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EPIDEMIOLOGIC PROOF IN TOXIC TORT LITIGATION

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INTRODUCTION

TOXIC tort[1] litigation has emerged as a major social and legal concern,[2] a development that has engendered numerous proposals for legal reform. Many of these reforms would require institutional

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1. This Article loosely defines toxic tort cases as those in which the plaintiff seeks compensation for harm allegedly caused by exposure to a substance that increases the risk of contracting a serious disease, but does not cause an immediately apparent response. These cases generally involve a period of latency or incubation prior to the onset of the disease. In most cases the increased risk of the disease does not diminish or dissipate, even with the cessation of exposure. The Article discusses exposure to radiation as well as to chemicals, and considers some cases involving drugs because many of the causation issues are similar to those in environmental or occupational cases. It also considers birth defect cases. The vast majority of toxic tort cases, however, are related to cancer and the issue of carcinogenesis, and thus, parts of this Article focus only on cancer and its causes.

2. One commentator notes that "[e]ven without a crystal ball, it is easy to see a wave of cancer litigation on the horizon." Shelton, *Defending Cancer Litigation: The Causation Defense*, For The Defense, January 1982, at 8, 14. Another cites asbestos litigation as indicative of the trend, and points out that "there are more than 15,000 asbestos related cases now pending, and additional cases are being filed at the rate of over 400 each month; it has been estimated that over 30,000 additional suits will be filed in the next 25 years." Olick, *Chapter 11—A Dubious Solution To Massive Toxic Tort Liability*, 18 Forum 361, 361 (1983). Also part of the trend are claims brought by people alleging harm from exposure to dioxin. *See* Long & Hanson, *Dioxin Issue Focuses on Three Major Controversies in U.S.*, Chem. & Eng'g News, June 6, 1983, at 23, 24. One accident involving dioxin at a West Virginia chemical plant has resulted in claims totaling 700 million dollars. Webber, *Dioxin Liability is Huge Problem for Companies, Courts*, Chem. & Eng'g News, June 6,

innovations, such as administrative funds from which claimants could obtain compensation with relatively little evidence of causation.[3] Most, however, would also allow recovery under existing tort theories.[4] Thus, questions about the application of common law principles in evaluating evidence of causation in toxic tort cases remain open.[5]

1983, at 57, 59. For other examples, see Note, *Establishing Causation in Chemical Exposure Cases: The Precursor Symptoms Theory*, 35 Rutgers L. Rev. 163, 164 n.2 (1982) [hereinafter cited as *Precursor Symptoms*].

3. *See, e.g.*, Ginsberg & Weiss, *Common Law Liability for Toxic Torts: A Phantom Remedy*, 9 Hofstra L. Rev. 859, 928-40 (1981); Milhollin, *Long-Term Liability for Environmental Harm*, 41 U. Pitt. L. Rev. 1, 16-25 (1979); Trauberman, *Statutory Reform of "Toxic Torts": Relieving Legal, Scientific and Economic Burdens on the Chemical Victim*, 7 Harv. Envtl. L. Rev. 177, 237, 243 (1983).

Perhaps the best known proposals for changes in the law are those made by the Superfund Section 301(e) Study Group (Study Group), which was appointed pursuant to Section 301(e) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, Pub. L. No. 96-510, 94 Stat. 2767 (1980) (codified at 42 U.S.C. § 9601 et seq. (Supp. V 1981)). The Study Group submitted a report to Congress in September 1982 that recommended the creation of rebuttable presumptions of causation to facilitate access to an administrative victim compensation fund. "Superfund Section 301(e) Study Group," 97th Cong., 2d Sess., Injuries and Damages From Hazardous Wastes—Analysis and Improvement of Legal Remedies—Report to Congress in Compliance with Section 301(e) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (P.L. 96-510) 213-25 (Comm. Print 1982) [hereinafter cited as 301(e) Study].

4. The only proposal of which the authors are aware that would eliminate tort law in the area of toxic exposure litigation is that of Ginsberg and Weiss. *See* Ginsberg & Weiss, *supra* note 3, at 932.

5. The 301(e) Study recommended the creation of rebuttable presumptions of causation in favor of plaintiffs seeking compensation from an administrative fund. See *supra* note 3. While the Study did not recommend that its presumptions carry over to tort actions, neither did it recommend against such a step. 301(e) Study, *supra* note 3, at 260. Some commentators have affirmatively proposed this. *See, e.g.*, Burcat, *Uncompensated Victims of Low-Level Radiation: Unnecessary Hostages of the Price-Anderson Act Debate*, 15 Forum 847, 859 (1980); Delgado, *Beyond* Sindell: *Relaxation of Cause-In-Fact Rules for Indeterminate Plaintiffs*, 70 Calif. L. Rev. 881, 899 (1982); Note, *The Inapplicability of Traditional Tort Analysis to Environmental Risks: The Example of Toxic Waste Pollution Victim Compensation*, 35 Stan. L. Rev. 575, 615 (1983) [hereinafter cited as *Environmental Risks*]; Note, *Tort Actions for Cancer; Deterrence, Compensation, and Environmental Carcinogenesis*, 90 Yale L.J. 840, 855 (1981) [hereinafter cited as *Tort Actions for Cancer*]. Other commentators have suggested evidentiary standards that are stacked in favor of plaintiffs though not couched in terms of presumptions. *See, e.g.*, Hall & Silbergeld, *Reappraising Epidemiology: A Response to Mr. Dore*, 7 Harv. Envtl. L. Rev. 441, 444-45 (1983); *Precursor Symptoms*, *supra* note 2, at 189-90.

Some commentators have proposed proportional liability as an alternative to the traditional all-or-nothing recovery approach. *See, e.g.*, Delgado, *supra*, at 899-902; Rizzo & Arnold, *Causal Apportionment in the Law of Torts; An Economic Theory*, 80 Colum. L. Rev. 1399, 1407-13 (1980); Robinson, *Multiple Causation in Tort Law: Reflections on the DES Cases*, 68 Va. L. Rev. 713, 755-58 (1982); Rosenberg, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System*, 97 Harv. L. Rev. 849, 881-87 (1984). The theory underlying these proposals

This Article focuses on the use of the traditional preponderance-of-the-evidence standard of proof in toxic tort cases in which a single substance is at issue.[6] Courts have found it difficult to apply this standard to the kind of evidence seen in toxic tort litigation, and as a result, have sometimes allowed recovery based on highly suspect evidence,[7] or conversely, have failed adequately to justify the exclusion of evidence.[8]

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has not been developed fully, however, and such a reform may be inappropriate for single-factor cases. Multi-factor cases may provide stronger justification for proportional liability, but its rational use would require the application of evidentiary tests very similar to the one proposed in this Article. See *infra* pt. V(B).

6. While many cases involve allegations that a number of substances combined to cause a plaintiff's disease, the analysis in this Article is confined to cases in which a plaintiff is exposed to a single identifiable substance and subsequently contracts a disease. The disease is known to arise without the identified exposure, but the plaintiff nonetheless links his or her case to that exposure. Examples include litigation about asbestos, Agent Orange and radiation. Of course, all of these cases involve at least two factors: the substance at issue and whatever other factor(s) (for example, diet or exposure to other substances) are responsible for the background incidence rate of the disease. They are single-factor cases in the legal sense, however, because liability will attach, if at all, to the one identifiable factor.

Cases in which the accused substance allegedly interacted with other factors involve issues not addressed in this Article. Also unaddressed are cases involving two or more identifiable factors, each independently sufficient to cause the injury at issue. When more than one factor is a source of potential liability, however, the epidemiologic concept of attributable risk, upon which this Article is based, still provides the only scientifically valid factual basis for legal analysis. For example, a lung cancer victim exposed to both benzene and cigarettes might be able to attribute 60% of the risk to cigarettes, 20% to benzene, and 20% to unknown factors. These attributable risks are either additive or multiplicative (in lay terms "synergistic"). If the former, the analysis of this Article can be applied with little further elaboration; if the latter, other rules of attribution are required.

7. The full extent of this problem is not revealed by published decisions. Often when a plaintiff with an unsubstantiated claim wins a verdict after presenting very questionable evidence, the defendant will simply settle. The lack of clear standards turns appellate review into a crapshoot with the dice loaded for the plaintiff. A recent example is instructive. In Grasso v. B.F. Goodrich Co., No. 78-1562 (D.N.J. Jan. 30, 1981), the plaintiff alleged that his liver cancer (angiosarcoma) had been caused by vinyl chloride (VC) from a factory located near his home. The expert witness called by the plaintiff to establish this theory of "neighborhood cancer" testified that, in addition to the plaintiff, eight documented cases of angiosarcoma had occurred within two miles of an industrial plant using VC. Trial Transcript at 97, *Grasso*. The expert, however, did not substantiate his conclusion about causation. Although he acknowledged that angiosarcoma could occur without any exposure, *id.* at 182, and that 75% of all cases were of unknown origin, *id.* at 121, he took the position that diagnosis of angiosarcoma and proximity to an emitting VC source would suffice to establish VC as the cause. *Id.* at 177-78. He also seemed unwilling or unable to distinguish between an explanation that is "more likely than not" correct and one that is "the most likely" of several explanations. *Id.* at 112-13. Despite this weak evidence, the jury returned a plaintiff's verdict that the trial court refused to set aside. An appeal was taken, but the case was settled before argument. A clear

These problems can be overcome, however, if courts apply recognized epidemiologic principles and concepts in conjunction with the traditional standard of proof. Epidemiology is the only generally accepted scientific discipline that deals with the integrated use of statistics and biological/medical science to identify and establish the causes of human diseases.[9] Its use enables scientific estimation of the percentage of the risk of a disease that is properly attributable to a given factor, such as exposure to an allegedly harmful substance. Thus, use of an epidemiologic standard would provide courts with a rational and consistent means for evaluating evidence of a causal relationship between exposure to a particular factor and the incidence of a disease.

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evidentiary standard might have prevented the initiation of a case like *Grasso*, and surely would have made it easier to take the case from the jury or to argue for reversal on appeal.

Counsel to one major chemical company has publicly lamented the ease with which plaintiffs can obtain settlements in toxic exposure cases. He attributes the problem to complexity and expense of defense as well as to the uncertainty of the outcome at trial. Sheridan, *Rethinking Mass Tort Defense*, Litigation, Summer 1983, at 29, 29-30.

8. Two recent cases in the District of Columbia, both involving the anti-morning sickness drug Bendectin, illustrate the problems courts have in justifying the exclusion of patently insufficient evidence at the outset of a trial, or in taking a case from the jury if no other evidence is introduced during the trial. In Koller v. Richardson-Merrell, Inc., No. 80-1258 (D.D.C. filed Feb. 25, 1983) the plaintiff alleged that her birth defects had been caused by Bendectin manufactured by the defendant and taken by her mother during pregnancy. The court, in a preliminary order, required that all statistical evidence be significant at a 95% confidence level. *Id.* at 1. This kept certain causation testimony out of the trial, which the plaintiff lost. Neither the order nor the memorandum opinion indicate what is meant by "significance," however. If the reference is simply to the existence of a significant difference between children of mothers who took Bendectin and those who did not, the ruling makes sense, but if the reference goes to complicated statistics such as the risk ratio, significance testing makes little sense. See *infra* pt. II.

In Oxendine v. Merrell Dow Pharmaceuticals, Inc., No. 1245-82 (Super. Ct. D.C. filed Sept. 1, 1983), the judge allowed the testimony that had been excluded in *Koller*. After a jury verdict for the plaintiff, however, the court granted a judgment n.o.v. The judge found no evidence that Bendectin could cause birth defects, *id.* at 2, although the plaintiff's expert testified that 21 of 1,000 children born to mothers who had taken Bendectin would have defects compared to no more than 20 of 1,000 children born to mothers who had not. Trial Transcript at 108-09, *Oxendine*. These statistics are evidence that the drug causes some birth defects, though at most only a very small percentage. The judge would have been on much firmer ground had he found the evidence insufficient to satisfy the more-likely-than-not test rather than finding that it showed nothing at all.

9. Epidemiology is a well-established science tracing its roots back at least 150 years. While not a required part of the typical medical school curriculum, it is taught at most schools. Epidemiologists are not necessarily medical doctors, but many do have M.D.'s. The discussion of epidemiology, *infra* pt. II, explains at some length the discipline's relationship to other sciences.

This Article's underlying premise is that a toxic tort plaintiff, like any other tort plaintiff, has the burden of proving each element of his case[10], including causation.[11] This burden includes the production of evidence from which the factfinder could reasonably infer that the accused substance "more likely than not" caused the plaintiff's harm.[12] The plaintiff must introduce evidence of both the substance's harmfulness at a given exposure level, and of his exposure to the

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10. "Burden of proof" is an unfortunately ambiguous term that incorporates both the burden of producing evidence and the ultimate burden of persuasion. *See* Laughlin, *The Location of the Burden of Persuasion*, 18 U. Pitt. L. Rev. 3, 3 (1956). Inasmuch as this Article deals with the sufficiency of evidence, it is about the burden of production. *See* Dworkin, *Easy Cases, Bad Law, and Burdens of Proof*, 25 Vand. L. Rev. 1151, 1160 (1972). Courts and commentators have considered a number of factors in discussions of how the burden of proof (production or persuasion) should be allocated. These can be grouped under a few broad headings: probability, access to evidence and policy. See *infra* notes 135-42 and accompanying text. The general rule is that the "burdens of pleading and proof with regard to most facts have been and should be assigned to the plaintiff who generally seeks to change the present state of affairs and who therefore naturally should be expected to bear the risk of failure of proof or persuasion." E. Cleary, McCormick's Handbook on the Law of Evidence § 337, at 786 (2d ed. 1972).

11. Like "burden of proof," "causation" has been the source of much confusion. The law distinguishes between "cause in fact" and "proximate cause." The former is simply a matter of what has, in fact, occurred. *See* W. Prosser, Law of Torts § 41, at 237 (4th ed. 1971). The latter is a matter of law. *Id.* § 42, at 244. This Article is concerned solely with the issue of cause in fact, on which:

> as on other issues essential to his cause of action for negligence, the plaintiff . . . has the burden of proof. He must introduce evidence which affords a reasonable basis for the conclusion that it is more likely than not that the conduct of the defendant was a substantial factor in bringing about the result.

Id. § 41, at 241 (footnote omitted). Although this language seems restricted to negligence actions, Dean Prosser made clear that causation is also an essential element for any other tort. *Id.*

The "substantial factor" concept was developed to enable the law to deal with situations in which two or more factors combine to bring about a plaintiff's injury. It does not apply to cases in which factors have acted independently. *See* Delgado, *supra* note 5, at 886-87 & n.26 (referring to "material and contributing" factors, but citing the discussion of "substantial factors" in W. Prosser, *supra*, § 41, at 240-41). Because this Article is restricted to fact patterns involving a single identifiable factor, the "substantial factor" element in Dean Prosser's analysis need not be addressed.

12. Even commentators who have advocated changes to make it easier for plaintiffs to recover in toxic tort cases have explicitly recognized that the more-likely-than-not test is the present rule. *See* Hall & Silbergeld, *supra* note 5, at 446; Trauberman, *supra* note 5, at 197; *Environmental Risks*, *supra* note 5, at 578; *Tort Actions for Cancer*, *supra* note 5, at 857 n.77; *see also Precursor Symptoms*, *supra* note 2, at 193, in which the author explicitly states that the Note's theory requires placing the burden of uncertainty on defendants. The rationale for placing the burden of proof on plaintiffs and for requiring evidence sufficient to establish that the plaintiffs' allegations are more likely than not true is discussed *infra* pt. III(A).

substance at or above that level.[13] Because most toxic tort cases involve diseases with long latency or incubation periods, and because many of these diseases may occur in the absence of any identifiable exposure, causation very often becomes a central and complex issue at trial.[14] To resolve this issue, plaintiffs usually must resort to expert witnesses[15] who, unfortunately, sometimes venture opinions unsupported by scientific data.[16] Moreover, while the outcome of many cases depends on the legal sufficiency of such evidence,[17] courts have not been able to decide the sufficiency issue either clearly or consistently.

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13. *See* 301(e) Study, *supra* note 3, at 70-71. The standard this Article proposes pertains principally, but not exclusively, to the harmfulness aspect of causation. Unlike proof of harmfulness, proof of individual exposure generally depends on more traditional evidence. For a case that turned on the distinction between harmfulness and exposure, see Besner v. Walter Kidde Nuclear Lab., 18 A.D.2d 952, 952, 237 N.Y.S.2d 585, 587 (1963) (holding that the plaintiff had not established causation because the only expert witness who testified about a causal relationship "based his opinion on a completely erroneous premise as to the length of exposure involved and/or a set of facts as to the amount, nature or duration of the alleged exposure unsubstantiated by the record"). The case was remanded and the plaintiff won again below. The defendant once more appealed, but the plaintiff prevailed. He had been able to establish exposure "for a substantial part of two periods and also at other times in various amounts." Besner v. Walter Kidde Nuclear Lab., 24 A.D.2d 1045, 1045, 265 N.Y.S.2d 312, 313 (1965).

14. *See Tort Actions for Cancer*, *supra* note 5, at 851-55; *see, e.g.*, Boldt v. Josten's, Inc., 261 N.W.2d 92, 94 (Minn. 1977); Miller v. National Cabinet Co., 8 N.Y.2d 277, 282-83, 168 N.E.2d 811, 813-14, 204 N.Y.S.2d 129, 132-33 (1960); Clark v. Workmen's Comp. Comm'r, 155 W. Va. 726, 731-34, 187 S.E.2d 213, 216-18 (1972).

15. *See* Taylor, *Occupational Disease: A Defense Attorney's Point of View*, 12 Forum 297, 299 (1976); Trauberman, *supra* note 3, at 189 n.4.

16. An example of this is provided by an expert who testified in several of the swine flu cases. See *infra* pt. IV(B)(3). In one case he opined that the plaintiff's arthritis had been caused by her swine flu inoculation. Gicas v. United States, 508 F. Supp. 217, 220 (E.D. Wis. 1981). The court found:

> that the overwhelming weight of the medical literature opposes a theory that associates Swine Flu vaccine to the plaintiff's injuries. No authority other than [the expert] has causally related rheumatoid arthritis with a swine flu inoculation. . . . [The expert] knows of no evidence other than this case that supports his theory.

