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Articles

***213** "RISK ASSESSMENT": A METHODOLOGY FOR DECIDING CLAIMS FOR INCREASED RISK OF CANCER

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I. Introduction

A. Purpose and Goal

The purpose of this article is to suggest a methodology for deciding claims for increased risk of cancer. [FN1] These claims arise when a plaintiff is exposed to a carcinogen because of the conduct of the defendant and the exposure increases the chances that disease will occur. In most cases, there is no present physical injury. The goal is to provide victims of toxic substance exposure with a remedy without forcing a business facing such claims into bankruptcy. In such a "no-win situation," victims are not paid or are paid a fraction of the damages they have suffered. Businesses can suffer such ruinous damages that bankruptcy is the only viable solution. A well-known example of a corporation seeking protection from toxic tort claimants is the Manville Corporation, formerly known as Johns-Manville. [FN2] The Manville Corporation filed a petition in ***214** bankruptcy in 1982, when it was faced with potential liability for many asbestos-related claims. [FN3] Bankruptcy, in turn, leads to impaired viability of the subject company, lost jobs, and economic disruption. The objective of this article is to provide a procedure in awarding damages for claims of increased risk of cancer. Doing so can turn a no-win situation into a "win-win" situation, where victims are ultimately paid and businesses remain solvent.

B. Problems and Difficulties in Litigating Toxic Exposure Cases

1. The Statute of Limitations

The cancer that ultimately may develop after exposure to a carcinogen [FN4] generally does not manifest itself until long after the exposure occurred. The period of time between exposure and disease manifestation is called the "latency period." This fact leads to a number of problems for the hazardous substance exposure victim: (a) the statute of limitations may have run; (b) the responsible company may be out of business; and (c) there are inherent difficulties in proving an injury that has occurred in the past twenty or thirty years. [FN5]

There are two major approaches to the statute of limitations problem. [FN6] The "traditional rule" states that a cause of action accrues at the time of invasion of the body. [FN7] The "discovery rule" states that a claim accrues when the plaintiff knows or through due diligence should have known of the injury. [FN8]

The inequity of the traditional rule is that for injuries that do not occur until a long latency period has elapsed, the statute of limitations may have run by the time the victim even realizes that he or she has been injured. ***215**[FN9] The trend of the courts has been to follow the discovery rule, because of the inequity of the traditional rule. [FN10]

2. The Indeterminate Defendant Problem

The indeterminate defendant problem arises in a situation where any one or more defendants in combination or in totality caused the plaintiff harm, and therefore, no one defendant can be identified. This problem arises in the context of products liability where a number of defendants manufacture a product that may have caused an injury to the plaintiff and a single defendant cannot be implicated. [FN11] Compounding the problem is the fact that the harm may occur naturally, without the

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intervention of the defendant(s); therefore, the injury may have occurred independently of any conduct by the defendant(s). [FN12] For example, cancer occurs in the general population without exposure to a hazardous substance.

Also, the problem of the indeterminate defendant arises in other contexts, such as: hazardous waste litigation and water and air pollution cases. Many different defendants may have contributed to a pollution problem and any one defendant cannot be identified with certainty. [FN13] In a water pollution case, for example, there may be a number of different industrial or agricultural sites contributing to the pollution problem, and no one defendant can be identified.

3. Proving Causation [FN14]

In toxic tort cases, the plaintiffs must prove general and specific causation: that the substance is capable of causing their disease and that the substance caused this particular plaintiff's disease. [FN15] A strong and ***216** weak version of the rule exists. [FN16] The strong version requires both scientific evidence of increased chance of disease of more than fifty percent and "particularistic" proof that exposure caused the disease in this particular plaintiff. [FN17] The weak version permits recovery based solely on statistical evidence. [FN18] Epidemiological studies determine the likelihood that a given type of cancer was caused by exposure to a particular substance and some courts accept this proof alone.

A problem related to particularistic proof of causation is that of the indeterminate plaintiff. [FN19] The epidemiological proof in toxic tort cases can only show that exposure to a toxic substance has caused an increased incidence of disease in a group of individuals. [FN20] Thus, it is unknown whether a particular plaintiff's disease was caused by exposure or by some other factor such as background risk or other intervening causes. This may also be expressed as the "baseline risk paradox," [FN21] which is bane to plaintiffs' attorneys and solace to defense attorneys. [FN22]

Baseline risk is the risk of occurrence of the plaintiff's injury in the absence of any conduct by the defendant adding to the risk. [FN23] Thus, the definition of baseline risk leads to the "baseline risk paradox," that is, it is unknown whether plaintiff's cancer resulted from defendant's conduct or from some other intervening cause or was attributable to background risk.

Essentially the concept of "baseline risk paradox" incorporates the "confounding factors," [FN24] of epidemiology studies, and incorporates them into the requirement of particularized proof in the standard of causation. Thus, many claims are defeated by operation of the "baseline risk paradox." No one can prove that a particular plaintiff's case of cancer is part of the baseline risk, or is part of the added risk contributed by defendant's conduct. Thus, it is advantageous for defense counsel to maximize the contribution of baseline risk. Plaintiff's counsel, of course, has an interest in minimizing it. [FN25] Alternate ways of approaching the "baseline ***217** risk paradox" will be discussed in the section on risk assessment below. [FN26]

Breaking down the elements of the causation standard and expressing them in risk assessment terms leads to the following generalization: to prove causation, a plaintiff must prove hazard (i.e. the substance is capable of causing the cancer complained of). Further, a plaintiff must be able to prove and quantify the chemical-specific dose-response relationship (i.e. what quantity of the substance causes the particular kind of cancer). This quantification is also referred to as the toxicological assessment. Furthermore, a plaintiff must quantify the exposure level of the plaintiff or class of plaintiffs (exposure assessment). The information from the toxicological and exposure assessments is combined to define "risk." [FN27]

C. The Three Major Types of Claims Brought in Toxic Tort Actions [FN28]

Plaintiffs' attorneys have prosecuted toxic tort cases by bringing three major types of claims: claims based on the fear of developing cancer, claims for medical monitoring or surveillance, and claims for the increased risk of cancer.

1. Fear of Cancer

Fear of future cancer claims (also referred to as "cancerphobia") are based on the idea that exposure to a carcinogen is an event in and of itself that may give rise to reasonable fear and anxiety. [FN29] The magnitude of the increased risk is relevant to determine whether the fear is reasonable. [FN30] This type of claim is defined for purposes of review only and will not be discussed in detail in this article. [FN31]

2. Medical Monitoring Damages

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Medical monitoring damages are awarded to hazardous substance exposure victims to cover the costs of continued medical monitoring or surveillance. Awarding these damages allows the plaintiff to obtain ***218** timely medical intervention if the cancer complained of should develop. Thus, the probability of surviving the cancer is increased. [FN32] The reasoning supporting these damages is that they allow the plaintiff to mitigate damages. [FN33] The plaintiff must show enhanced risk, but rigorous quantification of the enhancement is generally not required. [FN34] This type of claim is defined for purposes of review only and will not be discussed in detail in this article. [FN35]

3. Claims for Increased Risk of Cancer

Increased risk of cancer claimants seek damages for exposure to a substance that resulted in an enhanced risk of a future disease. They allege defendant's conduct caused the harm. In most of these cases there is no presently manifested physical injury. [FN36] Some commentators argue that increased risk itself is an injury requiring compensation. [FN37] There is a wide range of opinion on how these claims should be handled by the courts. [FN38]

In jurisdictions recognizing the single cause-of action-rule, which is also referred to as the rule against splitting a cause of action, the increased risk of cancer claim may be the only remedy for the plaintiff. [FN39] The rule against splitting a cause of action requires that toxic tort victims bring one action to recover past, present and future damages resulting from a single wrongful act. [FN40] Failure to include all related claims against a defendant in one action precludes a second action based on un-litigated but related claims. [FN41]

In the jurisdictions recognizing this rule, the increased risk claimant must bring the claim for future damages at the same time as the claim for past and present damages. However, because the disease, if it develops at all, does so only after a long latency period, the plaintiff cannot predict whether or not he or she will get cancer. In these jurisdictions, a later ***219** cancer claim would also be barred by the statute of limitations. [FN42] In response to the harshness of the single cause-of-action rule, some states allow the plaintiff to split the claim and sue on the cancer claim when the disease develops. [FN43]

Another aspect of claims for increased risk of cancer is the all-or-nothing rule of damages. Under this rule, if the plaintiff proves past, present or future harm to the appropriate standard of proof, he or she will be awarded 100 percent of his or her damages. [FN44] If the harm cannot be proved to the appropriate standard, the victim will receive nothing. [FN45]

Courts have articulated the standard of proof to decide increased risk claims in a number of different ways. Part II of this article discusses some of major approaches utilized by various courts in defining the standard of proof.

II. How Courts Have Decided Claims for Increased Risk of Disease

Typically, claims for increased risk of cancer arise in cases with the following generalized fact pattern: a governmental or industrial entity contaminates various environmental media, such as, groundwater, soil, air, or surface water with toxic substances that have the potential to cause cancer. [FN46] Then, various subpopulations, such as, residents, workers, or recreational users are exposed to the contaminants by ingestion or inhalation, or dermally. A plaintiff or a group of plaintiffs then sue on a theory that they should recover damages because their exposure to the carcinogen increased their risk of developing cancer in the future. [FN47] Much of the time the plaintiffs have no present injury other than the toxic exposure, which may or may not have a biological effect.

Courts have denied these claims by using a number of different rules. [FN48] Some courts recognize the claim in theory, but deny it, nonetheless. [FN49] Other courts require that the plaintiff show a greater than fifty ***220** percent chance of getting the disease. [FN50] Still others deny the claims, because the plaintiff did not establish the increased risk by a "reasonable certainty" or a "reasonable probability." [FN51]

An award of damages for increased risk of future disease is not unknown in the law. Some courts have allowed recovery in sporadic accident cases, not involving toxic substances, in which the plaintiff had another present injury.

