ESTABLISHING PROOF OF EXPOSURE

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The Fall 2010 issue of *Practice Perspectives: Product Liability & Tort Litigation* contained a thought-provoking article entitled "Genes for Justice? Using Gene Expression Analysis to Identify the Molecular Footprints of Environmental Hazards."[1] The authors of that article examined the possibility that in the future, genetic technology might be able to identify a "chemical footprint" in a person's genome, to provide evidence that the individual had exposure to a particular chemical. Though such a "footprint" would not be able to identify the source of the chemical, nor would it be able to prove that whatever disease or illness the individual had was "caused" by the chemical, it could provide evidence that exposure had occurred. Until such technology is available, however, courts are obliged to use a variety of ways to determine the nature and extent of exposure in cases of alleged chemically induced disease and illness. This article will examine how courts currently approach the exposure issue.

Many state courts, and certainly the federal courts, have articulated what is necessary in order for a plaintiff to prove causation in a toxic tort or product liability case alleging chemical exposure. One recent case articulated the requirements this way:

> In determining whether an alleged chemical exposure caused a particular disease or illness, an expert must establish the following criteria: (1) the toxic substance at issue must have been demonstrated to cause in humans the disease or illness suffered by the plaintiff; (2) the individual must have been exposed to a sufficient amount of the substance in question to elicit the health effect in question; (3) the chronological relationship between exposure and effect must be biologically plausible; and (4) the likelihood that the chemical caused the disease or illness in an individual should be considered in the context of other known causes.[2]

Stated another way, the burden is on the plaintiff to prove:

1) That the chemical at issue is capable of causing the disease or illness the plaintiff has (often referred to as "general causation"); and
2) That the chemical at issue did in fact cause the disease or illness this particular plaintiff has (often referred to as "specific causation").

In other words, "[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case."[3]

In many toxic tort cases, especially cases involving long-latency-period diseases like cancer, epidemiology is used to try to prove general causation (that a chemical is capable of causing a particular disease). However, numerous cases have held that epidemiology is the study of the occurrence of disease in populations and "does not in and of itself address the cause of an individual's disease."[4]


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Although defendants in toxic tort litigation routinely devote time and resources to understanding and addressing the epidemiological question—Can the chemical cause the disease?—it is equally important to devote resources to understanding the second question—To what amount of that chemical, if any, was the plaintiff exposed?

The science of individual exposure assessment, at least at the practical level, is one that is not very far advanced. Many courts have recognized this problem in the toxic tort context by saying that it is not necessary to quantify the amount of exposure with precision. However, what is clear, for most courts, is that exposure must be quantified in some way.

The most common ways of measuring potential exposure to a chemical or a physical agent are attempting to quantify it in the air or near a person's breathing zone (industrial hygiene testing) or measuring it in some bodily fluid, like blood or urine, or in tissue, like fat. These tests are almost all done in the work context, either as part of an industrial hygiene program or pursuant to certain requirements under the Occupational Safety and Health Act. (One exception may be periodic blood lead screening, but that is done almost exclusively on the small number of individuals perceived to be at high risk.) Therefore, when an allegation is made that exposure to a particular chemical occurred, objective evidence of the extent of, or even the existence of, exposure is almost never available.

How, then, do plaintiffs go about trying to prove exposure? One way has been by personal testimony. A plaintiff and/or others testify that they smelled something, saw something (like asbestos fibers), or were made sick by something. That type of evidence might suffice in a case in which the disease is a well-recognized entity caused by a particular material, such as asbestosis, caused by asbestos exposure. However, even in that situation, courts have insisted that for a plaintiff to recover against a particular asbestos supplier, there must be evidence that the plaintiff's exposure was on a regular basis over some extended period of time in proximity to where the plaintiff actually worked.[5]

