7 N.Y.3d 434 (2006)
857 N.E.2d 1114
824 N.Y.S.2d 584

ERIC PARKER, Appellant,

v.

MOBIL OIL CORPORATION et al., Respondents. (And Third-Party Actions.)

Court of Appeals of New York.

Argued September 5, 2006.
Decided October 17, 2006.

435 *435 *Kreindler & Kreindler LLP,* New York City (*Marc S. Moller* and *Blanca I. Rodriguez* of counsel), and *Baggett, McCall, Burgess, Watson & Gaughan,* Lake Charles, Louisiana (*William B. Baggett, Sr., Wells T. Watson* and *Jeffrey T. Gaughan* of counsel), for appellant.

436 *436 *Wilson, Elser, Moskowitz, Edelman & Dicker LLP,* Newark, New Jersey, and New York City (*Robert J. Kelly, Richard E. Lerner, Robert P. Scott* and *Suna Lee* of counsel), for **Mobil Oil** Corporation and another, respondents.

437 *Smith Mazure Director Wilkins Young & Yagerman, P.C.,* New *437 York City (*Joel Simon* of counsel), for Island Transportation Corporation, respondent.

Rivkin Radler LLP, Uniondale (*James Quinn, Jay D. Kenigsberg* and *Harris J. Zakarin* of counsel), for Getty Petroleum Marketing, Inc., respondent.

Locks Law Firm, PLLC, New York City (*Seth R. Lesser* of counsel), and *Val Washington* for American Trial Lawyers Association and another, amici curiae.

438 *438 *Metzer Law Group, APLC,* Long Beach, California (*Raphael Metzger* of counsel), for Council for Education and Research on Toxics and others, amici curiae.

Mayer, Brown, Rowe & Maw, Washington, D.C. (*Andrew J. Pincus, Charles A. Rothfeld* and *Rajesh De* of counsel), and *National Chamber Litigation Center, Inc.* (*Robin S. Conrad* and *Amar D. Sarwal* of counsel), for Chamber of Commerce of the United States of America, amicus curiae.

439 *439 *Malaby, Carlisle & Bradley, LLC,* New York City (*Robert C. Malaby* and *David P. Schaffer* of counsel), *Crowell & Moring LLP,* Washington, D.C. (*William L. Anderson* and *Jennifer G. Knight* of counsel), and *Shook, Hardy & Bacon, LLP* (*Victor E. Schwartz* and *Mark A. Behrens* of counsel), for Coalition for Litigation Justice, Inc., amicus curiae.

Michael A. Cardozo, Corporation Counsel, New York City (*Leonard Koerner, Fay Leoussis, Christopher G. King, Amy London* and *Elizabeth S. Natrella* of counsel), for City of New York and another, amici curiae.

440 *440 *Debevoise & Plimpton LLP,* New York City (*Anne E. Cohen, Robert D. Goodman* and *Genevieve A. Pope* of counsel), and *Hugh F. Young, Jr.,* Reston, Virginia, for Product Liability Advisory Council, Inc., amicus curiae.

Jordan and Moses, Saint Simons Island, Georgia (*Randall A. Jordan* and *Mary Helen Moses* of counsel), *Louis P. Warchot,* Washington, D.C., and *Daniel Saphire* for Association of American Railroads, amicus curiae.

441 *441 *National Legal Scholars Law Firm, P.C.,* Lyme, New Hampshire (*Anthony Z. Roisman* of counsel), for Margaret A. Berger and others, amici curiae.

Chief Judge KAYE and Judges ROSENBLATT, GRAFFEO, READ and R.S. SMITH concur; Judge PIGOTT taking no part.

442 *442 # OPINION OF THE COURT

CIPARICK, J.

