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HAZARDOUS SUBSTANCE LITIGATION IN MARYLAND: THEORIES OF RECOVERY AND PROOF OF CAUSATION

Ian Gallacher*

The bar is reminded that sound statistical analysis is a task both complex and arduous. Indeed, obtaining sound results by these means, results that can withstand informed testing and sifting both as to method and result, is a mission of comparable difficulty to arriving at a correct diagnosis of disease. We are no more statisticians than we are physicians, and counsel who expect of us informed and consistent treatment of such proofs are well advised to proceed as do those who advance knotty medical problems for resolution. Our innate capacity in such matters extends to "the inexorable zero" and perhaps, unevenly, somewhat beyond; but the day is long past . . . when we proceed with any confidence toward broad conclusions from crude and incomplete statistics. That everyone who has eaten bread has died may tell us something about bread, but not very much.1

I. INTRODUCTION

The recent torrent of lawsuits concerning exposure to hazardous substances2 shows no signs of diminishing. The ingenuity of the plaintiff's

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* Ian Gallacher is an associate at the law firm of Goodell, DeVries, Leech & Gray, LLP, where he practices in the areas of medical device, toxic tort, and complex litigation. An earlier version of this article was presented as part of a program on toxic tort litigation given by the Judicial Institute of Maryland in October, 1995. The author would like to thank Barbara Hammonds and the staff of the Judicial Institute for their assistance and permission to re-use some of that material. The author would also like to thank the following individuals for their insights and helpful suggestions: Charles P. Goodell, Jr., Hilary D. Caplan, Teri L. Kaufman, Kelly Hughes Iverson, Martin Freeman, John E. Griffith, Jr., and Colleen Rathbun. Thanks also to Julia McKinstry for her unflagging support.

2. "Hazardous substances" can be defined as occupational or environmental toxins or consumer products that are characterized by five properties. First, people are exposed to them in a chronic and relatively low-dose fashion. Second, exposed persons lack awareness of the toxic effect during the initial phase of the exposure. Third, the exposure is followed by
bar in developing new theories of recovery and methods of proof is matched only by the defense bar's ability to cast doubt on entire areas of proposed expert testimony offered to prove causation. As a result, courts are introduced to new and challenging concepts such as the "one hit" theory,\(^3\) "toxic soup,"\(^4\) "fiber drift" theory,\(^5\) and multiple chemical sensitivity syndrome.\(^6\)

This Article is intended to provide a framework for the analysis of the most commonly raised theories of recovery and modes of proof of causation offered in hazardous substance exposure cases, with particular reference to the law of Maryland. It first introduces theories of recovery that frequently have been offered by plaintiffs—present injury, multiple chemical sensitivity syndrome, post-traumatic stress disorder, increased risk of injury, fear of future injury, and medical monitoring—as well as some of the inherent problems of such theories. This Article then addresses the issue of standards of proof of causation and the central role of the expert witness in such proof. Lastly, analyzed in detail, is the nature of expert

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\(^3\) The principle behind the "one-hit" theory is that there is no quantity of a carcinogen that a person can be exposed to without incurring some increased risk. Marry v. Westinghouse Elec. Corp., 684 F. Supp. 847, 851 (M.D. Pa. 1988). In other words, one fiber of a carcinogenic substance coming into contact with the body causes an increased risk of cancer.

\(^4\) Courts face the "toxic soup" issue when a plaintiff is able to show that a variety of toxic substances were present at a site, but have been unable to show a causal connection between any one product and the specific injury they allegedly have suffered. See, e.g., McClelland v. Goodyear Tire & Rubber Co., 735 F. Supp. 172, 174 (D. Md. 1990). In Maryland, which has not adopted the "market share" theory of product liability, claims based on "toxic soup" are unlikely to survive summary judgement. \(\text{Id.}\)

\(^5\) "Fiber drift" theory posits that fibers of a carcinogen, such as asbestos, can become airborne and therefore drift to different locales within a site. Eagle-Pitcher v. Balbos, 326 Md. 179, 216-17 (1992). The "fiber drift" theory radically expands the potential liability of a defendant, because, if accepted, it can explain injuries suffered by workers who were not in direct contact with the carcinogen, but who worked in areas where the carcinogenic fibers could have drifted.

\(^6\) See discussion, infra, and notes 10-25 and accompanying text.
testimony typically offered, including areas of expertise and their rela-
tionship to the so-called Frye/Reed test\(^7\) and the advantages and disad-
vantages of the types of studies relied on by experts in these areas.

II. Theories of Recovery

Plaintiffs have offered many theories of recovery for exposure to haz-
ardous substances. The following are the most common theories and are
discussed here, starting with those which have the most apparent manifes-
tations of symptoms, and concluding with asymptomatic plaintiffs seeking
present recovery for future events.

A. Present Injury

The least complicated theory of recovery is presented by a plaintiff cur-
rently suffering from symptoms that are directly attributed to exposure to
a hazardous substance. The symptoms suffered by such a plaintiff are
typically serious or life-threatening. Such plaintiffs include workers ex-
posed to asbestos during their professional lives who now suffer from a
range of ailments, from microscopic changes in their internal physiology
to mesothelioma, as well as workers exposed to toxic chemicals and other
hazardous substances.\(^8\)

A plaintiff usually can prove with a simple diagnosis that he or she
suffers from the alleged medical condition. The more difficult issue in a
recovery for a present injury is attributing the medical condition to the
exposure to a hazardous substance.\(^9\)

\(^7\) The Frye/Reed test is familiar to practitioners as the test for the admissibility of
expert testimony before Daubert v. Merrell Dow Pharmaceuticals, Inc. 509 U.S. 579
(1993). The test was first established in Frye v. United States, 293 F.2d 1013 (D.C. Cir.
1923), and it requires that, in order for expert testimony regarding scientific testing to be
admissible, it must be sufficiently established to have gained general acceptance in the
particular field in which it belongs. Id. at 1014. The Frye standard was specifically adopted
by the Court of Appeals of Maryland, in a four-to-three decision, in Reed v. State, 391 A.2d
364, 368 (Md. Ct. App. 1978). Although Reed was a criminal case, the test also has been
applied in civil cases as well. See, e.g., Haines v. Shanholtz, 468 A.2d 1365 (Md. Ct. Spec.
App. 1984). Maryland has not adopted the Supreme Court's new formulation on the ad-
missibility of expert testimony set forth in Daubert. For a full discussion of Maryland evi-
dentiary issues, see JOSEPH F. MURPHY, JR., MARYLAND EVIDENCE HANDBOOK (1989).

\(^8\) See, e.g., Christophersen v. Allied-Signal Corp., 939 F.2d 1106, 1108 (5th Cir. 1991)
(worker exposed to nickel and cadmium fumes for 14 years died "as a result of rare, small-
cell form of cancer that originated in his colon and metastasized to his liver"); Ferebee v.
exposure to paraquat died from pulmonary fibrosis).

\(^9\) See Ferebee, 736 F.2d at 1534-35.
B. Multiple Chemical Sensitivity Syndrome

Multiple Chemical Sensitivity Syndrome ("MCSS")\(^{10}\) is perhaps the most controversial of all of the theories of recovery proposed by plaintiffs. MCSS is not easily defined. The Ad Hoc Committee on Environmental Hypersensitivity Disorders of the Ontario Ministry of Health proposed the following definition of MCSS:

[A] chronic (i.e. continuing for more than three months) multisystem disorder, usually involving symptoms of the central nervous system. Affected persons are frequently intolerant to some foods and they react adversely to some chemicals and to environmental agents, singly or in combination, at levels generally tolerated by the majority. Affected persons have varying degrees of morbidity, from mild discomfort to total disability. Upon physical examination, the patient is normally free from any abnormal, objective findings. Although abnormalities of complement and lymphocytes have been reported, no single laboratory test, including serum IgE, is consistently altered. Improvement is associated with avoidance of suspected agents and symptoms recur with re-exposure.\(^{11}\)

A scientist working in the MCSS field defined MCSS as:

[A]n acquired disorder characterized by recurrent symptoms, referable to multiple organ systems, occurring in response to demonstrable exposure to many chemically unrelated compounds at doses far below those established in the general population to cause harmful effects. No single widely accepted test of physiologic function can be shown to correlate with symptoms.\(^{12}\)

The symptoms of MCSS are as numerous as the definitions, including: "depression, irritability, mood swings, inability to concentrate or think clearly, poor memory, fatigue, drowsiness, diarrhea, constipation, sneezing, runny or stuffy nose, wheezing, itching eyes and nose, skin rashes, headache, muscle and joint pain, frequent urination, pounding heart, muscle incoordination, swelling of various parts of the body and even

\(^{10}\) MCSS is also known as ecological illness, environmental illness, chemical hypersensitivity syndrome, cerebral allergy, total allergy syndrome, or 20th-century disease.


\(^{12}\) Terr, supra note 11, at 168 (quoting Mark R. Cullen, M.D., The Worker with Multiple Chemical Hypersensitivities: An Overview, 2 Occupational Med. 655, 657 (1987)).
schizophrenia." Because MCSS is polysymptomatic with a multi-factorial etiology, it mimics numerous other medical conditions, and there is no test that clearly will identify it.

Patients with MCSS frequently are treated with chemical-exposure avoidance, diet manipulation, vitamins, and neutralization therapy. Practitioners "commonly recommend avoidance of perfumes, plastics, chlorine, alcohol, detergents, pesticides, smoke, natural gas fuel, carpets, gasoline, vehicle exhaust, restaurants, and synthetics." Practicing avoidance of these chemicals can involve relocation from an urban setting or withdrawal from many typically urban activities.

