

No. 16-3310

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**IN THE UNITED STATES COURT OF APPEALS  
FOR THE SIXTH CIRCUIT**

IN RE: E. I. DU PONT DE NEMOURS AND COMPANY  
C8 PERSONAL INJURY LITIGATION

CARLA BARTLETT,  
*Plaintiff-Appellee,*

v.

E. I. DU PONT DE NEMOURS AND COMPANY,  
*Defendant-Appellant.*

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On Appeal from the United States District Court for the Southern  
District of Ohio, Case Nos. 2:13-cv-00170 & 2:13-md-02433

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**BRIEF OF CHAMBER OF COMMERCE OF THE UNITED  
STATES OF AMERICA, AMERICAN TORT REFORM  
ASSOCIATION, AND AMERICAN CHEMISTRY COUNCIL AS  
*AMICUS CURIAE* IN SUPPORT OF  
DEFENDANT-APPELLANT AND REVERSAL**

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## TABLE OF CONTENTS

TABLE OF CONTENTS .....	i
TABLE OF AUTHORITIES.....	ii
STATEMENT OF IDENTIFICATION AND INTEREST .....	1
STATEMENT OF COMPLIANCE WITH RULE 29(C)(5) .....	3
SUMMARY OF THE ARGUMENT .....	3
ARGUMENT .....	8
I.    The District Court Conflates General and Specific Causation in Ways That Will Substantially Harm Tort Defendants. ....	8
A.    The Leach Agreement Reflects the Accepted Understanding About the Distinction Between General And Specific Causation.....	9
B.    The District Court’s Interpretation of the Leach Agreement is a Drastic Break With Established Causation Principles. ....	14
II.   The District Court’s Error in Interpreting the Leach Agreement Promises to Stifle an Innovative Alternative Approach to Causation Issues. ....	17
A.    The District Court Deprived DuPont of Its Bargained-For Specific Causation Defense.....	18
B.    The District Court’s Errors Endanger the Viability of a Cost-Efficient Method of Handling Mass-Tort Cases.....	21
CONCLUSION.....	25

## TABLE OF AUTHORITIES

### Cases

<i>Amchem Prods. Inc., v. Windsor</i> , 521 U.S. 591 (1997) .....	22, 23
<i>Best v. Lowe’s Home Centers, Inc.</i> , 563 F.3d 171 (6th Cir. 2009) .....	12
<i>Conde v. Velsicol Chem. Corp.</i> , 804 F. Supp. 972 (S.D. Ohio 1992) .....	10
<i>Downs v. Perstorp Components, Inc.</i> , 126 F. Supp. 2d 1090 (E.D. Tenn. 1999) .....	13, 19
<i>In re Meridia Prods. Liab. Lit.</i> , 328 F. Supp. 2d 791 (N.D. Ohio 2004) .....	9, 10
<i>Leach v. Dupont</i> , Civ.A. No.: 01-C-608 (W.Va. Cir. Ct. Nov. 22, 2004) .....	23
<i>McClain v. Metabolife Int’l, Inc.</i> , 401 F.3d 1233 (11th Cir. 2005) .....	16
<i>Norris v. Baxter Healthcare Corp.</i> , 397 F.3d 878 (10th Cir. 2005) .....	13
<i>Pluck v. BP Oil Pipeline Co.</i> , 640 F.3d 671 (6th Cir. 2011) .....	9, 15
<i>Rider v. Sandoz Pharms. Corp.</i> , 295 F.3d 1194 (11th Cir. 2002) .....	9
<i>Terry v. Caputo</i> , 875 N.E.2d 72 (Ohio 2007) .....	21
<i>Textileather Corp. v. GenCorp Inc.</i> , 697 F.3d 378 (6th Cir. 2012) .....	19
<i>United States v. Wood</i> , 877 F.2d 453 (6th Cir. 1989) .....	18

