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***103** HOW MANY MICE MUST DIE?

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I. INTRODUCTION

The Senate Committee on Environment and Public Works has reported that more than ninety percent of all hazardous chemical wastes produced in the United States have been disposed of improperly. [FN1] Widely publicized associations between cancer in humans and a number of environmental factors have produced an increased awareness of the potential for contamination. [FN2] Although the scientific community has linked chemical waste exposure to cancer and other latent diseases, toxicological and medical experts are often limited to statements that such exposure creates merely the probability of an increased risk of contracting a disease. [FN3]

This article will examine risk assessment, that is, the process of characterizing the adverse health effects of human exposures to environmental hazards. [FN4] Through risk assessment, experts attempt to answer: (1) whether a given chemical is capable of causing harm and (2) whether that specific chemical caused harm to a specific individual. [FN5] Unfortunately, this assessment process has been criticized for being less precise than five year weather forecasting. [FN6] Despite the intrinsic uncertainties and difficulties of risk assessment, the information generated by this process serves as the evidence which links a toxic chemical to a given plaintiff, and is essential for resolution of the causation issue in toxic tort litigation. [FN7]

Current case law [FN8] and legislation also will be examined to determine whether an ***104** increased risk of contracting a disease may now constitute a legally compensable injury. Regarding the case law and, particularly, the long awaited Ayers v. Jackson Township [FN9] decision, the question remains; is the judicial system reforming the common law barriers to permit recovery for toxic tort victims' post-exposure, pre-symptom injuries? Perhaps, the Superfund Amendments and Reauthorization Act of 1986 (SARA) [FN10] provisions requiring increased chemical assessment by the Agency for Toxic Substances and Disease Registry (ATSDR) [FN11] will make the plaintiff's task of proving causation an easier one.

[FN1]. S.REP. NO. 848, 96th Cong., 2d Sess. 3 (1980) (citing Environmental Protection Agency (EPA) estimates).

[FN2]. There have been widely publicized contaminations: Times Beach, Missouri and Love Canal, New York. Lesser known contaminations include Woburn and Lowell, Massachusetts; Triana, Alabama; and Jackson Township, New Jersey. SUBCOMMITTEE ON INVESTIGATIONS AND OVERSIGHTS, HAZARDOUS WASTE EXPOSURE VICTIMS COMPENSATION (ASSESSING RISK FROM AMBIENT, NON-WORKPLACE EXPOSURE AND NEED FOR ADDITIONAL REMEDIES), H.REP. NO. 642-5, 99th Cong., 2d Sess. 1 (1986) [hereinafter, SUBCOMMITTEE REPORT, VICTIMS COMPENSATION]; See also, Stenzel, The Need for a National Risk Assessment Communication Policy, 11 HARV.ENVTL.L.REV. 381 (1987); Wilkinson, Being More Realistic about Chemical Carcinogenesis, 21 ENV'T.SCI.TECH. 843 (1987) [hereinafter Wilkinson].

[FN3]. Increased risk is a risk greater than that born by the general population. Industrial Union Dept., AFL-CIO v. American Petroleum Institute, 448 U.S. 607 (1980); Note, Tort Actions for Cancer: Deterrence, Compensation and Environmental Carcinogenesis, 90 YALE L.J. 840, 850-51 (1981) [hereinafter Note, Tort Actions].

[FN4]. Risk assessment differs from risk management. Risk assessment is the process of characterizing the adverse health effects of human exposures to environmental hazards. Risk management involves evaluating and choosing among regulatory options based on economic, social, political and engineering factors, as well as information on risk. U.S. CONGRESS, OFFICE OF TECHNOLOGY ASSESSMENT (OTA), IDENTIFYING AND REGULATING CARCINOGENS, BACKGROUND PAPER, J.REP. NO. 952-67, 100th Cong., 1st Sess. 26 (1987) [hereinafter OTA, IDENTIFYING CARCINOGENS].

[FN5]. Farber, Toxic Causation, 71 MINN.L.REV. 1219, 1227-8 (1987).

[FN6]. SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 10 (statement by Dr. Vernon N. Houk, Director of the National Centers for Disease Control (CDC)).

[FN7]. See id. at 2, 5, 11-14.

[FN8]. This article involves specifically the increased risk claim in toxic tort litigation. However, litigators often simultaneously pursue other causes of actions which have proven to be more successful for plaintiffs than the increased risk cause of action. These claims include: fear of increased risk of developing a disease, decreased quality of life, and compensation for medical surveillance. The fear of potential health hazards was recognized as cognizable in Sterling v. Velsicol Chemical Corp., 647 F.Supp. 303 (W.D.Tenn.1986), appeal pending, No. 86-6087 (6th Cir. Oct. 6, 1986), see infra notes 183-190 and accompanying text, as was mental anguish due to the fear of developing cancer in Hagerty v. L. & L. Marine Services, Inc., 788 F.2d 315 (5th Cir.), modified on other grounds, 797 F.2d 256 (5th Cir.1986), see infra notes 163, 171-181 and accompanying text. But see, Ayers v. Jackson Township, 106 N.J. 557, 525 A.2d 287 (1987), see infra notes 9, 192-207 and accompanying text, and Anderson v. W.R. Grace & Co., 628 F.Supp. 1219 (D.Mass.1986), appeal docketed, No. 87-1405 (1st Cir. March 15, 1988), see infra notes 162, 164-170 and accompanying text, where plaintiffs' emotional distress claims were denied. See also, Stites v. Sundstrand Heat Transfer Inc., 660 F.Supp. 1516 (W.D.Mich.1987), see infra notes 153-160 and accompanying text, where fear of cancer claims were addressed on a case by case basis; some recoverable, some denied. See generally, Dworkin, Fear of Disease and Delayed Manifestation Injuries: A Solution or a Pandora's Box? 35 DEFENSE L.J. 1 (1986). Regarding claims for decreased quality of life, Ayers allowed compensation for losses associated with damage to property. Regarding recoverable medical surveillance costs, see, e.g., Ayers, Hagerty, Villari v. Terminix International, Inc. 663 F.Supp. 727 (E.D.Pa.1987), and Lykins v. Westinghouse Electric, No. 85-508 (E.D.Ky.), 2 Toxics L.Rep. (BNA) 1100, 2288 (1987-1988). But see, Wehner v. Syntex Corp., No. C-85-20383 (N.D.Cal.1987), 2 Toxics L.Rep. (BNA) 706, 895, 1101 (1987).

[FN9]. 106 N.J. 557, 525 A.2d 287 (1987).

[FN10]. Superfund Amendments and Reauthorization Act of 1986, Pub.L. No. 99-499, 100 Stat. 1613 (1986) (codified at 42 U.S.C.A. § 9658 (West Supp.1987)).

[FN11]. Section 110 of SARA, amending CERCLA § 104i, 42 U.S.C. § 9604i.

II. MAJOR BARRIERS TO VICTIMS COMPENSATION

Toxic tort law is far from settled. Statutorially and administratively, much confusion and inconsistency exists in monitoring the adverse health effects caused by hazardous substances, as over a dozen federal statutes and at least five federal agencies are involved. [FN12] With respect to hazardous wastes, the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), [FN13] which provides for the clean-up of hazardous releases, contains no provision for recovery of damages to personal property and real property resulting from exposure. Although Congress provided for creation of a Study Group to propose solutions to the problems of victim compensation, none of the Group's recommendations has been adopted in the SARA legislation, the first major revision of CERCLA. [FN14] The absence of substantive federal legislation, therefore, forces reliance upon the common law tort principles of the states. [FN15] Until a comprehensive ***105** governmental response occurs, creating some statutory or administrative mechanism, the judicial system must continue to serve as the forum for an exposed victim's remedy. [FN16]

Confronted with hazardous waste and toxic chemical litigation, the courts have attempted to adapt common law tort doctrines to the particular problems presented by these cases. [FN17] Most commentators believe that this accomodation has been, and continues to be, unsuccessful. [FN18] Numerous obstacles have prevented victims' recovery: state statute of limitations, loss of evidence, the "single controversy rule" and res judicata, as well as the inability to prove causation. [FN19]

The long latency period typical of illnesses such as cancer, often prevents a victim from discovering an injury and pursuing a cause of action until after the state statute of limitations for personal injuries has expired. [FN20] Some jurisdictions, including New Jersey and Pennsylvania, have remedied this problem with a "discovery rule," which tolls the statute until the injury is discovered. [FN21] In jurisdictions without such a rule claims have been barred due to untimeliness. The federal enactment of SARA, however, has removed the limitations obstacle. [FN22] Under SARA, a plaintiff is required to bring his

claim within three years from the date plaintiff knew, or reasonably should have known, that the hazardous substances caused or contributed to the injuries. [FN23] The existence of a federally required commencement date, now pre-empts state statutes of limitations which begin to run on an earlier date. [FN24] Consequently, future litigation should not be plagued by statute of limitations problems.