The dietary component of the treatment typically involves the "eating

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14. There are, however, several tests that frequently are performed by those practitioners who accept MCSS as a recognized condition. In particular, a "provocation-neutralization" test is frequently administered. In this test, a patient is administered doses of certain substances, usually including formaldehyde, phenol, alcohol, glycerin, saline, water, histamine, tobacco, newsprint, foods, food additives, and inhalant allergens. This list presumably would be supplemented were the practitioner to suspect that the MCSS was caused by workplace exposure to hazardous substances. These doses are administered either by intracutaneous injection or by sublingual drops. A test is determined to be positive if the patient shows subjective symptoms within ten minutes. If the patient does not show symptoms, higher doses are administered until a positive result is achieved. Terr, supra note 11, at 170.
15. Terr, supra note 11, at 170.
16. An example of the lifestyle of one diagnosed with MCSS can be found in Lawson v. Sullivan, No. 88C-10128, 1990 U.S. Dist. LEXIS 18758 (N.D. Ill. Oct. 30, 1990). The plaintiff, a senior editor for publications at Northwestern University, claimed social security benefits for her MCSS. Id. at *1. Since her treatment with Dr. Randolph, plaintiff has drastically changed her lifestyle and home environment. She now follows a Rotary Diet, she dines out much less, visits friends less, traveling is considerably curtailed, she bakes the newspaper every morning [Footnote 22 states plaintiff is highly sensitive to Toluene, a chemical found in printer’s ink. Dr. Randolph believes that plaintiff’s chemical sensitivity to Toluene was “probably picked up from printer’s ink exposure over the years as a galley proof reader.” Baking the newspaper apparently reduces the toxic effects of Toluene], no longer drives to gas stations to get gas in the car, and avoids supermarkets, fabric stores, movie theaters, copy centers and xerox machines. Air purifiers are found in her automobile and living room. She lives “in constant fear of getting zapped by someone’s deodorant or fabric softener.” When she “smelled formaldehyde in [her] polyester/cotton pillow cases, [she] switched to all-cotton.” She spends much of her time in her oasis — “a room made as pollutant-free as possible, with a portable electric heater.” Her husband is most sympathetic to her condition, and has changed his shampoo, toothpaste, bath soap, detergents, removed the carpeting from her ‘oasis,’ and changed the gas kitchen stove to an electric one.
Id. at *5-6 (footnotes omitted).
of only 'natural' foods purchased in specialty stores . . . in order to avoid pesticides and food additives." In particular, sugar is often avoided by patients with "candida hypersensitivity syndrome" because it is believed to enhance colonization of the intestinal tract by the fungus *C. albicans*. Vitamins often are prescribed in "megadoses," and mineral supplements, amino acids, and antioxidants also are frequently recommended. Drug therapy is avoided.

Other protocols also are used to treat MCSS. One such protocol, neutralization therapy, is an extension of the testing process in which the patient self-administers extracts of test substances in an attempt to relieve present symptoms or prevent anticipated symptoms.

Nevertheless, MCSS is a controversial theory that has received little support in mainstream medical circles. The Council on Scientific Affairs of the American Medical Association has found that there are "no well-controlled studies establishing a clear mechanism or cause for MCSS; and . . . there are no well-controlled studies providing confirmation of the efficacy of the diagnostic and therapeutic modalities relied on by those who practice clinical ecology." "Until . . . accurate, reproducible, and well-controlled studies are available, the Council . . . [has recommended] that . . . [MCSS] should not be recognized as a clinical syndrome."

The portmanteau-like quality of MCSS makes it an appealing theory of recovery to plaintiffs. As a condition, MCSS explains a number of objectively quantifiable symptoms that can be tied to a specific exposure. Additionally, because the patient is currently experiencing remission from symptoms of the syndrome, he is not attempting to recover for a possible future condition. Consequently, he is not faced with the proof problems inherent in litigating a possible future condition.

Although MCSS has not yet reached the reported decisions of the Maryland courts, its time for litigation has come. *The Baltimore Sun*, on October 11, 1994, reported the story of Mary Helinski, an employee of Bell Atlantic Corporation, who was suing her employer and four manufacturers of carbonless copy paper for causing her to suffer from MCSS.

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17. Terr, supra note 11, at 171.
18. Id.
19. Id.
20. Id.
22. Id.
In addition, *The Baltimore Sun*, on July 25, 1994, reported the case of a Catonsville woman, Lurie Bormel, who had a housing discrimination claim rejected by the Howard County Office of Human Rights. The report correctly observed: "So far, multiple chemical sensitivity has produced a trickle of lawsuits and discrimination complaints, but a flood is on its way."25

**C. Post-Traumatic Stress Disorder**

Post-Traumatic Stress Disorder ("PTSD") is another condition that plaintiffs have claimed is caused by exposure to hazardous substances. Courts that have become familiar with PTSD in the traumatic injury context might be surprised to see its use in hazardous exposure cases. However, PTSD offers a vehicle for recovery for present emotional and psychological damage that might otherwise be difficult to prove.

PTSD was re-defined recently by the American Psychiatric Association in the fourth edition of its Diagnostic and Statistical Manual ("DSM-IV") to require the satisfaction of several diagnostic criteria.27 These criteria

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24. Mark Guidera, *Housing Discrimination Case Rejected*, BALT. SUN, July 15, 1994, at B1. Ms. Bormel, a certified respiratory therapist, alleged that she could not work because the managers of the condominium complex where she lived did not give her adequate notice of spraying pesticides, herbicides, and other chemicals. *Id.* The Office of Human Rights, however, concluded that the condominium management made reasonable attempts to notify Ms. Bormel before spraying, and that the condominium association was looking into switching to organic or other alternative lawn care materials. *Id.* Accordingly, the Office concluded that there was insufficient evidence to continue with Ms. Bormel's housing discrimination case. *Id.*


27. These criteria are:

A. The person has been exposed to a traumatic event in which both of the following were present:

   (1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others;
   (2) the person's response involved intense fear, helplessness, or horror. . . .

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

   (1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions; . . . .
   (2) recurrent distressing dreams of the event; . . . .
differ from those as outlined in the DSM-III-R, the first edition of the DSM to recognize a PTSD diagnosis. In particular, the DSM-IV scope of the definition of the “stressor” or traumatic event necessary for a PTSD diagnosis has been narrowed significantly.

In addition to narrowing the circumstances under which a patient may

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). . . .
(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event;
(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma;
(2) efforts to avoid activities, places, or people that arouse recollections of the trauma
(3) inability to recall an important aspect of the trauma;
(4) markedly diminished interest or participation in significant activities;
(5) feeling of detachment or estrangement from others;
(6) restricted range of affect (e.g., unable to have loving feelings);
(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep;
(2) irritability or outbursts of anger;
(3) difficulty concentrating;
(4) hypervigilance;
(5) exaggerated startle response.

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than one month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.


29. The former definition of “stressor” was:

A. The person has experienced an event that is outside the range of usual human experience and that would be markedly distressing to almost anyone, e.g., serious threat to one’s life or physical integrity; serious threat or harm to one’s children, spouse, or other close relatives and friends; sudden destruction of one’s home or community; or seeing another person who has recently been, or is being, seriously injured or killed as the result of an accident or physical violence.

Id. at 250.
be diagnosed with PTSD, the American Psychiatric Association also has
developed a new diagnosis for a condition named “Acute Stress Disor-
der” (“ASD”). ASD is a milder form of PTSD, involving the same stres-
sor but with fewer dissociative symptoms required for a diagnosis and
with a definitionally limited duration.\textsuperscript{30}

Because of its broader definition, ASD may appear to be a more at-
tractive option for plaintiffs seeking recovery for the alleged psychologi-
cal reaction to exposure to hazardous substances than PTSD as defined in
the DSM-IV. However, the four week duration set on this diagnosis
should limit its appeal to plaintiffs in toxic exposure lawsuits.

30. The diagnostic criteria for ASD are as follows:
A. The person has been exposed to a traumatic event in which both of the fol-
lowing were present:
   (1) the person experienced, witnessed, or was confronted with an event or
      events that involved actual or threatened death or serious injury, or a threat
      to the physical integrity of self or others
   (2) the person’s response involved intense fear, helplessness or horror.
B. Either while experiencing or after experiencing the distressing event, the in-
dividual has three (or more) of the following dissociative symptoms:
   (1) a subjective sense of numbing, detachment, or absence of emotional
      responsiveness
   (2) a reduction in awareness of his or her surroundings (e.g., “being in a
      daze”)
   (3) derealization
   (4) depersonalization
   (5) dissociative amnesia (i.e., inability to recall an important aspect of the
      trauma).
C. The traumatic event is persistently reexperienced in at least one of the fol-
lowing ways: recurrent images, thoughts, dreams, illusions, flashback episodes, or
a sense of reliving the experience; or distress on exposure to reminders of the
traumatic event.
D. Marked avoidance of stimuli that arouse recollections of the trauma (e.g.,
thoughts, feelings, conversations, activities, places, or people).
E. Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping,
irritability, poor concentration, hypervigilance, exaggerated startle response, mo-
tor restlessness).
F. The disturbance causes clinically significant distress or impairment in social,
occupational, or other important areas of functioning or impairs the individual’s
ability to pursue some necessary task, such as obtaining necessary assistance or
mobilizing personal resources by telling family members about the traumatic
experience.
G. The disturbance lasts for a minimum of 2 days and a maximum of 4 weeks
and occurs within 4 weeks of the traumatic event.
H. The disturbance is not due to the direct physiological effects of a substance
(e.g., a drug abuse, a medication) or a general medical condition, is not better
accounted for by Brief Psychotic Disorder, and is not merely an exacerbation of a
preexisting Axis I or Axis II disorder.

DSM-IV, \textit{supra} note 27, at 431-32.
The United States Court of Appeals for the Sixth Circuit, in *Sterling v. Velsicol Corporation*, discussed the applicability of PTSD to hazardous substances exposure litigation. The court analyzed PTSD under the older, more liberal, diagnostic criteria of the DSM-III-R yet still determined that the district court had wrongly awarded damages.