A. Early Cases Denying Claims for Increased Risk of Disease

In Ayers v. Township of Jackson, the New Jersey Supreme Court acknowledged that a claim for increased risk of cancer

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against a municipality was compensable under the New Jersey Tort Claims Act. [FN52] However, the court held that if the plaintiff's expert could not establish the claim of future harm by a "reasonable probability," there could be no recovery. [FN53] The Court further required that plaintiff's expert be able to quantify the increased risk of harm. [FN54] The court concluded that the plaintiff had not met this burden. The dissent in Ayers viewed the injury as being exposure to the toxic chemical. The exposure had occurred and was not speculative nor a remote possibility. The dissent would have allowed recovery for increased risk of disease claims. [FN55]

Other cases denying such claims include: Amader v. Johns-Manville Corp. [FN56] and Bennett v. Mallinckrodt, Inc. [FN57] Amader is not a pure increased risk of cancer case. Rather in this case recovery was denied for someone claiming emotional distress as a result of watching a loved one having an increased risk of cancer develop the disease. In Bennett, plaintiffs suffering a radiological injury were denied recovery for increased risk of cancer because the future damage was not certain to occur. [FN58]

***221** B. Plaintiff Must Show a Greater than 50% Chance of Getting Disease

In Rabb v. Orkin Exterminating Co., [FN59] the plaintiffs argued that their claim for increased risk of disease was analogous to medical malpractice cases in which, a plaintiff could recover for a lost chance of survival caused by a medical practitioner's negligence. [FN60] Liability in the medical malpractice area was predicated on a probability of survival that was less than fifty percent. [FN61]

In Rabb, the Court declined to apply this theory of recovery to toxic tort cases, stating that the future consequences of an injury had to be reasonably probable. The Court deemed that a level of probability greater than fifty percent was required. [FN62]

C. "Reasonable Certainty" Cases

In Stites v. Sundstrand Heat Transfer, [FN63] the defendants failed to properly dispose of used chemicals. This conduct caused contamination of the plaintiffs' drinking water with trichloroethylene. The levels of exposure observed were higher than the EPA-recommended limitation. Therefore, plaintiffs brought a claim for increased risk of cancer. One of the significant aspects of this case was the nature of the analysis conducted by the experts of the parties. Defendant submitted a quantitative risk assessment showing a de minimis increase in total cancer risk because of the exposure. [FN64]

In contrast, plaintiffs' experts' assessment of the hazard was more qualitative in nature, citing that: 1) the EPA classified TCE as a "probable human carcinogen" and recommended that no TCE be allowed in drinking water; and 2) plaintiffs faced an aggregate risk that was well above the regulatory standard of 1 in a million, which was above the level defined by health officials as posing an unacceptable risk in either the workplace or domestic environments. [FN65]

The Stites court held that the plaintiff must demonstrate with "reasonable certainty" that the future consequences will occur. [FN66] The court concluded that they had not done so in this case. The court further stated that the quantitative assessment of defendants' expert was preferred to ***222** the qualitative one provided by plaintiffs' experts. [FN67] In short, the defense had shown that the likelihood that plaintiffs would get cancer was significantly less than the "reasonable certainty" standard required by Michigan law. [FN68] The Court also dismissed the fact that the plaintiffs demonstrated that the level of exposure exceeded regulatory standards, because such proof was insufficient to demonstrate that the Michigan standard of "reasonable certainty" had been satisfied. [FN69]

The "reasonable certainty" standard was quantified in Wilson v. Johns- Manville. [FN70] The court stated that the plaintiff must demonstrate a greater than fifty percent chance of injury to obtain full compensation for the injury. [FN71] The court commented that this was the traditional "all-or- nothing" approach and contrasted the traditional approach to the "pro-rata" approach used by the English courts. The "pro-rata" approach required defendants to pay a percentage of damages to the injured class based upon the probability of future harm.

The "reasonable certainty" standard has also been applied to increased risk claims in product liability actions. In Morrissy v. Eli Lilly & Co., [FN72] plaintiffs attempted to recover on claims for increased risk of cancer because they had been exposed to DES (diethystilbesterol) in utero while their mothers took the drug during pregnancy. The court thought that the

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connection between exposure to the drug and the possibility of developing cancer was insufficient and denied damages because they were not reasonably certain to occur. [FN73]

D. "Reasonable Probability" Cases

1. The Chemical Carcinogen Cases

In Anderson v. W.R. Grace & Co., [FN74] the defendant contaminated two wells in the Town of Woburn, Massachusetts with trichloroethylene and tetrachloroethylene. The plaintiffs, who were exposed to the contaminants in their drinking water, brought a claim for their increased risk of developing disease. [FN75] The Massachusetts courts had not ruled on the validity of this claim, therefore the District Court held that there had to be a "reasonable probability" the harm would occur to sustain the claim. Further, ***223** the court commented that a cause of action must have accrued at the time recovery is sought. [FN76] The court opined that this rationale was in harmony with the discovery rule, which was applied to latent disease cases. Under the discovery rule, the claim accrues with the manifestation of the disease. Furthermore, the court stated that the record did not indicate the magnitude of the increased risk or the diseases that the plaintiffs would suffer and cited the Ayers decision as requiring the same. [FN77]

The court also noted that although the plaintiffs complained of a variety of present illnesses, they had not demonstrated that the speculative future diseases would be part of the same disease process. In conclusion, the court stated that if future illness stemmed from the same disease process as the illness(es) the plaintiffs presently complained of, recovery must be sought in the instant action. [FN78] If the disease processes were different, the cause of action for the future illness would not accrue until the illness was manifested. [FN79]

Similarly in Hagerty v. L&L Marine Services, [FN80] the plaintiff was exposed to the known human leukemogen, benzene, and sought recovery on a claim for increased risk of cancer. The court held that recovery for increased risk of cancer would be allowed only where the plaintiff shows it more probable than not that exposure will [emphasis in original] lead to cancer. [FN81] Since the plaintiff had not shown his increased risk was greater than fifty percent, recovery was denied.

In Sterling v. Velsicol, [FN82] plaintiffs were exposed to the carcinogens, carbon tetrachloride and chloroform, after their well water had been contaminated by a hazardous waste landfill owned and operated by the defendant, a chemical manufacturer. The trial court sustained the claim for increased risk of cancer, but the appellate court reversed. The court held that based on Tennessee law, the potential risk of susceptibility to future disease must be medically, reasonably certain to follow from existing present injury. [FN83] Based on the facts of the case, the appeals court opined that an increased risk for susceptibility to cancer and other diseases of only twenty- five to thirty percent was not sufficient. Further, the Court commented that had plaintiffs' expert testified to a probability of greater than fifty percent the result may have been different. [FN84]

***224** 2. Asbestos Cases in Federal Court

The asbestos cases generally involved plaintiffs exposed to asbestos in the workplace, sustaining injuries such as asbestosis, lung cancer and mesothelioma. Claimants sued asbestos manufacturers on a theory of products liability as well as other theories, such as negligence.

a. Third Circuit

In Herber v. Johns-Manville, [FN85] the trial court did not permit plaintiff's expert to testify to Mr. Herber's increased risk of cancer. Apparently, trial counsel made a proffer of some sort concerning what the expert would testify to, but for some reason the record of the proffer was not clear. [FN86] The appellate court found that the expert did not have epidemiological data showing a class risk in excess of 50%, and that he was not prepared to offer an opinion that the plaintiff, more likely than not, would experience cancer. [FN87]

The Third Circuit noted a cause of action for anticipated future harm was recognized under New Jersey law, [FN88] provided there was a reasonable probability that the prospective consequences were expected to flow from the past harm. The Court also noted that a plaintiff could recover on a claim for increased risk under New Jersey law, if there was an underlying

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present physical injury. [FN89] The Third Circuit concluded that Herber had not proffered evidence of probable future cancer and denied recovery.

b. Fifth Circuit

1. Asbestos Cases Denying Recovery

The Court in Dartez v. Fibreboard Corp., [FN90] acknowledged that the Texas standard for damages in increased risk cases was a reasonable medical probability. Possibility was not enough to satisfy the preponderance of evidence standard; however, certainty was not required. [FN91] Although plaintiff's expert cited studies showing that persons in the same occupation as plaintiff had an increased risk of lung cancer, ranging from 35 to 60 percent, and an increased risk of mesothelioma ranging from 7 ***225** to35 percent, the Court held that this testimony did not rise to the level of a reasonable medical probability. [FN92] One factor that confounded the plaintiff's case was that he was a smoker and could have cut his risk by one-third if he had quit smoking. [FN93]

The facts in Adams v. Johns-Manville, [FN94] were less than supportive of the plaintiff-appellant's case. The plaintiff claimed he was troubled by shortness of breath, but he attributed this, in part, to lack of physical conditioning. During the two years before trial, he was able to play volleyball and touch football competitively and run for a mile and a quarter. [FN95] He was also overweight, smoked a pack and a half of cigarettes a day for ten years, and then smoked fifteen cigars a day from 1963 to 1979. [FN96] His pulmonary function tests were normal for a man of his age and his shortness of breath could have been attributable to his age, weight, and smoking history rather than asbestosis. [FN97]

Plaintiff did not complain of the symptoms of asbestosis (persistent shortness of breath, chest pain or cough) to either his own expert or to the defendant's expert. Plaintiff enlisted the aid of another expert who would testify that there was an increased risk of cancer in asbestos workers who had asbestosis, based upon a British study. [FN98] Further, the expert testified that there was an increased risk of cancer in persons occupationally exposed to asbestos, with a history of smoking. The expert, however, would offer no specific estimate of the plaintiff's probability of getting cancer. The court applied the reasonable probability standard and held that this evidence was insufficient. The court further commented that abstract statistics and generalizations would not suffice to satisfy the standard.

2. Asbestos Cases Allowing Recovery

The Fifth Circuit has permitted recovery on claims for increased risk of cancer in two asbestos cases: Gideon v Johns-Manville, [FN99] and Jackson v Johns-Manville. [FN100] In Gideon, the court applied the reasonable medical probability of cancer rule. Because the plaintiff's expert testified that the probability was greater than fifty percent that the plaintiff ***226** would die of an asbestos related cancer, the standard was satisfied. [FN101] The plaintiff in Jackson had asbestosis, which is interstitial fibrosis of the lung caused by inhalation of asbestos fibers, [FN102] and presented expert medical testimony of a greater than fifty percent chance of getting cancer. For these reasons, the plaintiff was allowed to recover.

E. Cases Allowing Recovery for Increased Risk of Disease

Claims for increased risk of disease have been sustained in sporadic accident cases. Most of these cases do not involve toxic substances. In all the cases, the plaintiff suffered from a present injury, even where the probability of future disease was small or unquantified. [FN103]

Although most of these cases did not involve toxic substances, Brafford v. Susquehanna Corp., [FN104] did involve a hazardous substance, radon gas. [FN105] The gas came from uranium mill tailings that the defendant had placed around the foundation of the plaintiffs' home. [FN106] The plaintiffs were exposed to levels of radiation well in excess of regulations. [FN107] There are two significant aspects of this case. First, the Court recognized that permanent chromosomal damage was a present, compensable injury. Second, although the expert had not quantified risk, he was prepared to present evidence supporting the increased risk claim. Specifically, plaintiffs' experts were prepared to testify to a reasonable degree of medical certainty that they suffered from present, permanent and irreparable genetic and chromosomal damage as a result of radiation exposure. Further, ***227** this sub-cellular damage was a present injury and that the trigger of cancer had been cocked.

