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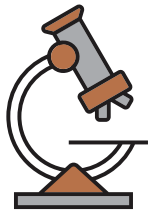


# Regulatory Risk Assessment vs. Tort Law Causation: A Distinction *with* a Difference

*by Kyle W. Mack and Eric M. Kraus*

## INTRODUCTION

Those of us in the business of defending clients in toxic tort and pharmaceutical product liability cases often take it for granted that pronouncements by regulatory agencies regarding risk are distinct and quite different in key ways from what is required in civil litigation for proof of causation. But *how* are they different, *why* are they different and *where* is the evidence that they are intended to be different? The answers are fundamental to defending cases in which a regulatory opinion regarding risk associated with a chemical or pharmaceutical product may be introduced to a jury, particularly if plaintiffs' attorneys try to use risk assessments and maximum exposure limits set by regulatory agencies as "proof" of causation. To a juror, and even to a judge, the simplicity of this approach can be appealing. People (generally) put faith in the opinions of agencies that are tasked with protecting their well-being. The problem is that the opinions of agencies about potential hazards from exposure to chemicals or pharmaceutical products are not opinions about causation as required in the courtroom. To appreciate the differences, we start with some fundamental concepts about how causation is established in a product liability or toxic tort action.

## CAUSATION IN CIVIL TORTS

To establish causation in a product liability/toxic tort action, a plaintiff must establish by a preponderance of the evidence that exposure to the alleged defective product/toxic substance is (1) generally capable of causing the injury alleged, and (2) that such exposure was the cause of their alleged injury.[1] What is important to remember is that "causation" is an inferential determination based on available facts.[2] Not all "facts" carry the same weight, and understanding the different types and grades of scientific evidence encountered in toxic tort/product liability litigation is necessary to come to any conclusions about a causal relationship between an exposure and an adverse outcome.

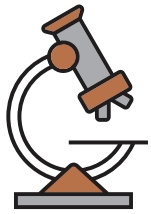
The hierarchy of evidence starts with human data, which are the most useful with respect to causal assessments. Mammalian animal studies[3] are more useful than non-mammalian, and data from any animal species is generally more useful than *in vitro* data.[4]

1 *Simpson v. Johnson & Johnson*, No. 5:20-cv-1237, 2020 WL 5630036, *1 (N.D. Ohio Sept. 21, 2020) ("In toxic tort cases, the causation inquiry is two-pronged. First, a plaintiff must show that the substance to which she was exposed *can cause* the type of injury alleged [general causation]. Next, a plaintiff must show that in her case, exposure to the substance *actually caused* the alleged injury [specific causation]." (citation omitted)).

2 Steve C. Gold, Michael D. Green & Joseph Sanders, *Scientific Evidence of Factual Causation: An Educational Module*, The National Academies of Sciences, Engineering, Medicine (Oct. 2016), https://www.nationalacademies.org/our-work/science-policy-decision-making-educational-modules/modules.

3 Primate studies being the most useful mammalian animal study.

4 Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, in Reference Manual on Scientific Evidence at 645 (3d ed. 2011). [As described by the Reference Manual at p. 645, "*In vitro* research concerns the effects of a chemical on human or animal cells, bacteria, yeast, isolated tissues, or embryos." (emphasis added). In other words, it is data acquired from the laboratory, not from humans in real-world settings].



Animal studies can be hypothesis-generating but alone cannot prove causation. Indeed, courts have often ruled that human epidemiologic studies are *required* to prove causation.[5] A statistically significant, positive animal study can raise questions as to whether the same effect may occur in humans, but does not prove it.[6] There are still several hurdles to clear before any conclusion can be reached with respect to applicability of the findings to humans. Some of these hurdles include disproportionately higher doses often used in animal studies,[7] and potential differences in the dose-response relationship between humans and the test animal(s).[8] There are a number of reasons for this dose-response variability that are based on the compound's physical properties and the pharmacokinetic/toxicokinetic characteristics (absorption, distribution, metabolism, excretion) specific to each species.[9] Some of the reasons for differences in pharmacokinetic/toxicokinetic characteristics across species include differences in metabolic rates, enzymatic function or expression, tissue distribution, genetic susceptibilities, etc. For these reasons, animal studies alone cannot carry the day for a plaintiff.[10] Indeed, plaintiffs' over reliance on animal studies involving excessive doses of the compound at issue has been rejected by courts.[11] Understanding the limitations of animal data, and how such limitations affect plaintiff's burden of establishing causation, are critical.

