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Troyen A. Brennan

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CAUSAL CHAINS AND STATISTICAL LINKS: THE ROLE OF SCIENTIFIC UNCERTAINTY IN HAZARDOUS-SUBSTANCE LITIGATION

Troyen A. Brennan†

Many commentators recognize proof of causation as the paramount obstacle to just resolution of tort claims based on injury from toxic substances.[1] Causation has thus become the focus of a number of essays and investigations.[2] Some authors have argued persuasively for a restructured tort law system that could compensate hazardous-substance injuries appropriately.[3] Others maintain that common law courts cannot cope with the sophisticated social and scientific problems raised by such injuries.[4] The latter group argues that a regulatory agency could better resolve hazardous-substance-induced injury claims.[5]

The debate between those who support modifications of tort law doctrine and those who support an administrative approach to

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† Instructor, Harvard Medical School, and Lecturer, Harvard Law School. B.S., Southern Methodist University, 1975; M.A., Oxford University, 1978; M.P.H., M.D., J.D., Yale University 1984.

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1 *See* Pierce, *Encouraging Safety: The Limits of Tort Law and Government Regulation*, 33 VAND. L. REV. 1281, 1298 (1980); Trauberman, *Statutory Reform of "Toxic Torts": Relieving Legal, Scientific, and Economic Burdens on the Chemical Victim*, 7 HARV. ENVTL. L. REV. 177, 197 (1983).

2 Robinson, *Probabilistic Causation and Compensation for Tortious Risk*, 14 J. LEGAL STUD. 779 (1985); Delgado, *Beyond Sindell: Relaxation of Cause-in-Fact Rules for Indeterminate Plaintiffs*, 70 CALIF. L. REV. 881 (1982); Robinson, *Multiple Causation in Tort Law: Reflections on the DES Cases*, 68 VA. L. REV. 713 (1982); Note, *Pursuing a Cause of Action in Hazardous Waste Pollution Cases*, 29 BUFFALO L. REV. 533 (1980).

3 Rosenberg, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System*, 97 HARV. L. REV. 851 (1984); Note, *Toxic Substance Contamination: The Risk-Benefit Approach to Causation Analysis*, 14 U. MICH. J.L. REF. 53 (1980).

4 Ginsberg & Weiss, *Common Law Liability for Toxic Torts: A Phantom Remedy*, 9 HOFSTRA L. REV. 859 (1981); Schwartz, *Products Liability, Corporate Structure, and Bankruptcy: Toxic Substances and the Remote Risk Relationship*, 14 J. LEGAL STUD. 689 (1985); Note, *Environmental Health: An Analysis of Available and Proposed Remedies for Victims of Toxic Waste Contamination*, 7 AM. J.L. MED. 61 (1981).

5 *See* Pierce, *supra* note 1, at 1320-21; Trauberman, *supra* note 1, at 215.

hazardous-substance injury has continued throughout the 1980s,[6] but has produced little in the way of legislative initiatives or judicial activism.[7] This inaction most likely results from the inability of judges and law-makers to discern a clear advantage between common law solutions and regulatory approaches. Meanwhile, there are increasing sigus of courts' inability to grasp scientific evidence of causation.[8]

Deciding between common law modifications and a regulatory approach might be facilitated if one alternative appeared clearly superior for resolving causation issues. Hazardous-substance litigation presents a myriad of procedural and substantive problems.[9] Discussion of these issues may, however, detract from an analysis of the central problem of the courts' difficulty with resolving toxic-substance-caused injury. Thus, in this Article, I heed the following advice from Professor Fleming James:

> What is needed is a separation between the issue of cause and all the other issues, which are often meritorious in themselves but too frequently parade meretriciously in the guise of cause. Perhaps this would affect substantive results only a little, but it would contribute much to clarity of thought.[10]

In both legal and scientific reasoning, causation is an epistemo-

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6 Congress initiated the most thorough discussion of alternatives to tort litigation as part of the original Superfund legislation. *See* SUPERFUND SECTION 301(E) STUDY GROUP, SENATE COMM. ON THE ENV'T AND PUB. WORKS, 97TH CONG., 2D SESS., INJURIES AND DAMAGES FROM HAZARDOUS WASTES—ANALYSIS AND IMPROVEMENT OF LEGAL REMEDIES (Comm. Print 1982) [hereinafter SUPERFUND STUDY]. A blue-ribbon committee convened by a not-for-profit organization produced a similar study. *See* KEYSTONE CENTER, POTENTIAL APPROACHES FOR TOXIC EXPOSURE COMPENSATION: A REPORT ON THE CONCLUSIONS OF A KEYSTONE CENTER POLICY DIALOGUE (1985) [hereinafter KEYSTONE REPORT].

7 There are indications that a hybrid approach to asbestos-induced disease compensation may be developed. *See* Wellington, *Asbestos: The Private Management of a Public Problem*, 33 CLEV. ST. L. REV. 375 (1984-85). Moreover, as jury awards to victims of hazardous substances increase, industry may actively support new, perhaps more rational, approaches to hazardous substance injury compensation. *See* Skeen v. Monsanto Chem. Co., Nat'l L.J., Dec. 29, 1986, at 4, col. 3 (S.D. Tex. Dec. 12, 1986) ($100,000,000 jury verdict for benzene exposed worker who died of leukemia).

8 *See infra* notes 130-71 and accompanying text.

9 *See generally* Goggin & Brophy, *Toxic Torts: Workable Defenses Available to the Corporate Defendant*, 28 VILL. L. REV. 1208 (1982-83) (theories for recovery keep increasing while defenses decrease; procedurally, patterns of jury instruction vary greatly); Shelton, *Defending Cancer Litigation: The Causation Defense*, 24 FOR DEF. 8 (1982) (discussing difficulties that defendants face in toxic substance litigation); Trauberman, *supra* note 1, at 189 (discussing problems arising from long latency periods); SENATE COMM. ON ENV'T AND PUB. WORKS, 96TH CONG., 2D SESS., SIX CASE STUDIES OF COMPENSATION FOR TOXIC SUBSTANCES POLLUTION: ALABAMA, CALIFORNIA, MICHIGAN, MISSOURI, NEW JERSEY, AND TEXAS (Comm. Print 1980) (detailing difficulties that victims have in gaining compensation).

10 James & Perry, *Legal Cause*, 60 YALE L.J. 761, 811 (1951).

logical construct that allows one to investigate the unknown and to define and overcome uncertainty. Legal notions of causation, however, differ in important ways from scientific causal concepts. Hazardous-substance litigation exaggerates these differences, and therefore often produces results that neither satisfy the goals of justice nor demonstrate an understanding of scientific knowledge. In this Article, I clarify the differences between legal causation and the scientific concepts of causation that underlie the evidence linking hazardous substances with injuries. I then argue that these differences do not permit just and efficient resolution of hazardous-substance-induced injury claims through tort litigation. I conclude by outlining the regulatory apparatus that could provide compensation for, and internalize the costs from, hazardous-substance-caused injuries.

At the outset, I outline the roles of causation and uncertainty in the epistemology of scientific investigation. The evolution of the philosophy of science has produced a different understanding of the relationship of uncertainty to investigation than existed a century ago. More important, the causal concepts that derived from Newtonian physics are quite similar to those that inform the law, although they are no longer essential to science. Thus, by tracing the evolution of the philosophy of science, one can juxtapose legal and scientific notions of causation.

I then apply insights gleaned from an analysis of this evolution to a discussion of the legal theory of causation. The function of causation in law is not simply to imitate current scientific knowledge; rather, the law uses causal concepts not only to link events, but also to provide moral prescriptions. Thus, attempts to combine the non-Newtonian causal concepts used by scientists with the legal notions of causation can produce disastrous outcomes, as my analysis of case law demonstrates.

Only a thorough understanding of the specifics of scientific uncertainty and causation can lead to the best formula for compensation and deterrence of hazardous-substance injury. After developing a framework for classifying the uncertainty involved in toxic-substance injury, I evaluate the advantage of a regulatory approach over common law tort actions for dealing with causation problems. Then, by analyzing several case studies, I corroborate conclusions that the theoretical model of uncertainty suggests.

My analysis focuses on hazardous-substance injury. I define a hazardous substance as a product, environmental pollutant, or occupational exposure that produces a disease process in human be-

ings.[11] This definition cuts across legal distinctions between products liability, environmental, occupational safety, and nuisance law.[12] The use of this broad definition is justified if the evidence used to link such substances to disease is similar enough to suggest generic approaches to compensation and deterrence.

I
THE HAZARDOUS SUBSTANCE PROBLEM

Proposing sweeping changes in the law is justified only if the problem is large enough to warrant such an effort. Is the threat represented by hazardous substances great enough to require reformulation of tort law, such as Rosenberg proposed,[13] or the development of a regulatory structure such as that proposed by the Superfund Section 301(e) subcommittee?[14] In the last ten years a large number of law journal articles have made gloomy predictions and called for various reforms.[15] Although the legal system has adopted few, if any, of these reforms, the system has not collapsed, nor are there clear indications of a current epidemic of hazardous substance-produced injury. Why then should the legal community be concerned?

There is no easy answer to this threshold question. Occupational toxins, environmental contaminants, and unsafe consumer products can cause an overwhelming variety of diseases including birth defects,[16] an assortment of pulmonary diseases,[17] skin dis-

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11 *See infra* text accompanying notes 172-74. Others have presented a different topology of toxic substance. *See* Page, *A Generic View of Toxic Chemicals and Similar Risks*, 7 ECOLOGY L.Q. 207 (1978). I avoid discussion of toxins that produce economic or aesthetic injury without harming human life. *See* Halter & Thomas, *Recovery of Damages by States for Fish and Wildlife Losses Caused by Pollution*, 10 ECOLOGY L.Q. 5 (1982); Wood, *Requiring Polluters to Pay for Aquatic Natural Resources Destroyed by Oil Pollution*, 8 NAT. RESOURCES L. 545 (1975).

12 This Article centers on the generic causation problems presented by hazardous substances, regardless of the legal doctrine under which they arise. Certainly, hazardous substance causation problems arise under many different doctrines. *See* SPECIAL COMM. ON THE TORT LIABILITY SYSTEM, AM. BAR ASS'N, TOWARDS A JURISPRUDENCE OF INJURY: THE CONTINUING CREATION OF A SYSTEM OF SUBSTANTIVE JUSTICE IN AMERICAN TORT LAW (1984).

13 Rosenberg, *supra* note 3, at 916-29.

14 *See* SUPERFUND STUDY, *supra* note 6, part IV, at 196-246.

15 *See, e.g.*, Pierce, *supra* note 1; Ginsberg & Weiss, *supra* note 4; Trauberman, *supra* note 1.

16 Anesthetic gases produce birth defects in laboratory rats. *See* Fink, Shepard & Blandau, *Teratogenic Activity of Nitrous Oxide*, 214 NATURE 146 (1967). Female operating room personnel have a higher incidence of spontaneous abortions. *See* Ad Hoc Committee on the Effect of Trace Anesthetics on the Health of Operating Room Personnel, American Society of Anesthesiologists, *Occupational Disease Among Operating Room Personnel*, 41 ANESTHESIOLOGY 321 (1974).

17 Asbestos is the most frightening example. *See infra* notes 215-31 and accompanying text. Coal workers' pneumoconiosis is a serious cause of morbidity in coal mining

eases,[18] neurological disorders,[19] heart diseases,[20] blood dyscrasias,[21] and gastrointestinal disorders.[22] Diseases caused by these substances produce an almost inestimable amount of morbidity and mortality.

Hazardous substances also cause cancer. Cancer is the second leading cause of death in this country. In 1984, cancer caused roughly 400,000 deaths.[23] Although the magnitude of morbidity and mortality represented by cancer is huge, the critical question for this inquiry is, what proportion of cancer does exposure to hazardous substances cause? Indeed the extent to which control of environmental and occupational carcinogens will significantly decrease the incidence of cancer has become a central issue in the debate over alternatives to tort litigation.[24]

The extent to which exposure to hazardous substances causes cancer is a subject of great debate. In a widely-cited study, Barth and Hunt asserted that environmental phenomena induce 85-90%

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areas. *See* Carpenter & Cochrane, *Death Rates of Miners and Ex-miners With and Without Coalworkers' Pneumoconiosis in South Wales*, 13 BRIT. J. INDUS. MED. 102 (1956). Silica causes debilitating silicosis in exposed sandblasters. *See* Samimi, Weill & Ziskind, *Respirable Silica Dust Exposure of Sandblasters and Associated Workers in Steel Fabrication Yards*, 29 ARCHIVES ENVTL. HEALTH 61 (1974). Grain workers and farmers develop reactive airway disease after chronic exposure to organic dusts. *See* Chan-Yeung, Schulzer, MacLean, Dorken & Grzybowski, *Epidemiologic Health Survey of Grain Elevator Workers in British Columbia*, 121 AM. REV. RESPIRATORY DISEASES 329 (1980).

18 Occupational toxins cause a wide variety of skin diseases. *See* H. MAIBACH & G. GELLIN, OCCUPATIONAL AND INDUSTRIAL DERMATOLOGY (1982).

19 Various solvents and heavy metals cause difficult-to-detect behavioral and neurological disorders. *See* Allen, *Chemical Neurotoxins in Industry and Environment*, in 2 THE NERVOUS SYSTEM: THE CLINICAL NEUROSCIENCES 235 (D. Tower ed. 1975); Feldman, Hayes, Younes & Aldrich, *Lead Neuropathy in Adults and Children*, 34 ARCHIVES OF NEUROLOGY 481, 483 (1977).

20 *See* Herd, Lipsky & Martin, *Cardiovascular Effects of 1,1,1-Trichloroethane*, 28 ARCHIVES ENVTL. HEALTH 227 (1974).

21 Benzene is the best recognized hematological toxin. *See* Askoy, Erdem & Dincol, *Leukemia in Shoe-Workers Exposed Chronically to Benzene*, 44 BLOOD 837 (1974). Other substances will also damage blood cells. *See* Crawford, *Aplastic Anaemia Due to Trinitrotoluene Intoxication*, 2 BRIT. J. MED. 430, 431 (1954).

22 *See* Cohn, Boylan, Blanke, Fariss, Howell & Guzelian, *Treatment of Chlordecone (Kepone) Toxicity with Cholestyramine*, 298 NEW ENGLAND J. MED. 243 (1978).

23 *See* 2 NATIONAL CENTER FOR HEALTH STATISTICS, PUBLIC HEALTH SERV., U.S. DEP'T OF HEALTH AND HUMAN SERVS., VITAL STATISTICS OF THE UNITED STATES, 1984, pt. A, at 226 (1987). Of this total, approximately 121,000 deaths resulted from lung cancer, 38,000 from breast cancer and 17,000 from leukemia. *See* 1 V. DEVITA, S. HELLMAN & S. ROSENBERG, CANCER: PRINCIPLES AND PRACTICE OF ONCOLOGY 168 (2d ed. 1985). Twenty-five years of progress in cancer chemotherapy, radiation therapy, oncological surgery and cancer biology have done little to blunt crude or age-adjusted incidence rates. Some writers have suggested that research shift from treatment to prevention. Bailar & Smith, *Progress Against Cancer?*, 314 NEW ENGLAND J. MED. 1226 (1986).

24 *See* Huber, *Safety and the Second Best: The Hazards of Public Risk Management in the Courts*, 85 COLUM. L. REV. 277, 300-01 (1985).

of all cancers.[25] Another important study, filed with the Occupational Safety and Health Administration (OSHA), conservatively estimated that occupational carcinogens alone accounted for 20% of cancer mortality.[26] In perhaps the most influential article of its type, Doll and Peto attacked the OSHA report and estimated that occupational carcinogens cause only 4% of cancer mortality[27] and environmental carcinogens cause somewhat less than 4%.[28] The mandate to reform tort law would be overpowering if it could save 300,000 lives per year; it is somewhat less so if only 32,000 lives are at stake.[29]

The imprecision of estimating the impact of environmental and occupational carcinogens derives from the central uncertainty surrounding the nature of carcinogenesis. Like the study of toxicology in general, the lack of clear insights into the disease's molecular basis hampers the study of carcinogenicity.[30] Cancer biologists have developed useful hypotheses of carcinogenicity, some of which are now guiding potentially useful research.[31] Nonetheless, the statement by a group of experts convened by the International Agency

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25 P. BARTH & H. HUNT, WORKERS' COMPENSATION AND WORK-RELATED ILLNESSES AND DISEASES 84 (1980).

26 *See* NATIONAL CANCER INST., NATIONAL INST. OF ENVTL. HEALTH SCIENCES & NATIONAL INST. FOR OCCUPATIONAL SAFETY AND HEALTH, ESTIMATES OF THE FRACTION OF CANCER IN THE UNITED STATES RELATED TO OCCUPATIONAL FACTORS 1 (1978); *cf.* SENATE COMM. ON ENV'T AND PUB. WORKS, 96TH CONG., 2D SESS., HEALTH EFFECTS OF TOXIC POLLUTION: A REPORT FROM THE SURGEON GENERAL AND A BRIEF REVIEW OF SELECTED ENVIRONMENTAL CONTAMINATION INCIDENTS WITH A POTENTIAL FOR HEALTH EFFECTS 19 (Comm. Print 1980) (finding that magnitude of health risk created by toxic waste to general population really not known).

27 *See* Doll & Peto, *The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today*, 66 J. NAT'L CANCER INST. 1191, 1244 (1981).

28 *Id.* at 1251.

29 Estimates are that some 400,000 deaths result from cancer each year. *See supra* note 23 and accompanying text. If environmental factors cause 80% of cancers, as Barth and Hunt suggest, then 320,000 deaths each year are due to environmental carcinogenesis. Using the Peto and Doll estimate, environmental and occupational factors account for approximately 8% or 32,000 of the 400,000 cancer deaths.

30 Since the turn of the century, scientists have been unraveling cancer's molecular basis. Important steps have included: discovery of the differences between direct carcinogens that do not require any in vitro metabolic activation; characterization of some carcinogens as initiators, substances that change normal cells into cancerous ones, and other carcinogens as promoters, substances that cannot alone cause cancer, but if preceded by an initiator would enhance the cancerous response; and, more recently, analysis of the genetic machinery of cells, the double-stranded helix of deoxyribonucleic acid (DNA) that makes up chromosomes. *See* Smuckler, *Chemicals, Cancer and Cancer Biology*, 139 W.J. MED. 55, 71-72 (1983).

31 These efforts have given rise to at least two hypotheses concerning carcinogenesis. In the somatic mutation hypothesis, substances disrupt the double helix of DNA, causing aberrant production of proteins which then cause cells to become malignant. The epigenetic hypothesis states that chemical disruption occurs further 'downfield' after ribonucleic acid (RNA) is processed from DNA, but before the RNA provides its signals to protein manufacturing ribosomes. *See* Cooper, *Cellular Transforming Genes*, 218

for Research on Cancer (IARC) in 1980 remains true today: "the mechanisms by which chemicals induce cancer and the developmental stages from initial exposure to frank neoplasia are poorly understood."[32]

Given this central uncertainty regarding the cause of cancer, any statement about the role of any agent as a carcinogen is hedged with assumptions and hypotheses. Because scientists do not yet understand the molecular model of carcinogenesis, it is impossible to state that a given carcinogen caused any individual tumor. With statistical information, however, scientists can estimate the proportion of preventable cancers.[33] Using this type of information, Doll and Peto have estimated that 75-80% of cancers in the United States are avoidable.[34]

The debate then turns to which cancers are preventable and which carcinogens should be controlled. Avoidable causes of cancer include diet, tobacco and alcohol use, environmental and occupational carcinogens and unsafe consumer products. Some scientists have argued that dietary carcinogens, for instance, are more deadly than occupational or environmental carcinogens.[35] Those who oppose tort reform or new regulations use these estimates. They argue that the keys to prevention are tobacco and dietary education, rather than law suits against chemical manufacturers.[36]

This debate, and indeed the larger debate over the threshold of

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SCIENCE 801 (1982). These remain only working hypotheses, but they have produced some new areas for research in molecular carcinogenicity. *See infra* note 174.

32 *See* INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, WORLD HEALTH ORG., LONG-TERM AND SHORT-TERM SCREENING ASSAYS FOR CARCINOGENS: A CRITICAL APPRAISAL 295 (1980) [hereinafter IARC REPORT].

33 Indeed, we have to rely on probabilistic evidence. *See infra* notes 149-70 and accompanying text. Over the past fifty years, it has become clear that there are large differences in the incidence of specific cancers in different geographic locations. Doll & Peto, *supra* note 27, at 1198. Moreover, it is now clear that migrants from one area to another usually reflect the new region's cancer rates. *Id.* at 1200. In addition, changes in incidence over time have been observed, usually correlating with changes in the local environment or living conditions. *Id.* at 1201.

34 Doll & Peto, *supra* note 27, at 1205.

35 *See* Ames, *Dietary Carcinogens and Anticarcinogens*, 221 SCIENCE 1256 (1983). *But cf.* Epstein & Schwartz, *Fallacies of Lifestyle Cancer Theories*, 289 NATURE 127 (1981) (scientific basis for lifestyle theory of cancer causation is scanty).

36 *See* Huber, *supra* note 24, at 278. Huber suggests that our society is overly focused on public risks, and not appropriately cognizant of private risks. *Id.* at 278. This kind of criticism is also made by those who argue that Americans are perversely risk averse in some areas. *See* M. DOUGLAS & A. WILDAVSKY, RISK AND CULTURE: AN ESSAY ON THE SELECTION OF TECHNICAL AND ENVIRONMENTAL DANGERS 151 (1982). These criticisms are not without merit. Nonetheless, it is in some ways proper, given the political nature of our state, to emphasize public risks. A liberal state protects a certain sphere of liberty, a negative freedom for the individual. *See infra* notes 43-45. Hazardous substances, as defined herein, trespass on that liberty. Smoking or dietary choices do not. Thus, in terms of liberalism, emphasizing curbs on public risks makes sense.

mortality necessary to justify restructuring the law, does not change the proposals I set out in this Article for three reasons. First, even Doll and Peto's conservative estimate of the mortality caused by environmental and occupational carcinogens[37] suggests that these carcinogens cause some 30,000 preventable cases of cancer each year. Without a doubt, our legal system should make every reasonable effort to pursue the laudable goal of preventing these deaths. Moreover, legal institutions should ensure that the firms or parties that produce the carcinogens internalize the costs for these preventable deaths, and that the families of the victims of these carcinogens are compensated appropriately. Thus, even the most conservative estimate of the carcinogenic threat posed by hazardous substances presents a challenge that the law cannot ignore.[38]

The second reason for reforming tort law, independent of the exact size of the threat that hazardous substances pose, involves economic predictability and corporate stability. Tort litigation, largely because of the courts' difficulties with scientific evidence,[39] produces inconsistent resolutions of claims deriving from the same hazardous substances. One claim for benzene-induced leukemia can produce an award of $2,800, while another produces a $100,000,000 award for the plaintiff.[40] Such inconsistency disrupts long-term corporate planning and drives insurance companies away from enterprises.[41] As a result, even a conservative estimate of hazardous-substance-induced disease presents sufficient motivation for reforming existing law from the industry's point of view.[42]

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37 *See supra* note 29.

38 Huber himself, despite his emphasis on private risks, refers to the devastation at Bhopal, which was the culmination of a public risk. *See* Huber, *supra* note 24, at 301. *See also* Iyer, *India's Night of Death*, TIME, Dec. 17, 1984, at 22.

39 *See infra* notes 118-70 and accompanying text.

