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Probability of Causation in Radiation Tort Litigation

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PROBABILITY OF CAUSATION IN RADIATION TORT LITIGATION

> There is found in the Summer a kind of spider called a Tainct, of a red colour, and so little of body that ten of the largest will hardly outway a graine; this by Country people is accounted a deadly poyson unto cowes and horses, who, if they suddenly dye, and swell thereon, ascribe their death hereto, and will commonly say, they have licked a Tainct. Now to satisfie the doubts of men, we have called this tradition unto experiment; we have given hereof unto dogs, chickens, calves and horses, and not in the singular number, yet never could finde the least disturbance ensue. There must be therefore other causes enquired of the sudden death, and swelling of cattell, and perhaps this insect is mistaken, and unjustly accused for some other. . . . Now although the animall may be mistaken and the opinion also false, yet in the ground and reason which makes men most to doubt the verity hereof there may be truth enough, that is the inconsiderable quantity of this insect. For that a poyson cannot destroy in so small a bulke, we have no reason to affirme.
>
> Sir Thomas Browne, Pseudodoxia Epidemica*

I. INTRODUCTION

Radiation[1] can "cause" cancer; radiation can "cure" cancer. Radiologists[2] are confronted with this tragic irony every time they prescribe a course of radiotherapy,[3] or radiation cancer therapy. The benefit to be achieved by radiotherapy is the death of cancer cells; the cost to be

* 1 SIR THOMAS BROWNE'S PSEUDODOXIA EPIDEMICA 282-83 (R. Robbins ed. 1981) (1st ed. 1646).

1. The term "radiation" includes ionizing and non-ionizing radiation. The term as used in this paper refers only to ionizing radiation or "radiation of either a particulate-or a wavelike character that removes charges from, or adds them to, electrically neutral atoms and molecules . . ." Beebe, *Ionizing Radiation and Health*, 70 AM. SCIENTIST 35 (1982).

2. A radiologist is "[o]ne skilled in the diagnostic and/or therapeutic use of x-rays and other forms of radiant energy." STEDMAN'S MEDICAL DICTIONARY 1188 (5th unabr. lawyer's ed. 1982).

3. Radiotherapy is the "medical specialty which relates to the use of electromagnetic or particulate radiations in the treatment of disease." *Id.* The medical treatment of cancer utilizes several forms of radiation: x-radiation, fast neutrons, protons, negative pi-mesons, helium ions and heavy ions. E.J. HALL, RADIOBIOLOGY FOR THE RADIOLOGIST 319-20 (2d ed. 1979).

weighed, the death or carcinogenesis[4] of surrounding normal cells. Exposure to radiation is intrinsically valueless; its value or harm is dependent on which cell it "hits" and how it "hits" it.[5] The radiologist, therefore, must weigh the radiation risk of the neoplastic transformation[6] of healthy cells against the successful diagnosis and treatment of cancer, maximizing radiation's curative effect, minimizing its destructive impact.

Society weighs the risks and benefits of other uses of radiation. The viability of nuclear energy as a source of energy rises or falls with the public's perception of the risks and benefits of its production. Nuclear weapons proliferate with society's fear of hostile attack rather than self-destruction. The disposal of nuclear waste threatens or abates depending on whose backyard becomes a waste disposal site. The uses of radioactive substances are diverse and pervasive—from nuclear weapons and nuclear energy[7] to diagnostic and therapeutic medicine[8] to scientific

4. Carcinogenic is defined as "causing cancer." STEADMAN'S MEDICAL DICTIONARY 223 (5th unabr. lawyer's ed. 1982). Cancer is a "general term frequently used to indicate any of various types of malignant neoplasms, most of which invade surrounding tissues, may metastasize to several sites, and are likely to recur after attempted removal and to cause death of the patient unless adequately treated. . . ." *Id.* at 216.

5. There are two primary models of the inactivation of molecules in a cell by ionizing radiation: the target theory and the diffusion theory. The target theory posits that the radiation directly damages or kills the cell by producing ionizations of the DNA (deoxyribonucleic acids). The diffusion theory addresses the likelihood of the interaction with molecules of water in the cell, as the cell is eighty percent water. According to the diffusion theory, radiation indirectly acts on the DNA of the cell through the formation of highly reactive free radicals H and OH from water. Hutchinson, *Molecular Basis for Action of Ionizing Radiations*, 134 SCIENCE 533 (1961). *See* E.J. HALL, *supra* note 3, at 10-12.

If the radiation "hits" the cell either through direct or indirect action, several possible effects occur: 1) the subcellular damage may be repaired by the cell, 2) the hit(s) could kill the cell, or 3) the damage could result in the carcinogenesis, or neoplastic transformation of the cell. Han, Hill and Elkind, *Repair of Cell Killing and Neoplastic Transformation at Reduced Dose Rates of ^{60}Co γ-Rays*, 40 CANCER RES. 3328 (1980).

6. Neoplasm is defined as:

> an abnormal tissue that grows by cellular proliferation more rapidly than normal and continues to grow after the stimuli that initiated the new growth cease. [Neoplasms] show partial or complete lack of structural organization and functional coordination with the normal tissue, and usually form a distinct mass of tissue which may be either benign (benign *tumor*) or malignant (carcinoma).

STEDMAN'S MEDICAL DICTIONARY 931 (5th unabr. lawyer's ed. 1982).

7. UNITED NATIONS SCIENTIFIC COMMITTEE ON THE EFFECTS OF ATOMIC RADIATION, SOURCES AND EFFECTS OF IONIZING RADIATION at 13-15, U.N. Doc. A/32/40, U.N. Sales No. E.77.IX.1 (1977) [hereinafter UNSCEAR].

8. *Id.* at 15-16. X-ray examinations are perhaps the most common source of non-natural radiation exposure. Though once used less discriminately as a diagnostic tool, x-ray examinations are kept at the minimum required for medical use. *Id.* at 17. "Among diagnostic procedures, the average dose absorbed by the bone marrow varies from about 0.01 rad for a chest x-ray to 0.90 rad for a barium enema . . . ." Beebe, *supra* note 1, at 35. High energy irradiation of cancer tumors through the use of linear accelerators and cobalt-60 units is the major source for radiation cancer therapy. H. JOHNS & J. CUNNINGHAM, THE PHYSICS OF RADIOLOGY 130 (4th ed. 1983).

research, food irradiation, and even smoke alarms and antistatic devices.[9] The efficacy of each use must be measured by the balancing of defined costs and benefits. How the scales tip depends on how the relevant factors are weighed and measured.

In balancing radiation costs and benefits to society, the cost of injury must be recognized and redressed. Cells comprise individuals; individuals, society. Radiation injury to a healthy cell can give rise to a cancer which can destroy the individual. As one of the roles of medicine is to avoid or remedy such an injury to the body, one of the roles of tort law is to prevent or redress possible harm to individual members of society incurred in pursuit of societal goals. Tort law defines and evaluates the costs of various societal aims by recognizing such harms as legally cognizable injuries, compensating the injured from the coffers of the tortfeasor. Such compensation deters the conduct which produces the harm if the costs outweigh the benefits, or spreads the costs among the beneficiaries if the benefits outweigh the costs. This system, however, often fails in radiation tort actions, not because the injury, cancer, is not legally cognizable, but because under traditional tort law the plaintiff is unable to show that radiation has "caused" the cancer.[10] The costs, therefore, are not fully counted in the cost/benefit analysis of radiation use.

The nemesis of legal redress is cause-in-fact, an unlikely source for what in essence acts as a determination of liability based on policy. In any tort the plaintiff must prove that the defendant's act or omission was a "necessary antecedent" or cause-in-fact of the injury.[11] Unlike the policy-laden concept of proximate cause,[12] cause-in-fact is assumed to be a neutral concept.[13] But in effect, it is cause-in-fact that precludes recovery from many radiation tort plaintiffs. Determining that radiation exposure caused any individual cancer is impossible.[14] What can be scientifically

9. UNSCEAR, *supra* note 7, at 13.

10. *See generally* Note, *Tort Actions For Cancer: Deterrence, Compensation, and Environmental Carcinogenesis*, 90 YALE L.J. 840, 846-47 (1981) (discussing the failure of tort law's role of compensation in cancer torts).

11. W. KEETON, PROSSER AND KEETON ON THE LAW OF TORTS § 42, at 272-73 (5th ed. 1984).

12. *Id.* § 41, at 265.

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

14. Biological effects of radiation are divided into two categories according to the dose received: chronic, or low dose effects, and acute, or extremely high dose effects. This paper is concerned only with cancer, a chronic effect which is probabilistic in its occurrence due to its subcellular damage. Legal causation problems do not occur with acute radiation injury. The doses are so high that the effects are fatal, immediate, and apparant at the tissue and organ level.

[T]here are several dose-dependent syndromes that cause fairly rapid mortality. At acute

determined through the use of epidemiological studies is an excess risk of cancer in populations exposed to radiation. Though such evidence is persuasive scientific evidence, it does not meet the requisite "but-for" or "substantial factor" tests of legal cause-in-fact.[15] Consequently, the radiation tort plaintiff is without compensation, and the defendant is without cost. Tort objectives of compensation to injured plaintiffs and deterrence of defendants' conduct are left unrealized. The essential element of injury costs is subtracted from the balancing equation that weighs the costs and benefits of the use of radiation.

The injustice and inequity of this effective bar to legal redress for injury will be examined through the following paradigm:

Dena Smith, a forty-one-year-old mother of two, has lived all her life in Grand Junction, Colorado. Grand Junction is a beautiful town in western Colorado that supported the industries of uranium mining and milling that flourished after the development of the atomic bomb. When Dena was a child, most of the Grand Junction community was in some way associated with the uranium industry. Few if any of the residents believed that there was any risk associated with the radioactive uranium mill tailings that were removed from the mill site and used in construction of buildings and houses throughout Grand Junction.[16] Dena lived in such a house from the ages of ten to twenty-one.

