THE CONFLICT BETWEEN LAW AND SCIENCE

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INTRODUCTION

In 1982 a small group of Cape Breton landowners sought a *quia timet* injunction to prevent the spraying of a dioxin-contaminated herbicide near their homes by a Swedish-owned multinational forest company.[i] They were concerned that the dioxin would irreparably damage their health and their natural environment. Following an exhaustive review of the voluminous evidence presented by both the plaintiffs and the defendant, the trial judge concluded:[ii]

> *This matter thus reduces itself now to the single question. Have the plaintiffs offered sufficient proof that there is a serious risk of health and that such serious risk of health will occur if the spraying of the substances here is permitted to take place?*

By confining the legal issue to burden and onus rules, the Nova Scotia Court merely echoed a long-standing precedent for resolving legal disputes.

This paper argues that litigants attempting to use traditional common law doctrines to minimize the potential adverse impact of environmental risk activities, such as spraying pesticides, invariably lose. Part II of this paper explains the inherent limitations in the law and the science of environmental risk activities which promote this result. Part III explores the desirability of extending tort doctrines to allow recovery in "environmental risk" lawsuits. Part IV briefly canvasses various mechanisms for moderating onus and burden rules in appropriate circumstances. Where appropriate, the Noval Scotia Herbicide Trial will be used for illustrative purposes.

I. PROOF OF CAUSE/EFFECT RELATIONSHIPS: LAW vs. SCIENCE

A. Legal Burdens and the Onus of Proof

Historically, the procedural backbone for resolving legal conflict in our society has focused on the rules dealing with proof of harm and burden of proof.[iii] Generally, the injured party has the onus of presenting to the factfinder enough information to prove the alleged injury, and to prove that the injury was in fact caused by the wrongful conduct of the injuring party. Criminal law affords an excellent example of these rules. A person is presumed innocent until the State can prove the guilt of that person beyond a reasonable doubt. If the accused person is not demonstrably proven guilty of the particular charges brought by the State, the case has not been made out. The accused must be set free, without restrictions. Thus, traditional dispute resolution principles irrebutably presume that the inability to establish enough information to prove guilt means conclusive evidence of innocence. In the result, an accused person who is *in fact* guilty of a crime will be declared innocent whenever the State cannot establish enough information to prove that the accused caused the illegal activity. Society consciously promotes this result because high priority is accorded to the principles of individual liberty and freedom from coercion by the State. These principles are exemplified in the maxim: "Better that 100 guilty people be declared innocent than one innocent person be declared guilty."[iv]

Civil lawsuits adopt similar notions of proof of harm and burden of proof. In a typical tort action, the party alleging injury must prove, on a balance of probabilities, three elements: injury in fact, unacceptable conduct by a particular defendant and a causal nexus between the injury and the unacceptable conduct of the defendant.[v]

The allocation of the burden of proof to the party alleging harm (the State or a plaintiff) normally represents an efficient and fair mechanism for regulating legal proceedings. The field of tort law is illustrative.[vi] Tort liability concerns itself with distributing losses which are inevitable by-products of organized society. Practically all human activities involve some risk of interference with the rights of others. But tort law does not attempt to shift the loss in every case where one person's conduct adversely impacts upon the interest of another. Rather, tort liability compensates a party who has been injured, by compelling the wrongdoer to pay for the damage that has been done to that injured party where the harm to the individual is greater than he ought reasonably be expected to bear in the circumstances. The function of tort law is to determine how best to allocate these losses, in the interest of the public good.[vii]

Procedural rules placing the onus and burden of proof upon the party alleging injury generally help to uphold these principles. A legal regime which allows a person to initiate a lawsuit without presenting appropriate information to justify a change to the status quo would be inefficient.[viii] Such a system would promote the unreasonable use of judicial processes because there would be minimal costs attached to the plaintiff's initiation of a lawsuit. The plaintiff would have little incentive to proceed with prudence. In addition, it would be difficult and costly to the judicial process, and to defendants, to assess the merits of the case. Defendants would be forced to speculate about the case of the plaintiff and produce volumes of evidence, much of which would be of doubtful relevance.

In addition to arguments of efficiency, considerations of fairness dictate that plaintiffs should not be able to restrain the conduct of a defendant simply by instituting a lawsuit. As one commentator observed:[ix]

> *[T]he plaintiff's starting point on the road to a tort recovery is to be able to pick the defendant out of the crowd; that is, to demonstrate factually that* there is a reason why this particular person is the defendant. (emphasis added)

The concept of fairness is most evident in criminal law. The presumption of innocence is the cornerstone of criminal law in most civilized societies.[x] Our society is opposed in principle to allowing the State to interfere with the freedoms and liberties of an individual without first showing good cause as to why that interference is justifiable. This is particularly so where the complex and sophisticated fact finding capability of the State gives a powerful advantage over an individual accused.

The following illustration highlights a reason for the development of burden and onus allocations. In many civil proceedings which may be characterized as "environmental" cases, a non-consumer of a resource is inevitably the injured party, while the consumer of a resource is inevitably the injuring party.[xi] For example, an individual living downstream from a pulp and paper mill may use the shared river for drinking purposes. The non-consumptive drinking use will never restrict the use of the water to carry off effluent. However, the mill's use of the water as a sewer (the consumptive use) will inevitably interfere with the use for drinking purposes. A legal system which allocates the burden of proof upon the non-consumptive user, *ex hypothesi* allows the consumptive activity to continue unabated until such time as a lawsuit is initiated. Further, the non-consumptive user must prove, on a balance of probabilities, injury and causation-in-fact by the resource consumer. In cases of doubt about any material component of the lawsuit, the consumptive user will prevail.[xii] Thus, the common law has traditionally encouraged economically productive activity by casting the onus and the burden of proof on the non-consumer. This preference reflected society's approval of industrial expansion, frontier settlement and economic growth over the conservation of natural resources. This was based in large part upon an understandable, but drastic misconception that natural resources were inexhaustible.

A final argument supports the historical allocation of burden and onus rules.[xiii] In the vast majority of legal disputes, the wrongful conduct of the defendant and the resulting damage to the plaintiff are contemporanious and obvious. Indeed, identification of the injury has rarely been a problem. Most lawsuits have dealt with accidental injuries, wherein a relatively sudden cause gives rise to an immediate and visible effect (injury) to person or property. In most tort cases the person alleging injury can point to identifiable harm over and above that which is associated with the everyday risks of life. In the result, the injured party can immediately gather all of the relevant information needed to prove the harm and to prove that the harm was caused by the wrongdoer. This information can then be preserved and presented to the decision-maker charged with resolving the dispute. In most disputes, the decision-maker would have available *all* of the relevant information needed to make an equitable decision. The decision maker could fairly conclude upon the *available* information that the plaintiff had proven the case or had not proven the case. Thus, the contemporaneous nature of the cause-effect nexus is well suited to allocating burden and onus rules upon a plaintiff.

B. Characterizing Environmental Risk Activities

Environmentalism in its modern form was born during the 1960s in the writing of Rachel Carson, René Dubos and Barry Commoner.[xiv] During this time there was general agreement over the nature of environmental insults and the methodologies needed to resolve them.[xv] This was so because the data needed to assess and resolve problems such as air pollution and the eutrophication of water systems was readily available and understandable. By the mid-1970s, observers of environmental disruption became more sophisticated. They began to realize that a special category of serious environmental insults had dimensions that were not amenable to simple solutions. Society was being confronted with newly recognized, poorly understood and amazingly complex environmental *risk* problems.