Plaintiff Eric **Parker** commenced this action in 1999 against **Mobil Oil** Corporation, Island Transportation Corporation and Getty Petroleum Marketing, Inc., alleging that exposure to benzene in gasoline caused him to develop acute myelogenous leukemia (AML). **Parker** had worked as a gas station attendant for 17 years and had been exposed to benzene through inhalation of gasoline fumes and through dermal contact with gasoline. There is no dispute that benzene is a known carcinogen.

Parker worked at several full-service stations between March 1981 and August 1998. As part of his duties, he pumped gasoline for customers, exposing him to gasoline vapors; the pumps were not fitted with vapor recovery systems to reduce exposure to fumes until the early 1990s. He was also exposed to fumes upon receipt of deliveries of gasoline and upon daily gauging of gasoline levels in the tanks and he was responsible for cleaning up gasoline spills, occasioning it to remain on his hands and clothing throughout the day. Defendants did not warn him of the dangers of benzene exposure or provide him with safety or protective gear. It should be noted that **Parker** was also exposed to therapeutic radiation.

Prior to the completion of discovery, and before the exchange of expert reports, defendant **Mobil Oil** and several third-party defendants moved to preclude **Parker's** expert testimony on the issue of medical causation. Defendants argued that the expert testimony was scientifically unreliable and should be excluded under *Frye v United States* (293 F 1013 [DC Cir 1923]). Further, defendants moved for summary judgment dismissing all claims, arguing that they lacked the necessary support in the absence of appropriate causation evidence.

In support of the motion, defendants introduced the opinions of two experts prepared for other litigations. The first, Dr. Gerhard K. Raabe—an epidemiologist and Director of Medical Information Health Risk Assessment for **Mobil**—
443 acknowledged *443 that there is an increased risk of AML for service station employees exposed to large amounts of benzene ("typically over 100 PPM TWA"[1]) over an extended period of time, but concluded that the low levels of benzene exposure resulting from gasoline service station work are "below the practical threshold for the dose necessary to initiate the leukemia process." Raabe cited to a National Institute for Occupational Safety and Health (NIOSH) study of benzene exposure for service station employees (the maximum concentration of benzene in gasoline was 2% with the greatest level of exposure 0.19 ppm TWA, which is less than the 1 ppm occupational standard set by the Occupational Safety and Health Administration [OSHA]); to a study of petroleum workers exposed to gasoline with a concentration of 2% to 3% benzene that did not show any additional risk of AML from exposure to gasoline; and to a European study of service station workers exposed to gasoline that was 3% to 5% benzene that did not find an elevated risk of AML. Defendants also provided a letter from Raabe responding to an expert opinion in another litigation citing a study he coauthored, which found an increased risk of AML for those exposed to "increasing cumulative doses of benzene above 200 ppm-years ... [and] no excess risk for AML for doses below" that level.

Defendants also offered the affidavit of Richard D. Irons, Ph.D., a toxicologist—likewise prepared for other litigation. Irons explained that the dose-related relationship was a unifying concept in the medical sciences and a cornerstone of pharmacology and toxicology; that there is usually a threshold below which no effect can be observed; and that the evidence of an association between chronic exposure to benzene and AML became less reliable as the dosage decreased; and that there was "virtually no reliable evidence to indicate that a causal relationship exists between chronic exposure to benzene at 10 ppm or lower and the development of AML." In order to determine causation, according to Irons, it is necessary to know the amount of benzene sufficient to cause AML and the amount of benzene to which the particular plaintiff was exposed. He noted that the plaintiff's expert in that case did not quantify the benzene exposure and
444 did not address studies finding no increased risk *444 of AML in service station or petroleum distribution workers. Irons also pointed out that AML has been known to develop in those who have been exposed to the drugs and chemicals used in chemotherapy.

