No. 23-30854

IN THE UNITED STATES COURT OF APPEALS
FOR THE FIFTH CIRCUIT

FLOYD RUFFIN,
Plaintiff-Appellant,

v.

BP EXPLORATION & PRODUCTION, INCORPORATED;
BP AMERICA PRODUCTION COMPANY,
Defendants-Appellees.

On Appeal from the United States District Court
for the Eastern District of Louisiana,
No. 2:20-cv-00334
Hon. Ivan L.R. Lemelle, United States District Judge

BRIEF OF AMICUS CURIAE PUBLIC CITIZEN IN SUPPORT OF PLAINTIFF-APPELLANT AND REVERSAL

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April 10, 2024

SUPPLEMENTAL CERTIFICATE OF INTERESTED PERSONS

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Pursuant to this Court's Rule 29.2 and Federal Rule of Appellate Procedure 26.1, amicus curiae Public Citizen submits this supplemental certificate of interested persons to fully disclose all those with an interest in this brief and to provide the required information as to their corporate status and affiliations.

The undersigned counsel of record certifies that the following listed persons and entities as described in the fourth sentence of Rule 28.2.1, in

addition to those listed in the brief of plaintiff-appellant Floyd Ruffin, have an interest in the outcome of this case. These representations are made in order that the judges of this Court may evaluate possible disqualification or recusal.

A. Amicus curiae **Public Citizen** is a nonprofit, non-stock corporation. It has no parent corporation, and no publicly traded corporation has an ownership interest in it of any kind.

B. The above-listed amicus curiae is represented by **Nicolas A. Sansone** and **Allison M. Zieve** of **Public Citizen Litigation Group**.

/s/ Nicolas A. Sansone
Nicolas A. Sansone

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April 10, 2024

TABLE OF CONTENTS

TABLE OF AUTHORITIES

Cases **Page(s)**

Other Authorities

INTEREST OF AMICUS CURIAE[1]

Public Citizen is a nonprofit consumer advocacy organization founded in 1971, with members in all fifty states. Public Citizen works before Congress, administrative agencies, and courts to advance the interests of consumers, workers, and the public. Public Citizen has a longstanding interest in promoting legal rules that more effectively enable workers and other members of the public to seek redress when corporate practices expose them to health or environmental hazards. To advance that interest, Public Citizen has filed briefs in the Supreme Court and the courts of appeals advocating for such rules, including in connection with the Deepwater Horizon explosion and oil spill. *See, e.g.*, *Atl. Richfield Co. v. Christian*, 140 S. Ct. 1335 (2020); *In re Deepwater Horizon BELO Cases* (11th Cir. Nos. 23-11535, 23-11538, 23-11539) (amicus brief filed Sept. 21, 2023). This brief explains that the district court's exclusion of plaintiff-appellant Floyd Ruffin's expert's opinion on general causation was based on reasoning that departed from scientific

[1] This brief was not authored in whole or part by counsel for a party, and no one other than amicus curiae or its counsel made a monetary contribution to the preparation or submission of the brief. Counsel for all parties have consented to its filing.

norms and that placed a far more stringent burden on Mr. Ruffin's expert than this Court's precedent demands.

SUMMARY OF ARGUMENT

Although experts seeking to determine whether a particular substance has adverse health effects cannot ethically conduct controlled clinical studies on humans, other methodologies may offer reliable inferential support showing or refuting a causal link. One approach that this Court has favored is observational epidemiology, which involves identifying real-world populations that have been exposed to the substance and comparing their experience to the experience of unexposed populations. If the comparison reveals an association between exposure and a given health condition, an expert can make a scientific judgment, based on a variety of factors, as to whether the association is likely causal. A single epidemiological study, however, rarely if ever provides a sufficiently clear picture to support a confident conclusion on causation. To make a reliable causal inference, then, an expert must typically examine the entire body of relevant epidemiological, toxicological, and other studies, assess whether the studies cohere with one another and

with accepted scientific background principles, and exercise professional judgment in drawing a conclusion from the overall weight of the evidence.