The term "environmental risk" describes an activity which has apparent benefits, but which has concomitant *potential* adverse consequences, the underlying probabilities of which are *uncertain* and knowable, if at all, at some future time.[xvi] This is so because the scientific information needed to assess the potential of a toxic substance to cause harm, and the extent of harm, is either equivocal, or simply not available. Examples of environmental risks include the risk of groundwater contamination near hazardous waste disposal facilities, the risk of leakage from toxic chemicals storage sites and the risk of using pesticides which may adversely affect human health or damage the receiving ecosystem.

The fact that the information necessary to define the boundaries of a risk activity is uncertain or unavailable is *fundamental* to understanding the conflict between law and science. Therefore, the next subsection will describe a series of legal paradigms which highlight the reasons *why* scientific information concerning risk activities is usually uncertain or unavailable, and its consequent impact in a courtroom setting.[xvii]

C. Environmental Risk Problems in Court

When attempting to establish causation, a litigant exposed to a toxic chemical may realistically encounter one of two scenarios. Either one will defeat the case if traditional procedural rules are adopted. In the first scenario, the injury of the plaintiff is obvious (cancer, birth defect, etc.), but the plaintiff cannot establish that it was more likely than not that a particular wrongdoer in fact caused the injury in question. In the second scenario, the defendant is obvious, but the plaintiff cannot establish that it is more likely than not that he has, or will develop, a particular injury. The reasons for, and the legal consequences of each paradigm are discussed below.

1. Indeterminate Defendants

a) One of a Number of Wrongdoers

In *Cook* v. *Lewis*,[xviii] the plaintiff suffered injury caused by one of two hunters, both of whom fired negligently in his direction. Unfortunately, the plaintiff could not prove which one of the two hunters in fact caused his injury. To assist the faultless victim, the Court shifted the burden onto each hunter to prove that he was not responsible for the plaintiff's injury. The consequence of this ruling is that liability was imposed on someone who did not cause the harm. The Court reasoned that it was impossible for the plaintiff to establish who caused the injury. As between an innocent victim and negligent defendants it would be fairer that the burden be placed upon each defendant to exculpate himself, even if he had not in fact caused the harm. As stated by Fleming, the "law prefers a 50% chance of doing justice to the certainty of doing injustice."[xix]

The doctrine developed in *Cook* v. *Lewis* was extended in the California Supreme Court, in *Sindell* v. *Abbott Laboratories*.[xx] The plaintiff, a cancer victim, was able to establish that her injury resulted from the ingestion of diethylstilbestrol (DES) administered to her mother during pregnancy. However, she was not able to produce any evidence to point to the particular manufacturer(s) from whom her mother purchased the drug. She therefore sued the nine major manufacturers of the drug, arguing that each should bear the burden of proving that it had not produced the drugs her mother had ingested. The Court upheld her suit as an extension of the *Cook* v. *Lewis* principle.[xxi] When many of the manufacturers were unable to absolve themselves, the court held each liable in proportion to its share of the DES market. The court stated:

> *[i]n our contemporary complex industrialized society, advances in science and technology create fungible goods which may harm consumers and which cannot be traced to any specific producer. The response of the courts can either be to adhere rigidly to prior doctrine, denying recovery to those injured by such products, or to fashion remedies to meet these changing needs [...] [W]e acknowledge that some adaptation of the rules of causation and liability may be appropriate in these recurring circumstances.*[xxii]

These cases indicate a willingness by the courts to expand causation principles to assist worthy plaintiffs who are unable, *through no fault of their own,* to identify the cause(s) in fact of their injuries.[xxiii]

b) Wrongdoer May Not Have Caused Injury

Exposure to toxic chemicals has the potential to cause a broad range of physical harm, including cancer, genetic mutations, central nervous system disorders, fetal and birth injuries, lung disease, and sterility.[xxiv] A tremendous difficulty that must be confronted by toxic tort litigants is the fact that virtually all of these adverse health impacts are also caused by "natural" events.[xxv] Science does not understand the etiology (the established pathways explaining how and why an exposure produces an effect) of many diseases.[xxvi] Thus, it is often impossible to distinguish between a disease induced by toxic chemicals and the normal background incidence of the same disease.

The inability to distinguish between natural causes of a disease (which would not be recoverable in a tort action) and human causes of a disease, reflects the inability of the natural sciences to establish cause-effect relationships with any degree of certainty.

Two types of evidence are used to identify toxic substances that may pose unacceptable health hazards: (1) epidemiologic evidence derives from studies of exposed human population, and (2) toxicological evidence derived from acute, short-term and long-term experiments on animals.[xxvii]

i) Epidemiological Proof of Probability

Epidemiology is the study of the distribution of disease and the search for causes of the observed distribution in human populations.[xxviii] The two main types of investigations used to develop evidence of a particular chemical hazard are "cohort studies" and "case-control studies".

Cohort studies involve the comparison of the rates of a particular disease in two groups that were differentially exposed to a substance. Ideally the two groups are identical in demographic makeup (age, sex, race, living and working environment, etc.) except that one group was totally unexposed to the chemical in question and the other was significantly exposed to the chemical.

Case-control studies involve a comparison of people who already exhibit a given disease with a group that is identical demographically, except that they do not exhibit the disease. The purpose of the study is to ascertain if the two groups differ in exposure to the chemical under investigation. Case-control studies are usually retrospective. Researchers take a group that has contracted the disease, and collect data back in time to compare characteristics of that group with those of the control group. The investigator then searches for distinguishing factors that might have caused the disease. Cohort studies can be either retrospective or prospective. In a prospective study, a sample population exposed to a toxic suspect chemical is followed forward in time. The incidence of disease is recorded and then compared with the disease rate of the control group. In a retrospective study, the researcher examines the historical record of a group of people exposed to the suspect chemical over a definite time period to establish their disease rate. That rate is then compared with the disease rate for a similar group not exposed to the chemical.

A number of practical and theoretical limitations severely restrict the value of epidemiological studies in a courtroom.[xxix] Retrospective studies require accurate and well-documented data. Information concerning the past and present whereabouts of individuals, exposed or diseased, lengths of exposure, and exposure rates is critical. Effective studies often need to go back in time for 10 to 20 years. In many cases, records have not been kept, data about past events is sketchy or unavailable and members of the exposed population are difficult to track. In addition, it is often impossible to segregate and measure the effect of a suspect chemical because of unknown exposures to other toxic substances. Retrospective studies are susceptible to bias because both the researcher and the subject are usually aware of the hypothesized causal connection between exposure and disease.

Prospective cohort studies have additional problems. Since many diseases of concern have lengthy latency periods, all cohort studies require large numbers of subjects and a long follow-up period. There are ethical problems involved in exposing subjects to suspect chemicals in prospective studies. In addition, the criteria and methodology may change over time as new information becomes available. Subjects may tire of the study and respond sporadically or not at all.

All epidemiological studies are extremely expensive and time consuming. In addition, the selection of control groups must be done with extreme care to ensure as much similarity as possible between the groups being studied.

