

Toxic Torts

COMMENTARY

REPRINTED FROM VOLUME 26, ISSUE 11 / JULY 2, 2008

DNA Evidence in Toxic-Tort Cases: Still Weak Proof of Exposure, Risk and Injury

By Anthony G. Hopp, Esq.

Ten years ago lawyers and judges dealing with personal-injury cases did not have to ask, What is an injury? Or what is evidence of damage? A broken arm or a fractured leg was an injury. Medical bills and lost wages were evidence of damage. Thanks to the growing availability of DNA testing, however, the questions of when a physical impact has occurred and when that impact rises to the level of compensable damage have become more complex. Even more complicated is the question of when a physical impact at a sub-cellular level increases a person's risk of future illness.

There are three ways in which DNA evidence has been used in recent years in toxic-tort cases: as proof of exposure, as proof of injury and as proof of increased risk of future injury. Unlike in the criminal context, however, where DNA evidence can be near-conclusive proof of guilt or innocence, DNA evidence in civil cases is much less probative.

DNA Evidence as Proof of Exposure

Over the past decade genetic studies have identified several DNA markers that can be used to prove exposure to toxic substances. Two of these are DNA adducts and cytokines. While increased levels of DNA adducts and cytokines are proof that the plaintiff has experienced an impact at the sub-cellular level, they are not specific to any particular exposure or time frame and therefore are only weak proof that a particular defendant's product or substance caused that impact.

DNA Adducts

Polycyclic aromatic hydrocarbons, or PAHs, are formed by the incomplete combustion of carbon-containing substances such as wood, paper, coal, oil and cigarettes.

Smoked and grilled foods also contain PAHs. When PAHs enter a person's body, they can bind with the proteins that make up a cell's DNA. In theory, these stray molecules, called DNA adducts, can interfere with the orderly replication of DNA and lead to mutations.

For the past 20 years scientists have been working on ways to identify and count the number of adducts that are formed when human and animal tissues are exposed to PAHs.¹ This work has been largely successful, and there are now several methods available to detect, quantify and characterize DNA adducts in PAH-exposed tissue.

Despite the advances in technology for finding and counting DNA adducts, they remain a very imprecise measure of PAH exposure for several reasons. First, while adducts can be formed by contact with foreign substances, some DNA adducts are formed by metabolic processes not involving external exposures.² So simply counting DNA adducts will not indicate how many came from an external exposure and how many resulted from background metabolic processes.

Second, DNA adducts are not specific to any particular PAH. All sorts of PAHs form DNA adducts. A smoker who eats grilled or smoked meats and who also is exposed to the defendant's PAH-containing product will likely have high levels of DNA adducts in his or her blood, but it will be impossible to separate the adducts from the alleged tortious exposure and the adducts that arise from the plaintiff's lifestyle (for example diet and smoking).

Finally, DNA adducts are most often measured in white blood cells. They can be measured in other tissues, such as lung or liver tissue, but these tests require biopsies. Biopsies are expensive and carry more risk of complication than

blood tests. White blood cells do not become cancerous, and they do not normally live more than a few months. So they can only be used to measure comparatively recent exposures. While DNA adducts in white blood cells are a convenient surrogate for underlying tissues, they may not accurately reflect the effect of long-term PAH exposure on the plaintiff's lungs, liver or other organs.

DNA adduct studies are useful, therefore, as rough proof of exposure to PAHs. Their value beyond proof of recent exposure, however, is open to debate.

Cytokines

The use of cytokines as an indication of exposure is much less advanced than the science of DNA adduct studies. Nevertheless, the use of this method is growing.³

Cytokines are signaling peptides that consist of water-soluble proteins. They are produced in tissues undergoing defense, growth, differentiation and repair processes.⁴ Infection and inflammation result in the induction of cytokine-mediated immune responses. By exposing red blood cells to various toxic substances, scientists have been able to identify the type of cytokine "signatures" created by such exposures. Thus far, the cytokine signatures for benzene and hexavalent chromium have been identified.⁵ Work is underway to identify additional signatures.⁶

DNA evidence has been used in toxic-tort cases as proof of exposure, injury and increased risk of future injury.

The major drawback for cytokine evidence as proof of exposure is its novelty. While its promoters claim that cytokine studies are based on the same type of DNA technology that has been widely accepted in criminal cases, the admissibility of cytokine evidence in civil cases simply has not been tested in the courts. Other limitations include the fact that cytokine test results cannot be used to tell when a particular exposure occurred, and they do not take into account factors like an individual's unique susceptibility to certain exposures and the cumulative effects of repeated exposures over time.⁷

DNA Evidence as Proof of Injury

Most states have some variation on the basic rule for when a personal-injury claim is fit for adjudication. The plaintiff must have an actual physical injury or an actual disease, pain or impairment of some kind.⁸ When the alleged injury or damage occurs at a sub-cellular level, however, courts are split with respect to whether the plaintiff's

claim warrants a hearing. While a handful of courts have allowed plaintiffs to proceed based on nothing more than damage to their DNA, the majority rule appears to be that a clinical symptom or disease is necessary before a personal-injury claim exists.

