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The Use of Risk Assessment Evidence to Prove Increased Risk and Alternative Causation in Toxic Tort Litigation

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THE USE OF RISK ASSESSMENT EVIDENCE TO PROVE INCREASED RISK AND ALTERNATIVE CAUSATION IN TOXIC TORT LITIGATION*

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I. INTRODUCTION

Tort litigation in the past decade has exploded to some extent, as victims allegedly injured by exposure to toxic substances such as asbestos and hazardous wastes have sought compensation for their injuries through the courts. This dramatic expansion of "toxic tort" litigation has been facilitated by important changes in tort liability rules recently adopted by courts. The changes, which include such doctrines as joint and several liability and strict products liability, have favored plaintiffs. These liability rules have now become established legal doctrine, and make it much easier for plaintiffs to recover compensation from defendants whose products or wastes demonstrably caused a plaintiff's injury.[1]

Much less settled are the legal rules by which plaintiffs must demonstrate that defendants caused their injuries. Causation often is not a serious problem in traditional tort litigation such as accident cases, since the injury is usually traumatic in nature and occurs contemporaneously with the accident. In toxic tort litigation, however, the injuries take the form of latent diseases such as cancer, which can take years or even decades to manifest. By the time the latent disease finally appears, much of the evidence on the defendant's actions and the victim's exposure may no longer be available.

A further problem plaintiffs encounter trying to prove causation is that most of the injuries caused by exposure to toxic substances, when they do appear, are non-specific in nature and not obviously attributable

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[1]For example, under strict liability, plaintiffs can recover damages from defendants whose actions have been reasonable and non-negligent, but have nevertheless caused the plaintiff's injuries.

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to a particular cause.[2] For these reasons, plaintiffs in toxic tort suits often face a formidable challenge in meeting their burden of proving by a preponderance of the evidence that their injuries were caused by toxic substances produced or discharged by a particular defendant.

Given the difficulties of proving causation in most toxic tort suits, plaintiffs recently have begun to develop and use scientifically sophisticated risk assessments as evidence in proving causation. Risk assessment involves the application of statistical methods of analysis to incomplete databases to reach conclusions about the probability and magnitude of injuries and property damage likely to arise from potentially hazardous industrial activities and products. First developed and used by federal agencies to establish regulatory standards, risk assessments now are being introduced by plaintiffs as evidence in toxic tort suits to establish the factual basis for proving causation of injury. Defendants have responded by developing and introducing their own risk assessments to demonstrate that they did not cause the plaintiffs' injuries.

These early uses of risk assessment in toxic tort actions have led to two new trends in tort liability. *First,* there is the trend in which risk assessment is used by plaintiffs to buttress claims for future injury or "increased risk." *Second,* there is the trend in which risk assessment is used by defendants to establish that other factors caused, in whole or in part, plaintiffs' injuries.

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[2]Exceptions are so-called "signature" diseases which are almost exclusively caused by a particular substance. Examples include mesothelioma caused by asbestos fibers and vaginal adenocarcinoma caused by use of the drug diethylstilbestrol ("DES") during pregnancy.

These two trends involving the use of risk assessment in toxic tort suits have the potential to alter significantly the liability of industrial firms which use or produce hazardous substances. The growing use of risk assessment to prove increased risk and alternative causation is also likely to effect further doctrinal change in tort causation and liability rules as applied by courts. These doctrinal changes in turn may further promote the use of risk assessment as evidence in toxic tort suits.

This paper will evaluate the two recent trends of using risk assessment in toxic tort litigation to prove increased risk and alternative causation. For each of the two trends, the paper will describe: (i) the applicable traditional legal doctrine; (ii) the recent case law establishing the trend; (iii) the manner in which risk assessment is used as evidence in the recent cases; (iv) the academic commentary on the trend; and finally (v) the likely future development of the trend. The paper concludes that although use of risk assessment by plaintiffs to establish increased risk and by defendants to prove alternative causation is relatively recent, it has already had a substantial effect on tort liability and will continue such impact in the future. Therefore, these uses of risk assessment in toxic tort litigation are clearly of considerable significance to alleged victims of industrial risk, to industrial firms, and to insurers who have been severely impacted already by previous changes in toxic tort liability.

II.
INCREASED RISK

A. *Traditional Legal Doctrine*

A fundamental premise of tort law is that a plaintiff must have a present injury before he or she can sue for damages. "The threat of future harm, not yet realized, is not enough."[3] However a plaintiff who has suffered a present injury is entitled to recover damages "for all harm, past, present and prospective."[4] Thus, a plaintiff is entitled to recover for the future consequences of a present injury.

The traditional legal doctrine for recovery of prospective harm has developed primarily in cases where a plaintiff has suffered a traumatic injury in, for example, an automobile accident, and seeks to recover damages both for present injury and for the risk of future complications. Two different theories have emerged from these cases for recovery of damages for possible future harm. The majority view can be described as the "all-or-none" approach. Under this approach, a plaintiff must prove that the future injury will occur with a "reasonable certainty" or a "reasonable

[3]PROSSER & KEETON ON TORTS, § 30 (5th ed. 1984).
[4]RESTATEMENT (SECOND) OF TORTS § 910.

probability."[5] Although the meaning of a reasonable probability or reasonable certainty is not often explicitly defined, most often it has been interpreted to mean a more likely than not standard.[6] Courts in a few states with a "reasonable certainty" standard, however, have applied a stricter standard and required near certainty that the future condition will occur. A plaintiff who is able to demonstrate that a future condition will occur with reasonable certainty or probability is entitled to recover full damages for the condition, as if it actually existed. A plaintiff who cannot meet the threshold of reasonable certainty or probability is denied any damages for the risk of future injury, however, and is precluded from presenting evidence of such a possibility to the jury. Thus, recovery is "all-or-none."

The second and minority view on damages for potential future injuries can be described as the "pro rata" approach. Under this approach, the plaintiff does not have to prove that the risk of future consequences is more likely than not before he or she can recover. Rather, the evidence for the increased risk is presented to the jury, which then awards damages in proportion to its estimate of the increased risk. Therefore, a plaintiff who can demonstrate that he or she is at an increased risk of 20% for a future condition would be awarded 20% of the full damages that would normally be given if the condition existed now.

Although most courts have applied the all-or-none approach,[7] the pro rata approach finds legal support in the language of the American Law Institute's Restatement of Torts.[8] The comments to the Restatement state that a plaintiff can recover for harm that "appears will be suffered in the future, if the risk of the future harm is substantial and the proof of the probable extent satisfies the rules as to certainty."[9] The principal condition imposed on the recovery of damages for prospective harm is the requirement of "reasonable certainty of proof."[10] The Restatement also suggests that a plaintiff should recover damages for a future injury in proportion to the probability of its occurrence: "When an injured person seeks to recover for harms that may result in the future, he is entitled to

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[5]According to one account, the courts in at least 32 states have adopted a "reasonable certainty" or "reasonable probability" standard. Note, *How Far Should Increased Risk Recovery Be Carried in the Context of Exposure to Hazardous Substances?,* 76 KY. L. J. 459, 462–63 (1987–88).

[6]Brachtenbach, *Future Damages in Personal Injury Actions—The Standard of Proof,* 3 GONZ. L. REV. 75, 77 (1968).

[7]*See* Note, *supra* note 5, at 462–63 (listing over 45 cases applying the reasonable certainty or probability standard for recovery of damages for prospective injuries).

[8]RESTATEMENT (SECOND) OF TORTS, § 910 comment a (1979).

[9]*Id.*

[10]*Id.*, comment b.

damages based upon the probability that harm of one sort or another will ensue and upon its probable seriousness if it should ensue."[11]

In summary, it is now well-established that plaintiffs can recover for future injuries, providing they establish two requisite elements of such a claim. The first element, on which all courts agree, is that a plaintiff can only recover for future harm if he or she has sustained a present injury. Courts, however, disagree on the second element of a future injury claim. The all-or-nothing approach requires a plaintiff to prove that the future injury is reasonably certain or probable, which is usually interpreted to mean a likelihood greater than fifty percent. The pro rata approach provides a much lower threshold for recovery, requiring only that a plaintiff show with reasonable certainty that he or she suffers from an appreciable increased risk.

B. *Recent Case Law*

1. Summary of Increased Risk Litigation

Most of the claims seeking damages for increased risk of future injury have been brought by workers who were occupationally exposed to asbestos fibers, although several cases involve toxic contaminants in groundwater, or *in utero* exposure to the drug DES. There exist many differences and inconsistencies between the approaches and holdings of different courts, which is characteristic of a rapidly evolving area of law.

This survey examined forty-three decisions of increased risk claims recovered from searches of the LEXIS and WESTLAW databases as well as from miscellaneous other sources. With respect to these cases, courts refused to allow recovery for increased risk in thirty-seven of the forty-three cases, while in six cases the courts either denied defendant's summary judgment motion on the plaintiff's increased risk claim or permitted damages to be awarded for increased risk.[12] Of the thirty-seven cases denying recovery for increased risk, only four rejected recovery because the plaintiffs had failed to establish one of the two necessary elements of a claim for future damages: (i) a present injury or (ii) a sufficient showing of increased risk.

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[11] *Id.*, § 912 comment e.

