SUPREME COURT OF THE STATE OF NEW YORK
COUNTY OF NEW YORK : IAS PART 12

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IN RE NEW YORK CITY ASBESTOS LITIGATION

Index No. 190315/12

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ARTHUR H. JUNI and MARY JUNI,

Mot. seq. no. 018

Plaintiffs,

DECISION AND ORDER

- against -

A.O. SMITH WATER PRODUCTS, *et al.*,

Defendants.

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BARBARA JAFFE, JSC:

For plaintiffs:
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For Ford Motor Co.:
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Defendant Ford Motor Company moves post-trial for orders: (1) striking the causation opinions of plaintiffs' expert witnesses, and (2) dismissing the action and entering judgment as a matter of law in favor of it based on plaintiffs' failure to establish a *prima facie* case at trial, or, alternatively (3) setting aside the verdict rendered against it at trial and granting a new trial; (4) granting it leave to renew its opposition to plaintiffs' motion to consolidate and upon renewal, denying the motion to consolidate and granting a new trial; (5) setting aside and remitting the verdict as excessive and contrary to the weight of the evidence; and (6) reducing the verdict by offsets from settlements before entering judgment. Plaintiffs oppose.

I. BACKGROUND AND TRIAL

Plaintiffs sued defendant, and others who have since settled, claiming that exposure to

asbestos from products manufactured or used by them or used at their premises caused plaintiff Arthur Juni (Juni) to develop and die from mesothelioma. The trial of this action was consolidated with two other actions, *Karl Fersch and Anna Fersch v Amchem Products, Inc., et al.*, Index No. 190468/12, and *Darryl W. Middleton and Belinda Middleton v Amchem Products, Inc., et al.*, Index No. 190367/12. Prior to trial, I granted defendant Volkswagen of America's motion for an order precluding expert testimony in the *Fersch* matter to the extent of ordering a hearing pursuant to *Parker v Mobil Oil Corp*, 7 NY3d 434 (2006). Before the hearing commenced, the Fersch plaintiffs settled their claims against Volkswagen.

A jury trial commenced, soon after which the Middleton plaintiffs discontinued their case in its entirety. Thus, the trial proceeded to verdict only in *Juni* and only as against defendant. After plaintiffs rested, defendant moved for an order striking the causation testimony of plaintiffs' experts and for a directed verdict based on the insufficiency of the evidence. I reserved decision.

At the charge conference, the parties agreed that the jury would be asked whether Juni was exposed to asbestos from brakes, clutches, or gaskets sold or distributed by defendant, and would be presented with three alternative theories of liability against defendant: (1) common law negligence, (2) strict products liability (failure to warn), and (3) products liability (negligence). While plaintiffs conceded that "[defendant] didn't manufacture brakes, clutches or gaskets . . . [defendant] manufactured cars," they argued that defendant could additionally be held liable for Juni's exposure to asbestos-containing replacement parts used in its vehicles. (Tr. 2396). Absent any evidence that defendant intended or required, within the meaning of *Berkowitz v A.C. and S., Inc.*, 288 AD2d 148 (1st Dept 2001), that asbestos-containing replacement components be used in

its vehicles, I declined to instruct the jury on whether defendant failed to warn Juni of the danger of components used in its vehicles. (Tr. 2401). (*See also Matter of New York City Asbestos Litig. [Dummitt]*, 121 AD3d 230, 251-252 [1st Dept 2014], *lv granted* 2014 NY Slip Op 92113[U] [no duty to warn absent evidence that defendant had active role, interest, or influence in types of products to be used with own product after placing it into stream of commerce]).

The jury rendered its verdict finding that: (1) Juni was exposed to asbestos from brakes, clutches, or gaskets sold or distributed by defendant; (2) defendant failed to exercise reasonable care by not providing an adequate warning about the hazards of exposure to asbestos with respect to the use of the brakes, clutches, or gaskets; and (3) defendant's failure to warn Juni adequately was a substantial contributing factor in causing his injury. It then considered whether liability should be apportioned among the 16 other entities listed on the verdict sheet, and found that Juni had been exposed to asbestos in all 16 of the other entities' products or by use of their products, but that only one of them, non-party Orange & Rockland Utilities (Orange and Rockland), had failed to exercise reasonable care by not providing an adequate warning about the potential hazard of exposure to asbestos, and that its failure to warn adequately was a substantial contributing factor in causing Juni's injury.

After apportioning 49 percent of the liability to defendant and 51 percent to Orange and Rockland, the jury found that defendant had acted recklessly. It awarded Juni $8 million for his pain and suffering from the onset of his symptoms to his death on March 15, 2014, and to plaintiff Mary Juni $3 million for her loss of consortium.

A. Juni's pertinent testimony

Juni's deposition testimony was read to the jury. Beginning in 1964, he worked for

Orange and Rockland in its Nyack garage as a third-class mechanic. (Tr. 623).

As a third-class mechanic, he pumped gas, changed oil, and greased vehicle parts. As a second-class mechanic, he changed car tires and assisted with one brake job a week. (Tr. 624, 625, 646).

First-class mechanics worked on brakes. (Tr. 625). In assisting them, Juni jacked up the vehicles and removed the tires. (Tr. 629). In removing the brake drums, the mechanics dropped them on the ground, leaving brake dust that Juni swept up each night. (Tr. 631, 1097-1099). Juni also assisted the first-class mechanics with clutch replacement. (Tr. 638). On defendant's F-600s, for example, first-class mechanics would remove the bell housing, thereby producing clutch dust. (Tr. 637).

The Nyack garage serviced different kinds of vehicles, including bucket trucks and defendant's dump trucks and service vans, on which mechanics would install replacement brakes. (Tr. 626, 627, 628). Juni also assisted the first-class mechanics with replacing the clutches on defendant's vehicles. (Tr. 632, 633, 639, 640).

In 1966, Juni began working at Orange and Rockland's Spring Valley garage as a second-class mechanic, performing weekly brake work (Tr. 641, 646), removing brake drums and dumping the dust on the ground, although he tried to dump it onto rags (Tr. 650). When he performed brake jobs, dust collected in the disc brake vent holes. (Tr. 1292). Juni also replaced gaskets (Tr. 1037, 1041), by removing parts of the engine, removing the gaskets using small Brillo pads (Tr. 1042, 1044), and clearing out the area with an air gun (*id.*). At the end of each workday, workers used compressed air to clean up the dust, and they swept up the dust with brooms. (Tr. 1586, 1587).

Juni was promoted to first-class mechanic in the late 1960s (Tr. 642). As a first-class mechanic, he serviced all kinds of vehicles manufactured by defendant. (Tr. 646, 991). Approximately 500 vehicles, mostly defendant's, were serviced at that garage during Juni's tenure. (Tr. 1052). Almost weekly, Juni performed clutch work on defendant's bucket trucks. (Tr. 657, 660, 661, 1507).

After Juni became a foreman in the 1970s, he assisted other workers with brake work on defendant's vehicles. (Tr. 991, 992). He performed manifold gasket work on defendant's bucket trucks, replacing the original gaskets (Tr. 994), and from 1970 to 1979, he assisted with clutch work once every three months. (Tr. 995). After 1979, the garage serviced a fleet of 16 to 18 of defendant's bucket trucks. Clutch jobs were performed once or twice a week. (Tr. 996, 999, 1000).

Juni personally replaced or assisted with replacing clutches and installing replacement gaskets on defendant's C-8000s (Tr. 1036-1038, 1042, 1512) and brakes on defendant's service vans, F-250s, and F-350s (Tr. 1299-1300), and performing intake manifold work on its C-800s and C-8000s (Tr. 1505-1507). He assisted when others installed gaskets. (Tr. 1595).

Juni also repaired his own and his family's vehicles, which included defendant's vehicles. He changed the engines and exhaust, and built a hitch on the back of one of defendant's 1965 F-100. (Tr. 1077, 1083, 1084). He twice changed the brakes. (Tr. 1086).

B. Expert evidence

1. Dr. Steven Markowitz

To establish general causation, plaintiffs called Steven Markowitz, MD, a board-certified physician specializing in internal and occupational medicine. As pertinent here, Markowitz

testified that asbestos fibers have the ability to bypass the lung's defense mechanisms, depending on the quantity and size of the fiber. (Tr. 289). He named chrysotile as the fiber most used in manufacturing brakes (Tr. 296), and opined that "no level [of exposure to asbestos] has been identified that separates out increased risk from no risk" (Tr. 308).

