 69 [2004], *supra*) and other asbestos cases (*see e.g. Penn v Amchem Prods.*, 85 AD3d 475, 476 [1st Dept 2011]; *Matter of New York Asbestos Litig.*, 28 AD3d 255, 256 [1st Dept 2006]) do not justify allowing a judgment in an asbestos case to stand based solely on a bare conclusion that because the plaintiff worked with the defendant's asbestos-containing products, those products were a contributing cause of the plaintiff's mesothelioma. The rulings in each of those cases are based on their{**148 AD3d at 239} discrete facts. Where the courts relied on evidence linking visible dust to the use of the particular defendant's product,

expert testimony established that the extent and quantity of the dust to which the plaintiffs had been exposed contained enough asbestos to cause the mesothelioma. In none of those case was the mere presence of visible dust considered sufficient alone to prove causation. For instance, in *Lustenring*, the evidence established that "both plaintiffs worked all day for long periods in clouds of dust," which the expert testimony stated "necessarily contain[ed] enough asbestos to cause mesothelioma" (13 AD3d at 70). Here, in contrast to the expert testimony in *Penn v Amchem Prods.* (85 AD3d at 476), the testimony of plaintiff's expert as to the contents of the dust to which the decedent was exposed was equivocal at best, and was insufficient to prove that the dust to which Juni was exposed contained any asbestos, or enough to cause his mesothelioma.

The trial court also correctly declined to adopt plaintiff's theory of cumulative exposure to support the verdict. Neither of plaintiff's experts stated a basis for their assertion that even a single exposure to asbestos can be treated as contributing to causing an asbestos-related disease. Moreover, reliance on the theory of cumulative exposure, at least in the manner proposed by plaintiff, is irreconcilable with the rule requiring at least *some* quantification or means of assessing the amount, duration, and frequency of exposure to determine whether exposure was sufficient to be found a contributing cause of the disease (*see Parker*, 7 NY3d at 449).

The dissent references a "consensus from the medical and scientific communities that even low doses of asbestos exposure, above that in the ambient environment, are sufficient to cause mesothelioma." We do not agree that the existence of any such consensus entitles a particular plaintiff to be awarded judgment against a particular defendant by merely establishing some exposure to a product containing any amount of asbestos. Rather, the standards set by *Parker* and *Cornell*require that a plaintiff claiming that a defendant is

liable for causing his or her mesothelioma must still establish some scientific basis for a finding of causation attributable to the particular defendant's product. Here, the experts' broad conclusions on causation lacked a sufficient foundation, and were therefore legally insufficient to establish that Juni's exposure to asbestos from brakes, clutches, or gaskets sold or distributed by defendant constituted a [*5]significant contributing factor in causing Juni's{**148 AD3d at 240} mesothelioma. There is therefore no valid line of reasoning or permissible inference which could have led the jury to reach its result.

Accordingly the judgment of the Supreme Court, New York County (Barbara Jaffe, J.), entered June 3, 2015, in favor of defendant Ford Motor Company, should be affirmed, without costs. The appeal from the order of the same court and Justice, entered April 13, 2015, should be dismissed, without costs, as subsumed in the appeal from the judgment.

Concurring opinion by Kahn, J.

Kahn, J. (concurring). I join fully in the opinion of the majority, and write separately solely to address an important issue of our state's jurisprudence.

In contrast to the federal government and most of our sister states, New York does not base its law of evidence on statutorily codified rules. Instead, we rely principally upon the common law, as articulated by the Court of Appeals.

We additionally part ways with the majority of other jurisdictions in how our courts determine the admissibility of expert scientific testimony. Thus, we have not adopted any rules addressing the reliability of expert witness testimony comparable to those codified in Federal Rules of Evidence (FRE) rule 702.[FN1] Further, we are among the small minority of states that have not adopted the rule of *Daubert v Merrell Dow Pharmaceuticals, Inc.* (509 US 579 [1993]) requiring that all scientific testimony, whether novel or not, be based

upon reliable scientific principles properly applied, and charging the trial judge to act as the gatekeeper to ensure that result. Under *Daubert*, the expert witness must explain the application of the particular scientific principle to the facts at hand, ruling out alternative hypotheses and arriving at logical conclusions. Neither speculation nor generalized conclusions will pass muster under *Daubert*.{**148 AD3d at 241}

New York has consistently resisted adopting the *Daubert* standard as a means of assuring [*6]the reliability of scientific evidence put before our juries (*see e.g. Giordano v Market Am., Inc.*, 15 NY3d 590, 601 [2010]; *People v LeGrand*, 8 NY3d 449 [2007]; *People v Lee*, 96 NY2d 157 [2001]; *People v Wesley*, 83 NY2d 417, 435-436 [1994, Kaye, Ch. J., concurring]). With respect to the admissibility and sufficiency of evidence to prove causation in a long-latency toxic tort case, however, the New York Court of Appeals established its own standard a decade ago in *Parker v Mobil Oil Corp.* (7 NY3d 434 [2006]). There the Court acknowledged the tension present in cases involving long-latency personal injuries from exposure over time to toxic substances:

"As with any other type of expert evidence, we recognize the danger in allowing unreliable or speculative information (or 'junk science') to go before the jury with the weight of an impressively credentialed expert behind it. But, it is similarly inappropriate to set an insurmountable standard that would effectively deprive toxic tort plaintiffs of their day in court. It is necessary to find a balance between these two extremes" (*Parker*, 7 NY3d at 447).

To achieve this balance, the Court announced the following standard:

"It is well-established that an opinion on causation should set forth a plaintiff's exposure to a toxin, that the toxin is capable of causing the particular illness (general causation) and that plaintiff was exposed to sufficient levels of the toxin to cause the illness (specific causation) . . . . [It] is not always necessary for a plaintiff to quantify exposure levels precisely or use the dose-response relationship, provided that whatever methods an expert uses to establish

causation are generally accepted in the scientific community" (*Parker*, 7 NY3d at 448 [citations omitted]).

The *Parker* Court went on to suggest a nonexclusive list of alternative methods for proving causation in such cases that could satisfy its balancing test, including establishing the intensity of the plaintiff's exposure, estimations using mathematical modeling, or, in an appropriate case, qualitative comparison of the plaintiff's particular exposure level to the exposure levels of subjects in other studies (*Parker*, 7 NY3d at{**148 AD3d at 242} 449). Reviewing the case before it, however, the Court of Appeals, while acknowledging the plaintiff's exposure to the carcinogenic substance, rejected the plaintiff's expert evidence as too "general, subjective and conclusory," and lacking in specific relation to the plaintiff's exposures, to satisfy its announced standard (*id*.).

In this case, the dissent urges that this Court create an exception to the settled rule of *Parker* as to proof of causation to permit a finding of liability in asbestos friction product use cases through a plaintiff's unquantified cumulative exposures to "visible dust" which contained an unknown amount of chrysotile asbestos fibers,[FN2] based principally on the scientifically settled general association between asbestos exposure and mesothelioma and without evidence of either general or specific causation. However, were we to carve such a gaping hole in the *Parker* [*7]standard of proof on causation, eviscerating its fundamental evidentiary requirements, we would effectively overrule the Court of Appeals' holding in *Cornell v 360 W. 51st St. Realty, LLC* (22 NY3d 762, 783 [2014]), which explained that references to risk, linkage and association are not sufficient in themselves to establish causation in long-latency toxic exposure cases.

The approach urged by the dissent, regardless of its laudable goal in seeking compensation for injured workers, is not available to us. In view of the singular role of the Court of Appeals in advancing policy changes in the common law

(*cf. People v Keta*, 165 AD2d 172, 177 [2d Dept 1991] [recognizing "the policy and rule-making function traditionally perceived as the exclusive domain of the Court of Appeals"], *revd on other grounds sub nom. People v Scott*, 79 NY2d 474 [1992]; Hopkins, *The Role of an Intermediate Appellate Court*, 41 Brook L Rev 459, 460, 467 [1974-1975]), and given the key role the *Parker* rule plays in our state's evidence jurisprudence on expert witness testimony, any change in this regard must be made by the Court of Appeals.

Feinman, J. (dissenting). In this products liability action based on defendant Ford Motor Company's failure to warn{**148 AD3d at 243} decedent of the dangers of asbestos-containing products it sold or distributed, the jury returned a verdict in plaintiffs' favor after a trial lasting 20 days. Among its findings, the jury determined (1) that decedent Arthur Juni, Jr. was exposed to asbestos from products sold or distributed by Ford Motor Company, (2) that Ford failed to exercise reasonable care by not providing an adequate warning with respect to the hazards posed by such exposure, (3) that Ford's failure to warn was a substantial cause of his injury, and (4) that Ford acted with reckless disregard for the safety of others. While the jury found that decedent was also exposed to asbestos from products or equipment manufactured, sold, distributed or used by some other entities, except for nonparty Orange & Rockland Utilities (O&R), it found that those other companies did not fail to exercise reasonable care by not providing decedent with adequate warnings, or that those companies' failures to warn were not a substantial contributing factor in causing decedent's injuries. The jury allocated fault, 49% to Ford and 51% to O&R, and awarded money damages.

