Tort Actions for Cancer: Deterrence, Compensation, and Environmental Carcinogenesis
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Most cases of cancer result from exposure to man-made chemicals in the environment.¹ These cancers are torts, injuries inflicted by one person on another. Nevertheless, cancer victims nearly always fail to recover damages in tort because they are unable to meet traditional standards of proof. Cancer symptoms are usually latent for many years after exposure to carcinogens, and therefore it is often difficult for victims to assemble evidence of the tortious exposure. Furthermore, limited scientific understanding of the etiology² of cancer makes it almost impossible to establish proximate cause.

In the absence of an effective tort remedy, current strategies in the war on cancer inadequately serve the important objectives of cancer prevention and victim compensation. In order to enhance the effectiveness of the tort mechanism, this Note proposes that state legislatures adopt a catalog of carcinogenic substances to facilitate proof of proximate cause in cancer tort suits. If a cancer victim shows that she was exposed by the defendant to a threshold quantity of a substance listed in the catalog, the burden would shift to the defendant to show that he should not be held liable. The proximate cause requirements would then serve the functional objective of deterring carcinogen production and reducing future cancer incidence. In addition to strengthening the tort remedy, this proposal would complement current efforts to control the cancer epidemic through governmental regulation and medical research.

I. Current Approaches and Their Limitations

It is now widely accepted in the scientific community that between sixty and ninety percent of all cancers result from exposure

¹ See P. Barth & H. Hunt, Workers' Compensation and Work-Related Illnesses and Diseases 84 (1980) (85% to 90% of all cancers due to environmental phenomena); Doniger, Federal Regulation of Vinyl Chloride; A Short Course in the Law and Policy of Toxic Substances Control, 7 Ecology L.Q. 497, 509 (1978) (60% to 90% of all cancers caused by exposure to chemical substances in environment).

² The etiology of a disease is the “group of conditions which form [its] cause.” W. Thompson, Black's Medical Dictionary 351 (32d ed. 1979).
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to factors introduced into the environment by man.\(^3\) The incidence of cancer therefore can be lessened substantially by reducing the exposure of human beings to carcinogenic substances.\(^4\) Nevertheless, the current strategies in the war on cancer—government regulation, medical research, and private tort actions—fail to achieve this goal.

A. Regulation of Carcinogenic Substances

The primary strategy for controlling human exposure to carcinogens is government regulation of the production, use, and disposal of such substances. Six major federal agencies, empowered by fifteen separate statutes, regulate carcinogenic and toxic substances.\(^6\) These

3. P. Barth & H. Hunt, supra note 1, at 84; Doniger, supra note 1, at 509.
4. Doniger, supra note 1, at 510; see Cairns, The Cancer Problem, SCIENTIFIC AM., Nov. 1975, at 64, 64 ("Because we can act to alter the environment, those cancers are potentially avoidable.")
5. The current effort to eradicate cancer has many of the features of a crusade. See 7 WEEKLY COMP. OF PRES. DOC. 89, 92 (Jan. 25, 1971) (President Nixon's State of Union Message calling for intensive campaign comparable to atomic bomb or moon projects, aimed at "conquering this dread disease"). The bills that eventually passed as the 1971 National Cancer Act, 42 U.S.C. §§ 281-286g (1976), were known as the Conquest of Cancer Bill, S. 1828, 92d Cong., 1st Sess. (1971), and the National Cancer Attack Bill, H.R. 11302, 92d Cong., 1st Sess. (1971).

agencies and their supporting statutory schemes all seek to control environmental carcinogenesis by enforcing specific deterrence measures. 7

Some statutes, called "no-risk" measures, prohibit any human exposure to specified carcinogenic substances. 8 This type of regulation reflects a collective decision that these carcinogens are to be eliminated from the environment at all costs. 9 Other statutes, however, establish a more complex regulatory calculus. They require agencies to evaluate, on a cost-benefit basis, the net social effects of regulating a carcinogenic substance 10 or of imposing a particular level of regu-

7. "Specific or collective deterrence" refers to accident cost reduction measures adopted through the political process. See G. Calabresi, The Costs of Accidents 68 (1970) (specific deterrence "involves deciding collectively the degree to which we want any given activity, who should participate in it, and how we want it done"). "General deterrence," by contrast, refers to methods for deciding what the accidental costs of activities are and letting the market determine the degree to which ... activities are desired given such costs .... [I]t involves giving people freedom to choose whether they would rather engage in the activity and pay the costs of doing so, including accident costs, or, given the accident costs, engage in safer activities that might otherwise have seemed less desirable. Id. at 69. This Note uses these terms to refer to methods for controlling all externalities of economic actors, not simply their accident costs.

8. See, e.g., Food, Drug, and Cosmetic Act, 21 U.S.C. § 348(c)(3)(A) (1976) (prohibiting certification for safe use of any food additive found to induce cancer when ingested by humans or animals—the Delaney Clause); Clean Air Act, 42 U.S.C. § 7412 (Supp. III 1979) (prohibiting air pollution that may reasonably be expected to result in increase in mortality or serious illness). See generally Kraus, Environmental Carcinogenesis: Regulation on the Frontiers of Science, 7 Envt'l L. 83, 124-26 (1976) (discussing Delaney Clause); Leape, Quantitative Risk Assessment in Regulation of Environmental Carcinogens, 4 Harv. Envt'l L. Rev. 86, 106-07 (1980) (discussing no-risk or "health only" statutory provisions and agency responses to these mandates).

9. For example, the Federal Water Pollution Control Act, § 307(a), 33 U.S.C. § 1317(a) (1976), until its amendment in 1977, barred the Environmental Protection Agency from considering economic factors in regulating toxic water pollutants. See H.R. Rep. No. 911, 92d Cong., 2d Sess. 113 (1972) ("The committee considers that the discharge of toxic pollutants are [sic] much too dangerous to be permitted on merely economic grounds"); cf. G. Calabresi, supra note 7, at 100-02 (specific deterrence is often used to control injurious activity when strong moral values are at stake); Leape, supra note 8, at 88 & n.7 (cost-benefit analysis is objective and therefore amoral, but consumers, labor, and environmental groups are concerned about health risks grounded in moral considerations).

10. See, e.g., Industrial Union Dep't v. American Petroleum Inst., Inc., 100 S. Ct. 2844, 2863 (1980) (Occupational Safety and Health Act requires Administrator to conduct cost-benefit evaluation as to whether standard is "reasonably necessary and appropriate to remedy a significant risk of material health impairment" before promulgating regulations for toxic materials in workplace); Federal Environmental Pesticide Control Act, 7 U.S.C. §§ 136(bb), 136a(c)(3)(C)-(D) (1976 & Supp. III 1979) (prohibiting sale of pesticides that pose "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide"). See generally Comment, Cost-Benefit Analysis for Standards Regulating Toxic Substances Under the Occupational Safety and Health Act: American Petroleum Institute v. OSHA, 60 B.U. L. Rev. 115 (1980) (discussing possible formulations of cost-benefit analyses required by American Petroleum Inst. v. Occupational Safety & Health Administration, 581 F.2d 493 (5th Cir. 1978)).
The resulting regulations attempt to approximate the efficient resource allocation that would result if the costs of producing carcinogens were directly accounted for in the production decisions of each producer.\(^1\)

Although regulation has removed several carcinogens from the environment,\(^2\) the effect on overall exposure to carcinogens has been insignificant.\(^3\) As with all specific deterrence measures, enforcement

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   (A) the effects of such substance or mixture on health . . . ,
   (B) the effects of such substance or mixture on the environment . . . ,
   (C) the benefits of such substance or mixture for various uses and the availability of substitutes for such uses, and
   (D) the reasonably ascertainable economic consequences of the rule, after consideration of the effect on the national economy, small business, technological innovation, the environment, and public health.

