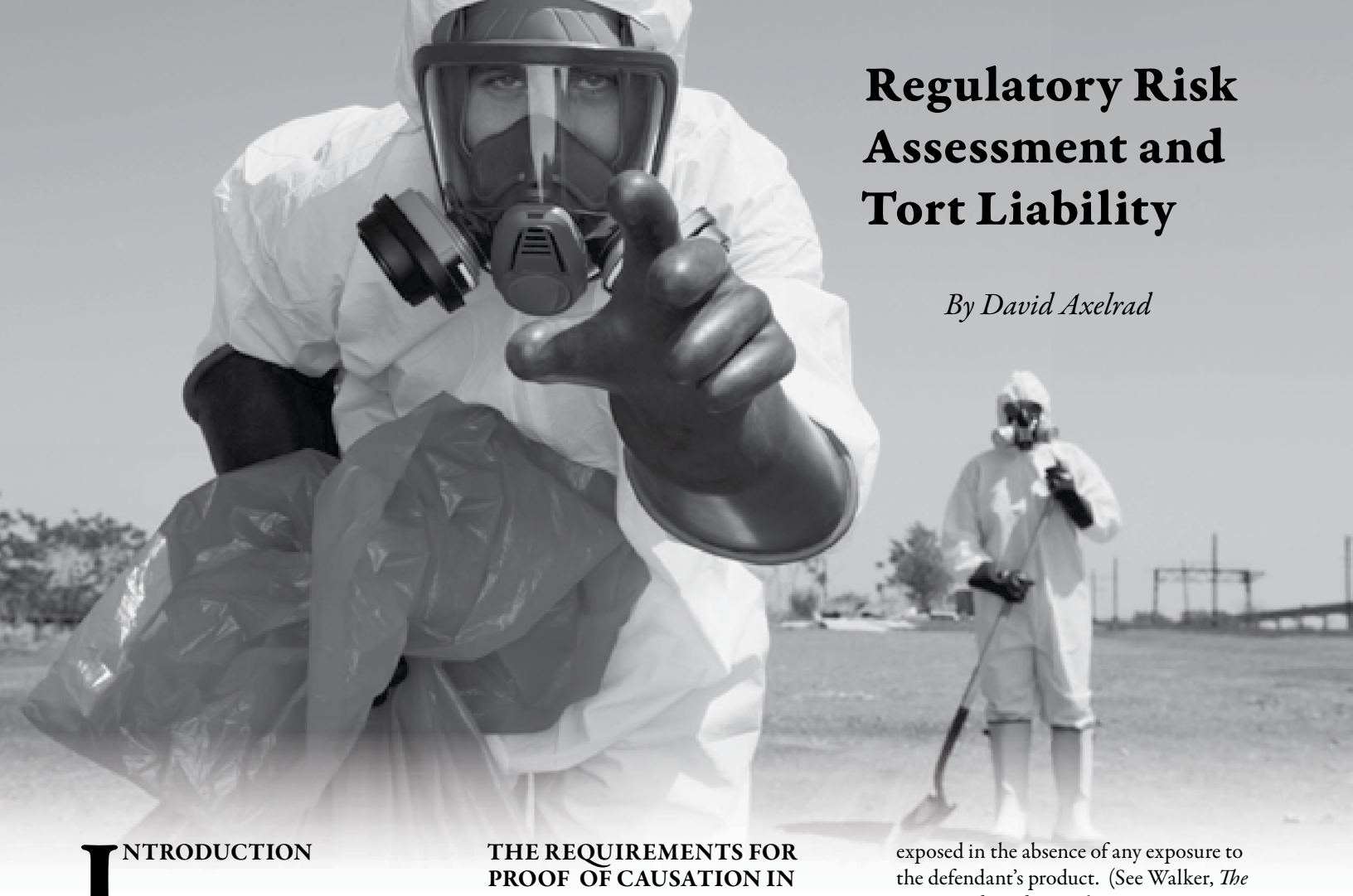

Regulatory Risk Assessment and Tort Liability

By David Axelrad

INTRODUCTION

Regulatory agencies often confront uncertainty or lack of data concerning the causal relationship between exposure to a particular chemical substance and a particular effect on human health. In these situations, regulators use risk assessment to estimate the extent to which exposure to a chemical will increase the incidence of a particular health effect. (See Reference Manual on Scientific Evidence (Third Edition 2011) p. 649; McGarity, On the Prospect of "Daubertizing" Judicial Review of Risk Assessment, 66 Law and Contemporary Problems 155, 157 (2003).)