Rainer v. Union Carbide Corp. involved claims by uranium enrichment workers who said they had been exposed to dangerous radioactive substances without their knowledge for many years.⁹ They were not suffering from clinical diseases but claimed that they had suffered sub-cellular damage to their DNA and chromosomes.

The court acknowledged that ionizing radiation is both toxic and carcinogenic. It has conclusively been demonstrated to cause bone cancer in radium (dial painters), lung cancer (uranium miners), leukemia (atomic bomb survivors), thyroid cancer (Chernobyl survivors) and various other malignancies (medical radiation workers).

Still, the plaintiffs in the *Rainer* case were not ill. They testified that they had recently undergone physical examinations and were found to be in good health.

One of plaintiffs' experts testified that the workers were nonetheless injured because they had suffered sub-cellular damage to their DNA. He conducted blood tests that revealed an 8 percent rate of chromosomal abnormalities, compared to a normal or "background" rate of 1.3 percent. Other experts said the disruption of the plaintiffs' DNA raised the risk of chromosome misrepair and increased the likelihood that the plaintiffs would develop cancer in the future.

While acknowledging the possibility of an increased risk of cancer, the court nonetheless rejected the plaintiffs' claims.¹⁰ The 6th U.S. Circuit Court of Appeals noted, "Given that negligently distributed or discharged toxins can be perceived to lie around every corner of the modern industrialized world, and their effects on risk levels are at best speculative, the potential tort claims involved are inherently limitless and endless."

The 6th Circuit went on to hold that accepting the plaintiffs' claims would create the possibility of litigation by any person experiencing even the most benign sub-cellular damage:

Based on the average American's exposure to chemically processed foods, toxic fumes, genetically modified fruits and vegetables, mercury-laden fish, and hormonally treated chicken and beef, this might encompass a very large percentage of the total population.

In addition to the "floodgates" rationale for not allowing such claims, the 6th Circuit noted that allowing such suits to

proceed would be of little benefit to plaintiffs in the long run. Many states have a “one claim” or “claim-splitting” rule that limits plaintiffs in tort cases to one chance to have their grievances redressed. A plaintiff seeking compensation for a sub-cellular or sub-clinical injury would be limited to the presumably nominal damages that he or she could recover on such a claim. If the plaintiff later became clinically ill as an alleged result of the same exposure, any subsequent claim would be barred.

The *Rainer* court said, “Allowing this suit to proceed would thus do a great disservice to those plaintiffs who might in fact later come down with the very diseases they so rightly fear.”

Finally, the court observed that calculating damages for a sub-cellular or sub-clinical injury would be difficult or impossible. The plaintiffs had not incurred any medical costs and were not in any pain or discomfort. Their alleged injuries had caused no financial losses or physical impairments. A trier of fact would not be able to assess damages in such a case without speculating. Other courts have reached similar conclusions.¹¹

A handful of courts have held that whether a sub-cellular or sub-clinical injury is a present, physical harm is a question of fact for the jury to decide.¹² In *In re MTBE Products Liability Litigation* the court considered the testimony of the plaintiffs’ expert that MTBE molecules, when ingested or inhaled, form DNA adducts.¹³ The expert further claimed that the presence of the adducts interferes with DNA replication, inducing mutations in the newly formed DNA. These mutations can lead to errant cell production and eventually tumors.

The court held that this evidence was sufficient to support emotional-distress claims but recognized that the courts that have declined to recognize sub-cellular damage as an injury have done so in the context of claims for physical injury rather than emotional distress.¹⁴

DNA Evidence as Proof of Increased Risk

In theory DNA damage should be a solid indication of increased risk of future disease. One might be tempted to think that this is particularly true of DNA adducts. After all, adducts interfere with DNA replication, and misreplication of DNA can lead to mutations and eventually to tumors. The reality is not so simple, for several reasons.

We all are under constant attack from DNA-damaging substances. PAHs are just one of many substances that are considered genotoxic. If each toxic insult to our DNA resulted in a tumor, none of us would live past infancy. We survive this constant assault because our bodies have

DNA repair mechanisms that clear DNA adducts before they can cause mutations or other diseases.

Further, only a select few chromosomes are responsible for the formation of tumors. DNA can be damaged in other locations and not lead to tumor formation. The truth is, while the human genome has been decoded, scientists still do not know what precise functions most genes serve. The mere fact of adduct formation, therefore, proves very little.