Dena has been diagnosed as having lung cancer. She is a former smoker. She is aware of the increased risk of lung cancer due to exposure to radon, a radioactive gas produced from the radioactive disintegration of radium and uranium in the mill tailings.[17] In fact, due to a governmental clean-up program, the Uranium Mill Tailings Remedial Action

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whole body doses of around 300 to 600 rad, mammals will encounter the hematopoetic syndrome and likely perish within 2 to 4 weeks because of certain blood changes which reduce the body's ability to fight bacterial infections. At doses of around 1000 rad or greater the GI tract becomes ulcerated, and the loss of fluids, electrolytes, and infection generally cause mortality within 1 week or less. Very high doses, say tens of kilorads, cause the hematopoetic and GI syndromes, but these do not have time to develop because mortality occurs too rapidly (< 1 day) owing to central nervous system (CNS) damage.

2 F.W. WHICKER & V. SHULTZ, RADIOECOLOGY: NUCLEAR ENERGY AND THE ENVIRONMENT 125 (1982). *See also* E.J. HALL, *supra* note 3, at 205-15.

15. W. KEETON, *supra* note 11, § 41, at 265-68.

16. U. S. DEP'T OF ENERGY, 1 REMEDIAL ACTIONS AT THE FORMER CLIMAX URANIUM COMPANY URANIUM MILL SITE GRAND JUNCTION, MESA COUNTY, COLORADO: FINAL ENVIRONMENTAL IMPACT STATEMENT 1 (DOE/EIS-0126F, December 1986).

17. Radon-222 is a progeny radionuclide of uranium-238. After uranium ore is mined, a uranium mill extracts uranium oxide from the raw ore so that it can ultimately be used as fuel in nuclear reactors and as the fissionable material necessary in nuclear weapons. The residues of the uranium milling process, called tailings, contain large quantities of radionuclides from the radioactive decay of uranium-238. Because of its gaseous state, Radon-222 is the source of the greatest health risk

Program (UMTRAP),[18] she has been informed that the radon levels in her former house were measured at 0.065 Working Levels (WL).[19] Dena is convinced that her lung cancer has been caused by her exposure to radon gas for eleven years. She therefore brings an action in tort against the uranium mill.

Under traditional tort law, Dena would be precluded from recovery. She is unable to prove that "but-for" her exposure to radon she would not have developed lung cancer. She is unable to show that radiation exposure was "a material element and substantial factor"[20] in bringing about her lung cancer. What Dena can prove through epidemiological evidence is the probability that her exposure to radon gas caused her lung cancer.

The perceived insufficiency of probabilistic proof under traditional tort law belies its notable veracity and dependability in scientific research. Such proof should not be precluded in radiation tort simply because the law rigidly requires only mechanistic proof of "out-of-control-Mack-truck-hits-and-kills-prudent-but-improvident-traveller" causation. Such evaluation of causation is obsolete in a time of complex and unknown disease etiologies and unidentified disease vectors. In order to

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from tailings. Radon, when inhaled, deposits energy from alpha particles in the lung. The resulting absorbed dose to the lung increases the risk of lung cancer. *See id.* at 1. 300,000 tons of uranium mill tailings were removed and used as construction material in approximately 3,500 houses in the vicinity of Grand Junction, Colorado. *Id.*

18. UMTRAP was formed under the aegis of the U.S. Department of Energy to clean up twenty-four former uranium mill sites and any property in the vicinity of the sites which had been contaminated with uranium mill tailings originating from those sites. Uranium Mill Tailings Radiation Control Act of 1978, Pub. L. No. 95-604, 92 Stat. 3021 (codified as amended in scattered sections of 42 U.S.C.).

19. The average indoor radon concentration of the 1065 vicinity properties monitored in Grand Junction, Colorado was 0.065 WL. COLORADO DEPT. OF HEALTH, REMPROG FILE, GRAND JUNCTION REMEDIAL ACTION PROGRAM. The National Research Council's Committee on the Biological Effects on Ionizing Radiations describes one working level as:

> [a]ny combination of ^{218}Po, ^{214}Pb, ^{214}Bi, and ^{214}Po (the short-lived progeny of radon) in 1 liter of air, under ambient temperature and pressure, that results in the ultimate emission of 1.3×10^5 MeV of alpha-particle energy. This is about the total amount of energy released over a long period of time by the short-lived daughters in equilibrium with 100 pCi of radon. . . .
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> . . . The working-level month (WLM) was introduced so that both the duration and level of exposure could be taken into account. The WLM is defined as the sum of the products of the WL times the duration of exposure during some specified total period. The unit WLM is equal to 170WL hours which corresponds to an exposure of 1WL for 170h (approximately 1 working month).

COMMITTEE ON THE BIOLOGICAL EFFECTS OF IONIZING RADIATIONS, NATIONAL ACADEMY OF SCIENCES, HEALTH RISKS OF RADON AND OTHER INTERNALLY DEPOSITED ALPHA-EMITTERS (BEIR IV) 140 (1988) [hereinafter BEIR IV].

20. W. KEETON, *supra* note 11, § 41, at 267.

realize the goals of tort law in modern society, a new test of causation should be adopted which would align legal cause-in-fact with scientific probability of causation. Under such a test, the radiation tort plaintiff would have to show a "causal linkage"[21] between radiation and a later cancer by proving by a preponderance of the evidence the probability that exposure(s) to radiation "caused" the cancer. The probability of causation should be estimated from the best scientific knowledge available from epidemiological studies of radiation-exposed populations. Upon proving the other prima facie elements of the tort by a preponderance of the evidence, the radiation tort plaintiff would then recover damages in proportion to the probability of causation proved. This would eliminate the unjust exclusion of plaintiffs harmed by radiation exposure and allow the actual costs of radiation use to be recognized and redressed.

II. SCIENTIFIC DETERMINATION OF RADIOGENIC CANCER

A. *Indeterminancy of Individual Cancer Causation*

Probability is interjected into the study of cancer caused by exposure to radiation at its most elemental level—radioactivity[22] itself. Radioactivity is the result of a process in which particles from unstable nuclei gain enough energy from chance collisions within the nuclei to escape the nuclear binding force.[23] In the disintegration process, energetic particles such as protons, neutrons, and alpha particles, and electromagnetic radiation, such as x-rays and gamma rays, can be emitted.[24] Escape of a nuclear particle is pure chance. Therefore, it is impossible to determine whether any particular nucleus will disintegrate.[25] All that can be determined is the probability that in a population of unstable nuclei a certain number will disintegrate in a given time.[26]

Further uncertainty results from the interaction of radiation with matter. The particles or rays interact with matter by the transfer of their escaped energy to atoms or molecules randomly encountered, and the

21. Calabresi, *supra* note 13, at 71.

22. This discussion concerns natural and not artificial radioactivity. Artificial radioactivity occurs in the decay of an unstable nuclide formed by the bombardment of stable nuclide with neutrons, high energy protons, deuterons, gamma rays or alpha particles. H. JOHNS & J. CUNNINGHAM, *supra* note 8, at 71.

23. H. JOHNS & J. CUNNINGHAM, *supra* note 8, at 71.

24. H. JOHNS & J. CUNNINGHAM, *supra* note 8, at 71.

25. H. JOHNS & J. CUNNINGHAM, *supra* note 8, at 71.

26. H. JOHNS & J. CUNNINGHAM, *supra* note 8, at 71.

formation of ions through the addition or removal of electrons;[27] hence, the name "ionizing radiation." Although the exact mechanism of human cancer induction by radiation is unknown, researchers assume that it is dependent on the absorbed dose—the mean energy absorbed per unit mass of tissue (expressed in rads).[28] A random "hit" of absorbed energy in a target cell can result in a range of effects, including neoplastic transformation (carcinogenesis).[29] Studies in cancer biology have hypothesized that carcinogenesis occurs when the energy absorbed by cells through the proper hit(s) alters the genetic apparatus of the cell.[30] One proposed mechanism of cell transformation posits that such hits could possibly activate oncogenes which in turn transform the genes of the cells and act as initiators or promotors or both in the production of cancer cells.[31] The process through which radiation not only "scores a hit" in a particular cell, but transforms the cell into a cancer cell is stochastic (a random process which can only be measured statistically). The probability that the neoplastic transformation of a cell will occur, therefore, can only be determined through the study of populations of cells.[32]

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27. H. JOHNS & J. CUNNINGHAM, *supra* note 8, at 134.

28. NAT'L INST. OF HEALTH, U.S. DEP'T OF HEALTH AND HUMAN SERVICES, PUB. NO. 85-2748, REPORT OF THE NATIONAL INSTITUTES OF HEALTH AD HOC WORKING GROUP TO DEVELOP RADIOEPIDEMIOLOGICAL TABLES 13 (1985) [hereinafter NIH TABLES].

> In general, the relative biological effectiveness (RBE) of a given absorbed dose of charged particle radiation depends on the spatial density of the ionizations (linear energy transfer, or LET) produced along the tracks of the radiation, the heavy particle radiations tending to produce very closely spaced ionizations (high LET), while electrons, x-rays and gamma rays tend to have fewer ionizations per unit length of track (low LET).

Id. at 14.

29. *Id.* at 13. Bond, *The Need for Probabilities in Cancer Litigation*, NUCLEAR NEWS, Aug. 1986, at 63. *See supra* note 5 and accompanying text.

30. NIH TABLES, *supra* note 28, at 13.

31. NIH TABLES, *supra* note 28, at 6. Researchers have postulated three successive stages of tumor development: initiation, promotion, and progression.

> During the initiation phase, the DNA of the target cell, which contains the genetic code, is presumed to be damaged or structurally altered as the result of exposure to radiation. . . .
>
> The promotional phase of cancer development is concerned with the subsequent changes in the initiated cell that lead to development of an overt tumor. . . . While a single exposure to an initiating substance can suffice to alter DNA, promoting effects typically are induced only by prolonged contact with the agent in question. . . . The progressive phase involves the outgrowth of progressively more malignant variants of the original neoplasm.

NIH TABLES, *supra* note 28, at 5.

32. Bond, *supra* note 29, at 63. Introducing the Radiogenic Cancer Compensation Act, Senator Hatch stated:

> A given charged particle's scoring just the right hit to initiate a cancer followed by the proper conditions for the development of that cancer are rare and substantially random events. However, the probability of these events occurring increases as the radiation dose increases, since more shots are being fired at the cells—so to speak. The process is stochastic, i.e. it is a random process that can be measured only statistically.