The significant lapse of time between exposure to a toxic substance and knowledge of injury through physical manifestation of disease, often causes other difficulties for plaintiffs. Evidentiary problems of actual loss of evidence and the increased difficulty in identifying the responsible parties, not to mention the risk that the identified parties are no longer solvent, have been attributed to this time delay. [FN25]

An additional obstacle often confronted in past toxic tort litigation is the "single controversy rule." This rule requires that a party include in one action all related claims against a given adversary. [FN26] Any failure to do so would preclude the maintenance of a ***106** second, later action. [FN27] Hence, an earlier action before the injury was discovered and, thus, not raised as a cause of action, would bar a future action when the disease manifested itself. Judicial decisions, however, have transformed this obstacle. Courts have held that "a timely-filed cause of action for damages prompted by the future 'discovery' of a disease or injury related to tortious conduct in prior litigation will not be precluded." [FN28]

Despite the legislative reform pre-empting state statute of limitations and the judicial accomodations of traditional tort doctrines, the burden plaintiff encounters in proving causation remains. In establishing that a sufficient nexus exists between defendants' conduct and plaintiffs' injury, two interconnected links must be proved. The first requires connecting the defendant with the release of the "harmful" substance. The second link requires tying an injured plaintiff to the given exposure. Many claimants are unable to produce sufficient evidence to prove these causal links and therefore fail. [FN29]

III. QUALITATIVE AND QUANTITATIVE RISK ASSESSMENT [FN30]

The first step in the causal chain requires an analysis of whether a chemical attributable to defendant is harmful. This entails a qualitative assessment of the potential toxic properties of the substance, that is, whether the chemical agent increases the incidence of cancer. [FN31] Depending on the stage of analysis and the assessor, scientist or federal agency, this might be called either a toxicological profile [FN32] or a hazard identification or assessment. [FN33] The second step, the quantitative risk assessment, for which dose-response and exposure assessments are the central components, identifies all the individuals or population subgroups that have been exposed to a chemical and calculates the actual doses received by analyzing the toxicological data with the exposure information. [FN34]

***107** A. Chemical Carcinogenicity--Is the Chemical Harmful?

Two million chemical compounds are known today, [FN35] with approximately 63,000 chemicals in common use in the United States and 1,000 newly synthesized chemicals entering international commerce annually. [FN36] Although many of the chemicals are not toxic, little is known about the possible health consequences stemming from exposure to these agents. [FN37]

The term cancer, applies to a family of diseases [FN38] characterized by unconstrained growth and cellular malfunction. [FN39] Scientists hypothesize that a change in either the genetic material or some other growth- regulating mechanism within the individual cell, is responsible for the cellular dysfunctioning. [FN40] Recognized as a highly complex, multifactorial disease, a variety of factors, both endogenous [FN41] and exogenous, [FN42] may cause the genetic and cellular changes. [FN43] Through a multistage process, these factors interact either simultaneously or in sequence to disrupt normal cell growth and division. [FN44]

Nonetheless, scientists have characterized two major stages to cancer: initiation and promotion. [FN45] Initiation is the process whereby a chemical or other agent damages the DNA of the cell. [FN46] Promotion consists of the subsequent progression and proliferation of the "transformed" cell through a variety of pathological states culminating in a malignant tumor. [FN47]

Those chemicals which increase the incidence of benign or malignant tumors are termed carcinogens. [FN48] Carcinogens, capable of directly damaging DNA are labelled as direct or genotoxic, while nongenotoxic or indirect chemicals act via mechanisms which are not entirely known and may even require other carcinogens to initiate a disease process. [FN49] The current regulatory policy does not distinguish between these types of carcinogens but has chosen to treat all carcinogens as

genotoxic. [FN50]

Some scientists, chemical manufacturers, and insurance companies argue that carcinogenicity should not be considered an inherent property of a chemical. [FN51] They emphasize ***108** that cancer is an outcome of the interaction between a chemical and a complex human biological system, which is influenced by many factors. [FN52] As a result, in evaluating a suspected carcinogen, "the scientist must observe the response it produces in some biological system: humans, laboratory animals or ... single cell s ." [FN53]

B. Qualitative Assessment

The qualitative assessment of carcinogenicity requires the determination of a chemical's cancer causing capabilities and its potency in quantitative terms. [FN54] Efforts to identify carcinogenic chemicals proceed on a case-by- case basis and mainly depend on the results of four types of studies. [FN55] These are (1) bioassays [FN56] with laboratory animals, (2) short-term tests involving microorganism or cell cultures, (3) epidemiological studies in human populations, and (4) chemical structure-activity [FN57] relationships. [FN58]

1. Bioassays With Laboratory Animals

Bioassays remain the most significant and practical experimental procedure for identifying potential carcinogens. [FN59] The scientist's control over experimental conditions and doses, as well as his ability to isolate the effect of a suspected carcinogen are primary reasons for this. [FN60] However, "many practical and theoretical uncertainties do exist, relating to both the design and conduct of the test itself, and the subsequent interpretation of the data." [FN61]

By way of procedure, rodents [FN62] exposed to very high doses of a chemical in a laboratory are compared with a control group of unexposed animals. [FN63] Ideally, young animals, some shortly after weaning, others after birth or even during fetal development, are treated with the chemical and are observed for tumor development and other signs of ***109** disease. [FN64] At the end of the study, the surviving animals are sacrificed and their tissues and organs, as well as those animals who died during the study, are examined. [FN65]

As detection of a risk of even one in a hundred persons requires over 10,000 animals, this process can become extremely expensive and impractical. [FN66] Therefore, "manageability and expense often limit the size of animal bioassays." [FN67] To circumvent these problems, scientists have relied on the use of two or three extremely high doses to generate a detectable response rate from a few hundred rather than a few thousand animals. [FN68] However, the use of such high doses has created a problem in judging a chemical as positive for carcinogenicity. [FN69] Although a large dose might yield a positive result, that is, a significant increase in tumor development, a dose selected at half that strength, would yield a negative result even though it is still well beyond the range of usual human exposures. [FN70]

The subsequent prediction of health consequences to humans exposed to very low doses is the primary purpose for collecting this animal data. However, the application of this data to humans must be construed to project what occurs at four or more orders of magnitude below the tested doses. [FN71] Although the scientist experimentally has determined the relationship for high doses and can plot this information graphically, since the response at low doses cannot be measured directly, he can only speculate on the nature of the dose-response relationship or curve at these low levels. [FN72] To aid in the estimation of these low dose effects, statistical models have been developed which allow the scientist to extrapolate from the high dose data points to the low portion of the curve. [FN73]

These dose-response models are, however, conflicting, for each represents a different, hypothesized mathematical relationship. [FN74] Although, no single extrapolation model is recognized as the most appropriate, a model which incorporates linearity is preferred, unless there exists information to justify the use of a non-linear model. [FN75] Additional debate surrounding these extrapolations involves whether a threshold level exists below which carcinogens will not evoke a response. [FN76] As a rule, the regulatory agencies endorse models that assume the absence of no-effect thresholds and linearity within the low-dose portion of the dose-response curve. [FN77]

An additional area of concern is properly assessing the relevance of these animal studies to humans. [FN78] It is questionable whether laboratory animal response is an adequate substitute for the human cancer response, and, more specifically, whether the presence of ***110** tumors in certain animal organs will directly correlate to human tumor development. [FN79] However, as these substances cannot be tested on humans, public policy dictates that "substances found to be carcinogenic in animals

be assumed to be carcinogenic in humans." [FN80]

Hence, both the extrapolations from high to low doses and from animals to humans may limit the usefulness of bioassay data in assessing the human carcinogenic effect. Despite these limitations, animal bioassays do provide strong evidence that a substance is carcinogenic. [FN81]

2. Short-Term Tests For Genotoxicity

Short-term tests for genotoxicity include microorganism and cell culture tests of both in vitro [FN82] and in vivo [FN83] assays. These tests examine chromosome aberrations, [FN84] unscheduled DNA synthesis, and sister chromatid exchange. [FN85]

By far the best known of these tests is the Ames Salmonella test. [FN86] Although validation studies suggest that this test yields false results ten percent of the time, a more recent study suggests that a positive result carries a high probability of seventy percent that a chemical will be associated with carcinogenic activity. [FN87] Of all the short-term tests, the Ames test appears to be the best predictor. [FN88]

Despite the predictability of the Ames test, it has become common practice not to rely on the results of one test alone, but to evaluate genotoxicity on the basis of the total weight of evidence from a battery of short-term tests. [FN89] As there exist considerable inconsistencies among the results of the different short-term tests, as well as little correlation between these tests and the results of the bioassays, the information derived from these batteries may be spurious. [FN90]

Nonetheless, given that these tests are conducted in a few days and are significantly less expensive than the animal studies, the short-term tests might be considered adequate substitutes for more sophisticated estimates of potency. [FN91] It is worth noting that these results are merely rough quantitative estimates and should be used with the knowledge of that limitation.