12. Absent regulation, the social costs of producing and using carcinogenic substances are not accounted for in the production decisions of producers because the costs of the production's adverse impact on health are borne by cancer victims rather than carcinogen producers. In economic terms, production of carcinogens results in an externality, that is, a divergence between the private costs and the social costs of carcinogen production. See Dahlman, *The Problem of Externality*, 22 J.L. & Econ. 141, 141 (1979). The presence of an externality reflects the existence of a market failure, in this case the improper pricing of the adverse effect on health and safety that is caused by production. Cost-benefit regulations limit a producer's consumption of society's good health to the amount that would be consumed if the right to damage health were appropriately priced. In cost-benefit analysis, the value of the incremental health benefits resulting from a particular level of regulation is weighed against the costs of implementing the regulation. See, e.g., Leape, *supra* note 8, at 106 (balancing statutes weigh risks posed by chemical to be regulated and compliance costs imposed by regulation). If, however, the externality is generated by an existing plant, even an accurate cost-benefit calculation will not necessarily lead to the socially optimal resource allocation. There is no necessary relationship between the costs of complying with the regulation and the magnitude of the existing externality. The total social cost of the existing plant's operation, measured by the total adverse health impact, may greatly exceed the plant's social benefit even though the regulation is set so that the compliance costs equal the marginal health benefits. Thus, a substantial externality may remain after complete implementation of the cost-benefit regulation.

13. See, e.g., S. Epstein, *supra* note 6, at 400-01 (Delaney Clause has been used to ban Flectol H and MOCA from food packaging, and dulcin, coumarin, safrole, oil of calamus, cyclamate, diethylpyrocarbonate, DES, mercaptopimidazoline, and various dyes as food additives); Doniger, *supra* note 1, at 526 (regulation or threat of regulation has forced vinyl chloride industry to reduce workplace airborne concentrations drastically, to reduce emission outside plant by 95%, and to reduce residual content of vinyl chloride in food packaging).

14. For case histories illustrating the failure of regulatory efforts, see S. Epstein, *supra* note 6, at 76-150 (workers developing cancer as result of regular occupational exposure to asbestos, benzene, and vinyl chloride), id. at 151-240 (consumers developing cancer as result of regular exposure to carcinogenic cosmetic dyes, saccharin, and estrogens used as food additives), and id. at 241-98 (cancers developing as result of nitrosamines and carcinogenic pesticides that pervade environment).

Compare National Institute for Occupational Safety and Health, Suspected Car-
of both no-risk and cost-benefit measures is expensive. Cost-benefit regulatory schemes in particular entail the additional burden of determining the socially optimal level of regulation. Moreover, producers have no incentive, other than the uncertain threat of token sanctions, to incur the costs of complying with the regulations. As a result, these regulatory measures commonly generate lengthy

CINOGENS at ix (2d ed. 1976) (more than 2,400 chemicals have been implicated as carcinogens) with S. Epstein, supra note 6, at 536-40 app. III (approximately 50 carcinogens are subject to regulation).

15. For example, in 1972, government expenditures for pollution control regulation and monitoring were estimated at $351 million, and an additional $306 million was expended in pollution-related governmental research. By 1975, expenditures under these two programs approached $1 billion. See U.S. Council on Environmental Quality, Environmental Quality: Sixth Annual Report 526, 530 (1975) [hereinafter cited as CEQ Annual Report].

16. "Cost-benefit" approaches require a method for comparing the complex health and economic consequences of proposed regulations. Assessing the health consequences of a particular level of regulation requires an estimation of the number of cases of cancer that would be prevented by the regulation and a valuation of the social benefits of preventing those cancers. Both steps in the analysis are likely to underestimate the magnitude of health benefits. See S. Epstein, supra note 6, at 412-15 (quantitative risk assessment is premature science employing economically simplistic terms); cf. Tribe, Trial by Mathematics: Precision and Ritual in the Legal Process, 84 Harv. L. Rev. 1329, 1361-65 (1971) (quantitative approaches to policy determinations will tend to underestimate importance of variables of high degrees of uncertainty in favor of more certain, though perhaps less significant, variables). On the other hand, costs incurred by industry are not likely to be understated because these costs are immediate, subject to relatively certain calculation, and fall upon highly vocal, influential, and cohesive subpopulations. See Doniger, supra note 1, at 516-18 (business incentives to exaggerate difficulties and costs of regulation combined with emphasis on short-term "dislocation costs" of regulation lead to overestimation of economic impact); Karstadt, Protecting Public Health From Hazardous Substances: Federal Regulation of Environmental Contaminants, 5 Envt'l L. Rep. 50,165, 50,172 (1975) (industry may respond to impending large regulatory costs by closing or threatening to close plants).

Even given an adequate determination of the costs involved, any approximation of the efficient resource allocation solution is, at best, a static optimum. Given technological change and advances in medical treatment, the information costs of maintaining socially efficient regulations are necessarily ongoing. See B. Ackerman, S. Rose-Ackerman, J. Sawyer, D. Henderson, The Uncertain Search for Environmental Quality 246-48 (1974) (economic changes necessitate continual adjustment of pollution regulations); Spence & Weitzman, Regulatory Strategies for Pollution Control, in Approaches to Controlling Air Pollution 202-03 (A. Friedlaender ed. 1978) (friction in adjusting to regulations makes dynamic regulatory process costly, time consuming, and perhaps infeasible).

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enforcement proceedings, sustained resistance, and very little compliance by the regulated industries.18

B. Cancer Research

The second major front in the war on cancer is an extremely expensive research program.19 This program adopts the traditional medical approach to fighting disease: it seeks a cure through basic scientific research and develops methods of treating existing cancer victims.20 Although this strategy had some success in reducing the number of cancer fatalities between the mid-1930s and the mid-1950s,21 the death rate has been virtually static since 1955.22 The incidence of cancer, moreover, has continued to rise.23 Thus, the narrow focus of current research programs shows little prospect of long-term cancer prevention.24

18. See J. Esposito, VANISHING AIR 114-18 (1970) (describing futile attempts by authorities over 14-year period to close small rendering plant in Maryland); S. Epstein, supra note 6, at 127-50 (describing industry resistance to attempts by Occupational Safety and Health Administration to revise standards for benzene exposure in workplace).

The complexity of environmental laws contributes to enforcement problems. See Speech by Rick Middleton, Sierra Club Legal Defense Fund, at Yale Law School (Sept. 22, 1980) ("When you consider the complexity of these enforcement procedures [under the Clean Air Act], it's a miracle there's any compliance at all.") (notes on file with Yale Law Journal).

Implementation and enforcement consists of three basic steps: translating general requirements into enforceable standards for individual sources, monitoring regulatees' conduct, and applying sanctions or other incentives to correct noncompliance and to prevent future violations. R. Stewart & J. Krier, supra note 17, at 536. "Each of these steps involves potentials for slippage or failure that can gravely impair the ultimate efficacy of regulatory controls . . . ." Id.

19. Congressional appropriations for the National Cancer Institute almost quadrupled between 1971 and 1977, increasing from $235 million to $815 million. R. Retig, CANCER CRUSADE 298-99 (1977). This massive funding produced "the largest, most extensive planning effort ever undertaken within biomedical research." Id. at 299.

20. See CEQ ANNUAL REPORT, supra note 15, at 34 (most effort has been directed toward cancer treatment and cure rather than prevention). In general, Americans spend more on treating diseases than preventing them. See S. Epstein, supra note 6, at 322 (of $48 billion spent on health care in 1978, 96% was allocated for treatment, 4% for prevention).

21. S. Epstein, supra note 6, at 8 (in mid-1930s, approximately one in five cancer patients survived for five years after diagnosis; by mid-1950s, five-year survival rate was one in three). Epstein reports that general advances in surgical and post-operative techniques, rather than improved medical techniques in treating cancer, were primarily responsible for the reduced mortality. Id. at 8-10.