The general-causation testimony that Mr. Ruffin submitted in this case from expert epidemiologist Dr. Benjamin Rybicki followed this accepted scientific practice. Dr. Rybicki carefully evaluated a body of observational studies regarding health outcomes among populations that had been exposed to certain chemicals found in crude oil, assessed that evidence against a backdrop of other scientific knowledge about how those substances act on humans and animals at the cellular level, and concluded that those substances can increase the risk of prostate cancer. Consistent with standard practice, in other words, Dr. Rybicki considered the full range of available evidence, produced a reasoned causal hypothesis that attempted to harmonize the evidence, and then used his scientific judgment to determine that there was a sufficient basis for accepting this hypothesis. Because Dr. Rybicki faithfully applied the methodology commonly employed by experts in his field, his opinion on general causation is sufficiently reliable to satisfy the liberal admissibility standards of Federal Rule of Evidence 702, even if a jury might choose to reject that opinion following an adversarial proceeding.

The district court's two reasons for excluding Dr. Rybicki's general-causation opinion misconstrue the science and the law. First, the court faulted Dr. Rybicki for failing to identify a precise quantitative dose at which exposure to the chemicals at issue can begin to cause harm, even though this inquiry has nothing to do with general causation and, in any event, falls within the province of toxicology, not epidemiology. Although the district court purported to derive its "harmful dose" requirement from this Court's opinions, no precedent of this Court imposes the rigid and unrealistic rule that the district court applied. Second, the district court criticized Dr. Rybicki for characterizing some epidemiological studies as more probative than others, but it entirely failed to address Dr. Rybicki's considered reasons for doing so. And despite the district court's criticisms of the studies upon which Dr. Rybicki chiefly relied—criticisms that were in any event irrelevant to general causation—the court disregarded the fact that those studies formed but one component of the broader mosaic of scientific evidence on which Dr. Rybicki based his opinion.

ARGUMENT

I. A reliable expert opinion on general causation typically rests on a cumulative body of evidence, no one part of which necessarily creates a strong causal inference on its own.

The "gold standard" methodology for drawing a scientific conclusion about the effect that a particular substance has on human health is a controlled study that exposes one group of people to the substance and compares outcomes for that group to outcomes for a similarly constituted control group that has not been exposed to the substance. *See* Michael D. Green, et al., *Reference Guide on Epidemiology* (hereafter, *Ref. Guide Epidem.*), *in* Fed. Jud. Ctr., Nat'l Research Council of the Nat'l Acads., *Reference Manual on Scientific Evidence* 549, 555 (3d ed. 2011), https://tinyurl.com/r373cjjt (hereafter, *Ref. Manual*). Researchers evaluating whether a new drug is safe and effective for use in humans, for example, undertake controlled clinical trials to compare outcomes for patients to whom the drug is administered and patients to whom it is not—after conducting preliminary laboratory and animal tests to ensure that the clinical trials will not expose the human participants to the risk of harm. *See* FDA, *Step 3: Clinical Research* (Jan. 4, 2018), https://tinyurl.com/yttstres. Researchers evaluating a causal link

between a particular substance and an *adverse* health condition, however, are precluded by ethical standards from conducting studies that would deliberately expose human subjects to the substance. *Ref. Guide Epidem.* at 555 & n.15. They therefore must typically instead draw inferences from observational epidemiological studies, toxicological studies, and other sources of biological knowledge. *See id.* at 556–65.

In observational epidemiological studies, a researcher does not control a preselected population's exposure to the potentially harmful substance under laboratory conditions but instead studies a group of individuals who have been exposed to the substance during the course of real-world events and compares that group to an unexposed group. *Id.* at 555–56. Because these studies usually "focus on individuals living in the [relevant] community," a researcher cannot control the characteristics of the individuals involved. *Id.* at 556. But a well-designed study that accounts for "the possibility of differences in the two populations being studied with regard to risk factors other than exposure" can provide reliable (although not definitive) information about whether exposure is associated with an observed health outcome and about the strength of any association. *Id.* at 556–57. Of course, the possibility always exists

that an association observed in a given study is the product of random chance, *id.* at 573, and researchers use the term "statistically significant" to indicate that "the probability … of observing an association at least as large as that found in the study when in truth there is no association" falls below a predetermined level (often 5 percent) called a *p*-value, *id.* at 576–77. That said, "any criterion for 'significance' is somewhat arbitrary," *id.* at 573, and even findings of a very strong association with a very high probability of being "true" (up to just shy of 95 percent, for example, where the *p*-value is 5 percent) can technically be deemed statistically insignificant.