Independent of practical limitations, a number of serious theoretical problems restrict the usefulness of these studies in a courtroom. The mathematical models used to quantify the risk of harm associated with exposure to particular substances are highly complex and fraught with uncertainty. There is no single correct protocol for analyzing the results of a particular epidemiological study. Different models, which are considered equally legitimate within the scientific community, may give rise to radically different conclusions.[xxx]

Even if an association between a chemical and a disease is found, it must always be understood that epidemiology, by definition, is a study of populations, not individuals.[xxxi] Epidemiology may assist in answering the question: "Could the substance have caused this disease?" It offers much less guidance when attempting to answer the question: "Did this particular substance cause the particular disease complained of by this particular plaintiff?" At best, epidemiology can only give statistical, probabilistic estimates of injury and causation.

The following example (which will be used throughout this paper) is illustrative.[xxxii] Suppose that a spill exposes a group of 100 workers (hereafter, the workers) to a chemical suspected of being a carcinogen. Epidemologists would assemble another group of 100 workers with similar demographic characteristics (age, sex, race, etc.) who were not exposed to the spill. The two groups would be observed in a prospective cohort study to determine the number of persons in each group that developed particular diseases. Assume that ten years after the spill, the investigators find the following results:

FIGURE I
Prospective Cohort Study:
Development of Liver Cancer Among
100 Exposed and 100 Unexposed Workers

GROUP	TOTAL	LIVER CANCER	NO LIVER CANCER
Exposed	100	15	85
Unexposed	100	10	90
Total	200	25	175

The risk of contracting liver cancer after ten years for the unexposed group is 10/100 or .10. This figure is extremely important; it establishes that the chemical in question cannot be the only cause of liver cancer since the disease occurs in the *unexposed* group as well. The exposed group has a risk of contracting liver cancer of 15/100 or .15. Thus, the increased risk of contracting liver cancer as a result of the exposure to the chemical is .15 minus .10, or .05 (5/100). Stated another way, it could be said that of the 15 liver cancers in the exposed group, 10 were caused by natural factors and 5 were caused by the exposure to the chemical. Therefore one individual in the exposed group can only show a 33% probability (5/15) that his particular injury was caused by the toxic chemical.

In a lawsuit against the chemical company, a victim could use the epidemiology study (assuming it could withstand procedural and theoretical scrutiny) to show that the defendant caused an increased incidence of liver cancer among a *group* of workers. However, the individual must prove, on a balance of probabilities, that his injury was in fact caused by the defendant's conduct. Two tests are commonly used to establish causation-in-fact.[xxxiii] Under the "but-for" test, the plaintiff must establish that but-for the defendant's conduct the injury would not have occurred. The "material and contributing factor" test requires that the plaintiff shows that the conduct of the defendant contributed materially to his injury. Our worker cannot satisfy either test because there is a 66% chance that the liver cancer was caused by an agency acting totally independent of the defendant. When the independent or the concerted conduct of two or more *tortfeasers* harm a plaintiff, caselaw such as *Cook* v. *Lewis* and *Abbott* v. *Sindell Laboratories* will assist the plaintiff in recovering for proven injury. But when one of two alternative causes does not involve unacceptable conduct (i.e., where the cause may be natural), the court has usually insisted that the plaintiff prove causation on a balance of probabilities.[xxxiv]

However, two cases lend support to modifying this traditional approach in appropriate circumstances. In *Cook* v. *Lewis*, the Supreme Court of Canada declared that a defendant who negligently "intermixes" human causation *with natural causation* so as to destroy the plaintiff's "power of proof" would bear the burden of disproof on the issue of causation.[xxxv] This statement was *obiter dicta,* however, as it was established that the injury was caused by one of two negligent human agents.

In *McGhee* v. *National Coal Board,*[xxxvi] the plaintiff contracted dermatitis while working in the defendant's brick works. The defendant conceded that it had breached its duty of care to the plaintiff by not installing showers. However, the plaintiff had not proven that failure to provide the showers had caused the harm. The medical evidence established that other factors may have caused the dermatitis.

The majority of the court acknowledged that the medical evidence made it impossible for the plaintiff to prove that the defendant's negligence had caused his harm. Nevertheless, the court held the defendant liable. Lord Wilberforce stated:[xxxvii]

> *[T]he question remains whether a pursuer must necessarily fail if, after he has shown a breach of duty, involving an increase of risk of disease, he cannot positively prove that this increase of risk caused or materially contributed to the disease while his employers cannot positively prove the contrary. [. . .] In my opinion, there are further considerations of importance. First, it is a sound principle that where a person has, by breach of duty of care, created a risk, and injury occurs within the area of that risk, the loss should be borne by him unless he shows that it had some other cause. Secondly, [. . .] why should a man who is able to show that his employer should have taken certain precautions, because without them there is a risk, or an added risk, of injury or disease, and who in fact sustains exactly that injury or disease, have to assume the burden of proving more: namely, that it was the addition to the risk, caused by the breach of duty, which caused or materially contributed to the injury? In many cases of which the present is typical, this is impossible to prove, just because honest medical opinion cannot segregate the causes of an illness between compound causes. And if one asks which of the parties, the workman or the employers, should suffer from this inherent evidential difficulty, the answer as a matter of policy or justice should be that it is the creator of the risk who, ex hypothesi, must be taken to have foreseen the possibility of damage, who should bear its consequences*.

ii) Toxicological Studies

As outlined above, epidemiological studies have a number of inherent limitations. In addition, the studies are most effective *after* a population has been exposed to a suspect chemical. Therefore, scientists have begun to rely on animal studies as an aid to predicting the potential of a chemical to cause unacceptable harm, prior to exposure.[xxxviii]

The effects of a particular chemical on an individual are dependent upon a number of complex, interrelated factors. The single most important factor that determines the effect of a chemical is the amount taken up by a living system (the dose). It is also important to consider how often the exposure occurs (the frequency). Other factors that influence the toxic effect of a chemical on humans include sex, age, state of health, nutrition, individual sensitivity and the presence of other chemicals. Different species of animals may respond to exposure to a chemical differently. Toxicity is also dependent upon the route of exposure. The three principle routes are the gastrointestinal track (oral), the skin (dermal) and the lungs (inhalation). The effect of a chemical itself may be immediate or delayed, and may occur in the exposed individual or in subsequent offspring.

Three types of tests are used to evaluate the toxicity of a particular chemical.[xxxix] Acute toxicity tests are designed to determine the amount of chemical that will cause death in 50% of a given number of test animals (the lethal dose - commonly written "LD_{50}").

Sub-chronic tests involve repeated administration of the chemical for up to 90 days. Two species of animals are used (usually rats and dogs) with three dose levels given to each of the two species. At the end of the test, all animals are subjected to complete postmortems, including microscopic examinations of all organ systems.

Chronic tests are those in which a low dose of the chemical is administered for a substantial portion of the lifetime of the test animal. Several species of animals could be used but mice, rats and hamsters are preferred. Their natural life-spans are short, they are easy to breed and handle in large numbers, they are relatively inexpensive and easy to care for. As well, scientists have a relatively good understanding of the biological mechanisms of these animals. This allows for a good assessment of background disease rates, susceptibility to specific diseases at specific organ sites, longevity, and response to husbandry systems. Established scientific protocol recommends that thorough chronic testing of the disease potential of a chemical must include two species of rodents, both sexes of each, adequate controls, a sufficient number of animals to allow for the detection of a potential effect, three dose ranges (including one level likely to yield maximum expression of disease potential), detailed pathological examination throughout the duration of the studies, observation extending to most of the lifetime of the animals, and statistical evaluation of the results.[xl]

In addition to acute, sub-chronic and chronic tests, special tests aimed at analyzing effects on reproduction, the immune system, skin, eyes and behaviour may be required.