Id. Faced with the same expert's testimony in another case, the court noted that "[t]he posture of the expert testimony in this case indicates the limited usefulness that such testimony offers a trier of fact." Latinovich v. United States, 537 F. Supp. 671, 676 (E.D. Wis. 1982). The court went on to list a number of other cases in which his theories had been rejected. *Id.* This expert was also explicitly rejected in Kubs v. United States, 537 F. Supp. 560, 563 (E.D. Wis. 1982).

17. A distinction must be drawn between sufficiency and admissibility. Insufficient evidence may be admissible, but if this is all that a plaintiff can offer, as a matter of law, he cannot prevail. For discussions of the distinction between sufficiency and admissibility, see Martin, *The Uncertain Rule of Certainty: An Analysis*

The first part of this Article examines the inconsistencies and deficiencies in cases that have addressed the issue of causation. Courts have recognized the need to infer causation in toxic tort cases from differences between exposed and unexposed populations. At the same time, they have tried to hold to basic tort law principles. Without a test to measure causal inferences against legal principles, however, their decisions have been ambiguous and confusing. The second part of the Article provides an introduction to the principles of epidemiology, which form the basis for a proposed standard that will enable courts better to distinguish insufficient from sufficient evidence. Part III establishes the basis for the premise that the preponderance-of-the-evidence standard should apply in toxic tort cases, and it then combines epidemiologic principles with this premise to formulate a standard for determining evidentiary sufficiency in toxic tort cases. The proposed standard would require the plaintiff to establish that more than fifty percent of the risk of developing the disease at issue be attributable to the substance at issue, and that certain fundamental epidemiologic postulates be satisfied. Part IV discusses precedents for the use of epidemiologic principles by courts, and possible requirements for witnesses who testify as expert epidemiologists. Finally, the Article addresses problems that might result from retaining the traditional burden of proof and using an evidentiary standard that requires the accumulation of data about populations before an individual can bring a successful action.

I. Causation in Cancer and Toxic Tort Cases

A. *Cancer Cases Involving Trauma or Irritation*

Legal inquiry into the causation of cancer pre-dates toxic tort law, and much of the early theory persists today. Plaintiffs often allege causation from either a traumatic injury[18] or exposure to an immedi-

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and Proposal For a Federal Evidence Rule, 20 Wayne L. Rev. 781, 797-802 (1974); Musslewhite, *Medical Causation Testimony in Texas: Possibility vs. Probability*, 23 Sw. L.J. 622, 622 (1969); Note, *Causation in Disease: Quantum of Proof Required to Reach the Jury*, 53 Nw. U.L. Rev. 794, 795-98 (1959).

18. *E.g.*, Kramer Servs., Inc., v. Wilkins, 184 Miss. 483, 496, 186 So. 625, 627 (1939) (plaintiff alleged that his cancer had been caused by a cut he received when broken glass fell on him); Stordahl v. Rush Implement Co., 148 Mont. 13, 14-16, 417 P.2d 95, 96-97 (1966) (cancer allegedly caused by blow to back); Casson v. A.C. Horn Co., 27 A.D.2d 966, 966-67, 279 N.Y.S.2d 244, 245 (1967) (lung cancer allegedly caused by inhaling paint fumes in work place accident); Hanna v. Aetna Ins. Co., 24 Ohio Misc. 27, 28, 259 N.E.2d 177, 178 (1970) (breast cancer allegedly caused by bruises suffered in automobile accident); Gambrell v. Burleson, 252 S.C. 98, 100, 165 S.E.2d 622, 622-23 (1969) (cancer allegedly aggravated by automobile accident). Most of the injuries are single, isolated traumas, though some are repeated

ately irritating or harmful substance, such as sand or sulfuric acid.[19] In adjudicating trauma claims, courts usually fail to recognize that cancers generally develop without identifiable prior traumatic events, and that incidence rates are no higher in groups that have suffered single traumatic injuries than in those that have not.[20] While appellate decisions sometimes acknowledge the uncertainty and ignorance that surround cancer, they often uphold plaintiffs' verdicts based on coincidences lacking statistical significance.[21] What little guidance medical science has provided about traumatic causation is frequently ignored or misinterpreted.

In 1926, Dr. James Ewing outlined criteria for attributing a particular cancer to a trauma.[22] Although these criteria were intended to provide guidance to courts, Ewing cautioned that "[t]he traumatic theory runs against too many general objections to permit its uncriti-

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traumas more akin to physical irritation. For purposes of this Article, trauma will mean single trauma.

19. *E.g.*, Hagy v. Allied Chem. & Dye Corp., 122 Cal. App. 2d 361, 363, 265 P.2d 86, 87 (1953) (cancer allegedly caused or aggravated by exposure to sulfuric acid); Bollinger v. Wagaraw Bldg. Supply Co., 122 N.J.L. 512, 514-15, 6 A.2d 396, 398-99 (1939) (plaintiff claimed that sand and ashes that had gotten into the decedent's shoes had so aggravated a pigmented mole on one of his feet that it developed into a cancer); Chalmers v. Dep't of Labor & Indus., 72 Wash. 2d 595, 597, 434 P.2d 720, 721 (1967) (cancer allegedly caused by fumes so irritating they once caused plaintiff's deceased husband to pass out); *see* Adelson, *Injury and Cancer*, 5 W. Res. L. Rev. 150, 168-69 (1954); Dyke, *Traumatic Cancer*, 15 Clev.-Mar. L. Rev. 472, 484-94 (1966); Comment, *Sufficiency of Proof in Traumatic Cancer: A Medico-Legal Quandary*, 16 Ark. L. Rev. 243, 256-67 (1962); Comment, *Judicial Attitudes Towards Legal and Scientific Proof of Cancer Causation*, 3 Colum. J. Envtl. L. 344, 354-68 (1977) [hereinafter cited as *Scientific Proof*]; Comment, *Sufficiency of Proof in Traumatic Cancer Cases*, 46 Cornell L.Q. 581, 581-82 (1961) [hereinafter cited as *Sufficiency of Proof*].

20. *See* Adelson, *supra* note 19, at 154-55; Auster, *The Role of Trauma in Oncogenesis: A Juridical Consideration*, 175 J. A.M.A. 946, 949 (1961); Russell & Clark, *Medico-Legal Considerations of Trauma and Other External Influences in Relationship to Cancer*, 6 Vand. L. Rev. 868, 875 (1953); Warren, *Criteria Required to Prove Causation of Occupational or Traumatic Tumors*, 10 U. Chi. L. Rev. 313, 318-20 (1943).

21. *E.g.*, Hagy v. Allied Chem. & Dye Corp., 122 Cal. App. 2d 361, 375-76, 265 P.2d 86, 95 (1953); Daly v. Bergstedt, 267 Minn. 244, 248, 126 N.W.2d 242, 245 (1964); *see Sufficiency of Proof*, *supra* note 19, at 582 & n.10. See *infra* note 104 for a discussion of what is meant by "statistical significance."

22. Ewing, *The Relation of Trauma to Malignant Tumors*, Am. J. Surgery, Feb. 1926, at 30, 31-34. The criteria set forth were:

(1) Authenticity and sufficient severity of the trauma.

(2) Previous integrity of wounded part.

(3) Identity of injured area with that giving origin to the tumor.

(4) Tumor of a type that could conceivably result from trauma.

(5) Proper time interval between receipt of the injury and appearance of the tumor.

Id.

cal acceptance."[23] Moreover, he premised his work on the assumption that the defendant has the burden of disproof,[24] thus further limiting the proper application of his postulates. By 1935, he had become still more conservative, acknowledging that "experimental data reveal the fact that cancer genesis requires quite peculiar factors which have not been found in the results of simple trauma."[25] Later work by others has further limited the Ewing approach.[26]

Ignorance and uncertainty make it virtually impossible, even with the aid of Ewing's criteria, to determine whether a single trauma, or a majority of irritating factors,[27] more likely than not caused the initiation of a latent disease such as cancer. Because plaintiffs bear the burden of proof, this dearth of evidence logically implies that plaintiffs should generally lose as a matter of law, but few courts have stated this explicitly.[28] Instead, decisions have generally been ill-reasoned and inconsistent.[29]

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23. *Id.* at 34.

24. *Id.* at 30.

25. Ewing, *The Modern Attitude Toward Traumatic Cancer*, 11 Bull. N.Y. Acad. Med. 281, 281 (1935).

26. *See* Auster, *supra* note 20, at 949. No one has suggested that the Ewing analysis can lead to a conclusion that a causal link is more probable than not. Rather, only possible inference is claimed. One commentator has explicitly stated that the postulates relate only to possibility. Adelson, *supra* note 19, at 156. Ewing's postulates may, however, be used to support defendants' verdicts because the plaintiff must at least satisfy them to prove causation. *See* Stordahl v. Rush Implement Co., 148 Mont. 13, 19-20, 417 P.2d 95, 99 (1966); Sikora v. Apex Beverage Corp., 282 A.D. 193, 196, 122 N.Y.S.2d 64, 66 (1953), *aff'd*, 306 N.Y. 913, 119 N.E.2d 601 (1954); Dennison v. Wing, 279 A.D. 494, 496-97, 110 N.Y.S.2d 811, 813-14 (1952).

27. *See* Auster, *supra* note 20, at 949. In some prolonged irritation cases it may be possible to infer causation with sufficient certainty. Ewing, *supra* note 25, at 314.

28. The only example of which the authors are aware is Tonkovich v. Department of Labor & Indus., 31 Wash. 2d 220, 195 P.2d 638 (1948).

29. *Compare* Daly v. Bergstedt, 267 Minn. 244, 248, 126 N.W.2d 242, 245 (1964) (upholding plaintiff's claim that a bruise on her breast had become cancerous) *with* Tonkovich v. Department of Labor & Indus., 31 Wash. 2d 220, 226-27, 195 P.2d 638, 641-42 (1948) (rejecting plaintiff's claim that fractured bones in his foot worsened into arthritis and intestinal cancer 10 years later).

Plaintiffs' verdicts in workers' compensation cases, even in the absence of reliable information, are perhaps understandable. The requirement that a disease be occupational conceptually parallels the tort law causation requirement, but it is not identical to it. *See* 1B A. Larson, Workmen's Compensation Law § 41 (1982 & Supp. 1983); *see, e.g.*, Cox v. Ulysses Coop. Oil & Supply Co., 218 Kan. 428, 432-33, 544 P.2d 363, 367 (1975) (in a workers' compensation case the claimant need only introduce evidence sufficient to convince the court that the award is proper); Deines v. Greer, 216 Kan. 548, 553, 532 P.2d 1257, 1262 (1975) (when injury shown to have arisen out of course of employment, every natural consequence of injury is compensable); Workmen's Comp. Appeals Bd. v. Bethlehem Steel Corp., 23 Pa. Commw. 454, 456, 352 A.2d 571, 572 (1976) (plaintiff need not prove injury caused by identifiable incident, but rather only that injury arose in course of employment). Some states

Daly v. Bergstedt[30] typifies the muddled reasoning employed in many trauma and irritation cases. The plaintiff brought a simple slip and fall tort action, straightforward except for her claim that a bruise on her left breast had caused it to become cancerous. The Minnesota Supreme Court affirmed the plaintiff's verdict, but the court's review of the evidence did not justify its holding. Six medical doctors testified that there was no causal connection between the bruise and the cancer, while one gave the opinion that the cancer could have developed from the trauma sustained in the fall.[31] Apparently realizing that science weighed heavily in favor of the defendant, the court chose to rely on the coincident location of the trauma and the cancer and the relatively short (14 months) time period between the two.[32] This approach totally ignores the absence of evidence that the incidence of breast cancer is higher among women who have suffered trauma than among women who have not.[33] The *Daly* case implies that it is appropriate to allow laymen to draw conclusions from information

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create presumptions that lessen the plaintiff's burden of proof. *See, e.g.*, Downes v. Industrial Comm'n, 113 Ariz. 90, 93, 546 P.2d 826, 829-30 (1976); Bolger v. Chris Anderson Roofing Co., 112 N.J. Super. 383, 394, 271 A.2d 451, 457-58 (1970), *aff'd per curiam*, 117 N.J. Super. 497, 285 A.2d 228 (1971). *Compare* Cox v. Ulysses Coop. Oil & Supply Co., 218 Kan. 428, 435-36, 544 P.2d 363, 369-70 (1975) (personal opinion of physician that causation is a "reasonable medical certainty" is sufficient to justify recovery) *with* Parker v. Employers Mut. Liab. Ins. Co., 440 S.W.2d 43, 45 (Tex. 1969) (causal connection must be clearly established between employment and injury to justify recovery).

30. 267 Minn. 244, 126 N.W.2d 242 (1964).

31. *Id.* at 248, 126 N.W.2d at 245. The court based its opinion on the Ewing Postulates. *Id.* However, the postulates had not, in fact, been satisfied. Ewing made it quite clear that only one type of breast cancer, carcinoma simplex, could be linked to trauma, and that "in each case the entire clinical history must be secured and the tumor and the entire breast must be examined by a competent tumor pathologist before the basis can be laid for an opinion." Ewing, *supra* note 25, at 320-21. There is no indication that Mrs. Daly produced such evidence. In fact, her expert had testified that she had a scirrhus carcinoma, not carcinoma simplex. 267 Minn. at 249, 126 N.W.2d at 246. Even if the postulates had been satisfied, the plaintiff would still not have established the causal link by a preponderance of the evidence. See *supra* note 10.

32. 267 Minn. at 247-51, 126 N.W.2d at 245-47. Other courts have held that while coincidence and expert testimony about possibilities by themselves are not enough, together they may be sufficient. *See* Hagy v. Allied Chem. & Dye Corp., 122 Cal. App. 2d 361, 371, 265 P.2d 86, 92-93 (1953) (quoting Fireman's Fund Indemnity Co. v. Industrial Acc. Comm'n, 93 Cal. App. 2d 244, 246, 208 P.2d 1033, 1034 (1949)). While plausible at first glance, this approach is in fact no better than that taken by the *Daly* court. An expert is assumed to know all the available facts relevant to causation, and if he cannot reach a suitably certain conclusion laymen should not be expected to do so. Stated another way, if proof of causation requires expert testimony, the expert's determination of how certain one can be ought to be determinative.

33. See *supra* note 20 and accompanying text.

found to be inadequate by experts, a rule that leaves little basis for a rational analysis of the legal sufficiency of evidence.[34]

Courts that have reviewed the sufficiency of expert testimony in trauma and irritant cases have tended to go little beyond the witness' expressed degree of certainty, distinguishing, for example, between the use of the words "possible" and "probable."[35] Often they uncritically defer to physicians,[36] whose training and experience typically do not qualify them to venture opinions about the probability that a particular factor caused a disease.[37] Focusing on the expressed certainty or supposed professional competence of physicians shifts attention from underlying uncertainty and permits at least apparent adherence to the more-likely-than-not standard, but it does not lead to consistent results.

The distinction between possibility and probability is not insignificant, but when reduced to a simple search for expressed certainty or for the blessing of a suitably credentialed expert, it often has no real effect. Judicial reluctance to examine the substantive basis of the testimony can easily permit unfounded expressions of certainty to carry the day. Pennsylvania, for example, requires that causation

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34. Other decisions have also been based on this kind of limited review. *See, e.g.*, Hagy v. Allied Chem. & Dye Corp., 122 Cal. App. 2d 361, 375-76, 265 P.2d 86, 95 (1953); Hanna v. Aetna Ins. Co., 24 Ohio Misc. 27, 32-33, 259 N.E.2d 177, 180-81 (1970); Valente v. Bourne Mills, 77 R.I. 274, 278, 75 A.2d 191, 194 (1950).

35. Cox v. Ulysses Coop. Oil & Supply Co., 218 Kan. 428, 435-36, 544 P.2d 363, 369-70 (1975); *see* Pucci v. Rausch, 51 Wis. 2d 513, 518-19, 187 N.W.2d 138, 141-42 (1971) (personal injury case in which the court required only that a doctor have sufficient certainty that his opinion is "correct to a reasonable medical probability. Other doctors may differ, but whether his opinion corresponds with that of another member of the medical profession does not go to admissibility of his opinion but to the weight the trier of the facts should give to his opinion."); City of Seymour v. Industrial Comm'n, 25 Wis. 2d 482, 491-92, 131 N.W.2d 323, 328 (1964) (medical testimony cannot be held "incredible because contrary to scientific facts or knowledge").

The *Pucci* court listed with approval a number of cases in which various forms of medical testimony had been either acceptable or unacceptable. 51 Wis. 2d at 519, 187 N.W.2d at 142. This approach can redound to the benefit of defendants as well as plaintiffs. *See* Casson v. A.C. Horn Co., 27 A.D.2d 966, 967, 279 N.Y.S.2d 244, 245 (1967) (medical testimony sufficient); Insurance Co. of N. Am. v. Myers, 411 S.W.2d 710, 714 (Tex. 1966) (medical testimony that causation was merely possible insufficient for recovery). *See generally* Annot., 66 A.L.R.2d 1082, 1118-24 (1959) (dealing with the issue of admissibility, not sufficiency, but citing many cases that relate to the sufficiency issue).

36. *See* McGrath v. Irving, 24 A.D.2d 236, 238, 265 N.Y.S.2d 376, 378 (1965) (plaintiff's expert testimony held sufficient based on his "medical qualifications").

37. When etiology is unknown, causation must usually be determined at least in part from statistical inferences. Biostatisticians deal with this numerical aspect of establishing causation, but they often lack a full appreciation of the biological aspect. It is the epidemiologist who specializes in using both statistics and biology to arrive at scientifically supportable conclusions about causation.

testimony be couched in very certain terms, but an expert in *Menarde v. Philadelphia Transportation Co.*[38] evaded this limitation simply by testifying that it was virtually impossible that the plaintiff's breast cancer had been caused by anything other than the minor injuries she had suffered in a trolley car accident.[39] This case clearly demonstrates how neatly an expert can tailor testimony to the requirements set forth in previous decisions. If certainty is needed, witnesses can be found who will profess it.

