

United States Court of Appeals
for the Fifth Circuit

United States Court of Appeals
Fifth Circuit

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Lyle W. Cayce
Clerk

No. 23-30854

FLOYD RUFFIN,

Plaintiff—Appellant,

versus

BP EXPLORATION & PRODUCTION, INCORPORATED; BP
AMERICA PRODUCTION COMPANY,

Defendants—Appellees.

Appeal from the United States District Court
for the Eastern District of Louisiana
USDC Nos. 2:20-CV-334, 2:22-CV-1006

Before ELROD, *Chief Judge*, and HIGGINBOTHAM and SOUTHWICK,
Circuit Judges.

JENNIFER WALKER ELROD, *Chief Judge*:

This toxic-tort case arising from the 2010 *Deepwater Horizon* oil spill presents the issue of what expert testimony must be introduced to establish causation. Plaintiff–Appellant Floyd Ruffin alleges that he was exposed to crude oil while employed as a clean-up worker after the spill. He was later diagnosed with prostate cancer and sued BP. The issue is whether the expert testimony that Ruffin submitted to prove that his alleged exposure caused his cancer was sufficiently “relevant” and “reliable” to be admissible under

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Federal Rule of Evidence 702 and *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993). BP says it was not, arguing for a rule that would require an expert to testify to the specific quantitative amount or “dose” of chemical exposure that would cause the plaintiff’s injury. We decline to adopt such a rule because it conflicts with our precedent. But because we agree with the district court that Ruffin’s expert testimony nevertheless suffers from fatal analytical flaws, we AFFIRM the district court’s exclusion of the testimony and its associated award of summary judgment to BP.

I

Ruffin worked as a shoreline clean-up worker in Louisiana for five months following the 2010 *Deepwater Horizon* oil spill. Five years later, he was diagnosed with prostate cancer. Ruffin sued BP in the Eastern District of Louisiana pursuant to the *Deepwater Horizon* medical-benefits class action settlement as a “Back-End Litigation Option” (BELO) claim for alleged injuries that manifest after the date of the settlement. Ruffin claimed that he was exposed to harmful chemicals that caused his cancer while working on the clean-up effort.

After discovery, Ruffin designated several experts. At issue here is Ruffin’s causation expert, Dr. Benjamin Rybicki, who is a genetic and molecular epidemiologist. Rybicki reported that Ruffin was exposed to “polycyclic aromatic hydrocarbons” (PAHs)—chemical compounds that are “ubiquitous in the environment” and occur “in coal, peat, crude oil, and shale oils”—and that at least one of these compounds can cause prostate cancer. Rybicki pointed specifically to a compound called “benzo(a)pyrene,” the “most prevalent PAH,” and reported that it causes cancer in humans. Relying on animal studies and occupational studies, Rybicki concluded that occupational exposure to PAHs is associated with a “modest” 1.5- to 2-fold increase in one’s risk of prostate cancer.

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Rybicki applied his research to Ruffin's case through a differential etiology—a process-of-elimination approach to determining the cause of Ruffin's prostate cancer. Rybicki first identified that Ruffin had a genetic risk for prostate cancer. Then, Rybicki ruled out possible exposure during Ruffin's prior twenty-year history as a truck driver. While Rybicki noted that exposure to diesel exhaust fumes (such as from large trucks) was reported to result in PAH exposure, the association is “modest at best.” Instead, Rybicki concluded that Ruffin was exposed to PAHs during his oil-spill clean-up work. Rybicki identified two instances of such exposure: when oil splashed onto Ruffin's body, face, mouth, and eyes while he was traveling by boat and when Ruffin once “fell face-first into the water while trying to retrieve a used, oil-saturated boom.” Ruffin reportedly “smelled strong fumes akin to diesel or petroleum” while working that “caused him dizziness and headaches” and constantly “cough[ed] up black soot.” While Ruffin's genetic background was his first “risk hit,” Rybicki concluded that “his oil spill exposures were the second and necessary hit to initiate his prostate cancer.”