***111** 3. Epidemiological Studies

Epidemiological studies are the only tool available for the assessment of the potential carcinogenic response in humans. [FN92] This identification of carcinogens occurs through correlations between the rates of a disease in a population and specific environmental factors. [FN93]

There are different designs of epidemiological studies which change based on the time period when the data is recorded and the groups and variables selected. [FN94] With respect to time there are both retrospective and prospective studies. A retrospective study begins with a group that already demonstrates the disease and involves uncovering factors in the group's history which correlate with the disease. [FN95] As a study it is considered less valid and reliable than prospective studies since it is prone to both researcher and subject bias. [FN96] The use of data that has already been collected may, in addition, lead to incomplete information. [FN97] Conversely, "prospective studies begin with an unaffected sample of the population who have certain factors present in their environment and ... record the occurrence of future events in relation to the presence or absence of those factors." [FN98]

Additionally, epidemiological studies are characterized as either following an incidence, prevalence, or case control approach. [FN99] The incidence approach follows a prospective timetable. [FN100] This type of study examines a population free of disease and measures certain attributes or factors related to the development of a particular disease. [FN101] Prevalence studies measure the presence or absence of diseases within a defined population and proceed to examine the relationship of specific variables between the diseased and non-diseased groups. [FN102] Lastly, in case control studies, individuals with a disease of interest are paired with a control group of persons without the disease and various factors are statistically compared. [FN103] This type of study is relatively inexpensive, more often performed and most likely to be offered in evidence for toxic tort litigation. [FN104] Common criticisms, however, involve the difficulty in selecting an appropriate control group and its retrospective design. [FN105]

A key issue addressed by epidemiological studies is the comparison between exposed and non-exposed groups or the relative risk. [FN106] This relative risk is a ratio of the incidence rate of disease in the exposed group divided by the rate in the non-exposed control ***112** group. [FN107] It should be noted that even for toxic substances with the strongest relationship between exposure and disease formation, the absolute risk of disease development is far less than fifty percent. [FN108]

Due to the significant role that statistics plays in analyzing epidemiological data, commentators suggest that studies should reach a sufficiently high level of statistical significance to guard against mere chance findings. [FN109] In most studies that are deemed significant, the "difference between the observed and expected result is large enough that such a difference would occur randomly only five percent of the time." [FN110] It is recommended that only highly significant epidemiological findings, those with a chance of random association of one percent (1 in 100) as opposed to five percent (1 in 20), be relied upon. [FN111]

"[P]otential difficulties in the design and implementation of [epidemiological studies] as well as problems in data collection, analysis and interpretation" may limit the usefulness of these results. [FN112] Although epidemiology cannot demonstrate the source of any particular individual's disease, it can provide the relative statistical relationship or probability between that disease and the population group involved in the epidemiological study. [FN113] Furthermore, these studies can never provide conclusive proof that a substance is not carcinogenic. [FN114] Nonetheless, epidemiological studies have gained wide judicial acceptance. [FN115] Of the four tests, they are viewed as the most relevant because they study phenomena actually occurring in humans under natural conditions. [FN116]

C. Quantitative Assessment

Evidence from the studies described above provides a qualitative assessment of the potential toxic properties of a substance without quantifying the risk imposed on an ***113** exposed individual. [FN117] It is the quantitative assessment which applies the qualitative results to an exposed population. [FN118] First, this assessment requires the establishment of the dose-response relationship. [FN119] Secondly, the magnitude and duration of the community's exposure is evaluated. [FN120] Finally, the dose-response model is applied to calculate the exposed individual's risk of developing a latent disease or cancer. [FN121]

1. Dose-Response Assessment

The dose-response assessment describes the relationship between the level of exposure, or the dose, and the incidence of disease. [FN122] While this step primarily consists of extrapolating from high to low doses and converting animal doses and responses to their equivalent human values, it also includes predicting from a given dose, the likelihood that a tumor will occur over the course of a lifetime. [FN123]

A variety of models have been proposed to predict this likelihood. [FN124] The probit model posits a logarithmic relationship between dose and response. [FN125] Since it predicts lower risks at low doses than the other models, it has not acquired general scientific and agency acceptance. [FN126] The multi-stage model, which derives from the theory that carcinogenesis results from a series of mutational steps, predicts the transition from one stage of cancer to the next as a function of both the dose and potency of the carcinogen. [FN127] The one-hit or linear model, the most strongly supported by governmental agencies, is based on the assumption that cancer is initiated by a single exposure of the carcinogen on the target cell. [FN128]

The use of these different models creates a significant divergence in the mathematical estimation of cancer risk for a suspected carcinogen. [FN129] Consequently, a significant criticism has been levied that the selection of the model often determines the result. [FN130] In addition, there are serious doubts as to whether the precise mathematical risk estimates are justified by the quality of the "qualitative" or toxicological data. [FN131] It is questionable, whether the procedural and analytic difficulties of the various tests, are consistent with attempts to quantify, precisely, the magnitude of the risk posed by the carcinogen.

2. Exposure Assessment

Another component of quantitative risk assessment is exposure assessment. In its purest form, exposure assessment identifies all individuals exposed to a chemical and assembles ***114** sufficient analytical chemistry data to allow quantitative calculation of the actual doses received by the exposed individuals. [FN132] These calculations quantitate separate contributions from oral, dermal and inhalation routes, describe the time dependence of the exposure and identify the source and toxicity of the chemical. [FN133]

There are two basic approaches to measuring exposure: passive dosimetry and biological monitoring. [FN134] Passive dosimetry is the measurement of the amount of chemical available through the lungs or skin. [FN135] These external

exposures are then converted using a conversion formula into a biologically available internal dose. [FN136] The other approach, biological monitoring, measures exposure by monitoring body fluids, typically blood or urine, for the chemical of interest. [FN137] In order to calculate the actual internal dose using this measurement, pharmacokinetics [FN138] of the chemical in the body must be known. [FN139] Once the level of exposure is determined, it is combined with the dose-response information to yield an estimate of the expected incidence of adverse health effects. [FN140]

IV. JUDICIAL RECOGNITION OF AN INCREASED RISK CAUSE OF ACTION

An examination of judicial willingness to recognize a cause of action for increased risk requires a discussion of whether the information obtained from risk assessment is sufficient to overcome the potentially insurmountable causation problems. It is well recognized that significant gaps exist in medical knowledge about cancer, the assessment process for determining chemical toxicity, and the measurement of exposure and the relationship of this exposure to future risk. [FN141] A recent Congressional committee reviewing victims' compensation, considered the inadequacy of existing risk assessment as the most significant impediment to individuals seeking relief through the courts for toxic chemical induced injuries. [FN142] Faced with the admitted inability of the experts to quantify the increased risk with exact scientific certitude, some commentators have questioned why juries should be permitted to speculate on matters that science does not yet fully comprehend. [FN143]

Courts often face the possibility that the articulated increases are so microscopically small as to be meaningless, or merely toxicological trivia. [FN144] In fact, some critics believe ***115** that the public's perception of risk and the fear of cancer is far greater than any demonstrable risk. [FN145]

In all, "no clear standard has yet emerged to determine when the data and analysis from risk assessment techniques are legally sufficient." [FN146] The courts have "sometimes allowed recovery based on highly suspect evidence, or conversely, have failed adequately to justify the exclusion of evidence." [FN147] For example, epidemiological studies are frequently deemed irrelevant, as they do not focus specifically on whether the individual plaintiff was injured, but instead, focus on large groups or populations which requires inferences to plaintiff's situation. [FN148]

Without some physical manifestation of illness, the courts, for the most part, have rejected sustaining damages based on enhanced risk albeit through the application of different standards. [FN149] What follows are particular toxic chemical cases which demonstrate these different standards.