22. Id. at 8-11. Since 1955, the five-year survival rates for relatively rare cancers such as Hodgkin's disease and acute lymphocytic leukemia in children have increased dramatically. Nevertheless, survival rates for the major cancer killers—lung, breast, and colon cancer—have not experienced significant improvement. Id.; see NATIONAL INSTITUTES OF HEALTH, CANCER PATIENT SURVIVAL 6-7, 62, 158, 157 (1976) (comparing survival trends for various cancers and discussing trends for colon, lung, and breast cancers).

23. See Devesa & Schneiderman, Increase in the Number of Cancer Deaths in the United States, 106 Am. J. EPIDEMIOLOGY 1, 1-5 (1977) (risk of developing cancer increased for every age group). Cancer is the only major killing disease whose incidence is rising. S. Epstein, supra note 6, at 11.

24. A research program aimed at controlling cancer should not be limited to the
C. The Tort System

Through the tort system, individual cancer victims, or their heirs, seek compensation from whoever they believe is responsible for their cancer. The defendant may be the plaintiff's current or former employer or employers, the manufacturer or supplier of a drug or other product, or a polluter.

A major objective of the tort system is to adjust "the conflicting claims of the litigating parties" and to compensate injured parties in a way that is just and fair. The tort system also promotes eco-traditional goal of finding a cure. See CEQ Annual Report, supra note 15, at 36-39 (modesty of improvements in cancer therapy effectiveness makes clear that increased emphasis on prevention is only prudent national cancer policy); R. Moss, The Cancer Syndrome 215-36 (1980) (describing obstacles to cancer prevention posed by current research policy). Scientists should also attempt to identify environmental carcinogens and the circumstances in which individuals are exposed to these substances. Such a comprehensive research program would offer a real prospect of long-term cancer prevention. Moreover, prevention of environmental carcinogenesis would be more efficient—and more humane—than merely treating and curing cancer victims. See Schneiderman, Sources, Resources, and Tsouris, in Persons at High Risk of Cancer 459 (J. Fraumeni ed. 1975) (prevention of cancer by simple public health methods is much more economical use of resources).


29. In Professor Calabresi's formulation, the tort system has two principal goals: it must reduce accident costs, and it must be just or fair. G. Calabresi, supra note 7, at 24. Calabresi declines to make a positive formulation of the "justice" goal. He evaluates particular proposals according to whether they comport with a general sense of fairness or are likely to create specific instances of injustice beyond those of current systems. Id. at 24-26.

In contrast to Calabresi's predominantly utilitarian approach, Professor Fletcher would have the tort system enforce a more explicitly moralistic standard of behavior. See Fletcher, Fairness and Utility in Tort Theory, 85 Harv. L. Rev. 537 (1972). Fletcher separates the question of whether the victim was entitled to compensation from the determination of whether it is fair to hold the defendant liable, and allows recovery from the defendant if the victim was injured by "non-reciprocal risks" created by the defendant's activities. Id. at 542.

Although this Note relies primarily on instrumental or utilitarian policy arguments to justify the imposition of liability, the moralistic approach of Professor Fletcher's "paradigm of reciprocity" provides a useful articulation of the justice goal of the tort system in the cancer context. The paradigm is based on a principle of fairness: all individuals in society have the right to roughly the same
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nomic efficiency by requiring a defendant to internalize the costs of injuries caused by his activities. A defendant who is forced to bear all the costs of his activities will, if behaving rationally, engage in those activities only to the extent that the economic benefits equal or outweigh the total costs.

Although cancer resulting from exposure to man-made carcinogens is a tort, few cancer victims have brought successful tort suits. As a result, a large percentage of the costs of cancer treatment is borne by victims and their families. The tort system's compensation goal is therefore not being met. Moreover, because employers, manufacturers, and polluters are not required to bear the full costs of production, they operate in an economically inefficient and socially harmful manner.

II. The Effects of Limited Scientific Knowledge on Tort Suits for Cancer

The ability of scientists to describe carcinogenesis is restricted to certain statistical statements about the relationship between cancer incidence and exposure to suspected carcinogens. Despite the emergence of security from risk. By analogy to John Rawls' first principle of justice, the principle might read: we all have the right to the maximum amount of security compatible with a like security for everyone else. This means that we are subject to harm, without compensation, from background risks, but that no one may suffer harm from additional risks without recourse for damages against the risk-creator.

Id. at 550 (footnote omitted). Scientists can describe cancer causation only in terms of the creation of additional risk above the background level. The paradigm of reciprocity justifies compensation for cancers that result from this additional risk. See pp. 855-59 infra (cancer victims should recover damages from defendant who exposed them to carcinogen that at least doubled risk of contracting cancer).

50. In the tort system, decentralized judgments by economic actors, considering accident costs as one of the many costs faced in choosing among different courses of action, minimize the sum of accident and accident prevention costs, thus contributing to economic efficiency. See Calabresi, Optimal Deterrence and Accidents, 84 YALE L.J. 656, 656 (1975). In the aggregate, the decisions of these economic actors result in optimal resource allocations if costs are accurately reflected in the decision processes. See G. CALABRESI, supra note 7, at 69-70.

51. See G. CALABRESI, supra note 7, at 73 (if costs of accidents are properly reflected in accident-causing activity, actors will shift to safer and less costly activities or make activities safer).

52. See, e.g., Mahoney v. United States, 220 F. Supp. 823, 841 (E.D. Tenn. 1963) ("Plaintiffs' exposures were well below the marginal limits for human safety."); Garner v. Hecla Mining Co., 19 Utah 2d 367, 371, 431 P.2d 794, 797 (1967) (citing statistical nature of plaintiff's case and "well known but unfortunate uncertainty as to the cause of cancer"). See generally S. Epstein, supra note 6, at 502-08 (describing cancer victims' difficulty in obtaining appropriate judgment or settlement).

53. In 1977, the direct cost of medical treatment for an individual with cancer averaged $20,000, not including indirect costs to the victim's family in the form of lost earnings. S. Epstein, supra note 6, at 5. Nationally, total direct and indirect costs of cancer in 1978 were $30 billion. Id. at 5-8.
of a scientific consensus on the descriptive and predictive strengths of these mathematical relationships, the courts have been reluctant to adapt tort requirements for proof of causation to accommodate evidence accepted by scientists as demonstrating cancer causation.

A. Scientific Understanding of Carcinogenesis

The basic characteristic of the group of diseases called cancer\(^3\) is the uncontrolled growth of cells.\(^5\) Although much has been learned about the medical aspects of this phenomenon, the etiology of cancer remains largely unknown.\(^6\) Given this limited understanding, carcinogenesis can be described only in terms of correlations between the observed incidence of cancer above the background level\(^7\) and exposure to suspected carcinogens.\(^8\) Regardless of the strength of this epidemiological evidence, the actual cause of cancer could be some other factor, whose correlation with the suspected carcinogen has not been noticed.\(^9\) Some of this uncertainty is mitigated by experimental

\(^3\) There are three major groupings of cancer: carcinomas, which arise in the epithelia, the cell layers that line the body and the glands; sarcomas, which affect fibrous tissue and blood vessels and are relatively rare; and leukemias and lymphomas, which are also rare and arise in the blood-forming cells of the bone marrow and lymph nodes. J. Cairns, CANCER: SCIENCE AND SOCIETY 20-22 (1978). The usual classification, however, is based on the organ in which the cancer originates. According to this typology, there are about 200 varieties of cancer. Id. at 22.

\(^5\) See id. at 15-33; M. Shimkin, SCIENCE AND CANCER 1-5, 45-63, 87-98 (1973).

\(^6\) See P. Barth & H. Hunt, supra note 1, at 84 (causes of cancer process have not been identified and understood).