According to Markowitz, when a worker develops mesothelioma or lung cancer, all instances of exposure to asbestos are "viewed as a whole," cumulatively contributing to and causing the illness, and "every part of that exposure," he stated, acts as a contributing factor. While Markowitz contended that no exposure may be discounted, no matter how remote the occurrence, as "it's the cumulative exposure that matters" (Tr. 334-335), he also testified that exposure to one of defendant's brakes in a year and a half would not be a substantial contributing factor to the development of a worker's mesothelioma, that exposure to two of defendant's brakes during the same period would "probably not" be a substantial factor, and that there is "some point" where exposure does not constitute a substantial factor. (Tr. 435-436). Still, Markowitz stated that "there's no magic number above which there's a substantial factor and below which there's not. The science doesn't permit us to say that. The more the exposure, the more contribution there is," and the more the exposure, the greater the risk. (Tr. 443-444).

Markowitz also opined that when a worker manipulates or works with asbestos-containing material and creates visible dust, asbestos is released into the air (Tr. 337), and that if it becomes airborne and is inhaled, the chrysotile fibers contained within friction products, such as brakes, clutches, and gaskets, can cause mesothelioma.

Markowitz based his opinion on:

1) "general knowledge" that chrysotile asbestos causes malignant mesothelioma;

2) certain industrial hygiene studies of workers using friction products, some of which showed "elevated levels of asbestos in the air of garage mechanics who are working with friction products";

3) case series (individual and group reports) of malignant mesothelioma occurring among garage mechanics or those who work with friction products in the vehicle repair setting, which he believes "speaks to the evidence of a causal relationship in this instance";

4) evidence of those who work with friction products in vehicle repair who develop asbestos-related non-malignant diseases or some asbestos-related scarring due to asbestos as the result of their work repairing brakes, removing engine gaskets, working with clutches, and performing other similar functions;

(5) peer-reviewed literature in which the previously-mentioned studies are examined; and

(6) statements and findings made by agencies that have studied the issue, including the Environmental Protection Agency (EPA), Occupational Safety and Health Administration (OSHA), and the World Trade Organization.

(Tr. 315-318).

The studies and literature on which Markowitz relied were neither identified nor offered in evidence, and on cross-examination, he conceded that the subjects of the industrial hygiene studies were factory workers who mass-produced friction products from raw asbestos and not garage workers, and that exposure to asbestos in the factory setting differs significantly from a mechanic's exposure to asbestos in a vehicle repair garage. (Tr. 397-398). Markowitz also admitted that he was not aware of any epidemiological cohort studies supporting his opinion that there is an increased risk of contracting mesothelioma from exposure to auto brakes, clutches or gaskets. (Tr. 380). Rather, he acknowledged that 21of 22 studies "do not show much evidence in support of a relationship between mesothelioma and exposure to friction products," and reveal that for those who work with friction products, there is no increased risk of developing mesothelioma.

Markowitz also allowed that it has been found that when asbestos fibers are mixed with certain resins used in manufacturing brakes, the fibers "would not be respirable" (Tr. 426), and that in the "vast majority" of studies assessing the composition of debris formed from work performed on brakes, it was found that almost all of the asbestos in the brakes had been converted to a non-toxic substance, and that any resulting dust is composed of less than one percent asbestos. (Tr. 457-458).

Notwithstanding the above concessions, and having discredited the 21 studies, *inter alia*, as based on data culled from a small number of subjects, Markowitz hewed to his opinion that working with friction products generally causes mesothelioma,. (Tr. 320-321, 520-522).

2. Dr. Jacqueline Moline

Dr. Jacqueline Moline, an expert in internal medicine and occupational and environmental medicine, testified, that based on her review of Juni's medical records and deposition transcripts (Tr. 1345), Juni's cumulative exposures caused his mesothelioma, stating that it is not possible to separate out or exclude any particular exposure. (Tr. 1367). In her opinion, "all" of Juni's occupational exposures constitute substantial contributing factors in causing his disease, and his cumulative lifetime exposure was sufficient to cause it. (Tr. 1369-70).

On direct examination, Moline was asked to assume that: (1) from 1964 to 1988, Juni personally and regularly assisted in performing brake and clutch work including on defendant's brakes and clutches; (2) Juni assisted in removing defendant's original brakes and clutches and replacing them with defendant's new brakes and clutches; and (3) Juni's work created and exposed him to visible asbestos dust. Assuming the truth of these facts, Moline opined, within a

reasonable degree of medical certainty, that Juni's "cumulative exposure to asbestos dust from [defendant's] brakes and clutches associated with [defendant's] vehicles was a substantial contributing factor to causing his mesothelioma." (Tr. 1370-2).

Moline based her opinion on the following:

> (1) her clinical experience interviewing and evaluating people whose exposures to asbestos were similar to Juni's;
>
> (2) industrial hygiene studies in which elevated levels of dust were found to have emanated from the manipulation of brakes, and thereafter asbestosis was diagnosed in brake mechanics, which shows that there was exposure to asbestos from the manipulation;
>
> (3) animal studies showing an association between mesothelioma and the type of asbestos used in brakes;
>
> (4) human studies showing an association between asbestos and mesothelioma; and
>
> (5) national and international research organizations holding the same opinion.

(Tr. 1372-3).

Moline equated Juni's testimony that he saw dust with evidence that he was in fact exposed to asbestos at levels above the minimum at which asbestos can cause disease. In her view, "visible dust is an important surrogate to show that someone has had significant exposure." (Tr. 1374). Although Moline acknowledged that Juni "might have had other exposures," she testified that all of the exposures "contribute to his cumulative exposure, whether it's from Company A or Company B, those are all part of his cumulative exposures." (Tr. 1381). She did not differentiate among Juni's exposures to asbestos emanating from products of different companies (*id.*), and studies showing no increased risk of mesothelioma in mechanics or garage or brake workers did not alter her opinion given what she generally characterized as

"discrepancies" in them. (Tr. 1383-4).

Moline acknowledged that the amount, duration, and frequency of exposure are critical factors in assessing the sufficiency of an exposure in causing an increased risk of developing a disease. (Tr. 1430). Absent any data, however, Moline did not know if Juni had worked with friction brakes, clutches, or gaskets sold or distributed by defendant during the time he worked for Orange and Rockland or how often he had been exposed to such products, nor did she attempt any dose reconstructions or assessments to quantify his exposure. (Tr. 1430-6).

While Moline did not use the term "each and every exposure," she opined that the regular use of products containing asbestos that results in exposure to it constitutes a substantial contributing factor in causing an asbestos-related disease. (Tr. 1433-4). And although she conceded ignorance of whether the fibers to which Juni was exposed were biologically active and had the potential of causing mesothelioma (Tr. 1477-8), and while she agreed that visible dust must contain asbestos to be dangerous (Tr. 1481), she advanced her opinion that the amount of asbestos to which Juni was exposed from brake-wear debris was a contributing factor to the development of his mesothelioma (Tr. 1478-1479).

II. CONTENTIONS

A. Defendant

Defendant argues that it is entitled to judgment as matter of law or a new trial as the opinions of plaintiffs' experts on causation are inadmissible absent a sufficient foundation, and are otherwise based on invalid assumptions.

Defendant alleges that the scientific evidence presented at trial demonstrates that exposure to friction products does not cause mesothelioma, and relies on the agreement of

plaintiffs' experts "that the chrysotile asbestos used in friction products differs from other forms of the mineral and is less carcinogenic than other forms of asbestos." It observes that 21 of 22 epidemiological studies each yields the conclusion that there is no increased risk of asbestos exposure in vehicle mechanics. Thus, defendant maintains that plaintiffs failed to lay a reliable foundation for the expert opinion as to general causation, i.e., that exposure to chrysotile asbestos contained within friction products can cause mesothelioma. (NYSCEF 493).