**Other Authorities**

Bernard D. Goldstein & Mary S. Henifen, *Reference Guide on Toxicology*, in FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (3d ed. 2011) ..... 12

Deborah R. Hensler, *The socio-economics of mass torts: What we know, don't know, and should know*, in RESEARCH HANDBOOK ON THE ECONOMICS OF TORTS (Jennifer Arlen, ed. 2013) ..... 22

Joseph Sanders, *From Science to Evidence: The Testimony on Causation in the Bendectin Cases*, 46 STAN. L. REV. 1 (1993) ..... 21

Margaret A. Berger, *The Supreme Court's Trilogy on the Admissibility of Expert Testimony*, in FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2d ed. 2000) ..... 9

Michael D. Green et al., *Reference Guide on Epidemiology*, in FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (3d ed. 2011) ..... 11, 12, 13

**Treatises**

RESTATEMENT (THIRD) OF TORTS (Am. Law Inst. 2010) ..... 9, 10, 12, 22

**STATEMENT OF IDENTIFICATION  
AND INTEREST**

*Chamber of Commerce of the United States of America* (“the Chamber”). The Chamber is the world’s largest not-for-profit business federation. For more than 100 years, it has represented American businesses of every size, in every sector of the economy, and from every region of this country. The Chamber represents 300,000 direct members and indirectly represents the interests of 3 million businesses and trade and professional organizations. An important function of the Chamber is to represent the interests of its members before Congress, the Executive Branch, and the courts. For this reason, the Chamber often files *amicus curiae* briefs in cases, such as this one, that implicate issues of significant importance to the business community.

*American Tort Reform Association* (“ATRA”). A broad-based coalition of businesses, corporations, municipalities, associations, and professional firms that have pooled their resources to promote reform of the civil justice system, the members of ATRA share the goal of ensuring fairness, balance, and predictability in civil litigation. For more than two decades, ATRA has filed *amicus curiae* briefs in cases

before state and federal courts that have addressed important liability issues.

*American Chemistry Council* (“ACC”). The ACC represents the leading companies engaged in the business of chemistry. ACC members apply the science of chemistry to make innovative products and services that make people's lives better, healthier and safer. The members of ACC are committed to common sense advocacy designed to address major public policy issues.

The district court's interpretation of the Leach Agreement, if allowed to stand, threatens to deter business defendants from relying on a mutually beneficial and efficient alternative model for resolving general causation issues in toxic-tort litigation. *Amici* represent many businesses that have been defendants in mass toxic-tort litigation. The economic costs of such litigation can be enormous, and agreements like the one at issue here could potentially reduce those costs—not only for business defendants, but also for plaintiffs and for the courts that must adjudicate these issues. More broadly, a correct understanding of general causation and its relationship to specific causation is critical in such litigation. It is therefore vital to the interests of *amici* and their

members that this Court reject the district court's erroneous construction of the Leach Agreement and its denial of DuPont's right to defend as to specific causation.

**STATEMENT OF COMPLIANCE  
WITH RULE 29(C)(5)**

*Amici* file this brief pursuant to Rule 29(a) of the Federal Rules of Appellate Procedure. Counsel for all parties to this appeal have been informed of the intended filing of this brief, and Appellant has consented to the brief. In an email to undersigned counsel on June 16, 2016, counsel for Appellee stated that Appellee takes no position at this time on whether the motion should be granted. Counsel for the Appellant did not author any portion of this brief. No party to this appeal has provided financial support to fund the preparation or submission of this brief.

**SUMMARY OF THE ARGUMENT**

The plaintiff, Clara Bartlett, sued DuPont, alleging that C8 from DuPont's Washington Works facility caused her kidney cancer. Ordinarily, the plaintiff's burden of proof would have included proving that C8 is capable of causing kidney cancer. If C8 does not cause kidney cancer, then it does not matter what level of C8 the plaintiff was



exposed to or what other factors might be responsible for her kidney cancer. Here, under the Leach Agreement, the Science Panel's finding of a "probable link" between C8 and kidney cancer, coupled with DuPont's agreement not to contest general causation, meant that whether C8, as a general matter, is capable of causing kidney cancer was off the table at the plaintiff's trial.