[12] Some of the cases denying recovery for increased risk are appeals court decisions overturning trial courts that had allowed recovery for increased risk. For example, in Sterling v. Velsicol Chemical Corp., 855 F.2d 1188 (6th Cir. 1988), the Sixth Circuit Court of Appeals overturned a federal Tennessee district court's decision to award damages to residents who were exposed to toxics from a chemical waste burial site for an increased risk of cancer. *Id.* at 1204–05.

a. Present Injury

All courts espouse the traditional doctrine that a plaintiff must sustain a present injury before recovering for increased risk of future harm. In the context of increased risk resulting from exposure to toxic substances, however, courts have adopted dramatically different theories of what constitutes a present injury. One important threshold difference is apparent as between those courts which require a physical manifestation of injury and those which do not. In eleven of the forty-three cases reported above, the court dismissed the plaintiff's claim for increased risk for failure to demonstrate a physical manifestation of a current injury. Overall, in fifteen cases the court either held or implied that a present physical manifestation of an injury was a necessary condition for recovery for increased risk, while in nine cases the court stated or implied that a manifest physical injury was not required. Courts did not reach this issue in eighteen other cases.

Several different theories were advanced in the nine cases that allowed recovery for increased risk without a physical manifestation of disease. For example, one court held that mere exposure to asbestos was sufficient to establish an injury to the plaintiff in the form of "inhalation of fibers and the invasion of his body by those fibers."[13] Several other courts held that an increased risk of cancer is itself a presently existing physical condition.[14] Other courts, however, have explicitly rejected the premise that an increased risk of future cancer is a present injury.[15] A third theory adopted by some courts for finding current injury when the plaintiff has exhibited no symptoms is that exposure to toxic substances results in invisible subcellular and genetic injuries. Again, several other courts have expressly stated that alleged genetic and subcellular damage from exposure to toxic substances is not sufficient to establish a present injury.[16]

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[13]Gideon v. Johns-Manville Sales Corp., 761 F.2d 1129, 1137 (5th Cir. 1985).

[14]*See, e.g.,* Herber v. Johns-Manville Corp., 785 F.2d 79, 82 (3d Cir. 1986); Ayers v. Township of Jackson, 106 N. J. 557, 525 A.2d 287, 305 (1987); Potter v. Firestone Tire & Rubber Co., No. 81723, slip op. (Cal. App. Dep't Super. Ct. 1987), *summarized in Two Families Awarded $3.9 Million; Firestone Held Strictly Liable for Disposal,* 2 TOXICS L. REP. (BNA) 862, 863 (1988).

[15]Adams v. Johns-Manville Sales Corp., 783 F.2d 589, 593 (5th Cir. 1986); Amendola v. Kansas City Southern Railway Co., 699 F. Supp. 1401, 1407 (W.D. Mo. 1988); Westrom v. Kerr-McGee Chemical Corp., No. 82C2034 (N.D. Ill. Oct. 4, 1983) (LEXIS, Genfed); Mink v. University of Chicago, 460 F. Supp. 713, 719 (N.D. Ill. 1978).

[16]*See, e.g.,* Jackson v. Johns-Manville Sales Corp., 781 F.2d 394, 412 n.22 (5th Cir. 1986) (relying on Schweitzer v. Consolidated Rail Corp., 758 F.2d 936 (3d Cir.), *cert. denied,* 474 U.S. 864 (1985)); Laswell v. Brown, 683 F.2d 261, 269 (8th Cir. 1982); Burns v. Jaquays Mining Corp., 156 Ariz. 375, 752 P.2d 28, 31 (Ct. App. 1987), *reviewed and dismissed,* 162 Ariz. 186, 781 P.2d 1373 (1989).

There is also considerable variation in the approaches of courts that do require a physical manifestation of disease before allowing recovery for increased risk. Some courts treat common, nominal symptoms such as dizziness, general malaise, headaches, and nausea as sufficient to establish a present injury for purposes of recovery for increased risk.[17] Other courts recognize a present injury for precursor conditions which are clinically detectable but may not cause any noticeable symptoms. Examples include pleural thickening in the lungs of those plaintiffs exposed to asbestos,[18] and an anatomical deformity known as a "vaginal hood" in females exposed to DES *in utero*.[19]

Still other courts require the present injury to be part of the same disease process as the future cancer for which the plaintiff is at an increased risk of developing. For example, in one case the court held that asbestoses and asbestos-related cancer are separate disease processes, even though they both result from asbestos exposure. Thus, asbestoses is not a present injury for purposes of recovering for increased risk of asbestos-related cancer.[20]

Finally, some courts require a present bodily injury of significant magnitude, although it does not have to be part of the same disease process as the potential future cancer. For example, one court held that subcellular damage or "psychosomatic injuries" such as headaches, depression, and insomnia are not an adequate physical impairment to support a claim for an increased risk of asbestos-related cancer.[21] Asbestoses, however, would be a sufficient physical injury for such a claim.[22]

In summary, the courts have adopted many different theories of what constitutes a present injury for purposes of recovering damages for an increased risk of cancer. Some of the variations require a physical injury with discernable symptoms, while other approaches do not. Therefore, parties in toxic tort suits often will be uncertain which theory will be applied by the court hearing their case. A plaintiff who can objectively demonstrate a present condition or symptom that is part of the same disease process as future cancer will be most likely to satisfy the present injury element of an increased risk claim.

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[17]*E.g.*, Hagerty v. L & L Marine Services, Inc., 788 F.2d 315, 317 (5th Cir. 1986); Villari v. Terminex Int'l, Inc., 692 F. Supp. 568, 573 (E.D. Pa. 1988).

[18]*Herber*, 785 F.2d 79, 88, 89 (3d Cir. 1986); Mauro v. Owens-Corning Fiberglass Corp., 225 N.J. Super. 196, 542 A.2d 16, 24 (N.J. Super. Ct. App. Div. 1988), *aff'd*, 561 A.2d 257 (N.J. Sup. Ct. 1989). *But see* Howell v. Celotex Corp., 904 F.2d 3 (3rd Cir. 1990) (holding that pleural thickening was not a present injury).

[19]McAdams v. Eli Lilly & Co., 638 F. Supp. 1173, 1178 (N.D. Ill. 1986).

[20]LaVelle v. Owens-Corning Fiberglass Corp., 30 Ohio Misc. 2d 11, 507 N.E.2d 476, 479 (C.P. Cayahoga County 1987).

[21]*Burns*, 156 Ariz. 375, 752 P.2d at 31–32.

[22]*Id.*

b. Sufficient Showing of Increased Risk

The second element of a claim for enhanced risk of a future injury is a sufficient showing of increased risk. There are often two requirements for a sufficient showing of increased risk. First, the plaintiff must be able to quantify its increased risk of developing future cancer. Second, the plaintiff often must prove that the magnitude of the increased risk is above a certain threshold. A plaintiff who is not prepared to establish both of these requirements with acceptable evidence will usually be precluded by the court from presenting a claim for increased risk to the jury.

In ten of the earlier cases, the court dismissed the increased risk claim of plaintiffs who were unable to quantify their increased risk of developing future cancer. Altogether, in twenty-four cases the court held or implied that plaintiffs must quantify their increased risk. Some courts have dismissed an unquantified increased risk of cancer even when plaintiffs' experts have testified that it is scientifically impossible to quantify the enhanced risk of the plaintiffs.[23]

A few courts have not required plaintiffs to quantify their increased risk of cancer. This more permissive approach significantly increases the feasibility of an increased risk claim, as evidenced by the fact that plaintiffs were allowed to recover for increased risk in all three cases in which the court did not require quantification.

The second requirement for showing sufficient increased risk is that the magnitude of the increased risk must usually be greater than a specific threshold. Most courts have adopted the "all-or-none" approach and require that the increased likelihood of future cancer be "reasonably certain" or "reasonably probable." Twenty-five of the forty-three cases studied required the increased risk to be "reasonably certain" or "reasonably probable." In fourteen of these cases, the court defined reasonable certainty to mean more likely than not to occur. In the other eleven cases requiring the future disease to be "reasonably certain," the court did not address whether this meant more likely than not. At least one case, however, suggested that the threshold would be significantly higher than a fifty percent probability.

Consistent with the traditional doctrine, courts apply the reasonable certainty standard as part of the all-or-nothing approach. Thus, "if plaintiffs could show that they were more likely than not to suffer cancer or other future illness, full recovery would be allowed for all plaintiffs . . ."[24] In at least one case, the plaintiff explicitly argued for the pro rata approach in which recovery for the likelihood of future cancer "would be

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[23]*See, e.g., Ayers,* 106 N.J. 557, 525 A.2d at 303; Elam v. Alcolac, Inc., 765 S.W.2d 42 (Mo. Ct. App. 1988), *cert. denied,* 110 S. Ct. 69 (1989) ("there is not enough information in science to do that" [quantify the risk of disease]); *Elam,* 765 S.W.2d at 162.

[24]Anderson v. W.R. Grace & Co., 628 F. Supp. 1219, 1232 (D. Mass. 1986).

proportionally reduced to reflect the probability that he will not contract cancer."[25] The court, however, rejected that plaintiff's argument "as fundamentally at odds with New Jersey's approach to compensable injury."[26] Nevertheless, at least two courts have rejected the more likely than not standard.[27]

In summary, most courts impose a heavy burden on plaintiffs to make a sufficient showing of increased risk. Unless plaintiffs are prepared to establish that they have a quantified increased risk of at least fifty percent of developing cancer in the future, most courts will dismiss their increased risk claims. Some courts may even require a higher threshold showing, such as proving that a future disease is a near certainty. A few courts have adopted a less burdensome approach, allowing recovery for an unquantified increased risk or for increased risks of less than fifty percent.