Many courts continue to follow the "reasonable medical certainty" rule. [FN150] This rule requires testimony from toxicologists and physicians establishing that damages consisting of future injury are "reasonably certain" to occur. [FN151] As a result, this standard usually requires some clinically recognizable disease or at the least a willingness by medical experts to state that they have seen the damage from the toxic chemical in the particular plaintiff and that there exists an increased risk of future disease. [FN152] Indicative of this approach is Stites v. Sundstrand Heat Transfer, Inc. [FN153]

In Stites, toxic chemicals, specifically trichloroethylene (TCE), leaked from a manufacturing plant into plaintiffs' drinking water. [FN154] The plaintiffs experienced prolonged and extensive exposure that greatly exceeded the EPA recommended limitations. [FN155] Although none of the plaintiffs actually contracted cancer, they claimed their exposure to TCE had caused them to suffer an increased risk of developing cancer. [FN156]

Despite what appeared to be epidemiological and animal bioassay evidence demonstrating that TCE is a human carcinogen, the experts at trial were unable to say, with a reasonable degree of medical and scientific certainty, that future cancerous consequences would occur. [FN157] Following the Michigan Supreme Court's approach in Larson v. Johns-Manville Sales Corp., [FN158] the Stites court stated that "a reasonable certainty is more than a reasonable probability," and, may be more accurately described as "the highest degree ***116** of probability" or "in all likelihood." [FN159] As none of the plaintiffs' experts were able to quantify the enhanced risk, the court upheld the cancer risk assessment model offered by the defendant, which established that the total cancer risk was "infinitesimal and virtually nil." [FN160]

The words "reasonably certain" have been interpreted inconsistently. [FN161] The most limiting interpretation is the requirement espoused by the Stites court. The least limiting interpretation requires that the future injury probably will occur. This slightly less stringent "reasonable probability" standard has been adopted by the courts in Anderson v. W.R. Grace & Co. [FN162] and Hagerty v. L. & L. Marine Services, Inc. [FN163]

In Anderson, defendants, W.R. Grace Co. and Beatrice Foods Co., were alleged to have contaminated the groundwater in certain areas of Woburn, Massachusetts with chemicals including trichlorethylene and tetrachlorethylene. [FN164] Within the group of thirty-three plaintiffs, eight either had contracted or died from leukemia, while twenty-five had not suffered any specific illnesses. [FN165] Regarding the increased risk claim, the court stated that the plaintiffs were entitled to compensation for all injuries that "reasonably were expected to follow" from the exposure, but not for those injuries that "possibly might follow." [FN166] Therefore, a recovery was deemed contingent upon establishing a "reasonable probability" that a harm would occur. [FN167]

Of specific interest were the policy concerns raised by the court. The first was that recognizing enhanced risks would create a flood of speculative law suits. [FN168] The court, also, was concerned that awarding damages based on a mere mathematical probability for such an award might significantly undercompensate those who did develop cancer, while representing a windfall to those who did not. [FN169] Since the plaintiffs' claim did not indicate the magnitude of the increased risk, the court delayed action on the increased risk claim, to avoid an award based on pure speculation. [FN170]

Comparably, in Hagerty, a seaman who was accidently soaked with toxic chemicals in the course of his employment, brought an action against his employer for the increased risk of contracting cancer. [FN171] Following its own precedent, as well as that of other ***117** jurisdictions, [FN172] the court concluded that a plaintiff could recover only where he could show that the toxic exposure more probably than not will lead to cancer. [FN173]

Consequently, the Hagerty standard requires evidence that the increased risk of cancer exceeds the fifty percent threshold. For most toxic substances the absolute risk of disease development is far less than fifty percent. [FN174] With this standard, it remains exceedingly difficult for a plaintiff to succeed. [FN175] In fact, recovery for potential future injury is either acknowledged or denied depending upon its likelihood. [FN176] This prevailing all or none approach is based on the increased risk exceeding fifty percent. [FN177]

In more traditional tort cases, some courts have allowed recovery through application of a standard based on the "extent of injury" doctrine. [FN178] This allows a plaintiff to recover damages for future illnesses that may result from a present injury, but that are not reasonably likely to develop. [FN179] Under this doctrine, a present injury still must be shown, but plaintiffs need only establish with "reasonable certainty" their enhanced risk of disease formation. [FN180] However, the courts have applied this rule only where gross physical injuries have occurred. [FN181]

For the "extent of injury" doctrine to apply to hazardous chemical litigation, recognition of genetic or cellular damage as the present injury would be required. [FN182] No court involved in hazardous chemical litigation has adopted such a view, although the Sterling v. Velsicol Chemical Corp. [FN183] court and Justice Handler in the Ayers dissent, have come closest to recognizing that such damage represents a present physical injury. [FN184]

In Sterling, a class action suit was brought by residents living within three miles of defendant's chemical waste burial site, as a result of the contamination of their home well water. [FN185] Velsicol operated the burial site for nine years, during which the site was filled with more than 300,000 fifty-five gallon drums of ultrahazardous chemical waste and hundreds of boxes of ultrahazardous dry chemical waste consisting of at least eleven ***118** highly toxic chemicals. [FN186] The five representative plaintiffs sought compensatory damages for increased susceptibility to disease. [FN187]

Following a traditional tort law approach, [FN188] the Sterling court was willing to define increased susceptibility as a presently existing condition, contingent upon support from scientific expert testimony. [FN189] The court stated that if a reasonable degree of scientific certainty exists to warrant a claim for a present condition of enhanced or increased susceptibility to disease, then, it would be for the jury to determine whether to award damages. [FN190] In this case, the court made a significant leap in recognizing enhanced susceptibility as an existing condition and not as a speculative future injury. Nonetheless, a limitation continues as the Sterling court still articulates a need for proof of a presently existing condition, even though this condition is that of enhanced susceptibility to disease.

Many commentators have strongly argued for judicial recognition of an increased risk right of recovery, independent of the need to prove a present injury. [FN191] With the Ayers litigation, there was much anticipation that the New Jersey Supreme Court would be the court to clear the way for victims to recover on the basis of increased risk alone. [FN192]

In Ayers, residents brought an action against the township for maintaining a landfill of toxic pollutants which leached into plaintiffs' well water causing contamination. [FN193] Without any evidence of disease, the plaintiffs brought a claim, inter

alia, for enhanced risk of future illness attributable to the exposure. [FN194] The trial court applied the reasonable probability test, and determined that an actual manifestation of physical injury attributable to ***119** the exposure was required before a damage claim could be sustained. [FN195] This approach was affirmed by the appellate court. [FN196] In a lengthy opinion, the Supreme Court debated whether increased risk standing alone is actionable. [FN197]

As in Anderson, [FN198] significant policy concerns, led the Ayers court to determine that the risks of recognizing a cause of action for unquantified enhanced risk outweighed the benefits. [FN199] The court was concerned that such a recognition would increase insurance rates, expose the tort system to substantial litigation that would be difficult to manage and resolve, and, given the speculative quality of an unquantified claim, would burden the judges and juries with damage assessments absent clear guidelines. [FN200] As such, the court declined to sustain the cause of action.

The Ayers court did, however, qualify their decision. Recognizing that some plaintiffs would not be compensated for their injuries due to the difficulties of proving causation, the majority requested a legislative solution. [FN201] The court suggested a statutory remedy which would ease the burden of causation in toxic tort cases, when statistically significant incidences of disease were established. [FN202] Additionally, the court emphasized that they were not deciding whether a plaintiff could maintain a claim of enhanced risk supported by testimony demonstrating that onset of disease is reasonably probable. [FN203] Instead, the majority left the door open, suggesting that a quantified risk supported by expert testimony might be actionable.

In a dissenting opinion, Justice Handler, criticized the majority's focus on the inability of measuring the risk rather than the fact of the contamination. [FN204] Comparable to the court in Sterling, the Ayers dissent argues that plaintiffs have suffered an injury; the injury of genetic and cellular damage caused by chemical mutagenic agents. [FN205] Although recognizing that genetic and cellular damage might constitute a new tort injury, Justice Handler noted that, "where new forms of injury have been put before the courts, the courts have developed procedures, standards and formulas for determining appropriate compensation." [FN206] As such, " t o deny ... redress for ... injuries merely because damages cannot be measured with precise exactitude would constitute a perversion of fundamental principles of justice." [FN207]

Unfortunately, Ayers exemplifies that the need to redress these newer forms of injury, has not yet overcome the courts continuing concerns over mere speculation and potentially limitless litigation and liability. Repeatedly courts, as in Ayers, deny compensation, while they request more precise quantification of increased risk and seek a legislative solution to the causation problem associated with the lack of risk assessment information.