But even if C8 is capable of causing kidney cancer as a general matter, it does not necessarily mean that C8 *did cause* this plaintiff's kidney cancer given her low exposure level. Under the accepted understanding of general causation, that concept is not tied to any particular dose or exposure level; it concerns whether a substance is capable *at all* of causing a given condition. The Leach Agreement, moreover, defined "general causation" in accordance with that general understanding, without mentioning dose or exposure level. And the Science Panel did not purport to find a probable link between C8 and kidney cancer at the plaintiff's exposure level or at all exposure levels. To the contrary, the substance of the Science Panel's work makes clear that it found *no* elevation in risk at the plaintiff's exposure level.

DuPont therefore should have been free to defend on the ground that C8 at the plaintiff's very low exposure level did not cause her kidney cancer. That defense did not dispute general causation because general causation is not tied to exposure level as a general matter or as defined in the Leach Agreement. And that defense in no way contradicted the Science Panel's report, which did not make a finding of a probable link at this plaintiff's exposure level. Yet the district court prevented DuPont from presenting such a defense, mistakenly concluding that a defense specific to the plaintiff's exposure level improperly disputed the Science Panel's finding.

Further, even assuming that whether C8 at the plaintiff's exposure level *was capable of* causing kidney cancer was somehow subsumed under the Science Panel's probable link finding, whether C8 at her exposure level was *likely* to cause kidney cancer was certainly not a general causation issue. At the very least, therefore, DuPont should have been free to defend on the ground that even if the plaintiff's exposure to C8 was capable of causing her kidney cancer, it was very unlikely to have done so. Bartlett, after all, had to prove that C8 more likely than not caused her kidney cancer; that C8 was merely capable of

doing so was insufficient to carry that burden. Whether an individual plaintiff's specific level of exposure actually caused her disease is the very definition of specific, not general, causation—both as a general matter and under the Leach Agreement. And the Science Panel's work strongly supported this defense, because it found an elevated risk only at much higher exposure levels.

Yet the district court prevented DuPont from presenting this specific causation defense. The court misconstrued both DuPont's agreement not to contest whether C8 is capable of causing kidney cancer and the Science Panel's finding and concluded that applying the probable link finding to a given class member "establishes that it is more likely than not that there is a link between *that class member's exposure* to [C8] *and his or her Linked Disease.*" Evidentiary Mots. Order No. 1 at 9–10 (July 20, 2015) (emphasis added). This conclusion transformed general causation into specific causation and essentially directed a verdict for plaintiff on causation—and transformed DuPont's carefully bargained-for agreement not to contest whether C8 is capable of causing kidney cancer into a forced capitulation to plaintiff's allegation that C8 caused her kidney cancer. The district court's rulings

were all the more unjust because the Science Panel—the expert body whose findings the court was supposedly deferring to—did not find a higher relative risk at plaintiff’s low exposure level.

The Leach Agreement was promising. In toxic-tort litigation like this, enormous resources are devoted to having experts analyze and dispute technical scientific issues for decision by a lay jury. By creating a Science Panel of expert epidemiologists and agreeing to be bound by their general causation findings, the parties here chose a different and potentially groundbreaking path. The Leach Agreement aimed to spare the plaintiffs, DuPont, the trial judge, and countless jurors from having to wrestle with the difficult issue of whether exposure to C8 is capable of causing a given disease, while leaving DuPont free to argue that exposure to C8 did not cause a particular plaintiff’s disease. This innovative approach had the potential to significantly improve the efficiency as well as the scientific validity of verdicts.