B. *Toxic Tort Cases*

In toxic tort cases, latency and the absence of an identifiable irritation or traumatic injury have made it more difficult than in trauma cases for courts to rely solely on coincidences.[40] Nevertheless, the focus on witnesses' expressions of certainty and the deference to medical experts seen in traumatic cancer cases have carried over to toxic torts. In *Boldt v. Jostens, Inc.*,[41] for example, the plaintiff claimed that her workplace exposure to fumes from heated glue caused her to contract Goodpasture's Syndrome, a pathologic condition in which the kidneys and lungs are attacked by one's own immune system. The doctor who testified for the plaintiff about causation acknowledged that the etiology of Goodpasture's Syndrome is unknown. He stated that it was thought to be an immunologic disease, that the antigen causing a reaction in a victim "can probably be many different things and different for different people,"[42] and that it is unknown whether the reaction is the result of one exposure or many.[43] Yet, he was willing to opine that the plaintiff's exposure to glue fumes "had a great deal to do with her illness, and certainly caused aggravation."[44] The Supreme Court of Minnesota held that this testimony sufficed to sustain a workers' compensation award, in part because "the truth of the opin-

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38. 376 Pa. 497, 103 A.2d 681 (1954).

39. *Id.* at 502, 103 A.2d at 684; *see* Peterson v. Kansas City Pub. Serv. Co., 259 S.W.2d 789, 794 (Mo. 1953).

40. *Scientific Proof*, *supra* note 19, at 354. *But see* Boney v. Gouverneur Talc Co., 77 A.D.2d 702, 702, 430 N.Y.S.2d 399, 399 (1980) (lung cancer found to have been caused by exposure to talc dust containing asbestos). The plaintiff in *Boney* admittedly had talcosis, a form of pneumoconiosis. But other than testimony that this condition might have predisposed him to contract cancer, there was apparently no evidence to support holding the defendent liable for the disease. It should be noted that while mesothelioma (which is not what the plaintiff had) is very clearly linked to asbestos exposure, other forms of cancer are not, at least not to the same high degree. This illustrates why specificity is so important in epidemiologic analysis. See *supra* note 89 and accompanying text.

41. 261 N.W.2d 92 (Minn. 1977).

42. *Id.* at 93.

43. *Id.*

44. *Id.*

ion need not be capable of demonstration."[45] Other cases indicate that in some circumstances a treating physician's testimony will be given special weight,[46] or that a specialist's testimony will be given more weight than a general practitioner's.[47]

When courts do go beyond simple deference to medical testimony, they generally do no more than subject it to the same cursory "probability versus possibility" analysis found in some trauma cases.[48] In exposure cases this has at least proven useful in culling claims in which a witness singles out one factor as the "most probable" of many. These situations occur because when diagnosing and treating a disease, doctors often cannot state with certainty which factor is its direct cause. They quite properly think in terms of finding the most likely cause instead of a factor that more likely than not is the cause.[49] Thus terms like "medical certainty" or "medical probability" often fail to satisfy legal requirements. In *Clark v. State Workmen's Compensation Commissioner*,[50] for example, the plaintiff established that the only clearly identifiable cause of her deceased husband's leukemia was his exposure to chemicals at the plant in which he had worked.[51] Her expert had also testified, however, that the etiology of the disease was unknown and that other factors could have caused it.[52] The court held that this evidence failed to satisfy the requirement that a workers' compensation claimant prove that his disease is job-related.[53]

Although scientific studies do not support the argument that trauma increases the incidence of disease,[54] data do exist that permit comparisons of disease rates in populations exposed to some substances with the rates in unexposed populations.[55] Such comparisons are a

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45. *Id.* at 94. *But see* Logan Co. v. Amic, 479 S.W.2d 1, 2-3 (Ky. 1972) (hypothesis of physician not sufficent evidence to justify plaintiff's recovery).

46. Long v. Martin Timber Co., 395 So. 2d 931, 934 (La. App. 1981); Groff v. Department of Labor & Indus., 65 Wash. 2d 35, 45, 395 P.2d 633, 639 (1964); *Sufficiency of Proof*, *supra* note 19, at 601.

47. Chalmers v. Department of Labor & Indus., 72 Wash. 2d 595, 598-601, 434 P.2d 720, 722-24 (1967); *Sufficiency of Proof*, *supra* note 19, at 601.

48. See *supra* notes 35-39 and accompanying text.

49. *See* Danner & Sagall, *Medicolegal Causation: A Source of Professional Misunderstanding*, 3 Am. J. Law & Med. 303, 304-05 (1977).

50. 155 W. Va. 726, 187 S.E.2d 213 (1972).

51. *Id.* at 728-29, 187 S.E.2d at 215.

52. *Id.*

53. *Id.* at 734, 187 S.E.2d at 217-18; *see* Schaefer v. Texas Employment Ins. Ass'n, 612 S.W.2d 199, 205 (Tex. 1981) (rejecting medical testimony that it was reasonably probable that workplace exposure caused decedent's cancer).

54. See *supra* note 20 and accompanying text.

55. *See, e.g.*, Doll & Peto, *The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today*, 66 J. Nat'l Cancer Inst. 1192

basic part of epidemiologic studies, and in a few cases, have led to causal inferences so strong that courts have found causation to be scientifically established without any analysis of the method and reasoning underlying that conclusion.[56] When the data are less conclusive, as usually occurs in toxic tort cases, the law has had far more difficulty in dealing with the evidence. A number of commentators have referred approvingly to the use of epidemiology or biostatistics,[57]

(1981); Wynder & Gori, *Contribution of the Environment to Cancer Incidence: An Epidemiologic Exercise*, 57 J. Nat'l Cancer Inst. 825 (1977).

56. The link between asbestos and mesothelioma (a form of cancer that attacks the lining of the pleural cavity) was not established until the early 1970's, just about the time that the growing flood of legal action began. Mehaffy, *Asbestos-Related Lung Disease*, 16 Forum 341, 344 (1980). Causation had been established by the epidemiologic work of Dr. Irving J. Selikoff and others, and in the litigation it has been substantially accepted. Without examining the methodology by which scientists reached their conclusions, courts accept causation almost as a matter of law. *See* Karjala v. Johns-Manville Prods. Corp., 523 F.2d 155, 158 (8th Cir. 1975); Bertrand v. Johns-Manville Sales Corp., 529 F. Supp. 539, 544 (D. Minn. 1982); Flatt v. Johns-Manville Sales Corp., 488 F. Supp. 836, 841 (E.D. Tex. 1980); Mehaffy, *supra*, at 341. *But see* Tretter v. Johns-Manville Corp., 88 F.R.D. 329, 332-33 (E.D. Mo. 1980) (court required plaintiff asserting causal link between asbestos and cancer to prove harmfulness).

In the DES litigation, the link between DES and clear cell adenocarcinoma is virtually certain, although established only epidemiologically. *See* Herbst, Ulfelder & Poskanzer, *Adenocarcinoma of the Vagina*, 284 N.E. J. Med. 878, 878 (1971); Note, *Market Share Liability: An Answer to the DES Causation Problem*, 94 Harv. L. Rev. 668, 669 (1981). Vinyl chloride exposure (at high enough levels) and one form of liver cancer have also been linked almost unequivocally through epidemiology. *See* Society of the Plastics Indus., Inc. v. OSHA, 509 F.2d 1301, 1305-06 (2d Cir.), *cert. denied*, 421 U.S. 992 (1975).

In the case of cigarettes and lung cancer, some early decisions indicated that epidemiologic evidence might be sufficient. *See* Lartigue v. R.J. Reynolds Tobacco Co., 317 F.2d 19, 22-23 (5th Cir. 1963); Pritchard v. Liggett & Myers Tobacco Co., 295 F.2d 292, 294-96 (3d Cir. 1961); *Scientific Proof*, *supra* note 19, at 369-73. Litigation about cigarettes, however, has been stifled by warning labels that preclude warranty claims, and by court holdings that until the labels were put on the packages the manufacturers could not have known about the harm cigarettes could cause and thus could not be held liable. *See* W. Prosser, *supra* note 11, § 99, at 660 & nn.82-83; *Scientific Proof*, *supra* note 19, at 369-73.

57. *See, e.g.*, Estep, *Radiation Injuries and Statistics: The Need for A New Approach to Injury Litigation*, 59 Mich. L. Rev. 259, 273-80 (1960); Forgotson, *Liability For Long-Term Latent Effects of Toxic Agents*, 50 A.B.A.J. 142, 142 (1964); Hall & Silbergeld, *supra* note 5, at 442-43; Henderson, *Medical Causation in Products Liability Disease Litigation*, Trial, June 1981, at 53, 55-57; Mobilia & Rossignol, *The Role of Epidemiology in Determining Causation in Toxic Shock Syndrome*, Jurimetrics J., Fall 1983, at 78, 82-86; Riley, *Toxic Shock Syndrome: Proving Causation Before Science Has*, 6 Am. J. Trial Advoc. 15, 19 (1982); Rosenberg, *supra* note 5, at 856-57, 869-74; Seltzer, *Personal Injury Hazardous Waste Litigation: A Proposal for Tort Reform*, 10 B.C. Envtl. Affairs L. Rev. 797, 815-21,

and a few courts have acknowledged the need to infer causation from comparisons between populations.[58] To date, however, neither commentators nor courts have provided guidance on how to mesh law and epidemiology in a consistent way.

A series of New York cases exemplifies both current developments and current confusion. In *Miller v. National Cabinet Co.*,[59] the New York Court of Appeals reversed an award of workers' compensation

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846-49 (1982-1983); *Tort Actions for Cancer*, *supra* note 5, at 857. *But see* Dickson, *Medical Causation by Statistics*, 17 Forum 792, 799-808 (1983) (noting shortcomings in the use of epidemiologic evidence); Dore, *A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-in-Fact*, 7 Harv. Envtl. L. Rev. 429, 431 (1983) ("Because of the confusing and complex nature of epidemiologic evidence, courts should . . . [limit] the use of such evidence as proof of causation . . . .").

The proponents of epidemiology give little guidance on how courts should use it, and except for Forgotson, none address the idea of requiring epidemiologic evidence. A number of commentators seem to have the impression that courts tend not to accept epidemiologic evidence. *See, e.g.*, Rosenberg, *supra* note 5, at 857-58, 869-74; Seltzer, *supra*, at 821-24; *Tort Actions for Cancer*, *supra* note 5, at 848; *see also* Trauberman, *supra* note 3, at 198 (author knows of no case in which an award has been based solely on epidemiologic evidence). Research, however, reveals no case in which a court has held against a plaintiff who has produced evidence sufficient to satisfy the standard proposed in this Article. A large part of the problem is that without a substantive standard, plaintiffs do not know how to present their cases. *Cf.* Schaefer v. Texas Employer's Ins. Ass'n, 612 S.W.2d 199, 205 (Tex. 1980) (plaintiff lost appeal because he failed to produce tests or data).

58. Traces of epidemiologic reasoning have appeared in a variety of cases. *See, e.g.*, Mahoney v. United States, 220 F. Supp. 823, 838 (E.D. Tenn. 1963) (court found for the defendant because there was only a 1 in 24 chance that the plaintiff's leukemia had been caused by radiation), *aff'd*, 339 F.2d 605 (6th Cir. 1964); Braden v. City of Hialeah, 177 So. 2d 235, 236 (Fla. 1965) (per curiam) (plaintiff's claim rejected because she did not show that workplace exposure to sun made probability of contracting skin cancer greater than that of persons with normal exposure to sun); Miller v. Olin Mathieson Chem. Corp., 398 S.W.2d 472, 472-73 (Ky. 1965) (plaintiff's claim rejected because physician's theory of chemical causation of leukemia contradicted by statistical data showing that the incidence of leukemia increased when presence in atmosphere of chemical compounds decreased); Miller v. National Cabinet Co., 8 N.Y.2d 277, 283-84, 168 N.E.2d 811, 814, 204 N.Y.S.2d 129, 133-34 (reference to need for medical statistics showing correlation between exposure to benzol and incidence of leukemia), *modified on other grounds*, 8 N.Y.2d 1025, 170 N.E.2d 215, 206 N.Y.S.2d 796 (1960); Collins v. National Aniline Div., 8 A.D.2d 900, 901, 186 N.Y.S.2d 979, 981 (1959) (reference to comparison of incidence rates of bladder cancer among those exposed to carcinogenic compounds and those not so exposed); Parker v. Employers Mut. Liab. Ins. Co., 440 S.W.2d 43, 47-48 (Tex. 1969) (testimony admitted but held not conclusive that persons exposed to radiation have a higher incidence rate of cancer than non-exposed persons); Ehman v. Department of Labor & Indus., 33 Wash. 2d 584, 595, 206 P.2d 787, 797 (1949) (court held for defendant because plaintiff could not show that but for his employment, he would not have contracted leukemia).

59. 8 N.Y.2d 277, 168 N.E.2d 811, 204 N.Y.S.2d 129, *modified on other grounds*, 8 N.Y.2d 1025, 170 N.E.2d 215, 206 N.Y.S.2d 796 (1960).

benefits to the widow of a worker whose death from leukemia had allegedly been caused by exposure to benzene (also known as benzol). The plaintiff's principal expert witness testified that the incidence of leukemia "is quite high in patients who have been exposed to benzol," and that "it is *possible* that this man's leukemia resulted from his alleged exposure to inhalation of benzol or benzene."[60] In holding for the defendant, the court relied principally on the possibility-probability distinction.[61] It pointed out, however, that "[t]he only possible basis for drawing an inference in favor of claimant . . . would be statistics indicating that in many instances leukemia follows benzol exposure without knowing why."[62]

The allusion in *Miller* to the consideration of statistics as a factor in the determination of causation represents a small step forward in toxic tort theory. Subsequent decisions in New York, however, have not furthered the development of this concept. Most opinions have been couched in terms similar to the plaintiff's argument in *Miller* and have failed to employ statistical data in arriving at their conclusions about causation.[63] In one case, decided for the plaintiff, the expert testified only that he knew at least some of the causes of the disease in question, and that the plaintiff had been exposed to one of them.[64] Two experts in another case stated, with little quantification, that people in the plaintiff's occupation ran a high risk of developing papillary tumors.[65] Again the plaintiff prevailed, though the facts were hardly distinguishable from those in *Miller*. In still another case, the court explicitly found that the statistical requirement had been met, only to be

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60. *Id.* at 282, 168 N.E.2d at 813, 204 N.Y.S.2d at 132 (emphasis added).

61. *Id.* at 282-83, 168 N.E.2d at 813, 204 N.Y.S.2d at 132-33.

62. *Id.* at 283, 168 N.E.2d at 814, 204 N.Y.S.2d at 133. How to use statistics and how to incorporate other information in drawing biological inferences remained unexplained, though the decision hinted that an eleven-fold increase in the incidence rate in an exposed population might not support a plaintiff's verdict. *Id.* at 285, 168 N.E.2d at 815, 204 N.Y.S.2d at 135.

63. *E.g.*, Shannon v. Grumman Aircraft, 29 N.Y.2d 786, 787-88, 277 N.E.2d 190, 190-91, 327 N.Y.S.2d 71, 72 (1971), *rev'g* 35 A.D.2d 230, 315 N.Y.S.2d 172 (1970); Boney v. Gouverneur Talc Co., 77 A.D.2d 702, 702, 430 N.Y.S.2d 399, 399 (1980); Smith v. Humboldt Dye Works, 34 A.D.2d 1041, 1042, 312 N.Y.S.2d 612, 614 (1970); Benenati v. Tin Plate Lithographing Co., 29 A.D.2d 805, 806, 287 N.Y.S.2d 528, 530 (1968); Amoroso v. Tubular & Cast Prods. Mfg. Co., 17 A.D.2d 1003, 1003-04, 233 N.Y.S.2d 909, 910-11 (1962), *aff'd*, 13 N.Y.2d 992, 194 N.E.2d 694, 244 N.Y.S.2d 787 (1963); Hassell v. Oxford Filing Supply Co., 16 A.D.2d 534, 536, 230 N.Y.S.2d 866, 868 (1962); *see, e.g.*, Yannon v. New York Tel. Co., 86 A.D.2d 241, 244, 450 N.Y.S.2d 893, 895 (1982); Berman v. Werman & Sons, 14 A.D.2d 631, 631, 218 N.Y.S.2d 315, 316 (196l).

64. Benenati v. Tin Plate Lithographing Co., 29 A.D.2d 805, 806, 287 N.Y.S.2d 528, 530 (1968).

65. Smith v. Humboldt Dye Works, Inc., 34 A.D.2d 1041, 1042, 312 N.Y.S.2d 612, 614 (1970).

reversed by the court of appeals, which found "no observable or acceptable correlation between exposure . . . and [disease]."[66] The decisions at both levels fail to indicate the standard by which statistical inference should be judged, or how biological inference should follow from statistics.

The *Miller* line of cases typifies the haphazard way in which courts have addressed the use of comparisons between exposed and unexposed populations to establish toxic tort causation.[67] No clear standard has yet emerged to determine when data and analysis are legally sufficient, or if statistical and non-statistical evidence have been properly integrated.[68] This has clouded legal analysis as well as factfinding.

C. *The More-Likely-Than-Not Test in Toxic Tort Cases*

Courts generally have not held that a toxic tort plaintiff bears a lesser burden of proof on the issue of harmfulness than does the traditional tort law plaintiff.[69] In fact, courts have explicitly adopted the preponderance test in a number of cases in which the harmfulness of a substance was at issue. In *Parker v. Employers Mutual Liability*

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66. Shannon v. Grumman Aircraft, 29 N.Y.2d 786, 788, 277 N.E.2d 190, 191, 327 N.Y.S.2d 71, 72 (1971), *rev'g* 35 A.D.2d 230, 315 N.Y.S.2d 172 (1970).

