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Making the Case for Causation in Toxic Tort Cases: Superfund Rules Don't Apply

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While causation is often a paramount obstacle to prosecuting a toxic tort claim, judicial interpretation of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)[1] generally has eliminated consideration of causation of actual injury or harm. Generally, under CERCLA's strict liability regime, no showing of causation of actual injury or harm at a site is required for liability to attach. This is important to understand, because too often parties presume that a potentially responsible party (PRP) under CERCLA also is liable to private parties under common-law tort theories, but this is more difficult than it seems. This Article compares the relative ease with which the government establishes liability of private parties under CERCLA to the more rigorous demonstration of causation that a plaintiff must make in a toxic tort case.

I. CERCLA—Causation

CERCLA was intended to address the damage presented at waste disposal sites where releases or threatened releases of contaminants had occurred and traditional negligence theories did not provide a basis for recovery against the parties responsible for placing the hazardous substances in the environment.[2] CERCLA imposes strict liability upon: (1) the current owner or operator of a facility from which there has been a release or threatened release of a hazardous substance; (2) any person who owned or operated a facility at the time the hazardous substances were disposed; (3) any person who contracted, agreed, or otherwise arranged for the disposal or treatment of a hazardous substance or arranged with a transporter for transport for disposal or treatment of a hazardous substance; and (4) any person who transported the hazardous substance to a facility from which there has been a release or threatened release.[3] The reasoning behind CERCLA strict liability is to shift the cost of the necessary environmental response from taxpayers to the parties who benefited from the intentional use of such sites.[4]

Establishing liability under CERCLA, therefore, often requires no more than a simple demonstration of ownership or operation history. To establish "arranger" liability, one often only has to show that an entity arranged for disposal or placement of a hazardous substance (such as for a generator of waste disposed at a dump site). Presently, there are approximately 800 specifically designated CERCLA hazardous substances identified in Table 302.4 in 40 C.F.R. §302.4, including synthetic and naturally occurring chemicals, such as copper, manganese, sodium, and zinc. Many common household items—including personal care products, furniture, flooring, and electronics, among others—contain hazardous substances, so this determination can be trivial. No demonstration of an unacceptable risk, injury, property damage, or other harm must be made to identify a PRP.

Once an entity is identified as a PRP, it can be compelled to investigate and possibly remediate a contaminated area or reimburse the government or third parties for past and future response costs.[5] The CERCLA cleanup process often begins with a procedure for identifying and ranking the hazards posed by contaminated sites. On the basis of that ranking system, CERCLA establishes a national priorities list that is intended to ensure that the most dangerous sites are remediated first. There are two types of cleanups recognized by CERCLA: (1) *removal* actions are short-term measures taken to minimize the dangers to human health and the environment on an emergency basis; whereas (2) *remedial* actions are long-term efforts that attempt to rid the site of dangers on a permanent basis.[6]

1. 42 U.S.C. §§9601-9675 (2007), ELR Stat. CERCLA §§101-405.
2. The U.S. Congress enacted CERCLA with the principal goal of addressing liability resulting from the need to clean up inactive hazardous waste disposal sites where hazardous substances have been released into the environment. *See* Pub. L. No. 96-510, 94 Stat. 2767 (1980); New York v. Shore Realty Corp., 759 F.2d 1032, 1040, 15 ELR 20358 (2d Cir. 1985).
3. 42 U.S.C. §9607(a).
4. Burlington N. & Santa Fe Ry. Co. v. Poole Chem. Co., 419 F.3d 355, 364 (5th Cir. 2005).
5. *See, e.g.,* Cooper Indus., Inc. v. Aviall Servs., Inc., 543 U.S. 157, 161, 34 ELR 20154 (2004).
6. 42 U.S.C. §9601(23), (24); *see, e.g.,* Schaefer v. Town of Victor, 457 F.3d 188, 195, 36 ELR 20139 (2d Cir. 2006).