Still, there is a general relationship between some PAH exposures and cancer. No one would deny that heavy cigarette smoking can increase a person’s risk of lung cancer. Likewise, smokers have high DNA adduct levels in lung tissue and white blood cells. But despite intensive study, researchers have not been able to correlate adduct levels with risk levels. As one commentator noted:

It is a common misconception that the magnitude of elevation of adduct levels in the tissue of smokers should reflect the magnitude of the increase in risk of developing cancer in that organ from smoking. It would be very convenient if life was that simple, but there is no direct relationship. Adduct levels in these circumstances are indicators of carcinogenic hazard but cannot, of themselves, be equated to quantifiable risk.¹⁵

The majority rule appears to be that a clinical symptom or disease is necessary before a personal injury claim exists.

The author goes on to note that due to the multistage nature of tumor formation, it would be surprising if a single parameter, such as adduct levels, could provide a quantitative estimate of cancer risk.

The most that anyone can say is that people who are exposed to high concentrations of PAHs tend to have high levels of PAH-DNA adducts and that people who have higher PAH exposure also tend to have higher rates of PAH-related cancers. It is not yet possible, and it may never be possible, to correlate adduct levels and risk levels.

Conclusion

Despite continuing advances in the field of human genomics, DNA evidence is still very weak proof in civil cases involving toxic torts. This is really not all that surprising when one considers the job DNA evidence is asked to do in civil cases as opposed to criminal cases. In criminal cases, DNA evidence is asked to make an identification:

Does blood sample A match the blood from person B? This is a discrete, if highly complex, question.

In civil cases, however, DNA evidence is being asked to answer questions regarding multistage processes that vary from exposure to exposure, person to person and illness to illness: Did substance A cause person B to develop tumor C? While it is possible that the science of genomics may one day be able to answer such questions, that day is likely still a long way off.

Notes

¹ E. Kriek *et al.*, *Polycyclic Aromatic Hydrocarbon-DNA Adducts in Humans: Relevance as Biomarkers for Exposure and Cancer Risk*, 400 *MUTATION RES.* 215-331 (1998).

² David H. Phillips, *DNA Adducts as Markers of Exposure and Risk*, 577 *MUTATION RES.* 284-92 (2005).

³ Bruce Gillis *et al.*, *Identification of Human Cell Responses to Benzene and Benzene Metabolites*, 90 No. 3 *GENOMICS* 324 (2007).

⁴ *Id.*

⁵ *Id.*; Igor M. Gavin *et al.*, *Identification of Human Cell Responses to Hexavalent Chromium*, 48 *ENVTL. & MOLECULAR MUTAGENESIS* 650-657 (2007).

⁶ Mark Hansen, *DNA Posed to Show Its Civil Side*, A.B.A. J. (Mar. 2008).

⁷ *Id.*

⁸ *Boyd v. Orkin Exterminating Co.*, 381 S.E.2d 295, 298 (Ga. Ct. App. 1989); *Henry v. Dow Chem. Co.*, 701 N.W.2d 684 (Mich. 2005); *Parker v. Wellman*, 230 Fed. Appx. 878 (11th Cir. 2007).

⁹ 402 F.3d 608 (6th Cir. 2005).

¹⁰ Citing *Wood v. Wyeth-Ayerst Labs.*, 82 S.W.3d 849, 857 (Ky. 2002).

¹¹ See *In re Rezulin Prods. Liab. Litig.*, 361 F. Supp. 2d 268 (S.D.N.Y. 2005) (under Texas law, sub-cellular damage absent any clinically manifest detriment is not a compensable injury); *Parker*, 230 F. Appx. 878 (sub-cellular damage resulting in sub-clinical injury does not constitute current, physical harm); *Schweitzer v. Consol. Rail Corp.*, 758 F.2d 936 (3d Cir. 1985) (sub-clinical injury resulting from exposure to asbestos is insufficient to constitute actual loss or damage); *Laswell v. Brown*, 683 F.2d 261 (8th Cir. 1982) (allegations of exposure to an "unusually high risk of disease is genetically passed cellular" damage was insufficient to state a claim); *Eagle-Picher Indus. v. Liberty Mut. Ins. Co.*, 682 F.2d 12 (1st Cir. 1982) (insurance policies do not cover sub-clinical claims).

¹² *Werlein v. United States*, 746 F. Supp. 887 (D. Minn. 1990), vacated in part on other grounds, 793 F. Supp. 898 (D. Minn. 1992); *Brafford v. Susquehanna Corp.*, 586 F. Supp. 14 (D. Colo. 1984); *Bryson v. Pillsbury Co.*, 573 N.W.2d 718 (Minn. Ct. App. 1998).

¹³ 528 F. Supp. 2d 303 (S.D.N.Y. 2007).

¹⁴ *Id.* at 315.

¹⁵ Phillips, *supra* note 2, at 288.

Anthony Hopp is a partner in **Wildman, Harrold, Allen & Dixon** in Chicago. He concentrates his practice in the representation of defendants of environmental and toxic-tort lawsuits.