Despite the extremely useful information that toxicology can generate to assist in estimating potential health risks, there are serious problems in using animal studies to demonstrate a causal link between harm and exposure to a particular substance.[xli]

The complexity and inexactitude of the experimental procedures listed above, including the amount and form of the test chemical, the routes of exposure, the selection of the test animals, the difficulty in assessing the results of postmortem examinations, the establishment of the protocols for the tests and the expense involved, all mitigate against the use of the final results to prove causal relationships. The predictive value to humans of adverse health responses in animal tests (the mouse-to-man extrapolation) is not universally accepted. In addition, the need to feed test animals much larger doses of a substance than humans could possibly consume has often been criticized.[xlii] Controversy also surrounds the selection of the mathematical models used to extrapolate the results from high dose to low dose levels in animals. Further extrapolation from animals to human beings compounds the uncertainty.[xliii] For example, the American National Academy of Sciences estimated that the expected number of bladder cancers resulting from the consumption of saccharin over a lifetime of exposure ranged between less than one to 1,144,000 cases, depending upon the particular extrapolation assumptions that were made.[xliv]

(c) Summary

The disciplines of toxicology and epidemiology are characterized by substantial uncertainties due to limitations in scientific understanding, data, models, and methodologies.[xlv] Contributing to the uncertainty are major difficulties in understanding the etiology of many human diseases, in determining expected levels of human exposure to specific chemicals, in estimating synergistic and antagonistic effects (interactions between two or more toxic chemicals), in estimating the length and effect of latency periods, and in understanding effects on sensitive populations such as children, pregnant women, the elderly and people with pre-existing sensitivities to diseases. The results of most tests are reduced to statistical, probabilistic statements, often with wide margins of error.

Given these limitations, litigants, through no fault of their own, encounter an insurmountable hurdle when forced to prove causation in fact using toxicological or epidemiological test results. As outlined in the next section, these scientific limitations also restrict a plaintiff's ability to establish injury in fact in certain situations.

2) Indeterminate Plaintiffs

In *Reserve Mining Co.* v. *EPA,*[xlvi] the plaintiff claimed that the continued dumping of mine tailings into the atmosphere and into Lake Superior, the source of the drinking water for the city of Duluth, would increase the incidence of various forms of cancer among the exposed poulation. Some of the tailings contained amosite asbestos fibers. The EPA brought an action to stop the discharge under the injunction provisions of the *Federal Water Pollution Control Act*. This Act requires that a pollution source be "presenting an imminent and substantial endangerment to the health of persons."[xlvii] The plaintiff argued as follows:[xlviii]

(a) Animal studies had demonstrated that asbestos fibers were carcinogenic;

(b) Human exposure to asbestos fibers at occupational levels has been scientifically established to increase the incidence of cancer;

(c) The tailings contained asbestos fibers, and taconite fibers that are substantially similar to, or identical to asbestos fibers;

(d) Therefore Reserve Mining's discharges into air and water represented a potential human health hazard; and

(e) Therefore the plaintiff was injured because he was exposed to a higher risk, beyond normal background levels, of developing cancer.

In *Palmer* v. *Stora Kopparbergs* the plaintiffs presented the following argument in an attempt to obtain a permanent injunction to prevent the future spraying of a dioxin-contaminated herbicide near their homes:[xlix]

(a) Dioxin has been demonstrated to produce cancer, mutation and birth defects in laboratory animals (toxicology tests);

(b) Epidemiology studies indicate that humans exposed to dioxin have a statistically significant increased incidence of cancer and birth defects;

(c) The herbicide to be applied by the defendants would drift onto the person and property of plaintiffs, and would contaminate water systems used by plaintiffs;

(d) Therefore, Stora Kopperberg's spraying activities represented a potential health hazard to plaintiffs;

(e) Therefore, the plaintiffs would be injured if spraying was allowed because they would be exposed to a higher risk of injury.

In essence, the position of the plaintiffs in *Reserve Mining* and *Palmer* was identical. In each case, the plaintiff could establish that the conduct of a particular defendant subjected the plaintiff to an uncertain risk of future injury. But they could not establish on a balance of probabilities that they would in fact develop the disease(s) of concern. Nevertheless, the decision of each court was radically different.

In *Reserve Mining,* the Court made the following determination:

> *[A]lthough Reserve's discharges represent a possible medical danger, they have not in this case been proven to amount to a health hazard. The discharges may or may not result in detrimental health effects, but, for the present, that is simply unknown. [. . .] The medical and scientific conclusions here in dispute clearly lie on the frontiers of scientific knowledge. [. . .] In assessing probabilities in this case, it cannot be forecast that the rates of cancer will increase from drinking Lake Superior water or breathing Silver Bay air. The best that can be said is that the existence of this asbestos contaminate in air and water gives rise to a reasonable medical concern for the public health. The public's exposure to asbestos fibers in air and water creates some health risk. [. . .] The existence of this risk to the public justifies an injunction decree requiring abatement of the health hazard on reasonable terms as a precautionary and preventative measure to protect the public health*.[l]

The Court then concluded:[li]

> *In fashioning relief in a case such as this involving a possibility of future harm, a court should strike a proper balance between the benefits conferred and the hazards created by Reserve's facility. [. . .] A court is not powerless to act in these circumstances. But an immediate injunction cannot be justified in striking a balance between unpredictable health effects and the clearly predictable social and economic consequences that would follow the plant closing. [. . .] The [lower] court abused its discretion by immediately closing this major industrial plant. In this case, the risk of harm to the public is potential, not imminent or certain, and Reserve says it earnestly seeks a practical way to abate the pollution. A remedy should be fashioned which will serve the ultimate public weal by insuring clean air, clean water, and continued jobs in an industry vital to the nation's welfare.*

The Court granted a permanent injunction, but stayed the execution of the injunction. The Court was clearly impressed with the undertaking by Reserve to spend $243 million to abate its discharges into air and water by developing environmentally acceptable on-land disposal facilities.

In *Palmer* the Court made the following observations:[lii]

> *The complete burden of proof, of course, rests upon the plaintiffs throughout for all issues asserted by them. If the spraying had actually occurred, they would have to prove by a preponderance of probabilities the essential elements of either or all of the alleged causes of action as I have set them out. However, the spraying has not occurred and this application is for a quia timet injunction. [. . .] That remedy also, however, is not without its limitations. [. . .] The plaintiffs must [. . .] prove the essential elements of a regular injunction, namely irreparable harm and that damages are not an adequate remedy. [. . .*]

Justice Nunn then questioned whether the plaintiffs have offered sufficient proof that there is a serious risk of harm to the health of the plaintiffs.[liii] He then continued:

> *A great deal of the evidence submitted related to animal studies where TCDD was reported to have caused various effects indicating it to be, among other things, fetotoxic, teratogenic, carcinogenic and to cause immunological deficiencies, enzymatic changes, liver problems and the like. Also it is alleged to bioaccumulate and be persistent both in soil and in tissue. [. . .] That TCDD has had all of these effects is undoubtedly true in the experiments described, but, in every case, the effect must be related to dose. In the animal studies the doses are extremely high and, in all cases, many, many thousands of times greater than any dose which could be received in Nova Scotia. [. . .] The human [epidemiological] information comes from a number of studies made in various countries of the world. [. . .] I am satisfied that in all these cases the exposure was massive, either through accident or industrial exposure or the Vietnam War. [. . .] I am satisfied that, on the whole of the evidence, where risk to health is claimed in any study, the circumstance has been one of massive exposure and such are not of significant probative value in light of the actual low possible exposure here*.[liv]

In the end, the Court refused to issue the injunction, holding that the Cape Breton landowners had not proven their case in accordance with long-standing legal principles.[lv]

D. Summary of Section II

Potential litigants contemplating common law action involving an environmental risk activity may be assisted by the courts when they cannot establish which of a number of wrongdoers caused a visible injury. However, where the malfeasance of the defendant may or may not have caused an obvious injury, or where the plaintiff cannot establish an injury or the potential for injury is on a balance of probabilities, the plaintiff will have a very difficult time convincing the court of the merits of the case. Section III of this paper will determine whether this state of affairs accords with the goals of tort law.