Plaintiffs' drinking or otherwise using contaminated water, even over an extended period of time, does not constitute the type of recognizable stressor identified either by professional medical organizations or courts. Examples of stressors upon which courts have based awards for PTSD include rape, assault, military combat, fires, floods, earthquakes, car and airplane crashes, torture, and even internment in concentration camps, each of which are natural or man-made disasters with immediate or extended violent consequences. Whereas consumption of contaminated water may be an unnerving occurrence, it does not rise to the level of the type of psychologically traumatic event that is a universal stressor.

The DSM-IV definition of "stressor" will make it even more difficult for a plaintiff to prevail on a PTSD theory in a hazardous substances exposure case. This is not to say that such a diagnosis is impossible, however, and it is likely that courts will be dealing with the interplay between PTSD, ASD, and hazardous substances for many years to come.

### D. Increased Risk of Injury

Under this theory of recovery, plaintiffs assert a claim for a present injury based on the prospect that they might suffer another injury in the future. The theory of enhanced risk of future injury has been described as "one of the most challenging causes of action [for a plaintiff] to prove," and is closely related to the "loss of chance" theory of recovery.

A plaintiff who sues under a theory of enhanced risk of future injury alleges that because of exposure to a hazardous substance, his or her potential for contracting a disease in the future is heightened. Furthermore, the plaintiff asserts that he should be compensated now for the possible future harm. Recovery under such a theory does not prevent the plaintiff

31. 855 F.2d 1188 (6th Cir. 1988).
32. *Id.* at 1210 (citations omitted).
from suing again if he or she, in fact, contracts the disease in the future.\(^3\)

In order to recover for enhanced risk of future injury in Maryland, however, a plaintiff must meet the Maryland standard for recovery of damages based on future consequences of injury, as articulated in *Pierce v. Johns-Manville Sales Corporation*.

In *Pierce*, the Court of Appeals of Maryland held that recovery could only be had if the consequences are reasonably probable or reasonably certain. Such damages cannot be recovered if future consequences are "mere possibilities." Probability exists when there is more evidence in favor of a proposition than against it (a greater than [fifty percent] chance that a future consequence will occur). Mere possibility exists when the evidence is anything less.\(^3\)

The Court of Appeals of Maryland recognized a cause of action based on increased risk of future injury in *Charlton Brothers Transportation Company v. Garrettson*, although not in the context of hazardous substances exposure.\(^3\) However, no court has cited this case in support of the proposition that an increased risk of future injury will support a present cause of action for almost fifty years. Accordingly, its precedential value, especially in the context of exposure to hazardous substances, is doubtful at best.\(^4\)

Moreover, the court's recognition of a cause of action for an increase in risk that was less than fifty-one percent indicates that

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35. One commentator has noted that "[a]ny recovery received in the second action would be discounted by damages received in the first action." Feldman, supra note 34, at 154 (citing Joseph H. King, Jr., *Causation, Valuation, and Chance in Personal Injury Torts Involving Preexisting Conditions and Future Consequences*, 90 *Yale L.J.* 1353, 1382 (1981) (suggesting that "[c]ompensation should be calculated in a way that avoids double recovery for the same injury") (emphasis added)). Why this result would obtain is unclear. If the increased risk of future harm is a separate cause of action, it is not the same injury, and therefore there is no question of double recovery. If the injury is the same, then the plaintiff should not be permitted to split the cause of action between currently manifested heightened risk of injury and actual injury manifested in the future.


37. *Id.* at 1026 (citation omitted).

38. 51 A.2d 642, 646 (Md. 1947).

39. In *Garrettson*, the plaintiff sustained a hernia when the street car in which he was riding collided with a truck. *Id.* at 644-45. Prior to the accident, the plaintiff had a 10% chance of suffering a recurring hernia; after the accident, the risk of a recurring hernia increased to 50%, even if he submitted to what the court termed a "formidable" operation. *Id.* at 646. The court of appeals held that the increased risk of suffering a recurring hernia was a present injury for which the plaintiff could recover. *Id.* "This increase in hazard is itself a permanent injury. Expert testimony hardly seems necessary to show that such an operation would leave plaintiff in a permanently weaker condition." *Id.*

40. Feldman, however, states that this case constitutes precedent for the recognition of a cause of action for increased risk. Feldman, supra note 34, at 158.
Garrettson could not support this theory of recovery after Pierce.\textsuperscript{41}

\section*{E. Fear of Future Injury}

The theory of recovery for a plaintiff's present fear of a future injury is linked closely to the concept of recovery for an increased risk of injury. However, the analysis of these two theories is not identical, and it is possible for a plaintiff to be unable to prove with sufficient probability that a future injury \textit{will} occur and yet still be able to recover for his or her present \textit{fear} that the injury will occur.\textsuperscript{42}

Present fear of future injury\textsuperscript{43} was analyzed recently by the Court of Appeals in Maryland in \textit{Faya v. Almaraz}.\textsuperscript{44} In \textit{Faya}, two former patients brought suit against a surgeon who operated on them without first in-

\textsuperscript{41} The court ruled that a 40\% increase in probability that the hazard would occur, for a total of 50\% likelihood, was sufficient to constitute a permanent, and, by implication, present injury. In fact, as discussed \textit{supra} at text accompanying notes 36-37, the court of appeals has held that a future circumstance must be "reasonably probable or reasonably certain." \textit{Pierce}, 464 A.2d at 1026. The court's own holding in \textit{Garrettson} makes clear that this standard was not met in that case, making it, at best, weak support for the proposition that a plaintiff can recover for an increase in risk of injury. \textit{Garrettson}, 51 A.2d at 646.

\textsuperscript{42} See, e.g., \textit{Sterling v. Velsicol Chem. Corp.}, 855 F.2d 1188, 1206 (6th Cir. 1988).

While there must be a reasonable connection between the injured plaintiff's mental anguish and the prediction of a future disease, the central focus of a court's inquiry in such a case is not on the underlying odds that the future disease will in fact materialize. To this extent, mental anguish resulting from the chance that an existing injury will lead to the materialization of a future disease may be an element of recovery even though the underlying future prospect for susceptibility to a future disease is not, in and of itself, compensable as it is not sufficiently likely to occur. \textit{Id.}

\textsuperscript{43} The "fear of future injury" issue also raises the issue of "phobia." The Sixth Circuit, in \textit{Velsicol}, opined that "[c]ancerphobia is merely a specific type of mental anguish." \textit{Id.} at 1206 n.24. Others agree, using less neutral language. "While it appears to be an entirely new development, in fact, cancerphobia is merely a branch of the garden-variety emotional distress tort. Cancerphobia 'grew' from emotional distress just like the story about the Little Shop of Horrors, where a man-eating monster 'grew' from a cute little flower." Martha A. Churchill, \textit{Medical Monitoring and Cancerphobia — The Rise of Fear Lawsuits, For The Def.}, Aug. 1993, at 2.

In fact, there clearly is a difference between the two concepts. "Fear" can be defined as "an unpleasant often strong emotion caused by anticipation or awareness of danger." \textit{WEBSTER'S NINTH NEW COLLEGIATE DICTIONARY} 453 (1986). By contrast, "phobia" can be defined as "an exaggerated [(usually)] inexplicable and illogical fear of a particular object or class of objects." \textit{Id.} at 883. In simple terms, fear is a rational response to danger while phobia is irrational. In the context of exposure to hazardous substances, plaintiffs most often claim either AIDS phobia, when exposed to blood products, used hypodermic needles, or other hospital waste, or cancerphobia, most often relating to exposure to asbestos or carcinogenic chemicals.

\textsuperscript{44} 620 A.2d 327 (Md. 1993).
forming them that he had AIDS. The trial court dismissed both complaints because the plaintiffs had failed to allege that the surgeon had used improper barrier techniques. Accordingly, there was no transmission mechanism by which either patient could have been exposed to the HIV virus.

The court of appeals reversed, stating that:

[W]e cannot say that appellants' alleged fear of acquiring AIDS was initially unreasonable as a matter of law, even though the averments of the complaints did not identify any actual channel of transmission of the AIDS virus. . . . [A] requirement that plaintiffs must allege actual transmission would unfairly punish them for lacking the requisite information to do so.

The court stressed that the plaintiffs' fear must be reasonable.

In reaching its conclusion, the Court of Appeals of Maryland rejected a test first articulated by the United States District Court for the Eastern District of Pennsylvania, in Burk v. Sage Products, Inc. In Burk, the court held that a plaintiff who had been stuck by the needle of a discarded syringe could not recover for his fear of contracting AIDS. The court based its decision on the plaintiff's inability to show that the needle had been used on a patient with AIDS. Furthermore, the plaintiff had tested HIV-negative five times in the thirteen months since the incident.

The Court of Appeals of Maryland declined to follow the requirement in Burk “that plaintiffs must allege actual transmission.” In fact, the Burk court merely required that a plaintiff demonstrate that he had been exposed to the AIDS virus. Exposure is very different from “actual transmission” and the court's misstated rationale could lead to many more “fear of future injury” cases in the Maryland courts, especially in the context of exposure to hazardous substances.

For example, the Burk court cited Cathcart v. Keene Industrial Insula-

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45. Id. at 328.
46. Id. at 330-31.
47. Id. at 336-37.
48. Accordingly, the court noted that there is a 95% chance that one will test positive for HIV contamination within six months after exposure, and held that, because the plaintiffs had tested negative for HIV more than one year after contact with the surgeon, they could only recover for their fear of contracting AIDS from the time they first learned of the surgeon's HIV status until the time they learned of their HIV-negative status. Id. at 337.
50. Id. at 286-88.
51. Faya, 620 A.2d at 337.
where the plaintiff sued for damages based on her fear of contracting disease. The plaintiff alleged that she was exposed to a hazardous substance when she inhaled the asbestos fibers on her husband’s work clothes. The Pennsylvania court held that the plaintiff could not recover, noting that until she was able to allege physical injury or a medically identifiable effect linked to her exposure to asbestos particles, her claim was not cognizable.