S. 921, 98th Cong., 1st Sess., 129 CONG. REC. S3921 (daily ed. Mar. 24, 1983) (Questions and answers entered into the record at the request of Sen. Hatch).

Whatever the etiology of radiation-induced cancer,[33] the effects are stochastic, subcellular, and unobservable. Though scientists hypothesize that various environmental factors act as initiators and promotors,[34] which carcinogen caused a particular cancer is unknown. It is impossible, therefore, to determine whether a particular cancer in a specific individual was caused by radiation exposure. Cancers which occur in individuals unexposed to radiation are indistinguishable from radiogenic, or radiation-induced, cancers.[35] The indeterminancy of cancer causation in the individual, consequently, mandates the use of population statistics in order to ascertain the probability that radiation exposure "caused" a particular cancer.

B. *Scientific Determination of the Probability of Causation*

Just as it is pure chance if radiation interacts with a particular cell and transforms it into a cancer cell, radiogenic cancer is similarly random with respect to the individual in which it may occur.[36] The carcinogenic effect of radiation can only be detected as randomly occurring excess cancers within populations exposed to radiation. The science of radioepidemiology determines this "prevalence and incidence" of radiogenic cancer[37] by comparing the number of cancers in unexposed and exposed populations. An excess number of cancers within an exposed population is evidence that radiation exposure is a determinant of cancer. An inference of causation, however, is not based upon a statistical association between radiation exposure and increased cancer incidence alone. Radioepidemiologists deem a statistical association between radiation exposure and cancer to be a causal association only if it meets certain, further epidemiological requirements: i.e., statistical significance, specificity,

33. "The etiology of a disease may be thought of as having a sequence consisting of two parts: (1) causal events occurring prior to some initial bodily response, and (2) mechanisms within the body leading from the initial response to the characteristic manifestations of the disease." B. MACMAHON & T. PUGH, EPIDEMIOLOGY 26 (1970).

34. Epidemiologists have inferred that 75-80% of fatal cancers in the United States are initiated or promoted by environmental factors, such as smoking, alcohol consumption, diet, pollution, occupational exposures, medical therapy and diagnosis, and viral infections. NIH TABLES, *supra* note 28, at 6-10.

35. NIH TABLES, *supra* note 28, at 17. Bond, *supra* note 29, at 62-63.

36. Bond, *supra* note 29, at 63. Catlin, *Determining Probability of Causation*, NUCLEAR NEWS, June 1986, at 73.

37. S. 921, 98th Cong., 1st Sess., 129 CONG. REC. S3922 (daily ed. Mar. 24, 1983) (Questions and answers entered into the record at the request of Sen. Hatch). Epidemiology in general is "the study of the *distribution* of disease and the search for the *determinants* of the observed distribution." B. MACMAHON & T. PUGH, *supra* note 33, at 1.

dose response, and consistency.[38]

No other cancer determinant has been studied as intensively as ionizing radiation.[39] Epidemiological studies of the atomic bomb survivors of Hiroshima and Nagasaki,[40] radium dial painters,[41] British spinal arthritis patients,[42] uranium miners,[43] the natives of the Marshall Islands,[44]

38. Statistical significance aids the radioepidemiologist in determining whether the observed difference in cancer incidence between the exposed and unexposed populations is a true difference or is the result of the sampling process. The radioepidemiologist will usually state the "null hypothesis" that there is no real difference in cancer incidence between the two populations. The statistical significance test computes the probability that the observed difference in cancer incidence is due to chance if the null hypothesis is, in fact, true. If the probability is small (5% is the common probability value), then the radioepidemiologist rejects the null hypothesis and concludes that the observed difference is a true difference. *See generally* J. FLEISS, STATISTICAL METHODS FOR RATES AND PROPORTIONS 33-49 (2d ed. 1981). Statistical significance "depends on the total number of cancer cases observed and the apparent size of the excess relative to the baseline risk." NIH TABLES, *supra* note 28, at 16. The specificity of the causal association is the measure of the probability that the excess cancer in the exposed population is the result of some factor other than radiation. The smaller the probability, the more specific the causal association between radiation exposure and cancer. NIH TABLES, *supra* note 28, at 16.

If there is a causal association, then cancer incidence should increase with increasing dose. While radioepidemiological studies have found a linear dose-response for high doses of radiation, excess cancer cannot be observed in exposed populations at low doses. Because cancers can arise from the random transformation of a single cell, it is presumed that there is no threshold dose below which radiation exposure is safe. The risk from lower-dose exposure, therefore, must be extrapolated from the linear, high dose-response relationship. This requires the use of a mathematical model. The model generally accepted by radiobiologists for low-LET radiation is the linear-quadratic model.

> The model assumes that two ionizing events are more likely to produce a biological effect if they occur very close together than if they are separated; because ionizing events tend to be widely spaced along low-LET tracks, closely-spaced events are likely to be at the intersections of different tracks and their probabilities are approximately proportional to the square of dose.

NIH TABLES, *supra* note 28, at 24. "The above considerations do not apply to high-LET radiation, for which close spacing of the ionizing events along the radiation tracks is the rule, rather than the exception. Both theoretical and experimental studies suggest that, for high-LET radiation, the dose-response tends to be linear. . . ." NIH TABLES, *supra* note 28, at 25.

Consistency in the associations observed between radiation exposure and increased cancer incidence from the study of different exposed populations adds credence to the inference that the statistical association is a causal association. More credence is placed "in associations that turn up frequently and under diverse circumstances of exposure," than in "isolated reports not verified by other experience." NIH TABLES, *supra* note 28, at 16-17.

39. Catlin, *supra* note 36, at 74.

40. NIH TABLES, *supra* note 28, at 15.

41. Radioepidemiological studies of workers who ingested radium while painting instrument dials with radium paint showed an increase in cancer incidence due to internal dose from alpha-particle radiation. NIH TABLES, *supra* note 28, at 15.

42. British patients who suffered from ankylosing spondylitis, a spinal arthritis, were treated with a course of x-ray therapy. An increase in cancer was noted in the group of patients. Beebe, *supra* note 1, at 37.

43. A causal association between lung cancer and the inhalation of radon has been observed in the radioepidemiological studies of uranium miners. NIH TABLES, *supra* note 28, at 15.

44. The Marshallese were exposed to radioactive fallout from nuclear weapons testing in the Pacific in 1954. NIH TABLES, *supra* note 28, at 15.

hyperthyroid patients,[45] and most significantly, patients exposed to therapeutic x-radiation[46] have well-documented the causal association between radiation and cancer. The data and conclusions from these radioepidemiological studies have been reviewed and analyzed by such eminent scientific advisory committees as the National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation, the National Council on Radiation Protection and Measurements Committee, and the United Nations Scientific Committee on the Effects of Atomic Radiation. These committees, through rigorous scientific evaluation of radioepidemiological studies, assess which dose-response[47] and risk projection model[48] best fit the data and determine the risk of cancer associated with a given dose of radiation.

The National Institute of Health working group relied upon these radioepidemiological studies for the production of probability of causation tables. The Orphan Drug Act, passed by Congress and enacted on January 4, 1983, required the Secretary of Health and Human Services to produce "radioepidemiological tables that estimate the likelihood that persons who have or have had any of the radiation related cancers and who have received specific doses prior to the onset of such disease developed cancer as a result of these doses."[49] The result of this legislation was the compilation of the best epidemiological estimates of cancer risk from radiation exposure[50] and the publication of these risk coefficients in

45. "The largest number of persons studied who were exposed to low-LET radiation at low dose and dose rate were patients who received oral iodine-131 for the treatment of hypothyroidism." NIH TABLES, *supra* note 28, at 15 (footnote omitted).

46. *See supra* note 8 and accompanying text.

47. *See supra* note 38 and accompanying text.

48. The expression of cancer generally requires a long latency period. Because most radiogenic cancers occur throughout a person's lifetime, none of the radiation exposed groups studied have been observed long enough to determine the full effects of their exposures. The result of this is that a risk projection model must be selected in order to assess the lifetime cancer risk due to radiation. The risk projection model can, therefore, estimate the risk for a longer period of time than currently available observation data will allow.

The two primary models used for lifetime projection of cancer risk are the absolute-risk model and the relative-risk model. The absolute-risk model projects the average number of excess cancer cases per unit of population, time, and dose. It is additive in that it adds the same average number or a constant incidence of excess cancer deaths to the natural incidence of cancer death across time. The relative-risk model projects the currently observed average percentage increase in cancer risk per unit dose into future years. It is multiplicative in that it multiplies the natural age-specific cancer risk by the percent increase due to radiation exposure. Because the natural incidence of most cancers increases with age, the relative-risk model projects greater lifetime risk from radiation exposure than that projected by the absolute-risk model. NIH TABLES, *supra* note 28, at 19-23.

49. Act of Jan. 4, 1983, Pub. L. No. 97-414, § 7(b)(1), 96 Stat. 2059, 2060.

50. "Forty of the 78 risk coefficients in the working group report were obtained from the 1980 report of the third National Academy of Sciences Committee on Biological Effects of Ionizing Radiation (BEIR III); an additional 38 coefficients were recalculated from 1984 data for lung, breast,

the Report of the National Institutes of Health Ad Hoc Working Group to Develop Radioepidemiological Tables (NIH tables).[51]

The primary assumption upon which the tables are based is that the probability of radiation exposure causing any individual cancer can be determined from the probability of increased cancer incidence within exposed populations. In other words, the NIH tables recognize that the only determination of a potential cause of a stochastic disease such as cancer is that observed through the study of populations exposed to the suspected carcinogen. The NIH tables express this increased risk as the probability of causation.

Probability of causation is defined as the cancer risk attributable to radiation divided by the total cancer risk from all causes.[52] It is estimated as the risk that radiation caused a particular cancer divided by the sum of the radiation risk and the background or baseline risk of the particular cancer.[53] Given that relative risk is equal to the radiation risk divided by the background risk, the probability of causation is equal to the relative risk divided by (1 + relative risk).[54] Consequently, probability of causation estimates are dependent on the radioepidemiological determination of relative risk, which in turn, is derived from population statistics.[55] For example, if a population of 10,000 exposed to a radiation dose of 15 rads manifests an incidence of 440 cancers, while a similar, but unexposed population of 10,000 shows only 400 cancers, the probability of causation would be the relative risk, 40/400, divided by (1 + 40/400), or 9%. Given that an individual is representative of the reference populations from which the radioepidemiological risk is derived, the probability that an individual exposed to 15 rads, who later develops cancer, is 9% that the cancer was caused by the radiation exposure.