In opposition to defendants' motion, **Parker** argued that whether benzene can cause AML is not novel scientific evidence subject to *Frye* review, and that there is a difference of opinion in the scientific community as to what level of benzene

exposure causes leukemia. To support his arguments, he produced reports from two experts. Philip J. Landrigan, M.D., a board-certified physician in occupational medicine and fellow of the American College of Epidemiology, detailed **Parker's** medical history as well as his exposure to benzene as a component of gasoline. Landrigan noted that **Parker** had received radiation treatment for a prior illness. The doctor also observed that, during his service station employment, **Parker** frequently had cuts or abrasions on his hands that would have increased the absorption of benzene directly into his bloodstream. Further, there was at least one instance where **Parker** was doused with gasoline but continued to work in his gasoline-saturated clothing for the remainder of the day.

Landrigan cited several studies that linked benzene exposure to leukemia. He noted that a NIOSH study of rubber plant workers in Ohio found a relationship between increasing cumulative benzene exposure and leukemia mortality. He concluded that the study showed a risk of mortality from leukemia of about "150 times above background" over a 40-year working lifetime from exposure to benzene at 10 ppm. At 5 ppm, the risk was 12 times over background and at 1 ppm (or 40 ppm-years) the risk was doubled. The expert went on to explain that "[e]xtensive mathematical modeling was conducted to determine the shape of this positive dose-response relationship. These analyses found that a linear model best explained the association. No evidence was found for a threshold level below which no leukemia occurs."

Landrigan further noted several studies that found an increased risk of leukemia in petroleum refinery workers and pointed out that the studies that did not find an increased risk of leukemia considered all refinery workers rather than specifically addressing only those exposed to benzene. He also stated that, in recognition of the carcinogenic nature of benzene, OSHA lowered the previous workplace standard from 10 ppm to 1 ppm. Landrigan found it unlikely that **Parker** would have
445 *445 contracted AML without his specific occupational exposure to benzene and therefore concluded "to a reasonable degree of medical certainty that Mr. **Parker** contracted his [AML] as a result of his personal occupational exposure to benzene."

Parker also submitted a two-page report from Bernard D. Goldstein, M.D., an expert in toxicology and epidemiology. Dr. Goldstein stated that **Parker** had greater levels of exposure to benzene than the workers in the refinery studies, as modern refineries function within the 1 ppm workplace standard and "[g]asoline has been approximately 2% benzene (i.e., 20,000 ppm)." He also noted that although a study of British refinery workers found no increased risk of leukemia, a "nested case-control study . . . [found] more than a doubling in the likelihood that those who did die of leukemia had been exposed to higher levels of benzene than appropriate controls." Finally, he observed that there was evidence that **Parker's** medical history—having received radiation treatment—made him more susceptible to leukemia from exposure to benzene. While Goldstein did give a number in ppm of how much benzene is in gasoline, neither of **Parker's** experts quantified **Parker's** exposure to benzene from gasoline.

Without conducting a *Frye* hearing (which neither party had requested), Supreme Court denied defendants' motion to preclude **Parker's** expert testimony. The court identified the issue as whether the causal relationship between benzene in gasoline and AML has general acceptance in the scientific community—particularly whether the experts used generally accepted principles and methodologies in arriving at their conclusions. The court recognized that **Parker's** experts did not cite to studies linking AML to exposure to benzene in gasoline or quantify **Parker's** exposure, but concluded that the experts distinguished the studies finding no increased risk of leukemia and that, while the failure to quantify exposure might require a hearing in some cases where there was less exposure, it was not necessary here.

Finally, the court determined that plaintiff's experts followed generally accepted principles and methodologies by detailing **Parker's** exposure, demonstrating the link between benzene and leukemia and presenting a dose-response relationship of 40 ppm-years (or the theory that there is no threshold of exposure under which there will be no negative effects to health). The court also found that Landrigan "track[ed]" the process of generating an opinion on causation in toxic tort cases
446 recommended *446 by the World Health Organization (WHO) and National Academy of Sciences (NAS).[2]

The Appellate Division reversed and dismissed the complaint, framing the issue as "to what extent the plaintiff was required to establish the precise level of his exposure to benzene in order to establish that his AML was caused by it through a scientifically-reliable methodology" (16 AD3d 648, 651 [2005]). The Court noted that neither of **Parker's** experts quantified his exposure to benzene—in particular, neither provided a time-weighted average in parts per million. Even if

the experts had established a threshold, they could not show that **Parker's** exposure exceeded it, and any conclusions as to the amount of **Parker's** exposure or whether the exposure caused his AML were therefore speculative.