In controversial areas of toxic torts, where the issue of dose, i.e., "how much is enough" to cause an alleged harm is disputed, plaintiffs frequently turn to regulatory risk assessment standards to fill in the evidentiary gap created by a lack of definitive science on the relationship between exposure to a particular product and the plaintiff's alleged injury. As explained below, these risk assessment standards are not designed and therefore should not be used to measure causal relationships for purposes of assigning tort liability.

THE REQUIREMENTS FOR PROOF OF CAUSATION IN TOXIC TORT CASES

Tort law assigns responsibility for harm to persons or property upon proof that the defendant's breach of a duty of care owed to the plaintiff was a substantial factor in causing harm. (See, e.g., *Weirum v. RKO General, Inc.* (1975) 15 Cal.3d 40, 46 ["The determination of duty . . . is the court's 'expression of the sum total of those considerations of policy which lead the law to say that the particular plaintiff is entitled to protection' "]; *Viner v. Sweet* (2003) 30 Cal.4th 1232, 1239 ["jury instructions on causation in negligence cases should use the 'substantial factor' test [which] subsumes the 'but for' test...."].

In the area of toxic and environmental torts, the law imposes rigorous requirements for proof of causation because of the scientific uncertainties associated with the consequences of human exposure to various chemical substances. Thus, to be held responsible in a toxic tort case, exposure to the defendant's product must have increased the risk of a particular harm above the baseline risk to which everyone is exposed in the absence of any exposure to the defendant's product. (See Walker, *The Concept of Baseline Risk in Tort Litigation* (1991) 80 Ky. L. J., 645-646, 673) ["[I]njuries resulting from the normal risks of life are not compensable because they are part of the danger inherent in living in society. 'Baseline risk' ... [is] the risk of occurrence of the plaintiff's injury or accident in the same or similar circumstances, *but* in the *absence* of any act of the defendant that in fact created an additional, unreasonable risk of the injury or accident.' ... Baseline risk is the floor or threshold risk, above which a defendant must have created an incremental risk in order to be found negligent."]

To satisfy this burden of proof, a toxic tort plaintiff must prove both general and specific causation. (E.g., *In re Hanford Nuclear Reservation Litigation* (9th Cir. 2002) 292 F.3d 1124, 1134 ["In order to prevail on their [toxic tort] claims, ... plaintiffs must establish both generic *and* individual causation" (original emphasis)]; see Bernstein, *Getting to Causation in Toxic Tort Cases* (2008) 74 Brooklyn L.Rev. 51, 52 ["American courts have reached a broad consensus on what a

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plaintiff must show to prove causation in a toxic tort case. First, a plaintiff must show that the substance in question is capable of causing the injury in question. This is known as 'general causation.' Second, a plaintiff must show that this substance caused *his* injury. This is known as 'specific causation. [Fn. omitted.]'"].)]

Proof of "general causation" establishes as a threshold matter that a particular chemical is capable of causing in humans the type of harm suffered by the plaintiff. (E.g., *In re Hanford Nuclear Reservation Litigation, supra,* 292 F.3d 1124 at 1133 ["General ... causation has been defined by courts to mean whether the substance at issue had the capacity to cause the harm alleged"].) If, for example, exposure to Chemical A can only cause headache in humans and plaintiff is complaining about skin rash there is no general causation and plaintiff's claim fails.

If a substance does have the capacity to cause the harm plaintiff claims to have suffered, then the plaintiff must prove "specific causation" by establishing a reasonable medical probability that plaintiff's actual exposure to the chemical in question was a substantial factor in causing this particular plaintiff's harm. (E.g., *In re Hanford Nuclear Reservation Litigation, supra,* 292 F.3d at 1133 ["'individual causation' refers to whether a particular individual suffers from a particular ailment as a result of exposure to a substance"]; *Bonner v ISP Technologies* (8th Cir. 2001) 259 F.3d 924, 928 ["the plaintiff must put forth sufficient evidence ... that the product was capable of causing her injuries, *and that it did*" (emphasis added)]; *Parker v. Mobil Oil Corp.* (N.Y.Ct. App. 2006) 7 N.Y. 3d 434, 448 [857 N.E.2d 1114] ["It is well-established that an opinion on causation should set forth a plaintiff's exposure to a toxin, that the toxin is capable of causing the particular illness (general causation) and that plaintiff was exposed to sufficient levels of the toxin to cause the illness (specific causation)"].) The key to proof of specific causation is dose, evidence that the plaintiff was exposed to the chemical at issue in sufficient quantity to produce the harm that particular chemical is capable of producing. (See, e.g., *In re Bextra and Celebrex Marketing Sales Practices and Product Liability Litigation* (N.D. Cal. 2007) 524 F.Supp.2d 1166, 1174 ["'all chemical agents are intrinsically hazardous-whether they cause harm is only a question of dose....'"]; *McClain v. Metabolife Intern., Inc.* (11th Cir. 2005) 401 F.3d 1233, 1242 ["'Dose is the single most important factor to consider in evaluating whether an alleged exposure caused a specific adverse effect'"].)