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Cleanup decisions are often based upon the nature and extent of contamination in various environmental media, e.g., soil, sediments, and groundwater, as compared to promulgated standards and the "risk" posed by such contamination. Risk at a CERCLA site is often based upon a determination of whether the contamination increases the lifetime risk of the population in question by more than a certain threshold.[7] The assessment of risk does not look at actual exposures but uses conservative exposure assumptions about groups of hypothetical receptors (such as residents, trespassers, workers, etc.) and relies, in most cases, upon toxicity factors generated from laboratory animals extrapolated to humans (with certain safety factors).[8] A determination is then made as to whether the contamination poses an "acceptable" risk to human health. The risk assessment process is considered sufficiently conservative to apply to even the most sensitive potential receptors. The specific health endpoint, or the baseline risk for the general population of developing the specific harm, is generally not considered. This approach does not evaluate risks to specific individuals. Therefore, a hypothetical CERCLA risk assessment is of little utility in the demonstration of actual causation of a specific harm (or even exposure) to an individual plaintiff.

While a CERCLA risk assessment may not establish causation, together with regulatory standards, it can establish that a specific exposure is not actionable.[9] In many courts across the country, defendants have been able to foreclose a plaintiff's recovery by establishing that the plaintiff was exposed to a level of contamination that is below a regulatory threshold. In these cases, the courts recognize that the regulatory agencies charged with protecting human health and the environment have determined that a certain level of contamination may be tolerated without creating a threat to human health. Examples of such regulatory standards are the maximum contaminant levels for public drinking water supplies under the Safe Drinking Water Act (SDWA).[10] In addition, the Agency for Toxic Substances and Disease Registry (ATSDR) has developed minimal risk levels for hazardous substances that represent a daily human dose of a hazardous substance that is not likely to pose an appreciable risk of human harm.

II. Disease—Causation

Causation in the toxic tort setting focuses on whether the individual's alleged harm was caused by exposure to the contamination in question. Most courts do not require a plaintiff to overcome the scientifically impossible burden of demonstrating causality with absolute proof; rather, most courts require a plaintiff to demonstrate causality with a reasonable degree of scientific certainty: that it is more likely true than not true that the harm was caused by the exposure at issue.[11]

When assessing causation to actual individuals, one must first establish general causation before moving to specific causation.[12] General causation answers the question of whether there is a scientific link between the contaminant in question and the alleged harm. General causation can be established by epidemiological literature linking exposures of a contaminant to a particular medical endpoint in humans. In forming these conclusions, epidemiologists generally rely on the Hill Criteria including: (1) strength; (2) consistency; (3) specificity; (4) temporality; (5) dose-response relationship; (6) plausibility; (7) coherence; (8) experiment; and (9) analogy.[13] These criteria are not intended to be used as checklists, but rather a list of all criteria that may be relevant to the question of causality. Examples of general causation