Defendant also asserts that neither of the expert opinions is based on a scientific expression of Juni's exposure to dust from friction products, that pursuant to *Parker v Mobil Oil Corp.*, 7 NY3d 434 (2006), a scientific expression of exposure is a required predicate for the admission of evidence of causation in any toxic tort case, that plaintiffs offered no evidence of the dose, frequency, and/or intensity of Juni's alleged exposures, and that neither expert compared Juni's exposures with those described in the studies on which they relied. As plaintiffs never quantified Juni's exposure to dust emanating from brakes, clutches, or gaskets that defendant sold or distributed, defendant contends, neither could Markowitz or Moline, who instead opined that all of his exposures, cumulatively, constituted a substantial contributing factor, a theory rejected by numerous courts. (*Id.*). According to defendant, neither the description of Juni's exposure as cumulative nor the allegation that he was exposed to undifferentiated visible dust constitutes a basis for finding that the dust contained asbestos, and in any event, neither satisfies the requirements set forth in *Parker*. Even if the experts' opinions were admissible, defendant maintains, that there exists a general connection between asbestos exposure and the development of mesothelioma constitutes insufficient evidence absent a showing of a causal connection between the disease and exposure to asbestos from a particular

friction product. (*Id.*).

B. Plaintiffs

Plaintiffs argue that their experts' opinions were admissible as they were supported by a well-established scientific consensus that chrysotile asbestos causes mesothelioma, and that there is no safe level of exposure to asbestos. They also maintain that the controlling legal precedent for the opinion that the inhalation of visible asbestos dust can cause mesothelioma and that the presence of visible asbestos dust is sufficient evidence of a substantial factor in causing asbestos-related disease, is *Lustenring v AC&S, Inc.*, 13 AD3d 69 (2004), *lv denied* 4 NY3d 708 (2005), and that defendant's disagreement with the experts' causation opinions does not warrant holding a *Frye* hearing (*Frye v US*, 293 F. 1013 [DC Cir 1923]), or striking the experts' testimony. (NYSCEF 580).

According to plaintiffs, *Parker* does not require that a plaintiff in a toxic tort case present a quantified dose-response relationship between a defendant's product and a plaintiff's illness, observing that numerous other justices of this court have rejected defendant's "narrow" reading of *Parker*. They also deny that epidemiological studies and studies of specific trades are required bases for finding causation. (*Id.*).

Plaintiffs assert that their experts established both general and specific causation based on: (1) Juni's history of working on brakes, clutches, or gaskets sold or distributed by defendant and the resulting exposures to brake dust; (2) Markowitz's testimony that exposure to visible dust produced by the manipulation of chrysotile-containing products can cause mesothelioma; and (3) Moline's testimony that Juni's cumulative exposure to asbestos from the work he performed on defendant's vehicles and the brakes, clutches, or gaskets sold or distributed by it

was sufficient to cause his mesothelioma. Plaintiffs thus maintain that their experts' testimony constituted a scientific expression of Juni's exposure and was sufficient under both *Lustenring* and *Parker*. (*Id.*).

C. Defendant's reply

In reply, defendant disputes that *Lustenring* is the controlling legal precedent, and observes that it and the other cases cited by plaintiffs do not address the sufficiency of expert testimony on causation. It also contends that evidence that asbestos fibers may cause cancer does not establish general or specific causation. (NYSCEF 614).

III. Applicable law

Pursuant to 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 on the ground that the verdict was not supported by legally sufficient evidence. In order to find that a verdict should be set aside as a matter of law, the court must determine that 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, Inc.*, 45 NY2d 493 [1978]; *Sow v Arias*, 21 AD3d 317 [1st Dept 2005], *lv denied* 5 NY3d 716). Thus, "it must appear upon a fair interpretation of the evidence that no valid line of reasoning or set of permissible inferences exist that would permit the jurors to arrive at the verdict reached." (*Zalinka v Owens-Corning Fiberglass Corp.*, 221 AD2d 830 [3d Dept 1995], *citing Cohen*, 45 NY2d at 499).

Here, in order to establish that defendant's failure to warn Juni adequately of the dangers of exposure to asbestos was a substantial contributing factor in causing his mesothelioma,

plaintiffs were obliged to prove not only that Juni's mesothelioma was caused by his exposure to asbestos, but that he was exposed to sufficient levels of the toxin to cause his illness as a result of his work on brakes, clutches, or gaskets sold or distributed by defendant.

In *Parker v Mobil Oil Corp.*, 7 NY3d 434 (2006), the Court of Appeals addressed the sufficiency of evidence of causation in toxic tort cases. In *Cornell v 360 W. 51st St. Realty, LLC*, 22 NY3d 762 (2014), the Court clarified its holding in *Parker*. Absent any dispute that wrongful exposure to asbestos constitutes a "toxic tort," consideration of both decisions is appropriate.

A. *Parker*

The plaintiff in *Parker* had worked as a gas station attendant for 17 years and was exposed to benzene contained in gasoline when he inhaled gasoline fumes and touched gasoline. He claimed that the benzene in the gasoline to which he had been exposed caused him to develop acute myelogenous leukemia (AML).

1. In Supreme Court

Prior to trial, the defendants moved *in limine* for an order precluding the plaintiff's expert from testifying on the issue of medical causation, and upon preclusion, for dismissal of the plaintiff's claims. In support, the defendants relied on the opinions of two experts. One, an epidemiologist, opined based on two studies that there is an increased risk of AML for gas service station employees exposed to large amounts of benzene over an extended period of time, but that the low levels of benzene exposure resulting from service station work are below the dose threshold necessary to cause AML. (7 NY3d at 442-3).

The other expert, a toxicologist, opined that a dose-related relationship is a cornerstone of toxicology and pharmacology, that there is usually a threshold below which no effect can be

observed, that evidence of an association between chronic exposure to benzene becomes less reliable as the dosage decreases, and that there is virtually no reliable evidence of a causal relationship between a low dosage and development of AML. He also testified that in order to determine causation, it is necessary to know the amount of benzene sufficient to cause AML and the amount to which a plaintiff was exposed. Absent quantification of the plaintiff's exposure to benzene or any contradiction of the studies finding no increased risk of AML in service station or petroleum distribution workers, the defendants' toxicologist opined that causation cannot be established. (7 NY3d at 443-4).

In opposition, the plaintiff argued that whether benzene can cause AML does not constitute a matter of novel science that would warrant the holding of a *Frye* hearing, relying on two expert reports. In one, a specialist in occupational medicine and epidemiology discussed the plaintiff's exposure to benzene and cited studies linking benzene exposure to leukemia. In a study, the National Institute of Occupational Safety and Health (NIOSH) reported that there existed a relationship between increasing cumulative benzene exposure and leukemia mortality, and found "no evidence . . . for a threshold level below which no leukemia occurs." (7 NY3d at 444-5). The expert also cited several studies demonstrating an increased risk of leukemia in petroleum refinery workers, and observed that in recognition of the harmful effects of benzene, the Occupational Safety and Health Administration (OSHA) had lowered the permissible workplace standard. Thus, the expert concluded to a reasonable degree of medical certainty that the plaintiff contracted AML as a result of his exposure to benzene. (*Id.*).

The plaintiff's other expert, a toxicologist and epidemiologist, affirmed that the plaintiff was exposed to greater levels of benzene than the workers described in the refinery studies, and

that while authors of another study of refinery workers found no increased risk of leukemia, in a case-control study, more than a doubling of the risk was found. Neither of the plaintiff's experts quantified the plaintiff's exposure to benzene from gasoline. (*Id.*).

The trial court denied the defendants' motion to preclude, framing the issue as whether the plaintiff's experts used generally accepted principles and methodologies in arriving at their conclusions, and finding that they did so by demonstrating a link between benzene and leukemia and expressing a dose-response relationship through the experts' view that there is no safe threshold level of exposure. The court also held that given the plaintiff's testimony detailing his exposure, there was no need for the plaintiff's experts to cite any studies linking AML to exposure to benzene in gasoline or to quantify the plaintiff's exposure. (7 NY3d at 445-6).

2. At the Appellate Division

The Appellate Division, First Department, reversed the trial court and dismissed the complaint, finding that neither of the plaintiff's experts had quantified the plaintiff's exposure to benzene, and that even if they had established a threshold, they could not show that the plaintiff's exposure had exceeded it. Thus, the experts' opinions of the plaintiff's exposure and whether the exposure caused his AML were held to be speculative. (16 AD3d 648 [1st Dept 2005]).

