

In the  
**United States Court of Appeals**  
**For the Seventh Circuit**

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No. 12-1902

JOANN EVELYN SCHULTZ, Individually and as  
Personal Representative of the Estate of  
Donald Walter Schultz,

*Plaintiff-Appellant,*

*v.*

AKZO NOBEL PAINTS, LLC, *et al.*,

*Defendants-Appellees.*

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Appeal from the United States District Court  
for the Eastern District of Wisconsin.  
No. 08-C-919—**Rudolph T. Randa**, *Judge*.

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ARGUED OCTOBER 30, 2012—DECIDED JUNE 26, 2013

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Before BAUER, FLAUM, and WOOD, *Circuit Judges*.

WOOD, *Circuit Judge*. Between 1981 and 1989, Donald Schultz worked as a painter for American Motors Corporation (which was acquired by Chrysler in 1987). Schultz's job was to paint equipment, floors, walls, ceilings, and pipes at company plants. In November 2005 he was diagnosed with acute myeloid leukemia (AML), a

disease that claimed his life in September 2006. Joann Schultz, his wife, acting on her own behalf and as the representative of her late husband's estate, sued Akzo Nobel Paints (formerly known as The Glidden Company, but we will refer to it under its current name, Akzo) and Durako Paint and Color Corp., alleging that these companies produced or distributed the paint Schultz used while working at Chrysler and that benzene from these paints caused his AML. Schultz offered reports from two experts to support his causation theory: Dr. Stewart, an industrial hygienist, who reconstructed Schultz's work with the paints in order to quantify his benzene exposure; and Dr. Gore, an oncologist, who testified that benzene is both generally known to cause AML and specifically was a substantial factor in the development of Schultz's disease.

The district court granted Akzo's motion for summary judgment on the ground that Dr. Gore's testimony was scientifically unreliable; without that crucial evidence, Schultz had no way of linking his disease to Akzo's paints. At the same time, the court granted Durako's motion for summary judgment. Schultz appeals both of these rulings. Because we find that the district court erred in excluding Dr. Gore's testimony, we reverse the grant of Akzo's motion for summary judgment. We affirm the judgment in favor of Durako, however, because of a lack of evidence indicating that Schultz was exposed to a Durako product.

**I**

Because this case turns for the most part on the rules governing expert witnesses, we will not dwell on Schultz's experience with Akzo, details about his medical history, or the source of the benzene, except insofar as these points bear on the issue before us. We turn instead directly to the two expert reports that Schultz proffered in an effort to avoid summary judgment.

Dr. Stewart reconstructed Schultz's quantitative exposure to benzene using Monte Carlo Analysis, a risk assessment model that accounts for variability and uncertainty in risk factors such as the likely variation in Schultz's exposure to benzene during different periods and at different plants. The U.S. Environmental Protection Agency (EPA) has endorsed this methodology as a reliable way to evaluate risk arising from environmental exposure. EPA, Office of the Scientific Advisor, *Guiding Principles for Monte Carlo Analysis*, <http://www.epa.gov/raf/publications/guiding-monte-carlo-analysis.htm> (last visited June 21, 2013) (noting "the EPA's position that such probabilistic analysis techniques as Monte Carlo analysis, given adequate supporting data and credible assumptions, can be viable statistical tools for analyzing variability and uncertainty in risk assessments."). Dr. Stewart interviewed Schultz's former co-workers and reviewed their deposition testimony to evaluate the extent of Schultz's exposure to Akzo paint. He then derived the chemical composition of the paints from material safety data sheets that Akzo had produced. He entered this data into the Monte Carlo model in order

to reconstruct Schultz's total benzene exposure. After twice revising his report to account for new information about the amount of paint used each day and to correct a typo in one of the numbers in the model, Dr. Stewart concluded that Schultz had been exposed on the job to a total of 24 parts-per-million years (ppm-years) of benzene. (This is equivalent to being exposed to 1 ppm of benzene each year for 24 years.)