7. 40 C.F.R. §300.430(e)(2)(i)(A)(2) (2009).
8. *See* U.S. EPA, Publ'n No. EPA/630/P-03/001F, Guidelines for Carcinogen Risk Assessment (2005).
9. *See* Thompson v. S. Pac. Transp. Co., 809 F.2d 1167 (5th Cir. 1988) (exposure to trichloroethane in drinking wells below the EPA-recommended maximum contaminant level was insufficient to establish causation); Brooks v. E.I. du Pont de Nemours & Co., 944 F. Supp. 448 (E.D.N.C. 1996) (contamination that meets state groundwater quality standards is not actionable in tort); Lamb v. Martin Marietta Energy Sys., Inc., 835 F. Supp. 959, 24 ELR 20575 (W.D. Ky. 1993) (concluding that de minimis levels of contamination were not sufficient for a nuisance cause of action); Iberville Parish Waterworks Dist. No. 3 v. Novartis Crop Prot. Inc., 45 F. Supp. 2d 934, 943 (S.D. Ala. 1999) (court dismissed trespass and nuisance claim because chemical levels were below regulatory standards); Adams v. A.J. Ballard Jr. Tire & Oil Co., 2006 WL 1875965, at *3 (N.C. Super. Ct. June 30, 2006).
10. 42 U.S.C. §§300f to 300j-26, ELR Stat. SDWA §§1401-1465.
11. *See, e.g.,* Hagerty v. L&L Marine Servs., Inc., 788 F.2d 315 (5th Cir. 1986); Dartez v. Fibreboard Corp., 765 F.2d 456, 467 (5th Cir. 1985); Gideon v. Johns-Manville Sales Corp., 761 F.2d 1129, 1137-38 (5th Cir. 1985); Laswell v. Brown, 683 F.2d 261, 269 (8th Cir. 1982), *cert. denied*, 459 U.S. 1210, 103 S. Ct. 1205 (1983); Mink v. Univ. of Chicago, 460 F. Supp. 713, 719 (N.D. Ill. 1978); Potter v. Firestone Tire & Rubber Co., 863 P.2d 795 (Cal. 1993); Hannan v. Pest Control Servs., Inc., 734 N.E. 2d 674 (Ind. Ct. App. 2000); Ayers v. Township of Jackson, 461 A.2d 184 (N.J. Super. Ct. 1983). *See also* Fed. Judicial Ctr., Reference Manual on Scientific Evidence (2d ed. 2000).
12. *See* Raynor v. Merrell Pharms. Inc., 104 F.3d 1371, 1376 (D.C. Cir. 1997) (holding that specific causation evidence is irrelevant where there is no general causation evidence indicating that Bendectin causes birth defects); Kelley v. Am. Heyer-Schulte Corp., 957 F. Supp. 873, 875 (W.D. Tex. 1997), *appeal dismissed*, 139 F.3d 899, 1998 WL 127822 (5th Cir. 1998); Snyder v. Upjohn Co., No. 94-1826-GHK, at 8 (C.D. Cal. May 20, 1997) (the only possible specific causes "are those for which general causation has already been established"); Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387, 1412-13 (D. Or. 1996) (courts "have recognized two levels of causation: general causation (i.e., can silicone gel cause disease in anyone?) and specific causation (i.e., did silicone gel breast implants cause disease in this plaintiff?)") (citations omitted); Jones v. United States, 933 F. Supp. 894, 900-01 (N.D. Cal. 1996) (plaintiff must show both "general causation," that defendant's conduct increased likelihood of injury, and "specific causation," that defendant's conduct was probable, not merely possible, cause of injury), *aff'd,* 127 F.3d 1154 (9th Cir. 1997); Rutigliano v. Valley Bus. Forms, 929 F. Supp. 779, 783 (D.N.J. 1996) ("Plaintiff's case requires expert testimony to satisfy her burden with respect to both general causation and specific causation") (citations omitted), *aff'd*, 118 F.3d 1577 (3d Cir. 1997); In re Silicone Gel Breast Implants Prods. Liab. Litig., 887 F. Supp. 1469, 1477 (N.D. Ala. 1995) (noting distinction between general causation and specific causation).
13. Austin Bradford Hill, *The Environmental Disease: Association or Causation?*, 58 Proc. Royal Soc'y Med. 295, 295-300 (1965).

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for environmental exposures include exposure to arsenic-causing skin cancer, exposure to asbestos-causing mesothelioma, and exposure to cigarette smoke-causing lung cancer. If published epidemiological studies do not exist linking a specific chemical exposure to the alleged health effect, then general causation often cannot be established. For example, since no scientific literature exists linking sugar to brain cancer, then a tort complaint making this allegation should not be actionable.

Once general causation has been established, specific causation must be demonstrated. This step evaluates whether an individual plaintiff has received a contaminant dose sufficient to cause an alleged harm. Sufficient information should be collected in discovery to determine the dose, which depends upon exposure concentrations, exposure frequency, exposure duration, and individual body weight for each plaintiff. For example, if exposures to contaminated groundwater are claimed, information regarding well location and depth, groundwater chemistry, dates of use of the well, duration of residence, and consumption rates of water should be collected. Then, toxicological factors (dose-response relationships) from epidemiological studies (rather than studies on laboratory animals) are paired with the specific exposure history (the dose) for each individual allegedly exposed to calculate risk. This risk is then compared with the baseline risk for contracting the alleged harm. Often, if an individual's risk is more than doubled, then it is established that the cause of the alleged harm (the health endpoint) is, more likely than not, the environmental exposure.[14] The concept of risk-doubling is clearly intended to provide the standard of proof required in tort cases and is unrelated to the CERCLA concept of an "acceptable" excess lifetime cancer risk.