Once an observational study has revealed an association between exposure to a substance and a particular health condition, a researcher must next assess the likelihood that the substance is a *cause* of the condition—in other words, the likelihood that the increased incidence of the condition among exposed individuals would not have been observed but for the fact of exposure. *Id.* at 597–98. An inference of causation, while "informed by scientific expertise," cannot be "made by using an objective or algorithmic methodology" and instead depends on a researcher's "judgment." *Id.* at 600. A set of nine factors known as the

Hill factors can guide epidemiologists in making causal inferences by prompting them to consider, for example, the temporal relationship between the exposure and the health outcome, the strength of the association between exposure and the risk of experiencing the outcome, and whether a causal relationship would cohere with existing knowledge about biological structures and processes. *Id.* at 599–600. But "no formula or algorithm … can be used to assess whether a causal inference is appropriate" based on the factors, and "there is no threshold number" of factors that must be met before such an inference can be drawn. *Id.*

In contrast to observational epidemiology, toxicology—another method of evaluating whether a substance causes a particular effect on the body—typically involves testing the substance on laboratory animals at varying dosages. *Id.* at 563. Toxicological studies are useful in assessing causation because they "can be conducted as true experiments," as "researchers control all aspects of the animals' lives." *Id.* But they have "two significant disadvantages." *Id.* First, anatomical differences between humans and other animals mean that an observed effect in an exposed laboratory animal will not necessarily occur, or occur in the same way, in a similarly exposed human. *Id.* Second, because animal studies

often involve direct exposure at high doses, even studies that suggest a causal link between a substance and a health effect may leave open the possibility that real-world human exposures would fall below a lower "threshold no-effect dose" and so would not be associated with any adverse health consequences. *Id.*

This Court has identified epidemiological studies as "the most useful and conclusive type of evidence" for establishing general causation in the toxic tort context. *Brock v. Merrell Dow Pharms., Inc.*, 874 F.2d 307, 311 (5th Cir. 1989); *see also, e.g.*, *Johnson v. Arkema, Inc.*, 685 F.3d 452, 467 (5th Cir. 2012) (per curiam). But "a single [epidemiological] study" will "[r]arely, if ever, … persuasively demonstrate a cause-effect relationship." *Ref. Guide Epidem.* at 604. Drawing a reliable causal conclusion instead "typically requires consideration of numerous findings, which, when considered alone, may not individually prove the [conclusion]." Margaret A. Berger, *The Admissibility of Expert Testimony*, *in Ref. Manual* 11 at 19–20. Accordingly, "many of the most well-respected and prestigious scientific bodies … consider all the relevant available scientific evidence, taken as a whole, to determine which

conclusion or hypothesis regarding a causal claim is best supported by the body of evidence." *Id.* at 20 (citing sources).

The First Circuit expressly recognized this point in a decision reversing the exclusion of an expert opinion that was based on the aggregate weight of several bodies of independently insufficient evidence. As that court explained, scientists tasked with drawing causal inferences can reliably "reason[] to the best explanation for all of the available evidence," even if no one body of evidence "itself … justif[ies] an inference of causation." *Milward v. Acuity Specialty Prods. Grp., Inc.*, 639 F.3d 11, 23 (1st Cir. 2011); *cf. Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 198 (5th Cir. 1996) (holding the "weight of the evidence" insufficient where the experts' conclusion was "at best weakly supported, if not contradicted, by the evidence on which they rel[ied]" and the experts "all declined to say that they would subject their findings to the test of peer review"). Ultimately, then, even where "no one line of evidence support[s] a reliable inference of causation," it does not follow that "an inference of causation based on the totality of the evidence [is] unreliable." *Milward*, 639 F.3d at 23.

II. Dr. Rybicki derived his opinion on general causation from a reliable methodology pursuant to which he assessed a body of epidemiological studies against a backdrop of scientific knowledge drawn from other sources.