\(^7\) "Individual human subjects in the population are exposed throughout life to a number of carcinogens, which may be considered to provide a background of carcinogenic risk; exposure to any amount of a single carcinogen, however small, is regarded as capable of adding to the total carcinogenic risk." Consumer Product Safety Commission; Environmental Protection Agency; Dep't of Health, Education, and Welfare; Food and Drug Administration; Food Safety and Quality Service, Dep't of Agriculture, Scientific Bases for Identification of Potential Carcinogens and Estimation of Risks, 44 Fed. Reg. 39,858, 39,876 (1979) [hereinafter cited as IRLG Report]. The term "background level," as used in this Note, refers to the expected incidence of cancer in the most general population appropriate for use in epidemiological studies of the particular type of cancer in question. See, e.g., Monson, Effects of Industrial Environment on Health, 8 ENV'TL. L. 663, 677 (1978) (epidemiological data comparing observed with expected deaths among vinyl chloride workers; "[e]xpected numbers based on proportional mortality ratios for United States white males"); id. at 685 (epidemiological data comparing observed and expected deaths from cancer among cadmium workers; "[e]xpected numbers based on incidence rates from the Birmingham (England) Regional Cancer Registry"); Comment, DES and a Proposed Theory of Enterprise Liability, 46 FORDHAM L. REV. 963, 964-65 (1978) (adenocarcinoma of vagina and uterus infrequently reported in female population prior to widespread use of DES).

\(^8\) See Monson, supra note 37, at 670 (epidemiological studies permit comparison of cancer incidence in population exposed to suspected carcinogen with incidence in control population).

\(^9\) See CEQ. ANNUAL REPORT, supra note 15, at 26 (epidemiological studies can seldom isolate single variables for study because of "complex mix of chemical agents to which
testing of chemicals on animals under controlled laboratory conditions.\textsuperscript{40} Although these tests may help to isolate the experimental agent responsible for the observed incidence of cancer,\textsuperscript{41} their results are still expressed as correlations between the experimental agent and cancer incidence.\textsuperscript{42}

Moreover, the relationship between the magnitude and duration of exposure to a carcinogen and the likelihood of contracting cancer must be inferred from indirect evidence. This relationship is investigated by exposing laboratory animals to high levels of the carcinogen.\textsuperscript{43} From the results of such experiments, human reactions to the carcinogen are predicted by the construction of dose-response curves, statistical models derived from observed correlations between known dosages and subsequent increases in the incidence of cancer.\textsuperscript{44} The extrapolations contained in dose-response curves permit the effects of exposure levels much lower than those used in the laboratory to be

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\textsuperscript{40} See Leape, supra note 8, at 93 n.44 ("[A]nimal bioassay is a laboratory procedure in which the scientists administer the test substance to one group of animals and compares [sic] their cancer incidence, or other response, to that of a 'control' group which has not been exposed to the substance.") See generally IRLG Report, supra note 37, at 39,862-69 (describing and evaluating bioassay research techniques).

\textsuperscript{41} See Leape, supra note 8, at 93; cf. IRLG Report, supra note 37, at 39,862-63 (specifying conditions under which test substance can be isolated as experimental variable in bioassay studies). Bioassay has other advantages as well. Latency periods ranging from five to forty years in humans are reduced to one to two years in the naturally short-lived laboratory animals used in bioassay. See U.S. Dep't of Labor, Occupational Safety and Health Administration, Identification, Classification and Regulation of Toxic Substances Posing a Potential Occupational Carcinogenic Risk, 42 Fed. Reg. 54,148, 54,157 (1977). Also, animals can be exposed to carcinogens in much higher dosages than humans commonly receive "to provide maximum detectability of carcinogenic effects within the already narrow confines of test sensitivity." IRLG Report, supra note 37, at 39,864.

\textsuperscript{42} See S. Epstein, supra note 6, at 65.

\textsuperscript{43} See Bates, Laboratory Approaches to the Identification of Carcinogens, 271 ANNALS N.Y. ACAD. SCI. 29, 30-32 (1976) (exposure of animals to high levels of suspected carcinogen is necessary because it is impractical to expose sufficient number of test animals to low dosages of suspected carcinogen to achieve statistically significant result). Critics frequently question the significance of laboratory test results based on high-level exposure to a suspected carcinogen. See S. Epstein, supra note 6, at 1-2 (public surprised and outraged by FDA decision to ban saccharin based on bioassays using extremely high doses). Nevertheless, the carcinogenic quality of a substance is independent of the level of exposure. See Council on Environmental Quality, Toxic Substances Strategy Committee Report to the President, 44 Fed. Reg. 48,134, 48,139 (1979) [hereinafter cited as CEQ Report to the President]; S. Epstein, supra note 6, at 52 ("The bottom line on carcinogenesis testing is this: You can drown an animal in a pool of some substance, suffocate an animal under a heap of it, or beat an animal to death with a sock full of it, but if it isn't carcinogenic, you can't give an animal cancer with it.") (quoting W. Hines and J. Randal, Washington journalists).

\textsuperscript{44} See IRLG Report, supra note 37, at 39,872-73 (describing procedures for constructing dose-response curves from experimental data).
estimated. These predictions, however, rely on two empirical assumptions: they assume that human beings and experimental animals respond comparably to the particular carcinogen, and that the responses observed under large laboratory dosages can be used to predict responses to the much smaller dosages that human beings are likely to encounter.

Despite these limitations, there is scientific agreement on a few key issues. There is agreement that most cancers result from exposure to environmental factors. In addition, scientists generally agree that there is no safe level of exposure to carcinogens, and that there is

45. See id. at 39,875 (close qualitative similarities observed in human and animal responses to carcinogens). The responses are comparable at least to the extent that all human carcinogens, with the exception of arsenic, have been found to cause cancer in animals. Karstadt, supra note 16, at 50,168. In addition, a number of models have been developed for translating animal response data into quantitative predictions of human response. See IRLG Report, supra note 37, at 39,875-76.

46. This extrapolation requires some explicit theory describing the relationship between cancer incidence and exposure to the suspected carcinogen over the relevant range of dosages. See p. 851 infra (linear relationship believed to exist between dosage and cancer incidence). To extrapolate from high level animal exposures, it is also necessary to assume that substances capable of causing cancer in large dosages are carcinogenic in very small amounts.

47. See P. BARTH & H. HUNT, supra note 1, at 84 (environmental phenomena cause most cancers); Doniger, supra note 1, at 509 (exposure to chemical substances in environment causes most cancers).

48. See Karstadt, supra note 16, at 50,174 (majority of cancer researchers conclude that only zero exposure to carcinogenic chemical should be considered safe for humans). But see Pelham, Government Tackles Tricky Question of How to Regulate Carcinogens, 86 Cong. Q. Weekly Rep. 967, 969-63 (1978) (industry objects to no-threshold view and insists upon “socially acceptable or permissible exposure levels”). Even if safe levels of exposure exist in theory, these levels cannot be adequately determined because of uncertainties in dose-response relationships, variability in susceptibility to cancer, and unpredictable interactions among cancer-causing agents. See CEQ Report to the President, supra note 43, at 38,139-40 (each human exposure to carcinogen adds to total carcinogenic risk); IRLG Report, supra note 37, at 39,876 (“The self-replicating nature of cancer, the multiplicity of causative factors to which individuals can be exposed, the additive and possibly synergistic combination of effects, and the wide range of individual susceptibilities [make it] currently unreliable to predict a threshold below which human population exposure to a carcinogen has no effect on cancer risk.”); Leape, supra note 8, at 101 n.116 (because no agent induces form of cancer that does not occur in absence of agent, there is no threshold). As a consequence, federal agencies have assumed, as a matter of policy, that there is no safe level of exposure to carcinogens. See Environmental Protection Agency, National Emission Standards for Hazardous Air Pollutants; Policy and Procedures for Identifying, Assessing, and Regulating Airborne Substances Posing a Risk of Cancer, 44 Fed. Reg. 58,642, 58,644-45 (1979). Policies based on this assumption have been upheld by the courts. See Society of Plastics Indus., Inc. v. Occupational Safety & Health Administration, 509 F.2d 1301, 1308 (2d Cir. 1979) (OSHA permitted to reduce permissible level of vinyl chloride to “lowest detectable level” because no evidence could be produced to identify safe levels of exposure due to deficiencies in existing methodology and research); Industrial Union Dep’t v. Hodgson, 499 F.2d 467, 474-75 (D.C. Cir. 1974) (OSHA permitted to set asbestos standards at lowest level feasible to protect workers from health impairment because insufficient data currently available to predict health effects of various levels of exposure to asbestos dust).
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a linear relationship between the degree of exposure and the likelihood of contracting cancer.\textsuperscript{49} By relying on these generalizations, it is possible to conclude that increased exposure to a particular carcinogen will result in an estimable increase in cancer incidence above the background level for the general population.\textsuperscript{50} Despite the predictive force of this conclusion, however, courts, in denying relief to cancer victims, continue to focus on the limitations of scientific understanding of carcinogenesis.\textsuperscript{51}