The final step in establishing causation is the elimination of confounding sources of risk. There are two types of confounding sources of risk. Multiple sources of the contaminant at issue can be present, and multiple exposure factors that increase the risk of the alleged harm can also be at play. Various metals are good examples of confounding sources of contamination. Lead and arsenic are common in current and historical airborne emissions from certain industries, but they are also commonly found in residential settings. Lead is nearly ubiquitous in paint on older housing stock, but was also historically used in pesticides/herbicides and was emitted from the exhaust of motor vehicles. Arsenic was also commonly used in pesticide and herbicide formulation, and is still present in some commercially available lawn fertilizer. The analysis of spatial patterns of contamination within a neighborhood can show whether chemicals are more likely to have originated from airborne emissions from a nearby industrial facility or from individual homeowner management of household chemicals. Other elements, such as boron and molybdenum, relatively mobile constituents in coal ash, are naturally occurring, often in concentrations greater than U.S. Environmental Protection Agency (EPA) action levels. Even chemicals such as dioxins are ubiquitous in the environment and have significant nonindustrial sources. According to EPA's most recent inventory of sources, the most significant sources of dioxins to the environment in rural areas are currently backyard burn barrels.[15]

In addition to confounding sources of contamination, confounding risk factors for the alleged harm must also be considered. If, for example, one is assessing causation for a tort case involving lung cancer allegedly due to exposures to hexavalent chromium vapor, one must consider the contribution from cigarette smoking and naturally occurring levels of radon in residential structures. Both of these potential sources of risk for developing lung cancer could easily exceed that of industrial exposures to hexavalent chromium, and therefore must be factored into the risk-doubling evaluation to meet the standard of proof.

In some tort cases, no actual injuries are manifested or alleged, but plaintiffs seek compensation for medical monitoring and/or fear of developing cancer or other latent health effects in the future as a result of past exposure. Generally, these causes of action would apply only to exposures to carcinogenic chemicals. Most noncarcinogenic compounds do not have latent effects. If the harm does not develop during the exposure, it will not develop at some later time. Therefore, a plaintiff would have to show, based on the scientific literature, that a specific noncarcinogenic compound has a known latent effect in order to justify a claim for fear of developing a disease or for medical monitoring for exposure to noncarcinogenic compounds.

A claim for medical monitoring may be justified if an exposure to a contaminant increases an individual's risk of developing future disease for which periodic preventative medical screening might be effective. There is no consensus on whether medical monitoring is a viable remedy or even an independent cause of action.[16] Some courts exclude recovery for medical monitoring unless present physical injury can be demonstrated.[17] Nonetheless, a claim or request for medical monitoring generally requires some form of exposure that creates an increased risk of developing a disease that has a treatment that is more effective when applied at the screen-detected stage.[18] It is important to point out that plaintiffs are often not required to go through the same rigor as required to establish disease-causation, as discussed above. In the medical-monitoring context, some courts have held that plaintiffs are only required to show a "significant" increase, as opposed to a showing that the latent disease will more likely

14. *See, e.g.*, In re Meridia Prods. Liab. Litig., 328 F. Supp. 2d 791 (N.D. Ohio 2004); Daniels v. Lyondell-Citgo Ref. Co., Ltd., 99 S.W.3d 722 (Tex. App. 2003).

15. U.S. EPA, Publ'n No. EPA/600/P-03/002F, An Inventory of Sources and Environmental Releases of Dioxin-Like Compounds in the U.S. for the Years 1987, 1995, and 2000 (2006).

16. *See* Metro-N. Commuter R.R. Co. v. Buckley, 521 U.S. 424 (1997).

17. *See, e.g.,* Mergenthaler v. Asbestos Corp., 480 A.2d 647 (Del. 1984); Henry v. Dow Chem. Co., 701 N.W.2d 684 (Mich. 2005); Badillo v. Am. Brands, Inc., 16 P.3d 435 (Nev. 2001).

18. *See, e.g.*, In re Paoli R.R. Yard PCB Litig., 916 F.2d 829, 21 ELR 20184 (3d Cir. 1990); Bocook v. Ashland Oil, Inc., 819 F. Supp. 530 (S.D. W. Va. 1993); Cook v. Rockwell Int'l Corp., 755 F. Supp. 1468 (D. Col. 1991).