II. THE GOALS OF TORT LAW

In Part II above, it was argued that procedural rules placing the onus and burden of proof upon the party alleging injury were developed to help promote the tort goals of compensation, deterrence, economic efficiency and fairness. As stated by Lord Reid in *McGhee* v. *National Coal Board*:

> *the legal concept of causation is not based on logic or philosophy. It is based on the practical way in which the ordinary man's mind works in the every-day affairs of life.*[lvi]

A number of tort scholars have argued convincingly that causation is not simply a blind mechanical formula to be insensitively applied by the courts.[lvii] Indeed, these writers state that courts will modify causation rules when equity or public policy considerations warrant such relaxation. *Cook* v. *Lewis, McGhee* v. *National Coal Board, Reserve Mining* v. *EPA* and *Sindell* v. *Abbott Laboratories* are all expressions of the court's insistence on an overiding policy of social justice. Ernest Weinrib, in a fascinating article entitled "A Step Forward in Factual Causation" makes the following observation in analyzing the *Cook* and *McGhee* cases:

> *The allocation of the burden of proof is not always to be the plaintiff's, but it must be subservient to compelling requirements of justice. And the primary evidential nature of cause in fact should not render it impervious to the considerations of policy, purpose and value*.[lviii]

Weinrib continues:[lix]

> *the very existence of the cause in fact inquiry is the expression of certain more abstract considerations of fairness. At stake is a balance between the innocent victim's claim to be compensated and the freedom of the defendant to be as wicked as he likes as long as no injurious consequences flow from that wickedness. The weighing of these competing interests is [. . .] not a value-free process [. . .] we must always be prepared to test the cause in fact process against the underlying policies and purposes that it embodies, and to adjust the ordinary method of dealing with cause in fact if it fails adequately to reflect our more basic notions of fairness.*

Does the status quo, which denies a remedy to a toxic tort litigant, then promote the principles of tort law? The answer is "no". As outlined above, a primary function of tort law is to compensate victims. If each of the 15 workers who had contracted liver cancer were permitted to recover fully for their damages, 10 would receive a windfall because it was more likely than not that the defendant did *not* injure them. However, denying recovery to all of the 15 individuals denies compensation to the five plaintiffs who were in fact injured by the defendant.[lx]

A second goal of tort law is to deter socially unacceptable conduct. Obviously, increasing the incidence of cancer from 10 to 15 cases in a group of 100 workers is socially unacceptable.

Granting a remedy to an individual worker in those circumstances would force the defendant to conduct his business in a more benign fashion.

Perhaps the most important reason for re-evaluating tort law principles involves the notions of fairness and justice. The rules governing onus and burden of proof were developed to protect individual rights and liberties. Therefore, traditional dispute resolution mechanisms presume that lack of proof of guilt (harm) means probable proof of innocence (safety).[lxi] Society accepts the potential for error when resolving traditional disputes (better that 100 guilty be freed than one innocent be convicted) because high priority is placed upon individual liberty. But when managing toxic chemicals, the primary concern for society must be with *public* health, not individual liberty or corporate growth.[lxii] When assessing environmental risk problems, it would be absurd to adopt the proposition that it is better that 100 harmful chemicals be declared safe than one safe chemical be declared harmful.[lxiii] When dealing with environmental risks, the inability to prove injury must *not* be viewed as presumptive proof of safety. The evidence that is equivocal or unavailable today may prove at some future date to have caused harm. And, in accordance with the characteristics of an environmental risk activity, the harm, if it does materialize, will be serious and irreparable. Thus, policy considerations dictate that, in appropriate circumstances, a toxic tort litigant should not be precluded from a remedy because, through no fault of his or her own, he or she cannot meet standards which were established to foster social policy goals different from the goals that are sought to be preserved in toxic tort litigation. Section IV of this paper will examine some mechanisms to affect appropriate changes to common law doctrine to allow recovery in toxic tort litigation.

III. SUGGESTED MECHANISMS FOR REFORM

A number of writers have suggested various approaches to overcome some of the inequities resulting from the insistence on using traditional burden and onus rules in toxic tort litigation. These remedies include, but are not restricted to, the establishment of no-fault insurance schemes,[lxiv] legislating a statutory cause of action for persons injured by toxic chemical exposure,[lxv] establishing governmental compensation boards,[lxvi] and adopting risk/benefit analysis to regulate specific chemicals.[lxvii] Many of these approaches are innovative and must be carefully examined to assess their merits, including the cost-effectiveness of implementation. No doubt the ultimate solution will incorporate a number of different approaches. Other papers in these proceedings deal primarily with the role of civil courts in adapting traditional common law approaches to standing, class action, damages and costs in toxic tort litigation. Therefore, this section will highlight some of the approaches that may be used to modify existing causation rules to assist deserving litigants. The ability to utilize any of these approaches will depend to a significant extent upon the particular facts of the case in front of the court.

A. Lower Plaintiff's Burden

Some writers have suggested that the courts could lower the burden of proof to allow recovery where the plaintiff establishes that causation is "possible", or "conceivable", rather than "probable".[lxviii] An interesting expression of this approach involves the replacement of the preponderance of evidence rule with proportional liability.

1. Proportional Liability

Proportional liability overcomes the difficulties associated with causal indeterminacy (plaintiff cannot establish on a balance of probabilities injury in fact, or injury caused by wrongdoing of a particular defendant) by holding the defendant liable in proportion to the probability that the defendant caused the injury.[lxix] In the liver cancer example, if 15 workers develop the disease and epidemiological studies causally link the defendant to 5 of those cancers, the principle of proportional liability would allow the court to conclude that there was a 33% chance (5/15) that the injury of each of the 15 victims was caused by the defendant. Proportional compensation would therefore allow each of the 15 plaintiffs to recover 33% of their damage claim. Proportional liability clearly undercompensates the five victims whose injuries were caused by the defendant, and provides a windfall for the 10 workers whose cancer was not caused by the defendant.[lxx] Nevertheless, to paraphrase Fleming, the law should prefer a 33% chance of doing justice, to 100% certainty of doing injustice.[lxxi] The tort goal of deterring socially unacceptable conduct is also addressed by the concept of proportional liability.[lxxii] The defendant could no longer expect to misuse toxic substances and remain exempt from tort liability. The remedy claimed by the 15 workers, each recovering 33% of their damages would, in the aggregate, equal the actual damage inflicted by the defendant. The threat of being made responsible for actual losses should deter the defendant from activities which give rise to liability.