In Maryland, under the holding of Faya, Cathcart presumably would have been decided differently. Because a Maryland plaintiff no longer must prove the mechanism whereby he or she has been exposed to a hazardous substance, Maryland courts must anticipate numerous cases where plaintiffs will attempt to determine the outer limits of the Faya decision.

Interestingly, the court in Faya did not resolve, or even address, the issue of how a plaintiff who cannot prove a mechanism for transmission of AIDS can satisfy the Pierce requirement that recovery of damages based on future consequences of an injury “may be had only if such consequences are reasonably probable or reasonably certain.” A plaintiff who cannot prove that he or she was exposed to a hazard, cannot prove that it is reasonably probable or certain that he or she will suffer future consequences. The court’s silence on this issue leaves the Maryland trial courts with no guidance in this area.

The Faya opinion has further significance in that it provides the most recent articulation of the law in Maryland regarding the necessity for physical injury when seeking emotional damages.

The Faya court began its analysis by noting that it was formerly the rule that there could be no recovery of tort damages for mere fright or mental suffering caused by negligence unconnected with physical impact or in-

53. Id. at 508.
54. Id.
55. Although Faya is a case involving exposure to AIDS and the HIV virus, the court of appeals was well aware of the case's implications for other hazardous substance exposure litigation. The court observed, in a footnote, that
   [m]ost cases addressing the viability of claims alleging fear of contracting disease concern exposure to asbestos, toxic chemicals, pesticides, and drug products. These cases often rely on the first holding of Burk: in the absence of facts demonstrating legitimate exposure to the disease-causing agent, there can be no recovery.
Faya, 620 A.2d at 335 n.7. Any comfort that might be derived by the defense bar from this remark, however, is tempered by the realization that it is precisely this holding from Burk that the court declined to follow.
56. Pierce, 464 A.2d at 1026.
The court noted, however, that subsequent cases departed from this rigid rule. The court then examined several cases that weakened the "physical impact" rule, finally focusing on Vance v. Vance. In Vance, the plaintiff, a woman married to the defendant in a religious ceremony, sought a divorce decree after the defendant left her for another woman. The defendant opposed the action on the grounds that he and the plaintiff were never legally married because he had not received a final divorce decree from his first wife prior to his "marriage" to the plaintiff.

The plaintiff had a negative reaction to this news:

She went into a state of shock, engaged in spontaneous crying and for a period seemed detached and unaware of her own presence. She was unable to function normally, unable to sleep and too embarrassed to socialize. In addition to experiencing symptoms of an ulcer, [plaintiff] suffered an emotional collapse and depression which manifested itself in her external condition, i.e., her significantly deteriorated physical appearance—unkempt hair, sunken cheeks and dark eyes.

From this evidence, the court concluded that the plaintiff had suffered an objectively manifested, definite nervous disorder which was sufficient to support a jury finding that she was physically injured as a foreseeable result of the defendant's negligent misrepresentation concerning his marital status.

Using Vance as an analogy, the Faya court concluded that the plaintiffs who alleged symptoms of headache, sleeplessness, "and the physical and financial sting of blood tests for the AIDS virus," could recover for their fear and mental and emotional distress. Thus, Faya teaches that the act of drawing blood to perform diagnostic testing combined with the act of paying for such testing is sufficient to meet Maryland's vestigial "physical impact" requirement. Distilled to its essence, the Faya opinion appears to eliminate the physical impact requirement in Maryland.

It is difficult to overstate the significance of Faya. Although this case legitimately can be seen as being restricted to its facts, and viewed as an exercise of judicial public policy, especially because it is an AIDS case.

57. Faya, 620 A.2d at 337 (citations omitted).
58. Id. (citation omitted).
59. 408 A.2d 728 (Md. 1979).
60. Id. at 729.
61. Id. at 734.
62. Id. (citation omitted).
63. Faya, 620 A.2d at 338.
64. In fact, it is possible to read Faya as a case that stands for little more than the
Faya can also be read as a case that dramatically expands the ability of a plaintiff to recover for his or her fear of future injury after exposure to a hazardous substance.

F. Medical Monitoring

Medical monitoring claims typically are brought when large groups of individuals have been exposed to a toxic substance.65 The exposure can be occupational66 or environmental.67 Because the exposure to toxic substances typically has been experienced by a relatively large group of individuals, medical monitoring relief usually is sought pursuant to class action certification.68 Courts have certified such classes when the defendant's liability clearly has been established, when the disease process has a long latency period, and when a prudent physician would order preventative testing in order to detect and treat the full-blown disease at its earliest manifestation.69

Although medical monitoring is thought of as equitable relief, and medical monitoring classes traditionally are sought under the Federal Rules of Civil Procedure 23(b)(2), the so-called "equitable class" provision,70 the proper theory of recovery is more complicated. Most state courts that have looked at the medical monitoring issue have analyzed the recovery in terms of measure of damages, awarded after a finding of liability after proof of an underlying tort.71 It is a well-established princi-

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68. See, e.g., Day, 851 F. Supp. at 869.

69. Id. at 880.

70. See, e.g., In re NLO, Inc., 5 F.3d 154, 159 (6th Cir. 1993).

ple that damages are legal, not equitable, in nature,72 and where the damages remedy is adequate, the courts should not grant equitable relief.73 Confronted with this problem, courts have declined to certify medical monitoring classes under Rule 23(b)(2).74

Courts are still grappling with some of the implications of medical monitoring relief. In particular, given the prohibition against splitting causes of action in many states, seeking relief for medical monitoring during the latency of a disease process may preclude a plaintiff from later seeking recovery for the injuries caused by a manifested disease.75 Because medical monitoring relief has the potential to cut-off further attempts at recovery later, counsel for both plaintiffs and defendants should be alert to the potential implications of an attempt to recover under a medical monitoring theory.

A plaintiff who seeks the remedy of medical monitoring typically must establish 1) significant exposure to a hazardous substance that was wrongfully caused by the defendant; 2) an increased risk of contracting a serious disease; 3) that periodic diagnostic examinations are reasonable and necessary because of the increased risk; and 4) that monitoring will be helpful in facilitating early detection and treatment.76

Courts that have rejected a cause of action for medical monitoring typically have done so on the basis of an absence of present injury77 or because a plaintiff has failed to show a reasonable probability that injury will occur.78 These same rationales for denying medical monitoring recovery were rejected by the New Jersey Supreme Court in the seminal medical monitoring case, Ayers v. Jackson Township.79

In Ayers, the New Jersey Supreme Court held that the use of courtsupervised funds to administer medical-surveillance payments in mass

74. See, e.g., Castano v. American Tobacco Co., 160 F.R.D. 544 (E.D. La. 1995), overruled on other grounds, 84 F.3d 734 (5th Cir. 1996) “[C]ertification of the medical monitoring claim in this case under Rule 23(b)(2) would infringe on the constitutional right to a jury trial. The court cannot and will not infringe on that inviolate right.” Id. at 552.
76. Paoli, 916 F.2d at 852.
toxic exposure cases was an appropriate exercise of the court's equitable powers. In particular, the court noted that if an individual was exposed to toxic chemicals, an early diagnosis could lead to improved prospects for cure, prolongation of life, relief of pain, and minimization of disability. In addition, the court held that it would be inequitable for an individual who wrongfully was exposed to dangerous toxic chemicals, but who was unable to prove that he likely would contract a disease, to be equipped to pay his own expenses when medical intervention was reasonable and necessary.

The state of a medical monitoring claim in Maryland is open to interpretation. On the one hand, the Court of Appeals of Maryland has not overtly recognized a cause of action for medical monitoring. Indeed, one commentator has stated that Maryland courts have . . . held loyal to traditional tort doctrine in restricting the viability of toxic tort claims. Given the difficulties in establishing probability of harm as a prerequisite to any remedy, no matter how progressive, it seems unlikely that the remedy of medical surveillance damages will be welcome in this jurisdiction in the near future.

On the other hand, a year after the above statement was written, the court of appeals handed down its decision in Faya. In that case, the plaintiffs were allowed to recover for the expense of testing for the HIV virus. Although the court did not explicitly recognize it as such, this was recovery for medical monitoring. The plaintiffs' recovery in Faya was limited to the testing necessary to confirm, with ninety-five percent accuracy, that they would not become infected with the HIV virus. Such a limitation could be interpreted, however, to mean only that further testing would not be reasonable or necessary, and that further testing would not aid in the early warning and treatment of health problems. Accordingly, in a disease process with a latency period measured in years rather than months, Faya could be used to support testing over the entire latency period.

It is unclear whether the court of appeals was fully aware of the impli-
cations of the Faya opinion when it was decided. It is certain, however, that the Maryland courts will be given many opportunities to define the parameters of that opinion in the years to come, and medical monitoring will be one of the issues at the forefront of that debate.

III. PROOF OF CAUSATION

Having identified the theories under which he may wish to pursue relief, the plaintiff must then decide how to go about proving causation. This is the most difficult burden for plaintiffs to overcome in toxic tort litigation.87

Ordinarily, proof of causation requires the establishment of a sufficient nexus between the defendant's conduct and the plaintiff's injury. In toxic tort cases, the task of proving causation is invariably made more complex because of the long latency period of illnesses caused by carcinogens or other toxic chemicals. The fact that ten or twenty years or more may intervene between the exposure and the manifestation of disease highlights the practical difficulties encountered in the effort to prove causation. Moreover, the fact that segments of the entire population are afflicted by cancer and other toxically-induced diseases requires plaintiffs, years after their exposure, to counter the argument that other intervening exposures or forces were the "cause" of their injury.88

Issues arising from a plaintiff's attempt to meet his burden of proving causation, and a defendant's attempt to discredit such proof, form the basis for the second half of this Article. In particular, the standards of proof required to make a *prima facie* case of exposure to hazardous substances are discussed. Finally, the importance of expert testimony, the appropriate standard for admissibility, and the types of experts and the studies upon which they rely, are analyzed.