C. *Arguments Underlying Court Decisions*

a. Statutes of Limitations and Res Judicata

The effect of statutes of limitations and the legal rule against claim-splitting known as res judicata were two important factors to courts considering increased risk claims. Statutes of limitations bar claims that are not filed within a specified time limit. For tort injuries, most states require a claim to be filed within one, two or three years of the time at which the cause of action accrues.

In traditional tort law, a plaintiff's cause of action accrues at the time of the injury-causing event. While accrual at the time of the event is usually straightforward for traumatic injuries, it prevents recovery for latent injuries caused by toxic substances, since those injuries usually do not appear until many years after exposure and long after the statute of limitations has expired. Therefore, most states have adopted a "discovery rule," in which a cause of action for injuries from toxic substances does not accrue until the plaintiff discovers or should have discovered the injury.[28] The 1986 Superfund Amendments now require all states to apply a discovery rule for toxic tort claims.[29]

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[25]*Herber,* 785 F.2d at 82.

[26]*Id.*

[27]Potter v. Firestone Tire & Rubber Co., *supra* note 14; Valori v. Johns-Manville Sales Corp., No. 6074 (D.N.J. Dec. 11, 1985) (WESTLAW, Allfed library).

[28]*See* Green, *The Paradox of Statutes of Limitations in Toxic Substances Litigation,* 76 CALIF. L. REV. 965, 979 n.65 (1988) (at least 43 states have adopted some version of the discovery rule).

[29]42 U.S.C. § 9658(a) (4) (A) (1988).

Even with the discovery rule, a plaintiff will still be barred from recovering compensation for cancer resulting from toxic exposure if the cause of action accrues at the time of discovery of an earlier disease or symptom. A few courts have sought to reduce the necessity of suing for increased risk of future diseases at the time the first symptoms become manifest by allowing for different times of accrual for distinct diseases resulting from the same toxic exposure. Furthermore, since statutes of limitations are created by state legislatures, courts are bound to follow legislation that mandates a different approach.[30] Therefore, uncertainty in many states about whether recovery for late onset cancer will be barred by the statute of limitations is likely to induce plaintiffs to bring actions for increased risk of cancer at the time the first symptoms of toxic exposure appear.

A somewhat similar but separate obstacle to recovery for latent cancer is the res judicata doctrine against claim-splitting. The rule against claim-splitting permits only one cause of action per "transaction."[31] The Restatement of Judgments states that "where one act causes a number of harms to . . . the same person, there is still but one transaction; a judgment based on the act usually prevents the person from maintaining another action for any of the harms not sued for in the first action."[32] This rule requires plaintiffs exposed to toxic substances to combine all claims for injury in a single cause of action.

The combination of the statute of limitations and the rule against claim-splitting can put a victim of toxic exposure in a "catch 22" predicament, as illustrated by *Hagerty v. L & L Marine Services, Inc.*.[33] In that case, the plaintiff was accidentally doused with toxic chemicals and experienced some immediate symptoms such as dizziness and leg cramps. He sued shortly after the accident. The court concluded that the plaintiff could not have postponed his suit until future cancer developed because his current symptoms were sufficient to start the statute of limitations running. By bringing a suit now, however, he would be precluded from a second suit if and when the cancer developed. Therefore, regardless of whether the plaintiff brought suit immediately, or waited to see if cancer developed, he would be denied a remedy for his damages for cancer. The plaintiff's only hope of recovering damages for cancer under these doctrines would be to commence an action for increased risk of cancer when the first symptoms of exposure appeared. The courts do have flexibility to make exceptions to the rule against claim-splitting, however. The rule does not apply when the "parties have agreed in terms or in effect that a

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[30]*See* PROSSER & KEETON ON TORTS § 30 (5th ed. 1984).

[31]RESTATEMENT (SECOND) OF JUDGMENTS § 24 (1982).

[32]*Id.*, comment c.

[33]*Hagerty,* 788 F.2d 315 (5th Cir. 1986).

plaintiff may split his claim, or the defendant has acquiesced therein," or when the "court in the first action has expressly reserved the plaintiff's right to maintain the second action."[34]

b. Other Arguments Cited by Courts

The major concerns of the courts are that increased risk claims are too speculative, are contrary to the court's own perceived role of providing certainty and resolution of social conflicts, and will greatly increase the case load of courts. In the words of one court, "to permit recovery for the increased risk of cancer would be to allow speculation."[35] The purpose of tort law is to compensate for injury, and thus it is the function of courts "to ensure that an award of damages is not made for an injury that probably will not be suffered."[36] Awarding damages for injuries that may never result would undermine the goal "that jury verdicts speak the truth."[37]

Many courts have also expressed concern that awarding damages for increased risk would over-compensate some plaintiffs while under-compensating others. An individual plaintiff who is awarded damages for increased risk but never develops the future disease will get a "windfall."[38] Under the pro rata approach, an individual who does develop cancer will be under-compensated by the previous award for increased risk that precludes additional remedies. Under the all-or-nothing approach, which awards full compensation for all plaintiffs if the increased risk is greater than fifty percent, the defendant would significantly over-compensate an injured class with an increased risk of over fifty percent.[39]

Finally, courts have expressed concern about the costs and consequences for defendant companies and their insurers if widespread recovery for increased risk of future disease is permitted. According to one court, "[t]o hold a defendant manufacturer responsible for the future occurrence of cancer would entail treating the manufacturer as an absolute insurer of its product."[40] Another court worried that awarding damages for increased risk would "lead to escalation of insurance rates."[41]

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[34]RESTATEMENT (SECOND) OF JUDGMENTS § 26 (1) (a), (b).
[35]*Lavelle,* 30 Ohio Misc. 2d 11, 507 N.E.2d at 478–79.
[36]*Herber,* 785 F.2d at 82.
[37]Eagle-Picher Industries, Inc. v. Cox, 481 So. 2d 517, 523 (Fla. Dist. Ct. App. 1985).
[38]*Anderson,* 628 F. Supp. at 1232 (citation omitted).
[39]Wilson v. Johns-Manville Sales Corp., 684 F.2d 111, 120 n.45 (D.C. Cir. 1982).
[40]*Lavelle,* 30 Ohio Misc. 2d 11, 507 N.E. 2d at 479.
[41]*Eagle-Picher Industries, Inc., supra* note 37.

D. *Evidence Used to Support Increased Risk Claims*

a. Present Physical Injury

Most courts require proof of a present physical injury as an element of an increased risk claim. When the plaintiff exhibits clinically diagnosable symptoms, it is relatively straightforward to satisfy this requirement by having a physician testify on the existence of the symptoms. The more difficult cases are where the plaintiff suffers no diagnosable symptoms, but attempts to establish a present injury in the form of alleged subcellular or genetic damage.

Plaintiffs claiming to suffer from subcellular or genetic damage have attempted to establish this fact by expert testimony that exposure to toxic substances results in such injury. None of the reported cases indicate, however, that a plaintiff was prepared to offer scientific evidence of actual molecular, genetic or cellular changes in an individual toxic exposure victim. As discussed earlier, a few courts have recognized "invisible" subcellular and genetic damage as a present injury, but most have not. An ability to objectively detect subcellular changes in individual plaintiffs would likely increase the acceptance of these types of changes as an adequate present injury to establish an increased risk claim.

One method for detecting genetic and subcellular changes in a plaintiff is chromosome screening. Easily observed chromosome aberrations and exchanges between chromosome arms (sister chromatic exchanges) may be indicative of exposure to toxic chemicals and increased risk of future cancer.[42] However, the health significance of these kinds of chromosomal changes is still questionable, significantly reducing the usefulness of this type of evidence.[43] Another problem is that chromosome aberrations are not specific for a particular substance, undermining any certainty about which agent caused the changes.

A more promising type of detectable subcellular change for measuring exposure to toxic substances is DNA adducts. DNA adducts are molecular or structural changes that occur when a foreign substance binds to DNA. These adducts "are widely suspected as a primary instigator of many cancers."[44] "The presence of specific DNA adducts in cells provides incontrovertible evidence that the individual from whom the cells came has been exposed to that chemical."[45] Several recently developed sophisticated analytical techniques now make possible "[t]he reliable and sensi-

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[42]Maugh, *Biological Markers for Chemical Exposure,* 215 SCIENCE 643 (1982).

[43]*Id.*

[44]Raloff, *Chemically Fingerprinting DNA Damage,* 135 SCIENCE NEWS 199 (1989). *See also* Ellinger, *DNA Diagnostic Technology: Probing the Problem of Causation in Toxic Torts,* 3 HARV. J. LAW & TECH. 31 (1990).

[45]Maugh, *Tracking Exposure to Toxic Substances,* 226 SCIENCE 1183 (1984).

tive detection of characteristic molecular markers for human exposure to chemical carcinogens."[46] Although more information is needed before DNA adducts can be used to accurately quantify the exposure or risks from toxic substances, they can (or soon will be able to) be used to provide reliable evidence that a person has been exposed to specific toxins.[47]

b. Proof of Increased Risk

In the past, plaintiffs suffering a traumatic injury were frequently able to establish causation or an increased risk of future injury simply by having a medical doctor testify that the disputed fact exists to a "reasonable medical certainty." This deference to medical experts resulted in numerous contradictory and aberrational decisions, because there was no consistent understanding of what the magic words "reasonable certainty" meant.[48] As one pair of authors summarized the problem with the traditional standard, "[i]f certainty is needed, witnesses can be found who will profess it."[49] A few courts that have not required increased risk to be quantified have accepted medical opinions as sufficient evidence in toxic tort enhanced risk cases. As discussed previously, however, most courts have required increased risk to be quantified. Conclusory statements by expert witnesses that attempt to quantify increased risk are not accepted by many courts unless supported by specific evidence establishing the risk quantification.[50] Therefore, plaintiffs now usually have to support their claims of increased risk with more sophisticated risk assessment evidence.