The trial court granted defendant Ford's postverdict motion to set aside the verdict, and dismissed the complaint, on the ground that there was legally insufficient evidence to establish that the decedent developed mesothelioma as a result of his exposure to asbestos-containing friction products sold or distributed

by Ford while he was working as an automobile mechanic. The majority now affirms. Because the trial court misapplied the standard of review for legal sufficiency, and misapplied the law concerning general and specific causation in asbestos cases, I would reverse and remand the matter to the trial court to determine the branches of Ford's postverdict motion it did not reach.[FN1] Simply put, affording the plaintiffs the benefit of every favorable inference, as we must when reviewing the legal sufficiency of the evidence, the jury's verdict was not "utterly irrational." Because the trial court and the majority have, in the guise of a legal sufficiency analysis, inappropriately substituted their assessment of the credibility of the witnesses for that of the jury, I dissent.

Trial Evidence

Decedent Arthur Juni, Jr. was diagnosed with mesothelioma in 2012 and died in early [*8]2014. At trial, his deposition was{**148 AD3d at 244} read to the jury. According to Juni, he worked from 1966 to 2009 as an auto mechanic in two garages owned by O&R, servicing predominantly Ford vehicles. For more than 25 years, he was exposed directly and indirectly to 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. In 1988, he was issued a respirator, and plaintiffs made no claim of respirable asbestos exposure thereafter.

Juni had previous exposure to asbestos when he worked as a driver for O&R in the summers of 1961, 1962 and 1963, because his truck was housed in a machine shop accessed through a one-room power station described by Juni as having asbestos "all over the place," on the floor and "everywhere." In addition, he was exposed to asbestos in 1963 and 1964, when he worked as a courier for O&R, delivering packages to the many company offices, one of which was at the same power station with the asbestos on the floor.

The rest of Juni's long work life was as a mechanic in the two O&R-owned garages. He began as a third-grade mechanic in 1964 in O&R's Nyack garage working the night shift. His primary work was pumping gas, changing oil and other regular maintenance of vehicles, but he was exposed to the dust from the work of other mechanics doing brake and clutch work on various types of mostly Ford vehicles. Juni's duties included sweeping up the asbestos-laden dust generated by the other mechanics.

In 1966, he was transferred to a much larger garage owned by O&R, and began as a second-class mechanic working at night where he helped service a fleet of up to 500 mostly Ford vehicles. On a weekly basis, he performed brake work, removing and dumping brake drums from Ford trucks to the floor, which raised asbestos-filled brake dust. He was promoted to first-class mechanic and became a foreman in 1970. Juni stated that he performed a lot of welding work for a couple of years, using an asbestos blanket for protection. He also installed new Ford brakes and removed old Ford brakes from a variety of Ford vehicles, and installed other brands of brakes in Ford vehicles and in other non-Ford trucks. The packaging of new brakes contained asbestos, and when Juni opened the packages, asbestos dust was released into the air. Sometimes he scuffed new brakes with sandpaper, which also released dust into the air, although he described this as a "quick process." {**148 AD3d at 245}Before 1979, Juni assisted with clutch work about once every three months, but thereafter he worked on a weekly basis with clutches, the bell housings of which contained asbestos, for Ford's 1979 C-8000 cab-over bucket trucks. The Ford clutches of the Ford trucks had to be replaced about once a month, and on average, a clutch job took about four hours. About once a month, he replaced clutches made by various companies on non-Ford backhoes. He did clutch work for 10 years.

He replaced Ford manifold gaskets in the Ford C-8000 cab-over bucket trucks. This involved taking the engine apart and removing and cleaning the

gaskets. To clean these parts, Juni and the other mechanics used a drill with a special brush that scraped off the dirt. Sometimes they used compressed air to clean the parts. Both processes spewed dust containing asbestos into the air.

In addition to Juni's deposition testimony and a video that showed his pain and suffering, the jury heard voluminous and sometimes quite technical scientific and statistical testimony over the course of 20 days from the parties' epidemiologists, toxicologists, medical doctors with specialization in occupational and environmental science, and others. They heard that mesothelioma is a rare and deadly disease, caused almost exclusively by respirable asbestos, and can take decades to manifest, but then killing within a year or two at the most. They heard that Ford's brakes, clutches, and gaskets (often referred to as friction products) were manufactured with asbestos, of a type called chrysotile, which is short-fibered and curly, and when inhaled can dissolve in the lungs or clear the lungs. The parties disputed its toxicity. Plaintiffs' epidemiologist, Steven Markowitz, M.D., testified that chrysotile is less harmful than other forms [*9]of asbestos, but is nonetheless hazardous. Defendant's toxicologist, Brent Finley, Ph.D., opined that it is not hazardous, but conceded that scientific organizations, including the National Institute for Occupational Safety and Health and the Occupational Safety and Health Administration, classify it as a human carcinogen. Documents issued internally by Ford, in the 1970s in particular, discuss the growing understanding of the health hazard associated with brake linings to workers installing and cleaning brakes in automobile repair shops, as well as in the automotive industry.