That type of testimony may also be sufficient in a case in which the exposure and the effect are very close in time. For example, one court said, "Under some circumstances … 'if a person were doused with chemical X and immediately thereafter developed symptom Y, the need for published literature showing a correlation between the two may be lessened.'"[6] However, even in acute exposure situations, other courts have excluded testimony that the exposure caused the effect. In *Moore v. Ashland Chemical Inc.*,[7] the court excluded the opinion of a pulmonary specialist that the plaintiff had developed reactive airways dysfunction syndrome ("RADS"), an asthma-like condition, as a result of exposure during a cleanup operation to spilled chemicals that contained, among other things, toluene. The court held that the absence of evidence regarding the dose of chemical the plaintiff actually received, the plaintiff's other risk factors for the type of disorder he had, and the fact that the doctor had never treated another case of RADS based on this type of exposure scenario made the doctor's opinion speculative at best.[8]

In the absence of objective evidence of exposure, many courts do what the New York Court of Appeals recently did in *Parker v. Mobil Oil Corp.*,[9] a case alleging that exposure to benzene in gasoline, in a service-station environment, had caused acute myelogenous leukemia. First, it acknowledged the problem:

> 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 benzene in the air at the service stations. Nor were they 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.[10]

Then, it articulated the rule: "[W]e 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 [specific] causation are generally accepted in the scientific community."[11]

Indeed, in *Parker*, the court pointed out a couple of ways this could be done. It said that:

> 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. [For example,] [c]omparison 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.[12]

It should be mentioned that all mathematical models, by definition, require inputs that are based on assumptions (about chemical concentrations in the air, the location of the plaintiff, ventilation, and air direction and speed, among others). In addition, the assumptions that go into a model should be as vigorously scrutinized by the court for scientific validity and reliability as the model itself before the expert is permitted to testify to a jury. The problem is that many such models are so technical that once presented to a jury, they run the risk of being overwhelming, and the rule of "garbage in, garbage out" is apt to be overlooked.

The other method discussed by the New York Court of Appeals—comparison to the exposure levels of subjects in other studies—is also fraught with problems. One of the main problems is that an expert may get on the stand and simply say the exposure of the plaintiff is comparable to the exposures of people in certain studies, without offering any real support for the statement. Indeed, that is what the New York Court of Appeals found had occurred in the *Parker* case. The court held that the "general, subjective and conclusory assertion" of one of the plaintiff's experts, based on the plaintiff's deposition testimony, that the plaintiff "had 'far more exposure to benzene than did the refinery workers in the epidemiological studies' is plainly insufficient to establish causation."[13] The court pointed out that such testimony neither stated the level of the refinery workers' exposure nor specified how the plaintiff's exposure (at service stations) exceeded it.[14] Similarly, the court rejected another expert's "quantification" as insufficient when the expert said that the plaintiff was "frequently" exposed to "excessive" amounts of gasoline and had "extensive exposures … in both liquid and vapor form."[15] The court also criticized that expert for equating the plaintiff's exposure to gasoline to exposure to benzene, which was, at most, a tiny percentage of the gasoline product in the case.[16]

Other cases have taken the same approach. For example, in the Vermont Supreme Court case of *Blanchard v. Goodyear Tire & Rubber Co., et al.*,[17] a plaintiff alleged that his exposure to benzene while playing on a ball field as a teenager in the late 1960s and early 1970s had caused him to develop a rare form of non-Hodgkin lymphoma ("NHL"). The ball field was on a portion of a Goodyear rubber-manufacturing plant that operated from 1936 to 1986. With respect to the exposure issue, the Vermont Supreme Court acknowledged that "in many, if not most, toxic tort cases it is impossible 'to quantify with hard proof—such as the presence of the alleged toxic substance in the plaintiff's blood or tissue—the precise amount of the toxic substance to which an individual plaintiff was exposed.' "[18] However, the court also recognized that "plaintiffs in toxic exposure cases must demonstrate specific causation by submitting evidence concerning 'the amount, duration, intensity, and frequency of exposure.' "[19] Furthermore, the court said that "courts generally preclude experts from testifying 'as to specific causation without having any measurements of a plaintiff's exposure to the allegedly harmful substance.' "[20] In this case, the court rejected three types of "evidence" of exposure and affirmed a lower court's grant of summary judgment for the defense. It rejected the testimony of the plaintiff and his boyhood friends regarding the amount of time the plaintiff had played on the ball field, the odors they had smelled, and the grass discoloration they had observed. It rejected the report and testimony of the project manager of an environmental firm that had been retained to conduct a site investigation, and it rejected the plaintiff's experts' testimony that occupational exposure to benzene is associated with NHL and that this plaintiff's NHL was not caused by any immunodeficiency disorder.