THE DISCONNECT BETWEEN CAUSATION REQUIREMENTS AND REGULATORY RISK ASSESSMENT STANDARDS

Exacting causation standards in toxic tort law ensure that only those specific persons whose conduct or products were a substantial factor in causing harm to a particular person will be held legally responsible to compensate the person harmed. In contrast, regulatory risk assessment standards are not meant to govern the legal relationships and responsibilities between particular plaintiffs and defendants. Instead, regulatory risk assessment standards are, as noted above, adopted to protect public health where there is uncertainty or lack of data concerning the relationship between exposure to a chemical and a particular health effect. (See Latin, *Good Science, Bad Regulation and Toxic Risk Assessments*, Yale J. on Reg. 89, 91-92 (1988) ["Toxic risk assessment suffers from fundamental uncertainties about causal mechanisms for cancer and other hazards.... These uncertainties generally preclude reliable assessments of relevant effects, and there is no scientific consensus on how they should be resolved.... [¶] Under current regulatory practices, Agency scientists produce risk assessments that seldom approach the level of reliability normally expected of scientific findings; indeed, many estimates are little more than educated guesses. [Footnote omitted]...."].) The process by which regulatory risk assessment standards are adopted illustrates the disconnect between such standards and the case-specific standards for proof of causation in a tort case.

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THE REGULATORY RISK ASSESSMENT PROCESS: ILL-SUITED TO PROOF OF CAUSATION

HAZARD IDENTIFICATION

There are four steps in regulatory risk assessment – "(1) hazard identification, (2) dose-response assessment, (3) exposure assessment and (4) risk characterization." (Donald W. Stever, *The Use of Risk Assessment in Environmental Law*, 14 Colum. J. Envtl. L. 329 (1989).) The first step, *identification of the hazard*, is roughly analogous to the general causation inquiry in tort litigation, i.e., can a particular chemical cause an adverse health effect? (See McGarity, *supra*, n. 7 at pp. 157-158.) Because there is little or no data concerning effects on humans (and hence the perceived need for a regulatory risk assessment), this inquiry is often based on an extrapolation from the results of animal studies to the supposed risks to humans. (See Reference Manual on Scientific Evidence (Third Edition 2011) pp. 563, 636, 644; Endicott, Interaction Between Regulatory and Tort Law in Controlling Toxic Chemical Exposure, 47 SMU L. Rev. 501, 504 (1994).)

Extrapolating from animal studies, while perhaps acceptable in the conservative prevention environment of regulatory risk assessment, is notoriously problematic when used as a foundation for proof of causation in a tort action. "Animal studies have two significant disadvantages.... First, animal study results must be extrapolated to another species – human beings – and differences in absorption, metabolism, and other factors may result in interspecies variation in responses." (Reference Manual on Scientific Evidence, *supra*, at p. 563 .) Second, animal studies typically use much higher doses than the doses to which humans are exposed, which makes it necessary to consider "the dose-response relationship and whether a threshold no-effect dose exists." (*Ibid.*) "Those matters are almost always fraught with considerable, and currently, unresolvable, uncertainty." (*Ibid;* see EPA, "Guidelines for Carcinogen Risk Assessment" (1986) at pp. 13-14 ["Low-dose risk estimates derived from laboratory animal data extrapolated to humans are complicated by a variety of factors that differ among species and potentially affect the response to carcinogens. Included among these factors are differences between humans and experimental test animals with respect to life span, body size, genetic variability, population homogeneity, existence of concurrent disease, pharmacokinetic effects such as metabolism and excretion patterns, and the exposure regimen"]; *Lynch v. Merrell-National Laboratories* (1st Cir. 1987) 830 F.2d 1190, 1194 [animal studies "do not have the capability of proving causation in human beings in the absence of any confirmatory epidemiological data"].)