67. Courts in other states have also touched upon the evidentiary use of statistical inference in determining toxic tort causation. *See, e.g.*, Miller v. Olin Mathieson Chem. Corp., 398 S.W.2d 472, 473 (Ky. 1965); Schaefer v. Texas Employers' Ins. Ass'n, 612 S.W.2d 199, 201 (Tex. 1981); Parker v. Employers Mut. Liab. Ins. Co., 440 S.W.2d 43, 49 (Tex. 1969); Garner v. Hecla Mining Co., 19 Utah 2d 367, 370, 431 P.2d 794, 796 (1967). *Garner* is the most interesting case because it involved the question how statistical evidence should mesh with non-statistical considerations, one of the principal concerns of epidemiology. A widow appealed the denial of workers' compensation benefits for the death of her husband, who had been a uranium miner. The widow introduced autopsy results showing that her husband's body had contained 34 times as much radioactive lead as the average non-miner's. She also introduced data indicating a high incidence of lung cancer in uranium miners. The court did not find this proof necessarily insufficient, but held that such evidence did not compel an award of benefits. *Id.* at 370, 431 P.2d at 796. The court noted that other factors might have caused the disease, specifically mentioning the fact that the decedent had smoked for approximately twenty years. *Id.* at 371, 431 P.2d at 796-97.

68. Only in a few of the cases that grew out of the 1976 swine flu inoculation program have courts made further progress, but the circumstances surrounding those cases were unique. The increased risk of Guillan-Barre Syndrome (GBS) related to swine flu shots lasted for only a few weeks. Most toxic tort risks are less reversible. Also, because of the number of people involved in the swine flu program and the careful monitoring of it by the Center for Disease Control, very good epidemiologic data were available. See *infra* pt. IV(B)(3).

69. The plaintiff must produce "proof which leads the jury to find that the existence of the contested fact is more probable than its nonexistence." E. Cleary, *supra* note 10, § 339, at 794. See *supra* note 10.

Insurance Co.,[70] for example, the plaintiff alleged that his cancer had been caused by workplace exposure to radiation. He was unsuccessful because he could only establish a low level of exposure, which merely suggested the possibility of causation. The court held that "a possible cause only becomes 'probable' when in the absence of other reasonable causal explanations it becomes *more likely than not* that the injury was a result of its action."[71]

In *McEwen v. Ortho Pharmaceutical Corp.*,[72] the plaintiff claimed that her blindness had been caused by birth control pills. The Oregon Supreme Court upheld her jury verdict, finding that the medical testimony had at least established that the inference of causation was "more probably correct than incorrect."[73] Other toxic tort decisions have been similarly based on the more-likely-than-not test,[74] but except in a few of the swine flu cases,[75] none has come close to considering either the need for epidemiologic evidence or how to analyze such evidence to insure that legal requirements are met.

With the dramatic increase in litigation over latent effects of toxic exposures, the failure to fit known facts into a legal context makes the need for a substantive evidentiary standard ever more pressing. The formulation of a test that will meet this need requires a basic understanding of the philosophy and methods of epidemiology. Properly used and evaluated, epidemiologic evidence will enable courts to adhere to both tort law and scientific principles.

II. Epidemiologic Principles

The elucidation of the relationship between a disease and a factor (e.g., a toxic substance) suspected of causing it lies within the domain of epidemiology.[76] The epidemiologist examines this relationship in the context of populations, comparing the disease experiences of people exposed to the factor with those not so exposed.[77] Although the epidemiologist utilizes statistical methods, the ultimate goal is to draw a biological inference concerning the relationship of the factor to the

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70. 440 S.W.2d 43 (Tex. 1969).

71. *Id.* at 47 (emphasis added).

72. 270 Or. 375, 528 P.2d 522 (1974).

73. *Id.* at 415 n.36, 528 P.2d at 541 n.36.

74. Sheptur v. Procter & Gamble Distrib. Co., 261 F.2d 221, 224 (6th Cir. 1958) (per curiam); Coburn v. North American Refractories Co., 295 Ky. 566, 174 S.W.2d 757 (1943); Grinnell v. Charles Pfizer & Co., 274 Cal. App. 2d 424, 435, 79 Cal Rptr. 369, 374-75 (1969).

75. See *infra* pt. IV(B)(3).

76. *See* Last, *Scope and Methods of Prevention*, in Maxcy-Rosenau Public Heath and Preventive Medicine 7-8 (J. Last ed. 1980).

77. *See* A. Lilienfeld & D. Lilienfeld, Foundations of Epidemiology 3 (2d ed. 1980).

disease's etiology and/or to its natural history.[78] 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."[79] It is an integrative, eclectic science utilizing concepts and methods from other disciplines, such as statistics, sociology and demography for the study of disease in populations.

To understand epidemiologic methods and reasoning, one must understand how epidemiology grew out of its component disciplines. The natural philosophers of the seventeenth century initiated a method of reasoning based on the premise that one can mathematically model a population's mortality experience.[80] This work developed into the modern fields of demography, vital statistics, and subsequently, epidemiology.

One of the tools that these scientists developed was the life table, known until the early 1900's as the "table of mortality."[81] The first life tables reflected only the aggregate mortality experience in a population.[82] They provided no record of individual diseases because the concept of specific diseases had not yet crystallized.[83] Indeed, al-

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78. Lilienfeld, *Definitions of Epidemiology*, 107 Am. J. Epidemiology 87, 89 (1978).

79. A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 4.

80. *See* Lilienfeld, *"The Greening of Epidemiology": Sanitary Physicians and the London Epidemiological Society (1830-1870)*, 52 Bull. Hist. of Med. 503, 504 (1979); Lorimer, *The Development of Demography*, in The Study of Population 124, 127 (P. Hauser & O. Duncan eds. 1959).

81. *See* Lilienfeld & Lilienfeld, *The French Influence on the Development of Epidemiology*, in Times, Places and Persons: Aspects of the History of Epidemiology 28, 28 (A. Lilienfeld ed. 1980). Figure I shows a typical life table, a tabulation of a given population's mortality experience.

FIGURE 1

A TYPICAL LIFE TABLE

Age	Population at Start of Age	Deaths
0-1	1,000	20
1-4	980	80
5-14	900	250
15-24	650	250
25-34	400	300
35 and over	100	100

82. J. Farren, Historical Essay on the Rise and Early Progress of the Doctrine of Life-Contingencies in England (London 1844); J. Francis, Annals and Legends of Life Assurance 87-97 (London 1853); *see* Lilienfeld & Lilienfeld, *supra* note 81, at 28.

83. *See* Temkin, *Comment on Hilt's "Epidemiology and the Statistical Movement,"* in Times, Places and Persons: Aspects of the History of Epidemiology 61 (A. Lilienfeld ed. 1980).

though the notion of statistically viewing the mortality experience of a population dates from the mid-1600's, not until the 1800's did the concept of disease specificity emerge. This development permitted scientists to make accurate correlations and to draw meaningful causal inferences.[84]

A. *The Definition of Disease*

Although concern about the exact definition of a disease began with communicable diseases, it is of equal concern when dealing with chronic diseases such as cancer, heart disease and stroke. The epidemiologist must begin his investigation with a clear, precise definition of the disease being studied.[85] Within the medical community, disease is viewed as an entity characterized by at least two of the following criteria: "a recognized etiologic agent (or agents); an identifiable group of signs and symptoms; [and/or] consistent anatomical alterations [that is, lesions or a pathologic state being present]."[86] This definition of disease does not differ markedly from that used by lawyers: "An illness or an abnormal state having a definite pattern of symptoms."[87] Neither statement, however, suffices for an epidemiologic investigation, which requires an exact definition of the disease being studied.

The definition of a particular disease depends on its nature and must be sufficiently precise to permit exclusion of all other diseases from consideration. The "gold standard" definition is that of the pathologist, as it is based on the histologic characteristics of the disease. For diseases defined by pathophysiologic changes, such as asthma, other characteristics, such as physiologic ones, may be used. Some diseases and syndromes, such as volvulus,[88] are best defined in terms of what is observed during surgical intervention. The internist

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84. The melding of the concepts of statistics and specificity was accomplished in Paris and London in the mid-nineteenth century by Pierre Charles Alexandre Louis and his English students. Louis' investigations of typhus, typhoid fever and tuberculosis are still considered classics in both epidemiology and clinical medicine. His insistence on accurate data remains a keystone of sound epidemiologic work. *See* A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 31 n.7. Louis and his students were concerned with the specificity of disease, i.e., a precise definition of the disease which excludes all other diseases from consideration. *See* Temkin, *supra* note 83, at 61.

85. A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 134-35.

86. Stedman's Medical Dictionary 401 (23d ed. 1976).

87. Black's Law Dictionary 420 (5th ed. 1979).

88. Volvulus is one form of intestinal blockage in which the intestine twists upon itself, thereby causing an obstruction. As the lesion in this condition is grossly visible upon surgical entry into the abdominal cavity, the surgeon can readily ascertain the pathology upon such intervention. Indeed, attempting to define this condition based on its histology is nearly impossible due to the macroscopic nature of its pathology.

seeks to relate these histologic and/or other characteristics to the clinical signs and symptoms exhibited by affected patients. The patient's disease is thereby diagnosed.

Because the epidemiologist depends on laboratory tests and those clinical signs and symptoms noted by the clinician, he needs a measure of the accuracy of these clinical indicators as they relate to the definition of the disease. The two most commonly used measures of the accuracy of clinical diagnoses are "sensitivity" and "specificity."[89] "Sensitivity" is defined as the proportion of correct diagnoses as ascertained by clinical signs or symptoms and/or laboratory tests of those afflicted with the disease. The percentage of instances in which the disease is not so diagnosed when it is in fact absent is known as "specificity."[90]

To determine the sensitivity and specificity of a particular clinical diagnosis or laboratory test, the epidemiologist selects individuals known to have or not to have the disease, then applies the test to these individuals. If either sensitivity or specificity is low, the quality of the epidemiologist's data is correspondingly diminished.

B. *Determining the Relationship between Incidence of Disease and Exposure to a Factor*

Once the epidemiologist has defined the disease of interest, he seeks to compare the rate of disease development (incidence rate) among

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89. A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 150.

90. The following figure illustrates these concepts:

FIGURE 2

INDICES TO EVALUATE THE ACCURACY OF A TEST OR DIAGNOSTIC EXAMINATION: SENSITIVITY AND SPECIFICITY

Test or Examination	Disease Present	Disease Absent
Positive (Indicating disease is probably present)	A (true positives)	B (false positives)
Negative (Indicating disease is probably absent)	C (false negatives)	D (true negatives)
Totals	A + C	B + D

Sensitivity is defined as the percent of those who have the disease, and are so indicated by the test. Thus,

$$\text{Sensitivity (in percent)} = \frac{A}{A + C} \times 100$$

Specificity is defined as the percent of those who do not have the disease and are so indicated by the test. Thus,

$$\text{Specificity (in percent)} = \frac{D}{B + D} \times 100$$

those exposed to the factor of interest with the rate among those not so exposed. The incidence rate is a measure of the probability that an individual will develop the disease. Hence, the epidemiologist is interested in determining if exposure to the factor changes the probability that an individual will develop the disease.[91] If there is a gradation in the degree of exposure, the possibility of a corresponding gradation in incidence rates exists and merits investigation. The two principal approaches to collecting and analyzing morbidity/mortality data for exposed and non-exposed individuals are the demographic study and the epidemiologic study. In the former, the subjects within the two groups are viewed in the aggregate, while in the latter the subjects are viewed individually.[92] The results of demographic studies are used to generate etiologic hypotheses, which are then tested through epidemiologic studies.

1. The Demographic Study

Demographic studies explore either morbidity, if the investigator seeks to explain sickness, or mortality, if the investigator seeks to explain death. In either case, a study initially seeks to determine the accuracy and completeness of the statistics being analyzed and then attempts to ascertain how such statistics are related to possible etiologic factors, such as age, sex, cigarette consumption or asbestos exposure. One might, for example, examine the relationship between annual asbestos use in the United States from 1910-1950 and the annual mortality rates for mesothelioma in the United States from 1940-1980. Before drawing conclusions from the relationship between asbestos exposure and mesothelioma, however, the epidemiologist must determine the accuracy of the available mortality and exposure data in order to ensure that there has not been under- or over-reporting of either asbestos use or mesothelioma mortality. Studies have indicated that such data are available and accurate and that there is a positive relationship between asbestos use and mortality from mesothelioma.[93] Although such a positive correlation is supportive of a possible causal relationship between the two, it is by no means conclusive.[94]

No matter how compelling the findings in a demographic study, it must be recognized that such observations refer to groups and not to the individuals within the groups. A correlation may exist between a

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91. *See* A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 14, 191.

92. *See id.* at vii, 191-94.

93. National Cancer Institute, National Institute of Environmental Health Sciences & National Institute for Occupational Safety and Health, Estimates of the Fraction of Cancer in the United States Related to Occupational Factors 8-11 (1978) [hereinafter cited as Occupational Factors].

94. *See* Goodman, *Ecological Regressions and Behavior of Individuals*, 18 Am. Soc. Rev. 663, 663 (1953); Robinson, *Ecological Correlations and the Behavior of Individuals*, 15 Am. Soc. Rev. 351, 351-52, 357 (1950).

factor and the incidence of a disease even though no causal relationship exists. The classic example of this phenomenon is the linear relationship between pig iron production in the United States and the birth rate in Great Britain.[95] Clearly, such an association is spurious. This problem is known as an "ecological fallacy," and it imposes an inherent limitation on the use of demographic studies in inferring a causal relationship between a factor and a disease.[96] Demographic studies are used mainly to focus attention on a possible association between a factor and a disease, the elucidation of which requires further, more refined modes of study. In order to demonstrate the association in terms of the individual members of a group, the investigator utilizes the epidemiologic study.[97]

2. The Epidemiologic Study

The epidemiologic study attempts to explore and clarify a possible association between a factor and a disease within individuals in a population. For epidemiologists, it represents the application of the scientific method to human populations. In the scientific method, the investigator observes the effect of a single modification in the environment of one of two otherwise identical animals. Similarly, in an epidemiologic study, one seeks to observe the effect of exposure to a single factor upon the incidence of disease in two otherwise identical populations.

There are two major types of epidemiologic studies: experimental and observational.[98] In experimental studies, the epidemiologist assigns the exposure status to individuals. If the assignment is not performed randomly, it is termed a "community trial." The use of fluori-

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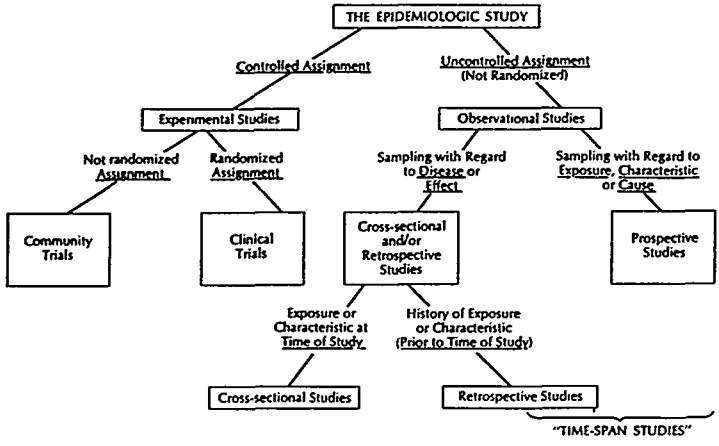
95. G. Snedocor & W. Cochran, Statistical Methods 189 (6th ed. 1967).
96. *See* A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 14.
97. *See id.* at 191.
98. *See id.* at 191-94. Figure 3 depicts the difference between the experimental and the observational study.

FIGURE 3
THE ANATOMY OF THE EPIDEMIOLOGIC STUDY



dation in water to prevent dental caries was tested in this way.[99] If the epidemiologist randomly assigns individuals to exposed and non-exposed groups, the study is a "clinical trial." The purpose of the randomization is to ensure that the only difference between the two groups is in the exposure; and that in all other respects, the groups are comparable.[100] Almost every new drug authorized for use by the Food and Drug Administration has been tested by such a clinical trial. While clinical trials are definitive studies,[101] they are not commonly encountered in toxic tort litigation because it is seldom possible to experiment by assigning individuals to an exposure.

The assignment of exposure, and thus an experimental study, is feasible only when it is ethical. It would be unethical, for instance, to assign individuals to exposure to cigarette smoking. The observational (non-experimental) study is uniquely suited to investigating situations in which controlled assignment is either unethical or difficult to achieve. In observational studies, the epidemiologist systematically observes the disease experience of individuals whose exposure status has been determined by themselves or by others in a nonrandomized manner. One might, for example, be interested in determining the difference in lung cancer incidence between smokers and non-smokers. If the epidemiologist views the population in terms of the individuals' exposure, the study type is "prospective." The investigator first determines if the individuals are cigarette smokers, then follows them over a sufficient number of years to see if their lung cancer incidence rate differs from that of non-smokers. If the epidemiologist views the populations in terms of individual disease status, the study is either "retrospective" or "cross-sectional." Retrospective studies focus on past exposure while cross-sectional studies consider current exposure. The investigator selects individuals who have or do not have lung cancer and then determines whether or not they are or have been cigarette smokers.

a. *Prospective Studies*

The prospective study is a powerful way to investigate the relationship between a factor and a disease because it closely approximates the classical scientific method. The investigator identifies two populations (or representative samples thereof), one composed of individuals who have been exposed to the factor and one of individuals who have not been so exposed.[102] Ideally, these populations will be otherwise identi-

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99. *See id.* at 5-6.

100. *See id.* at 257.

101. *See id.* at 256-57; D. Schwartz, R. Flemant & J. Lellouch, Clinical Trials (M. Healy trans. 1980).

102. A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 226; *see* J. Schlesselman, Case-Control Studies 14-15 (1982).

cal.[103] The investigator follows these populations for a period of time (possibly many years), observing the incidence rates of disease in each population. If the two groups are comparable, any difference in disease incidence can then be related either to the factor or to the sampling process, that is, to chance. Several statistical methods are available for assessing whether a difference in incidence rates results from sampling rather than from exposure to the factor.[104] After eliminating chance and determining that a statistically significant relationship between the disease and the factor exists, the epidemiologist's next task is to estimate the magnitude of the association. The accepted means of measuring such an association is the calculation of the

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103. If the two groups are not in fact comparable, statistical methods have been developed for adjusting the relative risk to account for the differences between them. J. Fleiss, Statistical Methods for Rates and Proportions 237-55 (2d ed. 1981); *see* Cochran, *Some Methods for Strengthening the Common* X^2 Tests, 10 Biometrics 417 (1954); Mantel & Haenszel, *Statistical Aspects of the Analysis of Data From Retrospective Studies of Disease*, 22 J. Nat'l Cancer Inst. 719, 730 (1959).