In this case, Mr. Ruffin submitted an expert report from Dr. Benjamin Rybicki, a genetic and molecular epidemiologist who has been "actively researching the genetic and environmental causes of prostate cancer" for decades. D. Ct. Dkt. No. 121-1 (Rybicki Rep.) at 3; *see id.* at 1. After examining an array of scientific evidence, Dr. Rybicki found "moderate to strong support" for the proposition that exposure to a certain class of chemicals found in crude oil—polycyclic aromatic hydrocarbons (PAHs)—can cause prostate cancer. *Id.* at 18. In reaching this conclusion, Dr. Rybicki drew on his considerable expertise and followed a reliable methodology for making causal inferences, thus satisfying the "liberal" admissibility standard for expert opinion evidence. *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 588 (1993) (quoting *Beech Aircraft Corp. v. Rainey*, 488 U.S. 153, 169 (1988)); *see* Fed. R. Evid. 702 advisory committee's note on 2000 amendment (emphasizing that "the rejection of expert testimony is the exception rather than the rule").

Dr. Rybicki began his analysis by observing that the International Agency for Research on Cancer has classified benzo[a]pyrene, "the most prevalent compound in PAH mixtures," as a human carcinogen based on "strong and extensive experimental evidence" from animal studies, "supported by … consistent and coherent mechanistic evidence" that "provide[s] biological plausibility" for the inference that the chemical causes cancer in humans. Rybicki Rep. at 9–10; *see, e.g.*, *id.* at 10 (detailing one study in which 80 percent of mice who were exposed topically to benzo[a]pyrene developed tumors, compared to 7 percent in a control group). Dr. Rybicki then reviewed "[a]ll human studies concerning PAH occupational exposure and prostate cancer risk that exist in the extant scientific literature," *id.* at 4, and found that "[a] significant epidemiological literature … support[s] a link between occupational exposure to PAH and increased prostate cancer risk," *id.* at 11. For example, a study of over 5,000 chimney sweeps with high exposure to PAHs found "significantly elevated" rates of prostate-cancer mortality. *Id.* And a study of automotive workers who were exposed to metal-working fluids known to contain PAHs "showed a linear increase in prostate cancer risk … associated with cumulative exposure." *Id.*

Dr. Rybicki acknowledged that some studies did not detect an association between occupational PAH exposure and prostate cancer, but he explained that this outcome could be attributable to aspects of study design or to "lower levels" of exposure among the studied populations. *Id.* at 12. Dr. Rybicki also emphasized that it is not surprising that PAH exposure does not necessarily affect all populations in the same way. One study, for example, found no overall association between PAH exposure and prostate cancer but *did* find an increased risk of prostate cancer among those workers who had the highest levels of exposure and who had an underlying genetic susceptibility to the condition. *Id.* Based on the entire body of epidemiological literature, Dr. Rybicki found support for the proposition that "higher levels, e.g. generally the highest quartile of the PAH exposure distribution, of occupational PAH exposure appear to increase prostate cancer risk, particularly in genetically susceptible individuals." *Id.* (formatting omitted).

Dr. Rybicki then tested this proposition against other sources of scientific knowledge. He noted, for example, that studies of earlier oil spills had detected chromosomal damage in clean-up workers and statistically significant increases in prostate cancer incidence in the

surrounding areas. *Id.* at 13. While identifying certain limitations in these studies, Dr. Rybicki considered them as "supplemental evidence" that could not "be wholly excluded." *Id.* Dr. Rybicki also observed that "the carcinogenic potential of PAH has long been known through in vitro studies of mouse prostate cells," and he explained that a recent "mechanistic study" identified certain cellular effects of benzo[a]pyrene and provided a "biologic rationale" as to how exposure could lead to DNA damage and ultimately to cancer. *Id.* at 14. Dr. Rybicki walked through this "biologic pathway" in great technical depth, detailing the chemical processes by which PAH can bind to DNA, creating "PAH-DNA adducts" that can cause mutations during cell division. *Id.* at 14–15. In one study, Dr. Rybicki noted, elevated levels of these adducts were associated with an increased prostate-cancer risk of 50 percent among men who, like Mr. Ruffin, are African American. *Id.* at 16; *see id.* at 7.