A. Standards of Proof

To establish causation, a plaintiff must prove that an identifiable hazardous substance caused his or her injuries. The Maryland courts use several standards of proof to determine whether a plaintiff has met his burden.

87. Ayers, 525 A.2d at 301 (citation omitted).
88. Id.
1. Substantial Factor

Maryland has adopted the "substantial factor" test as the standard by which a plaintiff's causation evidence must be evaluated. This test is derived from section 431 of the Restatement (Second) of Torts.

The Court of Appeals of Maryland has applied the "substantial factor" test in the asbestos setting for cases involving bystanders who were not direct users of the hazardous product. The court noted that in such a case, the test is fact specific to each individual's case, requiring an understanding of the interrelationship between the use of the product in the workplace and the plaintiff's activities in the workplace.

This requires an understanding of the physical characteristics of the workplace and of the relationship between the activities of the direct users of the product and the bystander plaintiff. Within that context, the factors to be evaluated include the nature of the product, the frequency of its use, the proximity, in distance and in time, of a plaintiff to the use of a product, and the regularity of the exposure of that plaintiff to the use of that product.

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90. Section 431 of the Restatement (Second) of Torts provides as follows:

The actor's negligent conduct is a legal cause of harm to another if
(a) his conduct is a substantial factor in bringing about the harm, and
(b) there is no rule of law relieving the actor from liability because of the manner in which his negligence has resulted in the harm.

RESTATEMENT (SECOND) OF TORTS § 431 (1965).

Comment (a) to section 431 expands on this definition:

The word "substantial" is used to denote the fact that the defendant's conduct has such an effect in producing the harm as to lead reasonable men to regard it as a cause, using that word in a popular sense, in which there always lurks the idea of responsibility, rather than in the so-called "philosophic sense" which includes every one of the great number of events without which any happening would not have occurred. Each of these events is a cause in the so-called "philosophic sense," yet the effect of many of them is so insignificant that no ordinary mind would think of them as causes.

Id. § 431 cmt. a.

91. Eagle-Picher, 604 A.2d at 459. In this case, the court of appeals includes an alternative formulation of the substantial factor rule from Prosser:

When the conduct of two or more actors is so related to an event that their combined conduct, viewed as a whole, is a but-for cause of the event, and application of the but-for rule to them individually would absolve all of them, the conduct of each is a cause in fact of the event.

Id. at n.11.

92. Id. at 460.

93. Id. (citations omitted).
The "substantial factor" test has been criticized as an insufficient standard to measure the relevance or sufficiency of evidence. Where the "but for" test clearly addresses the kind of relationship the law requires between an alleged cause and an alleged effect, the "substantial factor" test provides no guidance at all on this question. It was originally formulated to clarify proximate cause analysis, and its misuse in determining cause-in-fact is both unnecessary and ironic.

2. Significant but Unquantifiable Risk

Where a plaintiff cannot define with specificity the dose necessary to make a substance harmful, courts have adopted the concept of "significantly but unquantifiably enhanced risk" in order to permit litigation to


95. Id.

As an example of the "but for" test, Black proposes the following hypothetical: [If a man and a woman fire rifles simultaneously and negligently, and both of their bullets strike another person in the heart, the woman could claim her shot caused no harm because the victim would have died anyway. The man could make the same argument, meaning both defendants would escape liability. Id. (footnote omitted).]

It is this flaw in the "but for" test which, Black argues, led to the development of the "substantial factor" test. Black contends however, that a simple amendment to the "but for" test would permit its use without recourse to the inadequate "substantial factor" test:

One only need ask about each hunter whether it would have been necessary to eliminate his or her rifle shot for the plaintiff to have avoided death. Because either bullet would have been fatal by itself, the answer in each case would be "yes," making both the man and the woman causes-in-fact. Though the grammar may be clunky and unwieldy, the logic of the "reverse but for" test is quite clear. Id.

The effect of the adoption of this test instead of the "substantial factor" test can be seen by applying both tests to the facts of Eagle-Picher. In that case, expert witnesses testified that:

"[A]ll of [the] exposures to asbestos were a significant contributing causal factor to the mesothelioma," because the causation is "cumulative." ... Thus, the failure to warn on the part of any one supplier of an asbestos product to which a decedent was exposed can operate as a concurrent proximate cause with the failures to warn on the part of other such suppliers. Eagle-Picher, 604 A.2d at 459.

Accordingly, the "substantial factor" test was satisfied. However, under the "reverse but for" test, it would nor have been necessary to eliminate a specific manufacturer's asbestos-carrying product for the plaintiff to have avoided death, because exposure is cumulative, and there is no evidence to indicate that the plaintiff would not have been exposed to a sufficient quantity of asbestos to have caused him to contract mesothelioma in any case. Accordingly, under the "substantial factor" test, there was sufficient evidence for a jury to conclude that Eagle's activities were a proximate cause of the decedent's injuries. Under the "reverse but for" test, there was not.
proceed. For example, in *Merry v. Westinghouse Electric Corp.*, the court acknowledged that plaintiffs’ experts had not, and could not, quantify the plaintiffs’ chances of contracting an exposure-related disease. Nonetheless, the court held that the plaintiffs could escape summary judgement. Citing the plaintiffs’ expert’s conclusion that a medical monitoring program would be prudent based on the documented and “reasonably presumed” exposure to toxic substances, the court held that the plaintiffs had created an issue of fact as to the probability of contracting a serious illness. “It would be reasonable for a jury to conclude that the plaintiffs have a significantly but unquantifiably enhanced risk of serious disease and that such enhanced risk of disease justifies periodic medical examinations.”

In order to meet the “substantial factor” test, it is necessary that a plaintiff first prove that the substance to which he or she was exposed was hazardous or toxic. This task is at once simpler and more complex than might first be apparent.

On one level, proof of a product’s toxicity is extremely simple as *everything* is toxic to some degree. In fact, the truly relevant question is whether the dose of the substance under consideration was sufficiently large as to be harmful or poisonous.

The concept of a significant but unquantifiably enhanced risk is difficult to accept. If one cannot quantify the enhanced risk, how can one be sure that the risk is significant or was a “substantial factor” in causing plaintiff’s injury? The rationale behind this concept, the “one-hit” theory, provides that “there is no quantity of a carcinogen that a person can be exposed to without incurring some increased risk.” Although this may

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97. See also *In re Paoli R.R. Yard PCB Litig.*, 916 F.2d 829, 852 (3d Cir. 1990).
98. Id.
99. Id.
100. Id.
101. Id. at 851-52.
102. Paracelsus, a Renaissance physician and alchemist, first articulated the maxim: “All things are poisonous, for there is nothing without poisonous qualities. It is only the dose which makes the thing a poison.” Scott P. DeVries, *Effective Cross-Examination of Plaintiffs' Toxicologists and Immunologists in Mass Toxic Tort Litigation*, 4 TOXIC L. REP. (BNA) 818, 819 (1989).
103. Id.
be true, the issue should not be whether one molecule of a carcinogen can cause cancer or whether exposure to such a molecule causes some increased risk, but rather, whether that risk is legally significant. This issue, unresolved by the “one-hit” theory, is crucial when the numbers themselves are considered.

[T]here are a lot of numbers for increased risk that are greater than zero but that no one in their right mind would consider “significant.” Three examples of such numbers would be one octillionth, two octillionths, and three octillionths. Even jumping many orders of magnitude closer to 1.0 (certainty)—all the way to, say, a one-in-a-trillion increased lifetime risk of incurring cancer from a given dose of chemicals—one still is surely in the range of risk that is insignificant as a matter of law . . . , since a’ one-in-a-trillion risk equates to a risk that one extra person somewhere on earth exposed to such a dose will incur cancer sometime in the next 100 generations of humans, assuming that the global population approximately triples to a figure of fifteen billion by about the year 4,500 A.D. It seems indisputable that a plaintiff’s expert who is unable or unwilling to quantify the greater-than-zero increased risk implied by the “one-hit” theory cannot possibly claim to know whether the value for such alleged increased risk lies above or below the level of legal insignificance, whether that level be one in a trillion, or some higher or lower number.¹⁰⁶

Despite the logical and legal problems inherent in the “significant but unquantifiably enhanced risk” standard, the Maryland Court of Appeals has adopted this standard for use in asbestos cases.¹⁰⁷ There is no reason

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¹⁰⁶. *Endicott*, supra note 104, at 408-09. Endicott includes, in a footnote, a revealing example of how the “one-hit” theory can be attacked in a courtroom:

> When we pressed plaintiffs' toxicologists on cross-examination to quantify the plaintiffs' additional risk of cancer, they responded that quantification was not possible. Pressed further, they admitted they could not say whether the added risk was more or less than one-in-a-million. As every one of us has a one-in-three chance of contracting cancer in his or her lifetime, an added risk of one-in-a-million merely increases the total risk from \(0.333333\) to \(0.333334\), hardly anything worthy of note. Placing the \(0.333333\) on the blackboard and then erasing the last 3 and replacing it with a 4 was embarrassing to the plaintiffs' witness.


¹⁰⁷. *Eagle-Picher*, 604 A.2d at 459 (“The defendants' medical expert also believed that
to believe that the court would not also endorse the standard in other hazardous substances cases.