The first and often most difficult step in calculating increased risk is to measure the plaintiffs' exposure to the defendant's toxic substances. *Sterling v. Velsicol*[51] is an example of a case in which the plaintiffs used risk assessment techniques, attempting to estimate exposure and risk. Plaintiffs' experts used two different approaches for estimating exposure. The first approach relied on computer modeling to estimate groundwater levels of pollutants from defendant's facility. The estimated average an-

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[46]Shuker, *Reliable Exposure Assessment,* 329 NATURE 582 (1987).

[47]Raloff, *supra* note 44, at 199.

[48]*See* Black, *A Unified Theory of Scientific Evidence,* 56 FORDHAM L. REV. 595, 667–69 (1988).

[49]Black & Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation,* 52 FORDHAM L. REV. 732, 744 (1984).

[50]*See, e.g.,* Jackson v. Crane Packing Co., No. A-4459-87TS, slip. op. (N.J. Super. Ct. App. Div. 1989), *cert. den.,* 569 A.2d 1331, 117 N.J. 628 (1989), *summarized in* 4 TOXICS L. REP. (BNA) 35 (1989). In this case, a physician testified that the plaintiff "has maybe a 50 percent risk of getting cancer, a round ballpark figure." This evidence was an insufficient quantification of increased risk to allow a claim for recovery for increased risk to be submitted to the jury.

[51]Sterling v. Velsicol Chemical Corp., 647 F. Supp. 303 (W.D. Tenn. 1986), *rev'd in part,* 855 F.2d 1188 (6th Cir. 1988).

nual concentrations of toxic chemicals in the plaintiffs' drinking water were then used to calculate exposure via water consumption, bathing, and inhalation.[52] The second method for estimating exposure was to detail the ailments such as dizziness and vertigo that the plaintiffs experienced. Then, the dose was estimated by determining what dose it took to produce similar symptoms in workers exposed to known exposure levels of the same chemicals.[53] The trial court accepted the estimates from these two approaches to establish the range of likely exposure.[54]

The use of markers for toxic exposure, such as the DNA adducts discussed earlier, offer a potential means for measuring exposure in the future. The recent development of techniques to reliably detect DNA adducts caused by toxic chemicals may eventually make it "possible to measure with certainty the exposure to these chemicals."[55] Evidence indicates that the bodily concentrations of adducts are proportional to recent chemical exposure.[56] There remain unresolved questions such as whether these adducts persist for long periods of time which must be answered before DNA adducts can be used to provide quantitative estimates of exposure, however.[57]

Once plaintiffs have estimated exposure, they must then try to calculate their increased risk. The two major sources of data for calculating increased risk from exposure estimates are epidemiological studies and animal bioassays. Epidemiological data is best suited for determining increased risk, rather than individual causation, because it describes the probability that a substance will cause a specific disease rather than whether or not the substance actually caused the disease in a particular individual.[58] One disadvantage of epidemiological evidence is that relatively long periods of human exposure are required to develop data; therefore, there will not be any data available to early plaintiffs. Large sample sizes and high exposure levels are also required to obtain statistically significant results. Also, epidemiological data is most useful for cases in which an individual is exposed primarily to a single toxic sub-

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[52]*Sterling,* 647 F. Supp. at 469–70.

[53]*Id.* at 475–76.

[54]The trial court's acceptance of increased risk damages was overturned by the Sixth Circuit Court of Appeals because the estimated increased risk to plaintiffs was twenty-five to thirty percent, which does not constitute a reasonable degree of medical certainty. *Sterling,* 855 F.2d at 1205. The court of appeals did not, however, address the adequacy of the plaintiffs' exposure and risk estimates accepted by the trial court.

[55]Maugh, *supra* note 45.

[56]*Id.* at 1184.

[57]*Id.*

[58]Dore, *A Commentary On the Use of Epidemiological Evidence in Demonstrating Cause-In-Fact,* 7 HARV. ENVTL. L. REV. 429, 433 (1983).

stance such as asbestos, rather than cases involving a complex mixture of chemicals for which no data is usually available.

Courts have traditionally been reluctant to accept epidemiological data because it is based on statistics and probabilities.[59] In recent increased risk cases, however, courts have generally favored epidemiological evidence. Nevertheless, judges are not unanimous on the value of epidemiological data. A panel of the Fifth Circuit recently concluded that "[t]he proffer of abstract statistics and generalizations do not suffice to demonstrate that" a particular plaintiff is at a sufficiently increased risk to warrant recovery.[60]

The courts are much more divided on the use of animal studies to quantify increased risk. Several courts have rejected the use of animal data to demonstrate increased risk in humans. For example, one court held that animal data may be used to estimate risks for purposes of setting regulatory standards. Animal test data and *in vitro* carcinogenicity tests could not be introduced as evidence to demonstrate increased human risk, however, because "such an extrapolation does not reach the requisite level of acceptance within the scientific community to justify legal reliance."[61]

Conversely, several courts have accepted animal data evidence to establish increased risk. For example, animal studies were admitted as evidence of increased risk in *Villari v. Terminex International, Inc.*[62] on the basis of the court's finding "that a substantial portion of the scientific community relies on animal studies of this type in assessing health risks to humans."[63] Therefore, admissibility of animal studies data to prove increased risk varies from court to court.

One problem with the way the courts deal with risk assessment evidence in increased risk cases is the inadequate emphasis courts give to the uncertainty inherent in risk estimates. Estimates of risk can vary by many orders of magnitude, depending on the data used and the assumptions adopted. Yet plaintiffs frequently offer, and courts often seem to accept, apparently very precise claims of, for example, forty-three percent increased risk that involve no expression of uncertainty.[64] Given that under the all-or-nothing approach plaintiffs are entitled to full recovery if they establish an increased risk of fifty-one percent but receive no damages if they have an increased risk of forty-nine percent, it does not seem appropriate to give so little attention to the uncertainty and assumptions underlying risk estimates.

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[59]*See, e.g.*, Brennan, *Causal Chains and Statistical Links: The Role of Scientific Uncertainty in Hazardous-Substance Litigation*, 73 CORNELL L. REV. 469 (1988).

[60]*Adams*, 783 F.2d at 592.

[61]Arnett v. Dow Chemical Corp., No. 729586, slip op. (Cal. App. Dep't Super. Ct. Mar. 21, 1983).

[62]692 F. Supp. 568 (E.D.Pa. 1988).

[63]*Id.* at 571.

[64]*E.g., Valori*, No. 6074 (D.N.J. Dec. 11, 1985) (WESTLAW, Allfed library).

The courts also usually do not distinguish between an individual's total risk of developing cancer and the increased risk caused by defendant's activity. In at least one case, the fact that the plaintiff was a heavy smoker helped him to reach the fifty percent threshold for recovery for increased risk. The court seemed only to care whether the plaintiff's total risk of lung cancer was greater than fifty percent, and not how much of this total risk was due to asbestos and how much to the plaintiff's heavy smoking.[65] Under this approach, if two plaintiffs were exposed to asbestos and one smoked and the other did not, the plaintiff who smoked might be able to get to the jury with his claim for increased risk, while the non-smoking plaintiff could not.

E. *Other Ways of Introducing Evidence of Increased Risk*

a. Fear of Cancer

As discussed in the previous sections, most courts have not permitted plaintiffs to recover damages for an increased risk of cancer. Many courts, however, allow plaintiffs to introduce this same evidence of increased risk for the purpose of supporting a claim for fear of developing cancer.[66] The usual requirements for a claim for fear of contracting cancer or "cancerphobia"[67] include a "physical harm manifested by objective symptomatology" and proof "that a reasonable person would have suffered emotional distress" under similar circumstances.[68] Evidence on the increased risk of future cancer is usually admissible to show the reasonableness of the plaintiff's emotional distress.

While the requirements of a present physical injury and a reasonable risk of future cancer are similar to the elements of an increased risk claim, the courts are much more lenient in interpreting these requirements for a fear of cancer claim than for an increased risk claim. The first require-

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[65]*Gideon,* 761 F.2d at 1138.

[66]*E.g., Herber,* 785 F.2d at 85; *McAdams,* 638 F. Supp. at 1178; Wetherill v. University of Chicago, 565 F. Supp. 1553, 1561 (N.D. Ill. 1983); Sorenson v. Raymark Industries, Inc., 756 P.2d 740, 742 (Wash. Ct. App. 1988); *Lavelle,* 30 Ohio Misc. 2d 11, 507 N.E.2d at 481; Devlin v. Johns-Manville Corp., 202 N.J. Super. 556, 495 A.2d 495, 499; *Eagle-Picher Industries, Inc.,* 481 So. 2d at 529 (Fla. Ct. App. 1985).

[67]A few courts have distinguished fear of cancer from cancerphobia. For example, according to one court: "The former, which does not require an expert witness to prove its existence, is an idiosyncratic response to the knowledge that a person is at risk of contracting the disease, while the latter is a recognized psychiatric illness, which must be testified to by an expert witness. A phobia is an exaggerated, persistent, often irrational fear." *Eagle-Picher Industries, Inc.,* 481 So. 2d at 526 n.13. *See also Devlin,* 202 N.J. Super. 556, 495 A.2d at 499. However, most courts and commentators treat fear of cancer and cancerphobia as synonymous.