B. Why Tort Actions for Cancer Fail

To establish tort liability, a plaintiff must prove three elements: tortious conduct, injury, and proximate cause.\textsuperscript{52} If the plaintiff has cancer, she has obviously suffered injury. The failure to establish either or both of the other two elements, however, frequently stands in the way of tort recovery by the cancer victim.

1. Tortious Conduct

In the typical tort case, the tortious conduct and the resulting injury are contemporaneous. The plaintiff can immediately gather all the available evidence and preserve it for trial. In cancer actions, however, symptoms of the disease commonly appear many years after exposure,\textsuperscript{53} long after important evidence has been lost or destroyed.

\textsuperscript{49} See IRLG Report, supra note 37, at 39,871 (“The linear . . . dose-response model . . . appears to have the soundest scientific basis and is less likely to underestimate risk than other plausible models.”); Crump, Hoel, Langley, & Peto, Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment, 36 Cancer Research 2973, 2979 (1976) (“[L]inear dose-response relationships are likely to be approximately correct for many environmental carcinogens . . . .”); cf. Cornfield, Carcinogenic Risk Assessment, 198 Scr. 693, 695-96 (1977) (although scientific arguments supporting linear model are less than conclusive, model is supported by policy considerations).

\textsuperscript{50} See, e.g., Monson, supra note 37, at 675-98 (data on occupational groups indicate increased cancers above background level due to exposure to occupational carcinogens). \textit{But see} Maugh, \textit{Chemical Carcinogens: How Dangerous Are Low Doses?} 202 Sci. 37, 41 (constructing dose-response curves from existing data is difficult). \textit{See generally} IRLG Report, supra note 37 (describing specific guidelines for carcinogenic risk assessment relating excess cancer incidence to exposures, based on observed and experimental data).


\textsuperscript{52} See W. Prosser, supra note 28, at 143 (elements of negligence cause of action are duty, breach of duty, proximate cause, and injury). This Note uses the single term “tortious conduct,” rather than “duty” and “breach of duty,” to denote conduct that creates liability, whether the cause of action lies in intentional tort, negligence, or strict liability.

\textsuperscript{53} See J. Cairns, supra note 34, at 144 (cancer often does not appear until 7 to 20
Evidentiary problems complicate the plaintiff's case in two ways. First, it is sometimes difficult for the plaintiff to prove that she was exposed to a carcinogen or to establish the magnitude of her exposure. Many actions for occupational cancer, for example, have failed because the plaintiffs were unable to prove the magnitude of exposures they had received years earlier. Solutions for this problem lie outside the tort system. Second, it is often difficult for the plaintiff to identify, with any certainty, the particular defendant who caused the exposure. For example, years after consuming a prescribed drug, plaintiffs have been unable to identify the particular brand or its manufacturer. Thus, the element of tortious conduct is a significant obstacle to the cancer victim suing in tort. Nevertheless, it is not an unreasonable burden from either a doctrinal or a practical perspective.

Although both the compensation and the deterrence objectives of tort law can be achieved without identifying the specific tortfeasor, deterrence does require at least that the plaintiff identify the right class of defendants. Unless compensation is exacted from the class of years after exposure to carcinogen), The difficulty in proving causation as a result of an extended latency period is not unique to cancer torts, Cf. Note, Compensating Victims of Occupational Disease, 95 Harv. L. Rev. 916, 921-22 (1980) (workers less likely to be compensated for job-related illness than for injury due to job-related accident).


55. Adequate recordkeeping requirements should be developed to fill an important gap in current regulatory efforts. OSHA has promulgated regulations requiring employers to keep records on work-related injuries and illnesses. See [1979] 41 OCCUPATIONAL SAFETY & HEALTH REP. (BNA) 2111 (Ref. File) (describing recordkeeping requirements). But these regulations do not require employers to maintain, in the absence of a demonstrated hazard, long-term employment records that would reveal the chemicals to which workers have been exposed. Hence, currently available data are clearly inadequate to reconstruct the epidemiology of occupational cancers with long latency periods. See S. Epstein, supra note 6, at 368. Moreover, workers generally do not have the right to know the identity and nature of health hazards to which they are exposed in the workplace. New York Academy of Sciences, Cancer and the Worker 76 (1977). This information should be available to workers because, without it, workers are unable to protect themselves from hazards or to ensure that health regulations are enforced.

56. See, e.g., Gray v. United States, 445 F. Supp. 357, 338 (S.D. Tex. 1978) (granting summary judgment in favor of codefendant DES manufacturer on ground that plaintiff failed to identify codefendant as manufacturer of DES that her mother had ingested during pregnancy). In 1978, between 80 and 100 cases were pending in the United States against manufacturers of DES, a carcinogenic, man-made estrogen used by millions of pregnant women between 1947 and 1971. Comment, supra note 37, at 963-67. In the first case to go to trial, Barros v. E.R. Squibb & Co., No. 75-1226 (E.D. Pa. Jan. 27, 1978), the jury returned a verdict for the defendant, indicating in a special verdict that plaintiff had not proven that her mother had used Squibb's product. Comment, supra note 37, at 967-68.

57. See Calabresi, Concerning Cause and the Law of Torts, 43 U. Chi. L. Rev. 69, 81 (1975) (proximity of defendants to injury is useful in deciding which causally linked activities should be controlled, because proximate cause examines "relative susceptibility of various actions to modifications diminishing their riskiness"). Professor Calabresi ar-
defendants whose emissions of carcinogens can be influenced by potential liability, there will be no deterrent effect, and only further inefficiencies will result. Once the plaintiff has identified the right class, courts can apply one of three theories—alternative liability,\textsuperscript{58} enterprise liability,\textsuperscript{59} and market share liability\textsuperscript{60}—to shift the burden of identifying the specific tortfeasor to the class of defendants. Those defendants in the class who can show that they could not have caused the injury may be dismissed from the action,\textsuperscript{61} leaving an appropriate class of defendants to share the liability.