The Court also rejected Landrigan's position that there is no threshold below which leukemia would not occur as "the scientific reliability of th[at] methodology has flatly been rejected as merely a hypothesis" (16 AD3d at 653). The Court noted that the experts did not use the three-step process approved by the WHO/NAS and that although they used studies demonstrating a link between benzene and AML, they did not prove the causal connection between the exposure to benzene *in gasoline.* We now affirm.

Discussion

At issue in this case is the admissibility of **Parker's** experts' opinions. The parties dispute whether the opinions should be analyzed under *Frye.* The introduction of novel scientific evidence calls for a determination of its reliability. Thus, the *Frye* test asks "whether the accepted techniques, when properly performed, generate results accepted as reliable within the scientific community generally" (*People v Wesley,* 83 NY2d 417, 422 [1994]; *see also People v Wernick,* 89 NY2d 111, 115-116 [1996]). *Frye* holds that "while courts will go a long way in admitting expert testimony deduced from a well-
447 recognized scientific principle or discovery, the thing from which the deduction *447 is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs" (*Frye,* 293 F at 1014).[3] It "emphasizes `counting scientists' votes, rather than on verifying the soundness of a scientific conclusion'" (*Wesley,* 83 NY2d at 439 [citation omitted] [Kaye, Ch. J., concurring]).

The *Frye* inquiry is separate and distinct from the admissibility question applied to all evidence—whether there is a proper foundation—to determine whether the accepted methods were appropriately employed in a particular case (*Wesley,* 83 NY2d at 429). "The focus moves from the general reliability concerns of *Frye* to the specific reliability of the procedures followed to generate the evidence proffered and whether they establish a foundation for the reception of the evidence at trial" (*Wesley,* 83 NY2d at 429).

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.*

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.

One 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. Here, for example, defendants did not monitor the level of
448 benzene in the air at the service stations. Nor were they *448 required to do so by law or regulation. Further complicating the process of arriving at a specific quantification in this case is that a significant portion of **Parker's** benzene exposure was through dermal contact—a factor that would not be addressed in the air-based ppm-years 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) (*see e.g. McClain v Metabolife Intl., Inc.,* 401 F3d 1233, 1241 [11th Cir 2005]; *Wright v Willamette Indus., Inc.,* 91 F3d 1105, 1106 [8th Cir 1996]). Where we depart from the Appellate Division is that we find 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.

The argument that precise quantification is not necessary finds support in case law from other jurisdictions. For example,

the Fourth Circuit has noted that

> "while precise information concerning the exposure necessary to cause specific harm to humans and exact details pertaining to the plaintiff's exposure are beneficial, such evidence is not always available, or necessary, to demonstrate that a substance is toxic to humans given substantial exposure and need not invariably provide the basis for an expert's opinion on causation" (*Westberry v Gislaved Gummi AB,* 178 F3d 257, 264 [4th Cir 1999]; *see also Heller v Shaw Indus., Inc.,* 167 F3d 146, 157 [3d Cir 1999]; *Hardyman v Norfolk & W. Ry. Co.,* 243 F3d 255, 265-266 [6th Cir 2001]).[4]

Some cases requiring an expert to establish the dosage at which a substance is toxic and the amount of exposure a plaintiff actually experienced also appear to recognize that an exact number may not be necessary (*see Wright,* 91 F3d at 1107 ["We do not require a mathematically precise table equating levels of exposure with levels of harm, but there must be
449 evidence from which a reasonable person could conclude that a defendant's *449 emission has probably caused a particular plaintiff the kind of harm of which he or she complains"]; *McClain,* 401 F3d at 1241 n 6).