When BP deposed Rybicki, he acknowledged that he did not identify a specific level of PAH exposure that is capable of causing prostate cancer in a human. Rybicki also clarified that benzo(a)pyrene is “probably the only” PAH that is carcinogenic. Rybicki acknowledged that he did not specifically state that Ruffin was exposed to PAHs or benzo(a)pyrene and that his testimony was limited to crude-oil exposure. Nevertheless, Rybicki emphasized that PAHs were present in oil and reiterated that Ruffin's exposure to oil was “his most significant exposure [to PAHs] in terms of intensity.”

BP filed a *Daubert* motion to exclude Rybicki's testimony under Federal Rule of Evidence 702. After a hearing, the district court granted the motion. The court explained that Rybicki's testimony was inadmissible

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because it neither “identif[ied] the harmful level of exposure to a chemical” necessary to cause prostate cancer nor proved that Ruffin was “exposed to” that harmful level. The court also concluded that there was “an analytical gap . . . between the data and the opinions proffered” by Rybicki because his testimony suffered from several methodological flaws.

With Ruffin’s expert testimony excluded, the court determined that Ruffin lacked the evidence needed to satisfy the causation element of his claim and granted BP’s motion for summary judgment. Ruffin timely appealed.

II

A district court’s exclusion of expert testimony is reviewed for abuse of discretion and is not reversible unless it “is manifestly erroneous.” *Guy v. Crown Equip. Corp.*, 394 F.3d 320, 325 (5th Cir. 2004) (emphasis omitted). We review a district court’s grant of summary judgment *de novo*. *Rogers v. Bromac Title Servs., L.L.C.*, 755 F.3d 347, 350 (5th Cir. 2014).

III

Under Federal Rule of Evidence 702 and *Daubert*, expert testimony is admissible if “the reasoning or methodology underlying the [expert’s] testimony is scientifically valid” and can “properly . . . be applied to the facts in issue.” *Daubert*, 509 U.S. at 592–93. “In short, expert testimony is admissible only if it is both relevant and reliable.” *Pipitone v. Biomatrix, Inc.*, 288 F.3d 239, 244 (5th Cir. 2002). BP argues that Rybicki’s testimony was neither relevant nor reliable under *Daubert* and, therefore, that the district court properly excluded the testimony and granted summary judgment for BP.

To establish a defendant’s liability for a toxic tort, as with any tort, a plaintiff must prove causation. Specifically, for BELO claims like Ruffin’s,

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the medical-benefits class action settlement requires the plaintiff to “prove that the legal cause of the claimed injury or illness is exposure to oil or other chemicals used during the response.” *In re Oil Spill*, No. MDL 2179, 2021 WL 6053613, at *11 (E.D. La. Apr. 1, 2021). As both parties agree, Ruffin must establish both general causation and specific causation. “General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual’s injury.” *Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 351 (5th Cir. 2007) (quoting *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 714 (Tex. 1997));¹ accord Michael D. Green, D. Michal Freedman, & Leon Gordis, *Reference Guide on Epidemiology*, in Fed. Jud. Ctr., *Reference Manual on Scientific Evidence* 549, 623, 627 (3d ed. 2011).