3. At the Court of Appeals

In addressing the admissibility of the plaintiff's expert opinions, the Court observed that the pertinent inquiry is "whether there is a proper foundation - to determine whether the accepted methods were appropriately employed in a particular case." (7 NY3d at 447). The Court contrasted a *Frye* hearing, by which the trial court determines if the scientific procedure and results are generally accepted as reliable in the scientific community, and held that in the case

before it, the relevant inquiry was whether the methods employed by the plaintiff's experts led to a reliable result, "specifically, whether they provided a reliable causation opinion without using a dose-response relationship and without quantifying [the plaintiff's] exposure." (*Id.*).

Although the Court acknowledged that "[o]ne problem with establishing causation in toxic tort cases is that, often, a plaintiff's exposure to a toxin will be difficult or impossible to quantify by pinpointing an exact numerical value," it reiterated the well-established requirement that an expert opinion on causation 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). The Court also allowed that 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." (*Id.*). Those methods could include estimating the plaintiff's exposure through mathematical modeling based on a plaintiff's work history, or comparing the plaintiff's exposures with those reported in studies, provided that the expert specifically compares the plaintiff's exposure level with those of the other study's subjects. (*Id.* at 449).

In concluding that the plaintiff's experts had failed to demonstrate that the plaintiff's exposure to benzene contained in gasoline caused his AML, the *Parker* Court found that the general, subjective, and conclusory opinion that the plaintiff had "far more exposure" to benzene than did the refinery workers as reported in the studies was "plainly insufficient" and unsupported by epidemiological evidence to establish causation, given the absence of either a quantification of the other workers' exposure or evidence as to how the plaintiff's exposure

exceeded it. (7 NY3d at 449). It also held that the expert's opinion that the plaintiff was "frequently" exposed to "excessive" amounts of gasoline and had "extensive exposures" did not constitute a scientific expression of the plaintiff's exposure level, and that the demonstrated and undisputed connection between exposure to benzene and the risk of developing AML was insufficient, as the key issue was the relationship, if any, between exposure to gasoline containing benzene and AML. As neither expert was able to identify an epidemiological study finding an increased risk of AML as a result of exposure to gasoline, there was no evidence of a causal connection between gasoline containing benzine and AML, and standards promulgated by regulatory agencies as protective measures were deemed inadequate to establish legal causation. (7 NY3d at 449-450).

B. *Cornell*

The Court of Appeals summarized the factual and procedural background as follows, in pertinent part: The plaintiff in *Cornell* sued her landlord for exposing her to dampness and mold in her apartment, which she alleged caused various physical injuries. The landlord moved for an order summarily dismissing her claims, asserting that the plaintiff could prove neither that the mold at issue can cause the type of injuries alleged (general causation) nor that it caused the specific alleged injuries (specific causation). The landlord also sought to preclude the plaintiff's experts from testifying on causation. (22 NY3d 767, 768 [2014]).

The plaintiff cross-moved for an order granting her summary judgment, relying on the opinion of an expert in environmental and occupational medicine who specialized in mold-related illnesses. In an affidavit, the expert stated, in pertinent part, that exposure to damp buildings with excessive and atypical mold contamination is recognized as a cause of certain

respiratory health complaints and conditions in generally accepted and peer-reviewed literature.

In support, he cited a report that mold byproducts may all have effects adverse to humans, a report that the risk of certain respiratory conditions was higher in damp homes, a report finding that there exists sufficient evidence of an association between certain respiratory symptoms and building dampness and mold, and "suggestive" evidence of associations with other symptoms, a study finding that microbial agents in floor dust may be "a good surrogate measure" for dampness-related bioaerosol exposure, a study finding that epidemiological studies support the existence of a link between upper airway irritant symptoms and a damp indoor environment and mold growth, and a study finding that mold levels in dust are associated with asthma in a damp indoor environment and may increase the risk of building-related respiratory ailments. He also relied on government reports, guidelines, and public health initiatives that advise that mold exposure in indoor environments present a public health concern and recommend precautions, and several reports finding an association between building dampness and mold, that damp environments may be associated with work-related disease, that a cause-and-effect relationship between fungal exposure and respiratory disease is supported by epidemiological studies, and that indoor dampness alone seems to be associated with an increase in respiratory illness and symptoms. He performed a differential diagnosis on the plaintiff, using many diagnostic and laboratory tests, and concluded that the plaintiff suffered from particular respiratory ailments caused by her exposure to damp conditions in her apartment. (22 NY2d at 770-774).

The trial court dismissed the complaint, concluding that the plaintiff had failed to prove either general or specific causation. The Appellate Division, First Department, reversed, finding sufficient the plaintiff's expert's opinion relating her illnesses to the mold exposure based on the

opinion finding "some support in existing data, studies, and literature." The Court also suggested that "because '[i]t is undisputed that exposure to toxic molds is capable of causing the types of ailments from which [the plaintiff] suffers,' *Parker* teaches that threshold and actual exposure levels are not required to perform [a] differential diagnosis." (22 NY3d at 779).

The Court of Appeals reversed the Appellate Division, focusing on the data and evidence underlying the plaintiff's expert's opinion. After reiterating that standards promulgated by regulatory agencies are irrelevant since such standards are inadequate to demonstrate legal causation, the Court found that the expert's testimony did not establish general causation, as the reports and studies on which he relied were expressed in terms of "risk," "linkage," and "association," not causation, and that in equating association with causation, he had departed from the generally accepted methodology for evaluating epidemiological evidence when determining whether exposure to a toxin or agent causes a harmful effect or illness. The Court quoted from the federal courts' Reference Manual on Scientific Evidence, as pertinent here:

> [T]he first question an epidemiologist addresses is whether an association exists between exposure to the agent and disease . . . Although a causal relationship is one possible explanation for an observed association between an exposure and a disease, an association does not necessarily mean that there is a cause-effect relationship.

(22 NY3d at 783).

The Court concluded that because "studies showing an *association* between a damp and moldy indoor environment and the medical conditions [alleged by the plaintiff] do not establish that the relevant scientific community generally accepts that molds *cause* these adverse health effects," the Appellate Division was wrong in finding that the expert's opinion was sufficient to prove general causation based on "some support" in the record, and that the plaintiff had failed to

raise a triable issue as to general causation. (22 NY3d at 783 [emphasis in original]).

The Court also observed that while it had acknowledged in *Parker* that a precise quantification or dose-response relationship or an exact number is not required to show specific causation, "*Parker* by no means, though, dispensed with a plaintiff's burden to establish sufficient exposure to a substance to cause the claimed adverse health effect," and that it is "not enough for a plaintiff to show that a certain . . . agent sometimes causes the kind of harm that he or she is complaining of." (22 NY3d at 784). Rather, and "[a]t a minimum, . . . there must be evidence from which the factfinder can conclude that the plaintiff was exposed to levels of that agent that are known to cause the kind of harm that the plaintiff claims to have suffered." (22 NY3d at 784, *quoting Wright v Willamette Ind., Inc.*, 91 F3d 1105 [8th Cir 1996]).

The Court held that the plaintiff had failed to meet her burden as, among other issues, her expert made no effort to quantify her level of exposure and his differential diagnosis was inadequate. It thus found that the plaintiff had failed to raise a triable issue as to specific causation. (22 NY3d at 783-5).

The impact of the *Cornell* decision was recently addressed:

> While *Cornell* involved a claim that exposure to toxic mold caused a variety of personal injuries, its elucidation of the "general acceptance" standard as set forth in *Frye* [ ], and the foundation standard set forth in *Parker* [ ], is not limited to toxic tort cases. Rather, *Cornell* will have an impact in all tort cases where expert testimony is proffered to explain to the jury the mechanism of a plaintiff's injury.

(Michael J. Hutter, *Toxic Mold Case: Experts, Gatekeeping, Admissibility*, NYLJ, June 6, 2014 at 3, col 1; *see also Johnson v Guthrie Med. Group, P.C.*, 125 AD3d 1445 [4th Dept 2015] [*Parker* extended to medical malpractice cases]; *Muhammad v Fitzpatrick, M.D.*, 91 AD3d 1353 [4th Dept 2012] [same]; *Lugo v New York City Health and Hosp. Corp.*, 89 AD3d 42 [2d Dept

2011] [same]; *Kurz v St. Francis Hosp.*, 2014 WL 6992459 [Sup Ct, Nassau County 2014] [same]; *Nobre v Shanahan, M.D.*, 42 Misc 3d 909 [Sup Ct, Orange County 2013] [same]).