40 *See* Miller v. National Cabinet Co., 8 A.D.2d 281, 188 N.Y.S.2d 29 (1959), *rev'd*, 8 N.Y.2d 277, 168 N.E.2d 811, 204 N.Y.S.2d 129 (1960). *Cf.* Skeen v. Monsanto Chem. Co., Nat'l L.J., Dec. 29, 1986, at 4, col. 3 (S.D. Tex. Dec. 12, 1986). Small variations in jury awards present no problem. The problem arises from huge variations around the mean recovery, which vitiates any predictability in awards.

41 *See, e.g.*, Sparrow, *Hazardous Waste Insurance Coverage: Unexpected Past, Uncertain Future*, 64 MICH. B.J. 169 (1985); Sugarman, *Doing Away With Tort Law*, 73 CALIF. L. REV. 555 (1985). There are now very few firms offering environmental liability insurance. Angelo & Bergeson, *The Expanding Scope of Liability for Environmental Damage and Its Impact on Business Transactions*, 8 CORP. L. REV. 101, 116 (1985).

A subsidiary issue arises in products liability litigation. Manufacturers argue that their main problem is not inconsistency of verdicts, but unforeseen changes in products liability law. *See generally* Epstein, *Manville: The Bankruptcy of Product Liability Law*, REGULATION, Sept.-Oct. 1982, at 14; Wade, *On the Effect in Product Liability of Knowledge Unavailable Prior to Marketing*, 58 N.Y.U. L. REV. 734 (1983).

42 Asbestos may produce 8,000 deaths per year. 1 V. DEVITA, S. HELLMAN & S. ROSENBERG, *supra* note 23, at 109. This constitutes about 1-2% of the 400,000 cancer deaths that occur annually. Nonetheless, these deaths have caused serious dislocation in the insulation industry, not to mention the victims' extended families. *See* Winter, *Asbes-*

The third reason why the proportion of preventable deaths and illnesses due to hazardous substances should not determine whether we undertake reform of common law tort doctrines is that we live in a liberal state. The liberal state guarantees a certain amount of liberty, a set of prerogatives that the individual controls.[43] This "negative freedom," as Berlin has termed it, should be protected from encumberment.[44] The individual, on the one hand, has negative freedom to smoke, to eat certain foods, and to expose him- or herself knowingly to certain risks. On the other hand, negative freedom guarantees that the individual should not unknowingly expose him- or herself to the risks of hazardous substances at home or at work. As such, it makes little sense to argue that private risks such as smoking, however deplorable from a public health point of view, represent reasons for not controlling public risks such as environmental carcinogens.[45] Liberalism requires that we seek measures to reduce exposure to hazardous substances when we have not assented to those exposures, no matter what the size of the threat from the exposures to which we have assented.[46]

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tos Legal 'Tidal Wave' Is Closing In, 68 A.B.A. J. 397 (1982). Thus, even a relatively small threat can produce pressure for alternatives to litigation. *See* Wellington, *supra* note 7.

43 *See generally* J. MILL, ON LIBERTY (Watts & Co. ed. 1929) (1859). The liberal state celebrates the rights of individuals. As Ladd has noted, "As a matter of fact, the persuasive influence of lawyers in American public life—as noted by perceptive observers since de Tocqueville—reflects the principles of individualism, free enterprise and constitutional government characteristic of our polity." *See* Ladd, *Legalism and Medical Ethics*, in CONTEMPORARY ISSUES IN BIOMEDICAL ETHICS 1, 18 (J. Davis, B. Hoffmaster & S. Shorten eds. 1978).

44 I. BERLIN, FOUR ESSAYS ON LIBERTY (1970). As Berlin so succinctly expressed it:

> The first of these political senses of freedom . . . which I shall call the 'negative' sense, is involved in the answer to the question 'What is the area within which the subject—a person or group of persons—is or should be left to do or be what he is able to do or be, without interference by other persons?' The second, which I shall call the positive sense, is involved in the answer to the question 'What, or who, is the source of control or interference that can determine someone to do, or be, this rather than that?'

Id. at 21-22.

45 Public health policy must attempt to overcome those causes of disease that affect many similarly situated individuals. Nonetheless, we must recognize that a liberal state values individual prerogatives and that as a result, the utilitarian calculus is not straightforward. Mill believed that "[a]ll errors which [the individual] is likely to commit against advice and warning are far outweighed by the evil of allowing others to constrain him to what they deem his good." *See* MILL, *supra* note 43, at 94. Thus, under our current polity, ensuring that a nuclear power plant does not trespass on others' lifestyle is simply more politically acceptable than banning smoking.

46 Recognition that we live in a liberal state does not mean that individual tort litigation is the only way to protect negative freedom. Regulatory efforts can also guarantee liberty. *See infra* notes 272-92 and accompanying text.

II

CAUSATION AND UNCERTAINTY IN LAW AND SCIENCE

A. The Evolution of Causation in the Philosophy of Science

The courts' difficultly with handling evidence linking a hazardous substance to a disease is largely the result of the courts' inability to understand scientific notions of causation. The assumptions that courts make about causation very much resemble those that provide the foundation for Newtonian physics. Over the past century, science has come to rely on new assumptions about cause and effect. These new assumptions have not been integrated into legal reasoning. As a result, lawyers and judges are often confused when they address scientific causation issues.

A review of the evolution of causation within the philosophy of science helps expose the nature of this confusion. Newton introduced quantitative mechanics to physics, revealing the consistent operation of the laws of motion and acceleration in a variety of phenomena.[47] Locke best explicated the epistemological framework that accompanied these insights. He postulated the existence of fundamental particles in which inhered the "primary qualities," qualities that were the objects of perception.[48] Among these primary qualities was the power of one object to produce a change in another. Newtonian physics limited this power to a change in direction of particles following a collision. Thus, this theory limited causation to mechanical contacts between particulate objects.[49] Because incidents could be expressed mathematically, it followed that causation, too, simulated the rational structure of mathematics. This characterization of causation as collisions that follow the physical laws defined by mathematics and Newtonian physics is known as "corpuscularianism."[50]

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47 A discussion of Newton's contributions to physics is the proper starting point for tracing the evolution of the notion of causation in science. Newton's work helped to explain a variety of natural phenomena: falling objects, tidal actions, and cannon trajectory amongst others. *See generally* R. HARRÉ, THE PHILOSOPHIES OF SCIENCE (1972) (examining many scientists' theories); M. BUNGE, CAUSALITY: THE PLACE OF THE CAUSAL PRINCIPLE IN MODERN SCIENCE 59 (1959) (discussing Newton's law of motion—force equals mass times acceleration). These mechanical concepts of causation may be those that courts and the nonscientific public use when considering causal issues. The court's understanding, however, is salient in that the court must address scientific issues as presented by experts—the judge and jury cannot simply refuse to accept scientifically valid arguments. This tension between everyday views of causation and scientific causation is the focus of this paper.

48 *See* J. LOCKE, AN ESSAY CONCERNING HUMAN UNDERSTANDING, 555-56 (P. Nidditch ed. 1975) (4th ed. 1700); *see also* J. BENNETT, LOCKE, BERKELEY, HUME: CENTRAL THEMES (1971).

49 *See* R. HARRÉ, *supra* note 47, at 131.

50 *See* D. BOHM, CAUSALITY AND CHANCE IN MODERN PHYSICS 36 (1959). Corpuscularians believed that mechanical laws had general validity and that the entire universe

In the late nineteenth century, Mach outlined a corpuscularian theory of science that incorporated elements of positivism.[51] Positivism is the belief that scientific knowledge unceasingly expands.[52] Mach's disciple, C.G. Hempel, produced the most thorough description of this synthesis.[53] Hempel's discussion is interesting because the positivism-corpuscularian philosophy of science outlines many of the assumptions that underlie Newtonian physics. Courts, if not scientists, use these assumptions to understand scientific evidence.

Hempel argued that a scientific theory was merely the relationship between a covering law, defined as one of a general set of de-

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consisted of nothing but particles obeying the tenets of Newtonian physics. It followed that any previously unexplained phenomena could be reduced to an essence of particles and physical laws. Causal descriptions would follow from mathematical descriptions of mechanical events.

The reduction of causation to its essential nature initiated by Locke was completed by David Hume's radical empiricism. Hume argued that because there is no possible perception of causal primary qualities, causation must be thought of as a matter of habit on the part of the observer. *See* D. HUME, ENQUIRIES 80-96 (P. Nidditch 2d ed. 1902) (1778). The linkage of two events in a causal framework did not represent a necessary connection for Hume, because the linkage itself was imperceptible. Hume's attack on causal powers did little to loosen the dominance of Newtonian physics and corpuscularianism in science, but it did present the critical issue for epistemology and provoked Kant's Transcendental Deduction.

In the *Critique of Pure Reason,* Kant sought to find answers for Hume's empirical nihilism. *See* I. KANT, CRITIQUE OF PURE REASON 55, 606-07 (N. Kemp-Smith trans. 1929) (2d ed. 1787). Kant argued that, before any perception was possible, man had to posit his own existence. This initial step demonstrated that a person's experience was not only perception of sensory data, but also a set of posited ordering concepts. Causation was one of these ordering concepts. According to Kant, causation was a theoretical construct that allows comprehension of phenomena. Such theoretical constructs were not perceived, but rather were conceptual and belonged to the category of noumena. *See* T. WILKERSON, KANT'S CRITIQUE OF PURE REASON: A COMMENTARY FOR STUDENTS 180-200 (1976).

51 Mach incorporated positivisitic notions into a corpuscularian theory of science in the late 19th century. *See* E. MACH, POPULAR SCIENTIFIC LECTURES (T. McCormack trans. 1895). Mach argued that noumenal aspects of knowledge could not be profitably discussed; that the only reliable mode of knowledge was the scientific mode, based in mathematics; and that science unceasingly explained the unknown. Thus Kant's insights into the role that theoretical constructs play in ordering perceptions was lost. Moreover, Mach celebrated Hume's emphasis on the empirical, while ignoring the nihilism of his skepticism. Mach's positivism dominated the philosophy of science until the middle of the twentieth century, largely because of its common sense appeal and faith in scientific progress. *See* M. BUNGE, *supra* note 47, at 68-69.

52 Positivism is the theory that empirical natural sciences perfect knowledge. *See* Greene, *Biology and Social Theory in the Nineteenth Century: Auguste Comte and Herbert Spencer,* in CRITICAL PROBLEMS IN THE HISTORY OF SCIENCE 419-46 (M. Clagett ed. 1959). In the late 18th and early 19th centuries, scientific discovery and discussion continued at a remarkable pace. Drawing on Comte's work, philosophers and scientists alike argued that science would come to explain all phenomena and resolve all social and scientific questions. *Id.* Positivism was the term used to describe the belief in the unceasing advance of science, and the belief that science would overcome all questions.

53 C. HEMPEL, *Aspects of Scientific Explanation,* in ASPECTS OF SCIENTIFIC EXPLANATION AND OTHER ESSAYS IN THE PHILOSOPHY OF SCIENCE 331-410 (1965).

ductive principles, and an explanadum, the phenomenon that the covering law was to explain.[54] The relationship was formally logical, but Hempel expressed it in causal terms.[55] Thus, deductive causal analysis, as defined by covering laws, connected and explained phenomena. Deductive reasoning produced logical relationships between events, essentially enrolling phenomena into causal chains.

The covering law-explanadum model leads to the following corollaries. First, science progresses as deductively derived causal chains develop from applying covering laws to more phenomena. Uncertainty results from a phenomena without applicable covering law. Second, scientific knowledge constantly expands as causal chains connect previously unexplained phenomena.[56] Third, deductive reasoning takes precedence over inductive reasoning. The primary method of causal explanation, and thus of knowledge, was thought to be deductive reasoning.[57]

Hempel was criticized for failing to explain the increasing role of statistical evidence in science.[58] Ultimately, Hempel acknowledged that inductive-statistical evidence could be used in scientific explanation, but he doubted that such evidence could provide the basis for causal reasoning and thus afforded such evidence a subsidiary status.[59] This position proved untenable. Indeed, develop-

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54 Hempel has argued that scientific theories are deductive-nomological explanations. A deductive-nomological explanation effects a deductive subsumption of the explanation under principles that have characteristics of a general law. *Id.* at 337.

55 Thus scientific knowledge is logical and mathematical, but cloaked or expressed in causal language. Developments in geometry in the late 19th century reinforced this view of scientific information. *See generally* M. HESSE, THE STRUCTURE OF SCIENTIFIC INFERENCE (1974); D. SHAPERE, PHILOSOPHICAL PROBLEMS OF NATURAL SCIENCE (1965).

56 It is the sense of ever expanding and improving scientific knowledge that is positivism's influence in corpuscularianism. *See* Brennan & Carter, *Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease in Individuals,* 10 J. HEALTH POL., POL'Y & LAW 33, 37 (1985).

57 *See* C. HEMPEL, *supra* note 53, at 369.

58 Hempel's rendition of positivistic corpuscularianism was criticized because it failed to account for some important explanations. Foremost among these explanations were statistical analyses that are adequate for prediction, but not underpinned by a corpuscularian logical structure. I. SCHEFFLER, THE ANATOMY OF INQUIRY 42 (1963). Such statistical analyses and inductive reasoning could not be explained by a theory of science based on deductive, ever-expanding corpuscularian covering laws and a notion that uncertainty was simply a phenomenon awaiting deductive enrollment under a covering law. *See* Scriven, *Explanation and Prediction in Evolutionary Theory,* 130 SCIENCE 477 (1959).

59 As a result, Hempel acknowledged that in addition to deductive nomological reasoning, there existed inductive-statistical explanations. Hempel accorded subsidiary status to inductive-statistical explanations because he thought they were relative to certain knowledge situations and thus not universal like deductive nomological statements. Moreover, Hempel doubted that inductive-statistical statements could be accepted as a basis for causal propositions. Thus Hempel's mature positivism featured deductive reasoning from covering law to phenomena, with causal propositions as the commonsense stand-in for these deductive statements. In addition, as covering laws grew, uncertainty

ments in physics called into question the entire structure of positivism-corpuscularianism. At the end of the nineteenth century, the mechanistic physics and mathematics of Newton, upon which corpuscularian notions were so dependent, were undermined by field theories supported by integro-differential equations and matrix analysis.[60] This process continued in the twentieth century with the development of quantum mechanics and the theory of relativity.[61] Philosophers of science simply could not ignore the expanding role of inductive reasoning and statistical evidence in physics.[62] These developments in physics paralleled others in medicine, with epidemiologists William Farr and John Snow, among others, pioneering the use of probabilistic evidence.[63]

In the past twenty years philosophers have come to appreciate the importance of theory and hypothesis building in the enterprise of science. The philosophy of science now recognizes that scientists consciously use hypothesis testing to confront uncertainty, often using probabilistic evidence and inductive reasoning. Moreover, scientific progress is not understood as the relentless accumulation of phenomena under covering laws, but as a succession of theories or "problem shifts."[64] Scientists use theories to formulate hypotheses.

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was overcome. Statistical analysis fit only tenuously into this framework. *See* C. HEMPEL, *supra* note 53, at 177. *See also* R. VON MISES, POSITIVISM: A STUDY IN HUMAN UNDERSTANDING 188 (1951).

60 *See* M. BUNGE, *supra* note 47, at 75.

61 *See* D. BOHM, *supra* note 50, at 131. Modern physics thus lost much of the commonsense appeal that had made the corpuscularian analysis of causation so appealing. Uncertainty became a concept more meaningful than something simply outside the covering laws, especially as probability theory was incorporated into physics. Thus, as Suppes noted, "[B]y the late 1960s a general consensus had been reached among philosophers of science that the Received View [positivism] was inadequate as an analysis of scientific theories." F. SUPPES, THE STRUCTURE OF SCIENTIFIC THEORIES 4 (2d ed. 1977).

62 Much of the criticism of Hempel and corpuscularianism/positivism was directed at the role of the all important correspondence rules that related theory to fact. *See* Schaffner, *Correspondence Rules*, 36 PHIL. SCI. 280 (1969); *see also* Suppes, *What is a Scientific Theory*, in PHILOSOPHY OF SCIENCE TODAY 55 (S. Morgenbesser ed. 1967) (examining variety of concepts that define "scientific theory").

63 *See generally* M. SUSSER, CAUSAL THINKING IN THE HEALTH SCIENCES: CONCEPTS AND STRATEGIES OF EPIDEMIOLOGY 22-23 (1973).

64 In order to better understand science and the causal principles science produces, philosophers have returned to the epistemological issues Kant raised in answer to Hume. In addition, philosophers have paid closer attention to the manner in which scientists actually address uncertainty. The result is the so-called hermeneutic analysis of science. Hermeneutics is defined as the study of methodological principles of interpretation. WEBSTER'S THIRD NEW INTERNATIONAL DICTIONARY 1059 (1976). A hermeneutic approach allows a philosopher to understand the interplay of theory and phenomenal evidence, and leads to a renewed emphasis on the manner in which hypotheses guide scientists. In effect, Kant's noumenal aspects of reality are openly recognized. *See* T. WILKERSON, *supra* note 50, at 185. Lakatos, for instance, argues that scientific progress is a result of a succession of theories or a 'problem shift,' rather than a

They test these hypotheses by designing experiments that will provide anticipated results defined by the hypotheses. The whole process relies heavily on inductive reasoning. More important, scientists often based their reasoning on probabilistic evidence.[65] Science is not simply an extension of causal chain analysis or covering law deductions. As Hanson notes, "Causal chain analysis is generally unsatisfactory [in] that causal chain accounts are loaded with assumptions and theoretical presuppositions: these are such that without them the cause singled out would not be sufficient to produce the effect. . . . Causes are connected with events; but this is because our theories connect them, not because the world is held together by cosmic glue."[66] This also means that scientific progress is not simply the expansion of covering laws; rather science develops as certain hypotheses provide better explanation when tested and refinement improves theories.[67]

This is not to say that causal language is not scientific language.[68] Scientists use causal concepts to set up hypotheses. The method by which they test these hypotheses often involves using inductive reasoning and probabilistic evidence. Thus a scientific explanation is framed in terms of causality, but the evidence to support that explanation need not involve a neat, deductively derived causal chain. Indeed more often than not, the evidence is summarized by a probability statement in the form of a 'p' value.[69]

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simple matter of deductive enrollment of phenomena under covering laws. Lakatos, *Falsification and the Methodology of Scientific Research Programmes* in CRITICISM AND THE GROWTH OF KNOWLEDGE 118 (I. Lakatos & A. Musgrave eds. 1970); *see also* P. FEYERABEND, AGAINST METHOD (1971).

65 *See infra* notes 171-214 and accompanying text.

66 N. HANSON, PATTERNS OF DISCOVERY 64 (1958).

67 As Kuhn has emphasized, a phenomena which was once well explained by a covering law, could be understood in a completely different way following a scientific revolution. Thus it is theories that provide knowledge, not an uncovering of the essential deductive and logical nature of the universe, as the corpuscularian model held. Hermeneutic analysis supplies another critical insight of science concerning the growth of scientific knowledge. Science is no longer portrayed as an ever expanding set of information, linked by covering laws' deductive reasoning. Instead, science is conceived of as sets of competing theories that are analyzed in terms of their ability to explain observations. Scientific revolutions occur when a new theory supersedes others in terms of its explanatory power. *See* T. KUHN, THE STRUCTURE OF SCIENTIFIC REVOLUTIONS (1961). Hermeneutic analysis of scientific reasoning is not positivistic in its posture. There is no faith in science's ultimate ability to explain and solve all problems simply by expanding deductive reasoning and causal chain analysis. Rather, new theories are formulated and hypotheses tested—explanations are cast in terms of the best explanation, not as ultimate explanations.

68 To the contrary, discussions of highly probabilistic fields such as quantum mechanics are still framed in terms of causation. P. SUPPES, A PROBABILISTIC THEORY OF CAUSALITY 5 (1970).

69 *See infra* notes 194-97. A hermeneutic analysis of science accepts that causal language describes scientific understanding, but recoguizes that language is as often based

In summary, scientists understand that theories define uncertainty and provide the basis for hypothesis building. The process of hypothesis building and testing depends largely on inductive reasoning. Scientists often use probabilistic evidence to test hypotheses. As a result, statistical evidence deserves the same status as any other type of evidence. Moreover, one cannot expect science to provide deductive causal chains as the basis for all knowledge. Scientists recognize that the causal concepts they use often express probabilistic reasoning as deductive reasoning.[70]

B. Notions of Causation in the Law

1. *Causation and the Jurisprudence of Torts*

An empiricist posture toward causation has dominated Anglo-American jurisprudence of torts.[71] Although negligence does not result in liability unless it causes injury or damage, legal scholars have recognized that identification of a cause is seldom straightforward. As Fleming James and Roger Perry noted:

> Obviously the legal test includes a requirement that the wrongful conduct must be a *cause in fact* of the harm; but if this stood alone the scope of liability would be vast indeed, for "the causes of [causes] are infinite"—"the fatal trespass done by Eve was cause

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on probabilistic statements as it is on deductive ones. Hermeneutic analyses, unlike positivistic corpuscularian models of science, are not crippled by methodological opposition to inductive or probabilistic reasoning. Because the role of theory in defining uncertainty is fundamental to hermeneutic analysis, the inductive framing of a hypothesis and its rejection or acceptance following statistical analysis is readily accepted and indeed offers a better paradigm of scientific reasoning than does causal chain analysis. *See* Bohm, *A Suggested Interpretation of the Quantum Theory in Terms of "Hidden" Variables*, 85 PHYSICAL REV. 166, 166 (1952) ("Quantum-mechanical probabilities are [under the proposed quantum theory] only a practical necessity and not . . . a manifestation of an inherent lack of complete determination in the properties of matter at the quantum level.").

70 Causal language remains the vehicle for explaining physical phenomena while hermeneutic analysis recognizes the role of theories and the importance of context. In addition, probabilistic evidence is not subsidiary; indeed, testing and rejection of hypotheses based on statistical analysis is integral to science and offers a more realistic paradigm of scientific thought than does pure deduction. *See* M. SUSSER, *supra* note 63, at 69. The positivistic faith that science will soon explain the unknown has no place in a philosophy of science that focuses on how scientists actually work. Although causal language remains dominant in scientific explanation, probabilistic reasoning provides the basis for many of the theories that direct scientific inquiry and that lead to causal attribution. Theories and the explanations they offer are evaluated in terms of their cogency and efficiency, but are not expected to eliminate all uncertainty. It follows that science is not an unceasing march to total knowledge, but represents a competition of theories, with both deductive and probabilistic reasoning used to judge the competitors. The metaphor of science as a causal chain that is unceasingly elongated does not work. *See* Brennan & Carter, *supra* note 56, at 46-47.

71 Hume's skepticism regarding perception of a causal power is the epitome of empiricism. H. HART & J. HONORE, CAUSATION IN THE LAW 10-28 (1985).

of all our woe."[72]

The concept of proximate cause evolved to deal with the multiplicity of causes of events, as well as the contextual aspects of the event itself. Legal policies allow a jurist to discriminate between many so-called causes-in-fact and to identify a proximate cause for the purpose of determining liability. Borgo has called this the traditional legal doctrine of causation.[73] A long line of legal realists have supported it, starting with Professor Green.[74] As Borgo notes, the theory of proximate cause is inconsistent with the use of causality in ordinary language, where it is presumed that each event has an identifiable, individual antecedent cause.[75]

Jurists' confrontation with difficult cases can explain the law's willinguess to abandon the ordinary meaning of causation in favor of proximate cause. Innocuous events producing unexpected results can be used in a casual chain analysis in such a way that liability falls in an unjust manner. Indeed, the tragic-comedy of a causal chain of events recounted in a case like *Palsgraf v. Long Island Railroad*[76] provides the highlight of first year tort classes. The lack of an easily identifiable "cause in fact" in such cases forces a court to incorporate policy issues into causal attribution; hence, the evolution of proximate cause.[77]

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72 James & Perry, *supra* note 10, at 761.