Within the world of epidemiology, randomized placebo-controlled clinical trials are the most coveted, with other forms of primary human epidemiological studies (cohort studies, case-control studies, cross-sectional studies, and case series/reports) carrying progressively less weight for purposes of inferring causation in the courtroom.[12] The reliability of epidemiologic studies is another factor to consider with respect to application in a product liability/toxic tort claim. Prospective randomized controlled studies generally provide more reliable data for pre-specified outcomes. The reason for this is due to the ability of researchers to control for potential confounders prior to the commencement of the study, as opposed to mathematically adjusting for them retrospectively. Confounders are factors that may result in the outcome being studied, but are not related to the hypothesis being evaluated. For example, if a study is evaluating whether a diabetes medication is associated with an increased incidence of heart attacks, but another condition which is

5 *See In re Zoloft (Sertraline Hydrochloride) Prods. Liab. Litig.*, 26 F. Supp. 3d 466, 475 (E.D. Pa. 2014) ("Several courts have held that positive human epidemiological studies are required to reach reliable conclusions as to whether an agent is teratogenic in humans, and causation opinions based primarily upon *in vitro* and live animal studies are unreliable and do not meet the *Daubert* standard."). (*See also Wade-Greaux v. Whitehall Lab'ys, Inc.*, 874 F. Supp. 1441, 1453 (D. V.I. 1994) ("Although animal studies play a role in teratological investigations, it is scientifically invalid to extrapolate observations in animal experiments directly to human beings to determine human teratogenicity."), *aff'd*, 46 F.3d 1120 (3d Cir. 1994).

6 *See In re Zoloft*, 26 F. Supp. 3d at 481.

7 *In re Rezulin Prods. Liab. Litig.*, 369 F. Supp. 2d 398, 407 (S.D.N.Y. 2005) ("[T]he high doses often used in animal studies may not correspond to considerably lower concentrations of a drug or other substance to which humans are in reality exposed.").

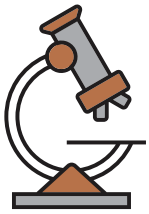
8 Goldstein & Henifin, *supra* note 4, at 646.

9 *Id.*

10 *See Wade-Greaux*, 874 F. Supp. at 1480 ("The notion that one can accurately extrapolate from animal data to humans to prove causation without supportive positive [human] epidemiologic studies is scientifically invalid because it is inconsistent with several universally accepted and tested scientific principles."). *See also In re Zoloft*, 26 F. Supp. 3d at 477.

11 *In re Rezulin*, 369 F. Supp. 2d at 421 & n.142 ("The Supreme Court found that the animal studies on which the experts relied, which involved exposing infant mice to massive doses of PCBs, 'were so dissimilar to the facts presented in this litigation that it was not an abuse of discretion for the District Court to have rejected the experts' reliance on them.'" (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 144-45 (1997)).

12 John B. Wong, Lawrence O. Gostin & Oscar A. Cabrera, *Reference Guide on Medical Testimony*, in Reference Manual on Scientific Evidence at 723-4 (3d ed. 2011).



known to be associated with heart attacks, such as high blood pressure (the confounder), is not controlled for, the data may be biased and the results of the study skewed. The reason for this is that the confounder could have been the cause of the observed outcome (subjects who experienced heart attack), which may result in erroneous calculation of statistical significance. Overall, epidemiologic data are only as sound as the study design and scientific rigor of the researchers conducting it. If researchers fail to identify and remove relevant confounders, or if the overall study design allows for potential errors in data collection and processing, the study may still be subject to false outcomes.

What is important to remember is that any single piece of evidence, regardless of its place in the hierarchy, can only provide facts for, or against, a causal determination. The type, quality, and to some extent the quantity, of the available scientific evidence in a case from each level of the hierarchy will drive the outcome of the causal determination.

## REGULATORY RISK ASSESSMENTS

Federal and international agencies conduct risk assessment analyses when a population may be exposed to a substance potentially harmful to health. Examples of such substances include heavy metals, air pollutants, carcinogens and radiation. Exposure limits are implemented if an agency's assessment of such compounds suggests the potential for harm across a population. Exposure limits and similar standards are based on a precautionary approach with built-in conservative safety margins and are not a definitive conclusion about a causal relationship.[13] A precautionary approach is used because agencies are tasked with protecting all people regardless of age, sex, race, preexisting medical conditions, genetic profile, economic status, living conditions, working conditions or location within the agencies' jurisdictions. To accomplish this task, a conservative blanket approach is a necessity, as all permutations of the above variables that may apply to each individual person cannot be accounted for. Considerations must also be taken for whether exposure to the substance is avoidable or inevitable.