There could be several other ways an expert might demonstrate causation. For instance, amici note that the intensity of exposure to benzene may be more important than a cumulative dose for determining the risk of developing leukemia. Moreover, exposure can be estimated through the use of mathematical modeling by taking a plaintiff's work history into account to estimate the exposure to a toxin. It is also possible that more qualitative means could be used to express a plaintiff's exposure. Comparison to the exposure levels of subjects of other studies could be helpful provided that the expert made a specific comparison sufficient to show how the plaintiff's exposure level related to those of the other subjects. These, along with others, could be potentially acceptable ways to demonstrate causation if they were found to be generally accepted as reliable in the scientific community.

Turning to the opinions offered by **Parker's** experts, although we reject the Appellate Division's requirement that the amount of exposure need be quantified exactly, we nonetheless conclude that the Appellate Division properly precluded them and properly deemed them insufficient to defeat summary judgment. The experts, although undoubtedly highly qualified in their respective fields, failed to demonstrate that exposure to benzene as a component of gasoline caused **Parker's** AML. Dr. Goldstein's general, subjective and conclusory assertion—based on **Parker's** deposition testimony—that **Parker** had "far more exposure to benzene than did the refinery workers in the epidemiological studies" is plainly insufficient to establish causation. It neither states the level of the refinery workers' exposure, nor specifies how **Parker's** exposure exceeded it, thus lacking in epidemiologic evidence to support the claim.

Dr. Landrigan's submissions were likewise insufficient. He reported that **Parker** was "frequently" exposed to "excessive" amounts of gasoline and had "extensive exposures . . . in both liquid and vapor form," which—even given that an expert is not required to pinpoint exposure with complete precision—cannot be characterized as a scientific expression of **Parker's** exposure level. Moreover, Landrigan concentrates on the relationship between exposure to benzene and the risk of developing AML—an association that is not in dispute. Key to this litigation is the relationship, if any, between exposure to
450 *gasoline* containing *450 benzene as a component and AML. Landrigan fails to make this connection perhaps because, as defendants claim, no significant association has been found between gasoline exposure and AML. Plaintiff's experts were unable to identify a single epidemiologic study finding an increased risk of AML as a result of exposure to gasoline. In addition, standards promulgated by regulatory agencies as protective measures are inadequate to demonstrate legal causation. Thus, the experts' opinions were properly excluded.

Parker's remaining contentions are without merit.

Accordingly, the order of the Appellate Division should be affirmed, with costs.

Order affirmed, with costs.

[1] PPM means parts per million—here, 100 parts benzene per one million parts of air. The TWA, or time-weighted average, is the average amount of a substance to which an individual is exposed over an eight-hour work shift. This measurement can also be expressed in ppm-years.

[2] Those steps are: (1) determining the plaintiff's exposure to the particular toxin; (2) general causation, which is proof that the toxin in question can in fact cause the illness, and the amount of exposure required to cause the illness (the dose-response relationship); and (3) specific causation—meaning the likelihood that plaintiff's illness was caused by the toxin, including eliminating other potential causes of the disease (*see* *Mancuso v Consolidated Edison Co. of N.Y., Inc.,* 56 F Supp 2d 391, 399 [SD NY 1999]).

[3] Although some amici urge the Court to adopt the federal standard (or some portions of it) as expressed in *Daubert v Merrell Dow Pharmaceuticals, Inc.* (509 US 579, 589-590 [1993] [requiring that scientific testimony be relevant and reliable in order to assist the trier of fact under Federal Rules of Evidence rule 702]), the parties make no such argument and acknowledge that *Frye* is the current standard in New York.

[4] We recognize that these cases employ a *Daubert* analysis. However, they are instructive to the extent that they address the reliability of an expert's methodology.