Having thus laid out the body of epidemiological and biomechanical evidence suggesting an association between PAH exposure and prostate cancer, Dr. Rybicki applied the Hill factors, *see supra* at 7–8, to assess whether this association could be causal. Rybicki Rep. at 16–18. While Dr. Rybicki acknowledged that the strength of the association revealed

in the epidemiological studies was "modest," he emphasized that such studies are "fraught with measurement error," which "often pushes associations toward the null," and that "considering underlying genetic risk and subsets of individuals at greater risk can reveal stronger associations." *Id.* at 16. And, critically, Dr. Rybicki explained that "[t]he epidemiological literature supporting a role of PAH-induced prostate cancer is strongly supported by molecular and laboratory findings," which have "demonstrated a strong association between PAH-DNA adduct levels and risk of prostate cancer progression" and have even begun to "elucidate the mechanism" by which exposure creates biological alterations that heighten the risk of disease. *Id.* at 17–18. Based on "the strength of [this] experimental evidence," Dr. Rybicki concluded that "PAHs must be considered a potential prostate cancer risk factor that can be cancer-causing in the occupational setting." *Id.* at 18 (formatting omitted).

Dr. Rybicki's conclusion on this point rested on his application of the accepted scientific methodology by which an epidemiologist draws causal inferences from a body of observational and laboratory studies. *See Daubert*, 509 U.S. at 594 (noting that the "[w]idespread acceptance"

of an expert's methodology, while not required for admissibility, "can be an important factor" in assessing reliability). To be sure, none of the sources from which Dr. Rybicki drew his opinion purported to establish a definitive causal link between PAH exposure and prostate cancer. But "in epidemiology hardly any study is ever conclusive," and an expert need not "back his or her opinion with published studies that unequivocally support his or her conclusions." *Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 354 (5th Cir. 2007). After all, given the impossibility of conducting experimental human studies in this context, assessing causation requires an expert to exercise "judgment about how the [relevant] study findings fit with other scientific knowledge." *Ref. Guide Epidem.* at 553.

Qualified experts might reach different inferential conclusions even after faithfully applying established principles. Under *Daubert*, however, the "appropriate means of attacking" a supposedly "shaky" conclusion is "[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof," not "wholesale exclusion." *Daubert*, 509 U.S. at 596; *see United States v. 14.38 Acres of Land*, 80 F.3d 1074, 1078 (5th Cir. 1996) (per curiam) (noting that "the trial court's

role as gatekeeper is not intended to serve as a replacement for the adversary system"). A jury should accordingly have the opportunity to test Dr. Rybicki's opinion on general causation during an adversarial proceeding and ultimately determine whether to credit it.

III. The district court abused its discretion in rejecting Dr. Rybicki's general-causation opinion as unreliable.

Upon reviewing Dr. Rybicki's testimony, the district court had no "doubt that there's any material, factual dispute or legal dispute that PAH is harmful to humans, that PAH has been proven through [the cited] studies to produce … prostate cancer." D. Ct. Dkt. No. 167 (Oral Arg. Tr.) at 31. The court nonetheless ruled that this compelling showing was not "enough" to render Dr. Rybicki's general-causation opinion admissible. *Id.* In the district court's view, this Court's precedent "requires a causation expert to identify the harmful level of exposure to a chemical to sustain the plaintiff's burden" on general causation, and Dr. Rybicki had not satisfied this supposed requirement. *Id.* at 110; *see id.* at 110–12. Additionally, the district court found it "problematic" that Dr. Rybicki relied on studies of "automotive industry workers [and] chimney sweeps," rather than "studies of petroleum workers," to support his opinion that PAH is capable of producing prostate cancer. *Id.* at 113. Because the

district court did not indicate whether it viewed either of these rationales as an independently sufficient ground for excluding Dr. Rybicki's testimony, reversal is required if either rationale reflects an abuse of discretion. As it turns out, both rationales are fatally flawed.

A. The district court imposed a "harmful dose" requirement that neither epidemiological practice nor this Court's precedents demand.

The district court's principal rationale for excluding Dr. Rybicki's general causation opinion was that he "did not identify a quantifiable level of exposure to [PAHs] capable of causing prostate cancer." D. Ct. Dkt. No. 168 (Summ. J. Op.) at 7. General causation, however, presents only the issue "whether a substance is capable of causing a particular injury." *Knight*, 482 F.3d at 351 (quoting *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 714 (Tex. 1997)). Where epidemiological studies demonstrate an observed association between a real-world exposure to the substance and human health, an expert can reliably resolve that issue in the affirmative, irrespective of whether some *other*, lower-level exposure might *not* trigger the health consequence at issue.