C. *Lustenring* and its progeny

Plaintiffs assert that *Lustenring v AC&S, Inc.*, 13 AD3d 69 (2004), *lv denied* 4 NY3d 708 (2005), is the controlling precedent for establishing the admissibility of expert evidence of causation in a personal injury action based on exposure to asbestos and asbestos-containing products. There, the Court upheld the trial court's decision entering judgment on a jury verdict in favor of two plaintiffs in an asbestos case. It found that "the evidence showed that both plaintiffs worked all day for long periods in clouds of dust raised specifically by the manipulation and crushing of defendant's packing and gaskets, which were made with asbestos," that "[v]alid expert testimony indicated that such dust, raised from asbestos products and not just from industrial air in general, necessarily contains enough asbestos to cause mesothelioma," and that the defendant's factual disagreement with the plaintiffs' causation theory did not require the holding of a *Frye* hearing. (*Id.* at 70).

The Appellate Division cited *Lustenring* in *Matter of New York City Asbestos Litig. (Marshall)*, also an asbestos case. There, the Court upheld a jury verdict against a defendant, finding that "the evidence demonstrated that both plaintiffs were regularly exposed to dust from working with defendant's gaskets and packing, which were made with asbestos." (28 AD3d 255, 256 [1st Dept 2006]). "The experts indicated that such dust from asbestos-containing products contained enough asbestos to cause mesothelioma" (*Id.*).

Similarly, in *Penn v Amchem Prods.*, the plaintiff testified that visible dust emanated from dental liners on which he worked as a dental student, and his expert testified that the dust

"must have contained enough asbestos to cause his mesothelioma." (85 AD3d 475, 476 [1st Dept 2011]). Finding this evidence sufficient to sustain the jury verdict, the Appellate Division reversed the trial court's contrary determination, relying on *Marshall*. (*Id.*). Although the underlying trial was held after the Court of Appeals issued its decision in *Parker*, neither the defendant-respondent (2010 WL 8758375) nor the Court cited it.

Lustenring, as well as *Marshall* and *Penn*, are based on discrete facts and evidence linking visible dust to the use of the particular defendant's product, and on expert testimony that the dust to which the plaintiff had been exposed contained or must have contained enough asbestos to cause the plaintiff's mesothelioma. In none of these opinions did the Appellate Division explain its findings or set a standard for the admissibility of expert evidence of causation, in an asbestos case or any other toxic tort case.

Courts ruling on the sufficiency of expert evidence in a variety of toxic tort cases have relied on *Parker.* (*See eg Sean R. v BMW of N. Am., LLC*, 115 AD3d 432 [1st Dept 2014] [medical and scientific literature offered by plaintiffs' experts did not support theory that exposure to gasoline fumes caused plaintiff's birth defects; thus plaintiff failed to show how exposure to constituent chemicals could have caused defects]; *Lindkvist v Travelers Ins.*, 111 AD3d 452 [1st Dept 2013] [expert testimony inadmissible as experts neither established that mold capable of causing injury nor quantified level of exposure necessary to cause plaintiff's illness]; *Rivera v Crotona Park E. Bristow Elsmere*, 107 AD3d 550 [1st Dept 2013] [plaintiff's case summarily dismissed as expert failed to provide scientific measurement or employ any accepted method of extrapolating measurement to establish plaintiff's exposure to specific toxic or allergen]; *Cleghorne v City of New York*, 99 AD3d 443 [1st Dept 2012] [complaint summarily

dismissed as expert adopted plaintiff's anecdotal allegations about exposure and failed to quantify level of exposure, and although he cited six studies, he neither compared plaintiff's exposure level with those described in studies, nor stated level of exposure necessary to cause injury]; *Nonnon v City of New York*, 88 AD3d 384 [1st Dept 2011] [summary judgment denied to defendants based on Court's finding, citing *Parker*, that triable issue raised as to causation; expert scientifically expressed exposure levels by conducting proximity analysis, a recognized substitute for dose-response analysis]; *Todman v Yoshida*, 63 AD3d 606 [1st Dept 2009] [expert's statement, based on manner in which plaintiff used the toxin-containing product, held insufficient as expert neither quantified exposure nor employed any methods for estimating it, such as mathematical modeling or comparing plaintiff's exposure level with those of study subjects]; *Jackson v Nutmeg Technologies, Inc.*, 43 AD3d 599 [3d Dept 2007] [*Parker* foundation requirement satisfied where expert coauthored report published in well-known, peer-reviewed medical journal detailing how causal connection between toxin and injuries found, compared exposures with those recorded in other studies, and cited other articles and publications in support of theory of causation]; *Zaslowsky v J.M. Dennis Constr. Co. Corp.*, 26 AD3d 372 [2d Dept 2006] [complaint summarily dismissed absent showing of causal connection between gas leak and injuries; experts neither quantified level of exposure to gas nor employed any other scientifically reliable method]).

And recently, another justice of this court, in deciding a post-trial motion to set aside a jury verdict in an asbestos case, addressed whether the plaintiffs had sufficiently established causation, citing *Parker*, not *Lustenring*, for the generally applicable legal standards, focusing on whether the plaintiffs' experts had established a "scientific expression" for the basis of their

opinions. (*Matter of New York City Asbestos Litig. [Dummitt]*, 36 Misc 3d 1234[A], * 8, 2012 NY Slip Op 51597[U] [Sup Ct, New York County 2012]). In finding that the experts had sufficiently demonstrated a scientific expression of the plaintiff's exposure levels, the court observed that one of the experts had measured the asbestos fibers released into the air from products identical to those produced by the defendant, and considered the plaintiff's experts' testimony in the context of evidence that the plaintiff's workplace contained hundreds of the defendant's products, thus finding that the evidence was sufficient to prove specific causation.

To the extent that plaintiffs rely on *Berger v Amchem Prods.*, 13 Misc 3d 335 (Sup Ct, New York County 2006), which was rendered before the Court of Appeals issued its decision in *Parker*, but after the Appellate Division issued its decision in *Parker*, for the proposition that *Lustenring*, and not *Parker*, controls, the *Berger* court neither cited *Lustenring*, nor discussed or distinguished the Appellate Division's decision in *Parker*, and it did not address whether the plaintiff's experts had established a sufficient foundation for their opinions or had established causation as a matter of law. *Berger* is thus inapposite. (*See Ratner v McNeil-PPC, Inc.*, 91 AD3d 63 [2d Dept 2011] [where experts offer no novel test or technique but intend to testify about theory of causation, and opinion is supported by generally accepted scientific methods, proper to proceed to foundation inquiry of admissibility]; *Matter of New York City Asbestos Litig. [Wiegman]*, 24 AD3d 375 [1st Dept 2005] ["(s)ince the parties argued over causation, no novel scientific technique or application of science was at issue, and a *Frye* hearing was not warranted"]).

D. *Parker* and *Cornell*, not *Lustenring*, govern

For all of these reasons, and given the role of the Court of Appeals in setting policy (*see*

Hynes v Tomei, 237 AD2d 52, 60 [2d Dept 1997], *revd on other grounds,* 92 NY2d 613 [1998], *citing People v Keta*, 165 AD2d 172, 178 [2d Dept 1991], *revd on other grounds sub nom, People v Scott*, 79 NY2d 474 [1992] [Court of Appeals is "the state's policy-making tribunal"]), and absent any reason articulated by plaintiffs for limiting *Parker* and *Cornell* to their facts, and deeming *Lustenring* controlling, *Parker* and *Cornell* are the controlling precedents in deciding whether the opinions of plaintiffs' experts are sufficient to prove causation as a matter of law in all toxic tort matters including asbestos cases. It is for the Court of Appeals alone to determine whether the link between mesothelioma and asbestos warrants relieving plaintiffs of the burden of establishing a foundation for the admission of an expert's opinion concerning general causation.