2. Risk/Benefit Analysis

In *Reserve Mining* the Court modified the traditional standards for obtaining an injunction where it was clear that the citizens of Duluth were subjected to an uncertain, but serious and irreparable risk of harm to their health. Using a risk/benefit analysis, the Court made a number of threshold findings to the effect that the scientific information needed to quantify the potential harm was unavailable through no fault of the plaintiff.[lxxiii] The Court then balanced the benefits to the community of the defendant's mine, against the seriousness of potential injury. The remedy, an injunction with a stay of execution pending defendant undertakings to eliminate the discharges, clearly accommodated the tort goals of fairness and deterrence.

B. Shift Burden of Proof onto Defendant

Several writers recommend that the burden of disproving causation be placed upon the defendant.[lxxiv] Once a plaintiff had presented a threshold case, showing, for example, that the injury or the cause of the injury is indeterminate, that epidemiological studies can permit reasonable probabilistic calculations of the increased incidence of disease, or disease potential, and that the plaintiff is within the risk zone (that is, the plaintiff was significantly exposed to the activity in question) the burden would shift to the defendant to disprove causation as to individual victims.

While the arguments supporting a shift in the burden of proof seem compelling at first glance, there are two reasons why extreme caution should be exercised before a Court adopts this approach. First, the limitations inherent in epidemiological studies which, at best, produce probabilistic estimates of causal relationships can often work to the advantage of a defendant.[lxxv] A worker may be able to demonstrate an increase over background levels, of five cancers, resulting in a 33% chance that his liver cancer was caused by the defendant. However, shifting the burden would allow the defendant to establish a 67% chance that he did not cause the plaintiff's injury!

Secondly, independent of probabilistic evidence (epidemiological and toxicology studies) a defendant might well be able to establish in certain cases that it is more likely than not that its activities are reasonable. For example, in the Nova Scotia Herbicide Trial, the Court made the following observations:[lxxvi]

> most countries, including Canada, have regulatory agencies, whose function it is to regulate and control the use of new chemical compounds before they are exposed to the environment. [. .]. In [Canada] reviews are made periodically after reviews of the literature and independent study by highly trained and competent scientists. In [Canada] registration for use is still in effect. [. . .] The provincial Department of Environment has not restricted the use of these herbicides. [. .]. To some extent this case takes on the nature of an appeal from the decision of the regulatory agency and any such approach through the Courts ought to be discouraged in its infancy. Opponents to a particular chemical ought to direct their activities towards the regulatory agencies, or, indeed, to government itself where broad areas of social policy are involved. It is not for the Courts to become a regulatory agency of this type. It has neither the training nor the staff to perform this function.

Thus, in circumstances where the concern for safety amounts to an uncertain risk of future harm, it may well be easy for a defendant to dispel a shifted burden, simply by showing that governmental regulatory agencies have approved the activity in question.

C. Science Courts

It has been suggested that a specialized court be created to minimize the difficulties associated with the law-science interface.[lxxvii] One proposal would allow scientific advocates on opposing sides of a technical issue to present evidence and argument to a panel of distinguished scientists. The panel would resolve the scientific issues. The Court would then utilize these factual conclusions, supply the relevant legal or policy judgments, and make a final decision. Another variation would allow litigants to argue their cases before a panel of judges with recognized scientific credentials.

The concept of the science court has generally fallen into disfavour.[lxxviii] Judges have historically shown great adeptness at understanding the essentials of complex litigations. In this regard, toxic tort litigation is not unique. In any event, the problem faced by litigants is not that the science of environmental risk is complex but that the data needed to establish the case is equivocal or unavailable. Thus, the science court would be faced with exactly the same difficulty in determining the cause-effect nexus faced by traditional law courts.

D. Summary

Social policy considerations dictate that, in appropriate circumstances, courts may wish to modify traditional burden and onus rules to accommodate deserving toxic tort litigants. To be effective, variations from the norm and the justification for such variations, must be clearly articulated. Part V concludes with some suggestions that may assist lawyers and judges in accommodating the broader social goals reflected in toxic tort litigation.

CONCLUSION

As separate disciplines, law and science each exert tremendous influence over the world as we understand it today.[lxxix] But the synergistic benefits of law and science acting in concert are much greater than the impact of each discipline acting independent of the other, or even worse, antagonistic to the other. If a goal of science is to seek physical truth, the goal of law is to seek justice - that is, truth tempered by mercy. In a very practical sense, when one discipline acts independent of the other, each is incomplete.

To a significant extent, the conflict between law and science is the result of misunderstanding and ignorance of one discipline by the practitioners of the other.[lxxx] Lawyers' lack of understanding of science, its role in society and methodologies is pervasive, and unacceptable. Scientists' ignorance of law, its role in society and its methodologies is equally pervasive and is also unacceptable. A pivotal first step in coming to terms with the conflict must be for each profession to begin to understand the strengths and limitations of the other. Professional associations must sponsor conferences with workshops and plenary sessions which specifically address the law/science interface. University curicula must encourage cross-disciplined education, at both the undergraduate and professional levels. The editorial boards of professional journals must solicit quality articles from various professional disciplines. Perhaps most importantly, lawyers must appreciate that they have a clear obligation to carefully assess the potential for winning toxic tort litigation, but only *after* they fully comprehend the implications of the science issues involved. In appropriate circumstances, lawyers *must* look at alternatives to litigation, including negotiation and mediation processes to resolve environmental disputes.[lxxxi] It is hoped that if some of these steps are taken, lawyers and judges will develop a better appreciation of the parameters of science. Armed with these new insights, they will undoubtedly be better able to accommodate the social goals which are at the very heart of tort law.

POSTCRIPT

The reader is cautioned that both caselaw and academic writing on this topic have evolved since these articles were written by Professor John G. Fleming: "Probabilistic Causation in Tort Law" (1989) 68 Can. Bar Rev. 661; and "Probabilistic Causation in Tort Law: A Postcript" (1991) 70 Can. Bar Rev. 136.

ENDNOTES

i. *Palmer* v. *Stora Kopparbergs Bergslags Aktiebolag* (1983), 12 C.E.L.R. 157 (N.S.S.C., T.D.) [hereinafter *Palmer* v. *Stora Kopparbergs* or *Palmer*].

ii. *Ibid.* at 230.

iii. See generally H. Hart & A. Honoré *Causation in the Law* (Clarendon Press, 1959); J. G. Fleming, *The Law of Torts*, 5th ed., (Sydney: The Law Book Company, 1977); E. J. Weinrib, "A Step Forward in Factual Causation" (1975) 38 Modern Law Rev. 518; J. Krier, "Environmental Litigation and The Burden of Proof" in Baldwin & Page (eds.,) *Law and the Environment* (Walker, 1970) at 105; G. Calabresi, "Concerning Cause and the Law of Torts: An Essay for H. Kalven, Jr." (1975), 43 U. Chicago L. Rev. 69; T. Page, "A Generic View of Toxic Chemicals and Similar Risks" (1978-79), 7 Ecology Law Quarterly 207; W.H. Pedrick, "Causation, The "Who Done It" Issue, and A. Becht" (1978), Washington U. L. Q. 645.

iv. Page, *supra* note 3, esp. at 233-234.

v. See generally sources cited in note 3, *supra.*

vi. See generally sources cited in note 3, *supra* esp. J. Fleming at 7-12.