¹ As a technical matter, the causation standard must be supplied by the applicable state or federal law. See *Newsome v. Int’l Paper Co.*, 123 F.4th 754, 761 & n.5 (5th Cir. 2024). BP applies the general-/specific-causation standard we described in *Knight v. Kirby Inland Marine Inc.*, 482 F.3d 347, 351 (5th Cir. 2007), which several of our unpublished cases concerning *Deepwater Horizon* tort claims have also applied. See, e.g., *Wunstell v. BP, P.L.C.*, No. 23-30859, 2024 WL 4100496, at *2 (5th Cir. Sept. 6, 2024) (citing *Knight*). However, *Knight*’s general-causation standard is drawn from Texas law, which neither party suggests applies here. *Id.* (citing *Havner*, 953 S.W.2d at 714); *Newsome*, 123 F.4th at 761. The medical-benefits settlement provides that it “shall be interpreted in accordance with General Maritime Law” while excepting disputed issues of “law applicable to the underlying claims.” Ruffin also characterizes his claims as arising under general maritime law. However, assuming *arguendo* that Ruffin is correct, we have still previously required general and specific causation for toxic-tort claims arising under general maritime law, and we see no reason to depart from that here. See *Seaman v. Seacor Marine LLC*, 326 F. App’x 721, 724 (5th Cir. 2009). Indeed, even though the *Knight* rule derives from state law, we nevertheless applied it there to a claim that arose under the Jones Act. *Knight*, 482 F.3d at 350; see also *Clark v. Kellogg Brown & Root L.L.C.*, 414 F. App’x 623, 624, 627 (5th Cir. 2011). We thus apply *Knight* “out of an abundance of deference to our Rule of Orderliness.” *Cuenca-Arroyo v. Garland*, 123 F.4th 781, 786 n.1 (5th Cir. 2024) (Elrod, C.J., concurring).

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We have adopted “a two-step process in examining the admissibility of causation evidence in toxic tort cases.” *Knight*, 482 F.3d at 351. “First, the district court must determine whether there is general causation.” *Id.* “Second, if it concludes that there is admissible general-causation evidence, the district court must determine whether there is admissible specific-causation evidence.” *Id.* The district court held that Rybicki did not provide admissible testimony for either. But because we agree that Rybicki’s testimony is inadmissible to demonstrate general causation, we need not address Ruffin’s specific-causation argument. *Id.*

A

BP principally contends that Rybicki’s testimony is inadmissible because it cannot support the general-causation element of Ruffin’s tort claim. To establish general causation, Ruffin must show, by expert testimony, that the chemicals he was exposed to are “capable of causing [his] particular injury or condition in the general population.” *Knight*, 482 F.3d at 351.²

1

BP argues that, to be admissible, an expert must “identify the minimum amount of a particular chemical necessary to cause a plaintiff’s alleged condition in the general population.” BP thus concludes that the district court correctly excluded Rybicki’s testimony on the basis that he

² Ruffin argues for an alternative “featherweight” causation standard or a more “lenient standard in the interest of equity.” But we have only applied the “featherweight” burden to Jones Act cases. *See Gautreaux v. Scurlock Marine, Inc.*, 107 F.3d 331, 335 (5th Cir. 1997) (*en banc*). “The standard for negligence under general maritime law is higher.” *In re Cooper/T. Smith*, 929 F.2d 1073, 1077 (5th Cir. 1991). Regardless, any “reduced burden” for causation would be “irrelevant” to whether evidence is admissible under Rule 702 and *Daubert*. *Seaman v. Seacor Marine LLC*, 326 F. App’x 721, 728 n.41 (5th Cir. 2009).

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“did not identify a quantifiable level of exposure to [PAHs] capable of causing prostate cancer.”

BP draws this rule from several of our unpublished cases addressing *Deepwater Horizon* toxic-exposure claims. Those cases require “the expert [to] ‘determine not only whether a chemical can cause certain health effects, but also at what level of exposure those health effects appear’” in order to establish general causation. *Wunstell v. BP, P.L.C.*, No. 23-30859, 2024 WL 4100496, at *2 (5th Cir. Sept. 6, 2024) (quoting *Braggs v. BP Expl. & Prod., Inc.*, No. 23-30297, 2024 WL 863356, at *2 (5th Cir. Feb. 29, 2024)); *see also Braggs*, 2024 WL 863356, at *2; *Smith v. BP Expl. & Prod., Inc.*, No. 23-30619, 2024 WL 3842571, at *2 (5th Cir. Aug. 16, 2024); *Barrington v. BP Expl. & Prod., Inc.*, No. 23-30343, 2024 WL 400191, at *2 (5th Cir. Feb. 2, 2024); *Prest v. BP Expl. & Prod., Inc.*, No. 22-30779, 2023 WL 6518116, at *2 (5th Cir. Oct. 5, 2023); *Byrd v. BP Expl. & Prod., Inc.*, No. 22-30654, 2023 WL 4046280, at *2 (5th Cir. June 16, 2023). In two of these cases, we characterized the excluded testimony as failing to identify the “necessary dose” of a chemical that could cause the relevant health condition. *Barrington*, 2024 WL 400191, at *2; *Wunstell*, 2024 WL 4100496, at *3.