104. *See* P. Armitage, Statistical Methods in Medical Research (1971); J. Fleiss, *supra* note 103. In both books, every chapter relates in some way to how statistical studies should be performed, but of particular interest on the question of sampling are chapters 3 and 4 in Fleiss and chapter 6 in Armitage.

The importance of statistical significance testing is that it enables the investigator to determine if the difference observed between two samples represents a true difference between the populations or if it is instead the result of the sampling process. *See* D. Barnes, Statistics as Proof—Fundamentals of Quantitative Evidence 143-45 (1983). *See generally* I. Hacking, Logic of Statistical Inference (1965).

The investigator will usually state the hypothesis that there is no actual difference as the "null hypothesis." For example, in a study examining the mortality of cigarette smokers compared to that of non-smokers, the null hypothesis (H_o) would be that the mortality rates for both groups are the same, and thus that cigarette smoking has no impact on mortality (the status quo). Alternatively, the null hypothesis can be viewed as the statement that the investigator is seeking to disprove. In either case, the conjugate of H_o is H_1 (also termed H_a). The statistical significance test provides the probability that the observed difference is due to chance if H_o is, in fact, true. If that probability is sufficiently small (5% being the most-commonly used level), then the investigator "rejects" H_o, concludes that its conjugate, H_1, is true, and completes his investigation using H_1 as an established fact. (This analysis is sometimes done using confidence intervals, which are fully equivalent to significance tests.)

It should be noted that with a sufficient number of observations from each population, a statistically significant result will be observed for even very small differences, which may represent little or no biological difference. It should also be noted that the statistical significance test does not have anything to do with the evaluation of the remainder of the investigation. The determination of the probability of the observed events being attributable to random events, that is, secondary to the sampling process, does not in fact assign a probability level to the results of the investigation being "correct." Once the investigator has determined that the differences he has observed are not in fact the result of random chance, he has made his inference as far as the statistical significance tests are concerned, and he then goes on to complete the remainder of his investigation, including the determination of biological inferences, without recourse to the probability figure that he derived in conducting the statistical significance test.

relative risk, which is the ratio of the incidence rate of disease in the exposed group divided by that rate in the non-exposed "control" group.[105] If there is no association between the factor and the disease, the relative risk is 1.0; that is, the incidence rates for the exposed and non-exposed groups are equal.

The greater the magnitude of the observed relative risk, the stronger the association between the factor and the disease. If the factor were the only cause of the disease, the relative risk would be infinite because the incidence of disease in the unexposed group would be zero. Because most diseases have multi-factorial etiologies, however, it is rare to observe a relative risk greater than 10. When a relative risk of 10 or more is observed, one can be reasonably certain that it represents a causal relationship. For example, the relative risk for mesothelioma from asbestos exposure, which is widely recognized as causal, is between 50 and 80.[106] By comparison, the relative risk for leukemia in children who have been irradiated *in utero* is only 1.6 times that of children who were not so irradiated.[107] This represents a relatively small increase in the risk of developing leukemia for the irradiated children, which reflects a relatively weak causal relationship.

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105. *See* J. Fleiss, *supra* note 103, at 64-65; A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 209; Cornfield, *A Method of Estimating Comparative Rates from Clinical Data: Applications to Cancer of the Lung, Breast and Cervix*, 11 J. Nat'l Cancer Inst. 1269, 1269 (1951); Mantel & Haenszel, *supra* note 103, at 730. See Figure 4.

FIGURE 4

EXAMPLE OF COMPUTATION OF RELATIVE RISK

1. Groups A and B are assumed identical except for exposure to Factor F. (If not identical, there are methods of adjustment that still allow valid comparisons).

2. Incidence of disease D in Group A (exposed to Factor F) is 50 per 100,000 population. Incidence of the disease in Group B (not exposed) is 5 per 100,000.

3. Relative risk (r) of exposed to non-exposed is 50/5 = 10.0.

106. Love, *Biological Aspects of Associations Between Environmental Exposures and Cancer*, 37 Am. Statistician 413, 417 (1983).

107. Lilienfeld, *Epidemiology of Infectious and Non-Infectious Disease: Some Comparisons*, 97 Am. J. Epidemiology 135, 141 table 3 (1973). It should be noted that this relative risk was estimated from data collected in a retrospective study. It is presented as an illustration of the importance of the magnitude of the relative risk in making epidemiologic/biological inferences.

The prospective study, although very reliable, is difficult and expensive to conduct. It is not always possible to identify populations that are exposed and not exposed to a factor. Frequently, the epidemiologist is unable to follow the two groups for the period of time required. Hence, epidemiologists have developed and extensively used the retrospective study.

b. *Retrospective Studies*

Whereas a prospective study investigates the disease experience of exposed and non-exposed groups, the epidemiologist performing a retrospective study begins with individuals who already have (cases) or do not have (controls) the disease under investigation.[108] He then determines whether or not each individual has a past exposure to the factor, presumably prior to the onset of the pathologic process resulting in the disease. Cases are usually ascertained in a hospital setting. Control groups are commonly selected in several different ways, including: (1) "hospital controls," in which hospital patients who are not cases, but have different diseases, serve as controls;[109] (2) "population" or "neighborhood controls," in which a random sample of the case's neighbors or other similar groups constitutes the controls;[110] and (3) "matched population" or "matched neighborhood controls," in which population or neighborhood controls are matched to the cases so that various factors known or suspected to be unrelated to the disease are similarly distributed in the case and the control populations.[111] The retrospective study is inherently limited because one cannot directly ascertain disease incidence rates among the exposed and non-exposed groups; hence, the relative risk cannot be calculated directly.[112] There is, however, a statistic known as the "odds ratio"[113] that approximates the relative risk in those instances in which the disease incidence rate in the non-exposed population is low. As the odds ratio increases, so does the relative risk.

Retrospective studies in which hospital controls are used, unlike prospective studies, may be subject to a major bias in the selection of the controls, known as a "Berksonian bias."[114] The bias results from

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108. A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 194; *see The Case Control Study: Consensus and Controversy*, 32 J. Chronic Diseases 1 (1979).

109. *See* A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 196-97 & table 8-4.

110. *See id.* at 197 table 8-4.

111. *See id.* at 197-98 & table 8-4.

112. Cornfield, *supra* note 105, at 1269.

113. *See* Fleiss, *Confidence Intervals for the Odds Ratio in Case-Control Studies: The State of the Art*, 32 J. Chronic Diseases 69 (1979).

114. A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 202; *see* Berkson, *Limitations of the Application of Fourfold Table Analysis to Hospital Data*, 2 Biometrics 47, 49-51 (1946).

the differing probabilities of admission into the hospital for cases and hospital controls. If the probabilities of admission for each of these two groups are equivalent, there is no Berksonian bias.[115] The maximum increase in the observed odds ratio that a Berksonian bias usually produces in the absence of any relationship between a factor and a disease is approximately three.[116] Hence, if an odds ratio is observed to be greater than three, it is unlikely to have resulted entirely from the operation of a Berksonian bias.

c. *Cross-Sectional Studies*

Epidemiologic studies usually are concerned with relating antecedent exposure with subsequent disease occurrence. There are, however, occasions when the epidemiologist is interested in determining the relationship between current exposure and current disease status. This association can be elucidated by the cross-sectional study.[117] However, as diseases involved in toxic tort litigation generally have significant latency periods, cross-sectional studies are usually of little use in determining causation.

d. *Attributable Risk*

Observational studies are all directed at determining the relative risk of developing a disease that is associated with exposure to a factor. The relative risk, however, expresses only the magnitude of that association.[118] The statistical measure of a factor's relationship to a disease in the population is the "attributable risk."[119] It was originally described as the percentage decline in the population's disease incidence that would occur if the population's exposure to the factor were eliminated.[120] For example, the risk of lung cancer attributable to smoking in the United States today is approximately eighty percent. In other words, if smoking were eliminated in the United States, the incidence of lung cancer would decline by about eighty percent.[121]

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115. *See* A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 199-202.

116. Lilienfeld, *The Maximum Relative Risk Produced by a Berksonian Bias* (unpublished manuscript 1983) (available in files of *Fordham Law Review*). *See* A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 201-02.

117. A cross-sectional study is identical to a retrospective one except that the investigator is concerned with current exposure status. Therefore, it shares the retrospective study's limitation in estimating relative risks.

118. A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 217-18, 302.

119. *Id.* at 217.

120. Walter, *Calculation of Attributable Risks from Epidemiological Data*, 7 Int'l J. Epidemiology 175, 175 (1978); *see* Levin, *The Occurrence of Lung Cancer in Man*, 9 Acta Unio Internationala Contra Cancrum 531, 536 (1953).

121. A. Lilienfeld, Foundations of Epidemiology 256 (1st ed. 1976).

Alternatively, the attributable risk may be viewed as representing the proportion of the disease that is statistically attributable to the factor.[122] Using the example of lung cancer and cigarette smoking, one could say that cigarette smoking accounts for approximately eighty percent of the incidence of lung cancer in the United States. The attributable risk, therefore, is a composite measure that takes into account both the relative risk of disease if exposed and the proportion in the population so exposed.[123] It is an essential tool in examining the sufficiency of epidemiologic evidence.

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122. Walter, *The Distribution of Levin's Measure of Attributable Risk*, 62 Biometrika 371, 371 (1975).

123. From the equation in Figure 5, it can be seen that for the attributable risk to be high for a given factor (i.e., greater than 0.5), both the relative risk (r) and the proportion in the population so exposed (b) must be relatively large.

FIGURE 5

CALCULATION OF ATTRIBUTABLE RISK

$$\text{Attributable Risk} = \frac{b\,(r - 1)}{b\,(r - 1) + 1}$$

b = proportion of total population exposed to factor
r = relative risk

The table in Figure 6 shows how attributable risk varies within these parameters. If an investigator restricts the definition of exposure, thereby increasing the relative risk, the proportion of exposed people in the population would be lower and the attributable risk would remain approximately the same.

FIGURE 6

ATTRIBUTABLE RISKS AS A PROPORTION FOR SELECTED VALUES OF RELATIVE RISK AND PROPORTION OF POPULATION WITH THE CHARACTERISTIC*

b = Proportion of Population with Characteristic (percent)	r = Relative Risk			
	2	4	10	12
10	.09	.23	.47	.52
30	.23	.47	.73	.77
50	.33	.60	.82	.84
70	.41	.67	.86	.89
90	.47	.73	.89	.91
95	.49	.74	.90	.92

*Attributable Risk = $\frac{b\,(r - 1)}{b\,(r - 1) + 1}$

C. *Biological Inferences from Epidemiologic Data*

Demographic and epidemiologic studies both facilitate the elucidation of the statistical association between a factor and a disease. In order to draw the biological inference that a causal relationship exists, however, the epidemiologist must integrate additional scientific information. The derivation of such an inference requires rigorous consideration of laboratory, experimental, demographic and epidemiologic data.[124]

A causal inference must be biologically plausible and must conform to generally accepted theories. With the advent of the germ theory, criteria for determining whether a given bacteria caused a disease became necessary. Thus the Henle-Koch Postulates, developed in the nineteenth century, permitted the inference that a given species of bacteria, such as *Vibrio cholera*, is the etiologic agent of a given disease, such as Asiatic cholera. These postulates were:

1. The organism must be found in all cases of the disease in question.
2. It must be isolated from patients with the disease and grown in pure culture.
3. When the pure culture is inoculated into susceptible animals or man, it must reproduce the disease.[125]

The success of epidemiology in elucidating the relationship between non-bacterial causes of disease in the 1930's to the 1950's necessitated extension of the Henle-Koch Postulates in order to to derive biological inferences about the relationship between a factor and a disease.[126] Much of the initial work on these modifications was conducted with a view to establishing the relationship between cigarette smoking and lung cancer. As the breadth of epidemiology expanded, these ideas were generalized. They have been stated formally by Evans[127] and are

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124. It should be noted that it is possible to have an inadequately developed biological inference regarding the relationship between a factor and a disease, yet still have a statistically plausible relationship. *See* A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 315-16. The necessary biological knowledge may not be available at the time that the statistical association is found. An example of this occurrence is the relationship between oral contraceptives and various circulatory diseases. *Id.* at 315-16. When an association was discovered, there was no laboratory evidence to support a causal inference. However, the statistical association provided direction for laboratory workers in their research. The resulting laboratory data provided the necessary biological facts for the causal relationship to be stated. *Id.* at 316.

125. *Id.* at 292.

126. For the purposes of this Article, the following definition of a causal relationship will be used: "A causal relationship would be recognized to exist whenever evidence indicates that the factors form part of the complex of circumstances that increases the probability of the occurrence of disease and that a diminution of one or more of these factors decreases the frequency of that disease." *Id.* at 295.

127. Evans, *Causation and Disease: The Henle-Koch Postulates Revisited*, 49 Yale J. Biology & Med. 175 (1976).

now known as the Henle-Koch-Evans Postulates. Widely accepted by epidemiologists as the valid criteria for arriving at biological etiological inferences,[128] the postulates are:

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 other 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.[129]

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128. A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 317-18.

129. *Id.* The first three postulates embody the same concept, that is, that the incidence of disease should be greater in those exposed than in those not exposed for cross-sectional, retrospective and prospective studies. Postulate 4 refers to the epidemic curve, an epidemiologic concept originally developed for infectious diseases that is also applicable to such chronic diseases as cancer. *See* A. Lilienfeld & D. Lilienfeld, *supra* note 77, at 54-56. Postulates 5 and 6 relate to "host responses,"

Satisfaction of these criteria enables the epidemiologist to move beyond a correlation to form a biological inference that is applicable to all contemporary situations. The importance of the last criterion of the Henle-Koch-Evans Postulates cannot be over-emphasized because only its satisfaction can translate statements of statistical associations into inferences understandable within a biological context (concerning a pathophysiological process with a defined cause).

The approach to epidemiologic problems described above is a generally accepted one. Although specific aspects of that approach, such as the extensions made by Evans to the Henle-Koch Postulates, have changed over time, the basic framework of reasoning has remained essentially unaltered since its inception in the nineteenth century. The major change over the past 150 years has not been in the epidemiologic approach to disease problems per se, but rather in the precision and refinement of the methods used to make biological inferences.[130]

III. An Evidentiary Standard Combining the More-Likely-Than-Not Test and Epidemiology

A. *Requirement that Plaintiff Prove that Allegations of Causation Are More-Likely-Than-Not True*

Basic to this Article is the premise that a toxic tort plaintiff bears the burden of proving causation by a preponderance of the evidence.[131] The plaintiff is regarded as the legal aggressor, the one who wants the court to change the present state of affairs.[132] Thus "policy consider-

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which include such phenomena as fevers, increases in the levels of antibodies to a bacteria or virus, or increases in the number of white cells.

130. Lilienfeld & Lilienfeld, *A Century of Case-Control Studies: Progress?*, 32 J. Chronic Diseases 5, 13 (1979).

131. See *supra* note 10 and accompanying text. The burden of proof encompasses the burden of producing evidence and the burden of persuasion. The former imposes on a party the obligaton to present evidence theoretically sufficient to sustain his version of the facts at issue; the latter determines which side loses if the factfinder is not sufficiently convinced at the end of the trial. E. Cleary, *supra* note 10, § 336, at 783-84; *see* Belton, *Burdens of Pleading and Proof in Discrimination Cases: Toward a Theory of Procedural Justice*, 34 Vand. L. Rev. 1205, 1213 (1981). See *supra* note 10. This Article focuses on the production rather than the persuasion aspect of the burden of proof; its concern is the sufficiency of evidence. The two burdens are conceptually linked, however, because a decision as to whether a party has satisfied the production burden cannot be made without considering the degree of certainty required to meet the persuasion burden. See *infra* pt. IV(B) for a discussion of why the more-likely-than-not test results in the appropriate degree of certainty.

132. Louisell, *Construing Rule 301: Instructing the Jury on Presumptions in Civil Actions and Proceedings*, 63 Va. L. Rev. 281, 285 (1977); *see* Belton, *supra* note 131, at 1213; Cleary, *Presuming and Pleading: An Essay on Juristic Immaturity*, 12 Stan. L. Rev. 5, 7 (1959).

ations of fairness suggest that [he] should be required to prove his claim to relief."[133] While there have been exceptions to this general rule in cases not involving toxic torts,[134] the principles on which the exceptions have been based do not indicate that toxic tort defendants should bear the "burden of disproof" when toxic tort plaintiffs cannot produce sufficient evidence of causation.

Commentators usually discuss reversal of the burden of proof in the context of presumptions, which are created for reasons of policy, fairness and convenience.[135] Judicial analysis, however, usually reduces to evaluation of probabilities and consideration of which party has superior access to proof.[136] Neither of these factors weigh against the typical toxic tort defendant. Consider the presumption that a driver acts in the course of his employment when he drives a vehicle that is owned by his employer. "Although it is known that employees

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The similarity between civil procedure and the scientific method in dealing with those who seek to change the status quo is also instructive. Both law and science do, after all, strive to determine as nearly as possible what "really" occurs or has occurred, and both have developed means for making decisions in the face of uncertainty. It is interesting that science, like the law, generally insists that a new finding be well-established by evidence before it is accepted as part of the body of scientific knowledge.

133. Belton, *supra* note 131, at 1213.

134. *See, e.g.*, Wells v. Metropolitan Life Ins. Co., 107 Ga. App. 826, 831-32, 131 S.E.2d 634, 638 (1963) (presumption in contract case in plaintiff's favor that her pregnancy extended nine full months); Johnson v. Secretary of State, 406 Mich. 420, 440-42, 280 N.W.2d 9, 14-15 (1979) (presumption of negligence stemming from automobile driver's flight from accident in violation of statute).