Plaintiffs offered Dr. Markowitz to establish that chrysotile asbestos in friction products, if allowed to become airborne and inhaled, can cause mesothelioma (general causation). His{**148 AD3d at 246} opinions were 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,[FN2] 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.

Dr. Markowitz testified that new friction products generally require beveling or shaving for proper placement in the vehicles. Mechanics are exposed to asbestos fibers in this process. They are also exposed when cleaning used products of dust and grime, including loosened asbestos, especially if cleaned with compressed air, but also with sandpaper, because asbestos-containing dust is released into the air. Asbestos dust is also shaken loose and becomes airborne when brake drums are dropped to the floor in the normal course of work.

Dr. Finley, defendant's toxicologist, testified that new brake linings are comprised of 50% asbestos by weight. Thus, if a truck brake lining weighed 12 pounds, 6 of those pounds would be asbestos, containing "billions" of fibers. He also testified that as to brakes and brake linings, although apparently *not* as to clutches or gaskets, chrysotile asbestos degrades with use due to the ongoing friction and heat from the braking process. It transforms into a nontoxic chemical called forsterite. According to Finley, there remains little to no actual asbestos fibers in used brake liners.[FN3]

Dr. Markowitz testified that even with degradation in used brakes, from one to about three percent asbestos remains present, and even this small amount contains millions of active fibers. He agreed that if asbestos fibers were mixed with certain resins during the brake manufacturing process, they would become nonrespirable, although it was unclear whether resins were used in manufacturing brakes and brake linings in the{**148 AD3d at 247} 1960s and

1970s, or a more recent addition. For example, plaintiffs later pointed to a study initiated in 1975 following a meeting between Ford and government agencies, among others, which stated that used brakes contain "typical chrysotile asbestos fibers along with a wide range of other forms of crystalline and fibrous materials."

[*10]

The jury heard from both parties' experts that studies have been undertaken to determine whether workers in certain environments where asbestos was typically and/or necessarily present, or who were engaged in certain asbestos-related work, were at a greater risk of asbestos-related disease. Defendant noted that there are 21 epidemiological studies finding no increased risk of mesothelioma among garage workers exposed to chrysotile asbestos. One recent study of brake mechanics showed a statistically significant risk, although the study was of a different type than the others.

Dr. Markowitz testified that in his opinion, the 21 studies were not relevant to Juni's work situation. He explained that there simply are no studies that have specifically looked at whether garage mechanics who regularly work with brakes and other friction products develop mesothelioma at a higher rate than others. The reasons are varied. One is that the number of garage mechanics working with friction products is relatively small and it is accordingly difficult to assemble a large enough number to undertake an occupational or cohort study. The majority of studies on this topic have used an alternative acceptable epidemiologic study, the case-control study, which compares people who develop mesothelioma and those who do not. However, until relatively recently, there was no medical classification for mesothelioma as a cause of death, thus skewing statistics to underreport deaths from mesothelioma. Such studies would take decades to complete, given the long latency from exposure to disease manifestation. Additionally, the fact that there is only a year or two at most from

the onset of mesothelioma until death further hampers the ability to collect high quality data concerning work life and exposure to asbestos products.

Dr. Markowitz's discussion of the studies was partly corroborated by defendant's occupational epidemiologist, Mary Jane Teta, Ph.D., who stated she was not aware that any of those studies were designed to track garage mechanics working with asbestos-containing friction products. The jury heard that Dr. Teta herself had conducted a study of "automobile {**148 AD3d at 248}repair and related services," in the 1980s, which found that working with friction products did not enhance developing mesothelioma. However, upon questioning, she conceded that she did not know whether any of her study's subjects were actually vehicle mechanics, and there may not have been any included.

Plaintiffs also offered Jacqueline Moline, M.D., an internal medicine and occupational and environmental science expert, to establish specific causation. She testified that based on her review of Juni's medical records and deposition testimony about the particulars of his work life, in her medical opinion, Juni had died from mesothelioma caused by cumulative exposures to asbestos. Dr. Moline's opinion was based on her extensive background with patients with asbestos exposure who had also worked with brakes and clutches and had similar descriptions of their exposures as Juni's; her knowledge of industrial hygiene studies finding elevated levels of dust from the manipulation of brakes; medical and scientific literature; animal studies; and studies by various professional, national and international organizations.