DOSE RESPONSE ASSESSMENT

The second step is a *dose response assessment* involving a determination, for risk assessment purposes, of the dosage level required to produce a particular health effect in humans. It is here that risk assessment is at its most cautious. Because the goal of risk assessment is protection of public health where there is a lack of causation evidence, risk assessors make unsupported conservative assumptions that tend to overestimate the actual risk of harm. "'[R]isk assessors may pay heed to any evidence that points to a need for caution, rather than assess the likelihood that a causal relationship in a specific case is more likely than not' '...." (*McLain v. Metabolife International, Inc.*, *supra*, 401 F.3d 1233 at 1249; see Latin, *supra,* at pp. 91-92, 94 ["Risk assessors often respond to scientific uncertainties by adopting conservative safety-oriented positions on some important issues while they use best-current-scientific-guess, middle-of-the-range, methodological-convenience, or least-cost treatments on other material issues"]; Endicott, *Interaction Between Regulatory Law and Tort Law in Controlling Toxic Chemical Exposure*, 47 SMU L.Rev. 501, 504-505 (1994) ["Generally, risk assessors, ... consciously seek to err on the side of standards that will be more, not less, protective of human health. This is a laudable goal, but the net

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result can be a risk estimate that varies from the actual risk by many orders of magnitude"]; Shapiro, *Politicization of Risk Assessment*, 37 Environmental Law 1083, 1089 ["The mandate of agencies to act on the basis of anticipated harm makes scientific uncertainty an unavoidable aspect of regulatory science...."].)

In short, risk assessors will utilize the most sensitive data sets and the most conservative assumptions in order to achieve the goal of protecting the public against all potential health effects, rather than determining the risk of harm to any actual person under a particular set of facts. (See *Baker v. Chevron USA, Inc.* (S.D. Ohio 2010) 680 F.Supp.2d 865, 880 [" '[R]egulatory levels are of substantial value to public health agencies charged with ensuring the protection of public health, but are of limited value in judging whether a particular exposure was a substantial contributing factor to a particular individuals' disease or illness' ... This is because regulatory agencies are charged with protecting public health and thus reasonably employ a lower threshold of proof in promulgating their regulations than is used in tort cases"]; *Sutera v. Perrier Group of America, Inc.* (D. Mass. 1997) 986 F.Supp. 655, 664 ["a regulatory standard, rather than being a measure of causation, is a public-health exposure level that an agency determines pursuant to statutory standards ... a regulator's purpose is to 'suggest or make prophylactic rules governing human exposure ... from the preventive perspective that agencies adopt in order to reduce public exposure to harmful substances' "]; see also Shapiro, *supra*.) As a result, " 'the procedures commonly used in 'risk assessment, ... are often ... of marginal relevance to estimating 'causation' in an individual—e.g., whether a particular chemical caused or contributed to a particular disease or illness in a given person." (Shapiro, *supra*.)

The process is also affected by the political and social policy bias of the government entity conducting the assessment. For example, the "acceptable" levels of exposure under the Carter and Reagan administrations were starkly different even though the government's knowledge of the risks of regulated chemicals did not materially change over that time. (See Latin, *Good Science, Bad Regulation, and Toxic Risk Assessments*, Yale J. on Reg. (1988) 89, 95-96 ["Under the Carter Administration, risks above one fatality per million exposed people were usually treated as 'unacceptable' if feasible control measures were available. Reagan Administration agencies have concluded that risks as high as one in ten thousand, or even one in a hundred in some settings, are tolerable. These risk-management decisions reflect different ideological preferences and different assumptions about the economic and political effects of toxic substances regulation. Similar considerations implicitly influence risk-assessment practices and resulting estimates of toxic hazards"]; see also Shapiro, *OMB and The Politicization of Risk Assessment*, Environmental Law, 37 Env. L. 1083, 1086 (2007) ["Administration officials at other agencies, however, have also asked or demanded that scientists change risk assessments because the results did not support policy outcomes preferred by the Administration."].)

The threshold levels of exposure used in setting regulatory risk assessment standards are often so low that virtually any exposure is considered significant. Substituting these conservative exposure levels for proof of causation in accordance with traditional tort principles undermines the predictability and fairness of tort law by creating the risk that persons whose conduct was not a substantial factor in causing a plaintiff's alleged harm nonetheless will be held responsible for the plaintiff's injury and required to pay damages. It is therefore not surprising that courts have repeatedly rejected the notion that there is "no safe level" of exposure to a chemical, and that evidence of exposure to any amount, however small, can establish causation. (See, e.g., *Parker v. Mobil Oil Corp.* (N.Y. App.Div. 2005) 793 N.Y.S.2d 434 [16 A.D.3d 648, 653], affd. (2006) 7 N.Y.3d 434 [857 N.E.2d 1114] ["[S]tating that any exposure to benzene is 'unsafe' is not tantamount to stating that any exposure to benzene causes [cancer]"]; *National Bank*