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[FN108]

III. Policy Concerns Regarding Claims for Increased Risk of Cancer

A. Policy Reasons For and Against Claims for Increased Risk of Cancer

There are recurring themes in the reasoning provided by the courts in the cases discussed in Section II. These are summed up well in Mauro v. Raymark [FN109] and elaborated upon by Schmauder. [FN110] Policy reasons supporting claims for increased risk of cancer include:

(P)ostponing redress of the claim until the disease is manifested would allow intervening causes to attenuate the chain of causation; [FN111]

not imposing liability thwarts tort law's capacity to deter improper use of toxic chemicals; [FN112]

applying the all-or-nothing rule rejects claims where risk may be substantial, that is 49% or less but falls short of the required 50%. [FN113]

The Dissenting Opinion in Mauro would have recognized these claims for several reasons. Actions brought long after the exposure occurred present enormous procedural and administrative obstacles to plaintiffs. [FN114] Evidence is lost, witnesses are unavailable, and memories fade. [FN115] Moreover, the transcripts needed for preparation, discovery, direct and cross- examination and record reconstruction disappear. [FN116]

Schmauder adds to this list of concerns that plaintiffs would have to prove causation a second time and face at least double the expenses in legal and expert witness fees. [FN117] In addition, the defendant may have gone bankrupt in the interim. [FN118] Plaintiffs would also be left with the problem of determining when the statute of limitations ran and whether her evidence met the standard of proof required by the court. [FN119] Further, the arbitrary line drawing of the all-or-nothing rule assured that many significant risks would not be redressed. Ultimately, this rule promotes ***228** under-compensation of tort victims, thus defeating the major goal of tort law, compensation for wrongs suffered. [FN120] Furthermore, the cost of risk-creating behavior would not be internalized into the price of the product, thus diminishing the deterrent effect of the tort law. [FN121]

The Court in Anderson v. W.R. Grace, [FN122] articulated several policy reasons against claims for increased risk. The court viewed claims for increased risk of cancer as speculative and for this reason avoided them. [FN123] Also, the court reasoned that, if the cancer complained of developed in the future, a claim could be brought at that future date. [FN124] Further, to award damages based on mathematical probabilities would significantly under-compensate those who actually developed cancer and would be a windfall to those who did not. [FN125] Furthermore, the Court opined that allowing recovery would create more lawsuits. [FN126] Klein [FN127] develops this argument by stating that courts have limited the availability of damages for increased risk in toxic tort cases out of concerns regarding judicial caseloads. [FN128] They reason that it makes more sense to award these damages in a few scattered head injury cases, [FN129] but not in a wave of asbestos cases. [FN130]

B. Advantages and Disadvantages of Claims for Increased Risk [FN131]

Legum [FN132] stated the advantages of allowing recovery for claims of increased risk: Evidence to establish causation and negligence is more readily available in an increased risk claim than in the typical cancer action. Even in states not recognizing the discovery rule, statutes of limitations would not be a complete bar to the action.

Then, too, defendants would more likely be solvent and extant at the time of the lawsuit, because the action can be brought shortly after exposure. [FN133] Additionally, lack of medical knowledge concerning the cancer mechanism will not confound the causation issue, because current science can establish whether or not a toxic exposure subjects a plaintiff to ***229** increased cancer risk. [FN134]

Substantial justice can be achieved between the parties, because plaintiffs unable to recover any damages can obtain at least

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limited damages; that is, some compensation is preferable to no compensation. Additionally, defendants are in a better position to internalize the costs of their risk bearing activity into the price of their products, than plaintiffs. [FN135] Also, the promptness of the increased risk claim enhances deterrence of irresponsible behavior on the part of risk creators. [FN136]

Further, Legum states that since American companies plan on the short-term, it is unlikely that the prospect of liability twenty or forty years in the future would induce them to modify behavior in the present. Furthermore, those making decisions concerning the handling of hazardous chemicals may not be the same people who own the corporation years later and the promptness of the increased risk action offers more deterrent effect and fairness. [FN137]

A disadvantage to plaintiffs of the increased risk claim is that increased risk judgments will not be enough to cover the costs associated with treating the cancer should it develop later. [FN138] For defendants, such a significant involuntary commitment of capital could lead to over-deterrence. [FN139] In extreme cases the impairment of capital could affect the viability of the business entity. Further, defendants may be held liable where science indicates that a substance is a possible or probable carcinogen, but which upon further evaluation is found to be non-carcinogenic. Furthermore, according to the "baseline risk paradox" defendants may be held liable for a result, which its conduct did not cause.

IV. Proposed Remedies for Handling Claims for Increased Risk of Disease

A. Limit the Claim

One commentator argues that courts should limit recovery for risk of disease to medical monitoring and emotional distress and not extend it to harm that may occur in the future. [FN140] If the cancer develops in the future, a lawsuit could be brought at that time. [FN141] This is consistent with ***230** traditional tort law, which seeks to provide compensation for all losses, which are the direct and proximate cause of a wrong. [FN142] Further, it is a logical approach, producing a fair and equitable system that compensates plaintiffs for loss, not chance. [FN143]

Another commentator articulated the problems with this approach. [FN144] Plaintiffs will have to prove causation a second time and the contribution of intervening causes will have to be minimized. Thus, plaintiffs will incur at least double the legal expenses and expert witness fees. The nature of the proof will change because evidence is lost and the memories of witnesses fade. Also, the defendant may no longer be a viable business entity. Further, the plaintiff has the arduous task of determining when the statute of limitations expires. Furthermore, the plaintiff must determine if risk exceeds the applicable standard of proof.

B. Recognizing Increased Risk Itself as the Harm

Other commentators would recognize that placing the plaintiff at increased risk is a harm that should be compensated. [FN145] One would define injury not as physical harm, but as the invasion of a protected interest of another. [FN146] He cites examples of currently protected intangible interests in privacy and nuisance. [FN147] The other, takes the position that increased risk of disease upon exposure to a toxic substance should be a legally actionable injury. [FN148]

C. Allow Recovery for Increased Risk Where Plaintiff Can Show Risk Has Been Doubled

Another commentator proposes that tort law should permit enhanced risk recovery on a proportional basis, but only when the plaintiff can demonstrate that toxic exposure has more than doubled her risk of contracting disease in the future. [FN149] This proposal is in essence a rewording of the standard requiring proof of future risk of more than fifty percent in epidemiological terms of relative risk (i.e. allowing recovery where relative risk is greater than two). [FN150]

***231** D. Insurance Remedies

Several commentators have proposed insurance-based remedies. Where a plaintiff can demonstrate present injury and a reasonable probability that a disease will occur in the future, one commentator argues that a court should use its equitable powers to order the defendant to pay the premiums of a policy to insure against the risk that the injury will occur in the future. [FN151] The article also supports viewing -sub-clinical injuries as a present injury for purposes of determining compensation for increased risk. [FN152]

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Viewing sub-clinical injuries as harms requiring compensation was examined in another article, which advocates recognition of the "extent of the injury" rule [FN153] in toxics cases. In applying this rule, courts would recognize genetic or sub-cellular damage as injury. [FN154] Further, this note comments on insurance packages for exposed plaintiffs. [FN155]Policies could be structured to pay medical treatment costs if and when a designated disease develops, provide supplemental income if the plaintiff is unable to work, and compensate the plaintiff's successors in the event of death. [FN156] Defendants may benefit as well, by being able to spread the costs over the latency period of the disease. [FN157] However, safeguards would be necessary to ensure that defendants paid premiums, or purchased pre-paid policies. [FN158]

Professor Rosenberg advocates the use of an insurance- fund remedy to compensate plaintiffs. [FN159] A liable defendant would be required to pay the mass-exposure plaintiff the premium that would purchase an insurance policy providing tort-type and tort-level damages when the ultimate harm occurs. [FN160] This remedy would be used in conjunction with the collectivization of claims discussed in Section IV F., below. [FN161] The insurance model would also use damage schedules that disregard the individualized determination of damages and other tort-related factors. [FN162]

Another article discusses insurance pools, which could be used ***232** when several defendants negligently expose a small number of workers to a carcinogen. [FN163] One insurance company could invest in determining the risk posed by that chemical and sell insurance policies to all the defendants, thus pooling risk and capitalizing on its investment in information. [FN164]

This article also discusses some of the problems with insurance remedies. [FN165] First, an upper limit on the policy payout would reduce the insurer's risk. [FN166] Also, a fixed-sum policy would lower the price of insurance to the defendant, while shifting risk onto the plaintiff. [FN167] Insurance would only be available if risk could be estimated reliably, which generally happens only when many people are exposed to the same risk. [FN168] Then too, for a risk estimate to be reliable all persons exposed would have to be exposed by the same defendant. [FN169] Although there are problems with insurance remedies, they should be explored and refined. Additionally, risk assessment can be used to calculate premiums.

E. The Escrow Fund

Another proposal is the establishment of an escrow fund, to which defendants would make payments to cover risk damages. [FN170] This would address the problems of judgment-proof defendants and would ensure that those plaintiffs not developing the disease would not be compensated, while those who do would be paid in full. [FN171]

This same article also points out the problems [FN172] with the escrow fund. First, such a significant involuntary commitment of capital could lead to over-deterrence. Second, if only a fraction of the pool of increased risk plaintiffs brought claims, the escrow account would contain only a fraction of expected damages. Also, defendants may go bankrupt, thus limiting recovery to the fund. If only a few plaintiffs sued, or if the plaintiffs suing had a higher incidence of the disease than the general plaintiff pool, there may not be enough money in the account to fully pay ***233** the plaintiffs who made risk-based claims. Thus, the escrow fund remedy would lead to the judgment- proof problem arising in non-risk-based suits. Additional problems arise if the estimate of risk or harm is inaccurate; i.e. an estimate that is too high will impair a defendant's capital and an estimate that is too low will leave plaintiffs under-compensated.