***120** Despite these judicial requests for legislative action and actual debate during the enactment of SARA, Congress has been and continues to be reluctant to find a remedy for easing victims burden of proving causation. [FN208] Two main fears associated with creating a legislative solution paralyze Congress. The first is that creating a global remedy would be tantamount to creating a national health insurance system for all sufferers of long-latency disease. [FN209] Comparable to the judicial concern, Congress' second concern is that the floodgates will open to excessive litigation. [FN210] Until some express legislative action occurs, victims must rely on judicial modification of the tort law system. [FN211] But, perhaps a legislative solution has occurred and more precise quantification is upon the horizon.

V. THE POTENTIAL IMPACT OF THE "NEW" ATSDR

With SARA, Congress has greatly expanded the health authorities section of CERCLA. [FN212] Although, the original 1980 Superfund Act established the Agency for Toxic Substances and Disease Registry (ATSDR), it is the 1986 SARA legislation which has imposed specific, nondiscretionary duties on the ATSDR. [FN213] Substantial new provisions call for the preparation of toxicological profiles and health assessments within a statutory timetable. [FN214] Additionally, the ATSDR is now the principal agency responsible for health related activities with respect to the release of hazardous substances in the environment. [FN215] Furthermore, inadequate information on the health effect of a substance, requires the ATSDR Administrator to initiate a research program to ascertain the necessary information. [FN216]

It is believed that the ATSDR studies will be of enormous value. [FN217] Despite the range of specialities required to collect and analyze the health effects information, this information will be compiled and located in one place and will be the latest available. [FN218] "At ***121** a minimum, this ... information will provide a solid basis for calculation of a person's increased risk," as well as, provide an increased source of data for litigants. [FN219]

Consequently, the SARA legislation may have indirectly provided a remedy to the causation barrier. Through the expanded

authority of the ATSDR, the potential exists for increased risk assessment calculability. The centralization of the hazardous substance health effects information within the ATSDR, can only lead to a decrease in the alleged knowledge gaps within risk assessment.

VI. CONCLUSION

Over the past 10-15 years there have been major advances in analytical chemistry. [FN220] Toxicity, now, is routinely discussed in parts per billion or even parts per trillion. [FN221] Increasingly, science is able to establish that a particular exposure subjects a given plaintiff to an increased risk of developing cancer. [FN222] Unfortunately, scientific information is not easily translated within toxic tort litigation. A lack of scientific certitude surrounding risk assessment, often prevents plaintiffs from proving that a given harmful chemical caused plaintiff's harm. All too often, proof of causation has been an insurmountable barrier to victim compensation.

With the SARA Amendments bolstering of the ATSDR, the potential for increased calculability of risk assessment through the ATSDR, certainly exists. More likely than not, this will result in increased scientific certainty. Nonetheless, whether the information to be obtained by the ATSDR will be judicially accepted as evidence sufficient to overcome the burden of causation, remains to be seen.

Presently, the judicial system appears to be yielding to pressure to redress injuries from toxic chemical exposure. The major step taken by the Sterling court and the Ayers dissent, recognizing that genetic and cellular damage are present injuries, should lead to victim compensation for the risk of future disease stemming from these present injuries. Whether other courts will adopt this perspective, also, remains to be seen.

Although the concept of risk has evolved to the point of allowing plaintiffs to recover when there exists strong evidence of exposure to a significant risk, hopefully, future courts will recognize as well, that absolute scientific knowledge is not required in order to compensate exposure victims. As per Judge Jenkins, "dispute resolution demands rational decisions, and not perfect knowledge." [FN223] The judicial focus must shift from excessive concern over certainty, reaching the fifty percent threshold of probability, and the fear of compensating an event, "the potential injury," that may never occur, to recognizing that an actual injury has occurred which must be redressed. The wealth of existent risk information coupled with the information gathering ability of the statutorily bolstered ATDSR should be sufficient to quantify the increased risk and meet plaintiffs evidentiary burdens to overcome proving causation. Therefore, following from Justice Handler, increasingly, the courts should move towards acknowledging that sufficient risk assessment information exists to allow the judge or jury to compensate victims where the evidence shows a quantifiable and reasonable probability for increased risk of disease.

[FNa]. Maria Matteo is a second year student at the Temple University School of Law and an Assistant Editor of the Journal. B.A., University of Pennsylvania, 1979.

[FN12]. Trauberman, Statutory Reform of "Toxic Torts": Relieving Legal, Scientific and Economic Burdens on the Chemical Victim, 7 HARV.ENVTL.L.REV. 177, 203-04 (1983).

[FN13]. CERCLA, 42 U.S.C. §§ 9601-9657 (1982) (commonly known as Superfund).

[FN14]. Ayers, 106 N.J. at 580-81, 525 A.2d at 298-99; See, SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 14-19; See generally, Zazzali & Grad, Hazardous Wastes: New Rights and Remedies? The Report and Recommendation of the Superfund Study Group, 13 SETON HALL L.REV. 446 (1983) [hereinafter Zazzali & Grad].

[FN15]. 106 N.J. at 581, 525 A.2d at 299.

[FN16]. Id.; See e.g., SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 20-4; Ginsberg & Weiss, Common Law Liability of Toxic Torts: A Phantom Remedy, 9 HOFSTRA L.REV. 859, 920-30 (1981) [hereinafter Ginsberg & Weiss]; Rosenberg, The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System, 97 HARV.L.REV. 851, 855-59 (1984) [hereinafter Rosenberg]; But see, Lynch, Benton & Pagliaro, On the Frontier of Toxic Tort Liability: Evolution or Abdication?, 6 TEMP.ENVTL.L. & TECH.J. 1 (1987).

[FN17]. 106 N.J. at 581, 525 A.2d at 299; See SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 11-15.

[FN18]. 106 N.J. at 581, 525 A.2d at 299.

[FN19]. Note, Increased Risk of Disease from Hazardous Waste: A Proposal for Judicial Relief, 60 WASH.L.REV. 635, 636-37 (1985) (authored by Brent Carson) [hereinafter Note, Increased Risk of Disease].

[FN20]. 106 N.J. at 582, 525 A.2d at 299; See Farber, supra note 5, at 1225; Note, Increased Risk of Cancer As An Actionable Injury, 18 GA.L.REV. 563, 575 (1984) (authored by Barton C. Legum) [hereinafter Note, Increased Risk of Cancer].

[FN21]. 106 N.J. at 582, 525 A.2d at 300.

[FN22]. Id.

[FN23]. CERCLA, 42 U.S.C. § 9612(d); SARA, 42 U.S.C.A. § 9658 (West Supp.1987)).

[FN24]. Id.; 106 N.J. at 582, 525 A.2d at 300.

[FN25]. See Note, Will SARA Smile On Citizen Groups?, 6 TEMP.ENVTL.L. & TECH.J. 97, 103-04 (1987) (authored by William J. Cluck) [hereinafter Note, Will SARA Smile?]; Note, Increased Risk of Cancer, supra note 20, at 575.

[FN26]. Aetna Ins. Co. v. Gilchrist Bros., Inc., 85 N.J. 550, 556-57, 428 A.2d 1254, 1257 (1981).

[FN27]. Id.; 106 N.J. at 583, 525 A.2d at 300.

[FN28]. Ayers, 106 N.J. at 583, 525 A.2d at 300 citing Hagerty v. L. & L. Marine Services, Inc., 788 F.2d 315, 320-21 (5th Cir.), modified on other grounds, 797 F.2d 256 (5th Cir.1986) and Eagle-Picher Indus. v. Cox, 481 So.2d 517, 519-21 (Fla.Dist.Ct.App. 1985).

[FN29]. Trauberman, Developments in the Law--Toxic Waste Litigation, 99 HARV.L.REV. 1458, 1617-30 (1986); Note, Tort Actions, supra note 3, at 853-54.