135. *See* Belton, *supra* note 131, at 1217; Cleary, *supra* note 132, at 11; James, *Burdens of Proof*, 47 Va. L. Rev. 51, 65 (1961); Louisell, *supra* note 132, at 292-93. *But cf.* Laughlin, *In Support of the Thayer Theory of Presumptions*, 52 Mich. L. Rev. 195, 219 (1953) (balance of probabilities should be used to determine whether plaintiff's burden has been fulfilled). One relatively recent case listed eleven factors to be considered in allocating the burden of proof. Nelson v. Hughes, 290 Or. 653, 658-59, 625 P.2d 643, 645-46 (1981).

Commentators have disagreed about whether presumptions operate to shift both the burden of persuasion and the burden of production, or only the latter. *See* Allen, *Presumptions in Civil Actions Reconsidered*, 66 Iowa L. Rev. 843, 862-67 (1981) (mechanical use of presumptions should be discarded); Hecht & Pinzler, *Rebutting Presumptions: Order Out of Chaos*, 58 B.U.L. Rev. 527, 547-58 (1978) (distinguishing three situations in which presumptions arise); Ladd, *Presumptions in Civil Actions*, 1977 Ariz. St. L.J. 275, 283-88 (questioning whether all presumptions should be treated alike). *Compare* Laughlin, *supra*, at 209-12 (only production burden should be shifted), *with* Morgan, *Presumptions*, 12 Wash. L. Rev. 255, 281 (1937) (both burdens should be shifted).

136. James, *supra* note 135, at 66; *see* International Bhd. of Teamsters v. United States, 431 U.S. 324, 359 n.45 (1977) ("Presumptions shifting the burden of proof are often created to reflect judicial evaluations of probabilities and to conform with a party's superior access to the proof."). *But see* Dworkin, *supra* note 10, at 1161 (policy and fairness are determinative); Laughlin, *supra* note 135, at 219 (only presumptions based on probability are necessary).

sometimes use their employers' vehicles for purely private missions . . . that would constitute a distinct minority of cases."[137] Similarly, because services rendered in the context of a business relationship are not often performed gratuitously, a defendant denying an obligation to pay for such services would have the burden of proving that the obligation did not exist.[138] Such a common sense analysis of what is probable does not support making an exception to the general rule on proof of causation in toxic tort cases. Most diseases, including cancer, do not usually result from tortious conduct, or from exposure to identifiable man-made substances.[139]

Courts have also justified shifting the burden of proof because a defendant has superior access to evidence, but only under unusual circumstances such as when goods are damaged in a bailee's possession.[140] Such circumstances do not exist in most toxic tort cases because the problem encountered in determining causation is not the inaccessibility of evidence, but rather its non-existence or insufficiency. Epidemiologic analysis, the proper basis for recovery, can be performed by either plaintiffs or defendants.[141] A defendant may already possess the necessary records or data for an epidemiologic study, but given sufficient grounds for initiating a suit and a sufficient showing of relevance, discovery rules would make these available to the plaintiff. Thus, neither access to evidence nor probability warrants shifting the burden of proof to defendants in toxic tort cases. Toxic tort plaintiffs should be held to the same requirements as plaintiffs in most other tort actions. They should be required to produce evidence sufficient to establish that the substance at issue more likely than not caused the injury or disease in question.[142]

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137. Laughlin, *supra* note 135, at 215.

138. E. Cleary, *supra* note 10, § 337, at 787.

139. See *infra* note 220. One court, in holding for a toxic tort defendant, has explicitly noted the lack of such a general relationship between exposure and disease. Miller v. Olin Mathieson Chem. Corp., 398 S.W.2d 472, 473 (Ky. 1966) (noting that while organic chemical usage had increased, the overall incidence of leukemia, the disease at issue, had decreased).

140. James, *supra* note 135, at 66.

141. The only barrier to equal accessibility might be disparity in financial capabilities. No theory, however, would impose on a rich defendant the duty to develop a case for a poor plaintiff.

142. Requiring that a plaintiff sustain the burden of proof by a preponderance of the evidence derives from the practical objective of maximizing the number of cases decided correctly. Unlike criminal law, which is skewed toward avoiding incorrect guilty verdicts, tort law seeks to allocate neutrally the cost of damages or injuries. In most cases its goal is to minimize misallocation, which is best accomplished by using the more-likely-than-not test. *See* Cleary, *supra* note 132, at 13; Kaye, *The Limits of the Preponderance of the Evidence Standard: Justifiably Naked Statistical Evidence and Multiple Causation*, 1982 Am. B. Found. Research J. 487, 496-503. Applied to single-factor toxic tort cases, the long-term result of this rule is the payment by

B. *The Addition of the Attributable Risk Test to the Henle-Koch-Evans Postulates*

The Henle-Koch-Evans Postulates do not, by themselves, provide a complete legal standard because the determination of legal causation requires consideration of the degree of certainty required to meet the plaintiff's burden of proof. This deficiency can be remedied, however, by requiring in addition that the attributable risk for the factor at issue be greater than .50. 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 epidemiologic perspective, the question has two parts: (1) is the factor causally related to the disease (satisfaction of 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.[143]

C. *Practical Application of the Evidentiary Test*

Consider a manufacturing plant that employs 1000 production workers. At some work stations widget grinders emit widget dust. Studies of people exposed to this type of dust for ten or more years at concentrations higher than 100 dust particles per cubic centimeter have indicated a relative risk of 2.5 (compared to non-exposed per-

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defendants, taken collectively, of the total cost of the injuries they have caused to plaintiffs, taken collectively. The rule may break down in multi-factor cases, or in cases in which a defendant has very probably caused many, but not all, occurrences of a given disease in a relatively large population. In the latter situation either under-compensation or over-compensation of the plaintiffs, as a group, may result. These issues are discussed in the context of proportional liability, *infra* pt. V(B).

143. In using the Henle-Koch-Evans Postulates as constrained by attributable risk, great care must be taken in defining the exposure and the exposed population. In some instances, the focus should be on the total exposure above a certain level; in other cases the extent of exposure at any given time may be more important. The population of interest should be limited to individuals exposed at or beyond the level or extent at issue. For example, if the defined population included all steelworkers, it would be difficult to make inferences about the effects of prolonged high exposure to blast furnace fumes. New steelworkers and those who worked in rolling mills would not have suffered the same level of exposure as long-time blast furnace workers. To appreciate fully the problems that can be caused by improperly defining a population, consider a numerical example. Suppose that 10 of 50 blast furnace workers have a lung disease, that 100 of 1950 other steel workers have the same disease, and that 50 of 1000 non-steelworkers have it. Comparing the blast furnace workers to the general population yields a relative risk of 4, but if all steelworkers are considered, the relative risk drops to 1.1.

sons) for megabonkoma, a deadly (though fictional) form of lung cancer. If one of the widget workers contracts this terrible disease, could he establish through an epidemiologic study that it more likely than not resulted from widget dust exposure at the factory? Answering this question requires determining if the study results satisfy the Henle-Koch-Evans Postulates, and if the worker in question was exposed to widget dust for a long enough period and at a high enough concentration.

To test evidence against the Henle-Koch-Evans Postulates one must consider a number of factors. For example, breathing dust is more likely to cause a lung disease, such as megabonkoma, than a bone disease. This would support the inference of a causal connection. Studies that indicate a correlation between megabonkoma in rats and exposure to widget dust would tend to confirm human data and would further support the inference. Such biological information, together with a sufficiently large population sample, an absence of serious biases and a consistent and verified relative risk of 2.5 would probably support the inference that widget dust causes some cases of megabonkoma. The widget worker, however, would still have to establish both exposure and a sufficiently high attributable risk.

If the worker in question had held his job for over ten years, and had worked in a part of the factory where widget dust exceeded 100 particles per cubic centimeter, exposure would be quite clear, and the attributable risk of .60 would easily satisfy the more-likely-than-not test.[144] For situations in which sufficient exposure is certain, any relative risk greater than 2 would lead to an attributable risk of more than .50.[145] More typical, however, is the situation in which exposure is questionable. Perhaps the worker performed a number of tasks at various locations in the plant or used different machines that emitted varying amounts of dust. Under these circumstances, one could estimate the probability that exposure exceeded the level in the study. If only sixty percent of the 1000 workers were heavily exposed, the attributable risk would drop to .47,[146] even with a relative risk of 2.5. This evidence would fail the more-likely-than-not test and would not support a plaintiff's verdict.

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144. $\frac{1.0\ (2.5 - 1)}{1.0\ (2.5 - 1) + 1} = .60$

See *supra* note 123 and accompanying text.

145. If the proportion of the populations exposed is 1.0, as in *supra* note 144, then:

$$\frac{1.0\ (2+z-1)}{1.0\ (2+z-1)+1} = \frac{(1+z)}{(2+z)}$$

which is greater than 0.50 for any positive z.

146. $\frac{.6\ (2.5-1)}{.6\ (2.5-1)+1} = .47$

Note that this example is somewhat oversimplified. It assumes that at any exposure less than 10 years and 100 particles per cubic centimeter the relative risk is 1.0.

A worker who contracted megabonkoma after high exposure for less than ten years might still be able to establish causation if he could produce evidence that the total amount of dust inhaled was an adequate measure of exposure. A person exposed at a relatively low level for more than ten years could make a similar argument. In no case, however, can evidence suffice to establish a causal link if it does not include at least reasonable estimates of exposure levels and durations, and data that reasonably indicate a relative risk greater than 2.[147]

IV. Precedents and Requirements for the Introduction of Epidemiologic Evidence

A party seeking to introduce scientific evidence faces two general requirements: The methods used to obtain data and to draw inferences therefrom must be legally acceptable, and the witnesses through whom the evidence is introduced must be suitably qualified.[148] Precedent supports not only admitting epidemiologic proof into evidence,[149] but also requiring that such proof be produced by a toxic tort plaintiff. Precedent also supports a rule requiring that a medical expert be qualified as an epidemiologist before testimony on causation is admitted in a toxic tort case.

A. *Precedents for Admitting Epidemiologic Proof into Evidence*

In cases involving diseases caused by viruses or bacteria, courts have generally accepted epidemiologic evidence with little difficulty,[150] and

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147. The foregoing discussion leaves open many questions about the detailed application of the Henle-Koch-Evans Postulates, and about what constitutes a reasonable indication of relative risk. In actual cases, expert witnesses would probe the many complications and subtleties that have been omitted. At least one court has recognized that there is "room for responsible epidemiologists to differ significantly on many of the key choices and assumptions to be made in analyzing [a] causal relationship." O'Gara v. United States, 560 F. Supp. 786, 789 (E.D. Pa. 1983). To be sufficient, however, the testimony of experts should fall within the proposed framework.

148. *See* 3 J. Weinstein & P. Berger, Weinstein's Evidence §§ 702[01]-[04] (1982).

149. For an excellent bibliography and discussion of the admissibility and use of scientific evidence, see *Symposium on Science and the Rules of Evidence*, 99 F.R.D. 187 (1983). *See generally* Gianelli, *The Admissibility of Novel Scientific Evidence:* Frye v. United States, *a Half-Century Later*, 80 Colum. L. Rev. 1197, 1235-45 (1980) (discussion of the standards used to determine admissibility); Korn, *Law, Fact, and Science in the Courts*, 66 Colum. L. Rev. 1080, 1108-1113 (1966) (discussion of the process through which courts incorporate scientific principles and discoveries); McCormick, *Scientific Evidence: Defining a New Approach to Admissibility*, 67 Iowa L. Rev. 879, 882-83 (1982) (same).

150. *See, e.g.*, Kehm v. Procter & Gamble Mfg. Co., 724 F.2d 613, 617-20 (8th Cir. 1983) (toxic shock syndrome case in which court admitted into evidence epidemiologic reports from the Center for Disease Control); Wolf v. Procter & Gamble Co., 555 F. Supp. 613, 624-26 (D.N.J. 1982) (same); Travelers Ins. Co. v. Donovan,

there exists no rationale for treating such evidence differently in toxic tort cases. In fact, even in some toxic tort cases, courts have alluded to the concept of comparing incidence rates.[151] Some commentators have objected to this approach because the evidence is not specific to the plaintiff,[152] but they ignore the fact that even " '[p]articularistic' evidence offers nothing more than a basis for conclusions about a perceived balance of probabilities."[153] Other commentators have lamented that courts tend not to accept epidemiology,[154] but the basis for this assertion is unclear. In fact, good epidemiologic evidence is not only accepted by courts; in at least one case, it has been required.[155]

B. *Precedents for Incorporation of Epidemiologic Postulates into an Evidentiary Standard*

A number of precedents amply support an evidentiary standard incorporating scientific principles and requiring that evidence conform to them. Some courts have even measured evidence against the Ewing Postulates,[156] despite serious questions about their validity in legal proceedings and problems in applying them objectively. The postulates of epidemiology are far better established than Ewing's, and should be more readily used as the basis for a standard against which to test the sufficiency of evidence. Insofar as epidemiology involves statistics, decisions in a number of cases, not all involving toxic torts, have demonstrated the ability of courts to judge intelligently the validity of statistical inferences.[157]

1. Discrimination Cases

In discrimination cases, which often hinge on the statistical significance of the difference between the composition of a population and

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125 F. Supp. 261, 262 (D.D.C. 1954) (tuberculosis case in which workers' compensation claimant was awarded recovery based on increased a priori risk), *aff'd*, 221 F.2d 886 (D.C. Cir. 1955); Sacred Heart Med. Center v. Carrado, 92 Wash. 2d 631, 637, 600 P.2d 1015, 1019 (1979) (hepatitis case in which recovery was allowed based on plaintiff's elevated a priori risk of contracting the disease).

151. See *supra* note 58 and accompanying text.

152. Dickson, *supra* note 57, at 799-808; Dore, *supra* note 57, at 431.

153. Rosenberg, *supra* note 5, at 870.

154. See *supra* note 57.

155. Heyman v. United States, 506 F. Supp. 1145, 1149 (S.D. Fla. 1981). See *infra* notes 180-82 and accompanying text.

156. *See, e.g.*, Stordahl v. Rush Implement Co., 148 Mont. 13, 19-20, 417 P.2d 95, 99 (1966); Sikora v. Apex Beverage Corp., 282 A.D. 193, 196, 122 N.Y.S.2d 64, 66 (1953), *aff'd*, 306 N.Y. 917, 119 N.E.2d 601 (1954); Dennison v. Wing, 279 A.D. 494, 496-97, 110 N.Y.S.2d 811, 813-14 (1952).

157. *See* Rosenberg, *supra* note 5, at 870-71.

the composition of a work force, jury panel or the like, courts have shown great understanding of the value of testing hypotheses against data. The Supreme Court, in two 1977 discrimination cases,[158] explicitly approved the type of significance testing used in the statistical part of an epidemiologic study. A third case decided that year involved similar though less explicit reasoning.[159] *Castaneda v. Partida*[160] dealt with grand jury selection practices in Hidalgo County, Texas. Although the population was approximately eighty percent Hispanic, grand jury participation over a ten-year period averaged only thirty-nine percent Spanish surnamed, with the highest annual figure just over fifty percent.[161] The chance of such disproportionate representation was extremely low, assuming no discrimination. The Court, therefore, rejected this null hypothesis[162] and held that, absent rebuttal evidence, the alternative hypothesis of discrimination should be accepted.[163] *Hazelwood School District v. United States*[164] and *International Brotherhood of Teamsters v. United States*[165] involved discriminatory hiring practices alleged to be in violation of Title VII of the Civil Rights Act of 1964.[166] In *Teamsters*, the Court explicitly approved the use of statistics to establish a prima facie case of discrimination, but did not delve into details of methodology.[167] In *Hazelwood*, however, it endorsed the more rigorous statistical approach used in *Castaneda*.[168]

These and subsequent cases[169] clearly establish the ability of courts to understand and use classical hypothesis testing techniques. They do not, however, address the basic issue of tort causation. A statistically significant difference in a discrimination case shifts the burden of

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158. Hazelwood School Dist. v. United States, 433 U.S. 299 (1977); Castaneda v. Partida, 430 U.S. 482 (1977).

159. International Bhd. of Teamsters v. United States, 431 U.S. 324 (1977).

160. 430 U.S. 482 (1977).

161. *Id.* at 486-87 & n.7.

162. *See id.* at 494 & n.13. See *supra* note 104.

163. 430 U.S. at 496 n.17. The Court noted that the likelihood that random selection would produce the jury panels actually selected in Hidalgo County was less than 1 in 10^{140}.

164. 433 U.S. 299 (1977).

165. 431 U.S. 324 (1977).

166. *Hazelwood*, 433 U.S. at 301; *Teamsters*, 431 U.S. at 328.

167. 431 U.S. at 339.

168. 433 U.S. at 308 n.14, 311 n.17.

169. *See, e.g.*, Plemer v. Parsons-Gilbane, 713 F.2d 1127, 1137 (5th Cir. 1983); Harris v. Birmingham Bd. of Educ., 712 F.2d 1377, 1383 (11th Cir. 1983); Chisholm v. United States Postal Serv., 665 F.2d 482, 494-95 (4th Cir. 1981); EEOC v. United Virginia Bank, 615 F.2d 147, 149-54 (4th Cir. 1980).

proof, and absent rebuttal evidence, establishes the plaintiff's allegations as facts. In a toxic tort case, the difference not only must be statistically significant, but also must be sufficiently large to make it more likely than not that the individual plaintiff's injury resulted from the defendant's substance. The standard proposed in this Article would also require consistency with the Henle-Koch-Evans Postulates.

The following example illustrates the difference between discrimination and toxic tort cases. Evidence that a company employs a workforce that is only fifteen percent black from a population that is twenty percent black might conclusively prove discrimination. A twenty percent disease rate in a population exposed to a chemical, however, would not prove tort causation in an individual case if the unexposed population experienced a fifteen percent rate. The maximum attributable risk would be only twenty-five percent.[170] Moreover, even if the exposed population had a disease rate of forty-five percent, and the attributable risk were as high as sixty-seven percent,[171] the Henle-Koch-Evans Postulates would still have to be satisfied.