F. Toxic Death Risk Index Tax

With toxic death risk index tax, industries would be taxed, based on the number of people likely to be killed or injured in a toxic accident. [FN173] Imposition of liability under this system would be based on actual risk created by the polluter and there would be an economic incentive for users and producers of toxic substances to emphasize safety. [FN174]

G. Collectivization of Increased Risk Claims

Professor Rosenberg advocates the collectivization of risk-based claims by use of the class action in mass exposure cases. [FN175] Collectivization would increase the deterrence value of these claims and notions of individualized justice would not be compromised, because, otherwise, unmarketable mass-exposure claims are given access to the tort system. [FN176] This remedy would be used with the insurance-fund remedy discussed in Section D. Professor Rosenberg views risk-based claims for medical- monitoring as unnecessary for deterrence purposes, because mitigation of damages is in the defendant's best

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interest anyway. [FN177]

V. Using Risk Assessment to Evaluate Claims for Increased Risk of Cancer

It is certainly true that the cancer complained of in claims of increased risk of cancer may not develop. However, risk assessment [FN178] provides a means for discriminating between trivial risks and more serious ones. [FN179] The EPA published Guidelines for Carcinogen Risk Assessment***234** in 1986. [FN180] The 1996 Proposed Guidelines for Carcinogen Risk Assessment [FN181] update the 1986 Guidelines.

A. The 1986 Guidelines for Carcinogen Risk Assessment

Under the 1986 guidelines the linearized multistage model [FN182], provides estimates indicating whether a given exposure poses a risk of one in a million people getting cancer, one-in-one-hundred thousand, one-in-ten- thousand, one-in-a-thousand, one-in-one- hundred or one in ten. So, if exposure to a given carcinogen only poses a one in a million chance of developing cancer, this risk may be deemed arguably too trivial to be recompensed. However, if exposure was associated with a one-in-ten risk, that is significant and damages arguably should be awarded. Alternatively, damages could be determined on a sliding scale. For a one-in-ten or one-in-one-hundred risk, substantial damages could be awarded. One in one million risk would be recoverable at a lower rate or be considered too remote to be recoverable at all. Damage cut-offs for the intermediate-risk levels could be defined as well. Thus, risk estimates could be used to draw lines concerning when damages should be awarded based on a stated policy of the court. After trial to determine liability and prove damages, damages could be apportioned based on the estimates of risk.

Another aspect of risk assessment that should be considered simultaneously with the quantitative estimates is the EPA Weight-of-Evidence Classification System for Carcinogenicity [FN183], which categorizes carcinogens according to their potential to cause cancer in humans.

This system categorizes carcinogens as follows:

Group	Description
A	Human carcinogen
B1 or B2	Probable human carcinogen B1 indicates that limited human data are available B2 indicates sufficient evidence in animals and inadequate or no evidence in humans
C	Possible human carcinogen
D	Not classifiable as to human carcinogencitiy
E	Evidence of noncarcinogenicity in humans

***235** For example, benzene a human carcinogen is given a Class A designation. [FN184] Thus, those individuals exposed to known human carcinogens have a more persuasive argument in support of the award of damages.

B. The 1996 Proposed Guidelines for Carcinogen Risk Assessment [FN185]

The major changes of the 1996 guidelines as compared to the 1986 guidelines are in the weight-of-the-evidence determination of carcinogenicity and the dose-response assessment. [FN186] The 1996 guidelines replace the Group A (human carcinogen) to Group E (evidence of noncarcinogenicity) alphanumeric descriptors, with the following descriptors: "known/likely," "cannot be determined," and "not likely." [FN187] The "known/likely" descriptor is further characterized by

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three subcategories. The first subcategory includes those agents known to be carcinogenic in humans based on epidemiological and experimental evidence. [FN188] The second includes those agents that should be treated as if they are human carcinogens, based on a combination of epidemiological evidence, showing a plausible causal association and strong experimental evidence. [FN189] The last subcategory includes those agents likely to produce cancer in humans because of production or anticipated production of tumors by modes of action that are relevant to human carcinogenicity. [FN190] This group can also be assigned modifiers of high end and low end when the weight of evidence for an agent of interest is compared to other weights of evidence typical for the group. [FN191]

The "cannot be determined" descriptor includes those agents with ***236** databases of evidence concerning tumor effects or other data that are suggestive or conflicting or limited in quantity. [FN192] Four sub- descriptors further delineate this group. The first two sub-descriptors are based on the nature of the evidence, that is, suggestive; or conflicting (some of the evidence is suggestive with other evidence not confirming this). The second two sub-descriptors are based on the quantity and/or quality of the data, that is, inadequate data and no data. [FN193]

Agents given the "not likely" descriptor are those agents with experimental evidence that suggests no basis for a human hazard concern. [FN194] There are four subcategories to this group. The first subcategory includes those agents that are not considered to be carcinogenic because negative results were obtained in two well-conducted studies, in two animal species. The second is composed of those agents that are carcinogenic in animals, but the carcinogenic effects exhibited by animals are not relevant to humans. The third subcategory includes those agents exhibiting carcinogenic effects that are dose- or route-dependent. An example of dose-dependency is an agent that is not likely to cause cancer below a certain dose. An example of route-dependency is an agent that is not likely to cause cancer by oral exposure, but is likely to cause the disease by inhalation. The last subcategory includes those agents with which extensive human experience demonstrates a lack of effect.

The reasoning for placing a chemical into one of these categories must be discussed in a narrative. [FN195] The guidelines present numerous examples of the various descriptors and their accompanying narratives. [FN196]

The 1996 guidelines also made changes to the dose-response assessment. Typically, environmental exposures are much lower than occupational or experimental exposures and certain extrapolations must be made to characterize the dose-response relationship. [FN197] Three general extrapolations are made. An extrapolation from high (typically experimental or occupational exposure levels) to low dose (typically the environmental exposure of interest) is made. Next, if the experiment being used to estimate risk is in a non-human animal species, an extrapolation from animal to human responses is made. Lastly, if an inhalation experiment is used to estimate risk for oral exposure (or vice versa) an extrapolation from one route of exposure to another is made. [FN198]

***237** Whenever data are sufficient, a biologically based or case-specific response model is used to extrapolate risk at low doses. [FN199] The guidelines, however, anticipate that only in rare cases will the data be sufficient to derive a case-specific response model. [FN200] Therefore, three default procedures were developed: linear, nonlinear and both linear and nonlinear (that is, the data for a particular chemical shows that it is characterized by both a linear and nonlinear approach). [FN201] The agent's mode of action determines which procedure is used. [FN202]

The linear default procedure is used, when a chemical has a mode of action demonstrating mutagenicity because of DNA reactivity. Likewise for those agents not reacting with DNA, but with insufficient evidence of nonlinearity, the linear procedure is used. Lastly the linear procedure is used where linearity can be inferred because the background of human exposure to the agent is on the linear part of the dose-response curve that is sublinear overall. [FN203]

Utilization of the nonlinear default procedure is indicated where no evidence of linearity exists and the dose-response relationship falls more quickly than linearly with dose. The nonlinear procedure is also used where tumor response appears to be secondary to a toxic response that has a threshold. [FN204] Lastly, the nonlinear procedure is used where the response of concern is a precursor to tumor effects e.g. changes in hormone levels or mitogenic effects. [FN205]

Both a linear and nonlinear approach is used where tumors are observed at different sites of the body and the modes of action are different for the individual sites. Generally, this approach is used for chemicals that are mutagens, but also exhibit

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nonlinearity for a particular tumor type at another site of the body. The tumor response at the second site may be secondary to a toxic response that has a threshold. [FN206]

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***238** For all of these procedures, that is the purely "linear", "nonlinear" and "both," if risk is estimated based on an animal study, "dose" is expressed as a human equivalent dose. [FN207] "Dose" is further defined as the cumulative dose received by the experimental group of animals in question over their lifetime, (lifetime-average-daily dose). [FN208] As alluded to above, non-tumor responses can be used to characterize the dose-response curve [FN209]; and are referred to as precursor effects. Precursor effects include: changes in DNA, chromosomes or key macromolecules; effects on growth signal transduction; induction of physiological or hormonal changes; effects on cell proliferation or other effects that play a role in the process. [FN210]

The lower 95% confidence limit [FN211] on a dose associated with a 10% tumor response or relevant nontumor effect LED10 is identified on the dose response curve as the point of departure for extrapolation from high experimental exposures to lower, environmental exposures of concern. [FN212] ***239** The point of departure is merely the point defined by the dose-response curve from which the extrapolation to low dose is made. [FN213]

In the linear procedure, a straight line is extended from the LED10 to the origin, i.e. zero dose, zero response. [FN214] The linear default is thought to produce an upper bound on risk at low doses, that is a 1/100,000 to 1/1,000,000 and produces numerical results that are about the same as the former linearized multistage model. [FN215] The linear multistage model is eschewed by the 1996 guidelines, because it gives an appearance of specific knowledge and sophistication, which is unwarranted. [FN216]

With the nonlinear approach, a margin of exposure analysis compares the LED10 with an environmental exposure of interest, by computing the ratio between the two [FN217] (i.e. divide the LED10 by the environmental exposure of interest). [FN218]

If, as in the case where carcinogenicity is secondary to another toxic effect that has a threshold, a margin of exposure analysis is the same as that done for a noncancer endpoint. [FN219] A factor of ten each may be used to account for human variability and interspecies differences in sensitivity when humans may be more sensitive than animals. [FN220] When humans are less sensitive, a 1/10 fraction may be applied to account for this decrement in sensitivity. [FN221]

The database for a given chemical, especially the human and animal studies defining the dose-response curve is examined to develop these analyses. Then, a single well-conducted study is used as a basis for the risk assessment. Alternatively, the risk estimate may be based on several different studies through meta-analysis. The dose-response data for a given chemical is plotted, as in Figure 3-1. The dose-response curve indicates ***240** what is expected to occur upon exposure to the chemical of interest. The human exposure of interest is defined by the exposure assessment, at a given hazardous waste site or other pollution source. [FN222] The human exposure of interest is then compared to the expected as defined by the dose-response curve.

For chemicals indicating linearity, if the exposure of interest falls on the projected linear portion of the dose-response curve, the upper bound on risk is thought to be 1/100,000 to 1/1,000,000. [FN223] In theory, at least, the linear approach produces numerical results that are about the same as the former linearized multistage model. [FN224] If the human exposure is toward the upper end of the projected linear portion of the curve, then exposure is more problematic. In these situations, the linearized multistage model should be run to determine if a given exposure level poses a risk of one-in-one- million, one-in-one-hundred-thousand, one-in-ten-thousand, one-in-one-thousand, one-in-one-hundred or one-in-ten.