[68]These requirements for recovery of fear of contracting cancer were first articulated in Payton v. Abbott Labs, 386 Mass. 540, 437 N.E.2d 171, 181 (1982).

ment for a claim for fear of cancer is a physical injury that either caused, or was caused by, the plaintiff's emotional distress.[69] Many courts have insisted on a physical manifestation of bodily injury to satisfy this requirement.[70] Other courts, however, have not required a physical manifestation of injury as a prerequisite for recovery for fear of cancer.[71]

The second element of a claim for fear of cancer, the reasonableness of the plaintiff's emotional anxiety, is usually established by introducing evidence of increased risk of cancer. Unlike claims for recovery for increased risk, where the judge usually does not allow evidence of enhanced risk to be presented to the jury unless it is above a certain threshold (usually fifty percent), courts usually allow evidence of any increase in risk to be presented to the jury to support a fear of cancer claim. In fear of cancer claims, "the central focus is not on the underlying odds that the future disease will in fact materialize."[72] The plaintiff's increased risk need only be reasonable, and "[r]easonable in this context is not equivalent to probability or certainty, but is for a fact-finder to determine."[73]

While the courts seem to be unanimous that a plaintiff's increased risk of cancer does not need to be "reasonably certain" in order to be introduced to demonstrate the reasonableness of the plaintiff's fear of cancer, there is some disagreement about whether the increased risk needs to be quantified. Some courts require the plaintiff's increased risk to be quantified. Other courts, however, have either not required or have not permitted increased risks to be quantified.

Claims for emotional distress from fear of cancer will likely increase the liability of defendant companies and their insurers in two ways. First, by allowing otherwise inadmissible evidence of increased risk to be presented to the jury, fear of cancer claims may prejudice juries and cause them to unconsciously or surreptitiously include compensation for increased risk of cancer in the plaintiff's damages. The second and most obvious way that claims for fear of cancer can increase the liability of defendants is by the substantial damages juries are awarding for the fear of contracting cancer. The size of damage awards in cancerphobia cases has been increasing in recent years from a few thousand dollars per plain-

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[69]*Id.*

[70]*E.g., Adams,* 783 F.2d at 593; Plummer v. Abbott Laboratories, 568 F. Supp. 920, 925 (D.R.I. 1983), *referencing* D'Ambra v. United States, 114 R.I. 643, 338 A. 2d 524, 531 (1975); *Burns,* 156 Ariz. 375, 752 P.2d at 31; DeStories v. City of Phoenix, 154 Ariz. 604, 744 P.2d 705, 709 (Ct. App. 1987).

[71]*See* Gale and Goyer, *Recovery for Cancerphobia and Increased Risk of Cancer,* 15 CUMB. L. REV. 723, 735 n.77 (1985).

[72]*Sterling,* 855 F.2d at 1206.

[73]*Lavelle,* 30 Ohio Misc. 11, 507 N.E.2d at 481.

tiff to up to $100,000 per plaintiff.[74] In one recent case, the jury awarded the plaintiff $1.2 million for "past and future mental pain and suffering" and "emotional distress."[75] Furthermore, given the relatively low burden to establish a fear of cancer, the number of plaintiffs who recover for fear of cancer may be very large. Finally, while a plaintiff who recovers for increased risk of cancer would be precluded from further compensation if and when cancer develops, plaintiffs who recover damages for fear of cancer may be able to bring a second suit for additional damages if and when cancer appears.

b. Costs of Future Medical Surveillance

The courts have also been relatively lenient in allowing plaintiffs who are at an increased risk of cancer to recover for costs of future medical surveillance. These costs will generally be awarded when:

> proofs demonstrate, through reliable expert testimony predicted upon the significance and extent of exposure to chemicals, the toxicity of the chemicals, the seriousness of the disease for which individuals are at risk, the relative increase in the chance of onset of disease in those exposed, and the value of early diagnosis, that such surveillance to monitor the effect of exposure to toxic chemicals is reasonable and necessary.[76]

Plaintiffs, however, are usually not required to quantify their increased risk or to have sustained an existing injury.[77]

Claims for costs of future medical surveillance are generally less controversial than claims for increased risk or fear of future cancer. The amount of damages involved is usually considerably less. Further, early detection of latent cancer by medical surveillance can reduce plaintiffs' injuries and defendants' liabilities.

c. Immunotoxicity Claims

A recent trend in toxic tort litigation is for plaintiffs to include in their suits a claim for injury to their immune system as a result of exposure to toxic substances. These claims for "chemically induced immune dysfunction" are similar to increased risk claims because the plaintiffs

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[74]*See* Willmore, *'Cancerphobia': A Toxic Tort Time Bomb,* Wall St. J., Sept. 14, 1988, at 34, col. 4 (eastern ed.).

[75]Wisner v. Illinois Central Gulf Railroad, 537 So.2d 740, 749–50 (La. Ct. App. 1989), *cert. denied,* 540 So.2d 342 (La. 1989).

[76]*Ayers,* 106 N.J. 557, 525 A.2d at 312.

[77]*Id.,* 106 N.J. 557, 525 A.2d at 313; Burns, 156 Ariz. 375, 752 P.2d at 33. *But see* Villari v. Terminex International, Inc., 663 F. Supp. 727, 735 (E.D. Pa. 1987) (requiring present physical injury for recovery for costs of future medical surveillance).

claim that the damage to their immune system increases their future risk of contracting cancer and other diseases. Evidence for immune system damage usually involves a battery of blood tests showing abnormal white blood cell count or abnormal immunological response. The theory of immune toxicity, however, is very controversial and has been rejected by many scientists and professional medical societies.[78]

F. *Academic Commentary*

Academic commentary has favored recovery for increased risk of cancer by exposure to toxic substances.[79] Commentators favor eliminating both of the elements courts have generally required for an increased risk claim—a present physical injury and a threshold showing of increased risk. Unlike traditional risk-creating activity, in which the risk and the activity are contemporaneous, the risks from exposure to toxic substances continue after the risk-creating activity ceases. The increased risk from latent disease is not mere statistical probability of harm but rather some permanent biological changes in the exposure victim. Therefore, these commentators argue that "increased risk" should itself be recognized as a present physical injury.[80]

Academic commentators are almost unanimous in supporting the pro rata approach over the all-or-none approach adopted by a majority of courts.[81] The all-or-none approach is criticized for over-compensating or under-compensating a class of victims. If the plaintiffs' increased risk is less than fifty percent, they recover nothing, even though some incidence

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[78]*See* Marshall, *Immune System Theories on Trial,* 234 SCIENCE 1490, 1492 (1986); Rothman & Maskin, *Defending Immunotoxicity Claims,* 3 TOXICS L. REP. (BNA) 1219, 1221 (1989).

[79]At least one court has recognized "that the overwhelming weight of the scholarship on this issue favors a right of recovery for tortious conduct that causes a significantly enhanced risk of injury." *Ayers,* 106 N.J. 557, 525 A.2d at 307 (1987).

[80]*See, e.g.,* Kanner, *Emerging Concepts of Latent Personal Injuries in Toxic Tort Litigation,* 18 RUTGERS L.J. 343, 359 (1987); Note, *An Analysis of the Enhanced Risk Cause of Action (Or How I Learned to Stop Worrying and Love Toxic Waste),* 33 VILL. L. REV. 437, 456 (1988); Note, *Increased Risk of Cancer as an Actionable Injury,* 18 GA. L. REV. 563, 589 (1984); Comment, *Increased Risk of Disease From Hazardous Waste: A Proposal for Judicial Relief,* 60 WASH. L. REV. 635, 640 (1985). *But see* Note, *How Far Should Increased Risk Recovery Be Carried in the Context of Exposure to Hazardous Substances?,* 76 KY. L.J. 459, 474–77 (1987–88) [hereinafter *How Far Should Increased Risk Recovery Be Carried?*] (supporting the requirement of a present physical injury other than the increased risk itself).

[81]Gale & Goyer, *Recovery for Cancerphobia and Increased Risk of Cancer,* 15 CUMB. L. REV. 723, 742 (1985); Robinson, *Probabilistic Causation and Compensation for Tortious Risk,* 14 J. LEGAL STUD. 779 (1985); Comment, *Increased Risk of Disease From Hazardous Wastes: A Proposal for Judicial Relief, supra* note 80, at 649–50; Note, *How Far Should Increased Risk Recovery Be Carried?, supra* note 80, at 470; Note, *Increased Risk of Cancer as an Actionable Injury, supra* note 80, at 589.

of cancer may be almost certain if the class is large. On the other hand, if the plaintiffs' risk is greater than fifty percent, all plaintiffs receive full compensation, and the class as a whole is over-compensated.[82] Furthermore, it is arbitrary to "equate a forty-nine percent chance of disease formation with speculation, yet treat a fifty-one percent probability as reasonably certain."[83] Another problem commentators see with the all-or-none approach is that plaintiffs will rarely be able to establish the fifty percent threshold for increased risk needed to recover.[84] Of course, this is probably one reason why most courts have adopted the all-or-none approach in the first place.