While these cases lack precedential authority because they are unpublished, they each rely on our decision in *Allen v. Pennsylvania Engineering Corp.*, which described “[s]cientific knowledge of the harmful level of exposure to a chemical” as a “minimal fact[] necessary to sustain the plaintiffs’ burden in a toxic tort case.” 102 F.3d 194, 199 (5th Cir. 1996). BP presses that these unpublished cases demonstrate that *Allen* establishes a rule that expert testimony must identify the minimum amount or “dose” of the chemical necessary to cause the relevant injury.

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2

We disagree with BP's conclusion for at least two reasons. First, *Allen*'s comment about "the harmful level of exposure to a chemical" was directed at what is "necessary to sustain the plaintiffs' *burden*," not what is necessary to admit expert testimony. *Allen*, 102 F.3d at 199 (emphasis added). "[T]he Federal Rules of Evidence and the applicable standard of causation are distinct issues and do not affect one another." *Seaman v. Seacor Marine LLC*, 326 F. App'x 721, 728 n.41 (5th Cir. 2009) (internal quotation marks omitted). *Daubert* and Rule 702 control whether evidence is admissible, and they each provide that evidence is admissible if, among other things, it is "relevant" to the applicable legal element. *Daubert*, 509 U.S. at 591, 597. Here, that element is general causation, which requires only that a plaintiff establish that a substance is "capable of causing" the relevant condition in the general population. *Knight*, 482 F.3d at 351.³ Although a specific quantitative "dose" that causes a condition might be sufficient to establish general causation, *Knight* does not require it. Expert testimony may thus be relevant to general causation and therefore admissible under Rule 702 and *Daubert* even if it does not provide a specific, quantitative dosage.

Second, even if *Allen* were construed to require more to establish general causation than *Knight*, *Allen*'s language does not require an expert to provide a specific, quantitative exposure dose. *Allen* characterized the minimum showing as "the harmful *level* of exposure to a chemical." 102 F.3d at 199 (emphasis added). For this proposition, *Allen* cited the Eighth

³ *Allen* also does not conflict with *Knight*'s general-causation standard because *Allen* affirmed the exclusion of expert testimony as unreliable under Federal Rule of Evidence 703, not as irrelevant to general causation. See *Allen*, 102 F.3d at 198–99; see also *Clark v. Kellogg Brown & Root L.L.C.*, 414 F. App'x 623, 629 (5th Cir. 2011) (characterizing *Allen* as stating an "uncontroversial principle" of causation).

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Circuit’s decision in *Wright v. Willamette Industries, Inc.*, which rejected “a mathematically precise table equating levels of exposure with levels of harm” and instead defined the standard as “evidence from which a reasonable person could conclude” that the chemical caused the condition. *Wright*, 91 F.3d 1105, 1107 (8th Cir. 1996).