With respect to the lay testimony, the court held that the testimony provided no evidence that benzene was on or in the ball field when the plaintiff was playing there. Perhaps even more significantly, the court held that:

> even if we were to assume that benzene-containing products made their way into the gully and through the field, there is no evidence indicating the amount or concentration of benzene that was present. Nor is there any evidence indicating plaintiff's level of exposure to any benzene that may have been present on the field. Nor is plaintiff able to point to studies indicating a risk of cancer posed by exposure to limited

> amounts of benzene from petroleum products in an outside environment.[21]

The court added, "Putting aside plaintiff's failure to demonstrate the presence of benzene in the field, a jury could only wildly speculate on the level of plaintiff's exposure to any such benzene and on the relationship between any such exposure and plaintiff's disease."[22]

Even cases involving chemical spills, and claims for "medical monitoring" based on no more than "increased risk of adverse outcomes," have been subjected to strict proof regarding exposure. In a case involving a train derailment and fire that resulted in an evacuation, the court held that "[m]ere residence in the impact zone is insufficient evidence of contamination and increased risk because it ignores any individual variables, most notably, at what level the named Plaintiffs were actually exposed [to the chemical]."[23]

Since it appears so difficult to establish exposure, how can it be done? Some courts basically just finesse the issue. One court, for example, disposed of an argument regarding exposure by saying:

> The defendants maintain that [the plaintiff's expert's] dose reconstruction is speculation because it presumes that [the plaintiff] consumed dust. Having considered the briefs, however, the court concludes that the dose reconstruction is specific and reasonable enough to take it beyond the realm of speculation, especially since it is undisputed that everyone consumes a given amount of dust each day.[24]

Another court held that the requirements for proving the requisite amounts of exposure from a Superfund site could be established by the use of a variety of types of indirect evidence. These included information from U.S. EPA and state site remediation reports that discussed soil contamination levels and methods of removal, the fact that the site was open for an extended period of time, evidence that airborne contaminants could travel several miles, reports that people other than the plaintiffs had complained of odors and symptoms, the fact that the plaintiffs spent time in a town park adjacent to the site, and the fact that all the plaintiffs lived within four miles of the site.[25] Finally, the court found that U.S. EPA had written with respect to the site that because "air emissions occurred during the excavation and likely occurred while the excavation was left open for two years, it appears to be likely that some exposure occurred to residents surrounding the Site."[26]

Here, although the court relied on numerous types of evidence, none of it dealt with the dose of chemicals the plaintiffs actually received.

Sometimes, to fill evidentiary gaps, legislatures step in. For example, one court decided a workers' compensation case against a firefighter who claimed he developed non-Hodgkin lymphoma by virtue of smoke inhalation during his work as a firefighter for the City of Burlington, Vermont. The court held, among other things, that the firefighter had not quantified his exposure sufficiently.[27] However, the court noted that the state legislature had recently passed a statute providing that when a firefighter dies from certain cancers, including lymphoma, "the firefighter shall be presumed to have suffered the cancer as a result of conditions in the line of duty."[28] That presumption could be rebutted, but the burden would be on the employer.

Similarly, Congress has periodically stepped in to ease the burden of proving exposure. For example, from the late 1940s until the early 1960s, the United States conducted above-ground tests of atomic weapons. These activities may have exposed to ionizing radiation a considerable number of individuals downwind of the testing ("downwinders"), but radiation-exposure levels were never quantified. In the 1970s and 1980s, members of that group (and others, who mined and milled radioactive materials, such as uranium) alleged that their exposure to radiation caused them to develop cancer more frequently than those who were not so exposed.