By reference to the NIH tables, it is therefore possible to determine the scientific probability that an individual's cancer was "caused" by exposure to radiation. The probability of causation estimated from the NIH tables is proportional to the radiation dose to the individual organ

thyroid, and salivary gland cancer." Jacobson, *Radioepidemiological Tables*, 257 J.A.M.A. 806, 808 (1987) (footnote omitted).

51. *See supra* note 28.

52. Jacobson, *supra* note 50, at 807.

53. Jacobson, *supra* note 50, at 807. Bond, *supra* note 29, at 64. The baseline or background risk of particular cancers were taken from the Surveillance, Epidemiology, and End Results Program. *See* NAT'L CANCER INST., NCI MONOGR. 57, SURVEILLANCE, EPIDEMIOLOGY, AND END RESULTS. INCIDENCE AND MORTALITY DATA: 1973-1977 (1981).

54. Jacobson, *supra* note 50, at 807.

55. Catlin, *supra* note 36, at 73.

or tissue and its associated risk coefficient, and inversely proportional to the background probability of developing the same type of cancer.[56] The NIH risk coefficients are a function of radiation dose to the relevant tissue, the time between exposure and diagnosis,[57] sex,[58] age at exposure,[59] and for some cancers, age at diagnosis.[60] Because the radiation dose and the type of cancer are specific to the individual, and the risk coefficient and background cancer risk are specific to "that person's age, sex, and type of tumor," the population statistics used in the probability of causation calculation are "as closely matched as possible to the person of interest."[61]

The probability that radiation exposure caused an individual cancer can be illustrated through the paradigm found in the introduction. Given Dena Smith's exposure to 0.065 Working Levels[62] for 11 years, and assuming a 75% occupancy of her house each day, 365 days a year, as well as a multiplicative interaction between radiation and smoking,[63] the relevant risk coefficient from the NIH tables[64] would estimate a 25% probability that her exposure to radon gas caused her lung cancer.[65] The scientific determination of causation, therefore, is that there is a 25%

56. Bond, *supra* note 29, at 64.

57. NIH TABLES, *supra* note 28, at 19-23.

58. NIH TABLES, *supra* note 28, at 17.

59. NIH TABLES, *supra* note 28, at 18-19.

60. NIH TABLES, *supra* note 28, at 57-60.

61. Bond, *supra* note 29, at 64.

62. *See supra* note 19 and accompanying text.

63. NIH TABLES, *supra* note 28, at 48-50. "Smoking of tobacco, particularly in the form of cigarettes, is generally recognized as the single most important external risk factor for human cancer, being estimated to cause 25-40% of all cancer deaths in the U.S. ." NIH TABLES, *supra* note 28, at 7.

The interaction between smoking and radiation in carcinogenesis is determined to be either additive or multiplicative. If the total risk from a radiation exposure and smoking is assumed to be "the sum of the excess risks from each of the two taken separately," the two factors interact additively. NIH TABLES, *supra* note 28, at 47. If the relative risk due to radiation exposure and smoking is "the product of the relative risks of the two factors taken separately," the two factors interact multiplicatively. NIH TABLES, *supra* note 28, at 48.

If the multiplicative interaction model holds, then the probability of causation for radiation exposure does not vary according to the smoking history. This model appears to fit uranium miner studies "as between exposure to radon measured in cumulative Working Level Months and cigarette smoking measured as accumulated pack-years." NIH TABLES, *supra* note 28, at 48.

64. The relevant risk coefficient is 1.2 percent per WLM. NIH TABLES, *supra* note 28, at 233. The BEIR IV committee has subsequently estimated the risk coefficient as 1.5 percent per WLM based on further evaluation of radioepidemiological studies of uranium miners. BEIR IV, *supra* note 19, at 40.

65. The relative risk is calculated as (.75) (.065 WL) (WLM/170 WL hr) (24 hr/d)(365 d/yr) (11 yr) (1.2%/WLM) = 0.33. The probability of causation equals the relative risk divided by (1 + relative risk), or 0.33 / 1.33 = 0.25 or 25%.

probability that Dena's lung cancer was caused by the radon gas exposure.

III. LEGAL DETERMINATION OF CAUSE-IN-FACT

Whatever tort theory Dena Smith decides to present in a court of law—negligence, strict liability, or product liability—she must prove that the radiation exposure was more likely than not the legal cause of her lung cancer. Legal or "proximate" cause differs from scientific cause in that it is a policy determination of whether the cause of the injury is one upon which the society wishes to impose liability.[66] Proximate cause seeks to limit legal responsibility "to those causes which are so closely connected with the result and of such significance that the law is justified in imposing liability."[67] An element of proximate cause is cause-in-fact. Cause-in-fact is a "necessary antecedent" causally connected to the injury.[68] Before any explicit limitations on liability may be imposed under proximate cause, the plaintiff must first prove that the radiation was in fact a cause of the injury.

The problem in proving causation in radiation tort cases is not any policy-imposed limitation on liability under proximate cause. In fact, the foreseeability and significance of injury due to exposure to radiation is certainly recognized, most obviously in the determination of nuclear energy as "abnormally dangerous"[69] and subject to strict liability in tort.[70] The problem with proving legal causation is in proving that the radiation exposure was in fact the cause of the injury.

Dena Smith has an impossible burden to prove. The best scientific proof of causation that she can offer is insufficient proof of cause-in-fact: 1) It fails because the radiation dose cannot pass the "but-for" and "substantial factor" tests of cause-in-fact of the cancer; and 2) Even if the evidence of probability of causation is deemed sufficient evidence of cause-in-fact, she is unable to prove that the radiation dose more likely than not (greater than 50% probability) caused the lung cancer. Cause-

66. W. KEETON, *supra* note 11, § 41, at 264.

67. W. KEETON, *supra* note 11, § 41, at 264.

68. W. KEETON, *supra* note 11, § 41, at 265.

69. W. KEETON, *supra* note 11, § 78, at 558-59.

70. This is not to say that there are no statutory limitations on liability in the event of a nuclear accident. The Price-Anderson Act, 42 U.S.C. § 2210 (1975), was passed by Congress in 1957 "in order to protect the public and to encourage the development of the atomic energy industry." 42 U.S.C. § 2012(i) (1982). Nuclear energy development is encouraged by limiting aggregate liability of the licensee in the event of the accident to $560 million. 42 U.S.C. § 2210(e) (1982).

in-fact, the "neutral" element of causation, in effect precludes recovery for many, if not all, radiation tort plaintiffs.

A. *The Inadequacy of the But-For and Substantial Factor Tests*

The two tests which determine cause-in-fact are the "but-for" and "substantial factor" tests. The "but-for" test identifies an act or omission as the cause of the event "if the event would not have occurred but for that conduct."[71] The "substantial factor" test allows the inclusion of situations in which two or more causes concur to give rise to an event. Under this test, an act or omission "is a cause of the event if it was a material element and a substantial factor in bringing it about."[72] In effect, the substantial factor test is an extension of the "but-for" test in that without two or more identified causes acting together, the event would not occur. The scientific evidence of radiation exposure as a cause of cancer would not meet the requirements of either test.

Dena Smith would be unable to show that but for her exposure to radon gas, she would not have developed lung cancer. Because there is no way to distinguish a radiation-induced cancer from other cancers, she could not introduce medical proof that her lung cancer was caused by the radon exposure. The clinical and laboratory tests available to physicians can only specify the type of tumor and the amount of damage produced by the tumor to normal cells.[73] There are no markers in the tumor cells which would distinguish them as induced by radiation rather than some other causative agent. "The proof of causation sought by the legal system through this traditional 'medical expert approach' is thus rendered impossible."[74]

Dena Smith would also fail in demonstrating that radon exposure was a substantial factor in causing her lung cancer. The "substantial factor" test allows for two or more causes, but not two or more independent causes.[75] Though radiation exposure alone is sufficient to

71. W. KEETON, *supra* note 11, § 41, at 266.

72. W. KEETON, *supra* note 11, § 41, at 267.

73. Bond, *supra* note 29, at 63. Bond states that clinical tests are only valuable for determination of causation of disease "due to the dysfunction of entire organs or tissues" because "many of these procedures also yield clues to or are specific for the causative agent." Bond, *supra* note 29, at 63. This is not the case when dealing with disease caused by the transformation of a single cell. "No trace of causative agent remains in the daughter cells that constitute the tumor." Bond, *supra* note 29, at 63.

74. Bond, *supra* note 29, at 62.

75. W. KEETON, *supra* note 11, § 41, at 266. *See also* Delgado, *Beyond* Sindell: *Relaxation of Cause-In-Fact Rules for Indeterminate Plaintiffs*, 70 CALIF. L. REV. 881, 887 (1982); Comment,

cause cancer, radiation can join with other factors to complete the malignant transformation of a cell.[76] In such case, the interaction between radiation and another factor would be synergistic, or dependent.[77] If all other possible independent "causes" of the cancer were eliminated, then each of the two dependent factors, if a material element in bringing about the cancer, would be a substantial factor and would meet the test for cause-in-fact.[78] For instance, if Dena could show that the interaction of radiation exposure and smoking caused her lung cancer, radiation could still meet the substantial factor test, even though the exposure or smoking alone might have been sufficient to cause the cancer. The problem is that Dena is unable to prove what has in fact caused her cancer. Given the natural or background occurrence of lung cancer, Dena would be unable to show that but for her exposure to radiation alone or combined with smoking, she would not have developed lung cancer. The substantial factor test as applied to tort claims of radiation-induced cancer produces the same legal conclusion as the but-for test.

B. *Proof of Causation*

Because Dena Smith would not be able to show direct cause-in-fact under the "but-for" and "substantial factor" tests, she would have to present circumstantial proof that her exposure to radiation was more likely than not the cause-in-fact of her lung cancer.[79] The obstacles inherent in demonstrating such proof lie in 1) the use of epidemiology as evidence of causation, 2) the long latency periods for cancer induction, and 3) the difficulty of showing a greater than fifty percent chance that radiation caused the cancer.