In another unpublished case applying *Allen, Clark v. Kellogg Brown & Root L.L.C.*, we held that expert testimony that (1) attests that a chemical was “a known general cause of” the relevant condition, (2) is supported by relevant literature, and (3) demonstrates that exposure to the chemical in certain amounts resulted in “statistically significant” increases in risk was admissible even though the testimony did not “quantify precisely the dosage of [chemical] that is hazardous.”⁴ 414 F. App’x 623, 628–29 (5th Cir. 2011); *see also* Bernard D. Goldstein & Mary Sue Heflin, *Reference Guide on Toxicology*, in *Reference Manual on Scientific Evidence*, *supra*, at 633, 638 (contrasting general causation, in which “dose” is “not . . . a central issue,” with specific causation, in which “the primary issue [is] whether there has been exposure to a sufficient dose to be a likely cause of [the condition]”). Moreover, the “Bradford Hill criteria,” commonly used for evaluating causation in epidemiological studies—which we have said are “the most useful and conclusive type of evidence” in toxic tort cases⁵—consider a “[d]ose-response relationship” as but one factor among others such as “[t]emporal relationship,” “[b]iological plausibility,” “[s]pecificity of the

⁴ *Prest*, one of the unpublished cases we cited above, discounted *Clark*’s reasoning because, by its characterization, *Clark* only assessed specific causation. *Prest*, 2023 WL 6518116, at *3 n.5. But *Clark* emphasized that the expert testimony at issue there did “meet [*Allen*’s] burden” of providing “the harmful level of exposure to a chemical.” *Clark*, 414 F. App’x at 629.

⁵ *Brock v. Merrell Dow Pharms., Inc.*, 874 F.2d 307, 311 (5th Cir. 1989), *modified on reh’g*, 884 F.2d 166 (5th Cir. 1989).

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association,” and “[c]onsistency with other knowledge.” *Green et al., supra*, at 599–600, 599 n.141. And even when an expert does provide a quantitative-dosage estimate, we have allowed a numerical “range” supported by other, non-dosage associations, such as the chemical’s “toxicological profile” and the “temporal connection” between workplace exposure to a chemical and the incidence of a condition. *Curtis v. M&S Petroleum, Inc.*, 174 F.3d 661, 669–70, 670 n.8 (5th Cir. 1999).

* * *

Simply put, our general-causation-standard caselaw is inconsistent with requiring a quantitative dosage for expert testimony to be relevant under Rule 702 or *Daubert*. It does not require that an expert process all of the pertinent facts, qualitative and quantitative alike, into an ultimate numerical output like “FORTY-TWO.” *See* Douglas Adams, *The Hitchhiker’s Guide to the Galaxy* 303 (1986). Assuming that the underlying methods are reliable, our cases illustrate that an expert can provide a harmful “level” of exposure to a chemical that is relevant to general causation by, for example, establishing a significant association between occupational exposure and the relevant condition or by providing qualitative examples of exposure, such as ingestion, that are generally known to cause the relevant condition. *Curtis*, 174 F.3d at 669–70; *Clark*, 414 F. App’x at 628–29. Indeed, such real-world exposures can be relevant to general causation precisely because they may be more typical of how the “general population” is exposed to the chemical than a quantitatively precise laboratory dosage can provide. *Knight*, 482 F.3d at 351.

3

Applying these principles here, Rybicki’s testimony is not irrelevant on the basis that it fails to provide a quantitative exposure dosage of PAHs that would cause prostate cancer. Ruffin claims that exposure to PAHs

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caused his prostate cancer. Rybicki, in turn, reported that PAHs are capable of causing prostate cancer in humans. *See Knight*, 482 F.3d at 351. Though his testimony was not entirely clear, Rybicki surveyed toxicological studies and found an increased cancer risk per incidence of oral lifetime exposure to a PAH mixture. Rybicki also surveyed studies that show an association between increased risk of prostate cancer and exposure to PAHs in various occupational settings and compared them to the reduced risk of prostate cancer from PAH exposure in non-occupational settings. While Rybicki did not provide a quantitative “dosage” of exposure to PAHs that would cause cancer, he did purport to show that PAHs were capable of causing prostate cancer and that this exposure led to increased levels of risk. Not only so, he examined the risks at different “level[s] of exposure,” such as between occupational and non-occupational exposure. *Allen*, 102 F.3d at 199.⁶ Assuming these representations were otherwise reliable and relevant (an assumption to which we turn next), they were consistent with our requirements for general causation, and Rybicki’s testimony is not inadmissible *per se*.