73 *See* Borgo, *Causal Paradigms in Tort Law*, 8 J. LEGAL STUD. 419, 421-22 (1979). Borgo identifies three distinct doctrines of causation in tort law: (1) the traditional theory of legal realists that identifies many causes in fact, but allows policy to indicate a proximate cause; (2) the Coasean model that requires interaction of two players; (3) Calabresi's functional analysis of causation. *Id.* at 421-25.

74 *See* L. GREEN, RATIONALE OF PROXIMATE CAUSE (1927); *see also* Morris, *On the Teaching of Legal Cause*, 39 COLUM. L. REV. 1087 (1939).

75 Borgo, *supra* note 73, at 421. Borgo argues that the "legal orthodoxy" holds that harms are not necessarily the result of individual antecedent events. *Id.* Of course, this implies that judges think about causation in a different way than ordinary individuals do. I believe this is true insofar as decisions involving proximate cause reflect policymaking. However, I would argue that judges revert to an emphasis on individual cases and antecedent events when presented with probabilistic evidence. *See infra* notes 149-70 and accompanying text.

76 248 N.Y. 339, 168 N.E. 99 (1928).

77 An epistemological analysis of causation does not clarify whether the legal realists' advocacy of proximate cause derived from the concepts of causation that superseded Humean skepticism. *See supra* note 46. The theories presented by Hume and Kant are not part of the legal realists criticism of cause in fact. Rather, the trouble with cause in fact seems to be in identifying where the causal chain should end, not that corpuscularian causal chain reasoning is flawed. The impetus for notions of proximate cause was not that science had revealed that causal chain analysis was not sophisticated enough, but that the causal chains themselves were too long and did not reliably indicate liability. Thus, the legal realists rejected cause in fact in favor of proximate cause, but this did not represent a hermeneutic understanding of science and causation. Deductively devised chains still dominate comtemporary analyses of causation, and legal orthodoxy depends on causal chain analysis.

The past ten to fifteen years have witnessed a great deal of ferment in tort law, especially regarding causation. Legal scholars have begun to explore the validity of causes founded on probabilistic evidence rather than deductive reasoning. Hart and Honoré have stated:

> It is easy here to be misled by the natural metaphor of a causal 'chain', which may lead us to think that the causal process consists of a series of single events each of which is dependent upon (would not have occurred without) its predecessor in the 'chain' and so is dependent upon the initiating action or event. In truth in any causal process we have at each phase not single events but complex sets of conditions, and among these conditions are some which are not only subsequent to, but independent of the initiating action or event.[78]

Unlike James and Perry's complaint about the length of causal chains, Hart and Honoré's statement evinces a recoguition that causation is not simply a matter of identifying causal links.

Calabresi has argued that in addition to cause in fact and proximate cause, the law uses a third notion of causation. Calabresi calls this "causal linkage" and characterizes it as follows: "[A] causal link [exists] between an act or activity and an injury when we conclude on the basis of the available evidence that the recurrence of that act or activity will increase the chances that the injury will also occur."[79] He contrasts this with the more familiar 'but for' and 'sine qua non' cause, the ordinary-language use of cause in which a causal chain points to a particular event as the cause.[80] Calabresi does not elaborate on the differences between these concepts in the abstract, but does note that they function differently when analyzed in terms of the functions of the tort system.[81]

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78 H. HART & T. HONORÉ, *supra* note 71, at 72.

79 Calabresi, *Concerning Cause and the Law of Torts: An Essay for Harry Kalven, Jr.*, 43 U. CHI. L. REV. 69, 71 (1975).

80 *Id.* at 72.

81 *Id.* Some other contributions to causal concepts in the law are worth mentioning. Shavell has built on Calabresi's distinctions between concepts of cause. *See* Shavell, *An Analysis of Causation and the Scope of Liability in the Law of Torts*, 9 J. LEGAL STUD. 463 (1980). He calls the "but for" cause a "cause in fact," and characterizes it in terms of the consequences being different if the cause in fact had not occurred. *Id.* at 467 n.14. Cause in fact corresponds to the use of causation in everyday language. Shavell defines causal linkage in much the same way as Calabresi did, and calls it probabilistic cause. However, Shavell explains that probabilistic cause follows the adequate cause theory of Von Kries and other European legal scholars. *Id.* at 468-69.

The adequate cause theory was developed by the German physiologist Von Kries in the late 19th century. Von Kries was interested in probability theory and sociology. He defined a harm's "adequate cause" as a contingency which satisfies two conditions: (1) it is a sine qua non cause of the harm; and (2) it significantly increased the objective probability of the harm. The latter condition was the focus of much attention by German legal scholars. H. HART & T. HONORÉ, *supra* note 71, at 467-78 (citing J. VON KRIES,

Before turning to that functional analysis, it is interesting to note that the distinctions between but for cause and probabilistic causation coincide neatly with those between Newtonian physics and modern science noted earlier.[82] But for causation theory assumes the existence of causal chain analysis, depends on a mechanistic understanding of causation, and coincides with everyday, common sense notions of causation. The corpuscularian thinker can deductively derive a cause in fact. The only problem with but for causation concerns the reach of the deductive reasoning: selecting from the causal chain *the* but for cause upon which liability falls can be difficult. In contrast, probabilistic causation relies on probabilistic reasoning, and is independent of a mechanistic understanding of causation. Furthermore, probabilistic reasoning does not coincide with conventional notions of causation.[83] Thus, but for cause or cause in fact is the concept of causation that is employed in corpuscularian science, while probabilistic cause is integral to scientific research.

Having uncovered similar tensions between but for and probabilistic cause in law and corpuscularian and more modern analysis in science, I return to Calabresi's critical discussion of the function of causal concepts in tort law. Calabresi recognizes two major goals of tort law: compensation and deterrence.[84] Each of these further divides into components.

Compensation goals include spreading the burden of injury costs and imposing costs on those with "deep pockets."[85] These goals ensure that the burden does not fall on those who cannot bear it. Calabresi argues that proximate causation better achieves compensation goals from a functional point of view than does but for or causal linkage.[86]

ÜBER DIE BEGRIFFE DER WAHRSCHEINLICHKEIT UND MÖGLICHKEIT UND IHRE BEDEUNTUNG IM STRAFRECHTE (1889). In particular, Traeger championed adequate cause theory, emphasizing the role of probability. His work, H. TRAEGER, DER KAUSALBEGRIFF IM STRAF- UND ZIVILRECHT (1904), is the most frequently cited work on causation in German civil courts. H. HART & T. HONORÉ *supra* note 71, at 471 n.29. Hart and Honoré note that the development of statistical laws in science supported an adequate cause theory, especially in quantum theory. Indeed, they allow that "some have maintained that it has now ceased to be even a theoretical ideal of science to discover causal laws in the sense of statements of conditions which are invariably followed by a given event without exception." *Id.* at 472.

82 *See supra* notes 47-70 and accompanying text.

83 They are, however, dependent on context and circumstances, as Traeger emphasized. H. HART & J. HONORÉ, *supra* note 71, at 476. Hermeneutic analysis rejected deductive, isolated causal chains in favor of context laden observation and inductive reasoning. *See supra* notes 64-68.

84 Calabresi, *supra* note 79, at 73-91.

85 *Id.* at 73-77.

86 Calabresi argues that courts can use proximate cause to select that party best able to spread the losses resulting from an injury. *Id.* at 74. Moreover, once a court has

Deterrence is a different story. Calabresi distinguishes market deterrence from collective deterrence.[87] Both types of deterrence attempt to balance injury and safety costs by avoiding those injuries harmful enough to justify avoidance. Collective deterrence accomplishes this goal through political, collective compromises of safety and injury costs.[88] Calabresi posits that causal linkage notions best accomplish collective deterrence. By contrast, market deterrence leaves those compromises to individual, atomistic market decisions.[89] Market deterrence utilizes but for causation, as it readily allows calculation of costs the cheapest cost avoider would incur.[90] Calabresi does not argue that market deterrence requires but for cause; rather, he notes only that market deterrence goals explain the primacy of but for causal notions in law.[91] Tort law attempts to deter costly injuries, and in a market economy, but for causal analysis most readily accomplishes this goal.

Calabresi's analysis seems paradoxical at this point. He allows that market deterrence "alone . . . can explain the virtual universality of the *but for* test."[92] Moreover, he notes that the but for requirement is often described as an "essential, almost categorical imperative."[93] Yet, Calabresi's functional analysis of torts relegates but for causation to a rather minor status. Thus it seems that Cala-

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established but for causation, it can also select proximate cause with regard to the deepest pocket. *Id.* at 76. Thus, proximate cause serves the functional goals of an efficient tort system.

87 *Id.* at 91-93, 101.

88 *Id.* at 78-84.

89 *Id.* at 84. Calabresi notes that the pure form of collective deterrence is a societal judgment that certain acts are intolerable. *Id.* at 78. Calabresi argues that tbe *Wagon Mound* case, Overseas Tankship (U.K.) Ltd. v. Morts Dock & Eng'g Co., [1961] App. Cas. 388 (N.S.W.), represents an example of collective deterrence in that the case focuses on the proscribed activities from a societal point of view, rather than the the individuals actors' activities. Calabresi, *supra* note 79, at 80. Market deterrence emphasizes the individual's actions, because it depends on case-by-case determinations of action and intent. Thus Calabresi distinguishes two conceptions of tort law: one centers on collective determination of proscribed activity; the other focuses on the actions of the individual. *Id.* at 84.

90 Calabresi thus creates an attractive symmetry. Causal linkage notions require analysis at the level of the group's desires and actions. Collective deterrence shares this analytic framework while market deterrence proceeds at the individual level. "By using the *but for* requirement, we tell the chosen loss bearer that its burden will equal those costs that, *but for* its bebavior, would not have been incurred . . . ." Calabresi, *supra* note 79, at 85-86.

91 *Id.* at 87.

92 *Id.* at 85.

93 *Id.* Calabresi's choice of words highlights the difference between his synthesis of causal notions and that of Richard Epstein. For Epstein, causation has moral force—moral force that is self-evident. *E.g.*, Epstein, *A Theory of Strict Liability*, 2 J. LEGAL STUD. 151 (1973) [hereinafter Epstein, *Strict Liability*]; Epstein, *Intentional Harms*, 4 J. LEGAL STUD. 391 (1975); Epstein, *Causation and Corrective Justice: A Reply to Two Critics*, 8 J. LEGAL STUD. 477 (1979). Kant based his moral theory on just this sort of categorical impera-

bresi acknowledges a dominant role for but for causation in legal reasoning, but accords it only a very subsidiary status in his functional analysis of tort law.

Perhaps one can explain this paradox by noting that Calabresi did not address the role of but for causation as a vehicle for considerations of corrective justice. In contrast with economic analyses of tort law, such as that suggested by Calabresi, some legal scholars have posited that tort law has a normative role, and should reflect the notions of individual morality contained within ordinary language. Foremost among these scholars is Richard Epstein, and a brief review of his work demonstrates what Calabresi's economic analyses of tort law lacks.

Epstein's central thesis is that ordinary language reflects certain moral ideals,[94] one of which is that people are responsible for the harm they cause.[95] It follows from this that liability in tort law should be based on causation, not on policies that determine the proximate cause. Epstein's approach leads naturally to certain corollaries, including a preference for strict liability and a refusal to accept positive duties.[96]

Several important points emerge from the mere existence of the debate between Calabresi and Epstein. First, Epstein's corrective justice model of causation is firmly grounded in the notions of political morality that guide the liberal state.[97] The state grants individuals the widest possible sphere of negative freedom,[98] and holds them responsible when their actions encroach on the negative freedom of others. Tort law based on corrective justice does not serve these goals, and within that framework, causation focuses on the individual.[99]

Second, Calabresi's functional approach accommodates proba-

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tive. *See* I. KANT, GROUNDWORK OF THE METAPHYSIC OF MORALS 61 (H. Paton trans. 1947) (2d ed. 1798).

94 Epstein, *Defenses and Subsequent Pleas in a System of Strict Liability*, 3 J. LEGAL STUD. 165, 166 (1974).

95 Posner summarizes Epstein's major tenets in a succinct, yet accurate, manner. Posner, *Epstein's Tort Theory: A Critique*, 8 J. LEGAL STUD. 457, 458-59 (1979).

96 A rigorous critique of the various tort law formulations proposed by Calabresi and Epstein is beyond the scope of this Article.

97 *See supra* notes 43-45 and accompanying text.

98 *Id. See* McPherson, *Maximization of Democracy*, in PHILOSOPHY, POLITICS AND SOCIETY 83 (P. Laslett & W. Runcimann eds. 1967). Nonetheless, the notion of liberty as negative freedom guarded by rights has provided the basis for much of American political theory in the last 15 years. *See* R. NOZICK, ANARCHY, STATE, AND UTOPIA (1974).

99 Epstein uses the rubric of the original position, or the pristine, fundamental society, as an analytic tool. *See* Epstein, *supra* note 94, at 198. The same sort of analysis is the cornerstone of Rawls's thoroughgoing description of the liberal state. J. RAWLS, A THEORY OF JUSTICE (1971). *See also* Hart, *Rawls on Liberty and Its Priority*, in READING RAWLS 230 (N. Daniels ed. 1975).

bilistic notions of causation, but does not incorporate corrective justice.[100] Probabilistic causation depends on concepts of likelihood and group activities or behavior,[101] whereas corrective justice concepts of causation rely on analysis of individual behavior. Assigning liability within Epstein's model requires a clearly drawn causal chain linking an individual with a particular harm.[102] Any system of tort law that relies predominately on corrective justice as a basis for causal assignment will probably fail to accommodate probabilistic causal concepts.[103]

In summary, legal notions of causation reflect a complex inter-

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100 Calabresi does not claim that his discussion of tort law is exhaustive. He does not, however, explicitly entertain concepts of morality within tort law. *See* Calabresi, *supra* note 79.

Rosenberg argues that corrective justice can be accommodated by probabilistic forms of causation. His perception of corrective justice is that "wrongdoers should make their victims whole." *See* Rosenberg, *supra* note 3, at 877. He would argue, therefore, that corrective justice is served by having manufacturers of hazardous substances compensate groups of victims according to the proportion of disease caused. Corrective justice, as Epstein uses the term, and as it is used in the liberal tradition, involves a "causal chain" type of causation, not probabilistic causation.

The link between injurer and injured is critical, and is not a probable link. This insight is the key to understanding the problem courts have with hazardous-substance litigation. Moral causation, which produces corrective justice, assumes mechanistic notions of causation. Rosenberg's belief that corrective justice can rely on probabilistic causation can be supported only when there is little uncertainty in the probabilistic attribution, as in the asbestos litigation. Most hazardous-substance cases involve much more uncertainty, and present a greater challenge to corrective justice theory. *See infra* notes 216-55 and accompanying text.

101 *See* Calabresi, *supra* note 79, at 71.

102 Indeed Borgo criticizes Epstein for relying on the covering law model as proposed by Hempel. *See* Borgo, *supra* note 73, at 434-45.

103 This may be especially true in criminal law. Devlin has compared criminal law to a citadel at the heart of the legal system, surrounded by the battlements of civil law. P. DEVLIN, THE ENFORCEMENT OF MORALS 1-25 (1965). Thus, criminal law may have a larger element of corrective justice and moral impact. Causation that relies on overtly probabilistic reasoning is especially suspect in the criminal setting. *See* Tribe, *Trial by Mathematics: Precision and Ritual in the Legal Process*, 84 HARV. L. REV. 1329 (1971) (rejecting statistical evidence in criminal law). *But see* Kaye, *The Laws of Probability and the Law of the Land*, 47 U. CHI. L. REV. 34 (1979) (arguing that probability theories could be used effectively and opponents should have burden of disproving theories' utility).

The problems raised by identification cases such as People v. Collins, 68 Cal. 2d 319, 438 P.2d 33, 66 Cal. Rptr. 497 (1968), have crystallized in litigation over parenthood. *See* Tribe, *supra*, at 1334-38 (discussing Collins case). The scientific technique of HLA typing allows one to exclude up to 90% of falsely identified men in paternity cases. It is, however, based on statistical techniques and courts have only slowly accepted it. *See* Cramer v. Morrison, 88 Cal. App. 3d 873, 153 Cal. Rptr. 865 (1979); Malvasi v. Malvasi, 167 N.J. Super. 513, 401 A.2d 279 (1979); *see also* Black & Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation*, 52 FORDHAM L. REV. 732 (1984) (asserting that toxic tort plaintiffs must prove by introducing statistical evidence that it is "more likely than not" that accused substance caused harm).

As an aside, Posner expressed Epstein's view in DePass v. United States, 721 F.2d 203, 207 (7th Cir. 1983) (Posner, J., dissenting), a case in which Posner embraces probabilistic evidence.

play of several concepts. But for causation or cause in fact, which reflects commonly held assumptions about causation as well as certain moral and political notions of responsibility, tends to dominate the disposition of tort claims. Moreover, this rendition of but for causation coincides neatly with that of corpuscularian science. Probabilistic linkage[104] is distinguished from but for cause, but has a nebulous role in Anglo-American legal reasoning. Probabilistic causal notions correspond to the causal notions that modern science employs in that they are based on probabilistic evidence rather than simple deductively derived causal chains. Legal scholars generally have not assumed the existence of a singular causal power, nor have they used probabilistic notions in analyses of causation, but rather have relied on the policy-laden concept of proximate cause to identify the bearer of liability.[105]

From this discussion of competing notions of causation in law and science emerges a hypothesis that explains why courts have so much trouble with causation issues in toxic tort litigation. The scientific association between a toxic substance and injury to a person relies on probabilistic evidence: epidemiological studies and statistical associations.[106] Philosophers of science readily accept such evidence and, indeed, acknowledge that probabilistic reasoning dominates much of physics and medicine.[107] In corpuscularian writing, probabilistic evidence is second best, if acceptable at all, and corpuscularian notions of causation coincide with but for concepts of causation in tort law. Both rely heavily on causal chain analyses and individual actions. Corrective justice aspects of tort law assume the existence of traceable causal chains leading from actor to harm. As a result, tort law tends to induce a corpuscularian approach to scientific evidence. Litigants bringing scientific issues to court are expected to show causes in fact or but for causes, with minimal support from the policies of proximate cause.

A corpuscularian judge would not want to deal with probabilistic notions, as he would regard these as inferior methods of reason-

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104 I use the term probabilistic linkage for Calabresi's causal linkage because I think it better expresses the nature of the concept.

105 It is not surprising that neither the notion of causal linkage nor Epstein's corrective justice are part of what Borgo calls the legal orthodoxy. *See* Borgo, *supra* note 73, at 425. The analysis I have offered, however, is skewed towards that of Borgo. Epstein's analysis of causation relies on strict causal chains supported by morality. Borgo's legal orthodoxy also heavily relies upon causal chain analysis. Causal linkage, on the other hand, embraces probabilistic notions of causation. Judges are guided by proximate causation, but moral concepts discussed by Epstein play a role in judges' thinking. Any incorporation of Epstein's concept of morality into legal reasoning leads to causal chain analysis playing a more central role.

106 *See infra* notes 171-214 and accompanying text.

107 *See* D. BOHM, *supra* note 50; P. SUPPES, *supra* note 68.

ing. Rather than accept probabilistic statements, a corpuscularian judge would delay a decision until deductive, mechanistic, but for causes are available. Nor would a corpuscularian judge welcome uncertainty in a scientific issue—uncertainty will be overcome according to positivism, and it is best to wait until this occurs. In addition, tort law's corrective justice aspects would not permit uncertainty in the causal assignment of responsibility.[108]

In this regard, common law courts are neither unscientific nor ignorant. Rather, they cling to conceptions of individual responsibility that coincide neatly with eighteenth century science's notions of causation. Thus, it is not enough simply to say that courts should adopt probabilistic reasoning. They must be instructed. But given the importance of the moral concept of individual responsibility in tort law, we can expect courts to accommodate only so much probabilistic reasoning.

Unfortunately, toxic substance injury cases cannot produce mechanistic, deductively-derived causal evidence, and a corpuscularian judge cannot process the available probabilistic evidence. Thus, the causation problem in toxic tort litigation could result from an epistemological quandary. Judges, using but for causation when analyzing tort claims, may slip into corpuscularian reasoning about scientific evidence, even when that evidence is primarily probabilistic.[109]

2. *Case Law*

The explanation of the causation problem in hazardous-substance litigation outlined above remains only a hypothesis, which is proved only if the case law indicates that judges avoid probabilistic evidence and demand but for causes. Judges could avoid probabilistic evidence in two ways. First, they could simply refuse to accept such evidence. Second, they could focus only on the aspects of the probabilistic evidence that resemble a causal chain, while ignoring the scientifically more important probabilistic evidence.[110]

The initial cases indicating corpuscularian attitudes toward sci-

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108 Even less uncertainty is permitted in criminal law. *See* Tribe, *supra* note 103, at 1372.

109 I do not mean to say that judges necessarily reach the wrong conclusion. Perhaps they are using a notion of causation akin to that outlined by Epstein. *See* Esptein, *Strict Liability*, *supra* note 93.

110 Although relatively few judicial decisions regarding injuries from toxic substances exist (probably because many cases are resolved without published opinions) more are becoming available. *See* Brennan & Carter, *supra* note 56, at 53-55. Statistical evidence provides the basis for claims in other areas of civil litigation. Black and Lilienfeld have exhaustively catalogued these. *See* Black & Lilienfeld, *supra* note 103, at 770-73.

ence and disease were the so-called traumatic cancer cases. Although science has not identified traumatic accidents as a cause of cancer, judges have relied on evidence of traumatic blows or dermal irritations when deciding issues of cancer causation.[111] This attitude evinces corpuscularianism: a mechanistic sense of causation allows a judge to identify a "but for" cause when the available scientific evidence is probabilistic in nature.

When a traumatic event was not available to provide a simple causal chain from exposure to injury, courts usually have chosen to ignore[112] or to sidestep causation issues.[113] Other courts have refused outright to consider any epidemiological evidence,[114] particularly evidence of low-level exposure integral to epidemiology.[115] Many of those courts that have taken into account probabilistic evidence have not used it to find causation.[116]

Some courts have discussed the relation of probabilistic evidence to legal causal evidence. For instance, the court in *Crim v. International Harvester Co.*[117] noted that "medical testimony that a particular type of work statistically increases the probability of getting valley fever does not constitute evidence of a causal connection between the disease and the employment, even when the doctor uses the magic phrase 'reasonable medical certainty.' "[118] In other words, statistical evidence cannot prove causation. *Miller v. National Cabinet Co.*[119] provides another example of corpuscularian judicial reasoning. In *Miller*, a wrongful death action was brought on behalf of a worker exposed to benzol who died of leukemia. The court noted that "[t]he only possible basis for drawing an inference in

111 *See* Black & Lilienfeld, *supra* note 103, at 741; Comment, *Judicial Attitudes Towards Legal and Scientific Proof of Cancer Causation*, 3 COLUM. J. ENVTL. L. 344, 349-54 (1977).

112 *See, e.g.*, Pritchard v. Liggett & Meyers Tobacco Co., 295 F.2d 292 (3d Cir. 1961) (suit against tobacco company for lung cancer injuries).