3. Product Identification

Before a plaintiff can recover damages for exposure to a hazardous substance, the plaintiff must be able to identify the hazardous substance to which he or she was exposed. Indeed, product identification has been described as "[t]he threshold requirement of any products liability action." In *McClelland v. Goodyear Tire & Rubber Co.*, Judge Smalkin of the United States District Court for the District of Maryland held that the plaintiffs, who had sued Goodyear for its allegedly tortious conduct in furnishing various toxic chemicals to Kelly Springfield, their employer, could not prevail as a matter of law. Magistrate Chasanow reviewed the parties' filings and concluded that plaintiffs presented no evidence that their injuries were caused by any particular, identifiable, Goodyear-supplied chemical. Judge Smalkin agreed:

Plaintiffs' attempts to hold Goodyear responsible, under product liability theories, for the total "toxic soup" they claim existed in their workplace, without the ability to adduce competent evidence that a reasonable fact-finder could view as showing a greater than 50% chance of a causal connection between any such product and the specific injuries they allegedly suffered, must fail. Maryland has not adopted the "market-share" theory of product liability, of which the plaintiffs here assert a microcosmic variant. Thus, the court concludes that the plaintiffs must suffer summary judgment on their product liability claims. In *Eagle-Picher Industries, Inc. v. Balbos*, a plaintiff attempted to establish the liability of an installer of asbestos products (the Porter Hayden Company, or "Porter") by proving that the installer was "almost an exclusive distributor" for Johns-Manville ("Manville") products, which contained asbestos. Although the plaintiff was able to show both that Porter sold Manville products and that the plaintiff's place of employment purchased Manville products, the court held that more direct evi-

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dence was required. This ruling reversed the Maryland Court of Special Appeals which had held that the jury reasonably could have inferred that "any Johns-Manville products identified at [plaintiffs place of employment] were Porter Hayden products."112

In Eagle-Picher, the Court of Appeals of Maryland also rejected the "fiber drift" theory as a possible theory of exposure.

"The 'fiber drift theory' as it is described by the plaintiffs here takes as its starting point that asbestos fibers may become airborne or re-entrained and thus be carried from their source to other areas. Under this theory, however, both the specific locale of the product's use and the specific areas of the plaintiff's employment become irrelevant. The substance of the fiber drift theory is that once an asbestos-containing product can be placed anywhere in the Firestone plant, any plaintiff working at any point within that plant is entitled to have the question of causation submitted to the jury because it is likely, given that fibers can drift, that a given plaintiff was exposed to fibers originating in a particular defendant's product." So extremely attenuated is causation in fact under the 'fiber drift theory' that it is inconsistent with the requirement of Maryland law that an actor's negligence be a substantial factor in causing the injury.113

Although the court of appeals did not cast its holding in terms of product identification, the logic of this portion of the Eagle-Picher case requires that a plaintiff be able to prove with specificity that a particular manufacturer's product caused his or her injury.

4. Proof of Exposure

Before deciding whether or not an identifiable hazardous substance was a substantial factor in causing plaintiff's injuries, proof of exposure to the hazardous substance is required. "Exposure . . . may be established circumstantially."114 Whether exposure, no matter how it is established, is sufficient to permit a jury to find substantial factor causation is a question specific to the facts of each individual case.

The finding involves the interrelationship between the use of a defendant's product at the workplace and the activities of the plaintiff at the workplace. This requires an understanding of the

113. Eagle-Picher, 604 A.2d at 463 (quoting Robertson v. Allied Signal, Inc., 914 F.2d 360, 376 (3d Cir. 1990)).
114. Eagle-Picher, 604 A.2d at 460 (citation omitted).
physical characteristics of the workplace and of the relationship between the activities of the direct users of the product and the bystander plaintiff. . . . Within that context, the factors to be evaluated include the nature of the product, the frequency of its use, the proximity in distance and time of a plaintiff to the use of a product, and the regularity of the exposure of that plaintiff to the use of that product.115

In Eagle-Picher, the court of appeals provided several examples of cases in which exposure was established. The Eagle-Picher court held that where a plaintiff could establish that he had worked in an engine room (the area of the most dense concentration of asbestos) for six months in the vicinity of asbestos installers who used “tons” of defendant's cement, exposure was proved.117

5. Expert Testimony

In order to prove causation, a plaintiff must offer the testimony of at least one expert witness. The rest of this Article discusses the varying theories of proof with which the expert should be familiar, the threshold standard for admitting the expert’s testimony into evidence, the various fields of expertise, and the types of studies relied on by experts in the field of hazardous substance exposure.

IV. Theories of Proof

Scientists and lawyers think very differently about the issue of proof of causation. Although the legal standard clearly applies in the legal setting, and although expert witnesses should be familiar with legal, as opposed to scientific, theories of proof of causation, the contrast between the two disciplines can lead to a lack of clarity in the testimony. A basic understanding of the theoretical underpinnings of the two theories of causation can help to dispel any confusion that might arise.118

The paradigmatic scientific method uses inductive reasoning, making a generalized conclusion from specific empirical observation. Under this paradigm,

knowledge is gained by attempting to disprove or falsify a hy-

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115. Id. at 460 (citations omitted).
116. Id. at 461.
117. Id.
118. A full treatment of this complex issue is beyond the limited scope of this Article. For a fuller discussion of these issues, see Brennan, supra note 2. This section of the Article owes a great deal to Brennan's article.
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... hypothesis based on empirical investigation. Scientific methodology today is based on generating hypotheses and testing them to see if they can be falsified; indeed, this methodology is what distinguishes science from other fields of human inquiry. Theoretically, therefore, hypotheses are not affirmatively proved, only falsified. Of course, if a hypothesis repeatedly withstands falsification, one may tend to accept it, even if conditionally, as true.119

By contrast, the law uses primarily deductive, or empirical, reasoning, for example, a negligent act that causes damage results in liability. Of course, there are an infinite number of "causes" for damage. For example, if X is driving a car and, through his negligence, strikes Y, X's negligence is the cause of Y's injury. However, the fact of the manufacture of X's car is also a "cause" of Y's injury, even though that manufacture was non-negligent, as is the fact that X's parents conceived him. Indeed, under this rationale, Y's conception was also a "cause" of his injury.120

No one would advocate extending liability to all who were involved because each was a potential "cause" of Y's damages. In order to limit the potentially infinite causal chain that could stretch out from each alleged tort, the law developed the concept of "proximate cause."121 Under this concept, the factfinder must determine if the alleged conduct was "so significant and important a cause that the defendant should be legally responsible."122

Although inductive theories of proof work well for scientists, and deductive theories of proof work well for lawyers, problems develop when the two concepts are forced to intersect in the court room. Viewed from an inductive, or scientific, standpoint, it is almost nonsensical to speak in

120. The reductio ad absurdum of the causative chain analysis in the law is found in Palsgraf v. Long Island R.R., 162 N.E. 99 (N.Y. 1928).
121. The word "proximate" is a legacy of Lord Chancellor Bacon, who in his time committed other sins. The word means nothing more than near or immediate; and when it was first taken up by the courts it had connotations of proximity in time and space which have long since disappeared. It is an unfortunate word, which places an entirely wrong emphasis upon the factor of physical or mechanical closeness. For this reason "legal cause" or perhaps even "responsible cause" would be a more appropriate term. There is, however, no present prospect that long ingrained practice will ever be altered by the substitution of either.
122. Id.
terms of “reasonable scientific certainty.” What is reasonable? What is certain? These are not scientific terms. It may be reasonable to say that particular results point towards a possible conclusion, but is this reasonable certainty? On the other hand, lawyers have little time for scientific doubt—either a particular dose of a chemical is toxic or it is not. The law requires, if not certainty, then reasonable probability, and cannot, or should not, indulge itself by living in a state of perpetual doubt.123

The problem with this pragmatic approach to issues of proof of causation comes when experts testify about matters where no reliable evidence points to a strong association between a product and an injury. For example, an expert who testifies that a plaintiff has a significant but unquantifiable increase in risk of cancer because of exposure to a hazardous substance arguably meets neither the scientific nor legal standard necessary to support the plaintiff’s case. In such a case, “there is a risk that the jury would make an irrational finding of causation based upon the siren-like allure of opinions stated by highly qualified experts.”124 Accordingly, the importance of a threshold determination of the admissibility of an expert’s testimony is crucial to a fair determination of liability.

123. Judge Joseph H. Young of the federal bench attempted to resolve this “proof” dichotomy.

Evidence or absolute scientific “certainty” may be impossible for either plaintiffs or defendant to obtain in a products liability case of this nature, as it is difficult to isolate one of a variety of factors which may have caused an injury. Further, proof of causation must be presented in scientific and medical terms, which may be misinterpreted in a legal setting. For example, reliable evidence of a strong association between a product and an injury may lead a reasonable juror to conclude that a causal relationship is “more probable than not,” even though the expert witness never mentioned causation and only testified to an “association.” Nevertheless, when presenting evidence of an association, just as with any other expert testimony, there must be some basis beyond mere theory and speculation for the opinion.


An expert can be found to testify to the truth of almost any factual theory, no matter how frivolous, thus validating the case sufficiently to avoid summary judgment and force the matter to trial. At the trial itself an expert’s testimony can be used to obfuscate what would otherwise be a simple case. The most tenuous factual bases are sufficient to produce firm opinions to a high degree of “medical (or other expert) probability” or even of “certainty.” Juries and judges can be, and sometimes are, misled by the expert-for-hire.

A. The Continued Viability of the Frye/Reed Test

The evidentiary world of the federal courts was thrown into confusion by the Supreme Court in the case of *Daubert v. Merrell Dow Pharmaceuticals, Inc.* Maryland has avoided such trauma, and there is no indication that the court of appeals will reject the familiar standard for the admissibility of expert testimony first enunciated in *Frye v. United States,* and adopted in Maryland in *Reed v. State.*

The Reed standard requires an expert's methodology to be "generally accepted as reliable within the expert's particular scientific field" to be admissible. This "methodology" standard has been criticized, but in Maryland it is unlikely to be altered.