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of Commerce v. Associated Milk Producers (E.D.Ark. 1998) 22 F.Supp.2d 942, 966-967 [criticizing the "no threshold" dose theory of plaintiff's experts, and concluding that "[t]his flawed logic is no substitute for reliable scientific proof of causation"]; *Sutera v. Perrier Group of America Inc.* (D.Mass. 1997) 986 F.Supp. 655, 666 ["[T]here is no scientific evidence that the linear no-safe threshold analysis is an acceptable scientific technique used by experts in determining causation in an individual instance"]; *McClain, supra,* 401 F.3d at pp. 1242-1243 ["O'Donnell offers no opinion about the dose of Metabolife that caused ischemic strokes in three plaintiffs and a heart attack in the other. He only said that any amount of Metabolife is too much, which clearly contradicts the principles of reliable methodology ...."].)

EXPOSURE ASSESSMENT

The third step is an *exposure assessment*, involving analysis of the magnitude, frequency, duration and route of exposure to a chemical for a particular population. The bias in regulatory risk assessment favoring maximum protection of public health generally means that in assessing exposure, the greatest possible exposure for the longest period of time will be assumed to have occurred, regardless of the relationship between that assumption and any actual exposures. (See *Asbestos Information Ass'n/North America v. Occupational Safety and Health Admin.,* (5th Cir.1984) 727 F.2d 415, 425-426 (5th Cir.1984) ["[A]lthough risk assessment analysis is an extremely useful tool, ... the results of its application to a small slice of time are speculative because the underlying data-base projects only long-term risks. Epidemiologists generally study only the consequences of long-term exposure to asbestos"]; Rodricks, *Risk Assessment, the Environment, and Public Health*, Environmental Health Perspectives, Volume 102, Number 3, March 1994, p. 259, *www.ncbi.nlm.nih.gov/pmc/articles/PMC1567122/pdf/envhper00391-0015.pdf* [last visited July 9, 2012]; see also Fitzsimmons, et al., "When 'Likely' Does Not Mean 'More Likely Than Not': The Dangers of Allowing Government Chemical Classifications and Numeric Risk Assessments at Trial," <*www.toxictortlitigationblog.com/uploads/file/Fitzsimmons_Quadrino_Article%5B1%5D.pdf*> [last visited July 9, 2012].)

The assumption will also be that exposures are generic, i.e., that the level of exposure is the same across all populations, regardless of actual differences in exposure that may exist from one group to another. (See Fitzsimmons, et al., "When 'Likely' Does Not Mean 'More Likely Than Not': The Dangers of Allowing Government Chemical Classifications and Numeric Risk Assessments at Trial," *supra*; Rodricks, *Risk Assessment, the Environment, and Public Health*, Environmental Health Perspectives, *supra*.) In the courtroom, however, actual exposure, rather than assumed exposure, governs causation analysis. (See *Borg-Warner Corp. v. Flores* (Tex. 2007) 232 S.W.3d 765, 773 (*Borg-Warner*) ["Defendant-specific evidence relating to the approximate dose to which the plaintiff was exposed, coupled with evidence that the dose was a substantial factor in causing the asbestos-related disease, will suffice ... '[I] t is not adequate to simply establish that "some" exposure occurred.... [T]here must be reasonable evidence that the exposure was of sufficient magnitude to exceed the threshold before a likelihood of "causation" can be inferred'"].)

OVERALL RISK CHARACTERIZATION

The final step in the regulatory risk assessment analysis is an *overall risk characterization*. Here, because the risk assessment is dealing with inherent uncertainties, risk assessors make assumptions concerning *theoretical* lifetime risks, i.e., what might occur given the conservative assumptions adopted for purposes of protecting public health. (See *Asbestos Information Ass'n/North America v. Occupational Safety and Health Admin., supra*; Rodricks, *supra*, Fitzsimmons, *supra*.) The resulting "acceptable" risk assumes maximum levels of exposure (at which no regulatory action is required) that are often negligible or near zero. This assumption has no place in a courtroom where, as noted above, exposure must be causally related to the plaintiff's injury.

CONCLUSION

The end result of regulatory risk assessment is a picture of what might be possible but not what is probable, or even likely for any particular person or population under any particular set of factual circumstances, or in other words, a result which does not satisfy the requirements for proof of causation in a tort case.

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