113 State Compensation Fund v. Joe, 25 Ariz. App. 361, 543 P.2d 790 (1975).

114 Prudential Ins. Co. v. Gourley, 267 F.2d 156 (5th Cir. 1959) (court ruled that probabilistic information did not bear on heart attack susceptibility issue).

115 *See* Clark v. State Workmen's Compensation Comm'r, 155 W. Va. 726, 753, 187 S.E.2d 213, 277 (1972) (court ignored expert testimony about possibility of chronic as well as acute exposures to toxic chemicals while deceased employed at chemical plant).

116 *See, e.g.*, Lartigue v. R.J. Reynolds Tobacco Co., 317 F.2d 19 (5th Cir. 1963) (court noted that jury never got past causation issue in lung cancer from smoking case); *see also* Mahoney v. United States, 220 F. Supp. 823, 840 (E.D. Tenn. 1963) (without considering impact of new evidence, court refused to accept "10,000 to 1" statistical probability that Hodgkins disease resulted from radiation at Oak Ridge National Laboratories); Amorosco v. Tubular & Cast Prods. Mfg. Co., 13 N.Y.2d 992, 194 N.E.2d 694, 244 N.Y.S.2d 787 (1963) (mem.) (plumbing supplies manufacturer's employee denied compensation, even though court accepted evidence of increased incidence of lung cancer among plumbers).

117 646 F.2d 161 (9th Cir. 1981).

118 *Id.* at 165.

119 8 N.Y.2d 277, 168 N.E.2d 811, 204 N.Y.S.2d 129 (1960).

favor of claimant . . . would be statistics indicating that in many instances leukemia follows benzol exposure without knowing why."[120] The court's inference is that one knows something only when a mechanistic causal chain is identified, and statistical inferences do not contribute to such knowledge. Courts want but for causes from science—causes that bear directly on the individual. For example, one court in discussing estimates of damages, stated, "Expectancy or statistical data about a group do not establish concrete facts about an individual. An attempt to make what is at best a sheer guess more precise by introducing another uncertain factor is not apt to improve the accuracy of a calculation . . . ."[121] Thus, courts have been willing to reject probabilistic evidence, even when that evidence exemplifies the hypothesis creation and observation that now form the basis of scientific research. Courts do not expect discussion of uncertainty from science; they want causal chains that lead to but for causes.

Over the past ten years, plaintiffs have brought a growing number of suits that rely on probabilistic proof of causation, thereby forcing courts to weigh such evidence more critically. Indeed, some courts have begun to discuss probabilistic evidence in a knowledgeable and insightful manner.[122] These decisions implicitly recognize the heterogeneity of scientific evidence and investigation and implicitly support the use of probabilistic causation. Some judges have recoguized that legal notions of causation may be out of step with scientific ones. For instance, in *Todd Shipyards Corp. v. Turbine Service, Inc.*,[123] the court made the following observation: "In doing so, he was not the only witness who exhibited how differently engineers and attorneys look at the concepts of fault. Technical witnesses, dealing in uncertainties and statistical probabilities, seemed at times to reflect a completely different view of responsibility from that which attorneys are accustomed to."[124]

Nonetheless, most courts have continued to insist on "but for"

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120 *Id.* at 283, 168 N.E.2d at 814, 204 N.Y.S.2d at 133.

121 Heckman v. The Federal Press Co., 587 F.2d 612, 617 (3d Cir. 1978); *see also* Garner v. Hecla Mining Co., 19 Utah 2d 367, 370, 431 P.2d 794, 796 (1967) ("While it seems logical that the unusually high incidence of lung cancer in uranium miners would indicate in the same ratio the higher probability than otherwise that such was the cause of the disease, it nevertheless falls short of compelling a finding that such was the cause in any individual case.").

122 *See* Punnet v. Carter, 621 F.2d 578, 586-87 (3d Cir. 1980) (court calls for better statistical methodology in radition induced birth defects case); *see also In re* High, 638 P.2d 818 (Colo. Ct. App. 1981) (court reversed lower court's decision to deny benefits to uranium mine inspector with lung cancer because he smoked a pipe); McCormick v. United Nuclear Corp., 89 N.M. 740, 557 P.2d 589 (1976) (recovery allowed for lung cancer suffered by uranium miner).

123 467 F. Supp. 1257 (E.D. La. 1978).

124 *Id.* at 1287 n.12.

or "sufficient" causes and have refused to accept the probabilistic evidence upon which physicians and scientists have long relied in understanding disease and treating patients. The court in *Union Carbide v. Industrial Commission*,[125] for example, stated that

> there has been some dispute over the 'sufficient to cause' wording of the *Smith* test. The problem is that the legal definition is at variance with the medical definition. The medical experts do not speak of concentrations which are 'sufficient to cause' occupational diseases, but rather refer to concentrations which 'increase the risk' of contracting a disease.[126]

In the past three years, the trickle of hazardous-substance injury suits has become a flood. Increasingly, these cases have required courts to examine statistical and epidemiological evidence of causation. A few courts have come to terms with the epidemiological evidence presented by plaintiffs and defendants.[127] Unfortunately, even when courts are willing to review such evidence, they show a tendency to fall back into a corpuscularian state of mind when the probabilistic evidence is confusing. When the epidemiological studies are in conflict, or when they have deficiencies in their design, courts are placing more weight on the "treating" physician's decision about the cause of the disease in the individual patient, regard-

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125 196 Colo. 56, 581 P.2d 734 (1978).

126 *Id.* at 61 n.6, 581 P.2d at 737 n.6.

127 In Johnson v. American Cyanamid Co., 239 Kan. 279, 718 P.2d 1318 (1986), for example, the Kansas Supreme Court closely examined the evidence of the risk presented by Sabin oral polio vaccine. Johnson was stricken with polio after his daughter was vaccinated. The vaccine manufacturer was aware of the minuscule risk that this could occur and warned the administering physician of this risk in the vaccine package insert. Notwithstanding the warning, a jury awarded the plaintiff $10,000,000. On appeal, the Kansas Supreme Court carefully reviewed the epidemiological evidence for the risk of polio to a vaccinated child's parent and overturned the jury verdict. This outcome should be applauded by public health advocates, given that the risk of such occurrences are far outweighed by the benefits of mass vaccination.

Oxendine v. Merrell Dow Pharmaceuticals, Inc., 506 A.2d 1100 (D.C. Cir. 1986), provides another example of a court carefully examining probabilistic evidence. In this case the District of Columbia Court of Appeals overturned a trial court's decision to grant the defendant's motion for judgment n.o.v. for a child who allegedly was harmed in utero by the anti-emetic drug, bendectin. The court demonstrated considerable understanding of toxicological evidence by reviewing the four principal grounds for identifying toxicity: structure-activity information; in vivo, or animal bioassay studies; in vitro or short term screening assays; and epidemiology. *Id.* at 1104-08. The court also analyzed in detail the expert witnesses' discussions of the epidemiological studies that provided the most important evidence for bendectin's teratogenic properties. *Id.* The opinion contains a long review of relative-risk calculations and the role of confidence intervals in decisions about statistical significance. *Id.* at 1108-09. This decision demonstrates that courts are slowly beginning to abandon insistence on "but for" causes, and are showing more willingness to review epidemiological evidence.

Other courts have analyzed epidemiological evidence and found some heterogeneity in the assumptions that underlie the epidemiological studies in question—a critical insight. *See* O'Gara v. United States, 560 F. Supp. 786, 790 (E.D. Pa. 1983).

less of whether the physician based that decision on an understanding of all the available evidence or on merely anecdotal information.

The litigation concerning victims of Guillain-Barre syndrome (GBS) following swine flu vaccination provides a good example of this tendency. In most of these federal district court cases, the judges relied on one epidemiological study by the Center for Disease Control (CDC) that revealed a peak increased risk for GBS in the second and third weeks following the vaccination.[128]

Claims based on episodes of GBS that occurred after the six-week period following vaccination were denied. In several cases, however, the plaintiff presented a different epidemiological study that had found an increased risk for GBS beyond the six-week cutoff advocated by the U.S. Attorney. In *Gaul v. United States*[129] the court reviewed the alternative study in detail and demonstrated a great deal of sophistication in its analysis of attack rates and the assumptions underlying the conflicting studies.[130] The court rejected the alternative study and the plaintiff's claim.[131] Likewise, in *Sulesky v. United States*,[132] a plaintiff suffering the onset of GBS after the six-week cutoff relied on the alternative study and disputed the CDC study. On this occasion the court, rather than delving into analysis of the probabilistic evidence, instead relied on the treating physician's opinion regarding the relationship between the vaccine and the disease. The court stated that "expert epidemiological testimony is not determinative of the issue of causation in this case. . . . Rather, . . . the Court finds that the resolution of the causation issue turns on the testimony of the treating and evaluating physicians."[133] Because the treating physician thought that the vaccine had caused the GBS, the court found for the plaintiff.[134]

The *Sulesky* court never explained the special expertise that enabled this physician to determine the outcome of the case. Indeed, the court leaves the impression that the treating physician can rely

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128 Grubbs v. United States, 581 F. Supp. 536, 539 (N.D. Ind. 1984) (citing "Schonberger study," Schonberger, Bregman, Sullivan & Bolyai, *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 AM. J. EPIDEMIOLOGY 105 (1979)). *See also* Gaul v. United States, 582 F. Supp. 1122, 1127-30 (D. Del. 1984) (rejecting independent statistical evaluation of CDC data that claimed risk was present for up to 16 weeks); Bean v. United States, 533 F. Supp. 567, 577 (D. Colo. 1980) (summarizing expert testimony regarding nexus between some flu vaccine and Guillain-Barre Syndrome).

129 582 F. Supp. 1122 (D. Del. 1984).

130 *Id.* at 1127-31.

131 *Id.* at 1130.

132 545 F. Supp. 426 (S.D. W.Va. 1982).

133 *Id.* at 430.

134 *Id.*

on any sort of anecdotal evidence, even if his expertise comes only from the fact that he treated the injured individual. Thus, the court refused to analyze in detail the probabilistic evidence and turns to the physician to provide a "but for" cause in this case. In a case like *Sulesky*, the courts' tendency to defer to the treating physician's opinion[135] demonstrates the corpuscularian distrust of epidemiological evidence.

This kind of outcome might be more frequent in the future as a result of the decision of *Ferebee v. Chevron Chemical Co.*[136] *Ferebee* concerned a wrongful death action brought on behalf of the family of an agricultural worker at a government research center.[137] Mr. Ferebee had died of pulmonary fibrosis, allegedly caused by long term exposure of his skin to the chemical paraquat.[138] A jury awarded the Ferebee family $60,000.[139] Chevron Oil Company, the manufacturer of paraquat, appealed to the Court of Appeals for the District of Columbia.

The court's opinion began by carefully reviewing scientific evidence presented at trial. It noted that two experts examined Ferebee, one of whom was identified as the chief of the pulmonary branch of the National Institutes of Health's Heart, Lung and Blood Institute.[140] Both experts testified that long-term dermal absorption of paraquat caused the deceased's pulmonary fibrosis.[141] Chevron countered that the side effects of paraquat were limited to acute toxicities[142] and that the concept of dermal absorption leading to pulmonary fibrosis represented a novel theory, wholly unsupported by epidemiological studies.[143]

The court insisted that the jury was entitled to decide the causation issue and could properly base its decision on the plaintiff's two expert witnesses' testimony. It further stated that courts can decide cases even when the expert opinion on which the court relies is not generally accepted by the scientific community. In an extremely important passage, the court noted:

> Thus, a cause-effect relationship need not be clearly established by animal or epidemiological studies before a doctor can testify that, in his opinion, such a relationship exists. As long as the basic

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135 There is little doubt that courts have a tendency to defer to medical expertise and especially to treating physicians. *See* Katz, *Informed Consent—A Fairy Tale?: Law's Vision*, 39 U. PITT. L. REV. 137, 148 (1977).

136 736 F.2d 1529 (D.C. Cir. 1984).

137 *Id.* at 1532.

138 *Id.* at 1533.

139 *Id.* at 1532.

140 *Id.* at 1533.

141 *Id.*

142 *Id.* at 1535.

143 *Id.*

> methodology employed to reach such a conclusion is sound, such as use of tissue samples, standard tests, and patient examination, products liahility law does not preclude recovery until a "statistically significant" number of people have been injured or until science has had the time and resources to complete sophisticated laboratory studies of the chemical. In a courtroom, the test for allowing a plaintiff to recover in a tort suit of this type is not scientific certainty, but legal sufficiency . . . .[144]

The court probably considered two issues in denying Chevron's appeal: the testimony of an expert witness who was truly a leading expert in the area of pulmonary pathology and the unfairness of penalizing the earliest victims of any hazardous substance.[145] Nonetheless, the court's decision does suggest a certain corpuscularian frame of mind, in that it downplays the probabilistic evidence that toxicologists utilize, and re-affirms the importance of the examining physician's conclusion. This kind of ruling enables other courts to ignore probabilistic evidence and to rely solely on the testimony of a treating physician, no matter how novel the causal chain upon which that physician relies.

The *Wells v. Ortho Pharmaceutical Corp.*[146] litigation demonstrates the kind of difficulties that the reasoning used in *Ferebee* can produce. In *Wells*, the parents of a child born with multiple birth defects brought a products liability action on behalf of the child against the manufacturer of a spermicide used by the child's mother before and after conception. At both parties' request, the case was tried without a jury. The court noted that the decision was difficult primarily because of the conflicting scientific evidence presented by the parties.[147] To illustrate the court's difficulty, it used over twenty pages in the opinion to review the testimony of fourteen key expert witnesses.[148] This review convincingly details the court's proposition that leading authorities in the field can disagree about the association between spermicides and certain birth defects.

This Article does not address the details of this scientific controversy, but instead focuses on the generic issues that the court raised. The first issue concerns the minimal weight that the court placed on defendant's evidence that no statistical association exists between the product and the injury. The court noted simply that "[a]lthough the studies on which defendant relied failed to detect an

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144 *Id.* at 1535-36.

145 These early victims, like Mr. Ferebee, would provide the data for studies upon which later victims could rely.

146 615 F. Supp. 262 (N.D. Ga. 1985), *aff'd*, 788 F.2d 741 (11th Cir.), *cert. denied*, 107 S. Ct. 437 (1986).

147 *Id.* at 266.

148 *Id.* at 269-91.

association between spermicides and birth defects, some of defendants' [sic] own experts testified that these studies do not rule out all possibility that spermicides can cause birth defects."[149] This finding could indicate that negative epidemiological studies will not sway a court. Unfortunately, it could also indicate that the party attempting to disprove an association between a product and an injury cannot do so on the basis of epidemiological evidence.

The second generic issue is the court's reliance on testimony concerning "mechanisms" or theories to demonstrate causation.[150] Specifically, experts testified about an amniotic-band syndrome and vascular-disruption hypotheses to explain the plaintiff's injury.[151] This testimony influenced the court largely because it was based on examination of the plaintiff. The court pointed out that it was concerned only with this plaintiff's injury.[152] Thus the court's opinion asserts that when epidemiologic evidence shows no association between the product and the disease, but hypothesized " 'mechanisms' of causation"[153] appear to support an association, the defendant is liable.

This interpretation of the *Wells* case shows how judges can be corpuscularian even when they are willing to consider epidemiological evidence. As noted above,[154] scientists use theories to develop hypotheses. They then test these hypotheses by experiments or by designing statistical or epidemiological studies. If the studies are sufficiently powerful,[155] and yet fail to show an association, then the scientist rejects the hypothesis. The *Wells* court could be reversing this line of reasoning. One can characterize the *Wells* court as embracing the hypothesis even though the epidemiological study failed to show an association. The court focused on the individual and relies on the mechanistic causal chain analysis evinced by the hypothesis.

The defendant appealed the *Wells* decision to the Eleventh Circuit, which affirmed the multimillion dollar verdict for the plain-

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149 *Id.* at 292.

150 *Id.*

151 *Id.* at 292-93.

152 *Id.* at 292.

153 *Id.*

154 *See supra* notes 68-70 and accompanying text.

155 *See infra* notes 210-14. *See also* Brennan, *Untangling Causation Issues in Hazardous Substance Litigation*, 107 ANNALS INTERNAL MED. 791 (1987). "Power" refers to the size of a sample. An epidemiological study may fail to demonstrate an association, but there are two possible reasons for such failure: (1) there is no association; and (2) the sample size was not large enough to reject the null hypothesis. A powerful study is one with a sample size that is calculated to be large enough to discriminate an association at a given level.

tiff.[156] On appeal, Ortho Pharmaceutical argued that the trial court had failed to consider adequately the epidemiological evidence of no association. The Circuit Court rejected this argument, relying squarely on *Ferebee*.[157] The court reiterated that a difference exists between legal and scientific causation.[158] Noting that it was most interested in the injury suffered by the individual before it, the court stated that "it does not matter in terms of deciding the case that the medical community might require more research and evidence before conclusively resolving the question."[159] Thus the *Ferebee* distinction between legal and scientific causation appears to allow courts to opt for the mechanistic causal chain reasoning that underlies a hypothesis even when experimental or epidemiological evidence has not proved that hypothesis.[160]

Such decisions create a great deal of uncertainty in the minds of litigants regarding liability because they cannot predict which mode of causation theory a court will use in evaluating conflicting evidence. One court might reject statistical data outright and choose a causal chain anecdote proposed by an expert; another might review the same evidence and accept a different anecdote. This kind of uncertainty over liability standards disrupts the future planning upon which corporations and their insurers depend.[161]

Common law courts' inability to incorporate probabilistic evidence into their legal analyses of responsibility and causation raises fundamental questions regarding their ability to adjudicate hazardous-substance injury cases. According to Epstein's corrective justice model of causation in torts, the court will seek mechanistic causal chains linking individual events to individual injuries.[162] The probabilistic evidence provided by toxicology and epidemiology cannot produce such causal chains.[163] Decisions like *Ferebee* and *Wells* suggest that the moral relationships between citizens in a liberal state are part and parcel of tort jurisprudence. These moral concepts will

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156 Wells v. Ortho Pharmaceutical Corp., 788 F.2d 741, 747 (11th Cir.) (affirming plaintiff's verdict, but reducing $5.1 million award to $4.7 million), *cert. denied*, 107 S. Ct. 437 (1986).

157 *Id.* at 745.

158 *Id.*

159 *Id.*

160 The *Ferebee* distinction played an important role in the case of Hawkinson v. A.H. Robins Co., 595 F. Supp. 1290 (D. Colo. 1984). In this case concerning injuries resulting from intra-uterine devices, the court intermixed preponderance of evidence issues with scientific uncertainty notions thereby obscuring the causation issue entirely.

161 Calfee & Craswell, *Some Effects of Uncertainty on Compliance with Legal Standards*, 70 VA. L. REV. 965 (1984); *see* Roe, *Mergers, Acquisitions, and Tort: A Comment on the Problem of Successor Corporation Liability*, 70 VA. L. REV. 1559 (1984).

162 See the discussion of Epstein's corrective justice model of causation, *supra* notes 94-103 and accompanying text.

163 *See infra* notes 206-14 and accompanying text.

force judges and juries into a corpuscularian state of mind, thus making satisfactory resolution of litigation involving probabilistic evidence of causation impossible.[164]

This is not to say that courts cannot understand statistical evidence.[165] In such diverse areas of the law as antitrust[166] and civil rights,[167] courts use probabilistic evidence. Moreover, courts have consistently found certain parties liable for hazardous-substance-induced injury on the basis of probabilistic evidence. Indeed, the "tort crisis" affecting industry and insurers has arisen from the massive liability that falls on asbestos companies and certain drug manufacturers.[168] Unfortunately, even these intensely litigated issues have not produced adequate discussions of causation. When a rare tumor is closely associated with a toxic substance, courts generally take the causation issue as a given. This is the case in the asbestos litigation[169] as well as in the diethylstilbestrol/vaginal adenocarcinoma cases.[170]

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164 Impossible, that is unless one argues that the moral theory underlying tort law is not liberal at all. Professor Robinson has developed an outline of this argument, based on a discussion of Kantian moral theory. *See* Robinson, *Probabilistic Causation and Compensation for Tortious Risk*, 14 J. LEGAL STUD. 779, 789 (1985). Professor Weinrib's discussion of Aristotlean morality and tort law is, I think, much more convincing, and implicitly supports Epstein's focus on the individual and mechanistic causation. *See* Weinrib, *Toward a Moral Theory of Negligence Law*, 2 LAW & PHIL. 37, 38-43 (1983).

165 *See* D. BARNES, STATISTICS AS PROOF (1983); W. CURTIS, STATISTICAL CONCEPTS FOR ATTORNEYS (1983).

166 *See* D. BARNES, *supra* note 165, at 294-95.

167 *See* Hazelwood School Dist. v. United States, 433 U.S. 299, 307-08 (1977); International Bhd. of Teamsters v. United States, 431 U.S. 324 (1977); Castaneda v. Partida, 430 U.S. 482 (1977). These cases do not demonstrate that the court had any grasp of probabilistic evidence. *See* Brennan & Carter, *supra* note 56, at 53-54.

In *Hazelwood*, *International Bhd. of Teamsters*, and *Castaneda*, all relatively early cases, the Court merely had to note the difference in proportions of minority groups in the population as opposed to that of the particular body at issue, such as the grand juries in *Castaneda*. This required little probabilistic reasoning. In more recent cases, the courts face more sophisticated analysis that includes regression analysis and multivariate techniques. The extent of the courts' ability to deal with this evidence is unclear, although there is a recent example of excellent judicial comprehension of these issues. *See* McNeil v. City of Springfield, 658 F. Supp. 1015 (C.D. Ill. 1987). A recent Supreme Court ruling on death penalty issues does not reveal the same level of comprehension. McClesky v. Kemp, 107 S. Ct. 1756 (1987).

168 *See* Rosenberg, *supra* note 3, at 853. *See generally* S. EPSTEIN, THE POLITICS OF CANCER (1979).

169 *See infra* text accompanying notes 215-31; *see also* Special Project, *An Analysis of the Legal, Social and Political Issues Raised by Asbestos Litigation*, 36 VAND. L. REV. 573, 607-25 (1983).

170 *See* Herbst, Ulfelder & Poskanzer, *Adenocarcinoma of the Vagina: Association of Maternal Stilbestrol Therapy with Tumor Appearance in Young Women*, 284 NEW ENGLAND J. MED. 878 (1971). Courts might be forced to review causation issues if there were evidence that conflicted with the original studies that produced the linkage of asbestos to asbestosis and lung cancer, or DES to adenocarcinoma. *See* Horwitz, *The Role of Susceptibility Bias in Epidemiological Research*, 145 ARCHIVES INTERNAL MED. 909 (1985).

Courts are troubled by the probabilistic evidence of causation with regard to hazardous substance injury. In this section I have outlined some theoretical explanations for these difficulties. Courts rely on mechanistic notions of causation and are confused by probabilistic ones. This reliance on mechanistic causes reflects the notions of responsibility inherent in liberalism. Moreover, courts assume that when mechanistic causes are not yet available, it is best to procrastinate, as science is constantly producing new mechanistic causes. As a result, courts are both corpuscularian and positivistic.

Having discussed the theoretical problems courts face when considering probabilistic evidence, I now consider the nature of such evidence and how its inherent probabilities and uncertainties challenge the norms of tort law.