Questions of dose can be relevant to *specific* causation—that is, whether the substance at issue "caused a particular individual's injury."

Id. (quoting *Merrell Dow*, 953 S.W.2d at 714). After all, for a jury to find that a specific exposure caused a plaintiff's health condition, there must be a basis for concluding that exposure under the plaintiff's particular circumstances could have produced the condition. The distinct general-causation inquiry, though, asks whether *any* exposure level could have done so. *See* Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, *in Ref. Manual* 633 at 638 (contrasting specific causation, where "the primary issue will be whether there has been exposure to a sufficient dose to be a likely cause of th[e] effect," with general causation, where dose is "not … a central issue"). As Dr. Rybicki explained, individual variables such as one's age and genetic vulnerabilities can influence the likelihood that a particular exposure will have an adverse health effect. *See* Rybicki Rep. at 16–17; *see also id.* at 25 (explaining that Mr. Ruffin "is at significantly higher genetic risk for prostate cancer than the average man" and could be more susceptible to environmental hazards). It therefore makes little sense to speak at the general-causation stage of a single threshold dose that is per se sufficient to cause injury.

The district court's requirement that an expert identify a specific "quantifiable level" at which a particular substance can cause harm,

Summ. J. Op. at 7, moreover has no basis in epidemiological methodology. Whereas *toxicological* tests may permit quantitative judgments about what level of exposure is harmful when a substance is administered at carefully calibrated doses in a clinical setting, *see supra* at 8–9, *epidemiological* studies rely on observed associations between actual human exposures and an adverse health impact. The uncontrolled, real-world circumstances that are the subject of epidemiological study may permit the sort of *qualitative* inferences about harmful exposure levels that Dr. Rybicki made here. *See* Rybicki Rep. at 12 (explaining that the risk of prostate cancer tends to increase at "the highest quartile of the PAH exposure distribution" (formatting omitted)); D. Ct. Dkt. No. 104-3 (Rybicki Dep.) at 179 (testifying at deposition that "[m]edium to high exposure" to PAHs can be harmful). Those circumstances do not, however, permit the sort of *quantitative* inferences about dose that are the hallmark of toxicology.

Dr. Rybicki made this point repeatedly. When asked whether he had "identif[ied] the level of exposure to PAHs that are necessary to cause prostate cancer," Dr. Rybicki explained that he "would not try to do that" because he is "not a toxicologist." Rybicki Dep. at 164. He testified that

he would "rel[y] upon … toxicologists" to perform any sort of "quantitative exposure assessment with dose." *Id.* at 193. And he emphasized that "[d]ue to variation in human susceptibility and exposure conditions and the observational nature of epidemiologic studies, identifying a precise threshold 'dose' of an exposure to PAHs which can cause prostate cancer … is impossible." D. Ct. Dkt. No. 121-2 (Rybicki Decl.) at 2.

The district court at no point disputed that the identification of a quantitatively ascertainable "harmful dose" is not a component of standard epidemiological methodology and, therefore, is not necessary for a general-causation opinion based on that methodology to be reliable. Instead, it took the view that identification of a harmful dose is simply an established "Fifth Circuit requirement[]" for general causation. Summ. J. Op. at 7; *see also* Oral Arg. Tr. at 119 (opining that "binding Fifth Circuit precedent" requires "scientific knowledge of the harmful level of exposure"). The bulk of the cases the district court cited for this supposed requirement, however, are unpublished and create no binding precedent. *See* Oral Arg. Tr. at 111 (citing *Byrd v. BP Exploration & Prod., Inc.*, 2023 WL 4046280 (5th Cir. June 16, 2023) (per curiam); *McGill v. BP Exploration & Prod., Inc.*, 830 F. App'x 430 (5th Cir. 2020)

(per curiam); *Seaman v. Seacor Marine LLC*, 326 F. App'x 721 (5th Cir. 2009) (per curiam)); *see also Avelar-Oliva v. Barr*, 954 F.3d 757, 765 n.2 (5th Cir. 2020) (noting that "unpublished decisions are not precedent").