Responding to these concerns, Congress enacted the Radiation Exposure Compensation Act ("RECA")[29] in 1990, which recognized that "the lives and health of [individuals] were involuntarily subjected to increased risk of injury and disease to serve the national security interests of the United States."[30] With respect to "downwinders," in the absence of quantifiable exposure levels, Congress established both temporal and geographic requirements for purposes of determining "exposure." Geographically, only residents in a defined "affected area" were eligible for compensation. The "affected area" was defined to include certain counties in

Utah and Nevada; "that portion of Clark County[, Nevada,] that consists of townships 13 through 16 at ranges 63 through 71; and that part of Arizona that is north of the Grand Canyon and west of the Colorado River."[31] Temporally, the claimant must have been present in the "affected area" for at least one year between January 21, 1951, and October 31, 1958, or continuously between June 30, 1962, and July 31, 1962.[32]

Similarly, following 9/11, Congress passed the September 11th Victim Compensation Fund of 2001 (the "9/11 Fund") as part of the Air Transportation Safety and System Stabilization Act.[33] To qualify for compensation under the 9/11 Fund, an individual was required to have been aboard one of the 9/11 flights or to meet the statutory requirements for an "eligible individual." An "eligible individual" (or his or her family) had to prove that: (1) the individual was present at the time of or in the "immediate aftermath" of a crash and that (2) he or she suffered physical harm or death (3) as a result of that crash.[34] As in RECA, Congress specified that "presence" at the crash site had both temporal and geographic requirements. Temporal proximity was straightforward because it turned on physical presence within a discrete time window—the first 96 hours after the crash for rescue workers and the first 12 hours for everyone else.[35]

In contrast, geographical proximity was harder to quantify because the three crash sites differed greatly. Interestingly, Congress left the determination of the geographical boundaries up to a "Special Master" established by the statute. The Special Master concluded that the Pentagon and Shanksville, Pennsylvania, sites were more isolated; thus, no rules were necessary to specify geographical proximity to them. However, the World Trade Center site required detailed specification. Some argued that any person on the island of Manhattan at the time of the attacks should be allowed to file for compensation. The Special Master's Office, however, took a narrower view. In reaching this conclusion, attorneys for the Special Master examined aerial photographs and maps of debris dispersal in New York City and determined that the "Pedestrian No Access Zone" enforced by the New York City Police Department in the days following September 11, 2001, was a fitting area. However, to err on the side of inclusiveness, a street block was added to the perimeter of the zone.[36]

The 9/11 Fund officially closed on June 16, 2004; however, the recent passage of the James Zadroga 9/11 Health and Compensation Act of 2010[37] (the "Act") reopened it. The Act expands the class of eligible individuals in a number of ways. First, the amendments expand the temporal requirement, enlarging the time window from the first 12 or 96 hours after the crash to the period ending May 30, 2002.[38] Second, the amendments expand the geographical boundaries to include not just the World Trade Center, the Pentagon, and the site of the Shanksville, Pennsylvania, crash, but also other buildings or portions of buildings that were destroyed as a result of the terrorist-related aircraft crashes; any area related to or along the routes of debris removal, such as barge routes and the Fresh Kills Landfill; and any contiguous area designated by the Special Master because of a demonstrated risk of physical harm at the site as a result of the crashes or their aftermath.[39]

In addition to the revisions to the 9/11 Fund, the Act creates another funding mechanism for 9/11 victims. This additional approach provides medical benefits and treatment to eligible individuals suffering from a "WTC-related health condition" as listed in the Act.[40] Under the Act, to be eligible for monitoring and treatment benefits, individuals must first qualify as "WTC responders" or "WTC survivors."[41] Those WTC responders and WTC survivors must also satisfy temporal and geographic requirements.[42]

As can be seen, exposure can be difficult to prove in tort litigation, and many courts have taken cases away from juries and ruled for defendants in cases in which plaintiffs have not quantified their exposure by the use of valid and reliable scientific methods. Sometimes, when courts have let cases proceed in the absence of quantifiable exposure data, they have done so on the basis of little more than *post hoc* rationalization. In certain situations, for sound public-policy reasons, legislatures have become involved, either to shift burdens of proof or to establish the prerequisites that are necessary in order to establish exposure. ■

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[1] *Jones Day Practice Perspectives: Product Liability & Tort Litigation*, Fall 2010, pp. 8–11, 42.