The academic commentators cite some additional arguments for favoring increased risk claims. First, the evidence about a defendant's allegedly tortious activity will have "decayed" by the time latent cancer develops twenty or more years into the future.[85] The evidence will be much "fresher" and intact if an increased risk claim is brought soon after the activity occurs. Second, since modern corporations are often driven by short-term economic losses and gains, "the deterrent value of legal penalties for managerial error depends heavily on the proximity of the penalties to the actions for which they are assessed."[86] Since an action for increased risk of cancer can be brought much earlier compared to waiting for cancer to appear and then suing, awarding damages for increased risk should enhance deterrence. Third, defendants may be insolvent by the time cancer appears, in which case plaintiffs who develop cancer will be denied compensation altogether if they were denied recovery for increased risk.[87]

Thus, academic commentators strongly favor a pro rata approach in which plaintiffs are awarded damages in proportion to their increased risk of cancer. This solution would be "actuarily fair" in that the total liability of defendants would be equal to the total expected loss attributable to their conduct. The pro rata approach would also significantly increase the number of plaintiffs who would be able to recover for increased risk. It remains to be seen whether the strong academic support for the pro rata approach will influence its judicial acceptance.

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[82]Note, *Increased Risk of Cancer as an Actionable Injury, supra* note 80, at 568.

[83]*See, e.g.,* Comment, *Increased Risk of Disease From Hazardous Wastes: A Proposal for Judicial Relief, supra* note 80, at 639 (citation omitted).

[84]*See, e.g., Id.* at 639; Note, *How Far Should Increased Risk Recovery Be Carried?, supra* note 80, at 468–69.

[85]*See, e.g.,* Robinson, *supra* note 81, at 783.

[86]*Id.* at 785.

[87]Note, *Increased Risk of Cancer as an Actionable Injury, supra* note 80, at 576.

F. *Future Developments*

Most courts have erected a very high threshold for recovery of damages for increased risk of future disease caused by exposure to toxic substances. The most burdensome requirement for recovery of damages for increased risk is that plaintiffs usually must demonstrate a greater than fifty percent probability of future cancer. Most exposure to toxic chemicals, however, results in cancer risks that are far lower than fifty percent. These risks are orders of magnitude less than what is needed to establish a claim for increased risk in most courts.

The increased use of quantitative risk assessment and the development of reliable markers for exposure such as DNA adducts should assist plaintiffs in proving a present injury and quantifying the increased risk. These developments are likely to increase plaintiffs' success somewhat in recovering for increased risk, to the extent that many of the increased risk cases in the past were decided against plaintiffs because of their failure to quantify risk and prove a present injury. Furthermore, the increased ability to accurately and reliably detect present subcellular injuries and to quantify exposure and risk may ease judicial concern that increased risk claims are too speculative. More courts may be willing, therefore, to allow claims for increased risk of less than fifty percent.

New risk information is likely to encourage more claims for increased risk in additional ways. As discussed earlier, plaintiffs have an incentive to file claims for increased risk because they fear that if they wait until cancer appears, their suit may be precluded by the statute of limitations and the rule against claim-splitting. The increased ability to detect subcellular changes such as DNA adducts that are part of the cancer process may be sufficient to start the statute of limitations running and increase pressure on plaintiffs to file an immediate claim for increased risk.

Greater use of new techniques to detect and measure exposure and risk will also promote more claims for fear of cancer. As seen, courts have imposed a much less burdensome standard for recovery of fear of cancer damages than recovery for increased risk. The number and size of these awards for fear of cancer can substantially increase the liability of companies producing or using hazardous substances. Estimates of increased risk provide the foundation of these claims. Finally, the increased data on toxic emissions that companies are now required to disclose under community right-to-know laws, such as Title II of the 1986 Superfund Amendments,[88] will provide more information about exposure and risks

[88]Emergency Planning and Community Right-to-Know Act, 42 U.S.C. §§ 11001–050, Chap. 116 (Supp. V 1987).

to residents. This information is expected to stimulate even more toxic tort cases, including claims for increased risk and fear of cancer.[89]

Insurers of such companies also face economic losses, since at least one court has already held that insurers with policies using the standard insurance industry definition of "personal injury" will have to defend claims for increased risk even if they are not compensable by the court.[90] Faced with this increased liability, defendants and their insurers are likely to rely increasingly on their own risk assessment evidence to demonstrate that plaintiffs' risks are lower than alleged. Defendants likely will also encourage courts to give greater scrutiny to the uncertainty and risks from other attributable factors contained in the plaintiffs' estimates of increased risk.

III.
ALTERNATIVE CAUSATION

A. *Traditional Legal Doctrine*

Plaintiffs in tort cases have the burden of proving all elements of their claims by a preponderance of the evidence, including the fact that the defendant caused the plaintiff's injuries. The doctrine of causation becomes complicated when two or more factors may have combined to produce a plaintiff's injury. Two different tests of legal causation are "but for" causation and "substantial factor" causation. Under the "but for" test of causation, "[t]he defendant's conduct is a cause of the event if the event would not have occurred but for that conduct."[91] Under the substantial factor test, "[t]he defendant's conduct is a cause of the event if it was a material element and a substantial factor in bringing it about."[92] Consequently, if an alternative contributing factor had a predominant effect on bringing about the plaintiff's injury, the defendant's activity may be relatively insignificant and would therefore not be a substantial factor.[93] A defendant's actions may also not be a substantial factor if there are many contributing factors which significantly dilute the effect of the defendant's conduct.[94]

It will generally be much easier for alleged victims of toxic substance exposure to prove that the toxic substance is a substantial factor in caus-

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[89]*See* Chadd & O'Malley, *Superfund Amendments Offer Hope for Plaintiffs in Toxic Tort Actions,* NAT'L L.J., March 21, 1988, at 16.

[90]Techalloy Co., Inc. v. Reliance Insurance Co., 338 Pa. Super. 1, 487 A.2d 820, 826 (Super. Ct. 1984).

[91]PROSSER & KEETON ON TORTS § 41 (5th ed. 1984).

[92]*Id.*

[93]RESTATEMENT (SECOND) OF TORTS § 433A commend d (1965).

[94]*Id.*

ing their injuries than it will be to prove "but for" causation. Plaintiffs will be subjected to many intervening causes and events between exposure to toxic substances and the development of latent disease many years later. Therefore, it will be very difficult to prove that their disease would not have developed if there had not been exposure to defendant's toxic substances many years before. Thus, decisions by courts on whether to apply the substantial factor test or the "but for" test for toxic tort causation will have a significant impact on liability.

When more than one cause is found to be a substantial factor in producing a plaintiff's injury, the liability may in some circumstances be apportioned among the various causes. In addition to cases in which there are two or more tortious causes, the apportionment rules also apply when the second contributing cause is the innocent conduct of another person, a force of nature, a pre-existing condition in the victim which the defendant has not caused, or the innocent or negligent conduct of the plaintiff.[95] The burden of proof for apportionment is on the tortious actor who seeks to reduce his liability.[96] Some kinds of harm, however, are "by their very nature . . . incapable of any logical, reasonable, or practical division."[97] Death and most types of personal injuries are traditionally viewed as indivisible harms.[98] For indivisible harms, each defendant can be held jointly and severally liable for the entire harm.[99] It is usually the function of the judge and not the jury to decide whether damages for a plaintiff's injuries can be apportioned.[100]

Evidence of possible alternative causes of a plaintiff's injuries will be introduced by defendants for three purposes.[101] First, defendants will attempt to absolve their actions of any responsibility by pointing to alternative causes that may have been the sole cause of the plaintiff's injuries. Second, if the defendant's activity is found to have contributed to the plaintiff's injuries, the defendant can still escape liability if its activity was not a substantial factor. Finally, if the defendant's activity is found to be a substantial factor, the defendant will seek to minimize its apportionment of liability by introducing evidence of other factors that also substantially contributed to the plaintiff's injuries.

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[95]*Id.*

[96]*Id.* at § 433B(2).

[97]*Id.* § 433A comment i.

[98]*Id.*

[99]*Id.*

[100]*Id.* § 434(1) (b).

[101]For a discussion of how to present an alternative causation defense, see McConnell, *Alternative Causation: The Best Defense is a Good Offense,* 4 TOXICS. L. REP. (BNA) 51 (1989); Royal, *The Defense of Medical Causation,* TRIAL, Oct. 1987, at 40.

B. *Recent Case Law*

a. Cases Involving Alternative Causation

The archetypical cases for alternative causation are those involving asbestos and cigarette smoking. It is well established that both asbestos and smoking can cause lung disease and cancer, and the effects of exposure to both cigarette smoke and asbestos can be multiplicative.[102] In several cases, asbestos workers who have developed cancer have sued asbestos manufacturers for recovery, but have had their damages discounted because they are heavy smokers. In other cases, evidence of alternative causation has been successfully used to protect a defendant from any liability. In one case, the defense presented evidence that the plaintiff's medical complaints were not caused by asbestos, but rather stemmed from the plaintiff's previous heart condition, shoulder problems, exposure to coke dust, hernia, and ulcers.[103]

A defendant's evidence of possible alternative causes of plaintiff's injuries can be very effective in reducing or preventing liability. Yet surprisingly, the number of reported toxic tort cases in which defendants introduce significant evidence of alternative causes is relatively small. It may be that the introduction of such evidence is often unnecessary or superfluous because the plaintiffs are unable to meet their burden of proof on causation in any event under the current legal doctrine.

b. Cases Suggesting Changes in Current Doctrine

Changes in legal doctrine that will ease plaintiffs' burden in proving causation can significantly increase the need and importance to defendants of introducing alternative causation evidence. One of the most important doctrinal questions is whether the substantial factor test should replace the "but for" test of causation. Courts are currently grappling with this question. The leading cases on this question are two recent decisions rendered in November, 1988: *Elam v. Alcolac, Inc.*[104] and *Menne v. Celotex Corp.*[105] In *Elam,* the Missouri court adopted the substantial factor test, concluding that the

> substantial factor standard—which ascribes liability to a cause which has played an important part in the production of harm, even though the harm may have occurred absent that cause—is

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[102]*See, e.g.,* Peto, Seidman & Selikoff, *Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment,* 45 BRIT. J. CANCER 124, 133 (1982).