III
TOXIC SUBSTANCE INJURY: EVIDENCE OF CAUSATION AND THE ROLE OF UNCERTAINTY

A. The Toxicology of Hazardous Substances

In the previous sections, I argued that courts are troubled by the probabilistic evidence involved in the proof of hazardous-substance injury. The difficulty that judges and lawyers, as well as juries, have with statistical and epidemiological evidence arises from the uncertainty involved in this kind of evidence, uncertainty that conflicts with causal chain analysis paradigms. A scientist develops hypotheses and then tests them by designing studies that indicate the strength of the underlying reasoning. When that test involves statistics, as it often does in biomedical science, the result is consciously hedged with uncertainty. Before courts can use probabilistic evidence to resolve legal problems raised by toxic substances, courts must understand the nature of the uncertainty in toxicological evidence.[171]

Toxicology is broadly defined as the science that deals with poisons and their effects.[172] In this Article, toxicology is restricted to those poisons that I have broadly termed "hazardous substances."[173] Hazardous substances are those occupational or environmental toxins or consumer products that are characterized by five properties. First, people are exposed to them in a chronic and

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171 I hope this discussion will also make clear the difficulties that arise in attempting to estimate the size of the threat posed by hazardous substances. *See supra* notes 13-36 and accompanying text.

172 WEBSTER'S THIRD NEW INTERNATIONAL DICTIONARY 2419 (1976).

173 *See supra* notes 11-13. This is distinguished from toxicology of acute poisonings. *See* CASSARETT & DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS (C. Klaassen, M. Amdur & J. Doull 3d ed. 1986).

relatively low-dose fashion. Second, exposed persons lack awareness of the toxic effect during the initial phase of the exposure. Third, the exposure is followed by a latency period before the disease or injury manifests itself. Fourth, the injury or disease at least produces chronic defects and is usually irreversible. Fifth, the hazardous substance is not left in the body in a way that firmly links the disease or injury with the substance.[174] The paradigm of hazardous substances is the occupational or environmental carcinogen. Substances such as teratogens, certain agents that cause chronic lung disease,[175] and heavy metals that produce neurological disease[176] exemplify these five characteristics of hazardous substances.

There are four methods for identifying carcinogens: 1) cluster analysis; 2) short term molecular assays; 3) animal bioassays; and 4) epidemiological studies. Variations of these methods are also used to identify all other toxic substances.

Cluster analysis, which analyzes diseases shared by members of a group exposed to a single hazardous substance, was the first method employed to identify carcinogens.[177] Recent examples of very rare tumors occurring in disparate populations linked only by exposure to a common chemical are clear cell adenocarcinoma of the cervix and vagina in women exposed in utero to diethylstilbestrol[178] and hepatic angiosarcomas in vinyl chloride workers.[179]

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174 One of the most exciting areas of toxicology is the use of molecular techniques to identify exposure. *See* Perera & Weinstein, *Molecular Epidemiology and Carcinogen-DNA Adduct Detection: New Approaches to Studies of Human Cancer Causation*, 35 J. CHRONIC DISEASES 581 (1982); Perera, Poirier, Yuspa, Nakayama, Jaretzki, Curren, Knowles & Weinstein, *A Pilot Project in Molecular Cancer Epidemiology: Determination of Benzo[a]Pyrene DNA Adducts in Animal and Human Tissues by Immunoassays*, 3 CARCINOGENESIS 1405 (1982) [hereinafter Perera & Poirier].

Talbot has used many of the same elements to distinguish "environmental risks" from "classic pollution." Talbot, *A Generic View of Toxic Chemicals and Similar Risks*, 7 ECOLOGY L.Q. 207, 207-08 (1978). I think that Talbot's dichotomy disintegrates, given certain examples, and that it is best to maintain the more general definition of hazardous substances.

175 Byssinosis, for example, is a chronic lung disease caused by exposure to cotton dust. *See* Bouhuys, Schoenberg, Beck & Schilling, *Epidemiology of Chronic Lung Disease in a Cotton Mill Community*, 154 LUNG 167 (1977); Merchant, Halprin, Hudson, Kilburn, McKenzie, Hurst & Bermazohn, *Responses to Cotton Dust*, 30 ARCHIVES ENVTL. HEALTH 222 (1975).

176 A variety of chronic neurological diseases result from long term exposure to lead. *See* Cullen, Robins & Eskenazi, *Adult Inorganic Lead Intoxication: Presentation of 31 New Cases and a Review of Recent Advances in the Literature*, 62 MEDICINE 221 (1983).

177 Percivall Pott used paradigmatic cluster analysis in uncovering the association between scrotal cancer and chimney sweeping: he noticed a rare tumor affecting an unexpectedly high number of people who shared a common exposure. Hermo, *Chemical Carcinogenesis: Tumor Initiation and Promotion*, 2 OCCUPATIONAL MED. 1, 6 (1987).

178 The association of clear cell carcinoma of the cervix with diethylstilbestrol was detailed in the early 1970s. *See* Herbst, Ulfelder & Poskanzer, *supra* note 170.

179 The association of vinyl chloride with hepatic angiosarcomas also became clear

The second method for identifying carcinogens relies on short-term screening assays.[180] Screening assays are designed to test for carcinogenicity prospectively and inexpensively. They are an initial hurdle that substances must clear; those that fail are then targets for further studies. Short-term assays are based on certain molecular theories of carcinogenesis regarding the disruption of deoxyribonucleic acids (DNA). Similar disruption is thought to occur in mutagenesis.[181] Therefore, attention has long focused on the correlation of carcinogenesis with mutagenesis.[182]

A mutation is an abrupt and heritable genetic change. Thus a mutation is any change in the genetic material of the cell that is passed to following generations. This may mean a change in a single nucleotide (individual molecule of DNA), in several nucleotides within the same gene (a functional group of DNA molecules within a chromosome), or in an entire chromosome. Mutations may change a normal gene into a mutant (forward mutation) or a mutant gene into a normal one (reverse mutation).[183]

Researchers use several different types of assays.[184] These tests

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in the 1970s. *See* Spirtas & Kaminski, *Angiosarcoma of the Liver in Vinyl Chloride/Polyvinyl Chloride Workers: 1977 Update of the NIOSH Register*, 20 J. OCCUPATIONAL MED. 427 (1978).

180 *See generally* IARC REPORT, *supra* note 32; STRATEGIES FOR SHORT TERM TESTING FOR MUTAGENS/CARCINOGENS (B. Butterworth ed. 1977) [hereinafter STRATEGIES]; CARCINOGENESIS AND MUTAGENESIS TESTING (J. Douglas ed. 1984); Ames, *Identifying Environmental Chemicals Causing Mutations and Cancer*, 204 SCIENCE 587 (1979).

181 Slater, Anderson & Rosenkranz, *Rapid Detection of Mutagens and Carcinogens*, 31 CANCER RES. 970, 971-72 (1971).

182 Although early studies failed to demonstrate this correlation, in the past 15 years many laboratories have shown that most known carcinogens are mutagens. This then justifies the assumption that the reverse is true: mutagenic agents are probably carcinogenic. *See* Haworth, *An Overview of Short-Term Testing* in CARCINOGENESIS AND MUTAGENESIS TESTING, *supra* note 180, at 1, 4; *see also* Slater, Anderson & Rosenkranz, *supra* note 181.

183 *See* Brusick, *Consequences of Genotoxic Effects*, in CARCINOGENESIS AND MUTAGENESIS TESTING, *supra* note 180, at 17, 23-24. There are several specific types of mutations. Point mutations are either base pair substitutions (one individual pair of basic chemicals that cross link the DNA double helix are replaced by another pair of such chemicals) or frame shift changes (several of the so-called base pairs are deleted). *Id.* at 21; *see also* Ames, *supra* note 180, at 589.

184 Bacterial genotoxicity assays use large amounts of bacteria to identify mutations following exposure of the bacteria to a chemical. For instance, the Ames test is a reverse mutation assay using salmonella bacteria. Ames, *supra* note 180, at 589. There are also tests that use E. coli in reverse mutation assays. Forward mutation assays and DNA damage assays are less frequently used forms of baterial genotoxicity studies. *See* Mohn & Ellenberger, *The Use of Escherichia Coli* K12/343/113 (λ) *as a Multi-purpose Indicator Strain in Various Mutagenicity Testing Procedures*, in HANDBOOK OF MUTAGENICITY TEST PROCEDURES 95 (B. Kilbey, M. Legator, W. Nichols & C. Ramel eds. 1977).

Mutagen assays can be completed using mammalian cells growing in a culture dish. The genetic markers that demonstrate a mutation are usually related to drug resistance: the culture becomes resistent to the normal effect of a drug and the mutant enzyme is resistant to the drug's effects. Bradley, Bhuyan, Francis, Langenbach, Peterson & Huberman, *Mutagenesis by Chemical Agents in V79 Chinese Hamster Cells: A Review and Analysis of*

have shown that 90% of known carcinogens are also mutagens.[185] Questions remain concerning how best to combine the tests to identify the substances that are mutagens, and thus presumably carcinogens.[186] At this point, short-term assays can roughly identify mutagens, and presumably carcinogens, in a prospective and relatively inexpensive fashion.

The next step up from short term assays in terms of cost, expediency, and accuracy of identification of carcinogens is the animal bioassay test. As the International Agency for Research on Cancer (IARC) has noted, "The essence of long term testing is to observe test animals for a major portion of their lifespan for the development of neoplastic lesions after or during exposure to various doses of a test substance by an appropriate route."[187] Scientists assume that substances that are carcinogenic in animals are carcinogenic in humans—an assumption reinforced by comparing animal bioassays and cluster analysis results.[188] Animal bioassays, although conceptually simple, are quite costly, require two to three years to complete, and require the commitment of trained personnel and scarce resource facilities.[189]

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the Literature, 87 MUTATION RES. 81 (1981). These types of tests have shown that some known carcinogens are mutagens even when bacterial genotoxicity tests failed. Yet another technique that can demonstrate point mutation is based on a cell's ability to repair DNA. *Id.*

Mutations occurring at a chromosomal level are detected by chromosome-aberration tests. Some mutagens will disrupt the structure of the chromosome, and these disruptions are observable using a light microscope. A special subset of such tests involves drosophila flies. Research has characterized the lethal recessive genes on the drosophila X chromosomes especially well. A mutation will allow expression of these recessive lethal genes—drosophila death thus marks mutagenesis. *See* Latt, Allen, Bloom, Carrano, Falke, Kram, Schneider, Schreck, Tice, Whitfield & Wolff, *Sister-Chromatid Exchanges: A Report of the Gene-Tox Program*, 87 MUTATION RES. 17 (1981).

185 IARC REPORT, *supra* note 32, at 295.

186 The options are "tier" and "battery" testing. In tier testing, an agent is tested further only if it is positive in a less sensitive test—a substance proceeds up the ladder of tests to a certain point, where it is termed a mutagen. In battery testing, each compound is tested in every test. *See* Butterworth, *Recommendations for Practical Strategies for Short-Term Testing for Mutagens/Carcinogens*, in STRATEGIES, *supra* note 180, at 89, 90.

187 *See* IARC REPORT, *supra* note 32, at 24.

188 PUBLIC HEALTH SERV., U.S. DEP'T OF HEALTH AND HUMAN SERVS., FOURTH ANNUAL REPORT ON CARCINOGENS: SUMMARY, 1985, at 9.

189 Of known human carcinogens, only arsenic fails to cause cancer in animals. IARC REPORT, *supra* note 32, at 24. In developing an animal bioassay, researchers must consider several factors. First, the researcher must carefully characterize the test substance, considering all possible impurities and special biochemical properties. *Id.* at 28-30. Second, the researcher must carefully select the animal species. Certain strains of mice or rats with known levels of normal tumor incidence have become the test animals of choice. *Id.* at 30-32. Next, the researchers must select the exposure, preferably using routes which most closely simulate human exposure. *Id.* at 33-35; *see also* Page, *Current Concepts of a Bioassay Program in Environmental Carcinogenesis*, in 3 ADVANCES IN MODERN TOXICOLOGY: ENVIRONMENTAL CANCER 87, 90-93 (H. Kraybill & M. Mehlman eds. 1977).

Perhaps the most controversial issue in constructing an animal bioassay study is the

Animal bioassays begin with an initial hypothesis of the carcinogen having no effect (the null hypothesis). Thus, researchers expect that in each treatment group, those not exposed to the substance (controls) and those exposed to it will have equal numbers of tumors. The amount by which the experimental results differ from the null hypothesis can be summarized in a "trend test statistic," which is simply the summation of the difference between the observed numbers of tumors and the expected (control group) number of tumors.[190] The trend test statistic can then be converted into a p-value.[191] The p-value is defined as the probability of observing an apparent effect of exposure as great or greater than that actually found if chance alone were responsible for any apparent effects seen in the data. Statistical significance is usually arbitrarily defined as a p-value of less than .05; that is, chance alone can explain the results of the experiment only five percent of the time.[192]

If an animal bioassay is positive, meaning the substance causes cancer in the laboratory animals, then researchers assume that the substance is carcinogenic in humans. Researchers then attempt to estimate the dose of carcinogen required to provoke the cancer response in humans. First, the researcher employs scaling factors that compare the dose of carcinogens received by animals to human exposure. After determining the morbidity and mortality associated with a carcinogen at a relatively high dose, the researcher "extrapolates" the data to a lower dose and mortality range. In order to extrapolate, the researcher must assume that the carcinogen dose and cancer response vary in a consistent manner that can be described by a mathematical equation. Researchers have proposed a number of such equations or models.[193] If a good "fit" is found, the re-

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carcinogen dose range. Scientific Committee, Food Safety Council, *Proposed System for Food Safety Assessment*, 16 FOOD & COSMETICS TOXICOLOGY, Dec. 1978, at 1, 101 (Supp. 2). Scientists doing animal bioassays usually try to calculate a maximum tolerated dose (MTD) from subchronic feeding and pharmacokinetic studies. This dose is usually proportionally much higher than the levels to which humans are exposed. This is necessary, however, because of the relatively short life spans of the bioassay tests, and because of the limited number of animals that can be tested. Once the study is in progress, animals must be evaluated carefully on a daily basis. Any deaths must receive critical pathological review. The same is true at termination of the study period when the test animals are sacrificed. *See* Saffiotti, *Identification and Definition of Chemical Carcinogens: Review of Criteria and Research Needs*, 6 J. TOXICOLOGY & ENVTL. HEALTH 1029 (1980).

190 T. COLTON, STATISTICS IN MEDICINE 116-17 (1974).

191 This conversion assumes that the trend statistics from an infinite number of identical bioassays would simulate a normal distribution—meaning that they would resemble a bell-shaped curve if graphed with one axis representing the probability of occurrence, and the other representing the trend statistic itself.

192 *See* J. FREUND, MATHEMATICAL STATISTICS 387-418 (3d ed. 1980); T. COLTON, *supra* note 190, at 115.

193 *See, e.g.*, Brown, Fears, Gail, Schneiderman, Tarone & Mantel, *Models for Carcino-*

searcher uses the mathematical function to calculate a response at a given low dose.

Unfortunately, researchers can usually "fit" data from a given bioassay to several models.[194] A bioassay experiment provides a limited amount of discrete data which, when represented on a graph, describe a curve. If the graph flattens out completely at low dose levels, it means that a further reduction of dose will not reduce cancer incidence at all—the so-called threshold effect. If the graph continuously declines as dose level decreases, it means that every decrement of dose further decreases cancer response. Unfortunately any one experiment's dose/response curve can be simulated by a number of dose/response models.[195] Thus many models fit a single set of data, ensuring controversy whenever such models are used.[196]

The fourth method for identifying carcinogens is epidemiology.

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genic Risk Assessment 202 SCIENCE 1105 (1978); Crump, *Dose Response Problems in Carcinogenesis*, 35 BIOMETRICS 157 (1979).

194 Certain assumptions and inadequacies attend each of the major dose-response models. Each model has different implications for setting threshold levels of carcinogens.

The Mantel-Bryan model assumes that response varies in a normal distribution with the logarithm of the dose. Although fairly simple to calculate, the Mantel-Bryan model does not fit data particularly well. The curve it describes becomes very shallow at low doses, thus underpredicting low-dose risks as compared to other models. *See* Mantel & Bryan, *"Safety" Testing of Carcinogenic Agents*, 27 J. NAT'L CANCER INST. 455 (1962). Just the opposite is true of the one-hit model, which assumes that response is directly proportional to the exponent of the carcinogen dose: it estimates unacceptable risks at even very low doses. *See* Crump, Hoel, Langley & Peto, *Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment*, 36 CANCER RES. 2973 (1976). The multistage model, a polynomial as well as an exponential equation, assumes a cell must go through several stages before becoming cancerous. Because of the algorithm used to fit the multistage model to data, the model has a diminished ability to predict dose-response curves that are flat low doses, but rise steeply with increased dose. *See* Peto, *Epidemiology, Multistage Models, and Short Term Mutagenicity Tests*, in ORIGINS OF HUMAN CANCER: BOOK C—HUMAN RISK ASSESSMENT 1403 (H. Hiatt, J. Watson & J. Winsten eds. 1977). The multihit model, which assumes a cell site must be hit by a certain number of toxic molecules before the cell becomes cancerous, requires the integration of the dose exponent. This complex equation may generate very high safe doses if more than one hit is required to initiate cancer. *See* Rai & Van Ryzi, *A Generalized Multihit Dose-Response Model for Low-Dose Extrapolation*, 37 BIOMETRICS 341 (1981).

195 At low dosages, the models tend to diverge and the experimental data fails—researchers cannot assemble a sufficiently large animal study group to allow calculation of morbidity rates at low doses.

196 This is further complicated by the fact that the current understanding of cancer biology does not allow discrimination between the models. As a result, many have advocated abandoning quantification of risks altogether, asserting simply that there are not safe levels of carcinogens. *See* Robbins, *Risk Assessment: Too Complex, Too Soon*, in MANAGEMENT OF ASSESSED RISK FOR CARCINOGENS 59 (Annals N.Y. Acad. Sci., Vol. 363, W. Nicholson ed. 1981) [hereinafter MANAGEMENT OF ASSESSED RISK]; S. Jellinek, *On the Inevitability of Being Wrong in Management of Assessed Risk for Carcinogens*, in MANAGEMENT OF ASSESSED RISK, *supra*, at 43; *see also* OSHA, *Identification and Control of Carcinogens in the Work Place*, 45 FED. REG. 5002 (1980) (codified at 29 C.F.R. pt. 1990).

Epidemiology applies statistical techniques and probabilistic reasoning to disease incidence. Epidemiologists study groups or populations of people and attempt to demonstrate statistically significant correlations or associations between certain clinical outcomes and personal habits, characteristics, or exposures to any number of variables.[197] When directed at the identification of chemical carcinogens, epidemiologists usually describe groups of people exposed to certain substances and compare these groups' health with the health of nonexposed controls.

An epidemiological study, unlike an animal bioassay study, is not really an experiment. Researchers cannot control the factors that affect the quality of the data. Exact quantitative estimation of exposure levels is usually impossible, and so dose/response curves are extremely difficult to complete. Thus epidemiology contains even more uncertainty than short-term assays, and animal bioassay studies.[198]

Epidemiological studies are either retrospective or prospective. A retrospective study focuses on a group of people with a given disease and attempts to discern the factors common to the group. A prospective study follows a group of people with a common exposure over a period of time looking for disease incidence. The best example of a retrospective study is the case-control study; cohort studies are the most widely-used prospective studies.[199] Cluster analysis is a descriptive type of the retrospective study. Although cluster analysis reveals nothing about the nature of the substance's carcinogenic action, the clear association between exposure and rare tumors given by cluster analysis usually leaves little doubt about the substance's carcinogenicity.[200] Cluster analysis has two major drawbacks. First, it is retrospective; cancers have already occurred when the cluster is noticed. Second, it has limited utility because the clear associations between substances and tumors that are susceptible to cluster analysis are quite rare. Cluster analysis can therefore identify only a small number of carcinogens.

The nature of an epidemiological study's attribution of carcinogenicity can be illustrated by outlining an epidemiologist's work in a

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197 B. MACMAHON & T. PUGH, EPIDEMIOLOGY: PRINCIPLES AND METHODS 13-27 (1970); G. FRIEDMAN, PRIMER ON EPIDEMIOLOGY 1-5 (2d ed. 1980).

198 Epidemiologists are quite aware of the uncertainty involved in epidemiological thinking, and of the nature of their causal attributions. *See* M. SUSSER, CAUSAL THINKING IN THE HEALTH SCIENCES: CONCEPTS AND STRATEGIES OF EFIDEMIOLOGY 45-48 (1973); H. BLALOCK, CAUSAL INFERENCES IN NONEXPERIMENTAL RESEARCH 5 (1964); *see also*, Buck, *Popper's Philosophy for Epidemiologists*, 4 INT'L J. EPIDEMIOLOGY 159 (1975).

199 *See generally* D. LILIENFELD, FOUNDATIONS OF EPIDEMIOLOGY 164-67, 194-95 (1976) (describing methodology of retrospective and prospective studies).

200 For example, cluster analysis revealed cases of very rare hepatic angiosarcomas in workers at vinyl chloride processing plants. Spirtas & Kaminski, *supra* note 179.

given study. Consider, for example, a cohort study of a number of employees in a particular hazardous occupation. The health status of the chosen group is defined at the outset, and then followed periodically over a number of years while researchers record characteristics of work history and exposure status. Throughout the study, incidence of disease in the study group is compared with that in a control group—a group similar to the study group except for exposure status. The study ends at a designated time and rates of disease occurrence as well as comparative risks of disease between the two groups are calculated. With this data, the researcher tests for statistical significance, and posits whether the risk she has identified is statistically significant.[201]

The information gathered in a cohort study of this kind also allows the calculation of an attributable risk or etiological fraction for the substance in question—that is, the proportion of all new cases of a disease that are due to an exposure to a certain toxic substance.[202] Attributable risk calculation produces a percentage range within which the proportion of disease caused by a toxic substance is explained by chance only five out of one hundred times, provided that the attributable risk calculation is statistically significant.[203] Of course, exposure to more than one hazardous substance complicates such calculations because such exposures may be additive (risk from one exposure adds to another) or multiplicative (risk from one exposure multiplies the other).[204] Yet, even in complex situations such as the multiplicative relationship between asbestos and cigarette smoke exposures and lung cancer, researchers could still calculate an attributable risk.

Epidemiological planning and analysis increases the statistical confidence that a certain exposure, for instance, to asbestos caused an increase in, for instance, the risk of lung cancer.[205] It does this

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201 *See* R. MONSON, OCCUPATIONAL EPIDEMIOLOGY 67-74 (1980). For a further discussion of confidence intervals, *see infra* notes 210-14 and accompanying text.

202 *See* Enterline, *Attributability in the Face of Uncertainty*, 78 CHEST 377 (Supp. 1980); Miettinen, *Proportion of Disease Caused or Prevented by a Given Exposure, Trait or Intervention*, 99 AM. J. EPIDEMIOLOGY 325 (1974); Walter, *Calculation of Attributable Risks from Epidemiological Data*, 7 INT'L J. EPIDEMIOLOGY 175 (1978).

203 This assumes the standard for significance is .05. *See* Monson, *supra* note 201, at 70-71.

204 *See* Koopman, *Interaction Between Discrete Causes*, 113 AM. J. EPIDEMIOLOGY 716 (1981); Walter & Holford, *Additive, Multiplicative and Other Models for Disease Risks*, 108 AM. J. EPIDEMIOLOGY 341 (1978).

205 Epidemiological planning attempts to eliminate bias from the study design. *See* Sackett, *Bias in Analytic Research*, 32 J. CHRONIC DISEASES 51 (1979). Moreover, epidemiologists try to uncover confounding variables that can obscure the truth regarding an association. *See* Greenland & Neutra, *Control of Confounding in the Assessment of Medical Technology*, 9 INT'L J. EPIDEMIOLOGY 361 (1980).