2. Proximate Cause

The current limitations of scientific knowledge about the causes of cancer create three major problems for plaintiffs attempting to prove proximate cause in cancer suits. First, courts frequently dismiss cases on the ground that the plaintiff has failed to establish that the suspected carcinogen was capable of causing the cancer.\textsuperscript{62} Second,

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even if the plaintiff succeeds in such a proof, courts may refuse to find proximate cause on the ground that the plaintiff's exposure to the substance was not of "a sufficient amount . . . to do bodily harm."93 In other words, many courts assume, contrary to current scientific understanding,64 that there is a safe threshold level of exposure to carcinogens. Third, even if the court is persuaded that the plaintiff was exposed to a sufficient amount of a carcinogen, it may deny relief because the plaintiff cannot prove that her cancer was caused by that particular exposure.65

These difficulties in establishing proximate cause stem from the courts' refusal to accept scientific evidence about carcinogenesis as legal evidence of causation. Cancer victims should not be precluded from recovering for their injuries solely because carcinogenesis is described by a statistical correlation rather than by a cause-and-effect mechanism. Rather, the statistical correlation should be incorporated into the causation requirement so that the tort mechanism can effectively deter carcinogen production and prevent future cancer incidence.66 It is a statistical certainty that producers of carcinogens are

64. See p. 850 supra (no safe threshold of exposure).
65. See, e.g., Lartigue v. R.J. Reynolds Tobacco Co., 317 F.2d 19, 22-23 (5th Cir. 1963) (affirming jury verdict that plaintiff had failed to prove that his lung cancer was specifically attributable to smoking); Garner v. Hecla Mining Co., 19 Utah 2d 367, 371, 431 P.2d 794, 797 (1967) (affirming denial of benefits to heirs of uranium mine worker who died of lung cancer, citing statistical nature of plaintiffs' case).
66. This Note adopts an instrumental, rather than a moralistic, theory of causation. See note 29 supra. Under this theory, causal requirements in a tort action for cancer would be fulfilled if the assessment of liability based on a finding of causation would further the objective of deterring carcinogen production and preventing cancer incidence. According to Professor Calabresi,

Causal requirements, like all other legal requirements, must ultimately justify themselves in functional terms . . .

In this sense many seemingly significant philosophical questions concerning cause become irrelevant to the use of that term in law. To amplify: so far as legal language is concerned, the "cause" of a disease would depend on how, at any given time, it could be most easily controlled.

Calabresi, supra note 57, at 105.

Professor Borgo provides an elaboration of Calabresi's instrumental conception of causation that is useful in considering legal problems of carcinogenesis:

A type of conduct is causally linked to a type of harm when an increase in the frequency with which conduct of that type is performed leads to an increase in the frequency with which harm of that type occurs . . .

. . . To say that smoking is causally linked to lung cancer is to say that an increase in the performance of that conduct alone will cause a greater incidence of lung cancer. And this is true even if more than one type of conduct is causally linked to the same type of harm. The fact that the incidence of lung cancer is
increasing the incidence of cancer in the general population. The mere fact that the etiology of the injuries is complex should not shield those producers from legal responsibility.

III. Standards of Proof in Tort Actions for Cancer

Defendants' current advantage in suits brought by cancer victims should be eliminated by translating scientific understanding of carcinogenesis into a form that will facilitate proof of proximate cause. A catalog of carcinogenic substances and their dose-response curves should be compiled by a federal agency and then adopted into law by state legislatures. Once a cancer victim has demonstrated that she has been exposed by the defendant to some threshold amount of a listed carcinogen, the burden of proof should shift to the defendant to prove that the exposure was not the cause of the plaintiff's cancer.

A. Conditions for Shifting the Burden of Proof of Causation

Congress should require an existing agency to construct and maintain, based on the best available scientific information, a catalog of carcinogenic substances and their dose-response curves. Agency determinations of whether to list a substance should be made according to established priorities and pursuant to strict standards for the

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68. Any of the six federal agencies that are responsible for regulating carcinogens, see note 6 supra, has sufficient technical expertise to implement this proposal. Because the proposal is not restricted to a generic subset of carcinogens, the agency experienced with the widest range of carcinogens—the Environmental Protection Agency or the Food and Drug Administration—would probably be the most appropriate choice.
69. A schedule for evaluating substances for possible listing in the catalog should be established to discourage lobbying efforts by industries with interests in delaying decisions on particular substances. For an example of such priority-setting, see Consumer Product Safety Commission, Classifying, Evaluating, and Regulating Carcinogens in Consumer
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evaluation of cancer research. These determinations should be made without reference to economic consequences that might flow from the identification of a substance as a carcinogen. The catalog should be promulgated by the federal agency in formal rulemaking procedures. The process—rigorous scientific study followed by almost certain challenge before the agency and in the courts—would necessarily be long and costly. In the long run, however, it would be more efficient to determine the carcinogenicity of a substance in one comprehensive proceeding than to litigate it repeatedly in individual tort suits.


70. A consensus on cancer research standards has been developed by a consortium of federal agencies, the Interagency Regulatory Liaison Group, acting under an inter-agency agreement. Environmental Protection Agency, Regulation of Toxic and Hazardous Substances, 42 Fed. Reg. 54,856 (1977). These agencies recently published a report containing a detailed evaluation of scientific methodologies used to assess carcinogenic risk. See IRLG Report, supra note 37.

71. A major premise of this proposal is that carcinogens will be produced at a socially optimal level if producers of carcinogens are required to internalize the full social costs of their activities. In order to communicate accurate information to producers about the costs of their cancer-causing activities, the scientific determination of carcinogenicity must be made without regard to the economic consequences of such a determination.

72. The agency would be bound by the “on the record” rulemaking requirements of the Administrative Procedures Act, 5 U.S.C. §§ 553, 556-557 (1976). The evidentiary record compiled before the agency would be subject to judicial review under a “substantial evidence test.” Id. § 706(2)(E). Traditional standards for judicial review of administrative procedures would require that the agency prepare a full record before it reaches a determination of carcinogenicity. The agency, however, would have substantial discretion in arriving at a decision. See Vermont Yankee Nuclear Power Corp. v. Natural Resources Defense Council, Inc., 435 U.S. 519, 543-45, 558 (1978) (absent constitutional constraints and barring extremely compelling circumstances, administrative rulings should not be overturned if agency meets minimum procedural requirements); Federal Power Comm’n v. Transcontinental Gas Pipe Line Corp., 423 U.S. 326, 331-33 (1976) (agency decisions may be remanded for inadequate record, but courts should normally defer to agency’s “administrative discretion”).

Courts have been particularly deferential to agency determinations in scientifically uncertain areas such as carcinogen regulation. See Industrial Union Dep’t v. Hodgson, 499 F.2d 467, 474 & n.18 (D.C. Cir. 1974) (promulgation of standards “on the frontiers of scientific knowledge” necessarily requires that agency be afforded “broad discretion to attempt to formulate a solution to the best of its ability on the basis of available information”); cf. Society of Plastics Indus., Inc. v. Occupational Safety & Health Administration, 509 F.2d 1301, 1308 (D.C. Cir. 1975) (act requires that protection be provided to workers “even in circumstances where existing methodology or research is deficient”).

73. Administrative findings of fact would be substantially secure from judicial reversal unless the underlying record developed by the agency is inadequate. See note 72 supra. By precluding litigation in tort cases on the factual questions of carcinogenicity and dose-response curves, the proposal would centralize the fact-finding process and open it for public participation. By minimizing the overall transaction costs of participants in the decisionmaking, the proposal would assure more efficient and comprehensive consideration of the issues involved than would a more decentralized approach. See F. Anderson, supra note 17, at 186-89 (“publicness and centralization” in environmental decisionmaking will
State legislatures should then incorporate the catalog into state tort law. State legislation should create two presumptions that would apply in tort actions for cancer: a conclusive presumption that any substance listed in the catalog is capable of causing cancer in human beings and a rebuttable presumption that a plaintiff’s exposure to any of the listed substances was the cause in fact of her cancer. This second presumption would be triggered when the plaintiff shows, by means of the cataloged dose-response curve, that the amount of exposure to a listed carcinogen exceeded a given threshold. The threshold should be set at a level greater than double the background rate; that is, any exposure that more than doubled the probability of the plaintiff’s developing cancer would exceed the threshold.