Regulatory risk assessments are generally based on some or all of the following elements:

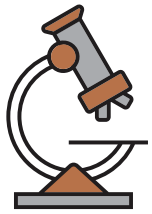
- Hazard identification;[14]
- Dose-response assessment;
- Exposure assessment; and
- Risk characterization.

These elements are derived from the National Research Council (NRC) risk management paradigm, conceived by the Committee on Institutional Means for Assessment of Risks to Public Health ("Committee") and published in 1983.[15] The Committee was commissioned by Congress

13 Gold, *supra* note 2, at 14-15 (footnote omitted) ("Most critical is the specific legal standard contained in the regulatory legislation – the 'risk trigger' – set by Congress as the threshold for regulatory action. Smaller risks than would likely be adequate to support specific causation may be appropriate for regulation, especially when large numbers of persons are exposed to the risk factor. Some statutes specify that regulations must be constructed conservatively so as to provide an adequate margin of safety, often referred to as the 'precautionary principle.'").

14 When agencies evaluate whether a chemical presents a hazard to human health, the initial approach is broad. Data associated with the chemical are evaluated to identify any potential/possible health effects. From there, agencies determine which effects, if any, have the strongest association based on the available evidence to guide categorization of the chemical. This is very different from legal causation. "The classic hazard identification where only one chemical is studied and all possible health effects are evaluated has generally been relegated to organizations that specialize in categorizing chemicals (e.g., carcinogen, neurotoxin) or those who identify safe levels of exposure [e.g., the Environmental Protection Agency (EPA), the International Agency for Research on Cancer (IARC), the Occupational Safety and Hazard Administration (OSHA), and the American Conference of Governmental Industrial Hygienists (ACGIH)]." Dennis J. Paustenbach, Human and Ecological Risk Assessment: Theory and Practice, John Wiley and Sons, Inc., (2002).

15 Nat'l Research Council, Comm. on the Institutional Means for Assessment of Risks to Public Health, *Risk Assessment in the Federal Government: Managing the Process*, (1983).



in 1981 — in response to public criticism of the risk assessment process and regulatory determinations made by federal agencies. The Committee was tasked to "strengthen the reliability and objectivity of scientific assessment," which serves as the foundation of regulatory policies regarding public health hazards.[16] The Committee determined that much of the public criticism stemmed from "the sparseness and uncertainty of the scientific knowledge of the health hazards addressed" at the time.[17] In response to this, the Committee ultimately proposed the standardization of the risk assessment process across federal regulatory agencies through development of a uniform set of inference guidelines.[18]

This model has been updated over time and, in 2009, the additional elements of "planning and scoping" and "problem formulation" were added by the NRC.[19] "Planning" relates to defining the purpose of the risk assessment, determining the resources needed to conduct the assessment, and developing an operating plan to see the assessment through. "Scoping" relates to establishing the boundaries of the risk assessment. This includes determining the potential harms, populations that may be affected, routes of exposure, duration of exposure, geographic factors and how the assessment will impact stakeholders.[20] "Problem Formulation" pertains to the development of a conceptual model and plan to move the risk assessment analysis forward.[21]

Following the NRC model, federal agencies consider various forms of data when conducting risk assessments. The forms of data and their overall weight can vary depending on multiple factors, including the route of exposure, duration of exposure, and the expected severity and nature of harm being considered by the agency. While such analyses can be useful for establishing precautionary exposure limits in order to protect populations as a whole from potential harm, they generally do not provide a clear picture with respect to determining whether a true causal association exists in humans, or the extent of potential risk the substance actually carries.

An example of this can be seen with the Environmental Protection Agency's (EPA) hazard identification for carcinogenic effects. The agency currently uses an assessment methodology that combines available data from humans (primarily epidemiological), long-term animal studies, and other supporting data including pharmacokinetic and pharmacodynamic studies, genotoxicity studies and chemical structure-activity relationships, etc.[22] Based on the outcome of such assessments, EPA classifies compounds into carcinogenic probability categories. Such assessments are "focused on the amount and quality of evidence regarding whether or not a substance is carcinogenic to humans, ***not on the***

16 *Id.* at iii.