Several epidemiologists have elaborated a set of operational criteria that gnide

by eliminating the effect of other variables such as smoking, diet, or increased age. Thus, epidemiology selects one condition from a list of several, and demonstrates that it is associated with a certain injury in a manner that could be explained by chance only five percent of the time or less—a probabilistic notion of causation.

B. Levels of Uncertainty

A great deal of uncertainty attends the statement "substance X is a carcinogen." This uncertainty is a subset of the uncertainty attending the statement "hazardous substance X has long term ill health effects." There are several reasons for this uncertainty.

First, no deductive/nomological or mechanistic understanding of cancer biology exists. Although science has begun to penetrate the molecular puzzle of cancer, deductive conclusions concerning carcinogenesis are not yet within its reach. Nor is current research likely to produce immediate breakthroughs.[206] Second, the study of chemical carcinogens relies heavily on statistical models and mathematical formulae. This includes not only the science of epidemiology, but also the statistical tests and risk assessment models used in animal bioassays and, increasingly, in short-term assays.[207] Thus, when stating that a chemical causes cancer, researchers use the term cause in a probabilistic sense.

Research into hazardous substances involves three different levels of uncertainty. An understanding of these levels of uncertainty can help clarify the nature of causation in science. The first and most fundamental level of uncertainty concerns so-called transscientific issues.[208] Although cast in scientific terms, these issues are not practically amenable to scientific resolution. For instance, scientists assume that animal models of carcinogenesis always apply to humans, but limited resources preclude undertaking a set of low-level-exposure animal studies on all suspected carcinogens. However, all but one of the widely-accepted known human carcinogens

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causal attribution. *See, e.g.*, D. KLEINBAUM, L. KUPPER & H. MORGENSTERN, EPIDEMIOLOGIC RESEARCH: PRINCIPLES AND QUANTITATIVE METHODS 32-34 (1982); *see also* Evans, *Causation and Disease: A Chronological Journey*, 108 AM. J. EPIDEMIOLOGY 249 (1978).

206 In the field of toxicology, the most exciting area of research concerns exposure issues. *See* Perera & Poirier, *supra* note 174. Tumor virns and recombinant DNA research promises new insights into the nature of carcinogenesis. *See* Israel, Helman & Miser, *Patterns of Proto-Oncogene Expression: A Novel Approach to the Development of Tumor Markers*, in IMPORTANT ADVANCES IN ONCOLOGY, 1987, at 87-104 (V. DeVita, S. Hellman & S. Rosenberg eds. 1987); Yunis, Frizzera, Oken, Theologides & Arnesen, *Multiple Recurrent Genomic Defects in Follicular Lymphoma: A Possible Model for Cancer*, 316 NEW ENGLAND J. MED. 79 (1987).

207 *See supra* notes 180-205 and accompanying text.

208 *See* McGarrity, *Substantive and Procedural Discretion in Administrative Resolution of Science Policy Questions: Regulating Carcinogens in EPA and OSHA*, 67 GEO. L.J. 729 (1979).

also causes cancer in animals. Most toxicologists therefore accept animal data when better evidence is unavailable. The same is true of the assumption that mutagenesis is a marker for carcinogenesis. Although toxicologists cannot prove this assumption in a mechanistic sense, the degree of correlation is sufficient for them to accept the proposition.[209]

The second level of uncertainty occurs when human epidemiological evidence is preliminary, unconfirmed, not statistically powerful, or generally provides weak evidence for rejecting the proposition being tested. Such evidence presents problems with confidence intervals, which are arbitrarily defined as the range within which the mean of a study parameter lies 95% of the time.[210] Of course one could set confidence intervals of 99% or 99.9%, or any other percentage, but 95% confidence intervals correspond to the usual statistical standard of $p = .05$ for significance.

To elaborate, the notion of a confidence interval derives from the same principles that underlie tests of significance. In a normal distribution, $\Pr(-1.96 \leq (\bar{x} - \mu)/(\sigma/\sqrt{n}) \leq 1.96) = .95$. This means that 95% of the observed values of a random variable, $\bar{x}$, drawn from a normal distribution with a sample mean $\bar{x}$, mean μ, standard deviation σ,[211] and a sample size of n, lie within plus or minus 1.96 standard deviations of the mean. This can be rearranged algebraically as $\Pr(\bar{x} - 1.96\,\sigma/\sqrt{n} \leq \mu \leq \bar{x} + 1.96\,\sigma/\sqrt{n}) = .95$. This means that if one sets out confidence intervals of $\bar{x} \pm 1.96\,\sigma/\sqrt{n}$, then in 95 out of 100 cases, or 95% of the time, the true mean of the sample population will lie within these limits.[212]

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209 Another trans-scientific issue is the correlation of mutagenesis with carcinogenesis. Despite a high degree of correlation between known carcinogens and mutagens, there is no way to prove that carcinogens are mutagens.

An example of the trans-scientific nature of animal studies is the carcinogenic effect of the pesticide chlordane. On the basis of at least 11 studies in laboratory animals regarding chlordane's health effects, the Pesticide Committee of the National Academy of Sciences warned that chlordane posed a carcinogenic risk to humans. *See* NATIONAL RESEARCH COUNCIL, NAT'L ACADEMY OF SCIENCES, AN EVALUATION OF THE CARCINOGENICITY OF CHLORDANE AND HEPTACHLOR (1977). Yet there are not any complete studies of humans exposed to chlordane that reinforce or detract from this animal data. *See* S. EPSTEIN, THE POLITICS OF CANCER 270 (1978). Thus the animal experiments, and the leap of faith concerning animal and human carcinogenesis, are the only evidence available when considering the risks from chlordane. This, then, represents the most profound level of uncertainty encountered by a finder of fact.

210 T. COLTON. *supra* note 190, at 126.

211 The mean of a sample is defined as the summation of all values, divided by the number of values. Standard deviation is the square root of the variance. Calculations of variance are usually based on the summation of the differences between the means and individual measurements. More specifically, variance equals $\Sigma (x - \bar{x})^2/(n-1)$, where $\bar{x}$ is the sample mean. *Id.* at 31-33. As a result, the standard deviation grows larger when there is a great deal of scatter, or divergence, in values of the various test measurements.

212 See *id.* at 126 for a discussion of confidence intervals.

Confidence intervals are wide when n, the sample size, is small or when σ, the standard deviation within the sample, is large. Thus, confidence intervals widen when few people participate in a study or when a great deal of variance occurs in the sample. The 95% confidence interval reflects the statistical assumption that .05 is the proper level for significance. Inherently conservative, statistics requires a less-than-one-in-twenty chance that random variation is responsible for the estimated value of the parameter.

Colton provides an example.[213] A group of one hundred cancer patients are treated with a drug. Previous experience with such cancer patients has shown that the mean survival is 38.3 months and the standard deviation is 43.3 months. The drug tested group has a mean survival of 46.9 months. The confidence interval for the related group is $46.9\ (1.96)(43.3/\sqrt{100} = 46.9 \pm 8.5$ months. If the sample had consisted of 10,000 patients, then the confidence intervals would be 46.9 + .85 months. With either sample, however, the mean of the untreated population lies outside the confidence interval, indicating significance at the .05 level.

Basing the confidence interval on a level of significance of .51 would decrease the size of confidence intervals, because the critical fraction would be .66 rather than 1.96. However, the researcher could then only state that the true mean of the population lies within the confidence intervals half the time. Statistical inference would find this amount of uncertainty with regard to the location of the true mean of the population unacceptable.

In summary, developing a proposition to be tested, also called formulating a null hypothesis, is essential when planning a study or experiment that involves statistical analysis. The null hypothesis usually consists of the opposite of the causal connection the researcher is attempting to prove. The researcher disproves the null hypothesis by developing statistically significant evidence that the null hypothesis is incorrect. Thus, the null hypothesis usually includes an assumption concerning the distribution of the data, often that the data are from a normal distribution. Therefore researchers expect the data to cluster about the mean in a characteristic manner. When using relatively few data points or individual measurements, the variance and standard deviation enlarge and widen the confidence intervals. In such a situation, although the evidence might lead to a statistically significant conclusion and a rejection of the null hypothesis, the wide confidence intervals detract from the study's conclusions.[214]

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213 *Id.* at 127.

214 *Id.* at 125-27. For instance, a preliminary study may calculate that a toxic substance increased a risk ratio for a given disease, but the confidence intervals might be

Wide confidence intervals indicate that the available data, although supporting the scientist's hypothesis, are less than completely reliable. Wide confidence intervals occur when the research included a relatively small number of observations, such as when the number of study subjects is small; they also occur when the method for making the measurements is imprecise, such as when exposure information is limited.

The third level of uncertainty attends any individual attribution based on group characteristics. This aspect of uncertainty is most salient when the epidemiological evidence is well controlled, based on large cohorts, and passes the various tests for bias. For example, an extraordinarily well done epidemiological study of a rare tumor and a clear exposure to a carcinogen for a large cohort of individuals might demonstrate that 45% of the lung cancers occurring in this group result from exposure to the carcinogen. As to each individual member of the cohort, however, it is uncertain whether the carcinogen or another factor caused the individual's lung cancer. Epidemiology makes statements only about the group, not the individual. Individual attribution involves uncertainty, because the epidemiological data produce only summary statistics applicable to the sample or to the population the sample represents.

C. Case Studies

All three levels of uncertainty present distinct problems for lawyers and courts accustomed to causation expressed in terms of causal chains. The court's ability to accommodate claims based on hazardous-substance-induced injury may vary according to the level of uncertainty that predominates. A review of several case studies of hazardous-substance injuries and potential claims may help indicate the kind of cases, if any, that a common law court could accommodate.

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quite broad, and the results proportionately less trustworthy. Similarly, the mathematical formulae used for dose/response curve extrapolations become imprecise at low doses and result in conclusions attended by wide confidence intervals. *See supra* note 194. Yet another example of overly broad confidence intervals occurs when exposures to potentially toxic substances or carcinogens are difficult to specify for a given population. Consider, for instance, a study of the relationship between chemical exposure and occurrence of the relatively rare brain cancers. Over the past decade, several studies have reported high rates of occurrence of gliomas in chemical workers. These studies have been hampered by the researchers' inability to assemble a large cohort of workers with precise exposure data and thus narrow confidence intervals in order to draw a clear cut association. *See* Olin & Ahlbom, *The Cancer Mortality Among Swedish Chemists Graduated During Three Decades*, 22 ENVTL. RES. 154 (1980); Thomas, Decoufle & Moure-Eraso, *Mortality Among Workers Employed in Petroleum Refining and Petrochemical Plants*, 22 J. OCCUPATIONAL MED. 97 (1980).

1. *Asbestos in the Workplace*

In 1972, the United States consumed 808,554 short tons of asbestos[215] for use in a variety of industrial cloths, insulation, cement, and composition materials. By the mid-1930s, both American and British medical journals were reporting the ill health effects of asbestos.[216] By 1950, research had established an association between asbestos exposure and lung cancer.[217]

In 1964, Selikoff and others reported data on the mortality experience of a cohort of asbestos insulation workers.[218] In 1979, they updated the mortality experience of these workers.[219] The insulators experienced a fairly consistent exposure to asbestos, estimated at dust levels of four to twelve fibers per milliliter of air.[220] Of the original 632 men registered as members of two locals in 1943, 478 were dead by December, 1976.[221] Ninety-three of these workers died of lung cancer.[222] Using age-specific United States death rates, only thirteen deaths from lung cancer would have been expected.[223] The researchers noted thirty-eight cases of mesothelioma, with less than one expected.[224] Total cancer deaths were almost four times more than expected.[225]

The overwhelming risk ratios, the ability to track an exposure given membership in a union, and the completeness of follow-up all contributed to the study's firm conclusion that asbestos causes cancer. The study has no problems with the first level of uncertainty: no animal studies or short-term tests were involved. Nor are there

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215 1 BUREAU OF MINES, U.S. DEP'T OF THE INTERIOR, MINERALS YEARBOOK: METALS, MINERALS, AND FUELS, 169 (1972).

216 *See* I. SELIKOFF & D. LEE, ASBESTOS AND DISEASE 23 (1979).

217 Much of this history, and indeed the duplicity of the asbestos industry has been documented in several asbestos products liability suits. *See* S. EPSTEIN, *supra* note 209, at 89-96.

218 *See* Selikoff, Churg & Hammond, *Asbestos Exposure and Neoplasia*, 188 J.A.M.A. 142 (1964).

219 *See* Selikoff, Hammond & Seidman, *Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976*, in HEALTH HAZARDS OF ASBESTOS EXPOSURE 91 (Annals N.Y. Acad. Sci., Vol. 330, I. Selikoff & E. Hammond eds. 1979) [hereinafter *Mortality Experience*]; *see also* INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, WORLD HEALTH ORG., BIOLOGICAL EFFECTS OF MINERAL FIBRES (J. Wagner & W. Davis eds. 1980); McDonald & McDonald, *Malignant Mesothelioma in North America*, 46 CANCER 1650 (1980); Hammond, Selikoff & Seidman, *Asbestos Exposure, Cigarette Smoking and Death Rates*, in HEALTH HAZARDS OF ASBESTOS EXPOSURE, *supra*, at 473 [hereinafter *Asbestos Exposure*]; Becklake, *Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice*, 114 AM. REV. RESPIRATORY DISEASE 187 (1976).

220 Selikoff, Hammond & Seidman, *Mortality Experience*, *supra* note 219, at 92.

221 *Id.* at 93.

222 *Id.* at 95.

223 *Id.*

224 *Id.*

225 *Id.* at 94.

problems with confidence intervals: the exposure and disease are well established in large numbers of people. The third level of uncertainty does, however, play a role: researchers cannot know whether a particular lung cancer death is from asbestos or from some other cause expected in any cohort of individuals. The tendency of asbestos workers to smoke and the multiplicative interaction of smoking and asbestos exacerbates this problem.[226] Thus, the individual attribution of asbestos-caused lung cancer is difficult, although the classification of asbestos as a carcinogen is unproblematic.

Asbestos producers have turned this problem of individual attribution of causation based on group characteristics into an effective weapon in litigation. The producers argue that there is no way to know if their product or the plaintiffs' smoking caused a lung cancer.[227] Plaintiffs could avoid this problem by presenting a case on behalf of the entire exposed group. Each member of the class would accept proportionate compensation based on the attributable fraction derived from the epidemiological study.[228] Unfortunately, such class action litigation often proves unwieldy and is chronically hampered by problems with incompleteness of the class.[229] Thus, a hazardous substance that presents only level three uncertainty can still represent a formidable challenge to common law procedure.

In addition to such procedural problems, claims involving level three uncertainty challenge the causal/moral nexus between actor and injury that Epstein argues is at the heart of the tort theory of causation.[230] In effect, resolution of level three uncertainty requires displacement of the essential nexus from producer/injured individual to producer/injured group. Although conceptually possible, this displacement would dilute the moral ascription aspects of tort

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226 *See* Hammond, Selikoff & Seidman, *Asbestos Exposure, supra* note 219, at 481-85, 488; Meurman, Kiviluoto & Hakama, *Combined Effect of Asbestos Exposure and Tobacco Smoking on Finnish Anthophyllite Miners and Millers*, in HEALTH HAZARDS OF ASBESTOS EXPOSURE, *supra* note 219, at 491, 495.

227 *See, e.g.*, Martin v. Owens-Corning Fiberglas Corp., 515 Pa. 377, 528 A.2d 947 (1987).

228 Class action or collateral estoppel doctrines are the best available methods for assembling the exposed group. *See infra* note 266.

229 *See infra* notes 267-69.

230 *See supra* notes 94-96 and accompanying text. Rosenberg has argued that corrective justice can accommodate probabilistic evidence of causation. *See* Rosenberg, *supra* note 3, at 877. I have argued that liberalism depends on notions of moral causation such as Epstein proposes, and that this sort of causation is conceptually dependent on mechanistic causal chains. *See supra* notes 71-107 and accompanying text. Although meaningful in the context of personal injury, moral causation lacks this meaning in the context of corporate liability for disease in a population of workers. This difference is even more salient in a case of air pollution than it is in asbestos litigation. *See infra* notes 249-55 and accompanying text.

litigation.[231]

2. *Chlorinated Organic Compounds in Drinking Water*

Halogenation of organic compounds produces a number of useful solvents and degreasers. Unfortunately, many of these compounds are toxic. For instance, chloroform (CCl_4) produces neurotoxicity as well as epithelial damage in humans. Moreover, it produces kidney and liver tumors in rats and mice. Trichloroethylene ($Cl_2 = CH-Cl$) and tetrachloroethylene ($CCl_2 = CCl_2$) are also neurotoxic. In addition, both compounds produce hepatocellular carcinoma in mice, but not in rats.[232] A cohort mortality (epidemiological) study of workers exposed to trichlorethylene revealed no excess cancer mortality, although the sample size was rather small.[233]

In May, 1979, the EPA found trichloroethylene, tetrachloroethylene, and chloroform in two out of eight municipal wells in Woburn, Massachusetts.[234] Subsequently, the Massachusetts State Department of Health found a significantly elevated rate of childhood leukemia in Woburn, with twelve cases diagnosed between 1969 and 1979 when only 5.3 were expected. A case control epidemiological study failed to identify any etiologic agent for the leukemias.[235]

In 1982, a coalition of concerned citizens, in concert with statisticians from the Harvard School of Public Health undertook a telephone survey and a review of hydrogeological data.[236] They found

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231 If tort theories provide no acceptable remedy, we can ill afford to adopt a do-nothing approach. A recent study estimated that 8,000-9,000 cancer deaths per year are attributable to asbestos alone. Nicholson, Perkel & Selikoff, *Occupational Exposure to Asbestos: Population of Risk and Projected Mortality—1980-2030*, 3 AM. J. INDUS. MED. 259 (1982). If tort law is not the answer, then society must try another approach. *See infra* notes 276-92 and accompanying text.

232 Craft, *Solvents and Related Compounds*, in ENVTL. & OCCUPATIONAL MED. 511, 520 (W. Rom ed. 1983); INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, WORLD HEALTH ORG., 20 MONOGRAPHS ON THE EVALUATION OF THE CARCINOGENIC RISK OF CHEMICALS TO HUMANS: SOME HALOGENATED HYDROCARBONS (1979); Weisburger, *Carcinogenicity Studies on Halogenated Hydrocarbons*, 21 ENVTL. HEALTH PERSP., Dec. 1977, at 7, 9 (oral administration of halogenated hydrocarbons produced hepatocellular carcinomas in mice, but had little or no effect on rats).

233 *See* Axelson, Andersson, Hogstedt, Holmberg, Molina & deVerdier, *A Cohort Study on Trichloroethylene Exposure and Cancer Mortality*, 20 J. OCCUPATIONAL MED. 194 (1978).

234 MASSACHUSETTS DEP'T OF ENVTL. QUALITY AND ENG'G, SPECIAL ANALYSIS: WOBURN, WATER SAMPLES TAKEN FROM WELLS G AND H BY MCCALL (May 14, 1979).

235 *See* MASSACHUSETTS DEP'T OF PUB. HEALTH, CANCER INCIDENCE AND ENVIRONMENTAL HAZARDS 1960-1978 (1981).

236 *See* Lagakos, Wessen & Zelen, *An Analysis of Contaminated Well Water and Health Effects in Woburn, Massachusetts*, 81 J. AM. STATISTICAL A. 583 (1986) (presenting and discussing results from survey).

positive statistical correlations between access to water from the contaminated wells and incidence rates of childhood leukemia, perinatal deaths, and two of five categories of congenital anomalies. Researchers estimated, however, that the well water explained only four to six of the ten to twelve excess cases of leukemia. All the conclusions of this study were qualified by the difficulties with precise hydrogeological (exposure) data and the possibilities for bias within the sophisticated study design.[237]

Is the polluter who dumped three chlorinated organic compounds responsible for four to six cases of leukemia? The answer is yes only if we recognize uncertainty on all three levels. First, we must accept that the positive animal bioassays for carcinogenicity apply to humans—a trans-scientific matter.[238] Second, we must realize that the inexact exposure data and possible bias within the study design create confidence interval problems. In other words, the epidemiology is inexact. Third, it is impossible to say which four out of twenty leukemias the chemical exposure caused. Thus, data on disease from an incident such as the one at Woburn involves all three levels of uncertainty, even though the epidemiological study itself is quite sophisticated.

Most salient in the Woburn example is level two uncertainty. The report used very sophisticated and somewhat controversial methods. The statisticians and epidemiologists who reviewed the study did not unanimously approve of these methods or the conclusions reached.[239] Despite the high level two uncertainty, the Woburn report represented a state of the art analysis of environmental toxins. Thus the question arises, will courts ever be able to deal with level two uncertainty of this degree, or will courts inevitably fail to analyze probabilistic evidence and instead formulate a causal chain explanation?[240]

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237 The report by Lagakos, Wessen & Zelen, *supra* note 236, was published with a series of commentary articles on the report's conclusions. Many comments dealt with highly technical aspects of the statistical methods used by the authors. *See* Swan & Robins, *Comment*, 81 J. AM. STATISTICAL A. 604 (1986). Others suggested that the study was tainted by bias, both with regard to recall, and with regard to the participation in the study of subsequent plaintiffs in a suit against several manufacturers located in the area. *See* MacMahon, *Comment*, 81 J. AM. STATISTICAL A. 597 (1986). For a discussion of bias, see *supra* note 205 and accompanying text, and Brennan & Carter, *supra* note 56, at 72 n.158. Still others noted the generic problems associated with doing cluster analysis around toxic waste sites. *See* Rogan, *Comment*, 81 J. AM. STATISTICAL A. 602 (1986). These comments and the report they criticize form a comprehensive review of the difficulties encountered when working with environmental epidemiology.

238 Moreover, the Woburn report, by Lagakos, Wessen & Zelen, *supra* note 236, involved cancers that the compounds in question have never caused in laboratory animals. *See* MacMahon, *supra* note 237, at 598.

239 *See* Swan & Robins, *supra* note 237; MacMahon, *supra* note 237.

240 The Woburn well contamination also demonstrates the difficulty of estimating

3. *Spermicides*

Spermicides are chemicals used in conjunction with diaphragms as a means of birth control. Because the effectiveness of these methods is low, each year 300,000-600,000 women become pregnant while using spermicides either alone or with diaphragms.[241] Spermicides are absorbed into the bloodstream in test animals.[242] In 1981, a retrospective epidemiological study raised the possibility of an association between vaginal spermicides and birth defects.[243]

Since the publication of the original study in 1981, no less than six studies have refuted the alleged association between spermicides and birth defects.[244] Several commentaries have summarized the existing literature and concluded that no association exists.[245] Moreover, one of the authors of the original study has reanalyzed the original data and found no association.[246]

The association between vaginal spermicides and birth defects presents even greater level two uncertainty than does the Woburn study. As a result of this great uncertainty, the majority of statistical

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the threat to public health presented by contamination of ground water with substances such as chlorinated organic compounds. The two wells were shut down only after these substances were detected in Woburn well water in 1979. It is difficult to predict how many other contaminated municipal wells exist. Moreover, the contamination at Woburn was at least partly caused by toxic chemicals buried years before. These chemicals were uncovered during excavation for a construction site, and they prompted further investigation as well as the extraordinary community cooperation that made the final epidemiological study possible. Thousands of such chemical burial grounds may exist in the United States—no one knows for sure. *See Developments in the Law—Toxic Waste Litigation*, 99 HARV. L. REV. 1460, 1462 (1986) [hereinafter *Developments*].