2. Identity Cases

Criminal law is another area in which courts have examined statistical evidence. Proving the identity of a criminal often involves the use of circumstantial evidence indicating that certain events would be very unlikely to occur by coincidence. Attempts to quantify this mode of proof through statistics, however, have generally foundered. The best known example is *People v. Collins*,[172] in which a white woman and a black man in a yellow car committed an assault and robbery. The defendants fit this description, and the prosecution introduced evidence that the probability of these factors occurring together by coincidence was extremely slight. The jury found the defendants guilty, but the Supreme Court of California overturned the conviction because the method used to compute the probability of coincidence was flawed, and because the unlikelihood of coincidence did not establish the probability that the accused couple was guilty.[173] In a large population, even a rare combination could be expected to occur more than just once. Therefore, without resorting to a controversial

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170. $\frac{1\ (20/15-1)}{1\ (20/15-1)+1} = .25$

See *supra* note 123 and accompanying text.

171. $\frac{1\ (45/15-1)}{1\ (45/15-1)+1} = .67$

See *supra* note 123 and accompanying text.

172. 68 Cal. 2d 319, 438 P.2d 33, 66 Cal. Rptr. 497 (1968).

173. *Id.* at 327-31, 438 P.2d at 38-40, 66 Cal. Rptr. at 502-05.

technique known as Bayesian analysis,[174] one cannot directly ascribe a probability to the hypothesis of guilt.[175]

The *Collins* problem limits all hypothesis testing. It is because statistics in themselves do not determine probability[176] that the non-statistical postulates of epidemiology are so extremely important. To reach a scientifically sound opinion that a causal link more likely than not exists, one must integrate other information. The overall epidemiologic approach and the need for a substantive standard are both illustrated by the swine flu cases, which constitute the best judicial use of epidemiology to date.

3. Swine Flu Cases

In 1976, fear of an impending influenza epidemic prompted rapid implementation of a swine flu inoculation program[177] before final testing of the vaccine could be completed. As a result, no drug company would manufacture the vaccine until the federal government agreed to assume all liability.[178] Thus, the swine flu cases were tried under the Federal Tort Claims Act[179] before federal district judges and without a jury, and many of the opinions did not reach the issue of

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174. *See* Finkelstein & Fairley, *A Bayesian Approach to Identificiation Evidence*, 83 Harv. L. Rev. 489, 498-501 (1970).

175. *Id. But see* Tribe, *Trial by Mathematics: Precision and Ritual in the Legal Process*, 84 Harv. L. Rev. 1329, 1365-76 (1971) (rejecting the use of Bayesian analysis).

176. For paternity cases, some courts, *e.g.*, Cramer v. Morrison, 88 Cal. App. 3d 873, 884-85, 153 Cal. Rptr. 865, 871-72 (1979); Malvasi v. Malvasi, 167 N.J. Super. 513, 515-16, 401 A.2d 279, 280 (1979); *see, e.g.*, Lascaris v. Laredo, 100 Misc. 2d 220, 221-23, 417 N.Y.S.2d 665, 666-67 (1979), legislatures, *e.g.* Ariz Rev. Stat. Ann. § 12-847(c) (West 1982), and even the ABA, Abbott, *Joint AMA-ABA Guidelines: Present Status of Serologic Testing in Problems of Disputed Parentage*, 10 Fam. L.Q. 247, 257 (1976), have embraced statistics without giving adequate attention to this problem. The accuracy of modern blood-typing techniques permits the exclusion of at least 90% of falsely identified men. That is, rejection of the null hypothesis of non-paternity is wrong only about 10% of the time. This does not, however, establish the probability that the alternative hypothesis is true. It only means that the null hypothesis does not conform well to the data. What then is the probability that a man not excluded is in fact the father of the child? The answer cannot be derived solely from the test results. Important assumptions about the number of possible putative fathers must be made. For a discussion of the misuse of the new testing techniques, see Ellman & Kaye, *Probabilities and Proof: Can HLA and Blood Group Testing Prove Paternity?*, 54 N.Y.U. L. Rev. 1131 (1979).

177. For a discussion of the history of the swine flu program, see *In re* Swine Flu Immunization Prods. Liab. Litig., 533 F. Supp. 567, 571-72 (D. Colo. 1980).

178. *See id.* at 572. The government agreed to assume liability because otherwise the manufacturers would be subject to strict products liability claims for any defects in the manufacture of the vaccine. *Id.*

179. 28 U.S.C. §§ 1346(b), 2401(b), 2671-2680 (1976 & Supp. V 1981).

legal sufficiency. They did, however, discuss in detail the judicial evaluation of the evidence involved.

Epidemiologic analysis figured decisively in most of the swine flu cases. For example, the court in *Heyman v. United States*[180] rejected the plaintiff's claim because she attempted to prove her case without epidemiologic evidence. The court found that clinicians generally cannot determine "whether a relationship exists between an illness and a preceding event such as a vaccination,"[181] and held that "without at least some reference to epidemiological studies, [the] plaintiff's position that her illness was caused by the swine flu shot amounts to nothing more than speculation."[182]

Central to the swine flu litigation was an epidemiologic study that indicated a relative risk of greater than 2 for Guillain-Barre Syndrome (GBS) up to ten weeks after swine flu inoculation.[183] If the exposed (vaccinated) population is perfectly defined, a relative risk of 2 corresponds to an attributable risk of 0.50.[184] The study induced the government to settle almost all cases in which the plaintiff contracted GBS within ten weeks of his swine flu shot.[185] Thus, the plaintiffs in virtually all of the reported cases either contracted GBS more than ten weeks after their inoculations, or contracted a disease other than GBS. One of these cases exemplifies the proper use of epidemiology; another shows the need for an epidemiologic evidentiary standard.

In *Cook v. United States*,[186] the plaintiff GBS victims experienced the onset of the disease approximately twelve weeks after their swine flu inoculations.[187] The district court disallowed their claims after a detailed and perceptive discussion of the use of epidemiology.[188] The judge discussed the connection between a relative risk of 2 and the more-likely-than-not standard, and also determined that the court should consider the two non-statistical factors of alternative explanations and biological credibility. [189]

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180. 506 F. Supp. 1145 (S.D. Fla. 1981).
181. *Id.* at 1149.
182. *Id.*
183. Schonberger, Bregman, Sullivan-Bolyai, Keenlyside, Ziegler, Retailliau, Eddins & Bryan, *Gullian-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 Am. J. Epidemiology 105, 112-13 (1979). The study also discussed attributable risk, *id.* at 111-13, but this was not used in any of the legal analyses.
184. $\frac{1\,(2\text{-}1)}{1\,(2\text{-}1)+1} = .50$
185. Hall & Silbergeld, *supra* note 5, at 446.
186. 545 F. Supp. 306 (N.D. Cal. 1982).
187. *Id.* at 307; *see* Padgett v. United States, 553 F. Supp. 794, 804 (W.D. Tex. 1982).
188. 545 F. Supp. at 315-16.
189. *Id.* at 314-15.

At the other extreme is *Sulesky v. United States*,[190] in which the plaintiff first exhibited signs of GBS more than three months after her injection.[191] She introduced epidemiologic testimony that conflicted with a government report that had previously been relied on in many cases.[192] This so confused the court that it turned to the testimony of treating and evaluating physicians, who apparently did not even discuss the disease's relative rate of occurrence.[193] Nonetheless, the court, relying on their testimony, held for the plaintiff.[194] Without a substantive standard for review, an appellate court faced with the *Cook* and *Sulesky* verdicts would have to uphold both, although only the first could be rationally explained. This Article's proposal would provide both trial and appellate courts with the required standard.

C. *Qualifications for Expert Witnesses Giving Testimony About Epidemiology*

No court has yet determined the qualifications necessary for a witness to offer expert testimony about epidemiology. In general, a witness need only have such "knowledge, skill, experience, training, or education" in the field at issue to "make it appear that his opinion . . . will probably aid the trier in his search for truth."[195] In *Jenkins v. United States*[196] it was held that a psychologist could, under some circumstances, give psychiatric testimony. The court cited an earlier case in which it had been held that "a general practitioner may testify concerning matters within a medical specialty if his education or experience, or both, involves demonstrable knowledge of the subject."[197]

Under the *Jenkins* test and the proposed standard, a medical doctor could testify about toxic tort causation only if he could demonstrate knowledge of epidemiology.[198] The preference often accorded treating physicians should not apply because a standard based on the drawing of inferences from populations does not require detailed knowledge of the plaintiff's individual case. Moreover, a medical degree would not

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190. 545 F. Supp. 426 (S.D. W. Va. 1982).
191. *Id.* at 429.
192. *Id.* at 429-30.
193. *See id.* at 430-31.
194. *Id.* at 431.
195. Fed. R. Evid. § 702; *see* Jenkins v. United States, 307 F.2d 637, 643 (D.C. Cir. 1962) (quoting McCormick, Law of Evidence § 13 (1954)).
196. 307 F.2d 637 (D.C. Cir. 1962).
197. *Id.* at 643-44 (citing Sher v. DeHaven, 199 F.2d 777, 782 (D.C. Cir. 1952)).
198. Sufficient knowledge might be established in a number of ways, including coursework or membership in appropriate professional organizations. In Kubs v. United States, 537 F. Supp. 560 (E.D. Wis. 1982), a witness' testimony was rejected in part because the work on which it was based had never been subjected to peer review. *Id.* at 562.

necessarily be required because many epidemiologists do not have one. The best witness, of course, would be a medical doctor thoroughly trained in epidemiology, because the need to integrate biology, statistics and common sense to draw proper inferences requires as broad a background as possible.

V. "First Case," Under-Compensation and Over-Compensation Problems

A. *The "First Case" Problem*

The use of epidemiology to determine the legal sufficiency of evidence would eliminate much of the inconsistency and irrationality from judicial decisions in which the causation of a latent disease is at issue. It would, however, also make it difficult for victims to prove causation prior to the development of adequate data, and thus would create a special problem for early victims.[199] In response to this "first case" problem, some commentators have proposed scientifically questionable rules to ease the plaintiff's burden of proof.[200] These proposals would, in effect, remove all rational limits on liability, a step for which most proponents give no theoretical justification beyond an unfocused desire to compensate.[201]

The unlimited liability that would result from relaxed evidentiary standards is best illustrated by one proposal that would require a

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199. Several of the swine flu cases involved claims that diseases such as polymyostis, Tabaczynski v. United States, 529 F. Supp. 156, 161 (E.D. Mich. 1981), *aff'd*, 711 F.2d 1059 (6th Cir. 1983), or arthritis, Gicas v. United States, 508 F. Supp. 217, 220 (E.D. Wis. 1981), were caused by swine flu inoculations. Most were rejected because a single isolated temporal coincidence is not sufficient evidence. One expert in *Tabaczynski* pointed out that a single case could not support statistical inferences. 529 F. Supp. at 62. *But see* Hasler v. United States, 517 F. Supp. 1262, 1271 (E.D. Mich. 1981) (onset of arthritis after inoculation was found not to be a coincidence), *rev'd*, 718 F.2d 202 (1983).

The modification of the statute of limitations in several states to allow a plaintiff to bring an action after a causal relationship is discovered is implicitly based on the recognition that scientifically establishing causation often requires time for the accumulation of data. *See* Stoleson v. United States, 629 F.2d 1265, 1269 (7th Cir. 1980).

200. *See, e.g.*, Hall & Silbergeld, *supra* note 5, at 442-43 (extrapolation of epidemiologic studies representing unusual subgroups in population to larger group to establish causation based on assumption that different species react similarly to different substances); *Tort Actions for Cancer*, *supra* note 5, at 855-59 (calling for government maintenance of a catalog of exposure levels at which particular carcinogenic substances will cause cancer; presumption of causation is created if plaintiff can show exposure above the threshold level).

201. *See* Burcat, *supra* note 5, at 857-59; Soble, *A Proposal for the Administrative Compensation of Victims of Toxic Substance Pollution: A Model Act*, 14 Harv. J. on Legis. 683, 768 (1977); *Precursor Symptoms*, *supra* note 2, at 194; *Environmental Risks*, *supra* note 5, at 587.

plaintiff to prove only exposure "significant enough to trigger disease."[202] According to some theories, significance could be found in very low exposures.[203] Thus adoption of the proposal could make nearly everyone potentially liable to countless people. Simply breathing releases traces of suspect organic compounds.[204]

Other proposals would use methods employed in making regulatory decisions to establish rebuttable presumptions of causation in tort actions.[205] Rebuttal, however, would require the same kind of studies needed to establish causation under the proposed standard. If the burden of proof were shifted to defendants in this way, the loss, in the absence of any information on causation, would be transferred to them.[206] Such a change would be at odds with recognized legal principles.

A clear distinction currently exists between the standard of proof used in regulation and the standard used in determining tort liability. Most legislation governing the regulation of potentially toxic substances requires far less convincing proof of harmfulness than would satisfy the more-likely-than-not test.[207] As a result, regulatory agencies have employed methods that would not meet the proposed test of evidentiary sufficiency. In particular, agencies have banned or limited certain substances on the basis of animal studies backed by little, if any, human data.[208] This type of analysis may be appropriate in protective regulation, but it does not satisfy the more-likely-than-not test[209] and should not, as some have argued, carry over to tort cases.[210]

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202. Hall & Silbergeld, *supra* note 5, at 445.

203. S. Epstein, The Politics of Cancer 3 (1978).

204. *See* ABA Section of Science & Technology, *Law, Science and Technology in Health Risk Regulation II*, 22 Jurimetrics J. 380, 381 (1982) (statement of Dr. Leon Golberg).

205. See *supra* note 5.

206. *See* Robinson, *supra* note 5, at 729 (placement of the burden of proof is dispositive of factual issue of causation); Rosenberg, *supra* note 5, at 866 n.65 ("shifting the burden would simply replace one bias with another").

207. *See* Reserve Mining Co. v. EPA, 514 F.2d 492, 520 (8th Cir. 1975) (reasonable medical concern for public health suffices to sustain agency action), *modified*, 529 F.2d 181 (1976); Environmental Defense Fund v. EPA, 510 F.2d 1292, 1298 (D.C. Cir. 1975) (the standard of "substantial evidence" means something less than the weight of the evidence); *see also* Maines, *Offensive Collateral Estoppel in Mass Tort or Products Liability Cases: The Potential for Corporate Catastrophe from Prior Administrative Proceedings*, 35 Admin. L. Rev. 327, 329-30 (1983) (lesser standard of proof in administrative hearings). *But see* Industrial Union Dep't v. American Petroleum Inst., 448 U.S. 607, 653 (1980) (more-likely- than-not test).

208. Environmental Defense Fund V. EPA, 510 F.2d 1292, 1299 (D.C. Cir. 1975).

209. Latin, *The "Significance" of Toxic Health Risks: An Essay on Legal Decisionmaking Under Uncertainty*, 10 Ecology L.Q. 339, 377-80 (1982).

210. *Id.*

The pitfalls of making conclusory legal leaps from mouse to man prevent rational extrapolation even in apparently extreme cases. Dioxin, for example, is a potent human toxin that may also be a carcinogen. In animals, its carcinogenic potency exeeds that of aflatoxin B, known as perhaps the most potent human carcinogen.[211] Its toxic effects, however, vary by a factor of 5000 in comparisons between tests using guinea pigs and hamsters.[212] If dose-response information for guinea pigs does not apply to another species of rodent, animal data are obviously not a reliable basis for making quantitative conclusions about exposed humans.[213] For regulatory purposes the existing evidence about dioxin may support the most stringent of limitations, but statements about likelihood in tort cases require more. Even within the regulatory context courts have recognized that human epidemiologic data should be given more weight than the results of animal testing. In *Dow Chemical Co. v. Blum*,[214] an epidemiologic study, albeit weak, sufficed to sustain an EPA order banning certain herbicides,[215] while in *Gulf South Insulation v. Consumer Product Safety Commission*,[216] the failure to consider epidemiologic evidence resulted in the reversal of a ban on the use of urea-formaldehyde foam insulation.[217]

The use of regulatory or other lesser standards in tort actions has been advocated by at least one proponent as necessary to achieve the tort system's goals of compensation, deterrence and retributive justice.[218] Relaxing evidentiary standards, however, would serve only the goal of compensation[219] and would unjustifiably single out the victims of certain diseases for special treatment. If society's only concern is compensation, why should lung cancer victims receive money when cystic fibrosis or multiple sclerosis victims do not? Even more to the point, why should a lung cancer victim who can demonstrate an exposure speculatively related to the disease receive compensation when other victims do not? If compensation is the only goal, it is best uncoupled from tort liability.

In addition to creating a crazy-quilt pattern of payments to disease victims, focusing only on compensation would seriously impair the

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211. Friedman & Weckesser, *Dioxin and Resource Recovery*, Envtl. F., Sept. 1983, at 44, 46.

212. Rawls, *Dioxin's Human Toxicity is Most Difficult Problem*, Chem. & Eng'g News, June 6, 1983, at 37; *see* Mays, *Dioxin: Deadly or Deceptive?*, Envtl. F., Feb. 1984, at 13, 14.

213. *See* Mays, *supra* note 212, at 13-14.

214. 469 F. Supp. 892 (E.D. Mich. 1979).

215. *Id.* at 907.

216. 701 F.2d 1137 (5th Cir. 1983)

217. *See id.* at 1146.

218. *Environmental Risks*, *supra* note 5, at 575.

219. *Id.*

other goals of the tort system. Retribution unrelated to fault and causation is meaningless. As to deterrence, defendants would have little incentive to alter their conduct because they would be held liable to many victims even when they did not in fact cause their injuries. Most potential targets of toxic tort litigation are industrial concerns, but cancer and other latent diseases would continue to occur even if they completely ceased production. Far fewer cancers are tied to specific substances or activities than many have assumed.[220]

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220. It is generally estimated that 60-90% of all cancers are linked in some way to the environment. Sixth Annual Report of the Council on Environmental Quality 33 (1975). This does not mean, however, that prevention of 60-90% is practicable. *See* Doll & Peto, *supra* note 55, at 1205-07; Higginson & Muir, *Environmental Carcinogenesis: Misconceptions and Limitations to Cancer Control*, 63 J. Nat'l Cancer Inst. 1291, 1296 (1979). Nor does it mean that man-made chemicals cause most cases of the disease, as some have concluded. *See Tort Actions for Cancer*, *supra* note 5, at 840-41. Even were this true, it would not justify relaxing evidentiary standards to facilitate almost universal recovery against chemical manufacturers when specific chemicals are not implicated.