Because all radioepidemiological proof of causation relies on a statistical association between radiation exposure and cancer, most criticism of this proof is directed at its statistical base. Traditionally, the courts

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Epidemiologic Proof of Probability: Implementing the Proportional Recovery Approach in Toxic Exposure Torts, 89 DICK. L. REV. 233, 242 (1984).

76. NIH TABLES, *supra* note 28, at 6. Radiation can act as an initiator or a promoter or both. It can therefore join with other carcinogens as an initiator or promoter and contribute to the development of a cancer cell. Or radiation can act as a complete carcinogen, fulfilling both functions of initiation and promotion. NIH TABLES, *supra* note 28, at 6.

77. *See supra* note 63 and accompanying text.

78. W. KEETON, *supra* note 11, § 41, at 267.

79. W. KEETON, *supra* note 11, § 41, at 269. "A mere possibility of such causation is not enough; and when the matter remains one of pure speculation or conjecture, or the probabilities are at best evenly balanced, it becomes the duty of the court to direct a verdict for the defendant." W. KEETON, *supra* note 11, § 41, at 269 (footnotes omitted).

treated statistical evidence as inadmissible hearsay.[80] Although it is now admitted as a hearsay exception, in many cases judges have not treated statistical "evidence with much more respect than if they had merely excluded it on the grounds of hearsay."[81]

The primary argument against the use of epidemiology is its use of population statistics to prove cause in an individual case.[82] It is argued that the only relevance epidemiological studies have to causation of the individual's cancer is a showing of increased risk of cancer, not actual cause of the individual's cancer.[83] Furthermore, only the subjective judgments of epidemiologists determine if a statistical association is a causal association and if the findings from a study population can be extrapolated to other populations.[84] Though such arguments can be countered by ensuring that the individual is representative of the studied population and that appropriate attention is given to the characteristics of the population studied,[85] several judges and commentators argue that an inference

80. P. RHEINGOLD, N. LANDAU & M. CANAVAN, TOXIC TORTS 403 (1977).

81. *Id.* at 410. *See also* McGovern, *Toxic Substances Litigation in the Fourth Circuit*, 16 U. RICH. L. REV. 247, 296-98 (1983). McGovern states:

> Although some attorneys have been successful in introducing epidemiological and other statistical studies into evidence, they must, however, overcome arguments suggesting that these studies are unreliable, irrelevant, unnecessary, hearsay and not subject to cross-examination. . . .
>
> . . . Defendants have argued that this type of testimony is inherently prejudicial because the defense cannot cross-examine the witness upon the controls, assumptions, soft variables, validity and other factors inherent in the studies that form the basis of the expert opinion.

Id. (footnotes omitted).

82. Dore, *A Commentary on the Use of Epidemiological Evidence in Demonstrating Cause-In-Fact*, 7 HARV. ENVTL. L. REV. 429, 433-35 (1983). "[E]pidemiology cannot determine which particular factor caused a particular person's disease, but only what factors are statistically associated with the occurrence of disease in groups of people." *Id.* at 433 (footnote omitted). *See* Catlin, *supra* note 36, at 73.

83. Dore, *supra* note 82, at 436. McElveen & Eddy, *Cancer and Toxic Substances: The Problem of Causation and the Use of Epidemiology*, 33 CLEV. ST. L. REV. 29, 60 (1984-85).

84. Dore, *supra* note 82, at 432-33. "Epidemiologists must make subjective judgments in deciding whether these biases are significant enough to preclude valid inferences to other populations." Dore, *supra* note 82, at 433 (footnote omitted). *See supra* note 38 and accompanying text. *See also* NIH TABLES, *supra* note 28, at 27-29.

85. Hall & Silbergeld, *Reappraising Epidemiology: A Response to Mr. Dore*, 7 HARV. ENVTL. L. REV. 441 (1983). The authors argue:

> Mr. Dore seems to have two problems with epidemiological evidence: (1) its application to individual cases, . . . and (2) its extrapolation from a restricted group to the population as a whole, which is primarily a scientific issue. Epidemiology by its nature attempts to design studies on representative populations. In some cases, these may represent unusual subgroups in the U.S. population, and Mr. Dore's restrictions on extrapolation are then appropriate. However, extrapolation from the groups studied to another larger group or to the U.S. population as a whole is valid when appropriate attention is given to characteristics of the population initially studied. The criteria and procedures for making such extrapolations are part of an epidemiologist's professional training.

Id. at 442. (footnotes omitted).

of causation based on population statistics is not a specific conclusion of causation in the individual.[86]

Another inherent difficulty in proving that radiation exposure has caused a specific cancer is the long latency period of cancer induction. The legal system incorporates the popular perception that an effect follows an identified cause close in time. Radioepidemiologists, conversely, determine the plausibility of a causal association between a radiation exposure and cancer according to whether the time from exposure to diagnosis coincides with the long latency periods observed in studies of exposed populations.[87] The minimum latency period for a radiogenic cancer is two-to-four years for bone cancer and leukemia, and ten or more years for other cancers.[88] These long latency periods undermine legal determination of causation by increasing the possibility of intervening causes,[89] while supporting scientific assessment of causation by reflecting appropriate temporal factors. The amount of time necessary for a tumor to reach the detectable size of millions of cells after an often protracted promotion stage in carcinogenesis[90] is frequently ignored as a relevant factor in a legal determination of cause and effect.

Finally, the plaintiff must prove that the radiation exposure was more likely than not the cause of the plaintiff's cancer.[91] This is generally interpreted as showing a probability greater than fifty percent.[92] As

86. Robinson v. U.S., 533 F. Supp. 320, 326 (E.D. Mich. 1982) ("[T]here is no epidemiological or biostatistical method which definitely establishes whether an individual case of GBS is caused by the individual's receipt of the swine flu vaccine or by other factors."); Employers Mut. Liab. Ins. Co. v. Parker, 418 S.W. 2d 570, 574 (Tex. Civ. App. 1967), *aff'd*, 440 S.W.2d 43 (1969) ("[A]n inference of causal connection can be no more than speculation and conjecture."); Garner v. Hecla Mining Co., 19 Utah 2d 367, 370, 431 P.2d 794, 796 (1967); Dickson, *Medical Causation by Statistics*, 17 FORUM 792, 801-05 (1982); Dore, *supra* note 82, at 433.

87. NIH TABLES, *supra* note 28, at 19-23.

88. Beebe, *supra* note 1, at 39.

89. Allen v. United States, 588 F. Supp. 247 (D. Utah 1984), *rev'd*, 816 F.2d 1417 (10th Cir. 1987) (on grounds of sovereign immunity), *cert. denied*, 108 S. Ct. 694 (1988). The court stated:

> The great length of time involved (*e.g.*, A irradiates B, who develops a tumor 22 years later) allows the possible involvement of "intervening causes," sources of injury wholly apart from the defendant's activities, which obscure the factual connection between the plaintiff's injury and the defendant's purportedly wrongful conduct. The mere passage of time is sufficient to raise doubts about "cause" in the minds of a legal system accustomed to far more immediate chains of events.

Id. at 406. *See* O'Toole, *Radiation, Causation, and Compensation*, 54 GEO. L. J. 751, 765 (1966).

90. NIH TABLES, *supra* note 28, at 19-23.

91. W. KEETON, *supra* note 11, § 41, at 269. *See* Delgado, *supra* note 75, at 887.

92. Rosenberg, *The Causal Connection in Mass Exposure Cases: A 'Public Law' Vision of the Tort System*, 97 HARV. L. REV. 851, 857 (1984); Black & Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation*, 52 FORDHAM L. REV. 732, 767 (1984). *Contra* Note, *Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence*, 96 YALE L.J. 376, 380-86 (1986). Author Steve Gold points out that the burden of proof consists of a factual burden and the amount of credence given that fact. When probabilistic evidence of causation is introduced, courts

in the case of Dena Smith, such proof is unlikely for most radiation tort plaintiffs. In order to show a probability of causation greater than fifty percent, the plaintiff would have to prove a statistical increase greater than one hundred percent of the natural incidence of cancer.[93] Given the high natural or background occurrence of cancer, this is very difficult to prove. The plaintiff would have to prove exposure to a very high radiation dose to meet a probability greater than fifty percent.[94] As there is no evidence of a threshold level below which radiation exposure is not potentially carcinogenic, many plaintiffs with probability of causation estimates below fifty percent will have cancers caused by radiation exposure, yet will lack the requisite proof.[95]

C. *Two Cases Representing Causation Problem*

In *Garner v. Hecla Mining Co.*,[96] the Utah Supreme Court rejected the use of radioepidemiological evidence of higher lung cancer incidence among uranium miners as proof that Garner, a uranium miner for over twenty years, developed lung cancer due to his work in the mines. The plaintiff presented additional evidence that Garner had thirty-four times as much lead-210[97] in his bones as normal and that the Hecla mine contained 2.5 times as much radon gas concentration as recommended by then existing federal government standards.[98] In addition, the plaintiff presented an expert witness who testified that "there was a very high

generally collapse the fact probability with the belief probability. Courts then determine that the fact probability (probability of causation) rather than the belief probability must be greater than fifty percent. "Where a traditional court would have sought a >50% belief in a yes-or-no fact, a 'collapsing' court seeks a yes-or-no belief in a >50% fact probability." *Id.* at 388.

93. *Allen*, 588 F. Supp. at 418. "The mechanical application of a 'greater-than-100%-increase' test in this context represents merely the refabrication of the 'but-for' test of causation in mathematical form: but for defendant's 50 plus percent share of the statistically identified injuries, plaintiff would probably not have been hurt." *Id.*

94. *See* S. 921, 98th Cong., 1st Sess., 129 CONG. REC. S3921 (daily ed. Mar. 24, 1983) (Questions and answeres enterred into the record at the request of Sen. Hatch).

95. It is also possible that plaintiffs with probability of causation estimates greater than fifty percent, though having requisite proof, will have cancers not caused by radiation exposure. *See* Green, *The Causal Relation Issue in Negligence Law*, 60 MICH. L. REV. 543 (1962). "A victim's hurt as the result, at least in part, of a defendant's conduct may be highly improbable and yet admittedly true, while on the other hand it may be highly probable and yet the result of other cause factors." *Id.* at 557.

