Personalized Toxic Tort Litigation?

by Jon Ferry

Proving causation in toxic tort litigation has bedeviled practitioners and courts for decades. Current work in the field of toxicogenomics, however, is closing in on ways to refine the practice, enabling a much more targeted approach to analyzing how a particular environmental hazard may have caused a particular plaintiff’s injury. Move over, personalized medicine. Personalized toxic tort litigation may be just around the corner.

**What is a Tort?** Tort cases are those in which the plaintiff claims to have been injured by the defendant’s negligent (or, rarely, intentional) misconduct. The best known examples are automobile accident and medical malpractice cases. In a toxic tort case, the plaintiff (or, often, a whole class of plaintiffs) claims that a sickness or injury is the result of exposure to some dangerous substance that the defendant negligently or intentionally put into the environment—the hundreds of thousands of asbestos cases that have been filed, for example.

**Causation and Toxic Torts.** In all tort cases, the plaintiff must show that the defendant’s conduct was what the law calls the “proximate cause” of his or her injury. This is especially tricky in a toxic exposure case. The plaintiff must prove two kinds of causation: that the substance in question can cause the kind of injury the plaintiff suffered (general causation), and that the substance *did in fact* cause the plaintiff’s injury (specific or individual causation). Proving specific causation is usually where the difficulties for the plaintiff arise.

Take a plaintiff claiming a lung disease as a result of exposure to asbestos on the job. It is widely accepted that exposure to asbestos can cause a variety of lung ailments. But how does a plaintiff prove that asbestos exposure more likely than not (the standard of proof in civil cases) *did* cause the lung disease from which he or she now suffers—as opposed to other workplace chemicals, smoking (a common issue in these cases), air pollution, genetics, or bad luck?

In most toxic tort cases, it is not possible to prove specific causation definitively. There is just no clear-cut way to prove that plaintiff X’s asbestos exposure caused his lung disease. So the question is, in the absence of definitive scientific proof, will the courts allow plaintiffs to introduce and rely on evidence establishing the probability that the toxic substance caused the plaintiff’s disease? Or more specifically, what level of probability will be required?

In order to prove causation – both general and specific -- plaintiffs frequently offer epidemiological studies designed to show whether an individual exposed to a potentially toxic substance is at an increased risk of developing a particular disease. A typical epidemiological study will be based on studies of large groups of individuals who have been exposed to the suspect substance. Researchers first attempt to determine an expected number of illnesses for the population as a whole. This number makes up the denominator in the relative risk ratio. The numerator in the ratio is the actual observed number of illnesses. Thus, if studies indicate the 100 out of every 100,000 persons in the non-exposed groups should fall ill and 200 out of every 100,000 persons in the exposed groups actually falls ill, the Relative Risk is 2.1

As this example makes clear, however, because there is a baseline occurrence of 100 illnesses in the general population, it is very likely that some number those with the illness in the exposed group would have become sick even without the exposure. Statistically, one half of those in the exposed group who got sick (200) would probably have fallen ill anyway, even without the exposure. What the statistics cannot reveal is whether the exposure caused the illness in any one specific individual in the exposed group. So as a matter of probability, out of any 200 sick plaintiffs before a court, 100 are likely to be entitled to recovery, 100 are not, and neither the court nor the jury can tell in which group an individual plaintiff belongs.

As one commentator suggested, allowing recovery under such circumstances turns the tort system into a lottery.2 A court in a breast implant case made a similar point: “If exposure to breast implants does not at least double the risk of injury, then more than half of the population suffering from the injuries allegedly caused by breast implants would be injured anyway (the background injury rate of injury), thereby disproving legal causation.”3

Some courts, on the other hand, have determined that a lower measure of relative risk may be submitted to the jury along with other evidence tending to prove causation. One federal court determined that a study need simply show a relative risk greater than one in order to be “statistically relevant” and qualify for submission to the jury as relevant evidence. *Pick v. American Medical Systems, Inc.*, 958 F.Supp. 1151, 1160 (E.D. La. 1997).4 Although rare, a court may also determine that a relative risk of 2 or greater is sufficient on its own to establish that it is more likely than not that the substance caused the plaintiff’s illness and support a finding of liability.5

**Enter toxicogenomics.** Genomic level analysis provides the prospect for significantly sharpening the concept of relative risk, and perhaps in some cases altogether supplanting population based risk analysis with proof of specific causation. In the first case, certain plaintiffs may have genetic characteristics that make them more susceptible to exposure of toxic substances than the general population. In the second case, analysis may reveal genetic mutations in exposed individuals that can be more directly traced to a specific causation of the plaintiff’s condition – a telltale genetic signature of the exposure.