The sole precedential authority the district court cited for its "harmful dose" requirement was this Court's decision in *Allen v. Pennsylvania Engineering Corp.*, 102 F.3d 194 (5th Cir. 1996). The fault that this Court found with the expert testimony in the relevant portion of *Allen* was that, because the experts lacked "evidence of the level of [*the plaintiff's*] exposure" to a suspected carcinogen, they had an insufficient factual basis from which to draw a reliable conclusion as to *specific* causation. *Id.* at 198. It was in this context that the Court cited *Wright v. Willamette Industries, Inc.*, 91 F.3d 1105 (8th Cir. 1996), for the proposition that "[s]cientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff['s] burden in a toxic tort case." *Allen*, 102 F.3d at 199. Read in context, nothing about this statement suggests that an expert opinion on general causation is unreliable if it fails to pinpoint with quantitative precision the threshold at which exposure to a particular substance becomes harmful. Rather,

Allen echoes *Wright*'s modest observation that a finding of specific causation must rest on evidence that "the plaintiff was exposed to levels of [a substance] that are known to cause the kind of harm that the plaintiff claims to have suffered." *Wright*, 91 F.3d at 1107. Indeed, even in the specific-causation context, *Wright* refused to "require a mathematically precise table equating levels of exposure with levels of harm" and demanded no more than "evidence from which a reasonable person could conclude that a defendant's emission has probably caused a particular plaintiff the kind of harm of which he or she complains." *Id.*

By misreading *Allen*, the district court created a legal requirement that has no connection to general causation and no basis in the epidemiological methodology that Dr. Rybicki employed. This misconstruction of the governing law was an abuse of discretion.

B. The district court's unfounded contention that Dr. Rybicki should have given certain studies more weight than others was not a valid basis for deeming his general-causation opinion unreliable.

In rejecting Dr. Rybicki's general-causation opinion, the district court stated that "studies of petroleum workers—rather than the cited studies of non-petroleum workers, such as chimneysweeps and automotive workers—would be more representative of Ruffin's activity"

and noted that "at least one petroleum-related study found no association between petroleum-work exposure and prostate cancer." Summ. J. Op. at 7. The court's concerns were misplaced for multiple reasons.

To begin, the district court's analysis again conflated general and specific causation. Whether epidemiological studies that demonstrate an association between PAH exposure and prostate cancer address contexts that are sufficiently "representative of Ruffin's activity," *id.*, might bear on the specific-causation question whether *Mr. Ruffin's* exposure caused *Mr. Ruffin's* cancer. But a level of dissimilarity between the facts of those studies and the facts of this case does not undermine those studies' tendency to demonstrate for purposes of general causation that PAHs are capable of contributing to the risk of prostate cancer.

Moreover, the district court failed to consider whether Dr. Rybicki's reasons for placing primary emphasis on studies outside the petroleum-worker context were consistent with a reliable scientific methodology. As Dr. Rybicki explained, the petroleum-worker studies to which the district court referred were unable to "make a determination of PAH exposure" in the workers being studied and generally included workers who had "little if any direct contact with petroleum products," thus "diluting the

effects of any potential petroleum exposures across the study population." Rybicki Decl. at 6. Although a jury would be entitled to question Dr. Rybicki's reasons for treating some studies as more probative than others, the district court offered no basis for concluding that Dr. Rybicki's exercise of his judgment in this respect fell outside the scope of acceptable scientific practice. *See Viterbo v. Dow Chem. Co.*, 826 F.2d 420, 422 (5th Cir. 1987) ("[Q]uestions relating to the bases and sources of an expert's opinion affect the weight to be assigned that opinion rather than its admissibility and should be left for the jury's consideration.").

Similarly, the fact that "at least one" study failed to demonstrate an association between petroleum-industry work and prostate cancer, Summ. J. Op. at 7, says nothing about the reliability of Dr. Rybicki's methodology. Again, Dr. Rybicki explained why epidemiological studies in general and petroleum-worker studies in particular might fail to detect an association that nonetheless exists. *See* Rybicki Rep. at 12 (explaining that low-dose exposures might not produce an observable association); *id.* at 16–17 (explaining that failure to disaggregate subsets of individuals within an exposed population might mask associations); Rybicki Decl. at 6 (explaining that petroleum-worker studies often include unexposed