[FN30]. Instead of the older terms, "qualitative" and "quantitative" risk assessment, the National Academy of Science (NAS) Committee on Risk Assessment uses the terms: hazard identification, dose-response assessment, exposure assessment, and risk characterization. These terms more clearly describe the separate analytic steps in a risk assessment. Hazard identification determines whether exposure to an agent increases the incidence of an adverse condition. Dose-response assessment describes the relationship between the level of exposure or the dose, and the incidence of disease. This requires extrapolating from high to low doses and converting from animal to equivalent human doses. Exposure assessment estimates the frequency, duration and intensity of human exposures to the agent. Lastly, risk characterizaion uses the information from both the dose-response and exposure assessments to estimate the expected incidence of adverse health effects. OTA, IDENTIFYING CARCINOGENS, supra note 4, at 27.

[FN31]. Note, Increased Risk of Disease, supra note 19, at 638.

[FN32]. A toxicological profile consists of results from analyses of a chemical for its adverse human health effects.

[FN33]. For the definition of hazard identification, See supra note 30. Whereas, hazard assessment describes the toxicological impact of exposure to chemicals in terms of critical toxicity values (CTV). These CTVs are considered an intrinsic property of a chemical. As specific toxicological endpoints, the acute CTV is the effect due to a single exposure, and the chronic CTV is an estimate of the integrated lifetime exposure. Severn, Exposure Assessment, 21 ENV'T.SCI.TECH. 1159, 1159-60 (1987) [hereinafter Severn].

[FN34]. Id. at 1159.

[FN35]. Tomar & Spielberg, New Developments in the Law of Toxic Torts in New Jersey, 18 RUTGERS L.J. 73 n. 3 (1987).

[FN36]. Id. at 73 n. 2.

[FN37]. Id. at 73 n. 3.

[FN38]. There are three major groupings of cancer: (1) carcinomas arise in the epithelial, i.e., skin, (2) sarcomas affect fibrous tissues and blood vessels, and (3) leukemias and lymphomas arise in the blood forming cells of the bone marrow and the lymph nodes. J. CAIRNS, CANCER: SCIENCE AND SOCIETY 20-22 (1978) as cited in Note, Tort Actions, supra note 3, at 848 n. 34.

[FN39]. Note, Quantitative Risk Assessment in Regulation of Environmental Carcinogens, 4 HARV.ENVTL.L.REV. 89 (1980) (authored by James P. Leape) [hereinafter Note, Quantitative].

[FN40]. Id.

[FN41]. Endogenous factors include metabolic or other imbalances associated with age or genetic makeup. Wilkinson, supra note 2, at 844.

[FN42]. Exogenous factors include diet, lifestyle, and exposure to chemicals both natural and synthetic. Wilkinson, supra note 2, at 844.

[FN43]. Id.

[FN44]. Office of Science and Technology Policy, Chemical Carcinogens: A Review of the Science and Its Associated Principles, 50 FED.REG. 10,371-442 (1985) [hereinafter OST Policy, Chemical Carcinogens]; Note, Quantitative, supra note 39, at 90.

[FN45]. Wilkinson, supra note 2, at 844.

[FN46]. OST Policy, Chemical Carcinogens, supra note 44; Note, Quantitative, supra note 39 at 90.

[FN47]. OST Policy, Chemical Carcinogens, supra note 44; Note, Quantitative, supra note 39 at 90.

[FN48]. Note, Increased Risk of Cancer, supra note 20, at 563 n. 4.

[FN49]. Wilkinson, supra note 2, at 845.

[FN50]. Id.

[FN51]. Id. at 844; See also Note, Quantitative, supra note 39, at 90- 91.

[FN52]. Id. at 844; See also Note, Quantitative, supra note 39, at 90- 91.

[FN53]. Note, Quantitative, supra note 39, at 91.

[FN54]. Id.

[FN55]. Environmental Protection Agency (EPA), Guidelines for Carcinogen Risk Assessment, 51 FED.REG. 33,992-34,014 (1986) [hereinafter EPA Guidelines].

[FN56]. A bioassay is a laboratory comparison of chemically exposed animals to unexposed animals for food intake, weight, clinical course, and any pathological condition (the presence of tumors or other toxicologic effects within tissues and organs). Other toxicological effects include suppression of the immune system, endocrine disturbances, and organ damage. Report of the Interagency Regulatory Liaison Group (IRLG), Work Group on Risk Assessment, Scientific Bases for Identification of Potential Carcinogens and Estimation of Risks, 44 FED.REG. 39,859, 39,865 (1979) [hereinafter IRLG Report, Scientific Bases].

[FN57]. In chemical structure-activity relationships (SAR), carcinogenicity is examined on the basis of the physical or structural aspects of the chemical. EPA Guidelines, supra note 55, at 33,994.

[FN58]. Note, Increased Risk of Disease, supra note 19, at 638 n. 17.

[FN59]. Wilkinson, supra note 2, at 845.

[FN60]. Note, Quantitative, supra note 39, at 93.

[FN61]. Wilkinson, supra note 2, at 845.

[FN62]. Mice, rats, hamsters, guinea pigs and rabbits are used extensively because (1) they have short life spans, (2) they are easier to breed, handle and care for in large numbers, (3) they are inexpensive, and (4) inbred strains exist which allow for control of "background" cancer rates. The background cancer rate is the rate of cancer found naturally in the animal population. IRLG Report, Scientific Bases, supra note 56, at 39,863.

[FN63]. See generally id. at 39,862-864.

[FN64]. Id. at 39,865.

[FN65]. OTA, IDENTIFYING CARCINOGENS, supra note 4, at 26.

[FN66]. Note, Quantitative, supra note 39, at 94.

[FN67]. Id. at 93.

[FN68]. Id. at 94.

[FN69]. See generally Wilkinson, supra note 2, at 845.

[FN70]. Id. at 846.

[FN71]. Id.; Orders of magnitude refers to differences that can be expressed in powers of ten. See generally OTA, IDENTIFYING CARCINOGENS, supra note 4, at 52-53.

[FN72]. Id. at 53; Note, Quantitative, supra note 39, at 101; See generally OST Policy, Chemical Carcinogens, supra note 44 at 10,371-442.

[FN73]. OTA, IDENTIFYING CARCINOGENS, supra note 4, at 53.

[FN74]. Note, Quantitative, supra note 39, at 101; IRLG Report, Scientific Bases, supra note 56, at 39,858-879.

[FN75]. OTA, IDENTIFYING CARCINOGENS, supra note 4, at 53-56.

[FN76]. Id. at 55-56.

[FN77]. Id. at 55.

[FN78]. Wilkinson, supra note 2, at 846.

[FN79]. Note, Quantitative, supra note 39, at 94.

[FN80]. Id.

[FN81]. Id. The results from the different tests are pooled together. Generally, positive findings in either epidemiological studies or in short-term tests override negative findings from animal bioassays. IRLG Report, Scientific Bases, supra note 56, at 39,871.

[FN82]. In vitro assays require administering the chemical agent to a cell outside of the living body and in an artificial environment.

[FN83]. In vivo assays are performed by administering a chemical in the living body of an intact microorganism or organism.

[FN84]. Chromosome aberrations involve breaks in whole chromosomes that may, after cell replication, be rejoined incorrectly, remain broken, or be lost. Maugh, Biological Markers for Chemical Exposure, 215 SCI. 643 (1983).

[FN85]. Sister-chromatid exchange consists of improper genetic material between complementary chromosomal strands. Id. at 644.

[FN86]. Note, Quantitative, supra note 39, at 95-96.

[FN87]. Id. at 96; Wilkinson, supra note 2, at 846.

[FN88]. Wilkinson, supra note 2, at 846.

[FN89]. Id.

[FN90]. Id.; See also Note, Quantitative, supra note 39, at 96.

[FN91]. Note, Quantitative, supra note 39, at 96.

[FN92]. Id. at 92.

[FN93]. Id.

[FN94]. Dore, A Proposed Standard for Evaluating the Use of Epidemiological Evidence in Toxic Tort and Other Personal Injury Cases, 28 HOW.L.J. 667, 679 (1985) [hereinafter Dore]. See generally Dore, A Commentary on the Use of Epidemiological Evidence In Demonstrating Cause-In-Fact, 7 HARV.ENVTL.L.REV. 429 (1983); Hall & Silbergeld, Reappraising Epidemiology: A Response to Mr. Dore, 7 HARV.ENVTL.L.REV. 441 (1983).

[FN95]. Dore, supra note 94, at 679-80.

[FN96]. Id. at 680.

[FN97]. Id.

[FN98]. Id.

[FN99]. Id.

[FN100]. Id.

[FN101]. Id.

[FN102]. Id.

[FN103]. Id.

[FN104]. Id. at 680-81.

[FN105]. See generally EPA Guidelines, supra note 55, at 33,995-996; IRLG Report, Scientific Bases, supra note 56, at 39,861-862.