Dr. Moline agreed that there were no measurements to quantify Juni's direct or indirect exposure to asbestos dust, at any location. She explained that Juni worked in locations where "historically" there have been various kinds of exposures to asbestos. It was the cumulative exposure to asbestos-containing products, over more than two decades of work, that was a substantial

contributing factor in causing his disease, she concluded. It is not possible to pinpoint which particular exposure to asbestos caused his mesothelioma. However, she explained, a general consensus in the scientific community has developed that there are indicia of dangerous levels of exposure to asbestos, in particular the presence of visible dust generated in the course of working with asbestos products. Visible dust, Dr. Moline explained, is "a surrogate" for an amount of asbestos dust known to be capable of causing mesothelioma, although it is expected that even at very low levels of exposure, with no dust visible, there is an increased risk of cancer. [*11]Juni's testimony, she noted, was that during his many years of working at the garages, he had breathed in visible dust generated in the course of his work and from the work of other mechanics. This alone is sufficient to establish that his work with the friction products, and the presence of other workers generating dust by working with asbestos products, caused Juni's mesothelioma. She agreed, however, that dust generated from friction{**148 AD3d at 249} products that did not contain asbestos would not contain asbestos and would not be a cause of mesothelioma. The majority suggests that her testimony that the visibility of dust was itself proof that there was a hazardous level of respirable asbestos was fatally undermined by her statement that studies have shown that there is only about one percent of asbestos fibers in worn brakes. This, however, merely reflects the existence of competing evidence to be assessed by the jury.

Dr. Markowitz testified in the same vein that there is no safe level of asbestos exposure. Exposure to asbestos causes an increased risk of disease, and it is the cumulative exposures that can ultimately cause disease.

Defendant strenuously disagreed. Its toxicologist, Dr. Finley, offered a different theory, that for every chemical, there is a dose below which there is no effect, and that chrysotile asbestos-exposure associated with brake repair is too low to cause any increase in the risk of disease.[FN4] In

contrast, *defendant's* occupational epidemiologist, Dr. Teta, testified that there is no known safe level of exposure to asbestos, an opinion in accord with Dr. Moline's.

The jury had before it internal documents, as mentioned above, from Ford written in the years 1973-1983 concerning asbestos found in a variety of automotive components, and worker safety. The documents show that in the early 1970s, Ford was aware, based on troubling air samples, that workers who did brake and clutch repairs in particular were exposed to a much greater risk of developing cancer and mesothelioma than other workers. Ford introduced internal safety procedures to reduce the amount of asbestos released into the air and to contain the asbestos to certain areas. It directed that the use of compressed air and dry brushing to clean brakes and clutches be completely discontinued. An August 1983 bulletin, from Ford's Employee Health Services, warned of the risks of asbestos dust, including mesothelioma, from clutch and brake linings. The bulletin described asbestos fibers as "nearly indestructible" and indicated that a potential health risk arises whenever asbestos fibers are released into the air as dust. Plaintiffs contended that Juni and his coworkers were not informed by Ford of the findings of its internal studies and recommendations.{**148 AD3d at 250}

Following the close of testimony, the trial court charged the jury that "there may be more than one cause of an injury," and the cause may be "substantial even if you assign a relatively small percentage to it." The jury found Ford 49% liable for causing Juni's mesothelioma.

Ford moved, posttrial, to vacate the verdict and dismiss the complaint as a matter of law, based on legal insufficiency. In the alternative, defendant sought to have the verdict set aside as against the weight of the evidence and a new trial ordered and/or other relief. The trial court granted the first branch of the motion, finding that plaintiffs had not introduced sufficient evidence tending to show

that Arthur Juni's exposure to asbestos from Ford's brakes, clutches, and gaskets was a significant contributing factor in causing his mesothelioma.

Discussion

The burden on a movant seeking to have a jury verdict set aside and judgment entered in favor of the moving party under CPLR 4404 (a) is a heavy one. A court must exercise [*12]considerable deference in exercising its discretionary power to set aside a jury verdict (*see Rose v Conte*, 107 AD3d 481, 484 [1st Dept 2013]). A jury verdict in favor of a party should not be set aside unless that jury "could not have reached the verdict upon any fair interpretation of the evidence" (*Lichtenstein v Bauer*, 203 AD2d 89, 89 [1st Dept 1994]; *see also Jackson v Mungo One*, 6 AD3d 236 [1st Dept 2004]). The court must view the evidence in a light most favorable to the nonmovant (*see Szczerbiak v Pilat*, 90 NY2d 553, 556 [1997]). The movant must persuade the court that there was "simply no valid line of reasoning and permissible inferences which could possibly lead rational [persons] to the conclusion reached by the jury on the basis of the evidence presented at trial" (*Cohen v Hallmark Cards*, 45 NY2d 493, 499 [1978]). Importantly, if 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" (*id.*; *see also Blum v Fresh Grown Preserve Corp.*, 292 NY 241, 245 [1944]; *Guiton v Gottlieb*, 236 AD2d 203 [1st Dept 1997]). The Court of Appeals has very recently reiterated the movant's high burden in *Killon v Parrotta* (28 NY3d 101 [2016]), again holding that a jury verdict is insufficient only when it is "utterly irrational" (*id.* at 108).