Referring to Figure 3-1, the picture for the judge or jury is simple. If the human exposure of interest falls on the projected linear portion of the dose response curve, there is no significant risk. If it falls at the upper portion of the projected linear portion of the curve, close to the LED10 the linearized multistage model should be used to define the risk.

For chemicals indicating that the nonlinear approach should be used a margin of exposure analysis is used. This analysis compares the LED10 with the environmental exposure of interest, by computing the ratio between the two [FN225] (i.e. by dividing the LED10 by the environmental exposure of interest). [FN226] As the human exposure of interest approaches the LED10, the margin of exposure gets smaller indicating an exposure that could pose a significant risk. If the exposure of

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interest falls above the LED10, the risk is significant.

Once again referring to Figure 3-1, the picture for the judge or jury is simple. The greater the margin of exposure is, that is the farther away the human exposure of interest is from the LED10, the lesser is the risk. As the human exposure approaches or exceeds the LED10, the risk is greater.

Since these tumor effects are thought to be secondary to other toxic effects, a threshold can be defined whereby exposures below the threshold probably do not cause tumors. Exposures above the threshold are, of course, more likely to cause such damages. For purposes of calculating damages, exposures below the threshold should not be compensated because ***241** they are unlikely to result in cancer.

Although it is not stated in the guidelines, the EPA has essentially made a policy decision concerning the way it will analyze carcinogens. Where the mode of action indicates genotoxicity, an approach that is more protective of public health, i.e., conservative, is warranted. This is the linear approach. However, the guidelines treat differently those chemicals with a different mode of action. Specifically, for those chemicals producing a tumor response, because of a toxicological reaction, defined by a threshold, a less conservative approach is warranted. This is because tumor response is not expected to be significant below the threshold. The new guidelines are a sensible approach, which is consistent with the philosophy that chemicals that are relatively safe when compared to others, should be regulated less stringently.

C. 1992 Guidelines for Exposure Assessment [FN227]

The goal of the exposure assessment is to estimate the dose of a chemical to which individuals or populations are exposed. The results of the exposure assessment are combined with the chemical-specific dose-response data of the toxicological assessment at a site to estimate risk. Making quantitative exposure [FN228] estimates can be approached in three different ways: 1) Point of contact; 2) scenario evaluation; and 3) by reconstruction through internal indicators.

1. Point of contact-measurements [FN229]

Point of contact measurements are taken while exposure occurs, at the interface between the person (at the outer boundary of the body) and the environment. Both exposure concentration [FN230] and contact time [FN231] are measured and integrated.

One example of point of contact of measurements is the radiation dosimeter. It is a small badge-like device that measures exposure to radiation ***242** as it occurs, providing an integrated estimate of exposure over a period of time. Another example is the Total Exposure Assessment Methodology (TEAM) studies conducted by the EPA. In these, a small pump with a collector and absorbent is attached to the person's clothing to measure exposure to airborne solvents or other pollutants.

2. Scenario Evaluation [FN232]

In a scenario evaluation, the assessor attempts to determine the concentration of chemicals in a medium. [FN233] The assessor then links this data with the time individuals or populations are in contact with the chemical. Exposure is estimated by evaluating exposure concentration and time of contact separately, that is at different times and locations. [FN234] Specifically, samples of the environmental media are collected at a site and are analyzed in the laboratory to determine concentration. A risk assessor sitting in her office hundreds of miles away from the site derives various time of contact hypotheticals.

An exposure scenario assumes how the contact may take place. For example, in a risk assessment for a hazardous waste site, the future use of the land often determines the scenarios to be considered. The risk assessor evaluates exposure of a hypothetical resident, construction worker, or recreational user. Certain assumptions are made about the duration of exposure, ingestion or inhalation rate, and body weight, to construct the scenarios. The hypothetical use of the land and the exposure assumptions constitute the exposure scenario.

3. Reconstruction by Using Internal Indicators [FN235]

Where exposure takes place and internal indicators such as biomarkers, [FN236] body burden, [FN237] or excretion levels

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[FN238] can be determined, ***243** dose [FN239] can be reconstructed or back-calculated using the indicator.

D. Risk Is Determined by Combining the Results of the Toxicological Assessment with the Exposure Assessment

Risk assessors generally consider two types of risk: individual and population. Individual risk is that borne by an individual within the population. [FN240] Risk managers are typically interested in the risk posed by high-end exposure estimates [FN241] and the central tendency. [FN242]

Population risk is the estimate of harm for a population and can be determined by summing the individual risks, if they are known, which is rarely the case, or by various methods to calculate risk using the arithmetic-mean dose. [FN243]

The dose estimated from the exposure assessment is combined with chemical- specific dose-response data, which are usually derived from animal studies, to estimate risk. [FN244] Courts and juries may use these risk estimates to determine risk on a pro-rata basis and calculate damage awards for claims involving increased risk.

Utilization of this approach makes even more sense when one considers that there is a wealth of information that has been compiled by the EPA, accessible on their website, concerning chemical-specific dose-response relationships. Furthermore, risk estimates have and are being derived for numerous hazardous waste sites as part of the Superfund remediation program. Attorneys, courts and juries, with the assistance of an appropriate expert, can utilize this data in calculating damages or insurance premiums for claims involving increased risk.

Additionally, although the risk estimates are not "actuarial" because they are not based on sufficient empirical data, they can be used as a guide to calculate damages. Many courts have already awarded damages ***244** for medical surveillance. If this medical surveillance data were deposited in a central registry, over time, the empirical data necessary for use of the estimates actuarially would be compiled. Also, there may be some databases for chemicals that have thorough epidemiological studies from which better estimates for human response could be derived. Then, too, better empirical data for use actuarially could be refined from exposure assessments based on point-of-contact measurements and reconstruction of dose methods.

Through time, better information concerning the carcinogenic effects of chemicals will be derived. If it is shown that risk estimates based on best available assessments made at the time of litigation show that a chemical is not as carcinogenic in humans as anticipated, then an appropriate institutional mechanism should be legislated whereby the money assessed from defendants in payment of such damages is returned to them in grants or tax relief to develop safer methods of handling substances and for improvements in pollution control technology.

E. The Problem of the Indeterminate Plaintiff Revisited

As alluded to in Section I. C., one can never be certain whether a particular person's cancer may be attributed to the risk caused by defendant's conduct or to baseline risk. As indicated by Walker, defense counsel wants to increase the contribution of baseline risk, because as baseline risk increases, the contribution of the defendant's culpable conduct may be minimized. [FN245] Plaintiff's attorneys desire to minimize baseline risk to show that defendant's conduct was the primary source of risk and the major cause of the plaintiff's cancer. [FN246]

From the perspective of both sides, there are a few things that should be considered. Questionnaires (plaintiff's initial interview memorandum or defendant's discovery demands) should characterize the plaintiff's present and past medical history, including medications, his or her lifestyle, diet, exercise regimen and present and past habits, including, smoking, drinking, and drug use.

The plaintiff's exposure to chemicals occupationally or in the environment (i.e. where the plaintiff works and lives) should be analyzed. Socioeconomic status and geographic location should be considered as well.

From the perspective of plaintiff's attorneys these factors should be evaluated, with the help of an expert, and a realistic estimate of the contribution of these factors to plaintiff's cancer risk should be made. This ultimately determines the likelihood of success upon litigation.

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***245** From the perspective of defense counsel, the contribution of the above factors should be maximized, so that the amount of additional risk that defendant's conduct caused can be accurately measured. As an example of the type of fact patterns plaintiff's counsel should avoid see Adams v. Johns- Manville. [FN247] In this case, intervening factors, which may have contributed to the plaintiff's condition, were not controlled by plaintiff's attorney and the experts. The plaintiff smoked, had poor nutrition, and was overweight. Moreover, the symptoms exhibited could have been attributed to other factors. Plaintiff's pulmonary function tests were normal. Furthermore, the plaintiff's activity level was inconsistent with a diseased state. Defense counsel seized upon these inadequacies and won. This case is a good example of the type of factual analysis that defense counsel should conduct in defense of these cases.

Although it is not possible to completely obviate the "baseline risk paradox." [FN248] There are some things that should be considered by plaintiff's counsel in his or her approach to this problem. The first thing to consider is whether the cancer complained of is manifested by a "rare tumor type," such as the development of mesothelioma upon exposure to asbestos. [FN249] It may be deduced that a "rare tumor type" is one that occurs infrequently in the general population. Thus, it is unlikely that the cancer occurred naturally and is attributable to background risk. An expert can provide the attorney with information regarding whether exposure to the chemical in question is associated with an increased incidence of a rare tumor type.

The EPA's website provides a great deal of information on the most commonly used chemicals by simply browsing through the topics. Also, the EPA's Integrated Risk Information System [FN250] has summaries of risk information and provides a weight-of-the-evidence determination of the carcinogenicity for many chemicals and chemical-specific risk estimates. Other useful repositories of hazard information include websites for: NIOSH (National Institute of Occupational Safety and Health), [FN251] ***246** ACGIH (American Conference of Governmental Industrial Hygienists), [FN252] NTP (National Toxicology Program), [FN253] ATSDR (Agency for Toxic Substances and Disease Registry), [FN254] and IARC (International Agency for Research on Cancer). [FN255]

Another approach to the "baseline risk paradox" is to use background exposure as a "proxy" for baseline risk. Specifically, in the risk assessments for many hazardous waste sites, background exposure to the chemical is evaluated by taking measurements for pollutants of concern in areas where little or no chemical contamination has occurred. Thus, background risk can be quantified based on background exposure to get a more accurate picture of how much risk is attributable to chemical contamination (i.e. defendant's conduct). In addition, for some sites there may be historical background values available as well.

Additionally, the epidemiological evidence should be evaluated carefully for confounding factors. If confounding factors are controlled in a study, a determination of how chemical exposure contributed to overall risk can be made. For example, smoking is a well-known confounding factor of the asbestos epidemiological studies. [FN256] However, some of the studies determine how smoking combined with exposure to asbestos to affect risk. [FN257] Also, perhaps a variable such as diet is considered and controlled in an epidemiological study and found to have no effect on risk. Obviously, an expert must evaluate the database for a chemical and make these determinations.