Unfortunately, the district court’s gross misreading of the Leach Agreement, and its departure from the accepted background principles and methods of causation analysis that the Agreement reflects, will likely deter future defendants from entering into similar mutually

beneficial and efficient agreements to resolve threshold general causation issues. If an expert panel's finding that exposure to a given substance is linked to a given disease can be twisted into a finding that an individual plaintiff's exposure more likely than not caused her disease—contrary to all available scientific evidence showing no elevation in risk at the plaintiff's exposure level—then no reasonable tort defendant will agree to refer causation issues to the judgment of an independent panel again.

## ARGUMENT

### **I. The District Court Conflates General and Specific Causation in Ways That Will Substantially Harm Tort Defendants.**

The Leach Agreement clearly incorporated accepted background principles of general and specific causation that govern toxic-tort cases. Yet the district court departed from this understanding and conflated the two separate analyses. The district court's error, if replicated in other cases, is likely to substantially prejudice tort defendants and, by overcompensating tort plaintiffs, raise costs for all consumers.

**A. The Leach Agreement Reflects the Accepted Understanding About the Distinction Between General And Specific Causation.**

Causation is “frequently the crucial issue” in toxic-tort actions. Margaret A. Berger, *The Supreme Court’s Trilogy on the Admissibility of Expert Testimony*, in FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 32 (2d ed. 2000). Such cases are often “won or lost on the strength of the scientific evidence presented to prove causation.” *Rider v. Sandoz Pharms. Corp.*, 295 F.3d 1194, 1197 (11th Cir. 2002). To prevail, a toxic-tort plaintiff must show both general and specific causation. *See, e.g., Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671, 676–77 (6th Cir. 2011) (“In a toxic-tort case, as here, the plaintiff must establish both general and specific causation through proof that the toxic substance is capable of causing, and did cause, the plaintiff’s alleged injury.” (citing *In re Meridia Prods. Liab. Lit.*, 328 F. Supp. 2d 791, 798 (N.D. Ohio 2004))). The distinction between general causation (exposure to a substance can cause a particular disease) and specific causation (exposure to a substance likely did cause that disease in a specific person) is well-grounded. *See, e.g.,* RESTATEMENT (THIRD) OF TORTS § 28 cmt. c. (3)–(4) (Am. Law Inst. 2010) (“RESTATEMENT”).

The first step for a plaintiff is to establish general causation, which answers an abstract question that is necessary, but not sufficient, for the plaintiff to prevail: is a substance (such as C8) *capable* of causing a particular disease? See RESTATEMENT § 28 cmt. c. (3) (“General causation’ exists when a substance is capable of causing a given disease.”). To make their general causation case, litigants turn to epidemiologists, who interpret large-scale public health data sets to determine whether there is a significant enough association between exposure to a substance and incidence of a disease to establish a likely causal link. See *Meridia Prods.*, 328 F. Supp. 2d at 800 (epidemiological analysis of public health data is the “primary generally accepted methodology for demonstrating a causal relation between the chemical compound and a set of symptoms or a disease” (quoting *Conde v. Velsicol Chem. Corp.*, 804 F. Supp. 972, 1025–26 (S.D. Ohio 1992))).

Causality has several hallmarks; the two most commonly litigated are “relative risk” and “dose-response.” Thus, epidemiologists often present their analysis of potential causal association in terms of the “relative risk” of the disease, *i.e.*, the strength of the association between incidence of the disease and exposure to a substance. A relative

risk of “1” denotes no association; “[t]he higher the relative risk, the greater the likelihood that the relationship is causal.” Michael D. Green et al., *Reference Guide on Epidemiology*, in FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 602 (3d ed. 2011) (hereinafter, “*Reference Guide*”).<sup>1</sup> In addition, epidemiologists will frequently identify the “dose-response” curve of the association, or whether and at what rate an increase in exposure increases the risk of developing a disease. *See id.* at 603 (“[A] dose–response relationship is strong, but not essential, evidence that the relationship between an agent and disease is causal.”).