241 Cordero & Layde, *Vaginal Spermicides, Chromosomal Abnormalities and Limb Reduction Defects*, 15 FAM. PLAN. PERSP. 16 (1983).

242 *See* Chvapil, Eskelson, Stiffel, Owen & Droegemueller, *Studies on Nonoxynol-9—Intravaginal Absorption, Distribution, Metabolism and Excretion in Rats and Rabbits*, 22 CONTRACEPTION 325 (1980).

243 Jick, Walker, Rothman, Hunter, Holmes, Watkins, D'Ewart, Danford & Madsen, *Vaginal Spermicides and Congenital Disorders*, 245 J. A.M.A. 1329 (1981). The report was consciously hedged with warnings that the conclusions were tentative. *Id.* at 1332. Another report identified an association between spermicides and Down's syndrome. *See* Rothman, *Spermicide Use and Down's Syndrome*, 72 AM. J. PUB. HEALTH 399 (1982).

244 Cordero & Layde, *supra* note 241; Bracken & Vita, *Frequency of Non-hormonal Contraception Around Conception and Association with Congenital Malformations in Offspring*, 117 AM. J. EPIDEMIOLOGY 281 (1983); Mills, Harley, Reed & Berendes, *Are Spermaticides Teratogenic?*, 248 J. A.M.A. 2148 (1982); Mills, Reed, Nugent, Harley & Berendes, *Are There Adverse Effects of Periconceptional Spermicide Use?*, 43 FERTILITY & STERILITY 442 (1985); Polednak, Janerich & Glebatis, *Birth Weight and Birth Defects in Relation to Maternal Spermicide Use*, 26 TERATOLOGY 27 (1982); Schapiro, Slone, Heinonen, Kaufman, Rosenberg, Mitchell & Helmrich, *Birth Defects and Vaginal Spermicides*, 247 J. A.M.A. 2381 (1982).

245 Bracken, *Spermicidal Contraceptives and Poor Reproductive Outcomes: The Epidemiological Evidence Against an Association*, 151 AM. J. OBSTETRICS GYNECOLOGY 552 (1985); Mills & Alexander, *Teratogens and Litogens*, 315 NEW ENGLAND J. MED. 1234 (1986).

246 *See* Watkins, *Vaginal Spermicides and Congenital Disorders: The Validity of a Study* (Letter to the Editor), 256 J. A.M.A. 3095 (1986).

and epidemiological experts reject the association. Yet, the original 1981 study stands as the primary evidence of causation in at least one court decision finding that spermicides cause birth defects.[247] Such a decision highlights the potential problems that courts encounter when faced with level two uncertainty and at least in part explains their tendency to substitute fictional causal chains when confused by the probabilistic evidence of causation.[248]

4. *Air Pollution Carcinogens*

A number of carcinogens escape into the ambient atmosphere as byproducts of industrial activity. The major carcinogens in air pollution are arsenic, asbestos, radioactive materials, benzene, and polycyclic hydrocarbons such as benzo[a]pyrene.[249] When attached to fine particulate matter, such hydrocarbons are potent carcinogens in animal models. Heavily polluted areas have much higher levels of benzo[a]pyrene in the air.[250] Thus air pollution probably causes cancer, especially respiratory tumors, in humans as well.

The link between air pollution and cancer, however, is very difficult to demonstrate, let alone quantify. Air pollutants diffuse rapidly in the environment after escape from industrial sources.[251] Thus, exposure to air pollution is diffuse, variable, and usually quite low level. As noted above, the difficulty in quantifying exposure widened the confidence intervals and diluted the certainty of the

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247 Wells v. Ortho Pharmaceutical Corp., 788 F.2d 741, 744 (11th Cir.), *cert. denied*, 107 S. Ct. 437 (1986).

248 The similarity of the uncertainty and the attendant causation problems that arise in the Woburn well contamination case and the spermicide situation demonstrate why I analyze these quite different legal situations together. Well contamination cases, such as the Woburn incident, spawn law suits that sound primarily in nuisance. Spermicide cases are products liability cases. The causation questions they raise are similar because both involve hazardous products.

Also, the spermicide question raises level three uncertainty: if spermicides have teratogenic effects, they still probably only cause a certain proportion of the birth defects in all users of spermicides. Thus, the court would have to calculate an attributable fraction and all exposed would have to receive proportionate compensation. *See supra* notes 202-03.

249 *See* OFFICE OF AIR QUALITY STANDARDS, U.S. ENVTL. PROTECTION AGENCY, REVIEW AND EVALUATION OF THE EVIDENCE FOR CANCER ASSOCIATED WITH AIR POLLUTION, at B-I app. (1984) [hereinafter EPA REPORT]; L. LAVE & A. SESKIN, AIR POLLUTION AND HUMAN HEALTH 290, 339 (1977).

250 The animal data on the carcinogenicity of benzo[a]pyrene is quite venerable. *See* Saffiotti, Cefis, Kolb & Shubik, *Experimental Studies of the Conditions of Exposure to Carcinogens for Lung Cancer Induction*, 15 J. AM. AIR POLLUTION CONTROL A. 23 (1965). The link to urbanization and increased death rates is even older. *See* Stacks & Campbell, *Lung Cancer Death Rates Among Nonsmokers and Pipe and Cigarette Smokers: An Evaluation in Relation to Air Pollution by Benzo-a-Pyrene and Other Substances*, 2 BRIT. MED. J. 923 (1955).

251 *See* R. BRODZINSKY & H. SINGH, VOLATILE ORGANIC CHEMICALS IN THE ATMOSPHERE: AN ASSESSMENT OF AVAILABLE DATA (1983); *see also* EPA REPORT, *supra* note 249, at III-32.

Woburn study.[252] This problem is several orders of magnitude larger with air pollution. In Woburn, the cross-sectional study involved a portion of a small town. In any study of air pollution, the cross-section may involve an entire metropolitan area or several metropolitan areas.[253] Thus the epidemiology is less precise.

The completed epidemiological studies on lung cancer and air pollution generally compare the rates of lung cancers in various geographic areas. Urban areas almost uniformly have higher rates of lung cancer, as well as higher levels of air pollution.[254] However, an attempt to control for the confounding effects of social class and cigarette smoking complicates studies of the relationship between air pollution and lung cancer. Nonetheless, one particularly sophisticated study estimated that ten percent of lung cancers in large cities could result from air pollution.[255]

Imagine that a group of plaintiffs suffering from lung cancer in Cleveland brought suit against the major industrial air polluters in that metropolitan area. All three levels of uncertainty would undoubtedly trouble the court hearing the case. Although uncertainty exists at the first level (benzo[a]pyrene is an animal carcinogen and therefore we assume it is a human carcinogen), extreme uncertainty exists at the second level (the very diffuse exposure-effect linkage causes huge confidence intervals and thus low confidence in the findings of a study that demonstrates the linkage). Moreover, uncertainty at the third level (individual attribution of cause) is profound for air pollution, especially when compared to the uncertainty surrounding the effect of chlorinated organic compounds in the Woburn water supply[256] or Selikoff's study of asbestos insulation

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252 *See supra* notes 238-39 and accompanying text.

253 *See* L. LAVE & A. SESKIN, *supra* note 249. Schlesselman has discussed the problems with a cohort study in air pollution. Schlesselman, *Sample Size Requirements in Cohort and Case Control Studies of a Disease,* 99 AM. J. EPIDEMIOLOGY 381 (1974). Case control studies on this subject are especially hard to complete. *See* Siemiatycki, Day, Fabry & Cooper, *Discovering Carcinogens in the Occupational Environment: A Novel Epidemiological Approach,* 66 J. NAT'L CANCER INST. 217 (1981); *see also* Haenszel, Loveland & Sirken, *Lung Cancer Mortality as Related to Residence and Smoking Histories,* 28 J. NAT'L CANCER INST. 947 (1962).

254 *See* L. LAVE & A. SESKIN, *supra* note 249; *see also* EPA REPORT, *supra* note 249, at app. A.

255 *See* Pike, Gordon, Henderson, Menck & Soottoo, *Air Pollution,* in PERSONS AT HIGH RISK OF CANCER: AN APPROACH TO CANCER ETIOLOGY AND CONTROL 225, 225-35 (J. Fraumeni ed. 1975); *cf.* Winkelstein, *The Relationship of Air Pollution and Economic Status to Total Morality and Selected Respiratory System Morality in Men,* 14 ARCHIVES ENVTL. HEALTH 162 (1967) (showing positive association between air pollution levels and total morality in older white men when economic status controlled). Similar studies show higher lung cancer rates in counties where smelters are located. *E.g.*, Blot & Fraumeni, *Arsenical Air Pollution and Lung Cancer,* 1975 LANCET 142.

256 *See* Lagakos, Wessen & Zelen, *supra* note 236.

workers.[257] Pollutants like benzo[a]pyrene would cause at most only one-tenth or one-twentieth of all lung cancers. Deciding which cases of lung cancers air pollution caused would thus be very difficult.

Certainly, plaintiffs in a class action based on injury from air pollution would frame their suit very differently than plaintiffs arguing injury from spermicides. Nonetheless, the problems regarding causation are similar because of the generic uncertainty involved in the probabilistic evidence. Hazardous substance litigation may proceed under a number of different legal theories; but they pose similar causation problems. Moreover, any solution to the courts' problems with causation must be broadly applicable. Having explained those problems and their relationship to the levels of uncertainty, I now turn to some solutions.

IV
SOLVING CAUSATION ISSUES IN HAZARDOUS SUBSTANCE LITIGATION

A. The Limits of Tort Law

Given the conceptual difficulties that courts have with causation and uncertainty in probabilistic evidence, what role should tort law play in the resolution of claims based on hazardous substance induced injury? Does tort law need to change to accommodate the special legal problems posed by hazardous substance litigation?[258] Or should we abandon tort law for a comprehensive regulatory approach to compensation and cost internalization?[259]

I believe that the answers to these questions are best found by adhering to the architecture of uncertainty outlined in the previous section.[260] Answers regarding level one uncertainty, that uncertainty that attends the trans-scientific questions about hazardous substances, require policy decisions.[261] Scientific experiments cannot resolve such questions. Instead, assumptions are made that em-

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257 *See* I. SELIKOFF & D. LEE, *supra* note 216.

258 The primary advocate of changing the tort law system is Professor Rosenberg. *See* Rosenberg, *supra* note 3, at 924-29 (proposing tort law shift to proportionality rule of causation and proposing extensive use of class action suits); *see also* Robinson, *supra* note 2, at 798 (arguing for further exploration of "risk" based, instead of "injury" based, tort liability).

259 *See* SUPERFUND STUDY, *supra* note 6; Pierce, *supra* note 1, at 1320-30.

260 This focuses the scientific causation issues and avoids the problems with multiple causation that also hamper hazardous substance litigation.

261 *See supra* notes 208-09 and accompanying text. Level one uncertainty involves trans-scientific issues—issues stated in scientific terms, yet not capable of scientific resolution. Examples of level one uncertainty include the applicability of animal models of carcinogenesis to human beings as well as the role of mutagencity tests as carcinogen assays. *Id.*

body policy: animal carcinogens are accepted as human carcinogens and mutagenic chemical are perceived as carcinogens. These assumptions, although stated in scientific terms, involve explicit policy choices.

Can tort law cope with level one uncertainty? A common law judge can decide policy as well as factual questions. Nonetheless, the policy questions that arise with level one uncertainty are so broad that poor resolution occurs through individual tort suits. The benefits gained by decisionmaking at the level of common law courts are outweighed by the anarchy that would result from reliance on an individual judge to decide whether, for example, animal carcinogens should be considered human carcinogens. Solutions to trans-scientific issues are only as valuable as they are consistent. Repetitive litigation of these issues in common law courts cannot provide this consistency. Decisions of this scope are best made at the macro level of regulatory agencies, not at the micro level of tort litigation.

What about level two uncertainty, characterized as confidence interval problems? Any attribution of cause based on statistical or epidemiological evidence involves some inherent uncertainty, best expressed as the range of values within which the true value of a risk of injury lies ninety-five percent of the time. This kind of uncertain causal attribution is the best that the probabilistic evidence of hazardous-substance injury can produce.

Deeply troubled by this kind of uncertain causal attribution, courts predominately adopt a corpuscularian attitude toward scientific evidence, expecting a clear causal chain analysis from science. Tort law's emphasis on the nexus of the liable individual with the injury he or she causes reinforces this expectation.[262] A tort system underpinned with moral theory and liberal values cannot easily accommodate probabilistic evidence.[263] Indeed, a review of cases dealing with hazardous substances demonstrates the conceptual difficulties that courts encounter with such evidence.[264]

Level three uncertainty, that uncertainty attending individual attribution of cause when the causal connection is based on group characteristics, is also problematic for common law courts. This kind of uncertainty also offends tort law's emphasis on the individual because the injurer/injured nexus is always uncertain.[265] Class

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262 *See supra* notes 77-81 and accompanying text.

263 Moreover, one would not want this type of evidence accommodated, as it would dilute the sense of morality incorporated into a finding of liability. *See supra* note 103.

264 *See supra* notes 112-60 and accompanying text. This author is very pessimistic about the successful resolution of hazardous-substance tort litigation.

265 *See supra* notes 77-99 and accompanying text.

action or collateral estoppel doctrines, however, bring the entire group of injured plaintiffs before the court.[266] Once presented with the entire group, a common law court might be persuaded that justice demands proportionate compensation for their injuries.[267]

Compiling these groups would not be a trivial task. Estimating the geography of exposure can be difficult, more so for a suit based on cancer from air pollution than for a suit based on cancer from a polluted drinking water source.[268] Any legal solution providing compensation for victims of hazardous substance injury must address these exposure problems. Class action tort litigation may be an especially unwieldy method for compiling the group of victims from any one substance.[269]

Because tort law appears unable to deal with level one and level two uncertainty, and deals rather inadequately with level three uncertainty, it makes sense to turn to a regulatory apparatus for resolution of the scientific questions that arise in litigation of hazardous-substance injury. Tort law possesses certain theoretical advantages over regulation;[270] however, these advantages in no way offset the

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266 For a thorough discussion of class action doctrine, see Rosenberg, *supra* note 3, at 910. Collateral estoppel also might work in hazardous-substance litigation. *See* Note, *Collateral Estoppel In Asbestos Litigation,* 14 ENVTL. L. 197 (1983) (authored by Troyen Brennan); Baldwin, *Asbestos Litigation and Collateral Estoppel,* 17 FORUM 772 (1982).

267 Rosenberg is fairly persuasive on this point. *See* Rosenberg, *supra* note 3, at 908. *See also* Note, *Class Certification in Mass Accident Cases Under Rule 23(b)(1),* 96 HARV. L. REV. 1143 (1983) (judicial interpretation of Rule 23(b)(1) preventing its use in mass accident cases conflicts with rule's underlying purpose—efficient handling of numerous related claims).

268 *See supra* Section III(C)(4). Hydrogeology is a very different endeavor. *See supra* Section III(C)(2). Moreover, consider the problems with bringing together all the litigants suffering from birth defects after exposure to spermicides. *See supra* Section III(C)(3). Any exposure problem leads to larger standard deviations and thus to wider confidence intervals.

269 Indeed, courts hearing class actions have not generally accepted products liability claims. *See In re* Federal Skywalk Cases, 680 F.2d 1175 (8th Cir.) (district court's mandatory class-certification order vacated because appeals court found that it violated Anti-Injunction Act, 28 U.S.C. § 2283 (1982)), *cert. denied,* 459 U.S. 988 (1982); *In re* Northern Dist. of Cal. Dalkon Shield I.U.D. Prods. Liab. Litigation, 693 F.2d 847 (9th Cir. 1982) (due to complexity of issues peculiar to individual claims, class certification denied to plaintiffs claiming injuries from I.U.D.'s), *cert. denied,* 459 U.S. 1171 (1983). Products liability litigants probably have the smallest problems with proof of exposure of any hazardous-substance litigants. If courts are wary of class actions in products liability cases, they will most likely be quite hostile to class certification in other types of bazardous-substance litigation.

270 Rosenberg notes: 1) judicial independence prohibits the sort of political capture that can occur with administrative agencies; 2) courts do not need full time expert staffs; 3) limitations on agency resources may preclude response to the many kinds of hazardous-substance injury; 4) tort damage actions are more discriminating than command and control regulation; and 5) there is value in allowing victims to initiate claims. Rosenberg, *supra* note 3, at 927. A properly designed administrative approach to hazardous-substance injury can preserve all of these virtues. *See infra* notes 272-91 and accompanying text.

fundamental problems that arise when common law courts are confronted with probabilistic evidence of causation.[271]

B. A Regulatory Approach to Hazardous-Substance Injury

The problems with scientific causation in tort law demand an administrative structure that provides scientific information on hazardous-substance injury. An administrative apparatus would free the system from the strong tort law emphasis on the individual and causal chain analysis. A regulatory approach that utilizes scientific experts could accommodate probabilistic evidence of causation. This in turn would produce the predictable outcomes that serve the interests of both plaintiffs and defendants.[272]

The structure of this administrative system is largely dictated by

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271 Before turning from tort law altogether, I consider the possibility of designing hybrid institutions that could assist common law courts with the difficulties presented by hazardous-substance litigation. As noted, common law courts need help with the complex probabilistic evidence that links hazardous substances with injuries. In addition, courts need some help assembling the group of litigants, in order to award proportionate compensation.

One way to accomplish these tasks would involve a screening panel for hazardous-substance claims. Many states have employed screening panels to help deal with medical malpractice litigation. *See generally* Sohn, *An Examination of Alternatives to Suit in Doctor-Patient Disputes*, 48 ALB. L. REV. 669 (1984). In the hazardous substance area, such panels could consist of consumers, jurists, and scientists, who would review the available evidence and reach some consensus on the probability of causation. Moreover, this panel could oversee the organization of the exposed group, in order to present to the court the proper group of plaintiffs.

Although attractive in the abstract, in reality constitutional and operational problems have plagued screening panels. Some have argued that screening panels represent a loss of rights for plaintiffs, and thus have subjected the legislation to constitutional challenges. *E.g.*, Note, *Medical Malpractice Mediation Panels: A Constitutional Analysis*, 46 FORDHAM L. REV. 322, 338 (1977); Learner, *Restrictive Medical Malpractice Compensation Schemes: A Constitutional "Quid Pro Quo" Analysis to Safeguard Individual Liberties*, 18 HARV. J. ON LEGIS. 143 (1981). Even when constitutionally permissible, such panels have largely failed to expedite the litigation of malpractice claims. Indeed, in many cases, the panels actually slowed litigation, without any real effect on the number or kind of suits brought. Note, *A Practical Assessment of Arizona's Medical Malpractice Screening System*, 1984 ARIZ. ST. L.J. 335, 346. At least with regard to medical malpractice, screening panels do not appear to have helped solve any of the problems posed by tort litigation.

Hybrid solutions for hazardous-substance compensation and deterrence have been offered as well. *See* Note, *Tort Actions for Cancer: Deterrence, Compensation, and Environmental Carcinogenesis*, 90 YALE L.J. 840 (1981). Many proposals before Congress include various hybrid solutions to the hazardous-substance problem. *See generally* Kircher, *Federal Product Legislation and Toxic Torts: The Defense Perspective*, 28 VILL. L. REV. 1116 (1982-83); Schwartz & Means, *The Need for Federal Product Liability and Toxic Tort Legislation: A Current Assessment*, 28 VILL. L. REV. 1088 (1982-83). None have won overwhelming support. In the next section, I present a different solution to the problems associated with probabilistic causation in tort law.

272 Absent a legal structure that deals appropriately with causation, the United States may eventually have to embrace alternative compensation schemes for injuries from hazardous substances. *See Developments*, *supra* note 240, at 1634; *see also* Kinsley, *Fate and Lawsuits*, NEW REPUBLIC, June 14, 1980, at 23. A silver lining to the hazardous-sub-

the problems with causation and uncertainty outlined in this Article.[273] A "Hazardous Substance Authority" should consist of scientific, enforcement, and compensation panels. A federal system is most appropriate. A federal agency would efficiently deal with the generic issues that arise in hazardous-substance-caused injury.[274] In addition, a federal agency would provide the kind of predictability in its determinations that industry requires.[275] A federal agency for

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stance cloud may be that industry will exert pressure on the government in support of national health insurance.

Under an administrative approach, Congress could order regulators to research hazardous substances' detrimental effects on health. There would be unavoidable lag time between the post-exposure onset of ill effects and the regulatory agency's ability to detect and verify causation. With a federal regulatory system, victims would receive compensation as soon as their injury's cause was verified. Operating after the fact, tort litigation will always lead to concerns about the effect of moral hazard on manufacturers' efforts to detect dangers in their products.

273 Indeed, I restrict myself to a discussion of the regulatory approach required to overcome causation problems. Other hazardous-substance issues may require different or additional mechanisms. Others have suggested examples of administrative compensation systems for hazardous-substance victims. *See generally* SUPERFUND STUDY, *supra* note 6, at 196-246; Trauberman, *supra* note 1, at 250 (comprehensive model for state or federal statute reforming toxic tort law by establishing victim compensation fund, financed jointly by industry and government and distributed on no-fault basis); Pierce, *supra* note 1, at 1320 (proposing creation of federal "Safety Enhancement and Compensation Agency" for widespread compensation of accident victims and for safety regulation); Reed, *Hazardous Waste Pollution: The Need for a Different Statutory Approach*, 12 ENVTL. L. 443, 466-67 (1982) (advocating comprehensive federal approach to hazardous waste problems, including compensation fund for personal medical injuries and creation of federal toxic tort based on strict and joint and several liability, and modified causation principles).

New Zealand's program, wherein no fault administrative compensation has generally replaced tort litigation, demonstrated the efficacy of such compensation systems. *See* Marks, *A First in National No Fault: The Accident Compensation Act of 1972 of New Zealand*, 47 AUSTRALIAN L.J. 516 (1973). A narrower approach, involving compensation of environmental injuries, exists in Japan. *See* Aronson, *Review Essay: Environmental Law in Japan*, 7 HARV. ENVTL. L. REV. 135 (1983).

274 Federalizing hazardous-substance-injury regulation and compensation could greatly enhance the system's efficiency. With such an approach, a single federal agency would collect and analyze scientific research on causation, aggregate personal injury statistics, and follow technological advances. The agency would rapidly disseminate this information along with its conclusions to state and local governments, manufacturers, and the scientific community; these parties could then fashion appropriate responses.

275 Much hazardous-substance litigation will involve federal jurisdiction based on diversity. For example, most spermicide manufacturers market their products in all 50 states. In contrast, many hazardous waste sites, and the claims they generate, will involve state jurisdiction. One could argue that states and their voters should determine the extent of liability for parties who placed hazardous substances into ground water. *See generally* Hathaway, *Hazardous Substance Victims Need a Federal Cause of Action*, 14 ENVTL. L. REP. 10,294 (1984). The federal government, however, has chosen to make this an area of federal concern by passing several overarching regulatory schemes. *Developments*, *supra* note 240, at 1470-76; *see also* Exxon Corp. v. Hunt, 475 U.S. 355 (1986) (Comprehensive Environmental Response, Compensation, and Liability Act, 42 U.S.C. § 9614(c) (1982) held to pre-empt New Jersey Compensation and Control Act provision permitting state to impose tax to support cleanup expenses).

analysis of hazardous-substance injury would also interact with decisionmakers at other federal agencies. This interaction would facilitate the development of uniform solutions to causation problems.

A Federal Hazardous Substance Science Panel would perform three functions: policymaking, adjudication, and boundary-drawing. These three functions would deal respectively with the first, second, and third levels of uncertainty.