F. Limitations of Risk Assessment

Some commentators have discussed the limitations of risk assessment. Gillette and Krier, [FN258] comment that one of the dangers in relying on risk assessment as currently practiced by administrative agencies is that all interest groups do not have equal access to the agency's decision- ***247** making process. [FN259] Since risk victims tend to be geographically dispersed it is more difficult for them to form coalitions to affect the public policy debate. [FN260] Whereas risk-producers often are organized into trade associations with substantial resources, making it is easier for them to affect agency policy. [FN261]

An additional limitation of risk assessment is that the risk assessor defines risk in terms of mortality or morbidity, i.e. body counts. [FN262] Lay people tend to have a more nebulous view of risk [FN263] and are concerned about risks that have catastrophic potential, that are unfamiliar, uncontrollable, involuntary, that threaten future generations, that would concentrate fatalities in time and space, that are distinctively threatening as opposed to shared by the general public, and that are manmade as opposed to natural. [FN264] These are risk judgments associated with "dread." [FN265] In spite of the limitations of risk assessment, the authors do not conclude that the information it provides is irrelevant; however, they argue

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it should not be exclusively or dominantly relevant. [FN266]

These authors also point out that risk assessors deal with trans-scientific issues, requiring judgment as well as quantitative analysis. [FN267] They often allow their emotions, biases, values and views about political and social issues to influence their risk decisions. [FN268] The authors also comment on "the blurring of the roles of the basic scientist, the technologist, and the entrepreneur," which "denies hazard management one of its strongest sources of early hazard identification--knowledgeable but independent basic scientists." [FN269] It is true that often the chief technical consultant at an environmental firm will be the person applying for the grant or responding to the request for bids. This person often does not want to be the bearer of bad news to a client and this may influence his or her risk analysis.

***248** Applegate [FN270] chronicled other limitations. He states that basic data needed for risk assessment of chemicals are limited, i.e., available information is scanty, and we do not have resources to test all chemicals and there are too many uncertainties involved in the data. [FN271] To an extent this is true, when one considers the great number of chemicals produced in an industrial society. However, the databases for some chemicals are quite extensive and the degree of professional confidence in the risk assessment is often characterized. [FN272]

Although Applegate states that estimating long-term risks is difficult and the results are highly subjective, [FN273] this is not true from the perspective of the risk assessor. These are not difficult calculations to make and the use of computers in calculation and data management makes the assessment less time consuming. Doing the fieldwork to obtain samples and the laboratory analysis of the data is costly; however, much of the data has to be acquired by any means to effectuate the cleanup of hazardous waste sites. Additionally, the results are not necessarily highly subjective; skilled risk assessors can be made to justify, to a fact finder, their reasoning and the assumptions used as required by the 1996 Proposed Guidelines, which seek to bring transparency to the risk-assessment process.

Applegate argues that other considerations should be made a part of environmental decision-making, such as public, cultural and historical values, quality of life and public anxiety. [FN274] Another important value is equity, i.e., risks are not distributed evenly across the population: wealth, race, neighborhood, advanced age and infancy make significant differences in susceptibility and exposure. [FN275] Intergenerational equity is also a concern. [FN276] Within the context of litigation, the function of the jury is to evaluate the facts. To an extent, jurors bring values with them, and through their deliberations these issues are fuel for the debate.

Applegate also argues that value judgments and political choices cannot be avoided in setting priorities. Risk assessment should not be used to create a false sense that environmental issues can be analyzed ***249** based on science alone. [FN277] Further, the numbers derived by risk assessment have a persuasive power and take on an unrealistic perception of precision and authority, which obscures the underlying uncertainty. [FN278] This perception may or may not be true and the 1996 guidelines have, therefore, eschewed the approach of the linearized multistage model, because it gave a false sense of scientific certainty where there was none. Values are always going to affect the risk assessor; they are human. However, the 1996 guidelines call for transparency in the reasoning process, which provides a method for ferreting them out of the risk assessment, if not the risk assessor.

VI. Conclusion

Attorneys, judges and juries should utilize risk assessment as a tool in deciding claims for increased risk of cancer. Risk assessment used in conjunction with the jury system can be made to weed out trivial claims and award damages where risk is significant. [FN279] Also, it can be delineated as a method to calculate damages and insurance premiums, allowing some of the remedies proposed by commentators to be effectuated. It can help effectuate the goal of compensation by providing plaintiffs, who otherwise would not have a remedy, with damages, while at the same time providing limitations on a defendant's damages.

Much data exists for a number of chemicals, and the contribution of chemical exposure to risk has been calculated for many waste sites. Courts and juries should be utilizing this data to decide claims for increased risk of cancer.

To implement such a result, courts and legislators must abandon the artificial all-or-nothing rule and antiquated notions of standards of proofs involving "reasonable certainty," "reasonable probability," and "greater than fifty percent chance of

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disease." These simply do not comport with best available scientific reasoning. Justice Cardozo stated [FN280]:
There should be greater readiness to abandon an untenable position when the rule to be discarded may not reasonably be supposed to have determined the conduct of the litigants, and particularly when in its origin it was the product of institutions or conditions which have ***250** gained new significance or development with the progress of years.

Thus, the all-or-nothing approach does not reasonably determine the rights and liabilities of plaintiffs and defendants in toxic tort cases. The rule originated from sporadic accident cases, in which injury is immediate and can be readily assessed. To apply this rule to cases involving a latency period ignores developments and progress in the fields of epidemiology, toxicology and risk assessment. A new rule should be implemented as soon as possible.

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[FN1]. See infra notes 36-38 and accompanying text.

[FN2]. David Rosenberg, Book Review: The Dusting of Asbestos--Carnage, Cover-up and Litigation. Outrageous Misconduct: The Asbestos Industry on Trial By Paul Brodeur, 99 Harv. L. Rev. 1693, 1693 (1986).

[FN3]. Id. at 1693.

[FN4]. MSN Learning and Research at http://encarta.msn.com/encnet/refpages/ISRPage.aspx?search=cancinogen&x=15&y=18. (Dec. 14, 2002). A carcinogen is any chemical, biological or physical agent that can potentially be a cause of cancer.

[FN5]. Tamsen Douglass Love, Special Project: Environmental Reform in an Era of Political Discontent: Deterring Irresponsible Use and Disposal of Toxic Substances: The Case for Legislative Recognition of Increased Risk Causes of Action, 49 Vand. L. REV. 789, 796 (1996). For a discussion of (b) see infra notes 109-20 and accompanying text. For a discussion of (c) see infra note 11, 109-20 and accompanying text.

[FN6]. Melissa Moore Thompson, Enhanced Risk of Disease Claims: Limiting Recovery to Loss, Not Chance, 72 N.C. L. Rev. 453, 468 (1994).

[FN7]. Id. at 468.

[FN8]. Id. at 469.

[FN9]. Id. at 470.

[FN10]. Id. at 469. See also Keith W. Lapeze, Comment, Recovery for Increased Risk of Disease in Louisiana. 58 La. L. Rev. 249, 258 (1977), and David P.C. Ashton, Decreasing Risks Inherent in Claims for Increased Risk of Future Disease, 43 U. Miami L. Rev. 1081, 1087-1088 (1989) (discussing difficulties of proof in matters happening in the distant past).

[FN11]. Love, supra note 5, at 818.

[FN12]. Richard Delgado, Beyond Sindell: Relaxation of Cause-In-Fact Rules for Indeterminate Plaintiffs, 70 Calif. L. REV. 881, 907 (1982).

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[FN13]. Barton Legum, Note, Increased Risk of cancer as an Actionable Injury, 18 Ga. L. Rev. 563, 795-96 (1984).

[FN14]. Cf. Ortiz v. Fibreboard, 527 U.S. 815 (1999) (stating that the Court for purposes of due process denied plaintiff's cause of action. Ortiz, inferentially overturns a cause of action based on the defendant's market-share liability however, this is unrelated to proving cause-in-fact through risk assessment). See also infra notes 175-177 and accompanying text.

[FN15]. Andrew R. Klein, A Model for Enhanced Risk Recovery in Tort, 56 Wash. & Lee L. Rev. 1173, 1196-97 (1999).

[FN16]. Id. at 1196-97.

[FN17]. Id.

[FN18]. Id.

[FN19]. Love, supra note 5, at 803.

[FN20]. Id. at 803.

[FN21]. Vern R. Walker, The Concept of Baseline Risk in Toxic Tort Litigation, 80 Ky. L.J. 631, 667 (1992).

[FN22]. See infra notes 245-53 and accompanying text.

[FN23]. Walker, supra note 21, at 646.

[FN24]. Jean Macchiaroli Eggen, Toxic Reproductive and Genetic Hazards in the Workplace Challenging the Myths of the Tort and Workers' Compensation Systems, 60 Fordham L. Rev. 843, 912 (1992) (quoting United States Congress, Office of Technology Assessment, Reproductive Health Hazards in the work place 3 (1985)).

[FN25]. Walker, supra note 21, at 665.

[FN26]. See infra notes 245-53 and accompanying text.

[FN27]. Risk is the probability of deleterious health or environmental effects. 57 Fed. Reg. 22, 888 (May 29, 1992) (see Glossary). See also EPA Proposed Guidelines for Carcinogen Risk Assessment (Apr. 1996) at http://www.epa.gov/ordntrnt/ORD/WebPubs/carcinogen/carcin.pdf.; Legum, supra note 11, at 575.

[FN28]. Not intended as an all-inclusive list.

[FN29]. Love, supra note 5, at 150.

[FN30]. Id.

[FN31]. See Shirley Duffy, "Cancerphobia" and Risk Assessment, 72 PA. Bar Assn. Quarterly 14 (2001).

[FN32]. Thompson, supra note 6, at 473.

[FN33]. Id. at 473.

[FN34]. Id.

[FN35]. Shirley K. Duffy, Medical Monitoring Damages OK'd,. J. Allegheny Co. Bar. Assn. Vol 1. No. 11. 3 at 12-13 (1999).

[FN36]. David Rosenberg, Symposium: The Institute of Judicial Administration Research Conference on Class Actions: Aggregation and Individual Justice and Collectivizing Risk-Based Claims in Mass-Exposure Cases, 71 Nyu L. Rrv. 210,

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218-19 (1996).

[FN37]. See Love, supra note 5, at 58 and Legum, supra note 13, at 592.

[FN38]. See infra notes 90-175 and accompanying text.

[FN39]. Ashton, supra note 10, at 1087.

[FN40]. Id. at 1087.

[FN41]. Id.

[FN42]. Schwegel v. Goldberg, 228 A.2d 405 (1967).

[FN43]. Thompson, supra note 6, at 472.

[FN44]. Ashton, supra note 10, at 1088.