These features of epidemiological analysis are merely ways of answering the abstract question posed above: is a substance *capable* of causing a given disease? Whether people exposed to a given substance are more likely, on a collective basis, to contract a given disease is a very different question from whether exposure to that substance actually caused the disease in a particular person. That latter question is beyond the domain of epidemiology and general causation. *See*

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<sup>1</sup> A relative risk of “1” means a 1:1 ratio of incidence of a disease among persons exposed to the substance and among persons not exposed, *i.e.*, the disease is equally common in both populations and exposure to the substance has no effect on a person’s risk.



*Reference Guide* at 609 (epidemiologists investigating general causation do not determine whether “an agent did cause a specific plaintiff’s disease”).

Rather, that inquiry is the province of specific causation. *See* RESTATEMENT § 28 cmt. c(4) (“‘Specific causation’ exists when exposure to an agent caused a particular plaintiff’s disease.”). Specific causation evidence may come from toxicologists, who testify about whether a plaintiff’s dose was enough that the exposure more likely than not caused the plaintiff’s disease, *see* Bernard D. Goldstein & Mary S. Henifen, *Reference Guide on Toxicology*, in FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 665–69 (3d ed. 2011), or from physicians, who may seek to rule out other potential causes through a methodology called “differential diagnosis,” *see, e.g., Best v. Lowe’s Home Centers, Inc.*, 563 F.3d 171, 178 (6th Cir. 2009).

These two approaches to specific causation analysis are closely related. If a plaintiff’s exposure, even if theoretically capable of causing her disease, was unlikely to have done so, then alternative potential causes are relatively more likely. Defendants thus frequently present both of these defenses together: the plaintiff’s exposure level was too

low to be a likely cause of the disease, and “other significant causes (including exposure to other substances, lifestyle, workplace, and genetic factors) of the individual’s clinical condition” are more likely to have caused the disease. *Downs v. Perstorp Components, Inc.*, 126 F. Supp. 2d 1090, 1095 (E.D. Tenn. 1999). This specific causation inquiry necessarily follows the general causation determination, because “without general causation, there can be no specific causation.” *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 881 (10th Cir. 2005).

The Leach Agreement clearly acknowledged this accepted distinction between general and specific causation. *Compare* Agreement § 1.25 (“‘General Causation’ [means] that it is probable that exposure to [C8] is capable of causing a particular Human Disease.”) *with id.* § 1.60 (“‘Specific Causation’ [means] that it is probable that exposure to [C8] caused a particular Human Disease in a specific individual.”). Based on this commonly accepted understanding, the Science Panel’s charge to determine whether there was a “probable link”<sup>2</sup> between exposure to C8 and kidney cancer was clearly not a mandate to assess specific causation in any one individual. The panel was composed of

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<sup>2</sup> “Probable link” is a lower standard than “general causation.” DuPont Br. 7

epidemiologists, not clinicians. *See Reference Guide* at 608–09 (“[S]pecific causation[] is beyond the domain of the science of epidemiology.”). The panel was also “free to consider all scientifically relevant data,” Agreement § 12.2.3(b), including animal and out-of-class human data based on a wide spectrum of exposures, some much higher than the .05 ppb required for class membership, *id.* § 2.1.1. *See also* Probable Link Evaluation of Cancer 10 (April 6, 2015) (hereinafter, “Evaluation”) (Science Panel noting that it looked beyond “data relating only to Class Members”). And, of course, based on that understanding of the Science Panel’s charge, DuPont agreed to “not contest the issue of General Causation” but “reserved[] the right to contest Specific Causation . . . as to any individual Class Member or plaintiff.” Agreement § 3.3.