B

Though we decline to adopt BP’s proposed quantitative-dose rule, we nevertheless agree that Ruffin has failed to establish general causation. Under *Daubert*, “[a] court may conclude that there is simply too great an analytical gap between the data and the opinion proffered” to admit an expert’s testimony. *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997). Rybicki’s testimony suffers from several.

⁶ Citing Rybicki’s deposition, both BP and the district court report that Rybicki “admitted that [benzo(a)pyrene] has not been shown to cause . . . prostate cancer,” but the referenced comment only recognized that the International Agency for Research on Cancer does not label it a carcinogen for prostate cancer.

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First, Rybicki's assertion that PAHs can cause prostate cancer is not supported for a basic reason: it is not what Rybicki's own analysis shows. To establish general causation, "the expert must . . . demonstrate that the chemical at issue is actually capable of" causing the plaintiff's condition in the general population. *Johnson v. Arkema, Inc.*, 685 F.3d 452, 469 (5th Cir. 2012). Importantly, the chemical at issue is the chemical that the plaintiff was exposed to. *Knight*, 482 F.3d at 355. Ruffin's complaint alleges that he was exposed to PAHs. However, while Rybicki purported to testify about PAHs, his testimony that PAHs cause prostate cancer was actually limited to only one kind of PAH, benzo(a)pyrene, which neither he nor Ruffin claim that Ruffin was exposed to.

Rybicki explained that PAHs comprise over 200 chemicals that are "ubiquitous in the environment." However, as Rybicki admitted, benzo(a)pyrene is "probably the only" PAH chemical that has been confirmed to be carcinogenic. Accordingly, Rybicki's analysis principally focused on the carcinogenic effects of benzo(a)pyrene rather than PAHs generally. For example, take Rybicki's testimony about a cancer toxicology study showing an increased risk of prostate cancer associated with oral PAH exposure. While Rybicki described the study as establishing a connection between "PAH exposure" and cancer, the study specifically found an excess risk of cancer from oral exposure per one milligram of benzo(a)pyrene.

Critically, neither Rybicki nor Ruffin claimed that benzo(a)pyrene was the specific chemical that Ruffin was exposed to. *See Knight*, 482 F.3d at 355. Ruffin's complaint attests only to the fact that "crude oil . . . contains . . . PAHs" and claims that his prostate cancer was caused "by exposure to oil, dispersants and other harmful chemicals." Rybicki similarly claims that "Ruffin was most likely exposed to PAHs." However, as Rybicki's testimony explained, PAHs can include any of over 200 chemicals, only one of which is benzo(a)pyrene. To be sure, Rybicki did report that

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benzo(a)pyrene is “generally the most prevalent PAH” chemical.⁷ But he also admitted that the chemicals present in a PAH mixture “vary depending on what the medium is.” The specific PAH chemicals present in a mixture, and their levels, vary when comparing samples from similar oil spills—even between samples from the same oil spill. Rybicki equivocates as to whether Ruffin was exposed to benzo(a)pyrene specifically even though, logically, it is the only relevant agent that could have even potentially caused his cancer according to Rybicki’s testimony.