As previously discussed, calculation of relative risk requires the comparison of the incidence of a disease in a control group versus exposed group. Genetic testing may show that certain individuals are more (or less) susceptible to illness from exposure thereby increasing (or decreasing) the relative risk assessment for particular plaintiffs within the exposed group. For example, genetic testing of workers exposed to trichloroethylene (TCE) has shown that workers with certain genetic attributes may be up to 10 times as likely to suffer kidney cancer after TCE exposure than workers without those genetic attributes. Specifically, a study found that workers with an active form of two types of genes were at 10 times greater risk of developing cancer than those without an active form of either gene. The study concluded that about 25-40% of the population might be in the higher risk category.6
**Personalizing Relative Risk.** Essentially, the utility of this genetic testing in the toxic tort context is to further segment the exposed group and determine if the disease in certain members of that group is more likely to have been caused by the exposure. A plaintiff with specific genetic characteristics might be in a position to show a much higher relative risk than an individual randomly chosen from the general population. Conversely, a defendant might be in a position to show that a specific plaintiff has a lower relative risk based on the presence of or lack of certain genetic characteristics.

From the toxic tort litigation perspective, an important result is likely to emerge from an increased understanding of the interaction between toxic exposures and genetic predisposition to illness. Plaintiffs who at one time could not show relative risk higher than 2 might now be able to show a significantly elevated relative risk based on their combined exposure and genetic profile. Flipping it around to the defendants' perspective, companies that expose people to substances may now be subject to liability from plaintiffs who before could not produce sufficient evidence of causation to withstand summary judgment.

**Expression Levels: From Relative Risk to Actual Causation.** Although not yet fully developed, the field of toxicogenomics holds some potential for eliminating the need for risk analysis entirely. Simply put, toxicogenomics seeks to measure a genetic response to specific toxic exposures by evaluating expression levels of relevant genes. The identification of a toxigenic response – finding gene expression levels in an individual's tissues consistent with a toxic exposure – may allow litigants to confirm or refute actual exposure to the substance at issue and determine, at least in the eyes of the court, if the substance at issue actually caused the subjects illness.

The first step in such an analysis requires identifying a unique response signature or gene expression profile associated with a potentially toxic substance. One hurdle in identifying this toxigenic signature is the vast amount of information that must be analyzed when monitoring the expression levels of the thousands of genes in human tissues. The ability to measure such expression levels is a developing technology that has advanced substantially in recent years, with well-documented advances in genomic sequencing and microarray technologies permitting the monitoring of the expression levels of thousands of genes at the same time.1

Showing that a particular plaintiff exhibits the same gene expression signature associated with exposure to a particular toxic substance may be sufficient to prove exposure, but proving that such expression is then the cause of the plaintiff’s illness is another step entirely. Linking the gene expression to toxicity indices is the next step required to show that exposure to the substance in turn caused the illness at issue. Ultimately, scientists may be able to determine that the specific exposure triggered the expression of specific genes which in turn caused the illness in question. For purposes of toxic tort litigation, that would amount to proving a direct chain of causation from exposure to the toxic substance to the plaintiff’s actual physical harm. This could, in turn, eliminate the need for evidence of increased risk within the plaintiff’s population, no matter how narrowly defined and enable truly personalized toxic tort litigation.

The emerging field of toxicogenomics presents important possibilities for refining a toxic tort litigant’s ability to prove causation, which is the critical step in establishing culpability. As with any new technology, the new tools of toxicogenomics will be subject to the gatekeeping functions of the court with regard to the admissibility of evidence. Ultimately, toxicogenomics holds the promise of providing more equitable outcomes by increasing the accuracy with which toxic exposures are linked to actual injuries and ensuring that such individuals are fairly compensated. More broadly, the trend toward personalized toxic tort litigation is reflective of the increasing prevalence of personalized genetic information in nearly every corner of society, including the courtroom.

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1 See Russelyn S. Carruth and Bernard D. Goldstein, Relative Risk of Greater Than Two in Proof of Causation in Toxic Tort Litigation, 41 Jurimetrics 195 (collecting cases).


3 In re Breast Implant Litigation, 11 F.Supp.2d 1217, 1226 (D. Col. 1998); see also, Carruth and Goldstein, supra note 3 at 200-202 (collecting cases).

4 Some courts have also found that an expert can come to a conclusion regarding causation without finding that the Relative Risk to an exposed person is two or greater. See, Ambrosini v. Labarraque, 101 F.3d 129, 135-137 (D.C. Cir. 1996) (rejecting district court's requirement that the expert witness opine that the relative risk to plaintiff is two or greater and admitting testimony by expert that he found an association between birth defects and drug); In re Joint Eastern & Souther District Asbestos Litigation v. Owen Corning Fiberglas Corp., 964 F.2d 92, 97 (2nd Cir. 1992 (finding no need for epidemiological studies showing relative risk to plaintiff in excess of two when additional evidence that factors our other known risk factors are also presented to the jury).  


7 Pierce, John R. and Terrence Sexton, Toxicogenomics: Toward a Future of Toxic Tort Causation, 5 North Carolina Journal of Law & Technology 33 (2003); Committee on Applications of Toxicogenomic Technologies to Predictive Toxicology and Risk Assessment, National Research Council, Applications of Toxicogenomic Technologies to Predictive Toxicology and Risk Assessment, pp. 139-141 (discussing prospects for universal standards in application of microarray methodology).