**B. The District Court’s Interpretation of the Leach Agreement is a Drastic Break With Established Causation Principles.**

The district court committed a fundamental interpretive error by losing sight of the limited nature of the Science Panel’s analysis and findings. The Science Panel found a probable link between C8 and kidney cancer. Evaluation at 10. This association, however, was weak:

relative risk as shown by worker-mortality studies was “not elevated compared to the U.S. population.” *Id.* And the dose-response curve demonstrated that the risk of kidney cancer due to C8 exposure was a no-association “1” at the lowest exposure levels and barely exceeded 1.5 even at higher exposure levels. *Id.* at 11–12 (Figures 1 & 2).

Given the Science Panel’s conclusion, the Leach Agreement barred DuPont from contesting general causation, *i.e.*, whether C8 *could* cause kidney cancer. But the district court went much further: it held that DuPont was barred from contesting whether C8 *at this plaintiff’s exposure level* materially increased her risk of kidney cancer and that applying the Probable Link Finding to an individual class member’s case “establishes that it is more likely than not that there is a link between *that class member’s exposure* to [C8] *and his or her Linked Disease.*” Evidentiary Mots. Order No. 1 at 9–10 (July 20, 2015) (emphasis added). This reading essentially took any specific causation defense off the table.

The district court’s error has severe consequences for businesses that find themselves frequent litigants in toxic-tort cases. The distinction between general and specific causation is fundamental to

these cases. *See Pluck*, 640 F.3d at 676–77. To carry their burden, toxic-tort plaintiffs must establish that a given exposure appreciably increased their individual risk of developing a disease. *See McClain v. Metabolife Int’l, Inc.*, 401 F.3d 1233, 1244 (11th Cir. 2005) (rejecting expert’s methodology for lack of a “show[ing] that taking Metabolife increases the risk [of disease]”). This necessarily requires establishing their individual exposure level and dose, *see id.* at 1242 (requiring plaintiffs to show “enough exposure to cause the plaintiff’s specific illness”), and in some cases showing that other individualized conditions or factors were less likely than the exposure to have caused their disease, *see id.* at 1233 (noting that expert must consider all potential “causes of a disease”).

The district court’s conflation of these concepts permits a plaintiff to short-circuit this well-established framework. Under the district court’s interpretation, plaintiffs can simply argue that, upon a showing that exposure *could* cause a certain disease, *any* exposure suffices to have caused that disease. Such an approach is not simply an unacceptably drastic break with established causation principles that govern all modern toxic-tort cases, *see supra* at 8–14; it is certain to

force tort defendants to wildly overcompensate plaintiffs for very minimal exposures that cause minute elevations of risks. These costs will inevitably be passed to consumers in the form of higher prices and reduced innovation and productive capacity.

## **II. The District Court's Error in Interpreting the Leach Agreement Promises to Stifle an Innovative Alternative Approach to Causation Issues.**

Despite the Leach Agreement's incorporation of the accepted distinction between general and specific causation and its express reservation of DuPont's right to defend on specific causation, the district court prevented DuPont from doing so. The court erroneously transformed the Probable Link Finding into a finding that the plaintiff's exposure to a very low level of C8 *more likely than not did cause* her kidney cancer. This error contradicted the plain text of the Leach Agreement, departed from settled causation analysis, and deprived DuPont of a bargained-for specific causation defense. This interpretive error promises to have grave consequences for future attempts to innovatively settle toxic-tort litigation.

**A. The District Court Deprived DuPont of Its Bargained-For Specific Causation Defense.**

Barring DuPont from contesting whether C8 at the plaintiff's very low exposure level could cause kidney cancer was a clear error. As explained above, general causation concerns whether exposure to a substance is *at all* capable of causing a given disease. *See supra* at 9–11. A key issue at trial thus should have been whether Bartlett's exposure to C8 at the lowest level reliably measured could have materially increased her risk of kidney cancer. DuPont Br. 32–34. The district court treated that issue as already resolved in the plaintiff's favor due to the Probable Link Finding, *see* Dispositive Mot. Order 7–10 (Dec. 17, 2014), even though general causation is ordinarily not tied to a particular exposure level. *See supra* at 9–11.