The *Daubert* standard, by contrast, requires the court to engage in what the United States Court of Appeals for the Ninth Circuit has termed: a difficult, two-part analysis. First, we must determine nothing less than whether the experts' testimony reflects "scientific knowledge," whether their findings are "derived by the scientific method," and whether their work product amounts to "good science." . . . Second, we must ensure that the proposed expert testimony is "relevant to the task at hand, . . . " i.e., that it logically advances a material aspect of the proposing party's case. There is a "common perception that *Daubert* liberalized the standard for admission of expert testimony." Surprisingly, more than half the cases

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126. 293 F. 1013 (D.C. Cir. 1923).
127. 391 A.2d 364 (Md. 1978). See Keene Corp. v. Hall, 626 A.2d 997, 997 n.2 (Md. Ct. Spec. App. 1993) ("[T]here is nothing in Maryland case law . . . that indicates that the Court of Appeals is considering [a move to the *Daubert* standard]."). *Keene* also formally resolved the issue of Reed's applicability in a civil case in the affirmative. *Id.* at 1002.
128. Reed, 391 A.2d at 368.
130. This standard also can prove difficult for the ill-prepared expert as the following question and answer illustrate:
Q: "Do you have a methodology or protocol for reaching determinations as to whether a particular agent is a human teratogen?"
A: "No."
Smith v. Ortho Pharm. Corp., 770 F. Supp. 1561, 1580 n.72 (N.D. Ga. 1991), quoted in Endicott, supra note 129, at 291 n.3. Summary judgment was granted based on this answer. *Id.*
131. Daubert v. Merrell Dow Pharm., Inc., 43 F.3d 1311, 1315 (9th Cir. 1995) (citations omitted). The first prong of the *Daubert* test is, in essence, the *Frye* test restated.
involving the admissibility of expert testimony reported during the year after Daubert was decided excluded the challenged expert testimony.\(^\text{133}\)

Although the Daubert standard has not been adopted in Maryland, at least one Maryland court has utilized a Daubert-type analysis in a case involving MCSS. In Bready v. G.D. Armstrong,\(^\text{134}\) Judge Durke G. Thompson was confronted with a motion in limine from defendants seeking to bar reference to MCSS as well as the testimony of two doctors who proposed to testify that the plaintiff suffered from MCSS. The plaintiff's attorney requested that the court accept the Daubert criteria.\(^\text{135}\) The defendant agreed that although the Frye/Reed test was still valid in Maryland, the Daubert factors might be considered by the court in a case involving cutting-edge science.\(^\text{136}\)

Judge Thompson resolved the dispute by finding that the Frye standard had not been abandoned in Maryland.\(^\text{137}\) Judge Thompson stated that “it is appropriate to incorporate the Frye rule into what the court finds is a more useful functional rule as encompassed by the Dalbert [sic] Supreme Court ruling.”\(^\text{138}\) The court held that under either test, however, there was insufficient agreement within the scientific community to permit tes

\(^{133}\) Id.


\(^{135}\) Mr. Nelson [for the plaintiff]: “I think what Dalbert [sic.] is really driving at is they are not going to permit a witness — I mean, basically under the Frye — under the old rule, the very conservative rule, the courtroom was always a generation or half a generation behind the science. And what Dalbert [sic] is saying is, well, that is not really fair; is it? If someone is injured and they can prove it?” Transcript of Motions Hearing, April 14, 1995, at 9-10.

\(^{136}\) Id. at 13. There was some dissension between defense counsel on this point. “[T]o answer the Court's question, I think that the standard that the Court has to apply currently is the Frye/Reed standard” Id. at 22.

\(^{137}\) Judge Thompson's lesson is confirmed by the Maryland Rules of Evidence adopted by the court of appeals on December 15, 1993, effective in Maryland since July 1, 1994. Rule 5-702, “Testimony by Experts,” provides that:

- Expert testimony may be admitted in the form of an opinion or otherwise, if the court determines that the testimony will assist the trier of fact to understand the evidence or to determine a fact in issue. In making that determination, the court shall determine (1) whether the witness is qualified as an expert by knowledge, skill, experience, training, or education, (2) the appropriateness of the expert testimony on the particular subject, and (3) whether a sufficient factual basis exists to support the expert testimony.

The Committee Note to Rule 5-702 makes clear that the rule is not intended to overrule the Frye/Reed test. “The required scientific foundation for the admission of novel scientific techniques or principles is left to development through case law.” Committee Note to Rule 5-702, cf., Reed v. State, 283 Md. 374 (1978), with Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579 (1993).

\(^{138}\) Id. at 31.
testimony about MCSS and its causal connection to the plaintiff's claims of damages. Accordingly, the court granted the defendant's motion in limine.\textsuperscript{139}

The lesson of \textit{Bready} appears to be that, while \textit{Frye/Reed} is still the appropriate evidentiary standard in Maryland, attorneys will raise \textit{Daubert} in an attempt to persuade courts that cutting-edge type evidence should be admitted. Because \textit{Daubert}, arguably, incorporates \textit{Frye/Reed}, it is possible for a court to make a ruling that combines the two standards if the final result is the exclusion of the proposed testimony. Admitting testimony under \textit{Daubert} that would be inadmissible under \textit{Frye/Reed}, however, is a practice which currently has no support under Maryland law.

\textbf{B. Areas of Expertise}

Courts are confronted with an array of separate areas of expertise in cases involving exposure to hazardous substances. The most commonly offered areas of expertise, however, are epidemiology and toxicology.

\textbf{1. Epidemiology}

The ultimate goal of epidemiology is:

to draw a biological inference concerning the relationship of [a] factor [for example, a toxic substance] to [a] disease's etiology and/or its natural history. Stated more formally, "epidemiology can be regarded as a sequence of reasoning concerned with biological inferences derived from observations of disease occurrence and related phenomena in human population groups."\textsuperscript{140}

Epidemiologists\textsuperscript{141} have a set of criteria that must be satisfied prior to

\textsuperscript{139} \textit{Id.} at 35-36.


\textsuperscript{141} An epidemiologist often is referred to as a doctor who can count. In fact, however, a medical degree is not necessary for expertise in the area of epidemiology. Black & Lilienfeld, \textit{supra} note 146, at 776. (Although more than 10 years old, this article is still an invaluable source for information on the relationship between epidemiology and litigation. For another, more recent, resource intended specifically for judges, \textit{see} \textit{FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE} (1994). Although emphasizing
an inference that an agent is the "etiologic agent of a given disease." These criteria, the Henle-Koch-Evans Postulates, for the two scientists who developed them in the nineteenth century, and Evans, who revised them in 1976, form the backbone of every epidemiological inference.

An epidemiologist now can draw an inference regarding the etiological relationship between an agent and a disease after satisfying the Henle-Koch-Evans Postulates. This alone is not sufficient to permit the epidemiologist to testify at a trial involving hazardous substance exposure. To testify about hazardous exposure, the epidemiologist must determine if the risk attributable to the hazardous substance (the factor) is greater

\[ \text{Daubert, this publication contains excellent reference guides on epidemiology, toxicology, survey research, forensic DNA evidence, statistics, multiple regression, and the estimation of economic losses in damages awards).} \]

142. Black & Lilienfeld, \textit{supra} note 140, at 762.
143. \textit{Id.} at 763. The Henle-Koch-Evans Postulates are as follows:

1. The prevalence rate of the disease should be significantly higher in those exposed to the hypothesized cause than in controls not so exposed (the cause may be present in the external environment or as a defect in host responses).
2. Exposure to the hypothesized cause should be more frequent among those with the disease than in controls without the disease when all risk factors are held constant.
3. Incidence of the disease should be significantly higher in those exposed to the cause than in those not so exposed, as shown by prospective studies.
4. Temporally, the disease should follow exposure to the hypothesized causative agent with the distribution of incubation periods on a log-normal-shaped curve.
5. A spectrum of host responses should follow exposure to the hypothesized agent along a logical biologic gradient from mild to severe.
6. A measurable host response following exposure to the hypothesized cause should have a high probability of appearing in those lacking this response before exposure (e.g., antibody, cancer cells) or should increase in magnitude if present before exposure; this response pattern should occur infrequently in persons not so exposed.
7. Experimental reproduction of the disease should occur more frequently in animals or man appropriately exposed to the hypothesized cause than in those not so exposed; this exposure may be deliberate in volunteers, experimentally induced in the laboratory, or demonstrated in a controlled regulation of natural exposure.
8. Elimination or modification of the hypothesized cause or of the vector carrying it should decrease the incidence of the disease (e.g., control of polluted water, removal of tar from cigarettes).
9. Prevention or modification of the host’s response on exposure to the hypothesized cause should decrease or eliminate the disease (e.g., immunization, drugs to lower cholesterol, specific lymphocyte transfer factor in cancer).
10. All of the relationships and findings should make biological and epidemiologic sense.

\textit{Id.} (footnote omitted).
than fifty percent (also expressed by epidemiologists as .50).\textsuperscript{144}

Epidemiological evidence has been deemed "[u]ndoubtedly . . . the most useful and conclusive type of evidence in a [toxic exposure] case."\textsuperscript{145} Epidemiological studies are regarded by the Environmental Protection Agency as "the only unreservedly persuasive evidence of causal associations between an agent and human health effects."\textsuperscript{146}

Despite the value of epidemiological evidence, however, it does not provide an unimpeachable or foolproof method of establishing causation.

Among the problems encountered by plaintiffs seeking to use [epidemiological] data is that [such] studies provide information regarding trends in groups rather than the occurrence of a disease in a particular plaintiff. . . . The study groups often involve levels of exposure to a substance far in excess of the level at which plaintiffs were exposed, possibly rendering the results of the study non-predictive or irrelevant.\textsuperscript{147}

Perhaps the most significant defects in epidemiological studies are "biases" which can invalidate a study's validity.\textsuperscript{148} Some of the most common "biases" are: selection bias ("the exposed group is selected in a way that makes it more or less [prone] to disease for reasons [unrelated to] exposure");\textsuperscript{149} diagnostic bias (when the disease is misdiagnosed); recall bias (the failure of subjects to recollect accurately details of exposure); and, the presence of unaccounted for confounders (an unrelated factor

\textsuperscript{144} Id. at 767.

Conceptually, the finder of fact must decide whether it is more likely than not that an individual plaintiff contracted a specific disease as a result of exposure to a factor for which the defendant is legally responsible. From an epidemiological perspective, the question has two parts: (1) is the factor causally related to the disease (satisfaction of the Henle-Koch-Evans Postulates), and (2) is the attributable risk greater than .50? If, in an exposed population, more than half the cases of a disease can be attributed to the exposure, and if the postulates are satisfied, then absent other information about a diseased individual, it is more likely than not that his or her illness was caused by the exposure.