96. 19 Utah 2d 367, 431 P.2d 794 (1967).

97. *Id.* at 369, 431 P.2d at 796. Lead-210 (Pb-210) is the end-product, or stable nuclide, that results from the disintegration of radon and its progeny. The amount of Pb-210 in Garner's body was proportional to the amount of radon inhaled. *See generally id.*

98. *Id.*

possibility"[99] that Garner's lung cancer was caused by the radon exposure. The court acknowledged that "[w]hile it seems logical that the unusually high incidence of lung cancer in uranium miners"[100] would indicate a higher probability than normal that the radiation exposure was the cause of Garner's cancer, "it nevertheless falls short of compelling a finding that such was the cause in any individual case."[101] The court, therefore, refused to accept epidemiological evidence of population statistics as proof of individual causation, even though in this case, the plaintiff was without doubt representative of the population.

In a more recent case, *Johnston v. United States*,[102] the United States District Court in Kansas found that evidence of radiation-induced cancers based on statistics "do not measure up to Prosser's test of proof of causation."[103] Four plaintiffs, former employees of an aircraft instrument and development plant, alleged that their respective cancers—leukemia, lung, thyroid, and colon cancer—were caused by their exposure to radium from aircraft instrument dials. The plaintiffs' expert witnesses used a probability of causation calculation which the judge determined to be "statistical speculation based upon speculative dose estimates and speculative risk assumptions. In other words, it is speculation based upon speculation."[104] The judge blurred the distinctions of methodology based on statistical analysis from medical opinion,[105] concluding that even a statistically based probability greater than fifty percent would not meet the standard of proving causation to a "reasonable degree of medical certainty."[106] It is unclear whether this opinion is an attack on probability of causation calculations based on epidemiological studies, or a diatribe against the plaintiff's expert's use of the calculation.[107]

99. *Id.* at 369-70, P.2d at 796.
100. *Id.* at 370, P.2d at 796.
101. *Id.*
102. 597 F. Supp. 374 (D. Kan. 1984).
103. *Id.* at 425.
104. *Id.* at 394.
105. *Id.* at 412-13.
106. *Id.* at 413. The court applied the standard for a medical opinion. No radiation tort plaintiff would be able to meet this standard of proof. *See* Bond, *supra* note 29, at 62-63.
107. This case is an excellent example of the "battle of the experts." Judge Kelly's frustration arose from the vast difference between the plaintiff's experts' probability of causation estimates and the defendant's. Because Judge Kelly did not believe the plaintiff's experts, he attacked the use of probability of causation calculations because of the ease of manipulation of dose and risk coefficient. Though this paper advocates the use of the same probability of causation calculation, the risk coefficient adopted by the NIH tables is presumed to be the best estimate of risk based on radioepidemiological data. The adoption of the PC coefficients from the NIH tables will therefore decrease the opportunity for manipulation of the PC calculation and will interject some consistency in radiation tort litigation.

IV. SUGGESTED REFORM IN LEGAL ANALYSIS OF CAUSATION

A. *Reevaluation of Cause-in-Fact*

Legal and scientific causation were not always so distinct. Before the 1920's, tort theory embraced the scientific notion of "objective causation"[108] which was cast in the mold of classical physics. Classical physics described a mechanical world in which cause and effect ruled. "Things moved for reasons. These reasons were based upon earlier causes for motion. Therefore, all motion was determined and everything was predictable . . . . The observer observed, never disturbed."[109] The evidence of cause and effect was particularistic—a "particular action . . . had the 'causal power' to cause an injury"[110]—and the role of the court was to identify the particular action, and hold the culpable actor liable for the injury caused. Also borrowed from classical physics was the "chain of causation"[111] in which each causal link brought about its determined effect. Liability for an injury resulting from a "chain of causation" would be imposed as long as no intervening cause disrupted its domino effect and thereby acquired the mystical causal power to produce the injury.[112] According to the theory of objective causation, the court *identified* rather than *selected* the culpable actor whose action indisputably caused the injury. Law and science were comfortable bedfellows, smug in a world of effects determined from certain causes.

The advent of quantum theory in physics and in particular the Heisenberg Uncertainty Principle signaled the end of this mechanistic era.[113] The Heisenberg Uncertainty Principle affirmed the imprecision and uncertainty of measuring or predicting atomic events. Heisenberg

108. Horwitz, The Doctrine of Objective Causation, in THE POLITICS OF LAW 201 (D. Kairys ed. 1982).

109. F. WOLF, TAKING THE QUANTUM LEAP 56 (1981).

110. Brennan & Carter, *Legal and Scientific Probability of Causation of Cancer and Other Environmental Disease in Individuals*, 10 J. HEALTH POL., POL'Y & L. 33, 53 (1985). *See also* Rosenberg, *supra* note 92, at 870. *See generally* Saks & Kidd, *Human Information Processing and Adjudication: Trial by Heuristics*, 15 LAW & SOC'Y 123 (1980-81).

> The concept of "particularistic" evidence suggests that there exists a form of proof that can provide direct and actual knowledge of the causal relationship between the defendant's tortious conduct and the plaintiff's injury. "Particularistic" evidence, however, is in fact no less probabilistic than is the statistical evidence that courts purport to shun. All knowledge of past as well as future events is probabilistic. Inevitably it rests on intuitive or more rigorously acquired impressions of the frequency with which similar events have occurred in like circumstances. "Particularistic" evidence offers nothing more than a basis for conclusions about a perceived balance of probabilities.

Rosenberg, *supra* note 92, at 870.

111. H. HART & A. HONORE, CAUSATION IN THE LAW 5 (1959).

112. *Id.*

113. F. WOLF, *supra* note 109, at 115.

determined that it was impossible to observe both the momentum and position of an atomic particle, because in an attempt to see the particle (its position), the measuring system (source of light) would transfer its momentum to the particle and the particle would move out of focus.[114] In other words, the observer disturbed.[115]

The impact of quantum theory was to strip away any pretense of objectivity in the scientific determination of cause and effect. What was external and objective became internal and subjective. The mysterious causal power once believed inherent in objects and events lay in the mind of the observer who inferred causation from the frequent conjunction of objects and events.[116] The predictable clockwork world of interlocking wheels and springs exploded into a world of randomness and probability.

Legal theorists in turn challenged the premise of objective or actual causation in tort law.[117] Proximate or legal cause supplanted objective causation by imposing or limiting liability to conduct which could foreseeably cause an injury. Foreseeability allowed the observer to disturb, and consequently, causal chains were woven into causal webs of social policy. With the advent of proximate cause, business, once protected by the fiction of objective causation, became the logical insurer of injuries foreseeably caused by its acts or omissions.[118]

The irony of the history of legal causation is that objective causation is still alive and well in the form of cause-in-fact. Courts still look to particularistic evidence of cause-in-fact, attributing causal powers to specific acts, totally ignoring the observer's role in drawing causal connections from observed frequencies of conjoined objects or events. For

114. F. WOLF, *supra* note 109, at 108-11. *See also* J. HONNER, THE DESCRIPTION OF NATURE 45 (1987).

115. F. WOLF, *supra* note 109, at 65. R. PETRUCCI, GENERAL CHEMISTRY 165 (2d ed. 1977). Petrucci suggests that a helpful analogy to understanding the effect of observation on the event observed is that of a basketball game and a movie. "In viewing a motion picture, no matter what the reaction of the audience, the plot is predetermined and is unaffected by this reaction. With a basketball game, on the other hand, the course and final outcome of the game can be affected by the response of the spectators." *Id.*

116. This epistemology of causation was articulated by Hume:

> [H]ow often must we repeat to ourselves, *that* the simple view of any two objects or actions, however related, can never give us any idea of power, or of a connexion betwixt them: *that* this idea arises from the repetition of their union: *that* the repetition neither discovers nor causes any thing in the objects, but has an influence only on the mind, by that customary transition it produces: *that* this customary transition is, therefore, the same with the power and necessity; which are consequently qualities of perceptions, not of objects, and are internally felt by the soul, and not perceiv'd externally in bodies?

D. HUME, A TREATISE OF HUMAN NATURE 166 (Selby-Bigge ed. 1888).

117. Horwitz, *supra* note 108, at 201.

118. Horwitz, *supra* note 108, at 210-11.

example, in a negligence claim against the landlord, the tenant who fell on the ice on the sidewalk of a common area, claims that but for the existence of the ice, she would not have fallen and injured herself. Though it can be argued that it is the particularistic evidence of seeing the ice which persuades us that its presence was the cause of the tenant's injury, a closer inspection of the reasoning reveals that it is the observed frequency of such falls in like circumstances which leads us to believe the relationship between the ice and the fall to be causal.[119] This evidence is probabilistic, not particularistic.

The danger of judical decisions in radiation or toxic tort cases which exclude probabilistic proof of causation is that they are often based on the assumption that, given time, science will reveal the specific, particularistic cause or causes of an event.[120] The fallacy of this reasoning is apparent in light of quantum theory and the Heisenberg Uncertainty Principle. "In a world of randomness, where there is no necessary connection between particular causes and effects, all we can hope to do is to statistically correlate acts and consequences in the aggregate."[121] The result of such decisions is that plaintiffs who have to rely on probabilistic proof of causation are not compensated for their injuries and the risk of doing business is not borne by the business nor adequately assessed by society.[122] Policy decisions so covertly operative can only portend disregard of the safety of many and protection of the interests of few.

B. *"Causal Linkage" Test of Cause-in-Fact*

The inadequacy of the traditional "but-for" and "substantial factor" tests of cause-in-fact has been shown in several judicial decisions.[123] To remedy this problem in cases of negligence, judges have simply shifted the burden of proof to the defendant once certain factual connections

119. *Contra* Note, *supra* note 92, at 384 n.42.

120. Brennan & Carter, *supra* note 110, at 54.

121. Horwitz, *supra* note 108, at 210.

122. Horwitz quotes Oliver Wendell Holmes, Jr. who noted that:

> [most injuries] with which our courts are kept busy today are mainly incidents of certain well-known businesses. They are injuries to person or property by railroads, factories, and the like. The liability for them is estimated, and sooner or later goes into the price paid by the public. The public really pays the damages, and the question of liability, if pressed far enough, is really the question how far it is desirable that the public should insure the safety of those whose work it uses.