[2] *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1156 (E.D. Wash. 2009), citing Eaton, D.L., "Scientific Judgment and Toxic Torts—A Primer in Toxicology for Judges and Lawyers," 13 *Journal of Law and Policy* 5, pp. 38–40 (2003).

[3] *Allen v. Pennsylvania Engineering Corp.*, 102 F.3d 194, 199 (5th Cir. 1996).

[4] *Beck v. Koppers, Inc.*, 2006 WL 270260 (N.D. Miss. 2006).

[5] *See Thacker v. UNR Industries, Inc.*, 603 N.E.2d 449, 457 (Ill. 1992), and *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1162–63 (4th Cir. 1986).

[6] *Bonner v. ISP Technologies, Inc.*, 259 F.3d 924, 931 (8th Cir. 2001), quoting *Heller v. Shaw Industries, Inc.*, 167 F.3d 146, 154 (3d Cir. 1999).

[7] 151 F.3d 269 (5th Cir. 1998).

[8] *Id.* at 278–79.

[9] 857 N.E.2d 1114 (N.Y. 2006).

[10] *Id.* at 1120.

[11] *Id.* at 1121.

[12] *Id.* In the recent case of *Nonnon v. City of New York*, 2011 WL 4089536 (N.Y.A.D. 1 Dept.), an appellate division of the New York Court of Appeals held that "the strength of the epidemiological data alone permits an inference of (specific) causation." That would seem to be insufficient, under the ruling in *Parker*, to prove individual causation, because epidemiology is generally acknowledged to be a study of populations, not of individuals, and epidemiology alone cannot prove causation in an individual case.

[13] *Id.*

[14] *Id.* at 1121–22.

[15] *Id.* at 1122.

[16] *Id.*

[17] – A.3d –, 2011 WL 3505236 (Vt. 2011).

[18] *Id.* ¶ 7, quoting *Plourde v. Gladstone*, 190 F. Supp. 2d 708, 721 (D. Vt. 2002).

[19] *Id.* ¶ 6, quoting *White v. Dow Chem. Co.*, 321 F. App'x 266, 273 (4th Cir. 2009).

[20] *Id.* ¶ 7, quoting *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1157 (E.D. Wash. 2009).

[21] *Id.* ¶ 11.

[22] *Id.*

[23] *Mann v. CSX Transportation, Inc.*, 2009 WL 3766056 (N.D. Ohio 2009).

[24] *Beck v. Koppers, Inc.*, 2006 WL 270260 (N.D. Miss. 2006).

[25] *Donaldson v. Central Illinois Public Service Co.*, 767 N.E.2d 314, 332–33 (Ill. 2002).

[26] *Id.* at 333, quoting CIPS's *Air Monitoring Report*.

[27] *Estate of George v. Vermont League of Cities and Towns*, 993 A.2d 367 (Vt. 2010).

[28] *Id.* Reiber, C.J., dissenting, p. 385, n.16.

[29] Pub. L. No. 101-426, 104 Stat. 920 (1990).

[30] *Id.* at § 2(a)(5).

[31] *Id.* at § 4(b)(1).

[32] *Id.* § 4(a)(1). Other temporal requirements also apply. *See* § 4(a)(1), 4(a)(1)(A), 4(a)(2), 4(b)(2).

[33] Pub. L. No. 107-42, 115 Stat. 230 (2001).

[34] *Id.* at § 405(c).

[35] 28 C.F.R. Part 104.2(b).

[36] Feinberg, K.R., *Final Report of the Special Master for the September 11th Victim Compensation Fund* (2004), vol. 1, p. 19, and n.58, *available at* http://www.justice.gov/final_report.pdf (last visited Dec. 14, 2011).

[37] Pub. L. No. 111-347, 124 Stat. 3623 (2011).

[38] *Id.* at § 201.

[39] *Id.*

[40] *Id.* §§ 3312(a) and 3322.

[41] *Id.* §§ 3311–3323.

[42] *Id.* § 3311(a)(2), § 3311(a)(1)(B), § 3306(7).