\textit{Id.} (footnote omitted). Black and Lilienfeld continue with a hypothetical example of the practical application of this so-called "evidentiary test." \textit{Id.} at 767-68.

\textsuperscript{145} Brock v. Merrell Dow Pharm., Inc., 874 F.2d 307, 311 (5th Cir. 1989), modified, 884 F.2d 166 (5th Cir. 1989). \textit{See also} Terrell v. United States, 517 F. Supp. 374, 379 (N.D. Tex. 1981) ("Epidemiological evidence is critical in discovering the etiology, or cause of diseases, particularly those which may have numerous causes.").

\textsuperscript{146} Whitehead & Espel, supra note 140, at 1043.


\textsuperscript{149} Green, supra note 119, at 649.
that nonetheless correlates with a greater or lower disease rate).\textsuperscript{150}

The importance of epidemiological studies, and the favor in which they are generally held by the courts, can have a detrimental effect on a plaintiff who cannot produce a supporting epidemiological study.\textsuperscript{151} Several plaintiffs have resorted to offering the re-analysis of already existent epidemiological data in an attempt to overcome this perceived proof problem. The practice of re-analysis, however, has received little support in the courts under the \textit{Frye} standard.\textsuperscript{152}

Epidemiological testimony should not be viewed as a panacea to all problems posed by causation problems in toxic litigation. Although a valuable tool under certain circumstances, and occasionally the only scientific evidence of causation that can be offered, problems of bias and methodology almost always will surface and be exploited by defense experts. A case that relies exclusively on epidemiological evidence for proof of causation is dangerously exposed to a strong defense attack.

2. \textit{Toxicology}

Toxicology is "a science that deals with poisons and their effect[s]."\textsuperscript{153} Toxicologists typically are used by plaintiffs to prove that the chemicals to which the plaintiffs were exposed were toxic, and that the chemicals were the source or exacerbating factor in the symptoms of which they complain.\textsuperscript{154} Toxicologists may use various studies upon which to base their opinions, including cluster analysis, short term molecular assays, and animal bioassays.\textsuperscript{155}

Cluster analysis studies "diseases shared by members of a group exposed to a single hazardous substance."\textsuperscript{156} An example of this form of analysis is Pott's discovery of an association between scrotal cancer and

\textsuperscript{150} Id. at 649-51.
\textsuperscript{151} Epidemiological studies are often lengthy, hence expensive, undertakings. In addition, the necessary data simply might not exist.
\textsuperscript{152} See, e.g., Brock v. Merrell Dow Pharm., Inc., 874 F.2d 307, 313-15 (5th Cir. 1989) (reanalysis of epidemiological studies generally accepted by the scientific community only when subjected to verification and scrutiny by others in the field); Richardson v. Richardson-Merrell, Inc., 857 F.2d 823, 830-31 (D.C. Cir. 1988). Although the issue of reanalysis of epidemiological data was raised by the appellant in \textit{Keene Corp.}, 626 A.2d 966 (Md. App. 1993), the court of special appeals held that the expert in that case merely summarized the epidemiological data and did not reanalyze it. Id. at 1005.
\textsuperscript{153} \textit{WEBSTER'S THIRD NEW INTERNATIONAL DICTIONARY} 2419 (1986).
\textsuperscript{154} DeVries, \textit{supra} note 102, at 819.
\textsuperscript{155} Brennan, \textit{supra} note 2, at 502. Brennan also identifies epidemiological studies as a fourth method for identifying carcinogens. These were discussed \textit{supra} note 2.
\textsuperscript{156} \textit{Id.}
chimney sweeping. Although cluster analysis is not considered to be an epidemiological study, it is however, open to the same problems of bias and confounders faced by epidemiologists.

Short term molecular assays test potentially carcinogenic substances. Substances that “pass” are deemed “non-carcinogenic.” Those substances that fail are then studied further.

Brennan acknowledges that the science of short term assay research is still being developed, and questions remain as to how best to combine the tests to identify the substances that are mutagens, and thus presumably carcinogens.

The fundamental assumption underlying animal assays is that substances that are carcinogenic in animals also will be carcinogenic in humans. These studies are typically conducted with a control group and a group intentionally exposed to the hazardous substance. The results between the two groups are then compared, and “[t]he amount by which the experimental results differ from the null hypothesis [a researcher’s initial assumption that the hazardous substance will have no effect] can be summarized in a ‘trend test statistic.’”

A mutation is an abrupt and heritable genetic change. Thus a mutation is any change in the genetic material of the cell that is passed to following generations. This may mean a change in a single nucleotide (individual molecule of DNA), in several nucleotides within the same gene (a functional group of DNA molecules within a chromosome), or in an entire chromosome. Mutations may change a normal gene into a mutant (forward mutation) or a mutant gene into a normal one (reverse mutation).

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157. Hermo, Chemical Carcinogenesis: Tumor Initiation and Promotion, 2 OCCUPATIONAL MED. 1, 6 (1987), cited in Brennan, supra note 2, at 502 n.177.
158. See notes 148-50 and accompanying text.
159. Brennan, supra note 2, at 503.
160. Short-term assays are based on certain molecular theories of carcinogenesis regarding the disruption of deoxyribonucleic acids (DNA). Similar disruption is thought to occur in mutagenesis. Therefore, attention has long focused on the correlation of carcinogenesis with mutagenesis.

A mutation is an abrupt and heritable genetic change. Thus a mutation is any change in the genetic material of the cell that is passed to following generations. This may mean a change in a single nucleotide (individual molecule of DNA), in several nucleotides within the same gene (a functional group of DNA molecules within a chromosome), or in an entire chromosome. Mutations may change a normal gene into a mutant (forward mutation) or a mutant gene into a normal one (reverse mutation).

Id. (footnote omitted).
161. Id. at 504 (emphasis added) (footnote omitted).
162. Although this is the “gold standard” for human epidemiological studies as well, for obvious ethical reasons, this form of research is impossible in hazardous substance research.
163. Brennan, supra note 2, at 505.
The trend test statistic can be converted into a p-value, or probability value, defined as:

the probability of observing an apparent effect of exposure as great or greater than that actually found if chance alone were responsible for any apparent effects seen in the data. Statistical significance is usually arbitrarily defined as a p-value of less than .05; that is, chance alone can explain the results of the experiment only five percent of the time.164

If a substance is shown to be carcinogenic in animals, the scientist assumes that it is carcinogenic in humans and attempts to calculate the dose necessary to provoke a cancer response in humans, using mathematical models.165

The problems with such animal tests in the litigation context are readily apparent. The degree of abstraction in this form of study—from the initial assumption that a substance that causes cancer in animals will also cause cancer in humans,166 through the calculation of p-values, to the estimation of a toxic dose in humans using mathematical models that are highly malleable—is high. Animal studies are slender reeds upon which to base a claim for damages.

Indeed, several courts have excluded testimony regarding animal studies.167 Furthermore, although the Environmental Protection Agency ac-

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164. Id. Brennan describes the conversion from trend test statistic to p-value as assuming that "the trend statistics from an infinite number of identical bioassays would simulate a normal distribution—meaning that they would resemble a bell-shaped curve if graphed with one axis representing the probability of occurrence, and the other representing the trend statistic itself." Id. at 505 n.191.

165. "Mathematical depiction of the process by which an external does more through various compartments in the body until it reaches the target organ is often called physiologically based pharmacokinetics. . . . [R]eliance on in vitro data for elucidating mechanisms of toxicity is more pervasive where positive human epidemiological data also exist." Reference Manual on Scientific Evidence, supra note 161, at 192.

166. This is a particularly dangerous assumption when the animal in question, for example the B6C3F1 mouse, has been bred for the specific purpose of developing cancer. DeVries, supra note 102, at 820.


There is no evidence that plaintiffs were exposed to the far higher concentrations involved in both the animal and industrial exposure studies. The animal studies are not helpful in the instant case because they involve different biological species. They are of so little probative value as to be inadmissible. They cannot be an acceptable predicate for an opinion under Rule 703.

Id. Lynch v. Merrell-Nat'l Lab., 646 F. Supp. 856, 865-66 (D. Mass. 1986) ("There are several limitations inherent in the use of the animal and other experimental data relied upon here by the plaintiffs, and these limitations have been conceded in prior testimony by plaintiffs' witnesses.") The court concluded that the studies were inadmissible because
cepts animal data as evidence of carcinogenity, even in the absence of epidemiological studies, the agency's decision is not significant in the litigation context.\textsuperscript{168}

V. Conclusion

The challenges facing the courts from hazardous substance litigation show no signs of abating, as experts in new areas of expertise are offered by attorneys seeking to recover for, or defend against, new legal theories spawned by scientific discoveries. Faced with the rapid pace of progress in the area of exposure to hazardous substances, courts must continue to evaluate the theories of recovery offered by attorneys, and the modes of proof of causation, according to well-established standards, neither rewarding parties for ingenuity without substance nor punishing them for creativity and innovation.\textsuperscript{169}

\textsuperscript{168} Commentators have noted that the EPA's decision is based on regulatory policy, and not "scientific evidence." Whitehead & Espel, \textit{supra} note 140. The reasons for this interpretation flow from the EPA's choice of language. By accepting animal studies in the absence of epidemiological studies, the EPA acknowledges that it cannot exclude bias or other confounding factors as a possible cause for the carcinogenic effect. "This position means that EPA assumes its answer to the question of probability. Such a regulatory assumption does not establish proof in tort litigation." \textit{Id.}

\textsuperscript{169} For an encyclopedic treatment of litigation and hazardous substances, see \textsc{Lawrence G. Cetrullo}, \textsc{Toxic Torts: A Complete Personal Injury Guide} (1993).