Horwitz, *supra* note 108, at 211 (quoting OLIVER WENDELL HOLMES, *The Path of the Law,* in COLLECTED LEGAL PAPERS 187 (1920)) (footnote omitted).

123. *See* Sindell v. Abbott Laboratories, 26 Cal. 3d 588, 607 P.2d 924, 163 Cal. Rptr. 132 (1980); Summers v. Tice, 33 Cal. 2d 80, 199 P.2d 1 (1948); Ybarra v. Spangard, 25 Cal. 2d 486, 154 P.2d 687 (1944).

between the defendant's conduct and the plaintiff's injury are made.[124] These factual connections in no way meet the traditional tests for cause-in-fact. For example, in *Summers v. Tice*, although there was a particularistic cause-in-fact relationship between the plaintiff's injury and the conduct of one of the defendants, the plaintiff was unable to prove which defendant's conduct was the cause of his injury.[125] The only factual connection established between the plaintiff and each of the defendants was that the defendants had each negligently fired a shotgun in the plaintiff's direction.[126] The justification for shifting the burden of proof of causation upon a showing of such factual connection is that both of the defendants were negligent and that they, therefore, should not be exonerated from liability. Though many consider the decision in *Summers v. Tice* to be a just one, the reasoning employed simply begs the question. The plaintiff, in essence, did not have to prove his prima facie case of negligence, because the defendants' conduct was negligent. Such arguments, unfortunately, become necessary in order to reach a just decision, as long as the tests for cause-in-fact are so limited.

Several commentators have urged that shifting the burden of proof to the defendant after some showing of factual connection is not only the answer to the problem of the indeterminate defendant, but also to that of the indeterminate plaintiff.[127] The plaintiff is indeterminate in radiation cases because the epidemiological evidence of proof of causation only directly proves the existence of excess cancer caused by radiation exposure to a population. Whether the plaintiff's specific cancer has been caused by radiation exposure or by "natural" causes, cannot be proven. However, in both kinds of cases, the plaintiff is unable to prove that the defendant was the cause-in-fact of the injury under traditional tort theory. The question then arises that if an exception can be made for the inherent uncertainty in the origin of causation, why not in its terminus?[128] This argument was adopted and applied by Judge Jenkins in *Allen v. United States*.[129]

In *Allen* the court shifted the burden of proof of causation in the

124. Thode, *Tort Analysis: Duty-Risk vs. Proximate Cause and the Rational Allocation of Functions Between Judge and Jury*, 1977 UTAH L. REV. 1, 5-7.

125. 33 Cal. 2d 80, 199 P.2d 1 (1948). *See also* Sindell v. Abbott Laboratories, 26 Cal. 3d 588, 607 P.2d 924, 163 Cal. Rptr. 132 (1980); Ybarra v. Spangard, 25 Cal. 2d 486, 154 P.2d 687 (1944).

126. Thode, *supra* note 124, at 6.

127. Delgado, *supra* note 75, at 881-83. *See* Hall & Silbergeld, *supra* note 85, at 445.

128. Delgado, *supra* note 75, at 883.

129. 588 F. Supp. 247 (D. Utah 1984), *rev'd*, 816 F.2d 1417 (10th Cir. 1987) (on grounds of sovereign immunity), *cert. denied*, 108 S. Ct. 694 (1988).

negligence action against the federal government brought by 1,192 plaintiffs who had resided in communities surrounding the Nevada Test Site.[130] The plaintiffs alleged that their exposure to radioactive fallout from the open-air nuclear testing at the test site was the proximate cause of their cancers.[131] After an exhaustive and cogent discussion of the insurmountable problem facing the radiation tort plaintiff in proving causation,[132] Judge Jenkins held that, in the interest of justice, the burden of proof of causation was shifted to the federal government due to its negligent conduct.[133] Jenkins wrote:

> Where a defendant who negligently creates a radiological hazard which puts an identifiable population group at increased risk, and a member of that group at risk develops a biological condition which is consistent with having been caused by the hazard to which he has been negligently subjected, such consistency having been demonstrated by substantial, appropriate, persuasive and connecting factors, a fact finder may reasonably conclude that the hazard caused the condition absent persuasive proof to the contrary offered by the defendant.[134]

While the extension of the indeterminate defendant exception to indeterminate plaintiffs appears logical and fair, it ignores the obvious fact that the problem is with the traditional tort tests and proof of causation. An extension of an exception to the rule palliates the problem; it does not cure it.

Another argument against the solution of shifting the burden of proof of causation is that it mingles cause-in-fact with negligence. The goal should be to make cause-in-fact as neutral as possible, forcing the limitation or extension of liability to be an explicit policy determination under proximate cause. In this instance, cause-in-fact does not have to be proved if breach of duty is proved. This ignores the fact that cause-in-fact is an element to be proven by the preponderance of the evidence in strict liability[135] and product liability[136] suits as well. Unfortunately, the reasoning employed in *Summers v. Tice* would not be as persuasive in strict liability suits. Would this in effect produce two different kinds of proof of causation: one for negligence and one for strict liability? The possible effect of shifting the burden of proof of causation emphasizes the necessity of keeping cause-in-fact separate from negligent conduct.

130. *Id.* at 257.
131. *Id.* at 257-58.
132. *Id.* at 404-15.
133. *Id.* at 415.
134. *Id.*
135. W. KEETON, *supra* note 11, § 79, at 560.
136. W. KEETON, *supra* note 11, § 98, at 692.

A better solution is to add an alternative test for cause-in-fact which would address these concerns. In his article *Concerning Cause and the Law of Tort: An Essay for Harry Kalven, Jr.*, Calabresi suggests the test of "causal linkage" as a determination of cause-in-fact.[137] He suggests that "[t]here is a causal link 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."[138] He further notes that the functions of tort law would lose little if causal linkage were the only test of causation allowed, as long as policy decisions to limit liability were explicitly left to proximate cause.[139]

The "causal linkage" test would not only solve the problem of the indeterminate defendant, but would solve the problem of the indeterminate plaintiff. For example, in *Summers v. Tice* it would not be difficult to show that the negligent firing of a shotgun by both defendants, if repeated in the future, will increase the likelihood of the kind of injury which occurred. In the case of our indeterminate plaintiff, Dena Smith, the "causal linkage" test is tailored for her probabilistic, epidemiological evidence of causation.[140] This test of causation should be adopted in order to augment the particularistic "but-for" and "substantial factor" tests of cause-in-fact and remedy their inadequacy in evaluating probabilistic proof of causation.[141]

C. *Probability of Causation as Proof of "Causal Linkage"*

The "causal linkage" test is an empirically based test that lends itself well to proof of cause-in-fact through radioepidemiological determination of cause.[142] For example, under the "causal linkage" test Dena Smith would have to prove that the use of uranium mill tailings as construction material in her house increased the likelihood of lung cancer. As mentioned above, the only proof available to Dena (as is typical of radiation tort plaintiffs) is her exposure to radon and the epidemiological evidence that inhalation of radon gas increases the risk of lung cancer.

137. Calabresi, *supra* note 13, at 71.

138. Calabresi, *supra* note 13, at 71

139. Calabresi, *supra* note 13, at 105-07. *See also* Brennan & Carter, *supra* note 110, at 52-53.

140. The relation of this theory of causation to scientific causation has been noted: "[The] appeal to probability in solving causal problems could be justified by the growing importance of statistical laws in science and by the fact that in quantum theory it appears impossible in principle to formulate them otherwise." H. HART & A. HONORE, *supra* note 111, at 417 (footnote omitted).

141. Brennan & Carter, *supra* note 110, at 52. "Causal linkage is a probabilistic theory of causation, while 'but for' causes are analogous to mechanistic corpuscularian causes." *Id.* (footnote omitted).

142. Brennan & Carter, *supra* note 110, at 52. *See also* Calabresi, *supra* note 13, at 71.

To prove cause-in-fact under the "causal linkage" test, the plaintiff would have to present the following evidence: (1) that the injury received is of a type known to be caused by radiation; (2) the radiation dose;[143] and (3) the relevant risk coefficient from the NIH tables.[144] From this evidence, the calculated probability of causation[145] would provide the proof of the "causal linkage" between Dena's exposure to radon and her lung cancer.

The available[146] risk coefficients from the NIH tables for the calculation of probability of causation should also be adopted. The risk coefficients in the NIH tables would provide the court with the best scientific estimate of risk[147] and a more simple and consistent method of calculating the probability of causation in radiation tort cases.[148]

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143. Alpha-track detectors are used to measure the annual average radon concentration in an enclosed structure. The dose to an individual residing in the structure would be dependent on the amount of occupied time.

Proof of dose, in general, is not an easy task. Radiation workers would have the easiest task of proving dose from the external exposure, as they wear film badges or pocket dosimeters to measure their dose and records of their cumulative dose are kept by the employer. For a discussion of potential dose assessment problems for radiation workers, see Meinhold, *The Impact of the Probability of Causation on the Radiation Protection Program*, 55 HEALTH PHYSICS 375-77 (1988).

Proof of dose becomes a problem when the unsuspecting plaintiff is not a radiation worker and has been exposed through environmental vectors, such as air or water. In the Nevada test site case, for example, elaborate radioecological modeling was required to attempt to reconstruct the dose the surrounding communities received from the fallout.

144. NIH TABLES, *supra* note 28, at 126-62. The reliability of the NIH tables depends on up-to-date review of new radioepidemiological data. The NIH tables are required to be updated "every four years, or whenever [the Secretary of Health and Human Services] deems it necessary to insure that they continue to represent the best available scientific data and expertise." Act of Jan. 4, 1983, Pub. L. No. 97-414, § 7(b)(3), 96 Stat. 2059, 2060.

The importance of such a provision is evidenced by a recent reevaluation of the dose estimates of atomic bomb survivors. Preston and Pierce, through their work with the Radiation Effects Research Foundation in Hiroshima, Japan, have reassessed the risk estimates for leukemia and solid tumor cancers as 75-85% higher than previously estimated in the BEIR III Report and the NIH tables. Preston & Pierce, *The Effect of Changes in Dosimetry on Cancer Mortality Risk Estimates in the Atomic Bomb Survivors,* 114 RADIATION RESEARCH 437-66 (1988).