vii. *Ibid.* See also R. Solomon, B. Feldthusen & S. Mills, *Cases and Materials in the Law of Torts* (Carswell, 1982) at 14-21.

viii. G. Robinson, "Multiple Causation in Tort Law: Reflections on the DES Cases", 68 Virginia L. Rev. 713 (1982).

ix. Thode, "Tort Analysis: Duty-Risk v. Proximate Cause and the Rational Allocation of Functions Between Judge and Jury" [1977] Utah L. Rev. 1, 2 quoted in R. Delgado, "*Beyond Sindell*: Relaxation of Cause-In-Fact Rules for Indeterminate Plaintiffs" (1982), 70 California L. Rev. 881, at 883 note 12.

x. See, for example, D. Stuart, *Canadian Criminal Law* (Carswell, 1982) at 32-41.

xi. Krier, *supra* note 3 at 107-111; J. Sax, *Defending the Environment* (A. Knopf, 1971) at 137-157. See also an excellent book by J. Dales, *Pollution Property and Prices* (U. of T. Press, 1968), esp. at 58-76.

xii. Krier, *supra* note 3 at 107-108.

xiii. See, for example, Note, "Tort Actions for Cancer: Deterrence, Compensation, and Environmental Carcinogenesis" (1981), 90 Yale L. J. 840; D. Large & P. Michie, "Proving that the Strength of the British Navy Depends on the Number of Old Maids in England: A Comparison of Scientific Proof with Legal Proof" (1981), 11 Environmental L. 555; R. Rabin, "Environmental Liability and the Tort System (1987), 24 Houston L. Rev. 27.

xiv. R. Carson, *Silent Spring* (Houghton Mifflin, 1962); B. Commoner, *The Closing Circle* (A. Knopf, 1971); B. Ward & R. Dubos, *Only One Earth* (W. Norton, 1972).

xv. W. Ruckelshaus, "Risk Science and Democracy" (Nov. 87) Chemtech; "Risk Assessment and Management: Framework for Decision Making," (Dec. 1984) U.S.E.P.A..

xvi. See Page, *supra* note 3 at 207-223; M. Gelpe & A. Tarlock, "The Uses of Scientific Information in Environmental Decision Making" (1974) 48 Southern California L. Rev. 371;

L. Silver, "The Common Law of Environmental Risk and Some Recent Applications" (1986), 10 Harvard Environmental L. Rev. 61.

xvii. A number of articles are extremely helpful in understanding the environmental science/law interface. They include:
Page, *supra* note 3; Delgado, *supra* note 9; Large & Michie, *supra* note 13; Council on Environmental Quality (Washington, D.C.: U.S. Government Printing Office), 6th Annual Report (Dec., 1975); K. Feinberg, "The Toxic Tort Litigation Crisis: Conceptual Problems and Proposed Solutions (1987), 24 Houston L. Revi. 155; J. Forstrom, "Victim Without a Cause: The Missing Link Between Compensation and Deterrence in Toxic Tort Litigation" (1987) 18 Environmental L. 151; Gelpe & Tarlock, *supra* note 16; Interagency Regulatory Liaison Group - Work Group on Risk Assessment, "Scientific Bases for Identification of Potential Carcinogens and Estimation of Risks" (July, 1979) 63 Journal of the National Cancer Institute 241; H. Latin, "The Significance of Toxic Health Risks: An Essay on Legal Decisionmaking Under Uncertainty" (1982) 10 Ecology L. Q. 339; J. Leape, "Quantitative Risk Assessment in Regulation of Environmental Carcinogens" (1980) 4 Harvard Environmental L. Rev. 86; J. McElveen & P. Eddy, "Cancer and Toxic Substances: The Problem of Causation and the Use of Epidemiology" (1984-85) 33 Cleveland State L. Rev. 29; "Rethinking Tort and Environmental Liability Laws: Needs and Objectives of the Late 20th Century and Beyond" (1987) 24 Houston L. Rev. 1; "Tort Actions for Cancer: Deterrence, Compensation, and Environmental Carcinogenesis" (1981) 90 Yale L. J. 840; H. Versteeg, "Environmental Risk Assessment: A Rational Approach to the Management of New Brunswick's Spruce Budworm Enigma" (1982) 11 Canadian Environmental L. Rep. 109; H. Versteeg, "The Spruce Budworm Spray Programme and the Perception of Risk in New Brunswick" (May 1984) Friends of the Earth; D. Harvey, "Epidemiologic Proof of Probability: Implementing the Proportional Recovery Approach in Toxic Exposure Torts" (1984) 89 Dick. L. Rev. 233; R. Rabin, "Environmental Liability and the Tort System" (1987) 24 Houston L. Rev. 27.

xviii. *Cook* v. *Lewis* [1952] 1 D.L.R. 1 (S.C.C.).

xix. Fleming, *supra* note 3 at 301. See also Weinrib, *supra* note 3.

xx. *Sindell* v. *Abbott Laboratories*, 26 Cal. 3d 588; 607 P. 2d 924.

xxi. The California Court extended principles developed in *Summers* v. *Tice*, 33 Cal. 2d 80 (1948). The facts in the *Tice* case were virtually identical to those in *Cook* v. *Lewis*. See Delgado, *supra* note 9 at 881-883.

xxii. *Supra* note 20. at 936.

xxiii. Delgado, *supra* note 9; Weinrib, *supra* note 3.

xxiv. See generally Council on Environmental Quality (CEQ) *Annual Reports supra* note 17; Versteeg, *supra* note 17 (Perception of Risk, esp. ch. 4 and 5). Note: This paper will focus on the potential adverse human health impact of toxic substances. However, it must be clearly understood that the potential impact on the natural environment is also profound. See, for example, Versteeg, *supra* note 17 (Perception of Risk, ch. 6) and CEQ *Annual*

Reports.

xxv. See CEQ *Annual Reports,* esp. the 6th Annual Report, ch. 2. See also S. Epstein, *The Politics of Cancer* (Garden City: Doubleday, 1979) esp. Part I.

xxvi. *Ibid.* See also Harvey, *supra* note 17 at 234-235; Leape, *supra* note 17; Interagency Regulatory Liaison Group, *supra* note 17.

xxvii. See, for example, Interagency Regulatory Liaison Group, *supra* note 17; Epstein, *supra* note 25.

xxviii. See, for example, A. Feinstein & R. Horwitz, "Double Standards, Scientific Methods, and Epidemiological Research" (Dec. 1982) 307, The New England J. of Medicine 1611; G. Hutchison, "The Epidemiological Method" in Schottenfeld & Fraumeni, *Cancer Epidemiology and Prevention* (1982); McElveen & Eddy, *supra* note 17, esp. at 37-47; Harvey, *supra* note 17, esp. at 236-240.

xxix. *Ibid.* But see esp. Harvey, *supra* note 17 at 241-242; McElveen & Eddy, *supra* note 17 at 47-56; Large & Michie, *supra* note 17; Feinberg *supra* note 17 at 159-164; C. Cranor, "Epidemiological and Procedural Protections for Workplace Health in the Aftermath of the *Benzene* Case" 5 Industrial Relations L. J. 372 at 379-393.

xxx. *Ibid.* esp. Cranor at 385-394.

xxxi. McElveen & Eddy, *supra* note 17 at 46-47.

xxxii. This example, somewhat modified, is taken from Harvey, *supra* note 17 at 237-240. See also Feinstein & Horwitz, *supra* note 28 at 1612.