75. Legislative imposition of presumptions of this sort is common in the context of occupational disease. See, e.g., 30 U.S.C. § 921(c) (Supp. III 1979) (establishing rebuttable presumptions that pneumoconiosis—black lung disease—arose out of employment if miner was employed in one or more mines for 10 years or more, and that “if a deceased miner was employed for ten years or more in one or more coal mines and died from a respirable disease . . . his death was due to pneumoconiosis’’); N.Y. WORK. COMP. LAW § 47 (McKinney 1965) (presumption that worker who develops any of several specified diseases during or after involvement in certain activities contracted disease as result of his employment). Legislation creating presumptions that apply in judicial proceedings has been commonly upheld against constitutional challenge. See, e.g., Usery v. Turner Elkhorn Mining Co., 428 U.S. 1 (1976) (upholding constitutionality of presumptions created by Black Lung Benefits Act); Mobile, J. & K.C.R.R. v. Turnipseed, 219 U.S. 35, 43 (1910) (presumption mandated by legislature does not violate due process or equal protection if rational connection exists between fact proved and ultimate fact presumed, and if inference of one fact from proof of another is not so unreasonable as to be purely arbitrary).

76. This threshold applies to evidence of exposure to carcinogens. It is designed to eliminate plaintiffs who cannot meet traditional tort standards of proof. Thus, this threshold imposes a legal standard that has no relation to the scientific threshold of safe exposure, the level below which exposure to a carcinogen will not increase the risk of developing cancer. See pp. 850-51 supra (scientific threshold).

77. As a matter of probability theory, setting the threshold at this level is equivalent, under certain simplifying assumptions, to requiring the plaintiff to demonstrate that her cancer was more probably than not caused by exposure to the defendant’s carcinogen. At the outset, in the absence of more particularized evidence, there are only two possible causes of the plaintiff’s cancer: either the cancer resulted from those processes that generate the background incidence of cancer in the general population, or it resulted from the demonstrated exposure to the defendant’s carcinogen. By hypothesis, the prospective probability of developing cancer from each of these causes is known: the background
Once the plaintiff has made a threshold showing, and has proved that the defendant was responsible for the exposure, the burden should shift to the defendant to rebut the presumption of causation. By introducing evidence of the plaintiff's exposure to other carcinogens, the defendant could show that exposure to their carcinogen was not the most likely cause of the plaintiff's cancer. Alternatively, the defendant could join other defendants—producers who had exposed incidence is simply the probability that any member of the general population will develop cancer, and the dose-response curves state the increase in likelihood, above the background probability, that the plaintiff will develop cancer given the level of her exposure to the defendant's carcinogen.

Once the plaintiff has developed cancer, determination of the cause requires a determination that one of the two possible causes was more probably responsible. To find the defendant responsible for the plaintiff's cancer is to find that the retrospective probability that exposure to the defendant's carcinogen was the cause is greater than one-half. Cf. Kaye, Probability Theory Meets Res Ipsa Loquitur, 77 Mich. L. Rev. 1456 (1979) (applying similar analysis to describe factual assumptions subsumed in doctrine of res ipsa loquitur).

If the plaintiff meets the threshold quantity of exposure required by the proposal, the prospective probability of developing cancer from the defendant's carcinogen would be greater than the background incidence. As a result, the retrospective probability would indicate that the cancer, more probably than not, resulted from the defendant's carcinogen. Under the proposal, the burden would be shifted to the defendant to supply evidence tending to undercut the presumption.

The relationships between prospective and retrospective probabilities assumed above are mathematically correct only under certain simplifying assumptions. For example, the fact that the potency of a carcinogen may be altered by interactions with other carcinogens violates the mathematical assumption that the two prospective causes of cancer are statistically independent. Moreover, the population whose cancer incidence is described by the dose-response curve is a subset of the general population used to establish background cancer incidence. Nevertheless, the danger of cancer to the public health justifies the use of these simplifying assumptions to further long-term prevention objectives, despite the absence of definitive scientific knowledge about these difficult factual questions.

78. See, e.g., Braden v. City of Hialeah, 177 So. 2d 235, 236 (Fla. 1965) (affirming denial of benefits because medical testimony did not exclude possibility that petitioner's pigmentation made her more susceptible to skin cancer or that she would not have developed cancer if she was not lifeguard); Garner v. Hecla Mining Co., 19 Utah 2d 367, 371, 431 P.2d 794, 797 (1967) (affirming denial of benefits to uranium mine worker who died of lung cancer, because evidence, including fact that decedent had been habitual cigarette smoker, was insufficient to prove that exposure by last employer had caused cancer); Olson v. Federal Am. Partners, 567 P.2d 710, 712-13 (Wyo. 1977) (evidence, including fact that decedent had been habitual cigarette smoker, insufficient to prove exposure by employer caused cancer).

These cases, relying upon demonstrations of cause-and-effect to establish liability, might well have resulted in different outcomes under a causation rule that focused on the increment to the plaintiff's risk of contracting cancer from exposure to the defendant's carcinogen. For example, if the defendant establishes that the plaintiff was exposed to another carcinogen, one that reacted synergistically with the defendant's carcinogen, the question would still remain as to who is legally responsible for the synergistic increment to the plaintiff's risk resulting from exposure to both carcinogens. If it is the defendant, then introducing evidence of the previous exposure will increase, rather than decrease, the increment for which the defendant is responsible.
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plaintiff to the same carcinogen or other carcinogens—in order to shift or share liability.\textsuperscript{79}

State adoption of the catalog and the presumptions should not preclude a plaintiff from bringing a conventional tort action.\textsuperscript{80} Thus, a plaintiff could attempt to demonstrate in court that her cancer resulted from exposure to a substance not listed in the catalog. Similarly, if the plaintiff was exposed to a cataloged carcinogen, but that exposure was not sufficient to meet the threshold, the plaintiff could still attempt to prove causation.\textsuperscript{81} In both of these cases, of course, the burden of proof would remain with the plaintiff.

B. Impact of the Proposal

The proposed catalog and judicial presumptions would overcome the advantage currently enjoyed by defendants in cancer actions. Cancer victims who could demonstrate significant exposures to carcinogenic substances would no longer be denied compensation because of science's limited ability to describe the etiology of cancer. The proposal would force producers to internalize the actual social costs of their activities, thereby providing an incentive for producers to reduce their emissions of carcinogens to socially optimal levels.

Not all producers of carcinogens would be equally affected under this proposal. Initially, a plaintiff would have to demonstrate that she had been exposed to a carcinogen. The proposal therefore would have its greatest impact in easily verifiable instances of exposure—routine exposures over long periods of time, such as those that occur

\textsuperscript{79} See Smoking, Not Asbestos, Caused Disease, Firm Claims in Suing Tobacco Companies, [1980] 10 OCCUPATIONAL SAFETY & HEALTH REP. (BNA) 463 (defendant asbestos company cross-claimed against six tobacco companies, alleging that smoking, not asbestos, caused plaintiffs' cancers).

\textsuperscript{80} The insufficiency of evidence to warrant listing a substance should not preclude a plaintiff from attempting to demonstrate that a substance is carcinogenic. First, although bioassay and epidemiology can demonstrate carcinogenicity with a high degree of accuracy, they cannot exonerate a substance. IRLG Report, \textit{supra} note 37, at 39,871. Second, the administrative determination of carcinogenicity would be a very lengthy process. Cancer victims should not be barred from bringing suit simply because the agency has not yet made determinations on the substances to which they were exposed. Third, many substances are not suspected to be carcinogens until patterns of incidence emerge. Restricting cancer suits to cataloged substances would discriminate against cancer victims exposed to substances recently discovered to be carcinogenic.

\textsuperscript{81} A plaintiff would be most likely to succeed if the cancer were a "marker tumor," that is, if it could be caused only by a particular chemical. Angiosarcoma of the liver, for example, is caused only by exposure to vinyl chloride. A plaintiff's exposure to vinyl chloride may have been only large enough to increase her prospective probability of developing angiosarcoma of the liver by 50\%. Nevertheless, the plaintiff might well convince a jury that her cancer resulted from that exposure, provided she was not exposed to significant amounts of vinyl chloride from any other source.