IV. ANALYSIS

> At issue in this case is the admissibility of Parker's experts' opinions. The parties dispute whether the opinions should be analyzed under *Frye* . . . Here, there is a question as to whether the methodologies employed by Parker's experts lead to a reliable result - specifically, whether they provided a reliable causation opinion without using a dose-response relationship and without quantifying Parker's exposure. There is no particular novel methodology at issue for which the Court needs to determine whether there is general acceptance. Thus, the inquiry here is more akin to whether there is an appropriate foundation for the experts' opinions, rather than whether the opinions are admissible under *Frye*.

(*Parker*, 7 NY3d at 446-447).

In arguing that mesothelioma, a signature disease, must have been caused by Juni's exposure to asbestos contained in brakes, clutches, or gaskets sold or distributed by defendant, plaintiffs suggest that they should be relieved of the burden of establishing some quantifiable level of exposure. In *Parker*, as here, and as in most asbestos cases, the plaintiff offered evidence that there is no safe level of exposure to the toxin; the plaintiff's exposure consisted

mainly of gasoline fumes and dermal contact.

Fumes are not visible, rendering the plaintiff's case in *Parker* difficult to prove. Nonetheless, and despite the close association between AML and benzene (*Parker*, 7 NY3d at 450; *U.S. v Apex Oil Co., Inc.*, 2008 WL 2945402 [SD Ill 2008] [exposure to benzene associated with development of cancer, especially AML]), the Court nonetheless required some quantification of the exposure to the benzene contained in gasoline, the particular product at issue. (*Parker*, 7 NY3d at 450-1). In other words, that mesothelioma is caused only by exposure to asbestos does not dispose of the issue of whether a defendant's product caused the mesothelioma, as it is not the association between mesothelioma and asbestos that is in issue when determining causation (*see Cornell v 360 W. 51st St. Realty, LLC*, 22 NY3d 762, 782-783 [2014]), but whether a defendant may be held liable for having caused a plaintiff's mesothelioma, which depends on the sufficiency of the exposure, if any, to asbestos in the defendant's product and whether that exposure is capable of causing mesothelioma. And, where an expert concedes that asbestos contained within friction products becomes degraded in the manufacturing process, and the plaintiff is alleged to have been exposed to numerous asbestos-containing products over many years, this issue may not be overlooked or ignored.

I thus analyze the evidence presented by plaintiffs using the standard set forth in *Parker* to determine whether Markowitz's and Moline's expert opinions on causation sufficiently established that Juni's exposure to asbestos contained within brakes, clutches, or gaskets sold or distributed by defendant was capable of causing his mesothelioma, and that Juni was thereby exposed to sufficient levels of asbestos to cause his mesothelioma.

A. General causation

Markowitz based his expert opinion that chrysotile asbestos contained within friction products can cause mesothelioma, *inter alia*, on the general knowledge that chrysotile asbestos causes mesothelioma, a proposition acknowledged by defendant. The issue, however, is 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 AML. (7 NY3d at 449-450; *see also Sean R. v BMW of N. Am., LLC*, 115 AD3d 432 [1st Dept 2014] [determining whether chemicals in gasoline can cause birth defects]). Thus, that chrysotile asbestos can cause mesothelioma, while probative of the issue here, does not dispose of it.

Although Markowitz cited industrial hygiene studies showing elevated levels of asbestos in the air breathed by garage mechanics working with friction products, the studies actually were of factory workers who produced friction products from raw asbestos. Markowitz then conceded that the factory exposure was significantly greater than that occurring in a vehicle repair garage. Thus, the studies he cited are not probative. In any event, the studies indicate only elevated levels of asbestos. However, proof of a risk, even an increased risk, does not constitute proof of causation. (*See Cornell*, 22 NY3d at 782-783 [reports and studies using terms like risk, link, or association do not establish general causation]).

On the other hand, in *Caruolo v John Crane, Inc.*, Markowitz testified that visible dust emanating from an asbestos-containing product contained enough asbestos dust to be hazardous. While Markowitz did not study the fiber release from the defendant's products, he testified that he was aware of two studies involving the products at issue, each of which measured the amount of asbestos fibers released by the products and showed that the amount was hazardous. The

Court thus found that Markowitz's testimony about the asbestos content in visible dust and about the two studies furnished an evidentiary basis for finding that the dust that allegedly came from the defendant's products contained hazardous levels of asbestos. (226 F3d 46 [2d Cir 2000]).

No such 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. And again, association is not causation. (*Cornell*, 22 NY3d at 782-783 [studies showing association do not establish that relevant scientific community generally accepts that particular toxic agent causes certain health effects]; *Ratner v McNeil-PPC, Inc.*, 91 AD3d 63 [2d Dept 2011] [case studies did not show that toxin caused injuries, only hypothesized that injuries were related to toxin]). Moreover, case reports or case studies are not generally accepted methods of establishing causation. (*Pullman v Silverman*, 125 AD3d 562 [1st Dept 2015], citing *Heckstall v Pincus*, 19 AD3d 203, 205 [1st Dept 2005] [case reports not generally accepted in scientific community on questions of causation]).

Peer-reviewed literature that only summarizes the aforementioned studies is also insufficient. (*See Castrichini v Rivera*, 175 Misc 2d 530 [Sup Ct, Monroe County 1997] [peer review of expert's articles relevant to reliability of opinion but does not indicate general acceptance in scientific community]). And the reports and findings of governmental agencies are irrelevant as they constitute insufficient proof of causation. (*Cornell*, 22 NY3d at 782 [standards promulgated by regulatory agencies as protective measures inadequate to establish legal causation]; *Parker*, 7 NY3d at 450 [same]; *see also Hamilton v Miller*, 23 NY3d 592 [2014] [in action alleging personal injuries from lead poisoning, court properly declined to take judicial notice of statutory provision wherein Congress justified legislation aimed at reducing lead by

noting that lead poisoning in children causes certain injuries; plaintiff could not avoid burden of proving general causation by relying on Congress's opinion on dangers of lead]).

For all of these reasons, Markowitz's opinions, either individually or collectively, do not establish that asbestos contained in friction products can cause mesothelioma, and as he conceded, he could identify no study to support his proposition that there is an increased risk of contracting mesothelioma from exposure to auto brakes, clutches, or gaskets or that there is an increased risk of mesothelioma from the use of friction products or work on friction materials in the automobile industry.

Plaintiffs rightly rely, however, on *Berger v Amchen*, 13 Misc 3d 335 (Sup Ct, New York County 2006), for the propositions that epidemiological studies specific to a profession, or even epidemiological studies in general, are not necessary to prove causation, and that an expert need not submit or cite to epidemiological studies related to the specific profession at issue, such as brake work, in order to prove causation. However, that epidemiological studies are not required does not mean that they are not probative, and here, Markowitz acknowledged that 21 of 22 epidemiological studies conducted of those who work with friction products yielded no evidence of an increased risk of developing an asbestos-related disease. (*See eg Ratner v McNeil-PPC, Inc.*, 91 AD3d 63 [2d Dept 2011] [where plaintiff's experts cited only case studies in support of theory of causation, analytical gap between plaintiff's scientific data and theory of causation widened by scientific articles submitted by defendant which contradicted theory of causation]; *see also Rowe v Fisher*, 82 AD3d 490 [1st Dept 2011] [court properly precluded plaintiff's expert testimony as to causation theory as expert's theories were contrary to medical literature on subject and therefore unreliable]; *Fraser v 301-52 Townhouse Corp.*, 57 AD3d 416 [1st Dept

2008] [plaintiff's expert evidence precluded as medical literature in record did not support expert's theory]).

And, while the absence of an epidemiological study is not fatal to proving causation (*see eg, Ratner*, 91 AD3d at 75 [absence of textual authority directly on point to support experts' opinion relevant to weight, not admissibility, of testimony]; *but see Parker*, 7 NY3d at 449-450 [key issue was relationship, if any, between exposure to gasoline containing benzene and AML, and plaintiff's experts failed to establish connection as they were unable to identify single epidemiological study finding increased risk of AML as result of exposure to gasoline]), here, the failure to offer in evidence any study to support Markowitz's opinion must be considered with the 21 studies which, Markowitz acknowledged, do not show "much evidence in support of a relationship between mesothelioma and exposure to friction products," and "showed that there was no increased risk of developing mesothelioma from occupational exposure to the products."