{**148 AD3d at 251}This litigation was essentially a battle of the experts. The parties produced conflicting expert evidence as to whether chrysotile asbestos in friction products can cause disease, whether asbestos causes disease

by cumulative exposures or only after a certain amount of exposure, and whether Juni had been exposed to a sufficient level of asbestos from Ford's products to cause his mesothelioma, among other issues. The jurors were also presented with differing methods to weigh the evidence. It is well established that it is within the province of the jury to reject or accept an expert's testimony in whole or in part; the weight to be given to opinion evidence and expert evidence is ordinarily entirely for the jury's determination (*see Matter of City of New York [Fifth Ave. Coach Lines]*, 22 NY2d 613, 630 [1968]).

A major issue before the jury, and the issue on appeal, was causation. *Parker v Mobil Oil Corp.* (7 NY3d 434 [2006]), addressing a claim that years of workplace exposure to benzene in gasoline had caused acute myelogenous leukemia, articulates what has become a well-established rule in New York in toxic tort cases, namely that "an opinion on causation should set forth a plaintiff's exposure to a toxin, that the toxin is capable of causing the particular illness (general causation) and that plaintiff was exposed to sufficient levels of the toxin to cause the illness (specific causation)" (*id.* at 448). *Cornell v 360 W. 51st St. Realty, LLC* (22 NY3d 762 [2014]), concerning a claim of illness from interior mold, holds that it is the plaintiff's burden "to establish sufficient exposure to a substance to cause the claimed adverse health effect" (22 NY3d at 784, citing *Parker* at 449). *Cornell* stated that, " '[a]t a minimum, . . . there must be evidence from which the factfinder can conclude that the plaintiff was exposed to levels of th[e] agent that are known to cause the kind of harm that the plaintiff claims to have suffered' " (*Cornell* at 784, quoting *Wright v Willamette Indus., Inc.*, 91 F3d 1105, 1107 [8th Cir 1996]).

New York's highest Court has not specifically addressed the sufficiency of proof needed to establish causation in an asbestos claim. This Court, and others, have accepted a consensus from the medical and scientific communities that even low doses of asbestos exposure, above that in the ambient environment, are

sufficient to cause mesothelioma. Mesothelioma is a "rare and deadly cancer," with 70 to 80% of all cases reporting a history of asbestos exposure at work (*Matter of New York City Asbestos Litig.*, 5 NY3d 486, 490 n 2 [2005]; *see also Dollas v Grace &*{**148 AD3d at 252} *Co.*, 225 AD2d 319, 320 [1st Dept 1996], quoting *O'Brien v National Gypsum Co.*, 944 F2d 69, 72 [2d Cir 1991] [mesothelioma is " 'an exceedingly rare disease . . . whose only known cause is exposure to asbestos' "]). We have been persuaded that medical science has been unable to determine a minimum level of exposure to asbestos below which it is not disease producing; every inhalation of visible asbestos-laden dust contributes to the cumulative dose responsible for [*13]the mesothelioma. Because of the lengthy gap between exposure and manifestation of disease, we recognize the difficulty in determining which of several asbestos-containing products used by a plaintiff was the cause of the disease or to what degree, and both before and subsequent to *Parker*, we have accepted not only an established link between asbestos and mesothelioma, but that visible dust released from an asbestos product contains high levels of fibers of asbestos capable of producing disease.

The leading case directly concerning the sufficiency of the evidence in a claim of asbestos exposure is *Lustenring v AC&S, Inc.* (13 AD3d 69 [1st Dept 2004], *lv denied* 4 NY3d 708 [2005]), decided prior to *Parker*. While *Lustenring* predates *Parker*, I do not read it as being in conflict with *Parker*, and maintain that it should continue to be followed.

In *Lustenring*, competent evidence was provided of long periods of daily working in dust laden with asbestos generated from products containing asbestos, and valid expert testimony that dust raised from manipulating asbestos products "necessarily" contains enough asbestos to cause mesothelioma (13 AD3d at 70). This Court agreed that there was no need for a *Frye* hearing because it is sufficiently established that visible dust from asbestos and

asbestos-containing products contains hazardous levels of asbestos, and allows a physician expert to testify that the product was a substantial factor in causing the disease. There was no novel scientific technique or application of science at issue, and it was the jury's duty to determine the credibility of the plaintiffs' arguments concerning causation as compared to the defendants' arguments.