workers). The district court gave no reason why sound scientific methodology required Dr. Rybicki to treat "at least one" study's failure to demonstrate an association between PAHs and prostate cancer, Summ. J. Op. at 7, as being more significant than the fact that multiple studies—including at least one petroleum-industry study involving more than 350,000 workers—*did* reveal an association. *See* Rybicki Decl. at 7 (citing the Wong and Raabe petroleum-worker study); Rybicki Rep. at 11–12 (citing studies outside the petroleum-industry context); *cf. Allen*, 102 F.3d at 195 (affirming district court's exclusion of expert general-causation evidence where "*no* epidemiological study ha[d] found a statistically significant link" between a particular substance and a particular condition (emphasis added)). To the extent that other experts might have weighed the various studies differently, it is for a jury to decide which expert's assessment is most persuasive.

More fundamentally, the district court's disagreement with Dr. Rybicki's treatment of the epidemiological studies failed to account for the fact that those studies formed but one part of his overall causation analysis. Importantly, the district court did not hold that the studies on which Dr. Rybicki relied failed to support his conclusion on general

causation; the court questioned only the *degree* of support that those studies provided. *See* Oral Arg. Tr. at 31 (accepting that Dr. Rybicki's studies doubtless showed a link between PAHs and prostate cancer). But the district court nowhere considered the wealth of other scientific evidence reinforcing the causal inference that Dr. Rybicki drew from the studies. Dr. Rybicki acknowledged that the epidemiological support for an association between PAHs and prostate cancer was "modest," but he felt secure opining on the likelihood of a causal relationship given the "strong[] support[]" that "molecular and laboratory findings" provided for such a relationship. Rybicki Rep. at 16–17. As explained above, *supra* at 13–15, Dr. Rybicki described studies demonstrating PAHs' cellular effects on humans and animals and tracing the biological pathways through which those effects increase the risk of prostate cancer. The "well established" biomechanical evidence that "PAH exposures can result in carcinogenic DNA adducts in humans," then, was a crucial foundation for Dr. Rybicki's analysis of the epidemiological studies. Rybicki Rep. 18.

The district court ignored this vital context. And it did so despite Dr. Rybicki's repeated testimony that reliable scientific practice required taking a holistic view. *See, e.g.*, Rybicki Dep. at 103 ("I think most

scientists would want to have some biologic mechanistic studies that would support the epidemiologic evidence, and they all work together in tandem."); *id.* at 145 (declining to "use" a single study "by itself"); *id.* at 157 (emphasizing that the epidemiological evidence of causation in this case is "not overwhelming" but that "there's also the molecular laboratory evidence"); *id.* at 184 (agreeing that "no epidemiologist should rely on a single study").

By wresting Dr. Rybicki's epidemiological evidence free from the remainder of his analysis and deeming that evidence alone insufficiently compelling to support a reliable causal inference, the district court flouted the standard epidemiological principle that assessing causation requires "judgment about how the [relevant] study findings fit with other scientific knowledge." *Ref. Guide Epidem.* at 553. This deviation from scientific norms was an abuse of discretion that, like the district court's imposition of an unjustifiable "harmful dose" requirement, creates an independent basis for reversal.

CONCLUSION

This Court should reverse the district court's ruling that Dr. Rybicki's opinion on general causation is inadmissible.

Respectfully submitted,

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CERTIFICATE OF COMPLIANCE

This brief complies with the type-volume limitation of Federal Rules of Appellate Procedure 29(a)(5) and 32(a)(7)(B)(i) because, excluding the parts of the brief exempted by Federal Rule of Appellate Procedure 32(f) and the Rules of this Court, it contains 5,571 words.

This brief also complies with the typeface and type-style requirements of Federal Rules of Appellate Procedure 29(a)(4), 32(a)(5), and 32(a)(6) because it has been prepared in a proportionally spaced typeface using Microsoft Word in 14-point Century Schoolbook.

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CERTIFICATE OF SERVICE

I hereby certify that I electronically filed the foregoing Brief of Amicus Curiae Public Citizen with the Clerk of the Court for the United States Court of Appeals for the Fifth Circuit on April 10, 2024, using the Appellate Electronic Filing system. I certify that all participants in this case are registered CM/ECF users and that service will be accomplished by the CM/ECF system.

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