Thus, Markowitz not only cited no study to support his opinion, but he also conceded that numerous studies contradict it. Given his failure to otherwise establish a causal connection between exposure to friction products and mesothelioma, defendant has established that plaintiffs offered insufficient evidence of general causation here.

B. Specific causation

Moline's opinion that Juni's exposure to asbestos contained in brakes, clutches, or gaskets sold or distributed by defendant was a substantial contributing factor in causing his mesothelioma was mostly based on hypothetical facts she was instructed to assume. Defendant contends that the hypothetical was not based on facts adduced at trial. I do not reach that issue. Rather, for purposes of determining specific causation only, I assume that the facts posed in the

hypothetical are based on the trial evidence.

Moline conceded that she could provide no scientific expression of Juni's exposure absent data, which was not provided and does not exist in the record. Moline also conceded that she did not know whether the dust to which Juni was exposed contained any asbestos, much less enough to cause mesothelioma, and she had no personal knowledge of the dust's composition, had done no tests or analyses, and was unfamiliar with what happens to chrysotile asbestos fibers during the brake manufacturing process, or whether the fibers to which Juni was allegedly exposed were biologically active and had the potential of causing mesothelioma. Absent knowledge of the amount, duration, or frequency of Juni's exposures to asbestos-containing dust from brakes, clutches, or gaskets sold or distributed by defendant, Moline could not and did not establish a dose-response relationship or even minimally quantify Juni's exposures. Moline also failed to use any other method identified by the Court in *Parker* and *Cornell* to express Juni's exposure scientifically, such as by estimating his exposure through mathematical modeling by taking into account his work history, and to the extent she mentioned or relied on studies, she did not and could not compare the exposures reported in the studies with Juni's exposures.

Moline thus failed to provide a scientific expression of Juni's exposure to asbestos from brakes, clutches, or gaskets sold or distributed by defendant, and therefore, plaintiffs failed to prove specific causation. (*See Cleghorne v City of New York*, 99 AD3d 443 [1st Dept 2012] [plaintiff failed to establish specific causation; only method expert used to quantify exposure was to accept plaintiff's allegations as to her exposure; expert provided no measurement of exposure and plaintiff offered no other evidence thereof; while expert cited six studies, he neither compared plaintiff's exposure to those of study subjects, nor stated level of exposure necessary to

cause injury]; *Todman v Yoshida*, 63 AD3d 606 [1st Dept 2009] [citing *Parker*, expert offered no scientific expression of plaintiff's exposure to toxic chemicals as he neither provided measurement of exposure nor employed any methods for reasonably estimating it, such as by mathematical modeling or comparing plaintiff's exposure level to those of study subjects]).

Evidence of Juni's "regular" exposure to brakes, clutches, or gaskets sold or distributed by defendant during his work life, absent any quantification of the exposure, is insufficient to constitute a scientific expression of his exposure, as were descriptions of the plaintiff's exposures in *Parker* as "frequent," "excessive," and "extensive" (7 NY3d at 449), and in *Cleghorne* as "high-level" (99 AD3d at 447).

C. Visible asbestos dust

To the extent that plaintiffs argue, based on *Lustenring v AC&S, Inc.*, 13 AD3d 69 (2004), *lv denied* 4 NY3d 708 (2005), that proof of visible asbestos dust constitutes or is a proxy for the scientific expression of causation, the evidence they offered is insufficient to prove that the dust to which Juni was exposed contained any asbestos or enough to cause his mesothelioma. As conceded by Markowitz, during the brake manufacturing process, when asbestos fibers are mixed with certain resins, they become nonrespirable, and the "vast majority" of studies assessing the composition of debris formed from work on brakes reflects that 99 percent of the asbestos is converted to a non-toxic substance during the process. Thus, when a worker claims exposure to a cloud of dust formed while working on a brake, that dust is composed of one percent asbestos.

Similarly, Moline agreed that for visible dust to be dangerous enough to cause an asbestos-related disease, it must contain asbestos. However, she did not know whether the

brakes, clutches, or gaskets sold or distributed by defendant, and to which Juni was exposed, produced asbestos-containing dust during a repair procedure.

And, even if *Lustenring* controls, the evidence there showed that "both plaintiffs worked all day for long periods in clouds of dust." (13 AD3d at 69). Here, by contrast, Juni never testified that he saw clouds of dust, or that he worked all day for long periods in clouds of dust. Thus, *Lustenring* is not factually analogous. The expert testimony in *Lustenring* also established that the dust necessarily contained enough asbestos to cause mesothelioma. Here again, neither Moline nor Markowitz knew whether the dust at issue contained enough asbestos to cause mesothelioma.

Therefore, to the extent that *Lustenring* established a standard of admissibility for expert evidence of causation based solely on proof of visible asbestos dust (*but see supra*, III.C., at 22-26), the proof introduced at this trial falls short of that standard as well.

D. Cumulative exposure

Alternatively, plaintiffs argue that evidence that Juni was regularly exposed over many years to asbestos contained in brakes, clutches, or gaskets sold or distributed by defendant renders unnecessary a quantification of his individual exposures to prove that his mesothelioma was caused by defendant. In testifying about their opinions on cumulative exposures, neither Markowitz nor Moline stated the basis for their opinion that no single exposure to asbestos can be discounted in evaluating whether an exposure contributed to causing an asbestos-related disease; they cited no study or authority to support their opinions. Defense expert Finley's tentative agreement on cross-examination that cumulative exposure to asbestos increases one's risk for developing an asbestos-related disease does not fill the evidentiary gap. (Tr. 2582-3).

The opinion that every single exposure constitutes a significant contributing factor because the exposures cumulatively cause the disease is irreconcilable with the well-recognized scientific requirement, acknowledged by Moline, that the amount, duration, and frequency of exposure be considered in assessing the sufficiency of an exposure in increasing the risk of developing a disease. In other words, the risk of developing a disease increases or decreases depending on the nature of the exposure, which depends on the amount, duration, and frequency of the exposure.

In asserting that the cumulative exposure controls, plaintiffs avoid the requirement of showing even an approximate quantification, not only as a matter of law, but as a matter of science, and fail to offer sufficient evidence that any specific exposure increases the risk of a disease and is thus a significant contributing factor to causing the disease. In this products liability case, the issue is whether products sold or distributed by defendant caused Juni's mesothelioma. It is not the association between mesothelioma and asbestos that is in issue when determining legal causation, but rather whether a particular defendant may be held liable for having caused a person's mesothelioma, which depends on the person's exposure to the defendant's product. (*See eg Burst v Shell Oil Co.*, 2014 WL 3893304 [ED LA 2014] [general causation question is whether exposure to gasoline containing benzene causes leukemia, not simply exposure to benzene; burden of proving general causation is to show that substance at issue is capable of causing harm; substance to which plaintiff was exposed was gasoline, not pure benzene]; *Henricksen v ConocoPhillips Co.*, 605 F Supp 2d 1142 [ED Wash 2009] [plaintiff argued that general causation not in issue as no dispute that benzene causes AML; in products liability action, product is at issue, and plaintiff was exposed to gasoline, not benzene alone, and

toxic effects of benzene differed among sources]). Thus, in *Parker*, the issue was the sufficiency of evidence of the plaintiff's exposure to benzene contained in the gasoline to which he was exposed. And here, the issue is whether Juni's exposure to brakes, clutches, or gaskets sold or distributed by defendant caused his mesothelioma.

Accepting the experts' theory that a cumulative and unquantified exposure proves causation means that if Juni was exposed to asbestos dust when working on one product at one time in his decades-long career, that exposure would be considered just as likely to cause mesothelioma as his greater and more frequent exposures to asbestos dust from other products. Again, such a notion is contrary to accepted science that it is the nature and degree of the exposure that affects the risk of developing a disease.

The Court of Appeals's direction in *Parker* and *Cornell* regarding the proof necessary to establish causation as a matter of law in a toxic tort case conforms with the case law in other jurisdictions addressing the issue of the sufficiency of evidence of cumulative exposure in asbestos cases. Many of those courts require specific proof of exposure and have rejected the so-called cumulative exposure theory and its variant, the "each and every" exposure theory.[1] An

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[1] In 2015, the Eastern District of Louisiana, in *Comardelle v Penn. Genl. Ins. Co.*, precluded an expert from testifying that all exposures to asbestos contribute to causing an individual's mesothelioma, finding that:

> Although there may be no known safe level of asbestos exposure, this does not support [the expert's] leap to the conclusion that therefore every exposure [the plaintiff] had to asbestos must have been a substantial contributing cause of his mesothelioma . . . This kind of blanket specific causation opinion is not based on or tied to the specific facts and circumstances of any of [the plaintiff's] exposures to asbestos and it elides any differences or nuances of duration, concentration, exposure, and the properties of the fibers to which he may have been exposed.