Subsequent decisions from trial courts and this Department often, but not always, reference *Lustenring*, and even when they do not, they employ the same understanding that a plaintiff's expert's opinion is based on established scientific opinion supplemented by the plaintiff's particular history of exposure (*see e.g. Matter of New York City Asbestos Litig.*, 24 AD3d 375, 375-376 [1st Dept 2005] [holding that "(t)he link between {**148 AD3d at 253}asbestos and disease is well documented"; jury to decide whether the asbestos contained in the defendant's product could be released in respirable form so as to cause disease]; *Matter of New York Asbestos Litig.*, 28 AD3d 255, 256 [1st Dept 2006] [regular exposure to dust from working with the defendant's asbestos-containing gaskets and packing; citing *Lustenring*]; *Matter of New York City Asbestos Litig.*, 143 AD3d 483 [1st Dept 2016] [electrician exposed to boilers and their concomitant dust, as well as dust created from mixing asbestos concrete powder which filled the air and settled on everything; citing *Lustenring*]; *Matter of New York City Asbestos Litig.*, 143 AD3d 485 [1st Dept 2016] [mechanic and electrician removed asbestos-containing insulation from valves, and mixed asbestos-containing insulation cement, both generating visible asbestos dust; citing *Lustenring*]).

In *Penn v Amchem Prods.* (85 AD3d 475 [1st Dept 2011]), cited by the majority as an example of a decision determining causation based on more than "the mere presence of visible dust," there was testimony by the plaintiff of visible dust emanating from working with asbestos-laden dental liners, and expert testimony that this dust "must have contained" enough asbestos to cause

the plaintiff's mesothelioma.[FN5] Such expert testimony is very similar to the testimony provided by plaintiffs' experts here, which was enough to show a scientific expression of the level of plaintiff's exposure, and Ford's liability.

I would note that we have held that a plaintiff need not show the precise causes of the decedent's damages, but only facts and conditions from which the defendant's liability can be reasonably inferred (*see Lloyd v W.R. Grace & Co.—Conn.*, 215 AD2d 177 [1st Dept 1995]; *see [*14]also Matter of New York City Asbestos Litig.*, 116 AD3d 545 [1st Dept 2014]). A plaintiff must provide evidence of a link between the claimed negligence and his or her injuries; in a product liability case, there must be shown a link between the injuries and a manufacturer's defectively designed product (*see Miller v Akronchem Corp.*, 276 AD2d 447 [1st Dept 2000], *lv denied* 96 NY2d 716 [2001]).

The majority agrees with the trial court that the proof in a claim involving disease caused by asbestos must be analyzed using the same method as that used to analyze exposure to{**148 AD3d at 254} benzene in gasoline, and or interior mold—*Parker* and *Cornell*, respectively. The trial court acknowledged that mesothelioma is caused only by asbestos exposure, but framed the question as "whether chrysotile asbestos, as contained within friction products, causes mesothelioma, an issue closely analogous to that addressed in *Parker*, namely, whether benzene, as contained in gasoline, causes [acute myelogenous leukemia]."

Defendant maintained that Juni's "regular" exposure to Ford-manufactured or distributed friction products required a quantification of the exposure and because plaintiffs' experts did not and could not quantify the dosage of decedent's exposure to asbestos from Ford products, they had not shown a scientific expression of Juni's exposure. Ford further argued that a "link" or an "association" between asbestos and mesothelioma is not, in itself, sufficient to

establish a foundation for an expert's opinion, and the presence of dust does not, in itself, prove that a hazardous dosage of asbestos fibers was inhaled.

Defendant posits a much too narrow foundation for establishing causation in an asbestos claim, which, if the trial court and the majority are correct, means no asbestos litigant will be able to prevail. *Parker* explicitly recognized that in toxic tort cases it is often "difficult or impossible to quantify" a plaintiff's exposure to the toxin (7 NY3d at 447). The Court explained that as long as the plaintiff's experts use a method generally accepted in the scientific community to establish causation, it "is not always necessary" to posit an "exact number" for the amount to which the plaintiff was exposed (*id.* at 448).[FN6] The method need not be quantification or dose-response analysis, and may include "qualitative" methods; the Court recognized that it is "inappropriate to set an insurmountable standard that would effectively deprive toxic tort plaintiffs of their day in court" (7 NY3d at 447).

The concurrence characterizes my position as creating an "exception" to *Parker*'s rule, in order to accommodate the specifics of asbestos claims. I disagree. I am merely reaffirming what has been for many years the basis of this Department's decisions in asbestos cases, a well-considered method for determining liability when the claim is injury caused by exposure to{**148 AD3d at 255} asbestos-containing products. It is a fact that, in most, if not all, asbestos exposure cases, numerical quantification of a plaintiff's exposure would be impossible. Because such quantification is not required either by the precedents of this Court or the Court of Appeals, the trial court's imposition of such a requirement, affirmed by a majority of this Court, represents an abrupt rupture in the asbestos jurisprudence of this state (*see* Michael Hoenig, Complex Litigation, *Ruling on Asbestos Experts a Potential Game Changer*, NYLJ, May 11, [*15]2015). This wrong turn ignores that mesothelioma takes decades to manifest, and the victim is generally long retired from the workplace where

exposure occurred. Witnesses have died. Work sites very often no longer exist, and when operational, most did not monitor and make records of air quality. The subject materials or products containing asbestos are no longer in existence and cannot be tested.