[103]Giovanetti v. Johns-Manville Corp., 372 Pa. Super. 431, 539 A.2d 871, 876 (1988).

[104]765 S.W. 2d 42 (Mo. Ct. App. 1988), *cert. denied,* 110 S. Ct. 69 (1989).

[105]861 F.2d 1453 (10th Cir. 1989).

> particularly suited to injury from chronic exposure to toxic chemicals where the subsequent manifestation of biological disease may be the result of a confluence of causes.[106]

The Tenth Circuit Court of Appeals recently adopted a different approach in *Menne v. Celotex Corp.*, in which the plaintiff tried to recover damages from several asbestos manufacturers for his malignant mesothelioma.[107] The plaintiff, who could not establish which of the manufacturers had caused his injuries, argued that Nebraska should substitute a substantial factor test for a test of "but for" causation.[108] The court rejected this argument, ruling that Nebraska law incorporated both a "but for" and substantial factor test of causation.[109] The court held, however, that proof of exposure to a defendant's product at the approximate time of injury and under circumstances that could have caused the injuries was enough to establish a prima facie case of causation. The burden of proof then shifted to the defendant to demonstrate that its product was neither a substantial factor nor a "but for" cause of the plaintiff's injuries.[110] All defendant asbestos manufacturers who failed to meet this burden would be held jointly liable and required to pay a pro rata share of the plaintiff's damages.[111] Since asbestos was the only alleged source of the plaintiff's injuries in this case, the court noted that it was expressing no opinion as to whether the same burden-shifting rule should apply where both asbestos and non-asbestos factors are alleged to have caused a plaintiff's injuries.[112]

These two causes may signal a new trend in which courts significantly ease plaintiffs' burden of proving causation in toxic tort cases. Either a substantial factor standard, as adopted in *Elam,* or a burden-shifting rule, as applied in *Menne,* would place additional pressure on defendants to demonstrate that some other cause was responsible for plaintiffs' injuries. Therefore, if these new doctrinal developments expand to other jurisdictions, defendants will have an incentive to develop and introduce more sophisticated and convincing evidence of alternative causation.

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[106]*Elam,* 765 S.W.2d at 174 (citation omitted). *But see, e.g.,* In Re Bendectin Litigation, 857 F.2d 290, 311 (6th Cir. 1988) (rejecting replacement of the "but for" standard with the weaker "substantial factor" standard).

[107]861 F.2d 1453 (10th Cir. 1988).

[108]*Id.* at 1460.

[109]*Id.*

[110]*Id.* at 1468–69.

[111]*Id.* at 1469.

[112]*Id.* at 1468, n.25.

C. *Evidence of Alternative Causation*

Defendants are advised to use a two-fold strategy for defeating a plaintiff's attempt to prove causation. The "two pincers" of the attack are "insufficient proof that the product caused the harm and proof of alternative etiology."[113] So far, defendants have used quantitative assessments of risks primarily to show that their product did not cause the plaintiff's injuries rather than to prove that some other factor did. The most frequent use of quantitative risk evidence by defendants, however, is the introduction of negative epidemiological studies to deny causation.

While defendants often rely on epidemiological evidence to support the first prong of their causation attack (i.e., that their product did not cause the harm), they have used quantitative risk information much less frequently in the second prong to demonstrate alternative causes. Because the plaintiff traditionally has the burden of proof of causation, defendants usually have found it sufficient to simply suggest to the fact-finder possible alternative causes of the plaintiff's injuries.

Given the increased sophistication of risk information used by plaintiffs attempting to prove causation, plus the possibility of changing legal doctrines that will put more of the burden of proof on defendants to disprove causation, defendants now have greater incentive to support suggestions of alternative causes with quantitative risk information. The two major types of quantitative evidence useful for demonstrating or disproving causation are human epidemiological studies and quantitative risk assessments based on animal data. The courts, however, have been very inconsistent in determining the weight and admissibility of both types of quantitative evidence.

In *Johnson v. United States,*[114] the court rejected quantitative risk assessments, produced by both the plaintiffs' and the defendant's expert witnesses, that were based on extrapolations from occupational epidemiological data. Plaintiffs' experts had estimated that the probability that plaintiffs' cancers were caused by radiation exposure from luminous dials at an aircraft instrument factory was 76.2 to 99.7%, while the defendant's experts estimated the same probability to be only .01 to 3%.[115] The court stated that this type of quantitative risk assessment "is statistical speculation based upon speculative dose estimates and speculative risk assumptions. In other words, it is speculation based upon other speculation."[116] Although calculations of probability of causation are not without value, "a court must consider statistical risk calculations with a

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[113]Royal, *Introduction to the Medical Causation Defense in Toxic Tort Cases,* 55 DEF. COUNS. J. 41, 46 (1988).

[114]597 F. Supp. 374 (D. Kan. 1984).

[115]*Id.* at 412.

[116]*Id.* at 394.

healthy degree of skepticism."[117] After rejecting both the plaintiffs' and defendant's risk assessments, the court found that the plaintiffs' diseases were not caused by radiation from the aircraft instrument facility.

The courts have been even more unwilling to rely on quantitative risk assessments based on animal study data for proving causation.[118] Perhaps the strongest rejection of the use of animal study data came from Judge Weinstein in the Agent Orange litigation. He held there that animal studies "are of so little probative force and are so potentially misleading as to be inadmissible."[119]

Ferebee v. Chevron Chemical Co.[120] stands out as the most lenient approach for the introduction of causation evidence. The D.C. Circuit Court of Appeals, in a case involving pulmonary fibrosis allegedly caused by exposure to paraquat, permitted the plaintiff's experts to testify as to causation without relying on either specific epidemiological or animal studies demonstrating a statistically significant linkage between paraquat exposure and pulmonary fibrosis. The court held:

> [A] cause-effect relationship need not be clearly established by animal or epidemiological studies before a doctor can testify that, in his opinion, such a relationship exists. As long as the basic methodology employed to reach such a conclusion is sound, . . . products liability law does not preclude recovery until a "statistically significant" number of people have been injured or until science has had the time and resources to complete sophisticated laboratory studies of the chemical.[121]

A recent series of cases alleging birth defects from *in utero* exposure to the anti-nausea drug Bendectin illustrates the inconsistent approaches adopted by different courts to deal with risk assessment evidence in toxic tort cases. Over thirty-five epidemiological studies have failed to find a statistically significant link between Bendectin and birth defects. In light of this negative epidemiological evidence, the District of Columbia, and the First and Fifth Circuit Courts of Appeals have refused to admit expert evidence offered by the plaintiffs that was based on a chemical analysis of Bendectin's composition, *in vitro* studies of the effects of Bendectin on cells, animal studies, and unpublished and non-peer reviewed

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[117]*Id.*

[118]For arguments on why animal data should not be admissible to prove causation in toxic tort litigation, see Landau and O'Riordan, *Of Mice and Men: The Admissibility of Animal Studies to Prove Causation in Toxic Tort Litigation,* 25 IDAHO L. REV. 5212 (1988–89).

[119]In re Agent Orange Product Liability Litigation, 611 F. Supp. 1223, 1241 (E.D. N.Y. 1985).

[120]736 F.2d 1529 (D.C. Cir. 1984), *cert. denied,* 469 U.S. 1062 (1984).

[121]*Id.* at 1535–36.

reinterpretations of epidemiological data.[122] These courts concluded that "in the face of the overwhelming body of contradictory epidemiological evidence," these types of evidence, singly or in combination, "cannot furnish a sufficient foundation for a conclusion that Bendectin caused the birth defects at issue."[123]

Epidemiological evidence is "the most useful and conclusive type of evidence" in toxic tort cases, and the lack of statistically significant epidemiological proof of Bendectin's teratogenicity was "fatal" to the plaintiff's case.[124] *Ferebee* was distinguished on the grounds that the scientific evidence on the teratogenicity of Bendectin, unlike the evidence on paraquat in *Ferebee,* was well-established and did not "stand at the frontier of current medical and epidemiological inquiry."[125]

However, other courts have reached different conclusions when deciding similar cases involving Bendectin. An appeals court in the District of Columbia held that the four types of data rejected by the three federal circuit courts, when taken together, formed a "mosaic" sufficient to establish that Bendectin caused a plaintiff's birth defects.[126] More recently, a federal district court in the Ninth Circuit denied summary judgment to the manufacturer of Bendectin on the grounds that epidemiological studies, which demand a high degree of certainty, may find a causal link to be insignificant when the legal system, applying a more relaxed standard of "significance," might find that such a link did exist.[127] The court suggested that animal studies and evidence from chemical analysis may be admissible to counter negative epidemiological evidence introduced by the defendant. Four other federal district courts have also recently denied motions for summary judgment by the Bendectin manufacturer, ruling

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[122]Richardson v. Richardson-Merrell Inc., 857 F.2d 823 (D.C. Cir. 1988), *cert. denied,* 110 S. Ct. 218 (1989); Ealy v. Richardson-Merrell, Inc., 897 F.2d 1159 (D.C. Cir. 1990); Lynch v. Merrell-National Laboratories, Inc., 830 F.2d 1190 (1st Cir. 1987); Brock v. Merrell-Dow Pharmaceuticals, Inc., 874 F.2d 307 (5th Cir. 1989), *modified,* 884 F.2d 166 (5th Cir. 1989), ("we stated [in our earlier opinion] that '[w]e find, in this case, the lack of conclusive epidemiological proof to be fatal to the Brocks' case.' We hereby change that sentence to read '[w]e find, in this case, the Brocks' failure to present statistically significant epidemiological proof that Bendectin caused limb reduction defects to be fatal to their case.'") *Brock,* 884 F.2d at 167, *reh'g. denied,* 886 F.2d 1314 (5th Cir. 1989), *cert. denied,* 110 S.Ct. 1511 (1990).