(__ F Supp 3d __, 2015 WL 64279 [ED La 2015]).

The highest state court of Texas held that proof of "some" or "any" exposure to asbestos is insufficient to establish causation for contracting mesothelioma, finding that:

> If any exposure at all were sufficient to cause mesothelioma, everyone would suffer from it or at least be at risk of contracting the disease . . . Acceptance of an any exposure theory would . . . ignore the importance of dose in determining a causative link, and impose liability even where, for all the jury can tell, the plaintiff might have become ill from his exposure to background levels of asbestos or for some other reason . . .
>
> More fundamentally, if we were to . . . accept that any exposure to asbestos is sufficient to establish liability, the result essentially would be not just strict liability but absolute liability against any company whose asbestos-containing product crossed paths with the plaintiff throughout his entire lifetime . . . And we have never embraced the concept of industry-wide liability on grounds that proof of causation might be difficult . . .
>
> The any exposure theory effectively accepts that a failure of science to determine the maximum safe dose of a toxin necessarily means that every exposure, regardless of amount, is a substantial factor in causing the plaintiff's illness. This approach negates the plaintiff's burden to prove causation by a preponderance of the evidence. . .

(*Bostic v Georgia-Pacific Corp.*, 439 SW3d 332 [Tex 2014]).

The highest state court of Virginia addressed the standard for proving causation pursuant to Virginia law in a mesothelioma case involving exposure to multiple sources of asbestos, and determined, in granting a new trial, that the plaintiffs' experts at the new trial "must opine as to what level of exposure is sufficient to cause mesothelioma, and whether the levels of exposure at issue in this case were sufficient." (*Ford Motor Co. v Boomer*, 736 SE2d 724 [Va 2013]).

The Supreme Court of Pennsylvania, its highest state court, in *Howard v A.W. Chesterton Co.*, reaffirmed several principles for establishing causation in asbestos cases:

(1) the theory that each and every exposure, no matter how small, is substantially causative of disease may not be relied upon to establish substantial-factor causation for diseases that are dose-responsive;
(2) in cases involving dose-responsive diseases, experts may not ignore or refuse to consider dose as a factor in their opinions;
(3) proof of *de minimis* exposure to a defendant's product is insufficient to establish causation; and
(4) when an expert witness addresses causation in a dose-responsive disease case, the witness must make a "reasoned, individualized assessment of a plaintiff's or decedent's exposure history . . . "

(79 A3d 605 [Pa 2013]; *Betz v Pneumo Abex LLC*, 44 A3d 27 [Pa 2012] ["any exposure" theory rejected as "one cannot simultaneously maintain that a single fiber among millions is substantially causative, while also conceding that a disease is dose responsive."]; *Gregg v V-J Auto Parts, Co.*, 943 A2d 216 [Pa 2007] ["generalized [expert] opinions [that any exposure to asbestos, no matter how minimal, is a substantial contributing factor] do not suffice to create a jury question in a case where exposure to the defendant's product is *de minimis*, particularly in the absence of evidence excluding other possible sources of exposure (or in the face of evidence of substantial exposure from other sources)."]).

While acknowledging the difficulties that toxic tort plaintiffs face in proving causation, the *Gregg* Court stated that:

> we do not believe that this is a viable solution to indulge in a fiction that each and every exposure to asbestos, no matter how minimal in relation to other exposures, implicates a fact issue concerning substantial-factor causation in every "direct-evidence" case. The result, in our view, is to subject defendants to full joint-and-several liability for injuries and fatalities in the absence of any reasonably developed scientific reasoning that would support the conclusion that the product sold by the defendant was a substantial factor in causing the harm.

(943 A2d 216).

Nevada's highest state court also held that evidence of any and all or cumulative exposure is insufficient absent evidence of the frequency, proximity, and regularity of exposure to a defendant's product. (*Holcomb v Georgia Pacific, LLC*, 289 P3d 188 [Nev 2012]).

An intermediate appellate court in Georgia found that an expert's opinion on specific causation was insufficient and inadmissible absent an opinion that any specific defendant's products caused the plaintiff's disease. Instead, the expert had testified that the plaintiff's cumulative exposure to all products contributed to causing his disease. (*Butler v Union Carbide Corp.*, 712 SE2d 537 [Ga App 2011]).

The Sixth Circuit Court of Appeals reversed a jury verdict against the defendant, as the plaintiff's expert testified that all of the plaintiff's exposures contributed to causing his mesothelioma, which the Court found did not establish that the plaintiff's exposure to the defendant's products in and of itself was a substantial factor in causing the mesothelioma. (*Moeller v Garlock Sealing Technologies*, 660 F3d 950 [6th Cir 2011]). The Court also observed that the plaintiff did not quantify his exposure to the defendant's products, and that there was evidence of his exposure to other asbestos-containing products, thus concluding that while exposure to the defendant's products may have contributed to his mesothelioma, the plaintiff did not prove that it was a substantial cause. (*Id.*; *see also Lindstrom v A-C Product Liability Trust*, 424 F3d 488 [6th Cir 2005] [expert opinion that every exposure to asbestos is substantial factor in

overview and analysis of such cases is set forth in several law review articles, including: Joseph Sanders, *The "Every Exposure" Cases and the Beginning of the Asbestos Endgame*, 88 Tul L Rev 1153 (2014); Megan A. Cedar, *A Dose of Reality: The Struggle with Causation in Toxic Tort Litigation*, 51 Hous L Rev 1147 (2014); William L. Anderson, *The "Any Exposure" Theory Round II: Court Review of Minimal Exposure Expert Testimony in Asbestos and Toxic Tort Litigation Since 2008*, 22 Kan J L & Publ Pol'y 1 (2012); David E. Bernstein, *Getting to Causation in Toxic Tort Cases,* 74 Brook L Rev 51 (2008); and Mark A. Behrens and William L. Anderson, *The "Any Exposure" Theory: An Unsound Basis for Asbestos Causation and Expert Testimony*, 37 Sw U L Rev 479 (2008).

Although there may be cases where it will be difficult or impossible to quantify a plaintiff's exposure to a toxin, the *Parker* Court held that some quantification is nonetheless necessary for a plaintiff to prove causation. (7 NY3d 434). Therefore, that the plaintiff's burden of establishing that a particular exposure to asbestos was the cause of his mesothelioma is satisfied by an expert's opinion that a cumulative exposure to asbestos, no matter how small and without any quantification, was a substantial contributing factor to the development of a plaintiff's mesothelioma, is contrary to New York law as set forth in *Parker* and *Cornell.*

V. CONCLUSION

Absent a sufficient foundation for the admission of the expert evidence, plaintiffs' evidence was legally insufficient to establish, *prima facie*, that Juni's exposure to asbestos from

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causing disease held insufficient absent proof related to each defendant at issue; "A holding to the contrary would permit imposition of liability on the manufacturer of any product with which a worker had the briefest of encounters on a single occasion."]).

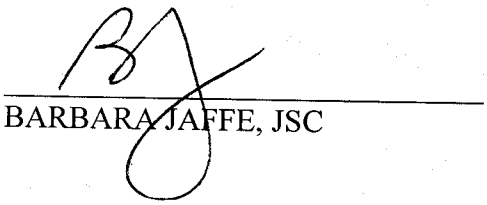
brakes, clutches, or gaskets sold or distributed by defendant constituted a significant contributing factor in causing Juni's mesothelioma. There is thus no valid line of reasoning or permissible inference which could have led the jury to reach its result.

Given this result, defendant's remaining arguments are not addressed.

Accordingly, it is hereby

ORDERED, that defendant's motion to set aside the verdict is granted, and the verdict is set aside in its entirety. Judgment is therefore rendered in favor of defendant, and the clerk is directed to enter judgment accordingly.

ENTER:



BARBARA JAFFE, JSC

Dated: April 13, 2015
New York, New York