xxxiii. See, for example, Fleming, *supra* note 3 at 179-191; Large and Michie, *supra* note 13 at 594-614.

xxxiv. *Ibid.* See also Delgado, *supra* note 9 at 887, esp. authorities cited in note 30.

xxxv. *Supra* note 18.

xxxvi. [1972] 3 All E.R. 1008 (H.L.).

xxxvii. *Ibid.* at 1012.

xxxviii. See generally authorities cited in note 17, *supra*. See also H. Schiefer, D. Irvine & S. Buzik, *You and Toxicology* (Saskatoon, 1986).

xxxix. Schiefer, *supra* note 35 esp. at 4-11 gives an excellent overview of the various tests used in toxicological studies. See also Interagency Regulatory Liaison Group, *supra* note 17 at 248-256.

xl. Interagency Regulatory Liaison Group, *supra* note 17 at 248.

xli. See generally authorities cited in note 17, *supra*.

xlii. The rationale for high dosage feeding has been gravely misunderstood by many lay people. There is little evidence that any chemical fed to animals in large quantities will cause cancer. However, there is clear scientific evidence that the higher the exposure of a subject to a chemical, the greater the likelihood the disease of concern will be contracted. Detecting an incidence of cancer as low as .01% in experimental animals (1 case in 10,000 animals - which is totally unacceptable in human populations!) would require hundreds of thousands of animals (1 case in 10,000; 10 cases in 100,000; 100 cases in 1,000,000). Since such experiments would be prohibitively expensive and impossible to manage, researchers *reduce*

the number of test animals but *increase* the dosage in order to increase the probability of inducing the disease in question in a particular animal (*or* to establish evidence of no observable effect). An illustration is helpful. Suppose that feeding the equivalent of 1,000 cans of saccharin-containing pop produces one significant cancer in 50 rats. The toxicologist may then assume, all other things being equal, that 100 cans will produce 1 tumor in 500 rats, 10 cans will produce 1 tumor in 5,000 rats, and 1 can will produce 1 tumor in 50,000 rats. Note that Justice Nunn in the Nova Scotia Herbicide Trial expressed grave concerns about the high dosage levels fed to animals in the dioxin studies - see text accompanying note 50, *infra*. See generally, Cranor, *supra* note 29 at 381; Epstein, *supra* note 25 at 61-64; CEQ, *6th Annual Report, supra* note 17.

xliii. Cranor, *supra* note 29 at 381.

xliv. *Ibid.* See also V. Covello, "Informing People About Risks From Chemicals, Radiation, and Other Toxic Substances", unpublished manuscript, submitted for publication. Available, subject to permission from author, from H. Versteeg.

xlv. *Ibid.*

xlvi. *Reserve Mining Co.* v. *EPA,* 514 F. 2d 492 (8th Cir. 1975) [hereinafter *Reserve Mining*].

xlvii. *Federal Water Pollution Control Act* 504(a), 33 U.S.C. 1364(a) (1982), quoted in Silver, *supra* note 16 at 560-561 88-89.

xlviii. See Large & Michie, *supra* note 13 at 560-561.

xlix. See J. F. Castrilli, "Problems of Proof and Credibility Issues in Relation to Expert Evidenc8e in Toxic Tort Litigation" (1984-85) 10 Queen's L. J. 71 at 73-74.

l. *Supra* note 46 at 506 *et seq.*

li. *Ibid.* at 535-537.

lii. *Supra* note 1 at 229-230.

liii. See text accompanying note 2.

liv. *Supra* note 1 at 234-235.

lv. *Ibid.* at 237. See also Castrilli, *supra* note 45; B. Wildsmith, "Of Herbicides and Humankind: Palmer's Common Law Lessons" (1986) 24 Osgoode Hall L. J. 161.

lvi. *McGhee*, *supra* note 36 at 1011.

lvii. See authorities cited in Delgado, *supra* note 9 at 891.

lviii. Weinrib, *supra* note 3 at 533.

lix. *Ibid.* at 530.

lx. See Harvey, *supra* note 17 at 245; D. Rosenberg, "Toxic Tort Litigation: Crisis or Chrysalis?" (1987), 24 Houston L. Rev. 183 at 190-193; Delgado, *supra* note 9 at 891-895.

lxi. See text accompanying note 4, *supra*.

lxii. See authorities cited in note 11, *supra*.

lxiii. Page, *supra* note 3 at 230-236; Versteeg, *supra* note 17 (Perception of Risk) at 86-91.

lxiv. Feinberg, *supra* note 17 at 167-174; Rabin, *supra* note 17.

lxv. Forstrom, *supra* note 17 at 163-168.

lxvi. Harvey, *supra* note 17 at 248; Forstrom, *supra* note 17 at 171-176.

lxvii. Note, "Toxic Substance Contamination: The Risk-Benefit Approach to Causation Analysis"

14 U. of Mich. J.L. Ref 53; Harvey, *supra* note 17 at 244.

lxviii. Harvey, *supra* note 17 at 244-246; Delgado, *supra* note 9 at 897; Gelpe & Tarlock, *supra* note 17.

lxix. See Delgado, *supra* note 9 at 899-908; Forstrom, *supra* note 17 at 168-169; Harvey, *supra* note 17 at 245-248; Rosenberg, *supra* note 56 at 190-194.

lxx. Rosenberg, *supra* note 56 at 192-193; Harvey, *supra* note 17 at 245.

lxxi. See text accompanying note 19, *supra*.

lxxii. Delgado, *supra* note 9 at 903; Harvey, *supra* note 17 at 258; Rosenberg, *supra* note 56 at 192.

lxxiii. See generally: Note, "Toxic Tort Contamination: The Risk-Benefit Approach to Causation Analysis", *supra* note 64 esp. at 60-67; Note: "Reserve Mining - The Standard of Proof Required to Enjoin an Environmental Hazard to the Public Health" (1975) 59 Minnesota L. Rev. 893 esp. at 901-913.

lxxiv. See for example Wildsmith, *supra* note 51 at 176-179; Castrilli, *supra* note 45 at 76-80; Krier, *supra* note 3.

lxxv. See Harvey, *supra* note 17 at 243-244.

lxxvi. *Supra* note 1 at 232-233.

lxxvii. See for example, A. Kantrowitz, "The Science Court Experiment: Criticisms and Responses" (April 1977); Bull. Atomic Scientists; Martin, "The Proposed Science Court" (1977) 75 Mich. L. Rev. 1058; R. Carpenter, "Ecology in Court, and Other Disappointments of Environmental Science and Environmental Law" 15 Natural Resources Lawyer 573 at 593.

lxxviii. See Carpenter, *supra* note 74 at 593.

lxxix. H. Markey, "Science and Law: A Dialogue on Understanding" (1982) 68 American Bar Association J. 154.

lxxx. *Ibid.* See also Carpenter, *supra* note 74; D. Bazelon, "Science and Uncertainty: A Jurist's View" (1981) 5 Harvard Environmental L. Rev. 209; W. Thomas (ed.) *Scientists in the Legal System* (Ann Arbor Publishers, 1974).

lxxxi. G. Bingham & L. Haygood, "Environmental Dispute Resolution: The First Ten Years" (1986) 41 Arbitration J. 3; Canadian Environmental Mediation Newsletter, published by Conflict Management Resources of York University, Toronto, Canada.