In its policymaking role and in dealing with level one uncertainty, the Science Panel would consult with the Environmental Protection Agency (EPA), the Federal Drug Administration (FDA), the Occupational Safety and Health Administration (OSHA), the Consumer Products Safety Commission (CPSC), and other federal agencies to develop a set of policies that address trans-scientific issues. Presently, a good deal of consensus exists on these policies, at least with regard to carcinogens.[276] The Science Panel would codify these policies and amend them as necessary.[277] These policies would then provide the basis for resolution of trans-scientific issues in hazardous substance injury.

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276 Fortunately, agencies have reached some harmony on level one uncertainty. In 1979, for example, experts from OSHA, EPA, and CPSC concurred on a set of cancer principles, setting out the role of animal bioassays as well as short-term tests. WORK GROUP ON RISK ASSESSMENT OF THE INTERAGENCY REGULATORY LIAISON GROUP, CONSUMER PROD. SAFETY COMM'N, ENVIRONMENTAL PROTECTION AGENCY, DEPARTMENT OF HEALTH, EDUC. AND WELFARE, FOOD AND DRUG ADM. & DEPARTMENT OF AGRIC., Scientific Bases for Identification of Potential Carcinogens and Estimation of Risks, *reprinted in* 44 Fed. Reg. 39,858 (1979). Despite some minor tinkering by the EPA, these principles remain in force. EPA Determination of Reportable Quantities for Hazardous Substances, 40 C.F.R. pt. 117 (1987); EPA Designation Reportable Quantities, and Notification, 40 C.F.R. pt. 302 (1987).

Regulatory agencies' decisions regarding these fundamental uncertainties and policies are made only after the input of literally hundreds of experts through notice-and-comment rulemaking. Moreover, relatively affluent and very well-respected environmental lobbying groups can advocate an approach that protects the public health. The resulting policies form the fundamental groundwork necessary to overcome level one uncertainty.

However, the federal agencies may be too fragmented to continue to develop coordinated policies. *See* COMMISSION ON LAW AND THE ECONOMY, AM. BAR ASS'N, FEDERAL REGULATION: ROADS TO REFORM 14 (1979); Tripp & Jaffe, *Preventing Ground Water Pollution: Towards a Coordinated Strategy to Protect Critical Recharge Zones*, 3 HARV. ENVTL. L. REV. 1, 15 (1979). In addition, the courts reviewing agency decisions are affected by the same corpuscularian state of mind that influences courts hearing common law actions. *See* T. Brennan, The Judicial Myth of Scientific Certainty: The Demise of Generic Rule-Making (unpublished manuscript available from author).

277 The panel could most appropriately achieve codification through notice-and-comment rulemaking, although delays in codification might necessitate the use of hybrid procedures. *See* Vermont Yankee Nuclear Power Corp. v. Natural Resources Defense Council, 435 U.S. 519, 543 (1978); *see also* Scalia, *Vermont Yankee: The APA, the D.C. Circuit and the Supreme Court*, 1978 SUP. CT. REV. 345, 406 ("While 'hybrid rulemaking' may no longer be devised by the courts under the APA, it will continue to flourish in a multiplicity of special statutes that modify the APA's dispositions . . . .").

As its second function, the Science Panel would review statistical and epidemiological evidence of injury caused by hazardous substances. The Panel would act as a "science court" and decide those questions arising from level two uncertainty.[278] This adjudicatory function would operate in two phases. In the first phase, the Science Panel would monitor scientific and especially toxicological literature for studies suggesting a causal relationship between hazardous substances and disease or injury. In addition, the Panel would review petitions from citizens, business enterprises, or community action groups aware or suspicious of hazardous-substance threats.[279]

In the second phase of the adjudicatory function, the Panel would consider the petition or agency suspicion on the basis of a predetermined threshold for action. This threshold would incorporate priorities based on the quality of the evidence available and the number of people exposed to the hazardous substance. The Science Panel would have three options. First, it could simply dismiss the petition as groundless. Second, it could initiate toxicological or epidemiological studies on the hazardous substance in order to establish or rule out causation.[280] As a third option, the Panel could

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278 *See generally* Bazelon, *Coping with Technology Through the Legal Process*, 62 CORNELL L. REV. 817 (1977) (endorsing goals of science court but expressing reservations about potential impact of its factual determinations.); *cf.* Martin, *The Proposed "Science Court"*, 75 MICH. L. REV. 1058 (1977) (cautiously supporting establishment of science court and setting out specifics for selection of judges, procedural matters, and opinion writing).

279 The panel would encourage these petitions, especially from business, because they would represent good faith efforts to reduce injury. Litigants could use the petitions as evidence in a later proceeding about liability. Allowing citizens to initiate proceedings under the regulatory approach parallels the citizen participation that Rosenberg cites as an asset of tort litigation. *See* Rosenberg, *supra* note 3, at 927.

280 In this situation, the Science Panel would also undertake ongoing medical followup of the exposed group or a sample of the exposed group. For instance, consider a toxic waste site where there were suggestions of increased incidence of cancer among citizens in the surrounding area. The science panel would initiate studies in the area and also complete follow-up exams on community members. A regulatory body is much better suited for this task than a common law court because the common law court is poorly equipped to order and monitor such follow-ups.

However, some commentators have encouraged recovery in common law courts from increased risk and fear of cancer. Note, *Increased Risk of Cancer as an Actionable Injury*, 18 GA. L. REV. 563 (1984); Note, *Emotional Distress Damages for Cancerphobia: A Case for the DES Daughter*, 14 PAC. L.J. 1215 (1983). Some courts have addressed recovery for emotional injury. *See* Ayers v. Township of Jackson, 189 N.J. Super. 561, 461 A.2d 184 (1983) (fear of cancer in itself insufficient basis for claim of emotional anguish); *cf.* Howard v. Mount Sinai Hospital, 63 Wisc. 2d 515, 217 N.W.2d 383 (1974) (no recovery for emotional stress from fear of cancer). The agency could monitor and compensate exposed citizens if disease eventually occurred. This ongoing monitoring would allay the potential victims' fears of receiving no compensation for their harm.

The Science Panel would obviously need a great deal of expertise, which the federal government need not employ directly. The Science Panel could contract with academics or private consultants for the design of appropriate studies, as well as for follow-up

certify the evidence cited in the petition as proving causation and establish the fraction of proportionate causation.[281] The Panel would reach all of these decisions by consensus among a group of scientists, consumers, workers, and business representatives on the Science Panel.[282] These decisions would be subject to de novo review on matters of law in an appellate court, whereas findings of fact would be granted deference by the reviewing court.[283]

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monitoring of an exposed population. The government would, however, have to undertake the training of more experts in preventive medicine.

281 Equitable treatment for both plaintiff and defendant requires proportionate compensation for injury from hazardous substances. The available scientific evidence provides an attributable fraction of the injury caused by a substance in question. *See supra* notes 202-04 and accompanying text; Brennan & Carter, *supra* note 56, at 58. This kind of compensation should occur even if the attributable fraction is less than fifty percent, the number common law courts rely on when applying the more-likely-than-not standard. *See generally* King, *Causation, Valuation, and Chance in Personal Injury Torts Involving Preexisting Conditions and Future Consequences,* 90 YALE L.J. 1353, 1376 (1981).

Precedent exists for agency developed proportionate compensation calculations. In 1983, Congress required the development of "radioepidemiological tables" to estimate the probability that a thyroid cancer victim developed his disease as a result of exposure to a previous dose of radiation. Orphan Drug Act, Pub. L. No. 97-414, § 7(b), 96 Stat. 2049, 2059 (1983). The commissioned study was published in 1985. AD HOC WORKING GROUP TO DEVELOP RADIOEPIDEMIOLOGICAL TABLES, NATIONAL INST. OF HEALTH, REPORT OF THE NATIONAL INSTITUTES OF HEALTH AD HOC WORKING GROUP TO DEVELOP RADIOEPIDEMIOLOGICAL TABLES (1985). Before its publication this report was reviewed by a special oversight committee. *See* NATIONAL RESEARCH COUNCIL, NATIONAL ACADEMY OF SCIENCES, ASSIGNED SHARE FOR RADIATION AS A CAUSE OF CANCER: REVIEW OF RADIOEPIDEMIOLOGIC TABLES ASSIGNING PROBABILITIES OF CAUSATION: FINAL REPORT (1984). The oversight committee had some problems with the assumptions used by the Ad Hoc Committee, but did not dispute the concept of assigned share compensation. Several other commentators have since reviewed the radioepidemiological tables, and expressed confidence that this method of dealing with uncertainty is better than establishing causation through litigation. *See* Lagakos & Mosteller, *Assigned Shares in Compensation for Radiation-Related Cancers,* 6 RISK ANALYSIS 345 (1986). *But see* Council on Scientific Affairs, American Medical Ass'n, *Radioepidemiological Tables,* 257 J. A.M.A. 806 (1987). The regnlatory approach can be generally compared with the common law courts' efforts by contrasting the above articles with the decision of Allen v. United States, 588 F. Supp. 247 (D. Utah 1984), *rev'd,* 816 F.2d 1417 (10th Cir. 1987). *See also In re* "Agent Orange" Prod. Liab. Litigation, 611 F. Supp. 1223 (E.D.N.Y. 1985) (struggling with admissibility of epidemiological evidence regarding relevant exposure to Agent Orange and alleged related health problems).

282 There is a fear that this group would become overly politicized and that the decisions would reflect only narrow interests. Given the current state of tort litigation, however, all parties should agree that their interests are served by consistent and just decisions regarding causation. More important, the independence of the Hazardous Substance Authority is integral to its functioning as an alternative to tort litigation.

In establishing "p" values, the Panel would employ the standard statistical levels of significance. "p" values of less than .05 would be acceptable and corresponding confidence intervals would be used.

283 Science Panel causation findings would present many of the same constitutional questions presented by the existence of medical malpractice screening panels. The constitutional quid pro quo should be quite apparent in this setting. The inability of tort law to accommodate the scientific evidence of causation in these cases necessitates the science panel and its findings. *See generally* Learner, *supra* note 271.

The third function of the Science Panel would involve describing the boundaries or limits of the "at risk" or exposed group. Given the heterogeneity of hazardous substances and the nature and number of injuries they cause, the Science Panel would set out broad guidelines of exposure for injury-causing hazardous substances. For instance, with a hazardous substance such as asbestos, the Science Panel could set the minimum number of months of exposure and degree of exposure to qualify as "at risk."[284] For a spermicide, the Science Panel could spell out the maximum number of days after exposure when a mother was still "at risk" for bearing an injured child. At a hazardous waste site, the Science Panel could define the geography of the exposed group.[285]

The determinations by the Science Panel could be used in a number of ways. First, administrative agencies could use the information on causation and exposures in planning future actions. For instance, the EPA could use the information gathered at various waste sites to improve its handling of Superfund sites. The FDA could use the evidence of teratogenesis from spermicides to develop a set of policies about post-marketing testing. Thus, the data devel-

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284 On this point, the studies on molecular genetics that record exposure could be most helpful. *See supra* note 174. Adding molecular exposure data to epidemiological studies would greatly increase those studies' accuracy of causal determinations. Without this exposure data, epidemiology can sometimes be less than clear. *See* Dore, *A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-in-Fact*, 7 HARV. ENVTL. L. REV. 429 (1983) (identifying problems with this type of evidence and recommending guidelines for its proper use); Hall & Silbergeld, *Reappraising Epidemiology: A Response to Mr. Dore*, 7 HARV. ENVTL. L. REV. 441 (1983) (arguing that epidemiological studies should play an important role as circumstantional evidence).

285 Again, a circuit court could review these decisions, but would defer to the Panel on factual findings. The regulatory approach would require clear and strong Congressional language on the matter of judicial review. Without this strong language, the regulatory approach would only add to the administrative costs and delays. *See* U.S. GEN. ACCOUNTING OFFICE, DELAYS IN SETTING WORKPLACE STANDARDS FOR CANCER-CAUSING AND OTHER DANGEROUS SUBSTANCES (1977) (details delays inherent in rulemaking procedures and offers suggestions for improvements).

Courts have reviewed the use of generic principles by regulatory agencies. The Food and Drug Administration's policy regarding the review of drugs for evidence of efficacy was the subject of a great deal of litigation in the 1970s. *See, e.g.*, SmithKline Corp. v. FDA, 587 F.2d 1107 (D.C. Cir. 1978) (review of FDA order denying new drug application on ground that clinical trials were deficient under FDA regulations). The OSHA Generic Cancer Policy was a thorough effort to abate exposure to workplace carcinogens. *See* OSHA, Identification, Classification and Regulation of Potential Occupational Carcinogens, 29 C.F.R. pt. 1990 (1987). The Supreme Court found OSHA's effort lacking in several areas. Industrial Union Dep't v. American Petroleum Inst., 448 U.S. 607 (1980). Nothing in either *SmithKline* or *Industrial Union*, however, prohibited the use of generic principles.

Reviewing courts must appreciate that exposure definitions are not easily standardized. Every significant study presented to the Panel would require review to determine if exposure data was meaningful. Unlike p values and confidence intervals, exposure issues are not readily quantifiable.

oped by the Science Panel could serve both command and control regulation and economic incentive approaches to regulation.[286]

In essence, the Science Panel would complement and supplement the activities of the scientific investigation departments of a number of federal agencies. Four major benefits accrue from this duplication of efforts. First, the Science Panel's expertise in toxicology and epidemiology would allow it to approach hazardous-substance injury in a coordinated fashion. Second, the Science Panel could develop a fully coherent approach to policy issues in science. Third, the heterogeneity of the substances of concern to the Science Panel would decrease the chances that one industry could effectively "capture" the Panel. Fourth, the Science Panel would move beyond general issues of quantification of risk and provide information useful to individual victims.

Litigants could employ the Science Panel's determinations. Both plaintiffs and defendants could rely upon Panel determinations regarding causation, attributable risk, and exposure. Of course, the evidence from the Science Panel would not necessarily be determinative. The trier of fact would weigh all the evidence without granting special significance to Science Panel data.

The Science Panel data would serve three purposes in tort litigation. First, litigants would have access to probabilistic evidence without having to pay for it themselves. This would lessen the inequities involved in litigation of hazardous-substance claims that pit individuals against large corporations. Second, the Science Panel's openly probabilistic evidence would serve to instruct courts in the nature of probabilistic causation. Introducing the Science Panel data as evidence would force judges and juries to consider confidence intervals and attributable fractions, thus accelerating the ongoing learning process.[287]

Third, Science Panel evidence in tort litigation would highlight the different evidentiary thresholds needed for setting prospective standards and for assigning post facto liability. The Science Panel may, for instance, find evidence that nuclear waste sites slightly increase the rate of thyroid cancer in residents living near the sites. The risk, in this example, would be slight, but widespread, resulting in a large number of victims with low attributable fractions.

This kind of evidence would be problematic for a court using notions of moral causation, but straightforward for an agency interested in preventing such injuries in the future. Level one and level three uncertainty are not as difficult for agencies to deal with as they

286 *See* Ackerman & Stewart, *Reforming Environmental Law,* 37 STAN. L. REV. 1333 (1985).

287 *See supra* notes 122-70 and accompanying text.

are for courts using notions of moral causation. In summary, Science Panel evidence would guide agency action without necessarily being determinative in tort litigation. Common law courts would retain their prerogative to judge evidence even as they grow accustomed to probabilistic causation and even as they determine how large a role such causation should play in the assigument of liability.

Circumventing tort litigation altogether is another alternative. The Science Panel could simply integrate their determinations into an overarching system that would deter injurers and compensate those injured by hazardous substances. A Compensation Panel could employ both the criteria for exposure and the proportionate causation fraction provided by the Science Panel, ensure that the applicant's injury is of the kind caused by the hazardous substance, and determine the amount of compensation for individuals. The Compensation Panel would provide money to pay for medical costs and also would provide disability payments commensurate with those available through Social Security.[288]

A General Compensation Fund would provide the compensation. Revenue for the Fund would be generated by a general tax on industry as well as funds accrued through actions against producers of hazardous substances.[289] Compensation for victims would not be delayed by actions instituted by hazardous-substance producers concerned about their liability. Once the Science Panel satisfactorily demonstrated causation, compensation would become available.[290]

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288 The Compensation Panel would attempt to make the injured party whole once again by providing for medical costs and rehabilitation. In addition, income insurance would be available in the form of disability payments. In order to minimize this system's cost, thereby rendering the approach economically and politically viable, victims would not receive punitive damages or compensation for emotional suffering.

Although the compensation system outlined could be quite expensive, raising the specter of the Black Lung Administration, opposition to the compensation scheme would be unfounded. The Compensation Panel would not abandon causation as did the Black Lung program. The Panel should at least ensure that the injury suffered by the applicant is the type caused by the substance. The Black Lung program did not accomplish this. *See* Lopatto, *The Federal Black Lung Program: A 1983 Primer*, 85 W. VA. L. REV. 677 (1983).

289 The compensation fund would resemble the clean-up fund established by Superfund. 42 U.S.C. §§ 9601-9657 (1982 & Supp. III 1985); *see Developments*, *supra* note 240, at 1472. The Hazardous Substance Authority would promote compromise by taking a liberal approach to settlements with manufacturers. *See generally* Miller, *EPA Superfund Enforcement: The Question Isn't When to Negotiate and When to Litigate, But How to do Either and How Often*, 13 ENVTL. L. REP. 10,062 (1983).

290 This postponement of review is akin to that accomplished by section 104 of CERCLA, 42 U.S.C. § 9604 (1982) before the 1986 overhaul of the Superfund program. *See, e.g.*, Wagner Elec. Corp. v. Thomas, 612 F. Supp. 736 (D. Kan. 1985) (court lacked jurisdiction to conduct pre-enforcement review of EPA's section 104 cleanups); *see also*, *Developments*, *supra* note 240, at 1486-89.

The Compensation Panel would also overcome the problem of third level uncer-

This comprehensive scheme would also need an Enforcement Panel. The Enforcement Panel would cooperate closely with similar sections at the FDA, EPA, OSHA, and the Justice Department in determining the liability of hazardous substance producers for the costs of compensation. Any awards in these suits would be funneled into the General Compensation Fund. In this manner, the Enforcement Panel would attempt to internalize the cost of production of hazardous substances to producers.[291]

This comprehensive regulatory approach to compensation and

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tainty by awarding proportionate compensation. Tort focuses on the individual and on the causal chain from that individual to the injurer. In hazardous substance litigation that chain is often severed, and the evidence available reveals only what proportion of the disease in a certain group is caused by the substance. The compensation panel can accommodate this probabilistic evidence and provide each injured party with compensation commensurate with the fraction of the disease caused by the hazardous substance in the exposed group. This satisfies justice and morality without reverting to a focus on the individual. The best available scientific evidence dictates this kind of proportionate compensation. *See* Brennan & Carter, *supra* note 56, at 56.

291 The enabling legislation would establish the standard for liability used by the enforcement department. Although there are several candidates, the main contenders are strict liability and negligence. CERCLA's failure to set a liability standard resulted in a great deal of litigation. *Developments*, *supra* note 240, at 1512. Litigation occurs not only over the standard of liability, but also over the scope of liability and the nature of causation. *Id.* at 1516-18.

Each choice for the standard of liability is buttressed by economic theories that demonstrate the efficiency provided by the particular standard. Schwartz, *Products Liability, Corporate Structure, and Bankruptcy: Toxic Substances and the Remote Risk Relationship*, 14 J. LEGAL STUD. 689, 692 (1985); Schwartz, *Foreword: Understanding Products Liability*, 67 CALIF. L. REV. 435, 483 (1979). Much of this debate focuses on the role of what the manufacturer knew or could have known. Wade, *On the Effect in Product Liability of Knowledge Unavailable Prior to Marketing*, 58 N.Y.U. L. REV. 734, 757 (1983). This choice, a matter of economic theory, is beyond the scope of this paper.

In addition to the liability standard, the enabling legislation should clarify several other issues. First, the legislation should empower the enforcement panel to pursue criminal actions against producers of hazardous substances who have acted in a grossly or willfully negligent manner. *See* Elliott, *Goal Analysis versus Institutional Analysis of Toxic Compensation Systems*, 73 GEO. L.J. 1357 (1985); *see also* Kraakman, *Corporate Liability Strategies and the Costs of Legal Controls*, 93 YALE L.J. 857, 869 (1984) (discussing imposition of noncriminal personal liability on management). Second, the legislation should encourage the enforcement panel to pursue settlements with producers. *See Developments*, *supra* note 240, at 1504.

Finally, the enabling legislation should state policies regarding both producers' bankruptcy and the retroactivity of the Science Panel's findings. There are numerous proposals dealing with liable parties' bankruptcy in mass tort claims. *See* Roe, *Bankruptcy and Mass Tort*, 84 COLUM. L. REV. 846 (1984); Hoffman, *Environmental Protection and Bankruptcy Rehabilitation: Toward a Better Compromise*, 11 ECOLOGY L.Q. 671 (1984). Similar policies should cover retroactive liability determinations, a major problem under CERCLA. *See Developments*, *supra* note 240, at 1540.

Finally, the enabling legislation would authorize the Enforcement Panel to collect a hazardous-substance tax. A broad-based tax on industry is perhaps in order to help internalize costs. Additionally, high-risk industries, such as oil companies and chemical manufacturers, might be encouraged to contribute larger amounts to the General Compensation Fund.

deterrence of hazardous-substance injury maintains many of the benefits of tort litigation.[292] Indeed, replacing tort litigation with the three federal Panels rather than duplicate the common law courts with an alternative system of compensation and deterrence makes the most sense.

The proposal for a Science Panel is nonetheless neutral on the question of replacing or maintaining tort litigation of hazardous substances. The Science Panel's determinations can be used to assist common law courts or can be integrated into an alternative system. To the extent that moral causation plays a role in both preserving certain notions of liberal justice in tort litigation and in assigning ex post facto liability, I argue that replacing tort litigation with a comprehensive regulatory approach would be a mistake. Perhaps the Science Panel should ideally play a less radical, more advisory role. The Science Panel's data could instruct common law courts about probabilistic causation and allow courts to reach a compromise between moral and probabilistic causation. At the same time, the Science Panel's information can gnide agencies in the design of more stringent regnlation and thus help prevent future injuries.

CONCLUSION

Causation issues represent a major impediment to the successful resolution of claims based on injuries from hazardous substances. Tort law nurtures a set of causal concepts that emphasize the individual and the elaboration of a causal chain from injured to injurer. The evidence linking hazardous substances to injuries, however, is largely probabilistic; forcing such evidence into a causal chain model generates great confusion.

A federal Science Panel could alleviate problems that occur when using probabilistic causation in the law. This Science Panel could make rational decisions with regard to the three levels of uncertainty presented by probabilistic evidence. The Panel's determination would also aid common law courts and regulatory agencies. Alternatively, the Science Panel could be integrated into a compre-

292 *See supra* note 270. The injured could still initiate actions, and damage awards could be tailored to fit tbe injury. The Panel requires a full time scientific staff, but contracting much of the work to university public health experts can minimize the Panel's size.

Moreover, the Hazardous Substance Authority would avoid the usual criticisms of regulatory approaches, such as those set forth by Trauberman. *See* Trauberman, *supra* note 1, at 203-06. The Authority would not be fragmented, but would cooperate with other agencies. The Authority would promote industry research into hazardous substance injury and reward prevention. Finally, the compensation process would be far less time consuming than that of tort law.

hensive system of deterrence and compensation for hazardous-substance injury. In either case, the Science Panel would help overcome the challenge presented to legal reasoning by probabilistic causation.