17 *Id.* at 6.

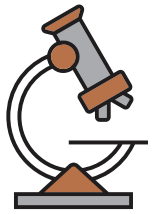
18 Inference guidelines are "an explicit statement of a predetermined choice among alternative methods (inference options) that might be used to infer human risk from data that are not fully adequate or are not drawn directly from human experience." *Id.* at 4.

19 Nat'l Research Council, Comm. on Improving Risk Analysis Approaches Used by the U.S. EPA, *Science and Decisions: Advancing Risk Assessment* (2009).

20 *See* Micah L. Berman, Taleed El-Sabawi & Peter G. Shields, *Risk Assessment for Tobacco Regulation*, *Tobacco Regulatory Science*, 2019 Jan;5(1):36-49.

21 *Id.*

22 EPA, *Dose-Response Assessment for Assessing Health Risks Associated With Exposure to Hazardous Air Pollutants*, (last updated Feb. 28, 2020), https://www.epa.gov/fera/dose-response-assessment-assessing-health-risks-associated-exposure-hazardous-air-pollutants.



***level of risk a substance might present***."[23] In other words, EPA's classification of a substance as a carcinogen speaks to the robustness of the evidence as to whether it carries the *potential* to be carcinogenic; not the likelihood that it actually will cause cancer.

## REGULATORY RISK ASSESSMENTS IN THE COURTROOM

The question remains: How is a regulatory risk assessment different from what is required in civil litigation to prove causation? As noted above, in the courtroom, a plaintiff generally must establish by a preponderance of the evidence that the substance in question (1) can cause the alleged adverse outcome, and (2) that it did cause the adverse outcome in their specific circumstance. In the regulatory realm, as noted above, determinations of risk are designed to protect populations, not to establish that a particular exposure will cause an adverse outcome. In the pharmaceutical context, an example comes from the International Agency for Research on Cancer's (IARC) classification of combined estrogen-progesterone oral contraceptives/menopausal therapy, which in IARC's classification scheme shares the same carcinogenicity categorization as plutonium, neutron radiation and products of nuclear fission (Group 1 – Carcinogenic to Humans).[24] This classification focuses on whether the combined hormone therapy carries any carcinogenic hazard; it does not assess the level of risk for specific cancers it actually conveys to a user of such therapy.[25] Furthermore, the use of categories to help define different degrees of "hazard" can be very misleading. With millions of person-years-of-use data available, we know oral contraceptives/hormone replacement therapies do not carry the same carcinogenic risk as plutonium or neutron radiation even though all of these substances are similarly categorized. Another example of this difference is IARC's 2016 categorization of processed meats as Group 1 carcinogens. The pronouncement of such impact required it to be walked-back in the press by IARC's parent entity, the World Health Organization, after being flooded with inquiries about what the categorization actually meant.[26] Examples such as these have resulted in medical experts openly criticizing IARC's categorizations, saying they are more confusing to policy makers and the lay public than they are helpful.[27]

The mission of regulatory agencies like the EPA and the Federal Drug Administration (FDA) make clear that the purpose of regulatory risk assessments is to protect human health in the broadest possible sense; it is not intended to fulfill the requirements of legal causation.[28] The mission of

23 EPA, *Risk Assessment for Carcinogenic Effects*, (last updated Apr. 7, 2021), https://www.epa.gov/fera/risk-assessment-carcinogenic-effects (emphasis added).

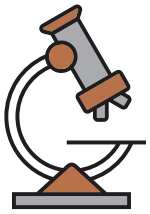
24 WHO, IARC Monographs on the Identification of Carcinogenic Hazards to Humans, *List of Classifications*, (last updated Mar. 26, 2021), https://monographs.iarc.who.int/list-of-classifications.

25 The most recent IARC Monograph on the cancer risk from combined estrogen-progestogen contraceptives characterized the combination drug as "*carcinogenic to humans (Group 1).*" *See*, WHO, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Pharmaceuticals, Volume 100A, A Review of Human Carcinogens, Combined Estrogen-Progestogen Contraceptives, 2012, at p. 311, https://publications.iarc.fr/Book-And-Report-Series/Iarc-Monographs-On-The-Identification-Of-Carcinogenic-Hazards-To-Humans/Pharmaceuticals-2012. However, the text of the Monograph contains an analysis of various studies with varying, sometimes inconsistent, data for different kinds of breast cancers, depending, for example, on duration of use, time that such therapy was initiated, was it continuous and, if not continuous, time since last use. These kinds of factors would be critical in any litigation matter but are not addressed in the ultimate assignment of a category by IARC.