[FN106]. Dore, supra note 94, at 694 n. 63 (refers to this calculation as a risk ratio); Note, Increased Risk of Disease, supra note 19, at 639 n. 20 (refers to this calculation as relative risk).

[FN107]. Note, Increased Risk of Disease, supra note 19, at 639 n. 20.

[FN108]. Id.

[FN109]. Dore, supra note 94, at 693-95.

[FN110]. Id. at 693.

[FN111]. Id. at 694-95.

[FN112]. Id. at 679; Farber, supra note 5, at 1220.

[FN113]. Dore, supra note 94, at 679 n. 5.

[FN114]. Note, Quantitative, supra note 39, at 92.

[FN115]. Dore, supra note 94, at 682-83.

[FN116]. Judicial consideration of qualitative evidence, specifically epidemiologic evidence appears in, Ethyl Corp. v. Environmental Protection Agency, 541 F.2d 1, 25, 26 (D.C.Cir.), cert. denied, 426 U.S. 941 (1976). Administratively, the EPA's consideration of this evidence is contained in EPA Guidelines, supra note 55 at 33,992. The categorization of the overall weight of evidence for human carcinogenicity appears at 34,000. This categorization is a three-step process:
(1) the weight of evidence in human or animal studies is summarized; (2) these lines of information are combined to yield a tenative assignment to a category--Category I consists of Group A & B designations, Category II consists of Group C designations, and Category III consists of Group D & E designations; (Group A are Known Human Carcinogens determined through sufficient epidemiological evidence to support a causal association; Group B, Probable Human Carcinogens, are designated through limited epidemiological evidence or no epidemiological data and sufficient evidence from animal studies; Group C, Possible Human Carcinogens, are determined through limited evidence in animal studies in the absence of human data; Group D, Not Classifiable as to Human Carcinogenicity, labelling occurs when there is either inadequate human and animal evidence or when no data is available; Lastly, Group E, Evidence of Non-Carcinogenicity for Humans, is the designation for agents that show no evidence for carcinogenicity in at least two adequate animal tests of different species or in both adequate epidemiological and animal studies; (3) all relevant supportive information is evaluated to see if categorization needs to be modified.
See also Whitehead & Espel, Legal Proof of Causation in Toxic Tort Litigation, 2 Toxics L.Rep.(BNA) 1040, 1042-43 (Feb. 24, 1988) [hereinafter Whitehead & Espel].

[FN117]. Note, Increased Risk of Disease, supra note 19, at 638 n. 18.

[FN118]. Id. at 638 n. 19.

[FN119]. IRLG Report, Scientific Bases, supra note 56, at 39,873-875.

[FN120]. Id.

[FN121]. Id. at 39,858.

[FN122]. See supra note 30.

[FN123]. IRLG Report, Scientific Bases, supra note 56, at 39,872-873.

[FN124]. Id.

[FN125]. Note, Quantitative, supra note 39, at 102.

[FN126]. Id.

[FN127]. Id.

[FN128]. Id. at 102-3.

[FN129]. Id. at 101-3.

[FN130]. Wilkinson, supra note 2, at 846; Panel Report, 225 SCI. 682-87 (1984).

[FN131]. Wilkinson, supra note 2 at 846; D. KREWSKI & J. VAN RYZIN, IN STATISTICS AND RELATED TOPICS, 201-31 (1981).

[FN132]. Severn, supra note 33, at 1159.

[FN133]. Id.

[FN134]. Id. at 1161.

[FN135]. Id.; Dermal exposure is manifested by the amount of the chemical on a pad on the skin. This is representational of the surface concentration, from which a calculation for all exposed body surfaces is made. Inhalation exposure is measured by the amount of chemical trapped on respirator pads or by analyzing the trapped material and estimating a breathing rate. Id.

[FN136]. Id.; There are various conversion formulas which include: (1) the surface-area rule, (2) the direct conversion rule, (3) the standard toxic conversion rule, and (4) the lifetime dose conversion rule. Id.

[FN137]. Id.

[FN138]. Pharmacokinetics is a general term for the various biochemical pathways in the body that activate, metabolize, detoxify, transport and excrete chemicals. OTA, IDENTIFYING CARCINOGENS, supra note 4, at 63.

[FN139]. Severn, supra note 33, at 1161.

[FN140]. See supra note 30, regarding risk characterization.

[FN141]. See SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2.

[FN142]. Id. at ix. (Comments from James L. Oberstar, Chairman of the Subcommittee on Investigations and Oversights).

[FN143]. Coll v. Sherry, 29 N.J. 166, 175, 148 A.2d 481, 486 (1959), as cited in Ayers, 106 N.J. at 578, 525 A.2d at 297-98.

[FN144]. Coll, 29 N.J. at 175, as cited in Ayers, 106 N.J. at 578, 525 A.2d at 297-98.

[FN145]. See Wilkinson, supra note 2, at 843-44, 847.

[FN146]. Black & Lilienfeld, Epidemiological Proof in Toxic Tort Litigation, 52 FORDHAM L.REV. 732, 749 (1984), as cited in SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 11, [hereinafter Black & Lilienfeld].

[FN147]. Black & Lilienfeld, supra note 146, at 735, as cited in SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 11. See also Whitehead & Espel, supra note 116.

[FN148]. SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 13.

[FN149]. See generally Note, Increased Risk of Disease, supra note 19.

[FN150]. Id. at 637-38.

[FN151]. Id. at 638.

[FN152]. SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 13.

[FN153]. 660 F.Supp. 1516 (W.D.Mich.1987). This case is particularly interesting for judicial use of a summary jury to help decrease excessive litigation. See Enslen, Alternative Dispute Resolution: Summary Jury Trial in a Toxic Tort Case, 2 Toxics

L.Rep.(BNA) 1015 (Feb. 17, 1988).

[FN154]. Id. at 1517.

[FN155]. Id. at 1517-18.

[FN156]. Id. at 1519; Additionally, plaintiffs brought a fear of cancer claim. See supra note 8.

[FN157]. Id. at 1524.

[FN158]. 427 Mich. 301, 317, 399 N.W.2d 1, 8 (1986).

[FN159]. King v. Neller, 228 Mich. 15, 22, 199 N.W. 674, 676 (1924), as cited in Stites, 660 F.Supp. at 1524.

[FN160]. 660 F.Supp. at 1519, 1524.

[FN161]. Note, Increased Risk of Disease, supra note 19, at 635 n. 15; Note, Increased Risk of Cancer, supra note 20, at 568 n. 17.

[FN162]. 628 F.Supp. 1219 (D.Mass.1986), appeal docketed, No. 87-1405 (1st Cir. Mar. 15, 1988), 2 Toxics L.Rep.(BNA) 1147 (Mar. 23, 1988). Previously, plaintiffs claimed that defendant's attorneys had suppressed evidence. Their request for a new trial was denied. 2 Toxics L.Rep. (BNA) 561 (Oct. 14, 1987).

[FN163]. 788 F.2d 315 (5th Cir.), modified on other grounds, 797 F.2d 256 (5th Cir.1986).

[FN164]. Anderson, 628 F.Supp. at 1222. Beyond the increased risk of developing future illnesses, plaintiffs brought actions for wrongful death, pain and suffering, emotional distress, recovery for illnesses and other damages and nuisance. See supra note 8.

[FN165]. Id.

[FN166]. Pullen v. Boston Elevated Railway Co., 208 Mass. 356, 357-58, 94 N.E. 469 (1911), as cited in Anderson, 628 F.Supp. at 1222.

[FN167]. Anderson, 628 F.Supp. at 1231.

[FN168]. Id. at 1232.

[FN169]. Id.

[FN170]. Id.

[FN171]. Hagerty, 788 F.2d at 316; Although the scope of this article is limited to litigation involving victims of ambient exposure, Hagerty is included, as plaintiff's exposure was accidental. Other actions brought by plaintiff included claims for cancerphobia and medical expenses. See supra note 8.

[FN172]. See, e.g., Dartez v. Fibreboard Corp., 765 F.2d 456 (5th Cir.1985); Gideon v. Johns-Manville Sales Corp., 761 F.2d 1129 (5th Cir.1985); Laswell v. Brown, 683 F.2d 261 (8th Cir.1982); But see Martin v. City of New Orleans, 678 F.2d 1321 (5th Cir.1982), cert. denied, 459 U.S. 1203 (1983).

[FN173]. Hagerty, 788 F.2d at 319.