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in the workplace or those that are due to environmental contamination;\textsuperscript{82} massive exposures, such as those that result from disasters;\textsuperscript{83} and products liability cases.\textsuperscript{84} By contrast, the proposal would have less impact on carcinogenic activities that result in exposures of relatively minor magnitude, duration, or potency. For example, the evidence that saccharin causes cancer in laboratory animals\textsuperscript{85} would trigger the conclusive presumption of carcinogenicity. Occasional consumption of low-calorie soft drinks, however, would be unlikely to reach the threshold of exposure required to activate the proposal's presumption of causation.\textsuperscript{86} Similarly, the presumption of causation would not be triggered if there were no evidence of the magnitude

\textsuperscript{82} Environmental contamination often goes unnoticed, but when it is discovered its presence and its effects are relatively easy to document. See, e.g., Brown, \textit{Love Canal, U.S.A.}, N.Y. Times. Jan. 21, 1979, \S 6 (Magazine), at 25 (describing contamination by toxic and carcinogenic wastes of Niagara Falls, N.Y., neighborhood). Once occupational hazards are recognized, they can be documented. See, e.g., P. \textsc{Barth} & H. \textsc{Hunt}, supra note 1, at 15-60 (describing incidence of workers' exposure to carcinogenic and toxic substances). But cf. note 55 supra (lack of recordkeeping requirements hinders identification of source of occupational disease).

\textsuperscript{83} Accidents that expose individuals to carcinogenic substances will frequently be sufficiently manifest to alert potential plaintiffs to collect evidence long before symptoms arise. See, e.g., \textit{Experts Monitoring Radiation's Effects}, N.Y. Times. Apr. 22, 1979, \S 1, at 25 (scientists gathering data on effects of radiation leakage at Three Mile Island).

\textsuperscript{84} Although the fungibility of different producers' products often makes it difficult years later to identify the specific manufacturer of an injury-causing product, tort law in some jurisdictions will permit actions against all or most of the members of the relevant industry. See, e.g., \textit{Hall v. E.I. Du Pont de Nemours \& Co.}, 345 F. Supp. 353 (E.D.N.Y. 1972) (allowing suit against all domestic manufacturers of blasting caps by children injured by unidentified caps); Sindell \textit{v. Abbott Laboratories}, 26 Cal. 3d 588, 607 P.2d 924, 163 Cal. Rptr. 132, \textit{cert. denied}, 101 S. Ct. 236 (1980) (plaintiff cancer victim permitted to sue 6 of 200 manufacturers of DES on ground that 6 represented 90\% of market).

\textsuperscript{85} See Coca-Cola Co., \textit{Tab Bottle Label} (Conn. ed. 5-cent refund) ("[T]his product contains saccharin which has been determined to cause cancer in laboratory animals."); S. \textsc{Rep.}, No. 95-535, 95th Cong., 1st Sess. 4-6, \textit{reprinted in} [1977] U.S. \textsc{Code Cong. \& Ad. News} 3921, 3924-26 (summarizing evidence leading to passage of Saccharin Study and Labeling Act, \textsc{Pub. L.} No. 95-203, \textit{§} 4, 91 Stat. 1451 (1977) (codified at 21 U.S.C. \textit{§} 343 (Supp. III 1979)). See also S. \textsc{Erstein, supra note} 6, at 194-95 (results of laboratory tests demonstrate that saccharin causes cancer in animals); Smith, \textit{NAS Saccharin Report Sweetens FDA Position, But Not by Much}, 202 Sci. 852, 852 (1978) (National Academy of Sciences report concludes that saccharin poses potential carcinogenic risk to humans, but fails to resolve political issue of FDA ban on saccharin).

\textsuperscript{86} The potency of saccharin is relatively low compared to that of other carcinogens; therefore greater quantities of saccharin exposure are required to induce comparable numbers of tumors in laboratory animals. See \textit{Maugh, Estimating Potency of Carcinogens Is an Exact Science}, 202 Sci. 38 (1978) (comparing quantities of different carcinogens required to induce cancer in 50\% of animals in bioassay study). Ubiquitous carcinogens of relatively low potency, such as saccharin and other food additives, are more properly the subject of specific deterrence control mechanisms such as regulation or prohibition. See Doniger, \textit{supra note} 1, at 655-57 (per se or no-risk prohibitions are more appropriate regulations for food additives).

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or frequency of exposure,\(^8^7\) or of the fact of exposure.\(^8^8\)

By restricting the availability of the presumptions to cases in which the exposure at least doubled the plaintiff’s risk of contracting cancer, the proposal would prevent over-deterrence of producers by protecting defendants from liability for cancers they probably did not cause.\(^8^9\) At the same time, the presumption would effectively deter defendants whose activities increased the incidence of cancer by a statistically substantial amount. To the extent that producers’ liability in tort approximates the actual social costs of their carcinogenic emissions, those emissions would be reduced to socially optimal levels.

A successfully functioning tort mechanism would also complement the effectiveness of the regulatory and research efforts in the war on cancer. The general deterrence effect of tort liability would encourage carcinogen producers in the private sector to conduct research in cancer prevention as part of their efforts to avoid future liability.\(^9^0\) This would encourage producers to conduct more systematic evaluations of the carcinogenic potential of alternative production processes, as well as general research in the fields of cancer treatment and cure.

A tort remedy would also complement government regulatory efforts by encouraging producers to reduce emissions of carcinogens.\(^9^1\) Economically rational producers would seek to reduce carcinogenic emissions in order to minimize their potential tort liability. Conse-

87. Evidence of exposure to a cataloged substance would trigger the conclusive presumption of carcinogenicity, but not the presumption of causation. Thus, the proposal would have helped the plaintiff in Garner v. Hecla Mining Co., 19 Utah 2d 367, 370-71, 431 P.2d 794, 796-97 (1967), who failed to persuade the court that exposure to uranium causes cancer. It would not have helped the plaintiff in Parker v. Employers Mut. Liab. Ins. Co., 440 S.W.2d 43, 47-48 (Tex. 1969), who persuaded the court that exposure to radioactive materials could cause cancer, but who had no evidence of the extent of exposure.

88. Without evidence of an exposure, there is no way to assign liability to a defendant. Cf. pp. 852-53 supra (general deterrence requires identification of right class of defendants).

89. The threshold requirement allows the proposal to approximate the traditional tort proof standard of “more likely than not.” See note 77 supra (probability theory explanation of threshold requirement). A lower threshold could result in a bandwagon effect: once a particular defendant has been found to be vulnerable, he could find himself sued by many plaintiffs able to demonstrate only a small exposure and whose cancers, therefore, probably were not caused by that defendant. Liability in such cases would unduly burden the target defendant while allowing more likely causes of the cancers to go undeterred.

90. Cf. Spence & Weitzman, supra note 16, at 216 (combination of regulatory and market approaches to emission reduction would provide incentive for technological growth that accommodates pollution abatement objectives as well as economic growth).

91. Thus, a carcinogen producer’s compliance with regulatory provisions should not be a defense in tort actions for cancer. Such a defense would undercut the deterrence function of the tort mechanism, destroying the complementary effect on the regulatory system. Cf. W. Prosser, supra note 28, at 203 (compliance with statute or regulation does not prove lack of negligence).
quently, producers would no longer regard the costs of emission control mandated by government regulation as deadweight losses, and they would comply with the regulations more willingly. As a consequence, the enforcement of government regulations would be more effective and would be achieved at a lower cost to the government.

Conclusion

Existing scientific and regulatory strategies in the war on cancer do not effectively deter the introduction of carcinogens into the environment and do not adequately compensate victims. Courts, applying traditional causation requirements, reject current scientific understanding of the etiology of cancer as proof of legal causation in tort suits for cancer, thereby preventing the tort mechanism from performing its deterrence function. The current limitations of the tort system could be overcome, however, by a shift in the burden of proof on the issue of proximate cause once a plaintiff has demonstrated a threshold exposure to a substance listed in a legislatively enacted catalog of carcinogens. An effective tort system would both compensate victims and deter the production of carcinogens while rendering more effective the other policy approaches to the cancer problem.