Moreover, even if benzo(a)pyrene were the chemical at issue, Rybicki’s testimony falls short on another front. For general causation, the chemical must be “capable of causing a particular injury” (*i.e.*, the plaintiff’s injury) in the general population. *Knight*, 482 F.3d at 351. As the district court aptly explained, this means that the expert must “establish a link with the specific cancer in which the plaintiff suffers and not cancer generally.” While Rybicki’s testimony does link benzo(a)pyrene to cancer, his connection of it to prostate cancer, Ruffin’s condition, is lacking. Rybicki claims that benzo(a)pyrene is a “human carcinogen” and causes the development of unidentified “tumors” and “[s]kin papillomas” in animals. However, that does not demonstrate that it causes prostate cancer. *See Allen*, 102 F.3d at 196 (“[T]he fact that [a chemical] has been classified as a carcinogen . . . is not probative of the question whether [the plaintiff’s] cancer

⁷ Though it was to support his claim that Ruffin was exposed to PAHs, Rybicki pointed to a study providing circumstantial evidence that, a year after the *Deepwater Horizon* spill, there were “increased tissue levels” of “benzo(a)pyrene mutagenic equivalents” in “Gulf menhaden fish.” Yet he provided no explanation of how this evidence might support Ruffin’s exposure to benzo(a)pyrene, such as by comparing the exposures’ proximity in time, amount of chemical, method of exposure, or other factors. *See, e.g., Green et al., supra*, at 599–600 (discussing various factors that guide epidemiological studies in assessing causation between exposure to an agent and the development of a disease).

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was caused by [the chemical].”). Rybicki’s support for an association with prostate cancer again relies on studies of PAHs generally, rather than benzo(a)pyrene specifically.⁸

Ruffin and Rybicki repeatedly make PAHs the “chemical at issue,” but Rybicki’s testimony does not support that PAHs cause cancer in the general population. The one PAH chemical that he identifies as a human carcinogen, benzo(a)pyrene, lacks a strong association with prostate cancer. Rybicki’s testimony is thus irrelevant to establishing general causation and was properly excluded by the district court. *A fortiori*, that exclusion was not manifestly erroneous, so we cannot sustain Ruffin’s first point of error.

IV

Without Rybicki’s testimony, Ruffin cannot demonstrate general causation.⁹ Summary judgment is required when there is no genuine issue of material fact and the moving party is entitled to judgment as a matter of law.

⁸ Though it was to support his claims about “PAH and Prostate cancer,” Rybicki did reference a study that found that “administer[ing] benzo(a)pyrene to mice through gavage for 9 months” led to “prostate mutagenesis.” We have “noted ‘the very limited usefulness of animal studies when confronted with questions of toxicity’” and the need for “careful[ly]” analysis to demonstrate their “explanatory potential for human beings.” *Allen*, 102 F.3d at 197 (quoting *Brock*, 874 F.2d at 313). Once again, Rybicki did not elaborate or connect that study to the human “general population” relevant to general causation. *Knight*, 482 F.3d at 355.

⁹ After the district court excluded Rybicki’s testimony, Ruffin proffered an alternate causation expert, Dr. James Clark. Clark reported that PAHs were toxic based on EPA methodologies and could cause “various cancers” after “[l]ong-term, or chronic, exposure.” The district court did not exclude Clark’s testimony but held it unresponsive because (1) it did not evaluate a connection between PAHs in oil and prostate cancer (general causation) and (2) Clark conceded that he did not offer a specific-causation opinion. Here, Ruffin does not brief Clark’s findings except by stating that they “confirmed” Rybicki’s findings. Because Ruffin does not address the district court’s holding that Clark’s testimony was inapplicable, he has forfeited that issue. *Rollins v. Home Depot USA*, 8 F.4th 393, 397 (5th Cir. 2021).

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Fed. R. Civ. P. 56(a). The “complete failure of proof concerning an essential element of the nonmoving party’s case necessarily renders all other facts immaterial.” *Celotex Corp. v. Catrett*, 477 U.S. 317, 323 (1986). The district court thus properly granted BP’s motion for summary judgment. *See, e.g., Barrington*, 2024 WL 400191, at *2 (“Given that [the plaintiff’s] general causation expert’s opinion was properly excluded, summary judgment was therefore due.”).

V

For the reasons stated above, we AFFIRM the district court’s exclusion of Rybicki’s testimony and award of summary judgment to BP.