[123]*Richardson,* 857 F.2d at 830.

[124]*Brock,* 874 F.2d at 311, 313.

[125]*Richardson,* 857 F.2d at 832.

[126]Oxedine v. Richardson-Merrell, Inc., 506 A.2d 1100 (D.C. 1986).

[127]Lonsmore v. Merrell-Dow Pharmaceuticals, Inc., 737 F. Supp 1117 (D. Idaho 1990).

that the jury must consider all relevant evidence and decide whether the drug causes birth defects.[128]

In summary, the courts are split on the use of epidemiological data to prove causation. A majority of courts, however, do favor such evidence, and the trend is clearly towards acceptance of epidemiological data. Nevertheless, a few recent decisions have still required causation to be proven by expert medical opinion that represents "the thought processes and considerations which a trained physician would engage in."[129] Furthermore, most courts have rejected the use of data from animal studies to prove causation. Similar evidentiary standards probably would apply to defendants attempting to use quantitative data to prove alternative causation.

Finally, new types of scientific evidence such as biological markers of exposure and genetic predispositions to disease may soon be available to prove alternative causation. Biological markers of exposure, such as the DNA adducts discussed above, may soon be available for allocating liability for toxic harm.[130] These markers will provide a toxicological "fingerprint" that indicates (and perhaps some day will quantify) a plaintiff's exposure to the defendant's products and possible alternative causes.

New molecular genetic techniques have led to the development of several hundred genetic markers for inherited diseases or susceptibilities. It may soon be possible to identify persons who are genetically predisposed to develop diseases that can also be caused by exposure to hazardous substances, such as anemias, dermatitis, asthma, emphysema, and several types of cancer.[131] Defendants who gain access to a plaintiff's medical records during discovery may be able to use the plaintiff's genetic "hypersusceptibility" to demonstrate alternative causation or to reduce liability through apportionment.

D. *Academic Commentary*

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[128]*See* 18 PROD. SAFETY & LIAB. REP. (BNA) 284, 285 (1990). Courts in several other toxic tort cases have also rejected the requirement that a plaintiff must use epidemiological evidence to prove causation. *See, e.g.,* Christopherson v. Allied-Signal Corp., 902 F.2d 362 (5th Cir. 1990).

[129]*E.g.,* Lee v. A.C. & S. Co., Inc., 542 A.2d 352, 355 (Del. Super. 1987).

[130]*See* Johnson, *Biological Markers in Tort Litigation,* 3 STATISTICAL SCIENCE 367, 368 (1988); Ellinger, *supra* note 44.

[131]Hunt, *The Total Gene Screen,* N.Y. Times, Jan. 19, 1986, Sec. 6 (Magazine), at 33. *See also* Ellinger, *supra* note 44; Kolata, *Genetic Screening Raises Questions for Employers and Insurers,* 232 SCIENCE 317 (1986); Murray, *Genetic Testing at Work: How Should It Be Used?,* TECHNOLOGY REVIEW, May/June 1985, at 51.

Many academic commentators believe that current legal doctrine imposes too high a burden on plaintiffs to prove legal causation.[132] Accordingly, there are many suggestions and strong pressure for doctrinal changes that ease a plaintiff's ability to demonstrate causation. Perhaps the most popular proposal is for proportional recovery, in which plaintiffs could recover damages in proportion to the likelihood that a defendant's activities caused their injuries.[133] Others have proposed that the burden of disproving causation should shift to the defendant once the plaintiff has demonstrated a prima facie case.[134]

With respect to the types of evidence that should be admissible to show causation or alternative causation, most commentators have favored the use of epidemiological data.[135] A few commentators believe, however, that courts should restrain the use of such evidence because it deals only with groups of people, and says nothing about whether a particular factor caused a specific plaintiff's injuries.[136] Therefore, commentators seem to split along the same lines as the courts, with the majority supporting the use of epidemiological evidence and a significant minority opposing its use.

The majority of scientists also appear to agree with the courts that animal data cannot yet be used to establish causation in humans. Although animal data has long been used by regulatory agencies to set standards, the policy basis for such use is different than in the toxic tort context. Neither the courts nor most commentators believe that animal data should be used to determine causation in toxic tort litigation at this time. If, however, further information reveals that animal studies data are a reliable predictor of human health effects, the courts will likely be more receptive to this type of evidence. Thus, animal study data may be admissible in the

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[132]*See, e.g.,* Farber, *Toxic Causation,* 71 MINN. L. REV. 1219, 1226 (1987); Trauberman, *Statutory Reform of "Toxic Torts": Relieving Legal, Scientific, and Economic Burdens on the Chemical Victim,* 7 HARV. ENVTL. L. REV. 177, 197 (1983).

[133]*See, e.g.,* Landes & Posner, *Tort Law as a Regulatory Regime for Catastrophic Personal Injuries,* 13 J. LEGAL STUD. 417, 425–26 (1984); Rosenberg, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System,* 97 HARV. L. REV. 849, 881–82 (1984).

[134]*E.g.,* Delgado, *Beyond Sindell: Relaxation of Cause-in-Fact Rules for Indeterminate Plaintiffs,* 70 CALIF. L. REV. 881, 899–900 (1982).

[135]"An epidemiological study reveals the correlation between some factor and a significant excess in the number of deaths or injuries above that which would otherwise have occurred—that is, above normal background levels." Dore, *A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-In-Fact,* 7 HARV. ENVTL. L. REV. 429, 443 (1983). *See also* Black & Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation,* 52 FORDHAM L. REV. 732 (1984); Brennan, *Causal Chains and Statistical Links: The Role of Scientific Uncertainty in Hazardous-Substance Litigation,* 73 CORNELL L. REV. 469, 506–09 (1988); Hall & Silbergeld, *Reappraising Epidemiology: A Response to Mr. Dore,* 7 HARV. ENVTL. L. REV. 441 (1983).

[136]*E.g.,* Dore, *supra* note 135, at 431.

future, especially considering the results of recent studies which found that the human risk estimates extrapolated from animal studies are strongly correlated with the risk estimates calculated from human epidemiological data.[137]

E. *Future Developments*

Currently, plaintiffs face a formidable barrier in proving causation in toxic tort litigation. Defendants are frequently able to defeat causation by a two-prong attack which consists of (1) introducing quantitative evidence that their products or wastes did not cause the plaintiffs' injuries and (2) suggesting alternative causes of the plaintiffs' injuries. Given the obstacles that plaintiffs have in proving causation, defendants usually only need to offer non-quantitative evidence of alternative causation to succeed on the causation issue. The difficulties plaintiffs face in proving causation are likely to spawn two trends that will put growing pressure on defendants to develop and introduce quantitative evidence of alternative causation.

The first likely trend is that plaintiffs will use increasingly sophisticated evidence attempting to prove causation. Improved risk quantification techniques and the promising development of exposure markers will probably be relied on by plaintiffs who, facing the bleak prospect of proving causation, see such evidence as their best hope. The use of more sophisticated risk evidence by plaintiffs will put pressure on defendants to respond with more refined evidence of their own. One possible response defendants could offer is to introduce quantitative evidence of alternative causation.

The second likely trend is that courts will adopt doctrinal changes that ease the burden of plaintiffs in proving causation. Courts have already experimented with eliminating the traditional "but for" causation requirement and shifting the burden of proof on the causation issue to the defendant. If a greater burden is imposed on defendants to show that their products did not cause a plaintiff's injuries, defendants may respond by introducing more convincing evidence of alternative causation. Thus, use of quantitative risk data to prove alternative causation in toxic tort litigation is likely to become increasingly prevalent in the future.

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[137]*See, e.g.*, Allen, Crump & Shipp, *Correlation Between Carcinogenic Potency of Chemicals in Animals and Humans*, 8 RISK ANALYSIS 531 (1988).

IV.
CONCLUSION

For many years now, federal agencies have relied on quantitative risk assessments to establish regulatory standards. Recently, use of such quantified risk information has commenced in toxic tort litigation. The courts, however, have been inconsistent in deciding which types of evidenc. to accept and what legal rules to apply. The variable and inconsistent holdings by the courts are indicative of a rapidly evolving area of law. Evolutionary changes in toxic tort litigation are taking place today because of shifts in traditional legal doctrines and the availability of more sophisticated methods for developing quantified risk information.

Changes in legal doctrine go hand-in-hand with changes in the available evidence. The availability of more reliable risk information may encourage courts concerned about speculation to adopt new legal doctrines which favor claims for increased risk. Conversely, doctrinal changes in causation requirements that lessen the plaintiff's burden will promote increased development and use of quantitative evidence of alternative causation by defendants.

Already, quantitative risk information is being used to support increased risk claims by plaintiffs, and, to a lesser extent, to demonstrate alternative causation by defendants. The combination of improvements in risk methodologies and evolving legal doctrines ensures that this type of risk information will be used even more frequently in the future. As the scientific and legal barriers to the use of quantitative risk information diminish, legal claims and defenses relying on such information will become more attractive. Consequently, claims for increased risk and defenses of alternative causation will become increasingly common and sophisticated in the future.