For these reasons, the standard being adopted by the majority erects an insurmountable hurdle requiring plaintiffs to recreate the work environment, to establish precise exposure levels, dust and fiber counts, air quality levels throughout the day, and so on, or to test the asbestos-containing materials or items so as to demonstrate how much asbestos was present and subject to release into the air through the work process, becoming respirable. Indeed, this is why experts in asbestos litigation rely not only on the clear link between mesothelioma and asbestos, but indicia such as the visible presence of asbestos-containing dust to establish quantity. As noted by plaintiffs, the well respected Reference Manual on Scientific Evidence (3d ed 2011), compiled by the Federal Judicial Center and National Research Council of the National Academies, indicates in a footnote 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 n 111, citing *Lohrmann v Pittsburgh Corning Corp.*, 782 F2d 1156, 1162-1164 [4th Cir 1986], and *Gregg v V-J Auto Parts, Co.*, 596 Pa 274, 291, 943 A2d 216, 226 [2007]). That is what occurred at this trial.

The jury's verdict finding Ford 49% responsible for causing decedent's mesothelioma after 25 years of exposure to asbestos-containing products sold or distributed by Ford was based on a fair interpretation of the totality of the evidence and an assessment{**148 AD3d at 256} of the credibility of the experts, and was not "utterly irrational" (*Killon*, 28 NY3d at 108). The evidence, viewed in the light most favorable to plaintiffs, was legally sufficient and the

disputed issues were properly submitted to the jury for factual determination. By setting aside this verdict the trial court and the majority have usurped the jury's function and redefined the nature of proof required to establish specific causation in asbestos cases.

Accordingly, I would reverse the trial court's order setting aside the verdict on the grounds of legal insufficiency and would remand this matter for consideration of the remaining grounds for posttrial relief sought by Ford, but not addressed by the trial court (*see Stewartson v Gristede's Supermarket*, 271 AD2d 324, 325 [1st Dept 2000]).

Tom, J.P., concurs with Saxe, J.; Kahn, J., concurs in a separate opinion; Feinman, J., dissents in a separate opinion.

Judgment, Supreme Court, New York County, entered June 3, 2015, affirmed, without costs. Appeal from order, same court and Justice, entered April 13, 2015, dismissed, without costs.

Footnotes

Footnote 1:FRE 702 provides:

"A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if:

"(a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;

"(b) the testimony is based on sufficient facts or data;

"(c) the testimony is the product of reliable principles and methods; and

"(d) the expert has reliably applied the principles and methods to the facts of the case."

Footnote 2: Typically, chrysotile asbestos fibers are less than two microns in length and are, therefore, not visible to the naked eye (*see* BioMed Central, Environmental Health, *Quantification of Short and Long Asbestos Fibers to Assess Asbestos Exposure: A Review of Fiber Size Toxicity* [July 21, 2014], available at https://ehjournal.biomedcentral.com/articles/10.1186/1476-069X-13-59 [accessed Feb. 10, 2017]).

Footnote 1: The remaining branches in Ford's motion seek a weight of the evidence review and a new trial (as opposed to dismissal), renewal of the court's prior order on the consolidation motion, remittitur, and offsets from settlements prior to entry of judgment.

Footnote 2: Dr. Markowitz explained that a case series is a report published in the medical literature focusing on persons in the same industry with a particular disease and suggesting a causal relationship between the disease and the industry.

Footnote 3: It was unclear whether Dr. Finley was testifying about modern brakes and brake linings, or brakes and brake linings produced and used in the 1960s and 1970s, the health hazards of which were becoming apparent in the 1970s, as seen in internal Ford documents discussed below.

Footnote 4: It bears noting that defendant attempted to question whether Juni's mesothelioma was caused by his exposure to asbestos in the 41/2 years working as a driver and a courier in the early 1960s, before he began working with Ford products at the O&R garages.

Footnote 5: As to the differences between the plaintiff's description of the dental liners and the codefendant representatives' description, we held it "simply raised a credibility issue for the jury" (*id.* at 476).

Footnote 6: We have held that "general acceptance" does not "necessarily mean that a majority of the scientists involved subscribe to the conclusion, but that those espousing the theory or opinion have followed generally accepted scientific principles and methodology in reaching their conclusions" (*Nonnon v City of New York*, 88 AD3d 384, 394 [1st Dept 2011]).