To be sure, the parties *could* have incorporated into the Leach Agreement's definition of general causation an exposure level (such as the lowest detectable level of .05 ppb) or a range of exposure levels, and thus departed from the general understanding of that term. But they did not, and the district court's imposition of that exposure level into the general causation definition was clear error. *See United States v. Wood*, 877 F.2d 453, 457 (6th Cir. 1989) (settlement agreements are

contracts); *Henry v. Chesapeake Appalachia, L.L.C.*, 739 F.3d 909, 912 (6th Cir. 2014) (courts must give effect to “the plain language of the contract unless that language is ambiguous” (quoting *Textileather Corp. v. GenCorp Inc.*, 697 F.3d 378, 382 (6th Cir. 2012))). Moreover, the district court’s imposition of an exposure level on the agreement’s definition of “general causation” contradicts the Science Panel’s findings, since the evaluation makes crystal clear that the panel found an elevated risk only at the highest exposure levels. DuPont Br. 23. No such exposure level can possibly be read into that finding when it contravenes the substance of the Science Panel’s report.

The district court’s error is even clearer when it comes to DuPont’s defense that C8 was not likely to have caused the plaintiff’s kidney cancer. Even if, contrary to the Science Panel’s findings, exposure at her very low level could actually cause kidney cancer, her low level of exposure is still not *likely* to have caused her kidney cancer, which the Science Panel’s evaluation makes clear. DuPont Br. 23. And DuPont was prepared to offer credible evidence that “other significant causes (including . . . lifestyle . . . and genetic factors)” posed greater risks. *Downs*, 126 F. Supp. 2d at 1095; DuPont Br. 32–34. Yet the court



prevented DuPont from defending on this critical point. If “specific causation” is to mean anything, then it must mean that DuPont can present these defenses. They were explicitly reserved by DuPont in the Leach Agreement, and fully consistent with (indeed, strongly supported by) the Science Panel’s findings.

The court’s rulings basically eliminate DuPont’s unambiguously reserved specific causation defense. By denying DuPont the right to present this evidence—essentially directing a verdict for Bartlett on causation—the district court denied DuPont the benefit of its bargain under the Leach Agreement. DuPont Br. 35. The Leach Agreement was not obtained cheaply: Among other provisions, DuPont made payments directly to the class and for medical monitoring, Agreement §§ 9.1, 12.2.2, & 12.3.2, and forfeited the right to contest general causation whenever the Science Panel found a probable link, *id.* § 3.3. In return, DuPont “reserve[d] the right to contest Specific Causation . . . as to any individual Class Member or plaintiff.” *Id.* By simply reading this provision out of existence, the district court has essentially created a new contract, one that no reasonable corporation would ever sign.

**B. The District Court's Errors Endanger the Viability of a Cost-Efficient Method of Handling Mass-Tort Cases.**

The district court's error could emanate far beyond this one toxic-tort case or even the 3,500 cases in this MDL. The Leach Agreement might have been a model for future mass-tort litigants, an example of efficient bargaining that could save litigants, jurors, and judges time, expense, and frustration. But unless the district court's error is corrected, the Leach Agreement may very well be the last of its kind.

Establishing causation “involves a scientific inquiry,” and so plaintiffs normally must establish causation through the testimony of expert witnesses. *See Terry v. Caputo*, 875 N.E.2d 72, 77 (Ohio 2007). General causation is usually shown by applying complex statistical methods to large-scale public health data sets. *See Meridia Prods.*, 328 F. Supp. 2d at 800. This inquiry often involves complicated and competing expert testimony about dose-response curves, regression analysis, and the strength of statistical associations, among other issues. *See Reference Guide* at 599–600. This is a highly technical enterprise that many non-experts are ill-equipped to confront with confidence. *See Joseph Sanders, From Science to Evidence: The Testimony on Causation in the Bendectin Cases*, 46 STAN. L. REV. 1, 5–

12 (1993) (reporting that juries delivered verdicts contrary to the scientific evidence in 40 percent of Bendectin cases); RESTATEMENT § 28 cmt. c (1) (“[S]ome courts [] distrust juries’ ability to resolve cases based on conflicting general expert-opinion evidence.”). Cleaving off general causation for determination by independent experts thus reduces the complexity of the issues that juries must confront, while potentially improving the scientific accuracy of decisions.