[FN174]. Note, Increased Risk of Disease, supra note 19, at 639 n. 20.

[FN175]. See Note, Increased Risk of Cancer, supra note 20, at 568-69.

[FN176]. Id.

[FN177]. Id. at 568 n. 19.

[FN178]. Note, Increased Risk of Disease, supra note 19, at 639-40.

[FN179]. Id.

[FN180]. Id. at 640.

[FN181]. Id.

[FN182]. Id.

[FN183]. 647 F.Supp. 303 (W.D.Tenn.1986), appeal pending, No. 86-6087 (6th Cir. Oct. 6, 1986); excerpts of appellate briefs appear as follows: Velsicol's, 2 Toxics L.Rep.(BNA) 482 (Sept. 23, 1987), Sterling's, 2 Toxics L.Rep.(BNA) 516 (Sept. 30, 1987), and Velsicol's reply brief, 2 Toxics L.Rep.(BNA) (Oct. 7, 1987).

[FN184]. Note, Increased Risk of Disease, supra note 19, at 640-41; See generally Comment, Damages in Genetic Mutation and Chromosomal Breakage: Tort Actions, 26 ST. LOUIS L.J. 105 (1981) (authored by Robert Herman).

[FN185]. Sterling, 647 F.Supp. at 306. Additional claims included trespass, nuisance, common law negligence and strict liability actions. Compensatory damages were sought for inconvenience and disruption of plaintiffs' normal life; fear, distress or emotional injury; as well as increased risk or increased susceptibility. See supra note 8.

[FN186]. Id. Some of the eleven primary chemicals include: carbon tetrachloride, chloroform, chlorobenzene, napthalene, toluene and tetrachlorethylene. Id. at 308.

[FN187]. Id. at 307-8.

[FN188]. See Sterling, 647 F.Supp. at 322, citing Feist v. Sears Roebuck & Co., 267 Or. 402, 517 P.2d 675 (1973) (increased risk of meningitis); Schwegel v. Goldberg, 209 Pa.Super. 280, 228 A.2d 405 (1967) (enhanced risk of epilepsy); Lindsay v. Appleby, 91 Ill.App.3d 705, 414 N.E.2d 885 (1980) (increased risk of seizures); Starlings v. Ski Roundtop Corp., 493 F.Supp. 507 (M.D.Pa.1980).

[FN189]. Sterling, 647 F.Supp. at 322.

[FN190]. Id. at 321-22. This case is being closely watched to see if the Sixth Circuit will reverse or uphold the District Court's actions. Some commentators believe the lower court's findings are not indicative of a new trend in toxic tort litigation and predict a reversal with respect to the increased risk cause of action. See Mitchell, Sterling v. Velsicol Chemical Corp.: Two Views of the Evidence--Exposure and Dose, 2 Toxics L.Rep. (BNA) 900-01 (Jan. 20, 1988). But see Rodricks, Sterling v. Velsicol Chemical Corp.: Two Views on the Evidence--The Case for Causation, 2 Toxics L.Rep. (BNA) 901-02 (Jan. 20, 1988).

[FN191]. See Ayers, 106 N.J. at 596, 525 A.2d at 307; See, e.g., Gale & Goyer, Recovery for Cancerphobia and Increased Risk of Cancer, 15 CUM.L.REV. 723 (1985); Ginsberg & Weiss, supra note 16; Rosenberg, supra note 16; Note, Increased Risk of Disease, supra note 19; Note, Increased Risk of Cancer, supra note 20.

[FN192]. Tomar & Spielberg, supra note 35, at 75-76.

[FN193]. Ayers, 106 N.J. at 565, 525 A.2d at 291.

[FN194]. 106 N.J. at 577, 525 A.2d at 297. Although the court denied the increased risk claim, plaintiffs are not entirely uncompensated. The court held the Township residents were entitled to damages for medical surveillance costs, structured as a court supervised fund, and for quality of life damages, which represented compensation for losses associated with damage to property. 106 N.J. at 572, 606, 525 A.2d at 294, 312. The majority distinguishes the claim for medical surveillance expenses from the claim based on enhanced risk, as they "stand[ ] on a different footing." 106 N.J. at 599, 525 A.2d at 308. However, some may view the medical surveillance award, the $8.2 million court supervised fund, as the actual remedy for the increased risk cause of action. This author purports that the claims seek different forms of compensation, are separate and

were addressed as separate causes of action by the majority. The increased risk claim seeks compensation for an actual sustained injury, comparable to damages for loss of a limb, whereas, the medical surveillance claim seeks compensation for expenses incurred. Furthermore, the majority individually treated these claims. "Despite, [their] dismissal of the enhanced risk claim ..., the court viewed the cost of medical surveillance as an appropriate item of damage...." 106 N.J. at 602, 525 A.2d at 310. This author views compensation for enhanced risk of disease as separate and distinct from compensation for medical surveillance, and, therefore, uncompensated by the Ayers majority.

[FN195]. Ayers v. Jackson Township, 189 N.J.Super. 561, 567-68, 461 A.2d 184, 187, as cited in 106 N.J. at 578, 525 A.2d at 297.

[FN196]. 202 N.J.Super. 106, 493 A.2d 1314.

[FN197]. Ayers, 106 N.J. at 557, 525 A.2d at 287.

[FN198]. Anderson, 628 F.Supp. at 1232.

[FN199]. 106 N.J. at 597-598, 525 A.2d at 307-08.

[FN200]. Id.

[FN201]. 106 N.J. at 598-99, 525 A.2d at 308.

[FN202]. Id.

[FN203]. Id.

[FN204]. 106 N.J. at 613, 525 A.2d at 316. Justice Handler concurred in part and dissented in part. Of primary interest with respect to this article, is Justice Handler's dissent.

[FN205]. 106 N.J. at 615, 525 A.2d at 316-17.

[FN206]. 106 N.J. at 618, 525 A.2d at 318.

[FN207]. 106 N.J. at 617, 525 A.2d at 318, citing Berman v. Allan, 80 N.J. 421, 433, 404 A.2d 8 (1979).

[FN208]. Legislators have toyed with the Zazzali & Grad suggestions of a two-tiered relief system, the first tier being a newly created state supervised administrative mechanism that would compensate victims, the second tier would remain the existant tort law system. The use of rebuttable presumptions and shifting burdens of proof have also been suggested. SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 14-19; See generally Zazzali & Grad, supra note 14; Note, Will SARA Smile?, supra note 25, at 102-06. Some commentators have suggested different compensation schemes. See Farber, supra note 5, (most likely victims gets full compensation, while the least likely victim gets nothing); Note, Increased Risk of Cancer, supra note 20, (following a proportional basis formula, the percentage risk of future illness would yield the percentage of damages).

[FN209]. SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 19 (statement by Leslie Cheek of Crum & Forster's).

[FN210]. See generally id. at 18.

[FN211]. Id. at 21-24, 26.

[FN212]. Section 110 of SARA, amending CERCLA § 104(i), 42 U.S.C. § 9604i; See, e.g., Note, Will SARA Smile?, supra note 25, at 106-10; Rogers, Potential Use of Toxicological and Exposure Information Developed Under the Superfund Amendments of 1986, in Toxic Tort Suits, ALI-ABA SEMINAR, HAZARDOUS WASTES, SUPERFUND AND TOXIC SUBSTANCES, (October 29-31, 1987) 231, 234-36 [hereinafter Rogers]; Siegel, Agency for Toxic Substances and Disease Registry Health-Related Activities, ALI-ABA SEMINAR, HAZARDOUS WASTES, SUPERFUND AND TOXIC SUBSTANCES, (October 29-31, 1987) 17, 21 [hereinafter Siegel].

[FN213]. Rogers, supra note 208, at 235-36.

[FN214]. Id. at 236; See also Note, Will SARA Smile?, supra note 25, at 107-09; Siegel, supra note 208, at 21.

[FN215]. Siegel, supra note 208, at 21.

[FN216]. Rogers, supra note 208, at 242.

[FN217]. Id. at 241.

[FN218]. Id.; Information will be coordinated from experts in the areas of toxicology, pathology, pharmacology and statistics.

[FN219]. Id. at 251.

[FN220]. SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 5.

[FN221]. Id.

[FN222]. Note, Increased Risk of Cancer, supra note 20, at 576-77.

[FN223]. Allen v. United States, 588 F.Supp. 247 (D.Utah 1984) as cited in SUBCOMMITTEE REPORT, VICTIMS COMPENSATION, supra note 2, at 22-23.

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