[FN45]. Id.

[FN46]. Various diseases generally referred to in the popular media as "cancer."

[FN47]. Increased risk of future cancer is only being considered in this article. Increased risk claims can also arise with developmental and reproductive, neurological and generalized toxic effects. The legal analysis is the same for all of the aforementioned effects; however, risk assessment methodologies differ and will be considered in forthcoming articles. Carcinogen risk assessment only is considered in this article.

[FN48]. The organization of this section was derived from Joseph C.K. Kearfott and D. Alan Rudlin. ALI/ABA Environmental and Toxic Tort Matters: Advanced Civil Litigation. Case Management and Health Claims in Toxic Tort Litigation. (Jan., 2000).

[FN49]. See notes 51-56 and accompanying text.

[FN50]. See notes 57-61 and accompanying text.

[FN51]. John C. Cummings, Comment, How Far Should Increased Risk Recovery Be Carried in the Context of Exposure to Hazardous Substances, 76 Ky. L.J. 459, 462 n. 28 (1987) (arguing that there is a distinction between the "reasonable certainty" and "reasonable probability" standards; with the former closely approximating the standard of proof required in a criminal action and the latter only requiring a preponderance of the evidence). See notes 61-100 and accompanying text.

[FN52]. 525 A.2d 287, 297 (N.J. 1987).

[FN53]. Id. at 297-98.

[FN54]. Id. at 303.

[FN55]. Id. at 717. See also Mauro v. Raymark, 116 N.J. 126, 132 (N.J. 1989) (The Court applied the Ayers rule to private entity defendants in asbestos cases.)

[FN56]. 514 F. Supp. 1031 (E.D. Pa. 1981).

[FN57]. Bennett v. Mallinckrodt, Inc, 698 S.W.2d 854 (Mo. Ct. App. 1985).

[FN58]. Bennett, 698 S.W.2d at 869. Bennett could also be classified as a "reasonable certainty" case.

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[FN59]. 677 F. Supp. 424, 426 (D.S.C. 1987) (citing Waffen v. U.S. Dept. of Health & Serv., 799 F.2d 911 (4th Cir. 1986)).

[FN60]. Waffen, at 923.

[FN61]. Id. at 426-27.

[FN62]. Id. at 427.

[FN63]. 660 F. Supp. 1516 (W.D. Mich. 1987).

[FN64]. Id. at 1519-20. Exposure to a level of 12 ppm resulted in an increased risk of .186% to .00000006%.

[FN65]. Id. at 1521.

[FN66]. Id. at 1523.

[FN67]. Id. at 1524-25.

[FN68]. Id. at 1524.

[FN69]. Id. at 1525.

[FN70]. 684 F.2d 111, 120 (D.C. Cir. 1982).

[FN71]. Id. at 119.

[FN72]. 394 N.E.2d 1369 (Ill. App. Ct. 1979).

[FN73]. Id. at 1376.

[FN74]. 628 F. Supp. 1219 (Ma. Dist. Ct. 1986).

[FN75]. Id. at 1222.

[FN76]. Id. at 1231.

[FN77]. Id.

[FN78]. Id. at 1232.

[FN79]. Id.

[FN80]. 788 F.2d 315 (5th Cir. 1986), overruled and modified en banc by 797 F.2d 256 on an issue not pertinent to present discussion.

[FN81]. Id. at 319.

[FN82]. 855 F.2d 1188 (6th Cir. 1987).

[FN83]. Id. at 1204.

[FN84]. Id. at 1205.

[FN85]. 785 F.2d 79 (3d Cir. 1986).

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[FN86]. Id. at 81.

[FN87]. Id.

[FN88]. Coll v. Sherry, 148 A.2d 481, 486 (N.J. 1959).

[FN89]. Lorenc v.Chemirad Corp., 179 A.2d 401, 411 (N.J. 1962).

[FN90]. 765 F.2d 456, 466 (5th Cir. 1985).

[FN91]. Id. at 466.

[FN92]. Id. at 466-467.

[FN93]. Id. at 467.

[FN94]. 783 F.2d 589 (5th Cir. 1986).

[FN95]. Id at 591.

[FN96]. Id.

[FN97]. Id.

[FN98]. Id. at 592.

[FN99]. 761 F.2d 1129 (5th Cir. 1985).

[FN100]. 781 F.2d 394 (5th Cir. 1986), overruled on another point, see Centennial Ins. Co. v. Ryder, 149 F.3d 378, n.10 (1998).

[FN101]. One may argue that most of the cases cited herein indicated that persons with asbestosis have a less than 50% probability of getting cancer, and, that, inferentially, this expert's opinion is bogus. Presumably this expert reviewed plaintiff's medical history, X-rays, etc. in coming to this conclusion and based upon these considerations could more appropriately give an estimate on an individual basis.

[FN102]. Miller-Keane Medical Dictionary (2000) (defining asbestosis as "lung disease caused by inhalation of asbestos fibers, characterized by interstitial fibrosis associated with mesothelioma and brochogenic carcinoma), available at http://webmd.com.

[FN103]. See Starlings v. Ski Roundtop Corp., 493 F. Supp. 507 (M.D. Pa. 1980) (unquantified increased risk of arthritis with present degenerative changes in the knee); McCall v. United States, 206 F. Supp. 421 (E.D. Va. 1962) (3-25% chance of developing epilepsy with severe brain injury); Lindsay v. Appleby, 91 Ill. App. 3d 705, 414 N.E.2d 885 (1980) (unquantified increased risk of seizures and head trauma); Schwegel v. Goldberg, 209 Pa. Super. 280, 228 A.2d 405 (1967) (one in twenty chance of seizure disorder with traumatic head injury that left the brain scarred); Feist v. Sears, 267 Ore. 402, 517 P.2d 675 (1973) (unquantified increased susceptibility of getting meningitis with basal skull fracture). The Court in one of these cases (Schwegel) found expert testimony drawing from a database of similar injuries after such accidents particularly persuasive.

[FN104]. Brafford v. Susquehanna Corp., 586 F. Supp. 14 (Co. Dist. Ct. 1984).

[FN105]. Id.

[FN106]. Id. at 15.

[FN107]. Id.

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[FN108]. Id. at 18.

[FN109]. Mauro v. Raymark, 116 N.J. 126 (1989).

[FN110]. Diane Schmauder, An Analysis of New Jersey's Increased Risk Doctrine, 25 Rutgers L.J. 893 (1994).

[FN111]. Mauro, 116 N.J. at 141-42.

[FN112]. Id. at 142.

[FN113]. Id.

[FN114]. Id. at 151.

[FN115]. Id.

[FN116]. Mauro, 116 N.J. at 151.

[FN117]. Schmauder, supra note 111, at 921.

[FN118]. Id. at 922.

[FN119]. Id.

[FN120]. Id. at 924-25.

[FN121]. Id. at 926.

[FN122]. Anderson v. W.R. Grace, 628 F. Supp. 1219 (Ma. Dist. Ct. 1986).

[FN123]. Id. at 1232.

[FN124]. Id.

[FN125]. Id.

[FN126]. Id.

[FN127]. Klein, supra note 15, at 1511.

[FN128]. Id. at 1201.

[FN129]. See supra notes 102-07 and accompanying text.

[FN130]. Klein, supra note 15, at 1511.

[FN131]. Not intended as an all-inclusive list.

[FN132]. Legum, supra note 13, at 576-85.

[FN133]. Id. at 576.

[FN134]. Id.

[FN135]. Id. at 577.

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[FN136]. Id. at 584.

[FN137]. Id. at 585.

[FN138]. Legum, supra note 13, at 582.

[FN139]. Note, Latent Harms and Risk-Based Damages, 111 Harv. L. Rev. 1505, 1519 (1998).

[FN140]. Thompson, supra note 6, at 476.

[FN141]. Id.

[FN142]. Id.

[FN143]. Id. at 477.

[FN144]. Schmauder, supra note 111 at 921-22.

[FN145]. Love, supra note 5; Legum, supra note 13.

[FN146]. Id. at 588.

[FN147]. Legum, supra note 13, at 588 n. 98.

[FN148]. Love, supra note 5, at 796.

[FN149]. Klein, supra note 15, at 1194.

[FN150]. Id. at 1198.

[FN151]. Ashton, supra note 10 at 1119-39.

[FN152]. Id. at 1113. This approach is consistent with U.S. EPA 1996, supra note 26, at 110.

[FN153]. See notes 102-07 and accompanying text.

[FN154]. Brent Carson, Comment, Increased Risk of Disease from Hazardous Waste: A Proposal for Judicial Relief, 60 Wash. L. Rev. 635, 637 (1985).

[FN155]. Id. at 650.

[FN156]. Id.

[FN157]. Id.

[FN158]. Id. at n. 85.

[FN159]. Rosenberg, supra note 36, at 219.

[FN160]. Id. at 219.

[FN161]. See notes 173-75 and accompanying text.

[FN162]. Rosenberg, supra note 36, at 246.

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[FN163]. Id. at 1521-22.

[FN164]. See Note, Latent Harms, supra note 139, at 1521.

[FN165]. Id. at 1521.

[FN166]. Id.

[FN167]. Id. at 1521.

[FN168]. Id.

[FN169]. This is not necessarily so, if a careful meta-analysis is performed (Meta-analysis is the combination of many trial results; a method designed to increase the reliability of research by combining and analyzing the results of all known trials of the same product or experiment on the same subject) at http:// encarta.msn.com/encnet/features/dictionary/DictionaryResults.aspx? refid=561537082.

[FN170]. See Note, Latent Harms, supra note 139, at 1518-19.

[FN171]. Id.

[FN172]. Id. at 1519.

[FN173]. Love, supra note 5, at 811.

[FN174]. Id.

[FN175]. Rosenberg, supra note 36, at 237.

[FN176]. Id.

[FN177]. Id. at 234-35. However, these damages could be used to register, track and insert data on claimants into a database for future actions. See Duffy, supra note 35, at 12.

[FN178]. Estimating the probability that contact with a chemical will harm people now and in the future at http:// www.epa.gov/superfund/programs/risk/commeng.htm (Dec. 14, 2002).

[FN179]. U.S. EPA. Risk Assessment Guidelines for Superfund, Vol. 1. Human Health Evaluation Manual (Part A). Interim Final. Office of Emergency and Remedial Response, Wash. D.C. EPA#<<backslash>>540<<backslash>>1-89<< backslash>>002.

[FN180]. U.S. EPA. Guidelines for Carcinogen Risk Assessment. 51 Fed. Reg. 33992 (Sept. 24, 1986).