Further, mechanisms such as the Leach Agreement can also be valuable cost-savings tools that redound to the benefit of all litigants, including plaintiffs. Expert services are costly for all parties, and these costs contribute to the shocking expense of mass tort litigation. *See Amchem Prods. Inc., v. Windsor*, 521 U.S. 591, 631–32 (1997) (Breyer, J., concurring in part and dissenting in part) (noting that 61 cents of every dollar spent in asbestos litigation goes to transaction costs); Deborah R. Hensler, *The socio-economics of mass torts: What we know, don’t know, and should know*, in RESEARCH HANDBOOK ON THE ECONOMICS OF TORTS 289–92 (Jennifer Arlen, ed. 2013) (reporting that Merck spent \$1 million per day in Vioxx litigation). These costs are often borne indirectly by plaintiffs, whose eventual settlement payouts

are reduced to account for expert expenses. *Cf. Amchem*, 521 U.S. at 631–33 (Breyer, J., concurring in part and dissenting in part) (noting ability of a settlement to “make more money available for plaintiffs”). Finding sensible, cost-saving ways of approaching these complex causation issues is thus a likely net benefit to litigants on both sides of a mass-tort case.

The Leach Agreement, and others like it, seek to reduce these costs. The Leach Agreement promised to alleviate “problems of proof and [the] possibility of modifications to applicable law,” sparing both parties the “time, expense, and distraction of embroilment [in litigation].” Joint Mot. Prelim. Approval Settlement 4–5, *Leach v. Dupont*, Civ.A. No.: 01-C-608 (W.Va. Cir. Ct. Nov. 22, 2004). By resolving the issue of general causation prior to trial, the Leach Agreement promised to spare both plaintiffs and DuPont alike millions in discovery and litigation costs. *See id.* at 4–5. With the Science Panel’s probable link finding (or lack thereof) dispositive of general causation, the parties could avoid protracted litigation, complicated epidemiological testimony, and the fees entailed by both.

The district court's error short-circuited this process for determining causation issues by essentially directing a verdict for Bartlett. No rational defendant would agree to delegate away its entire causation defense to an independent panel in this manner. By interpreting the Leach Agreement to deprive DuPont of its unambiguously reserved specific causation defense as well as its general causation defense, the district court has all but ensured that no litigant will enter into a similar agreement in the future.

## CONCLUSION

For the foregoing reasons, this Court should reverse the district court's judgment and remand for a new trial.

Respectfully submitted,

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## CERTIFICATE OF COMPLIANCE

This brief complies with the type-volume limitation of Fed. R. App. P. 32(a)(7)(B) because, as determined by Microsoft Word 2010, it contains 4,548 words, excluding the parts of the brief exempted by Fed. R. App. P. 32(a)(7)(B)(iii). This brief complies with the typeface requirements of Fed. R. App. P.(a)(5) and the type style requirements of Fed. R. App. P. 32(a)(6) because it has been prepared in a proportionately spaced typeface using Microsoft Word 2010 in 14-point Century Schoolbook font.

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## CERTIFICATE OF SERVICE

I hereby certify that I electronically filed the foregoing with the Clerk of the Court for the United States Court of Appeals for the Sixth Circuit by using the appellate CM/ECF system, which will effect service on all parties, on June 20, 2016.

*/s/ Jeffrey S. Bucholtz* \_\_\_\_\_

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