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than not be caused by the exposure, as is often required in the traditional toxic tort setting.[19]

While legislation and judicial opinion vary greatly from state to state concerning when damages for medical monitoring may be awarded, one decision by the Louisiana Supreme Court summarized the elements that we believe are necessary to bring a claim for medical monitoring.[20] The criteria required by the Louisiana Supreme Court are:

1. Significant exposure to a proven hazardous substance;
2. As a proximate result of the exposure, plaintiff suffers a significantly increased risk of contracting a serious latent disease;
3. Plaintiff's risk of contracting a serious latent disease is greater than
   a. the risk of contracting the same disease had he or she not been exposed, and
   b. the chances of members of the public at large of developing the disease.
4. A monitoring procedure exists that makes the early detection of the disease possible;
5. The monitoring procedure has been prescribed by a qualified physician and is reasonable and necessary according to contemporary scientific principles;
6. The prescribed monitoring regime is different from that normally recommended in the absence of exposure; and
7. There is some demonstrated clinical value in the early detection and diagnosis of the disease.[21]

In addition to a demonstration of "significantly" increased risk, the Louisiana Supreme Court required a demonstration of risk-doubling (with confounding risk factors for the specific individual to be considered), and a number of medical criteria that must be demonstrated, including the opinion of a reasonable physician that medical monitoring is reasonable, necessary, and likely to be effective.

Therefore, when faced with a claim for medical monitoring associated with a demonstrated increased risk of latent disease, it is also important to determine whether early screening is feasible, and whether screen-detected stage treatment is more effective than treatment applied after symptoms have led to diagnosis. It is well-recognized that early detection and treatment increases survival in screened populations for breast cancer, colorectal cancer, and cervical cancer. For many other types of cancers (lung cancer, for example), there are limited data to suggest that early detection increases survival rates, and it is often difficult to assess the effectiveness of early treatment. While the effectiveness of early detection is not always outcome-determinative, it can be a compelling factor in determining whether to award medical monitoring.[22]

In addition to claims for medical monitoring, many plaintiffs seek damages for emotional distress due to exposures to contaminants. The standard of proof for causation of emotional distress when no manifestation of injury has occurred varies from jurisdiction to jurisdiction. The only numerical or definitive standard we are aware of is the requirement that the exposure will more likely than not result in the development of the feared disease, as expressed by California courts.[23] This is a significantly higher standard of proof than that which is typically required in the toxic tort setting, but it ensures that spurious claims for emotional distress are not brought before the courts.

III. Conclusion

The determination of CERCLA liability, and associated predictions of hypothetical risk of exposure related to chemical emissions from a CERCLA site, does not establish that actual exposure and injury to the nearby population has actually occurred. There are significant additional barriers of proof required to support claims for tort damages of injury, medical monitoring, and emotional distress arising from exposure to environmental contaminants that are not typically considered in a CERCLA evaluation.

19. *See, e.g.,* Hansen v. Mountain Fuel Supply Co., 858 P.2d 970 (Utah 1993). In *Hansen*, the Utah Supreme Court held that:
    > [T]he plaintiff must prove that the exposure was of sufficient intensity and/or duration to increase his or her risk of the anticipated harm significantly over the plaintiff's risk prior to exposure. No particular level of quantification is necessary to satisfy this requirement of significantly increased risk. We reemphasize what should be apparent from our earlier discussion: Because the injury in question is the increase in risk that requires one to incur the cost of monitoring, the plaintiff need not prove that he or she has a probability of actually experiencing the toxic consequence of the exposure.

    Id. at 979.
20. *See* Bourgeois v. A.P. Green Indus., Inc., 716 So. 2d 355 (La. 1998).
21. *Id.* at 360-61.
22. *See Paoli R.R. Yard*, 916 F.2d 829 (requiring proof that there actually are medical tests in existence that make early detection and treatment of disease possible); *Hansen*, 858 P.2d at 979 (requiring proof "that a treatment exists that can alter the course of the illness").
23. *See* Potter v. Firestone Tire & Rubber Co., 863 P.2d 795 (Cal. 1993).