26 *Id.*

27 *Id.*

28 See, for example, the mission statements of EPA (EPA, Our Mission and What We Do, https://www.epa.gov/aboutepa/our-mission-and-what-we-do (last visited Apr. 21, 2021)); FDA (FDA, What We Do, https://www.fda.gov/about-fda/what-we-do (last visited Apr. 21, 2021)).



IARC is even further removed from legal causation as its stated mission is focused on cancer research, the promotion of international collaboration and cancer prevention.[29] It is the task of these agencies to protect the health and welfare of the population as a whole — or at least as a group — by limiting, or preventing, even *potentially* harmful exposures. To achieve this goal, agencies are held to a lesser standard of proof than a plaintiff in a courtroom.[30]

In contrast, legal causation seeks to definitively determine if a particular individual experienced a particular harm due to a particular substance. As articulated by the Southern District of New York in *Mancuso v. Consolidated Edison Co.*:

> The distinction between avoidance of risk through regulation and compensation for injuries after the fact is a fundamental one. In the former, risk assessments may lead to control of a toxic substance even though the probability of harm to any individual is small and the studies necessary to assess the risk are incomplete; society as a whole is willing to pay the price as a matter of policy. In the latter, a far higher probability (greater than 50%) is required since the law believes it unfair to require an individual to pay for another's tragedy unless it is shown that it is more likely than not that he caused it. … Regardless of what regulatory action would be justified by current evidence … such evidence would not be sufficient to find an individual defendant liable to an individual plaintiff.[31]

As the court noted in *Mancuso*, even when facing a limited scientific record, an agency's responsibility is still to protect the population based on the evidence at hand, even if that evidence is limited. In the courtroom, however, limited evidence may very well mean that the claim should not be able to proceed.

Regulatory conclusions provide easy ways for plaintiffs' lawyers to circumvent the rigorous burden of proof required in the courtroom. Often, the misleading use of regulatory findings are the subject of motions *in limine*. When these efforts fail, sometimes the only way to rebut these efforts is with a careful presentation of the scientific evidence that demonstrates that the characterization of a substance by EPA, IARC, FDA or other regulatory bodies, does not address the ultimate question jurors must answer: based on a preponderance of the available and credible scientific evidence, does this particular substance — in the form and dose in which humans are actually exposed — cause negative outcomes in humans, and did this particular substance by its alleged exposure route, duration and quantity, cause the plaintiff's *particular* alleged injury?[32]

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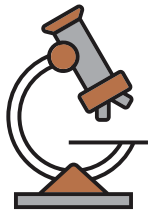
29 WHO, *IARC's Mission: Cancer research for cancer prevention*, https://www.iarc.who.int/about-iarc-mission/ (last visited Apr. 21, 2021).

30 *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 198 (5th Cir. 1996) (citing *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996)). See also *Yates v. Ford Motor Co.*, No. 5:12-CV-752-FL, 2015 WL 2189774, at *23 (E.D.N.C. May 11, 2015) (quoting *Mitchell v. Gencorp Inc.*, 165 F.3d 778, 783 n. 3 (10th Cir. 1999)) ("The methodology employed by a governmental agency results from the prevention perspective that the agencies adopt in order to reduce public exposure to harmful substances. The agencies' threshold of proof is reasonably lower than that appropriate in tort law, which traditionally makes more particularized inquiries into cause and effect and requires a plaintiff to prove that it is more likely than not that another individual has caused him or her harm.").

31 *Mancuso v. Consol. Edison Co. of N.Y.*, 967 F. Supp. 1437, 1448 (quoting *In re Agent Orange Prod. Liab. Litig.*, 597 F. Supp. 740, 781 (E.D.N.Y. 1984), *aff'd in relevant part*, 818 F.2d 145 (2d Cir. 1987)).

32 *Mitchell*, 165 F.3d at 779-80.