Despite the complexity of environmental carcinogenesis, advocates of reducing the plaintiff's burden of proof have based their proposals in part on estimates that 20-40% of all cancers result from workplace exposures. The source of this estimate and its infiltration into legal commentary is an interesting story in itself. In 1978 a group of scientists from several federal agencies put together a report that contained the 20-40% figures. Occupational Factors, *supra* note 93, at 22. This report was widely criticized, and at least two of the authors later conceded that they had "relied on some assumptions about data that have been shown subsequently to be incorrect." Davis, Bridbord & Schneiderman, *Estimating Cancer Causes: Problems in Methodology, Production, and Trends*, 9 Banbury Rep. 285, 308 (1981). Nonetheless the estimates were defended by others, including Dr. Samuel Epstein, one of the most vocal critics of the American industrial establishment's use and control of suspect substances. Epstein & Swartz, *Fallacies of Lifestyle Cancer Theories*, 289 Nature 127 (1981). Dr. Epstein's book, The Politics of Cancer, *supra* note 203, formed the basis for many of the assertions on which one of the proposals for shifting the burden of proof was based. *See Tort Actions for Cancer*, *supra* note 5, at 848-50 (analysis focusing on the overall relationship of cancers to chemicals but implicitly incorporating Dr. Epstein's use of the high occupational estimates); Note, *Occupationally Induced Cancer Susceptibility: Regulating the Risk*, 96 Harv. L. Rev. 697, 697 n.3 (1983) (dealing with regulation rather than tort law, citing the original 20-40% estimate). Thus, legal commentators persist in propagating scientific overstatement.

To obtain such high figures, one must unrealistically assume that all workers are exposed to potential carcinogens at the highest reported rates. *See* Doll & Peto, *supra* note 55, at 1240-41. One realistic epidemiologic analysis of the occupational cancer issue indicates that 4% is a far more appropriate estimate. *Id.* at 1245. Other reasonable estimates range from 1% to 10%. Wynder & Gori, *supra* note 55, at 830. It has not been determined what portion of this 1-10% receives legal compensation, but it is clear that for occupationally-caused cancer the potential for tort system dysfunction is, at worst, far less than the actual dysfunction assumed by supporters of relaxed standards. Furthermore, environmental exposures are generally much less concentrated than those experienced in the workplace, indicating that the overall dysfunction is exaggerated as well.

Thus, the first case problem does not warrant changes in tort law principles. It does, however, still require that the difficulty of collecting sufficient data to satisfy an evidentiary standard derived from epidemiology be addressed. There are legal and institutional reforms that would reduce this burden without compromising principles or creating unlimited liability. One of the most irrational barriers to recovery results when the statute of limitations precludes a claim because a disease manifests itself too long after exposure,[221] or when scientific knowledge linking exposure and disease comes too late after manifestation. As is already the law in many states,[222] the statutory period should commence with a plaintiff's illness, if the causal link is known at that time, or when causation becomes reasonably apparent.[223]

Another appropriate legal reform would be the adoption of procedural changes at the state level that would facilitate joint collection of evidence by plaintiffs. Within the federal court system, consolidation of cases for purposes of discovery has already proven useful in mass personal injury cases.[224] So, too, has the federal class action device.[225]

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221. *See, e.g.*, Steinhardt v. Johns-Manville Corp., 54 N.Y.2d 1008, 430 N.E.2d 1297, 446 N.Y.S.2d 244 (1981). In *Steinhardt*, the New York Court of Appeals held that an action for disease resulting from occupational exposure to asbestos was barred by the statute of limitations because it was commenced more than four years after the plaintiff's last employment-related exposure. *Id.* at 1010, 430 N.E.2d at 1298-99, 446 N.Y.S.2d at 245-46.

222. Annot., 1 A.L.R. 4th 117, 127-34 (1980).

223. *See, e.g.*, Large v. Bucyrus-Erie Co., 707 F.2d 94, 96-97 (4th Cir. 1983); Grabowski v. Turner & Newell, 516 F. Supp. 114, 118-20 (E.D. Pa.), *aff'd*, 651 F.2d 908 (3d Cir. 1980) (per curiam). Locke v. Johns-Manville Corp., 221 Va. 951, 958-59, 275 S.E.2d 900, 905 (1981). For a discussion of this rule as applied in federal courts, see Davis v. United States, 642 F.2d 328, 331 (9th Cir. 1981), a case involving polio vaccine. The *Davis* court refused to delay tolling of the statute until negligence as well as causation was discovered.

224. *E.g.*, *In re* Swine Flu Immunization Prods. Liab. Litig., 446 F. Supp. 244, 246 (J.P.M.D.L. 1978) (per curiam), *vacated*, 687 F.2d 14 (1982); *In re* A.H. Robins Co., "Dalkon Shield" IUD Prods. Liab. Litig., 419 F. Supp. 710, 712 (J.P.M.D.L. 1976) (per curiam); *In re* A.H. Robins Co., "Dalkon Shield" IUD Prods. Liab. Litig., 406 F. Supp. 540, 542 (J.P.M.D.L. 1975) (per curiam); *see* Note, *The Judicial Panel and the Conduct of Multidistrict Litigation*, 87 Harv. L. Rev. 1001, 1002-09 (1974).

225. *E.g.*, *In re* Three Mile Island Litig., 87 F.R.D. 433, 442 (M.D. Pa. 1980); Payton v. Abbott Labs., 83 F.R.D. 382, 387-88 (D. Mass. 1979). Other cases have denied class certification. *E.g.*, *In re* Northern Dist. of Cal., Dalkon Shield IUD Prods. Liab. Litig., 693 F.2d 847, 850-51 (9th Cir. 1982), *cert. denied*, 103 S. Ct. 817 (1983); *In re* Federal Skywalk Cases, 680 F.2d 1175, 1182-83 (8th Cir.), *cert. denied*, 103 S. Ct. 342 (1982); Ryan v. Eli Lilly & Co., 84 F.R.D. 230, 234 (D.S.C. 1979); *see* Seltzer, *Punitive Damages in Mass Tort Litigation: Addressing the Problems of Fairness, Efficiency and Control*, 52 Fordham L. Rev. 37, 69-71 (1983). For a good bibliography on class actions, see McGovern, *Management of Multiparty Toxic Tort Litigation: Case Law and Trends Affecting Case Management*, 19 Forum 1, 9 n.18 (1983).

Not all states permit such combined efforts, and to the extent that they do not, more liberal rules should be adopted.[226]

Of course, scientific research is not optimally conducted with the primary aim of preparing data for litigation. For the long term, a coordinated research effort is required, an effort which certain institutional changes would promote. Increased funding for governmental agencies that collect and analyze epidemiologic data would be a first step, but government agencies should not do all the work.[227] The establishment of a fund for research at universities or by other relatively disinterested private sector individuals or groups would diversify the information gathering effort. This fund could be provided at least in part by interested industries. Few institutional mechanisms for such participation now exist, but a number of changes are possible. These range from direct payments by industry for research, made perhaps in conjunction with labor unions, to the establishment of an umbrella organization to distribute money paid according to some form of cost allocation system.[228]

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226. 301(e) Study, *supra* note 3, at 257.

227. *See* Letter of Lilienfeld & Lilienfeld to the editors of Science, 198 Science 250-53 (Oct. 21, 1977) (suggesting a sort of Brookings Institution for science).

228. Research will not, of course, solve all causation problems, but even initial studies may have legal uses. In the case of some occupational diseases, it may be possible to establish a relationship between working in a particular industry and the incidence of the diseases, though great care must be exercised in interpreting the data. In one Finnish study of how cancer incidence varied by occupation, almost all of the differences were found to be associated with different cigarette consumption habits of the people who tended to go into the occupations under investigation. Pukkala, Teppo, Hakulinen & Rimpela, *Occupation and Smoking as Risk Determinants of Lung Cancer*, 12 Int'l J. Epidemiology 290, 293-95 (1983). For an example of how the performance of certain tasks within an occupation may be implicated, see American Iron & Steel Inst. v. OSHA, 577 F.2d 825, 832 (3d Cir. 1978) (relationship between lung cancer and exposure to coke oven emissions), *cert. dismissed*, 448 U.S. 917 (1980).

Evidence derived from occupational studies can facilitate proof within the workers' compensation context even without identifying a particular substance, and it can assist in further pinpointing the cause. The rubber industry provides an excellent example of how research studies can be done through industry-university cooperation without government funding or coercion. The 1970 union contract with the rubber industry provided for a comprehensive occupational research program. The Schools of Public Health at Harvard University and the University of North Carolina at Chapel Hill contracted with both the United Rubber Workers and the major U.S. rubber companies to do the work. They found, among other things, that certain cancers were more common in rubber workers than in the general population, though overall, the excess mortality for all cancers was minimal. *See* McMichael, Andjelkovic & Tyroler, *Cancer Mortality Among Rubber Workers: An Epidemiologic Study*, 271 Annals of the N.Y. Acad. of Science 125, 136 (1976); Monson & Fine, *Cancer Mortality and Morbidity Among Rubber Workers*, 61 J. Nat'l Cancer Inst. 1047 (1978). Similar industry-wide efforts should be encouraged in the future.

In addition to increasing and improving research, steps to insure better investigation of suspect cases of certain diseases could also be taken. The interests of society have already led to statutory requirements that certain deaths of unclear origin automatically fall under the jurisdiction of a coroner. These requirements greatly aid in collecting the information necessary to determine or prove if a crime has been committed. Similarly, to insure that occurrences of a disease possibly related to a toxic tort are properly and adequately documented, they should, by statute, come within the coroner's or some other health officer's jurisdiction.[229] This would not only make it easier to determine causation in the particular case under investigation, but would also generate data for general use in drawing epidemiologic inferences.

B. *Under-Compensation and Over-Compensation*

In some circumstances an epidemiologic standard would cause a radical shift from non-compensation to over-compensation. Until sufficient evidence of causation is developed, all plaintiffs would lose; afterwards, assuming there exists adequate proof of exposure and other necessary elements of the legal theory being pursued, all would likely win.[230] To avoid this dichotomy, and to allow some recovery when the burden of proof is not met, a few commentators have suggested proportional recovery.[231]

Under the proportional approach, if thirty percent of the a priori risk of a disease were attributable to a defendant, the plaintiff would recover thirty percent of his damages from that defendant. Likewise,

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229. Autopsy of *all* deaths should be encouraged. Autopsy, which often reveals false diagnoses, is now on the decline in the United States, a trend that should be reversed. *See* Lundberg, *Autopsies as the Doctor's—and Patient's—Best Friend*, J. A.M.A., September 2, 1983, *reprinted in* Baltimore Sun, Oct. 30, 1983, at K5.

230. Whether collateral estoppel on the issue of causation would be applied is an open question. The issue has been considered in the context of the asbestos litigation. *See* Baldwin, *Asbestos Litigation and Collateral Estoppel*, 17 Forum 772, 781-83 (1982); Comment, *An Examination of Recurring Issues in Asbestos Litigation*, 46 Alb. L. Rev. 1307, 1330-31 (1982); Note, *Applying Offensive Collateral Estoppel to Asbestos Cases: A Viable Alternative*, 16 Suffolk U. L. Rev. 687, 702-06 (1982). Even without collateral estoppel, however, once sufficient evidence is collected for one case, it will probably be available for use in other cases. *See* McGovern, *supra* note 225, at 8. This militates against hasty expansion of the scope of collateral estoppel. *See generally* Maines, *supra* note 207 (discussing problems that might be created by expansion of the scope of collateral estoppel based on administrative findings).

231. *See, e.g.*, Estep, *supra* note 57, at 281-86; Rizzo & Arnold, *supra* note 5, at 1407-13; Robinson, *supra* note 5, at 743-49. One recent proposal for proportional recovery would combine the concept with rebuttable presumptions. *Environmental Risks*, *supra* note 5, at 614-15. For the reasons discussed *supra* notes 135-42, this proposal is highly questionable.

if the risk were seventy percent, recovery would be limited to that percentage of the damages. Such verdicts are not possible today.[232] Although some courts have apportioned liability among several defendants when the harmfulness of the substance involved was not at issue,[233] no such award has ever been based on the probability of harmfulness.[234]

Proportional recovery would shift the focus of legal analysis from the individual case to the tortfeasor who has caused many, but not all, injuries. It would allow some plaintiffs to recover who, given perfect information, would not. It would also mean something less than complete recovery for those who would receive full compensation under the traditional rules. The tortfeasor would pay the full cost of the damage it had caused, but not necessarily to the parties it actually injured. One commentator has described this result as being "actuarially fair,"[235] and another has justified it on the ground that the law should prefer inexact justice to manifest injustice.[236]

Whether proportional recovery would in fact be more just than the present all-or-nothing rule remains an open question. Adoption of the theory would not dramatically reduce the difficulties faced by toxic tort plaintiffs. Its rational implementation would require epidemiologic evidence similar to that required to satisfy the proposed standard. Attributable risks of less than fifty percent would not preclude recovery, but data to support reasonable estimates of attributable risk would still be required.

The net effect of proportional recovery would depend on factors that require further investigation and research. The feasibility of detecting small attributable risks must be determined. It must also be determined whether more cases involve attributable risks above fifty percent or below fifty percent. If relatively small impacts on incidence rates defy detection, a theory intended to assist plaintiffs who cannot

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232. Apportionment today depends primarily on the nature of the plaintiff's injury, rather than on the conduct of defendants. *See* W. Prosser, *supra* note 11, § 52, at 314.

233. A good example is Sindell v. Abbott Labs., 26 Cal. 3d 588, 607 P.2d 924, 163 Cal. Rptr. 132, *cert. denied*, 449 U.S. 912 (1980), a DES case in which the court used a "market share" theory of liability. *Id.* at 611-13, 607 P.2d at 937, 163 Cal. Rptr. at 145; *see* Comment, *DES and a Proposed Theory of Enterprise Liability*, 46 Fordham L. Rev. 963, 995-1000 (1978). *But see* Sheffield v. Eli Lilly & Co., 144 Cal. App. 3d 583, 592-99, 192 Cal. Rptr. 870, 875-80 (1983) (refusing to apply the market share theory).

234. One court, however, by denying a defendant's motion for summary judgment, implied that the *Sindell* theory might apply to the issue of harmfulness as well as to identity of the party responsible for exposing the plaintiff. Pereira v. Dow Chem. Co., 129 Cal. App. 3d 865, 872-73, 181 Cal. Rptr. 364, 368 (1982).

235. Robinson, *supra* note 5, at 747.

236. Delgado, *supra* note 5, at 895.

meet the more-likely-than-not test would actually do them little good, and what relief it did provide would come at the expense of other plaintiffs who would recover more under the traditional test as incorporated in the proposed standard. If in the majority of cases, the attributable risk is above fifty percent, adoption of the theory might do more injustice than justice, even if small impacts were detectable. These potential problems may prove to be more imagined than real, but until they have been carefully researched and considered, the law should not rush to embrace the theory of proportional recovery.[237]

CONCLUSION

In both toxic tort and cancer cases, courts have generally done a poor job in determining whether evidence of causation is sufficient to meet the plaintiff's burden of proof. Failure to formulate and apply substantive standards has led to irrational and inconsistent results, a problem that need not continue. Accepted legal principles governing the burden of proof, combined with the principles of epidemiology provide an excellent basis for a standard that rationally conforms to general tort law principles and for which there is ample precedent.

Requiring that a plaintiff's evidence satisfy the postulates of epidemiology would work to the disadvantage of the first victims of a

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237. For single-factor cases, the traditional preponderance of the evidence rule may, as a practical matter, be the best possible standard. Multiple factor cases, however, present additional issues. Consider a plaintiff who has suffered high level exposures to a number of substances, each produced by a different defendant. If the plaintiff has a disease linked to all the substances, proving by a preponderance of the evidence that any single defendant caused it may be difficult. Depending on the levels of exposure and the relative risks for the substances considered separately, each defendant might escape liability only because of the other defendants.

The alternative liability theory, developed in Summers v. Tice, 33 Cal. 2d 80, 84-86, 199 P.2d 1, 2-5 (1948), would not apply to such a situation unless extended. *See* Molloy & Thomas, *Causation Problems in Design Defect Litigation*, Legal Notes & Viewpoints, Feb. 1983, at 35, 44-45 (the doctrine has only rarely been applied in product liability cases). In many toxic tort cases, strict liability rather than negligence is involved, and it is uncertain if all potential defendants have been included. With few exceptions, the case law indicates that this makes alternative liability inapplicable. Other theories formulated to reach multiple defendants would also not apply. These theories are: (1) "concert of action," requiring a common plan or design, which will not often occur in multiple defendant toxic tort cases involving multiple substances; (2) "enterprise liability," requiring industry-wide standards which, unless a single product such as blasting caps is implicated, is unlikely to be useful, and which would therefore be inapplicable in multiple-substance cases; and (3) "market share liability," which is the theory adopted in Sindell v. Abbott Labs., 26 Cal. 3d 588, 611-13, 607 P.2d 924, 936-38, 163 Cal. Rptr. 132, 144-45 (1980). It is difficult to see how a single relevant market could be defined for multiple substances, and thus how the theory could apply to cases involving more than one substance. Thus, some form of causal apportionment among the defendants might well be the best way to allocate liability in a case involving multiple high-level exposures, though further research is required.

substance that eventually proves to have harmful effects. The magnitude of this problem is not, however, as great as many have assumed, and the legal and institutional reforms appropriate to its solution do not involve reducing the plaintiff's burden of proof. Using an epidemiologic standard could cause a sharp shift from under- to over-compensation, but adoption of a proportional recovery standard to ameliorate this problem could create even more serious problems. Further theoretical development of this concept is necessary before it can be recommended. In any event, consistent and rational resolution of toxic tort claims requires that the law incorporate the principles of epidemiology and that legal reforms conform to epidemiologic reality.