[FN181]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17960, available at http://www.epa.gov/ordntrnt/ORD/WebPubs/Carcinogen.

[FN182]. See U.S. EPA. Lay Description of the Linearized Multistage Model, available at http://www.epa.gov/otaq/regs/toxics/airtoxf.pdf. In theory other models to estimate extra cancer risk may be used, but this model was most commonly used prior to the 1996 Proposed Guidelines for carcinogen risk assessment.

[FN183]. 1986 Guidelines, supra note 180.

[FN184]. See U.S. EPA RAGS, supra note 179, at 8-7.

[FN185]. This article primarily reviews the guidance for cancer risk assessment, which is in a state of constant flux. It does

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not examine in detail changes and amendments to the guidance that have taken place. These are to be evaluated in an updated article for the Fall 2003 Edition.

[FN186]. 1996 Proposed Guidelines, supra note 181, at 5.

[FN187]. Id. at 5-6.

[FN188]. Id. at 83.

[FN189]. Id. at 84.

[FN190]. Id.

[FN191]. Id. at 84.

[FN192]. U.S. EPA. Guidelines for Carcinogen Risk Assessment. 51 Fed. Reg. 33992 (Sept. 24, 1986).

[FN193]. Id. at 84.

[FN194]. Id. at 85.

[FN195]. Id. at 86.

[FN196]. Id. at 86-104 and Appendix A.

[FN197]. Id. at 110.

[FN198]. U.S. EPA. Guidelines for Carcinogen Risk Assessment. 51 Fed. Reg. 33992 (Sept. 24, 1986).

[FN199]. Id. at 7 and 11. See p. 111 for examples of these.

[FN200]. Id. at 111.

[FN201]. Id .at 8 and 113-18.

[FN202]. Id. at 113.

[FN203]. U.S. EPA. Guidelines for Carcinogen Risk Assessment. 51 Fed. Reg. 33992 at 67 and 113 (Sept. 24, 1986).

[FN204]. 1,2,4-Trichlorobenzene demonstrates a carcinogenic response as secondary to a toxic response. The mouse liver tumors observed in experiments using 1,2,4-trichlorobenzene may be caused by a secondary mechanism due to liver toxicity, available at http://www.epa.gov/chemfact/tcben-sd.txt.

[FN205]. Id. at 32; IARC Monographs Vol. 73 Atrazine (2003). For example, mammary tumors associated with exposure to atrazine may involve a non- reactive, hormonally mediated mechanism.

[FN206]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17960, 17980 (Apr. 23, 1996) (providing an example of a tumor response that is secondary to a toxic response).

[FN207]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17961; Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17993.

[FN208]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17993.

[FN209]. To illustrate the concepts of dose-response curve, LED10., experimental (observed) and environmental (human)

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exposures, see Figure 3-1 of the Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17995, available at http://www.epa.gov/ordnt/ORD/WebPubs/carcinogen.

[FN210]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17960; 17993 (Apr. 23, 1996).

[FN211]. "A '95% confidence interval' is a range of hypothesis values that should not be rejected on the basis of sample results, provided one is prepared to be wrong in as many as 5% of the cases" (i.e. one is willing to accept that perhaps one time in twenty the confidence interval would not contain the true value). Walker, supra note 21, at 659.

[FN212]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17960, 17967 (Apr. 23, 1996); Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17993; Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17995.

[FN213]. See Figure 3-1 of the 1996 Proposed Guidelines.

[FN214]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17962; Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17993.

[FN215]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17962.

[FN216]. Id.

[FN217]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17960, 17967 (Apr. 23, 1996).

[FN218]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17993; See Fig. 3-1 of the 1996 Proposed Guidelines for Carcinogen Risk Assessment. Available at http:// www.epa.gov/ordntrnt/ORD/WebPubs/carcinogen/FIG-31.TIF

[FN219]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. at 17996.

[FN220]. Id.

[FN221]. Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17960, 17967 (Apr. 23, 1996).

[FN222]. See supra notes 227-39 and accompanying text.

[FN223]. See supra note 213, at Fig, 3-1.

[FN224]. Id. at 8.

[FN225]. Id. at 31-32.

[FN226]. Id. at 114.

[FN227]. Guidelines for Exposure Assessment, 57 Fed. Reg. 22888 (May 29, 1992).

[FN228]. Exposure is the contact of a chemical, physical or biological agent with the outer boundary of an organism. Id. Glossary.

[FN229]. Id. at § 2.2 and 2.2.1.

[FN230]. Encarta World English Dictionary (2002). Concentration is the" amount of a substance dissolved in another." Also available at Encarta.http:// msn.com.

[FN231]. Contact time is the actual time periods (events or episodes) during which actual exposure occurs. Contrast exposure

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duration, which is a time interval of interest for assessment purposes during which exposure occurs, either continuously or intermittently. In the case of intermittent exposure contact time would be zero during some defined units of time. Guidelines for Exposure Assessment, 57 Fed. Reg. 22888 (May 29, 1992).

[FN232]. Id. at § 2.2 and 2.2.3.

[FN233]. For example, medium of concern at a hazardous waste site might be surface soils, groundwater, surface water, etc.

[FN234]. For example, at a hazardous waste site, field work (the actual gathering of samples of the medium of concern) may occur months before a risk assessor in the home office refines the final exposure scenarios for a site.

[FN235]. Guidelines for Exposure Assessment, 57 Fed. Reg. 22888 (May 29, 1992) at § 2.2 and 2.2.3.

[FN236]. An illustration of a biomarker would be a chemical that reacts or binds to the hemoglobin molecule found in blood. This binding changes the chemical composition of the molecule and perhaps its shape and the hemoglobin- chemical complex can be measured in the blood, id., at § 2.2.3.

[FN237]. Body burden is the amount of a particular chemical stored in the body at a particular time, especially a potentially toxic chemical, as a result of exposure. Body burden can be the result of long-term or short-term storage. For example the amount of metal in bone, the amount of a lipophilic substance such as PCBs in adipose tissue, or an amount of carbon monoxide (as carboxyhemoglobin) in blood. Id. at Glossary.

[FN238]. For example, an amount of a chemical or its metabolite in urine.

[FN239]. "Dose" is the amount of a substance available for interaction with metabolic processes or biologically significant receptors after crossing the outer boundary of an organism. Guidelines for Exposure Assessment, 57 Fed. Reg. 22888 (May 29, 1992), Glossary.

[FN240]. Id. at § 2.3.1.

[FN241]. A high end exposure estimate is a plausible estimate of the individual exposure for those persons at the upper end of the exposure distribution. Id. at § 2.3.1.

[FN242]. The central tendency is an average or median exposure or dose. Id. at § 2.3.1.

[FN243]. Id. at § 2.3.2

[FN244]. Risk is the probability of a deleterious health or environmental effect. Id. at Glossary

[FN245]. Id. at 665.

[FN246]. Id.

[FN247]. Adams v. Johns-Manville, 783 F.2d 589 (5th Cir. 1986), supra note 95-99, and accompanying text. This is not to say that some of these cases may not be successful or have settlement value; they just are not easy cases, providing minimal problems for plaintiff's counsel.

[FN248]. At least in theory, the linearized multistage model, calculates extra lifetime risk of cancer, because the transition rate of a cell line to cancer is taken into account in the model. See Lay Description, supra note 182, at F-3. Presumably, the same results would be obtained under the new guidelines, because the linear default provides similar results.

[FN249]. International Agency for Research on Cancer Monographs, Suppl. 7 Asbestos. at http://www.iarc/htdocs/monographs/Suppl.7/asbestos.html.

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[FN250]. Integrated Risk Information Systems, at http:// www.epa.gov/iris/index.html.

[FN251]. National Institute of Occupational Safety and Health, at http:// www.cdc.gov/niosh/homepage.html (last visited May 2003).

[FN252]. American Conference of Governmental Industrial Hygienists, at http://acgih.org/home.htm (last visited May 2003).

[FN253]. National Toxicology Program, at http://ntp-server.niehs.nih.gov (last visited May 2003).

[FN254]. Agency for Toxic Substances and Disease Registry, at http:// www.atsdr.cdc.gov (last visited May 2003).

[FN255]. International Agency for Research on Cancer, at http//www.iarc.fr (last visited May 2003).

[FN256]. International Agency for Research on Cancer Monographs. Suppl. 7 Asbestos, available at http://www.iarc.htdocs/monographs/suppl7/asbestos.html (last visited May 2003).

[FN257]. Id. at 3.

[FN258]. Clayton P. Gillette and James E. Krier, Risk, Courts and Agencies, 138 U. Pa. L. Rev. 1027, 1065-67 (1990).

[FN259]. Id. at 1068.

[FN260]. Id.

[FN261]. Id. This concept is also referred to as 'agency capture'. As a check on agency capture, the foreign scientific literature and the International Agency for Research on Cancer should be consulted. Some scientists and courts look at the foreign literature with disdain. Counsel's expert, however, should be able to evaluate the foreign literature and discern the quality studies from poorly conducted ones and be able to argue why the study is supportive and why it is a quality study.

[FN262]. Gillette and Krier, supra note 258, at 1073.

[FN263]. Id. at 1073.

[FN264]. Id.

[FN265]. Id. at n. 126.

[FN266]. Id. at n. 157.

[FN267]. Id. at 1088-89.

[FN268]. Id. at 1088-99.

[FN269]. Id. at n. 229.

[FN270]. John S. Applegate, Risk Symposium: A Beginning and not an End in Itself: Role of Risk Assessment in Environmental Decision Making, 63 U. Cin. L. Rev. 1643 (1995).

[FN271]. Id. at 1658.

[FN272]. The high, medium and low indicators in EPA's IRIS, supra note 250, indicates confidence in the study from which the risk estimate was derived, the database and the estimate.

[FN273]. Applegate, supra note 270, at 1659.

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[FN274]. Id. at 1661-62.

[FN275]. Id. at 1662-63.

[FN276]. Id. at 1663.

[FN277]. Id. at 1667.

[FN278]. Id. at 1668.

[FN279]. The weight-of-evidence determination along with the quantitative risk estimate should be used as probative evidence in deciding these claims. Agents that are Class A (old guidelines) or "known/likely" under the 1996 guidelines, should be awarded damages. The risk estimates can be used as a method to calculate damages.

[FN280]. Benjamin Cardozo, The Nature Of The Judicial Process 151 (1921), cited in Wilson v. Johns-Manville, 684 F.2d 111 (D.C. 1982).

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