This issue was recently addressed in the *In re Roundup Products Liability Litigation*. MDL No. 2741 (N.D. Cal. Feb. 18, 2019). District Court Judge Chhabria held that IARC's classification of glyphosate was an admissible fact, but the monograph created by the agency regarding its *interpretation* of the glyphosate data was not. Particularly, Judge Chhabria stated "discussion of the IARC classification will be restricted under Rule 403 to avoid wasting time or misleading the jury, as ***the primary inquiry is what the scientific studies show, not what IARC concluded they show***."[33] Although modified before ultimately being presented to the jury, Judge Chhabria's proposed jury instructions clearly and eloquently articulated why regulatory positions do not, and cannot, speak to the question of legal causation in a civil trial:

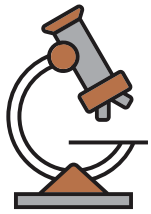
> IARC's decision to classify glyphosate as a probable carcinogen, even if you agree with it, is not sufficient on its own to support a conclusion that glyphosate is capable of causing non-Hodgkin's lymphoma at exposure levels similar to what [plaintiff] experienced. As IARC explains, its monographs 'evaluate[ ] cancer hazards but not the risks associated with exposure. ***The distinction between hazard and risk is important. An agent is considered a cancer hazard if it is capable of causing cancer under some circumstances. Risk measures the probability that cancer will occur, taking into account the level of exposure to the agent.*** The Monographs Programme may identify cancer hazards even when risks are very low with known patterns of use or exposure.'[34]

Thus, in Judge Chhabria's articulation of the differences, a regulatory determination as to whether a substance is hazardous is a qualitative assessment, while the litigation requirement of establishing that a substance carries a risk is both a qualitative *and* a quantitative one. IARC's decision to classify glyphosate as a carcinogen cannot speak to whether, in the manner and in the doses used by humans, it is capable of causing cancer generally or whether it caused a specific plaintiff's cancer, because IARC did not complete a quantitative evaluation of the scientific evidence to weigh carcinogenic risk associated with exposure to the compound. To establish causation in a product liability/toxic tort action, a plaintiff must be able to convince a jury that they were exposed to a sufficient quantity of the substance for a sufficient duration – which they also must have already established can cause the harm generally – to induce the alleged injury, and in some circumstances, that they were also exposed to the sufficient quantity during a requisite timeframe in their life (e.g., birth defect litigation). In a toxic tort case, expert testimony on the issue of general causation meets Daubert's "fit" or relevance requirement "only if the testimony includes an opinion that (1) exposure to the particular substance at issue, (2) in the dose to which plaintiff was exposed, (3) for the duration in which plaintiff was exposed, (4) can cause the particular condition(s) of which the plaintiff complains."[35] Dose is one of the most important factors a litigator must

33 Pretrial Order No. 81: Ruling on Motions *in Limine* at 1, *In re Roundup Prods. Liab. Litig.*, No. 3:16-MD-02741 (N.D. Cal. Feb. 18, 2019), ECF No. 2775 (emphasis added).

34 Pretrial Order No. 77: Court's Proposed Phase 1 Substantive Jury Instructions and Verdict Form at 2, *In re Roundup Prods. Liab. Litig.*, No. 3:16-MD-02741 (N.D. Cal. Feb. 12, 2019), ECF No. 2706 (second alteration in original) (emphasis added).

35 *Amorgianos v. Nat'l R.R. Passenger Corp.*, 137 F. Supp. 2d 147, 163 (E.D.N.Y. 2001), aff'd, 303 F.3d 256 (2d Cir. 2002).



consider in a product liability/toxic tort case.[36] Without taking into account quantity of exposure, IARC's conclusion does not address a fundamental element for establishing causation in product liability/toxic tort civil litigation and, therefore, does not speak to the ultimate issue the jury must decide upon.

## CONCLUSION

While plaintiffs' attorneys may attempt to introduce hazard assessments by regulatory agencies before a jury, such hazard assessments were never meant to establish legal causation. They are conducted to prevent potential harms to the population at large. As discussed above, they generally do not address the elements that plaintiffs are required to establish in the courtroom. The weight of an agency's determination alone is insufficient with respect to satisfying causation standards in civil litigation, particularly specific causation. Care should be taken either to keep regulatory determinations away from juries altogether — because of the likelihood that they can mislead jurors or — to explain how and why they are different, and that the regulatory determination does not answer the causation question that plaintiffs must answer in the courtroom. ■

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36 "Scientific knowledge of the harmful level of exposure to a chemical plus knowledge that plaintiff was exposed to such quantities are minimal facts necessary to sustain the plaintiff's burden in a toxic tort case." *Mitchell*, 165 F.3d at 781 (quoting *Allen*, 102 F.3d at 199).