145. PC = Dose x Risk Coefficient/(Dose x Risk Coefficient) + Bkg Cancer Incidence.

146. Several primary cancer sites and age groups have been excluded from the NIH tables due to lack of sufficient data. Catlin, *supra* note 36, at 75. When risk coefficients are not available in the NIH tables, the plaintiff could still calculate the probability of causation from available radioepidemiological data.

147. Catlin, *supra* note 36, at 74. Jacobson, *supra* note 50, at 809. S 921, 98th Cong., 1st Sess., 129 CONG. REC. S 3922. (daily ed. Mar. 24, 1983) (Questions and answers entered into the record at the request of Sen. Hatch).

> These tables and formulas will free the courts from dependence upon haphazardly selected epidemiological 'experts'. Judges are not scientists, and it is unlikely that they would become competent in radioepidemiology via the conflicting testimony in what is essentially an adversary procedure or a tort case. . . . HHS's radioepidemiological tables should represent a consensus among the best scientists in the field.

129 CONG. REC. S3922 (daily ed. Mar. 24, 1983). *Contra* Jose, *The Probability of Causation Approach*, 55 HEALTH PHYSICS 371-73 (1988).

148. Bond, *supra* note 29, at 67.

D. *The Argument for Proportional Liability in Radiation Tort Litigation*

The adoption of the "causal linkage" test in conjunction with epidemiological proof of that causal linkage shifts the emphasis from *whether* the defendant's conduct caused the plaintiff's injury to the *likelihood* or *probability* that the defendant's conduct caused the plaintiff's injury. Because liability under traditional tort law mirrors the "either-or" proof of cause-in-fact by awarding the plaintiff "all or nothing" of the proven damages, a different method of imposing liability is needed for awards based on proof of the probability of causation.[149] The answer to this concern is proportional liability based on the probability of causation proved by the plaintiff.[150] It is the probability that the plaintiff's cancer was caused by the exposure to radiation that is proved, not that radiation did or did not cause the cancer. It appears logical, therefore, that the plaintiff should recover in proportion to the probability of causation. Consequently, the plaintiff, upon proving a prima facie case, would be awarded the proven damages multiplied by the probability of causation. For example, if Dena Smith were successful in proving the other elements of her tort cause of action, she would recover twenty-five percent of her total damages.

The equity of proportional liability in radiation tort cases is apparent if one views the harm caused by the defendant as a harm to a population of which the plaintiff is a member. This concept appears foreign in a tort system traditionally concerned with compensation of the individual plaintiff.[151] A plaintiff must identify with a "subgroup of a larger population" which has been exposed to radiation and has substantially the same characteristics as the plaintiff in order to successfully implicate the defendant as the cause-in-fact of the cancer.[152] From an epidemiological perspective, the characteristics of an individual can only be represented by the "statistically average" person.[153] The plaintiff, therefore, assumes the probability of causation estimate of the harm caused the "statistically

149. Comment, *supra* note 75, at 254.

150. Comment, *supra* note 75, at 254. *Contra* Note, *supra* note 10, at 859.

151. This comment does not address the question of whether radiation exposure suits should be handled as class action suits rather than as private actions. For a complete discussion on the advantages and disadvantages of "public law," see Rosenberg, *supra* note 92, at 859. *See also* Delgado, *supra* note 75, at 881.

152. Bond, *supra* note 29, at 64. *See also* Jacobson, *supra* note 50, at 807.

153. Catlin, *supra* note 36, at 73.

average" person only to the extent that the plaintiff epitomizes the "statistically average" person.[154] Though in reality, the radiation exposure either did or did not cause the plaintiff's cancer, the cause can only be expressed in terms of the causal association of excess cancer incidence in an exposed population. The plaintiff, in essence, becomes a microcosm of that population by assuming "gradations of cancer that can increase with the amount of exposure."[155]

If recovery is viewed in light of this reliance upon population statistics, it becomes apparent that recovery should be allocated to the exposed population, not the individual. For example, assume that there are ten plaintiffs exposed to the same dose of radiation who later develop cancer, and the probability that the radiation exposure caused each plaintiff's cancer is ten percent. In fact, only one plaintiff's cancer has been caused by the radiation dose. Under proportional recovery, the defendant pays ten percent recovery to each of the ten plaintiffs, thereby paying a total of one one hundred percent recovery for the one plaintiff who was actually harmed by the defendant.[156]

Under this system of proportional liability, the "rights" role of the torts system to perform "corrective justice" that preserves the rights of the plaintiff against the "wrongful infringement" of the defendant[157] interfaces with the utilitarian role of providing the "optimal deterrence"[158] of the tortious conduct. Proportional recovery based on the probability of causation insures that all the plaintiffs potentially injured by the defendant's conduct can recover some of their damages.[159] The population as a whole is compensated and the defendant is held liable for the actual harm caused: the excess cancers in the population.[160] Proportional liability thereby insures optimal deterrence because the cost to the defendant is the redress of the actual harm caused to the population, though the

154. Catlin, *supra* note 36, at 73.

155. Bond, *supra* note 29, at 63.

156. In a private tort action, it is unusual to look at an individual plaintiff's harm and recovery as based on a population. It appears that compensation of the plaintiff does not occur in this situation. Indeed, the one thing we know for certain is that the probability of causation does not reflect the actual probability that each individual plaintiff developed cancer due to exposure to radiation. What occurs on an individual basis is that nine plaintiffs receive a windfall of ten percent of damages even though their cancers were not caused by the radiation exposure, and one plaintiff is undercompensated ninety percent of damages because the cancer was in fact caused by the radiation exposure. The alternative for the plaintiff, however, is no recovery under traditional tort theory. *See* Delgado, *supra* note 75, at 892-93.

157. Rosenberg, *supra* note 92, at 859-60.

158. Rosenberg, *supra* note 92, at 862.

159. Delgado, *supra* note 75, at 893. *See also* Comment, *supra* note 75, at 256.

160. Delgado, *supra* note 75, at 893. *See also* Comment, *supra* note 75, at 258.

actual victims are unidentifiable.[161]

The importance of the redress of injury costs is underscored when the defendant is a business (as is often the case) that conforms safety investments to profit margins.[162] This is particularly true when the defendant's business involves radioactive materials. That radiation exposure can cause cancer is not disputed.[163] But if cost of the injuries is not borne by the party who benefits from the use of radiation, there is no economic incentive for that party to invest in safety. From the defendant's point of view, no harm is done if it is not attributable to the defendant.

The failure of causation in the traditional tort system to attribute the risk of cancer to the sources of radiation which would otherwise be liable has made investment in safety less economical and, therefore, more lax.[164] Recent reports by the Ohio environmental protection agency estimate the release of 298,000 pounds of uranium wastes into the air, and the deliberate dumping of 167,000 pounds of hazardous waste into a river by National Lead of Ohio, the contractor at a uranium processing plant in Fernald.[165] Uranium-238 has a half-life of 4.5×10^9 years.[166] The effect of its dispersal will be felt for millions of years.[167] Though a class action was filed against National Lead in 1985 by the Fernald area residents, the plaintiffs limited their prayer for damages to lowered property values and emotional trauma, because they were "[a]ll too aware that radiation exposure is difficult to link conclusively with specific health problems."[168] One of the plaintiffs, who attributes the cancers of his two sons, ages eight and two to the contamination of the air and soil in the family's vegetable garden with uranium-235 from the Fernald plant, painfully articulates the need for legal redress:

> I would like to see, just like it was an individual, that they'd just admit they screwed up, that they were willing to right their wrongs . . . There is a lot of damage they can't undo. But if they deny responsibility, and you have a Government that is not accountable to its citizens, then you do not have a republic.[169]

161. Rosenberg, *supra* note 92, at 866.
162. Rosenberg, *supra* note 92, at 855. *See also* Note, *supra* note 10, at 843 n.12.
163. *See supra* notes 22-65 and accompanying text.
164. Cramer, Leavitt & Nash, *"They Lied to Us,"* TIME, Oct. 31, 1988, at 61-65.
165. *Id.* at 64.
166. UNSCEAR, *supra* note 7, at 13. Because it is not possible to predict when any given atom will disintegrate, half-life is used to determine, on the average, the amount of time in which half of the atoms of a nuclide will disintegrate. H. JOHNS & J. CUNNINGHAM, *supra* note 8, at 28.
167. UNSCEAR, *supra* note 7, at 13.
168. Cramer, Leavitt & Nash, *supra* note 164, at 64.
169. Cramer, Leavitt & Nash, *supra* note 164, at 65.

V. CONCLUSION

Radiation by its very nature presents a risk of injury to society; it is a known carcinogen. Several radioactive elements have such long half-lives that their exposure period extends for many millions of years. The gravity of the risk together with the increased probability of exposure due to the long half-lives of many radioactive elements should enter into any balancing equation concerning the costs and benefits of the use of radioactive substances.

Because society relies primarily on the tort system to prevent injury-generating activity and to compensate victims of such activity, the costs of radiation use must be recognized and remedied by imposing liability. The inaccessibility of a remedy in tort for the radiation tort plaintiff shifts the balance away from investments in safety because no injury costs are weighed.

Tort remedy often remains inaccessible to radiation tort plaintiffs due to the outdated legal notions of causation. The rejection of probabilistic evidence of cancer causation in radiation torts due to its failure to meet the requirements of legal cause-in-fact should result in the reevaluation of legal cause. To remedy this deficiency, the legal concept of cause-in-fact should be expanded to allow for probabilistic evidence that provides a "causal linkage" between the radiation exposure and the development of cancer. The science of radioepidemiology can provide that evidence through the determination of the probability of causation.

If the plaintiff succeeds in proving each element of the tort by a preponderance of the evidence, the liability of the defendant should be proportioned according to the probability of causation proved. In this way, the injury costs resulting from the use of radiation will accurately reflect the actual harm caused by the radiation. The cost component in society's balancing test will then be accurately weighed in decisions concerning any future use of radioactive substances.

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