In order to show that this degree of exposure was, as a scientific matter, a substantial factor in the development of Schultz's AML, Schultz presented Dr. Gore's report. Dr. Gore is both a practicing oncologist and a Professor of Oncology at the Comprehensive Cancer Center at Johns Hopkins University. He has been on the Johns Hopkins Medical School faculty since 1990; before that, he spent three years as a Senior Clinical Fellow in Oncology at Johns Hopkins University School of Medicine. He received a Master's degree in pharmacology and a M.D. from Yale University, and he has published more than 75 articles, most relating to the biology and treatment of leukemias, lymphomas, and other diseases of the blood. Dr. Gore explained that as part of his "day job" as a clinical oncologist, he diagnoses and treats dozens of leukemia patients every year, many with AML. Dr. Gore's standard diagnostic practice is to take an extensive history from each patient, reviewing his occupation, family history, lifestyle, and other life activities that may have led to exposures to chemicals or environmental risk factors, in order to assess whether any factors can be identified that might have con-

tributed to the patient's disease. He has used this process, called differential diagnosis, to assess the causes contributing to the diseases of several hundred AML patients. Dr. Gore explained that oncologists regularly rely on differential diagnosis to identify causal factors in order to treat patients, because a patient whose leukemia was caused by exposure to a known chemical is treated differently from one whose leukemia arose from an unknown cause. Dr. Gore's report concluded that Schultz's history of smoking and exposure to benzene were both significant causes of Schultz's AML.

In his deposition, Dr. Gore explained:

[W]orkers who had greater than eight to sixteen per million years exposure to Benzene, are estimated to be [*sic*] a six-fold increase of leukemia, compared to people who don't. And, if it is greater than sixteen parts per million years, the relative risk was a hundred-fold. So, either way with these estimates, Mr. Schultz was well within these diagnosis risk exposures. And, it's my understanding that Dr. Stewart was only estimating the risk from six years of exposure. And, in fact, the gentleman worked in these plants for considerably longer than that. So minimally, we think he is exposed to a very toxic and dangerous level within six years of exposure. And, that's ignoring the other years of painting that he did.

At a different point, Dr. Gore testified that "[s]ix or less parts per million year exposure, greater than fifteen years, one can argue that they don't seem to be at in-

creased risk epidemiologically. But, those with—it looks like eleven parts per million years, do.” Finally, Dr. Gore opined that Schultz’s smoking history probably also contributed to his AML, but he found no evidence that any other risk factor played a role. He explained that “[t]he fact that Mr. Schultz’s cigarette smoking may have contributed to his AML in no way undermines my conclusion that his benzene exposure played a substantial role in the development of the disease.”

To refute Dr. Gore’s conclusion that Schultz’s AML was caused in part by benzene exposure, Akzo introduced a report of its own expert toxicologist, David Pyatt, who concluded that benzene exposure was unlikely to have contributed to Schultz’s AML. To support this conclusion, Pyatt cited a study finding that only workers exposed to greater than 40 ppm-years benzene were at a higher risk for developing AML. Pyatt also stated that the risk of developing AML decreases as time passes following exposure to benzene. Based on this opinion, Akzo argued that Schultz’s AML was not likely to have been caused by exposure to benzene: his exposure was less than 40 ppm-years, and there was a 15-year latency period between Schultz’s exposure to benzene and the time he developed AML.

In his deposition, Dr. Gore responded to Pyatt’s assertions. He explained that the hypothesis of a 40 ppm-year threshold originated in a study based on just nine cases of leukemia, only six of which were AML. Dr. Gore pointed to a larger study, Hayes *et al.*, “Benzene and the Dose-Related Incidence of Hematologic Neoplasms in

China,” which was introduced as an exhibit to his deposition. In the Chinese study, which involved more than 30 cases, leukemogenic effects were observed at exposures well below 40 ppm-years. The authors concluded that “[r]isks . . . are elevated at average benzene-exposure levels of less than 10 ppm and show a tendency, although not a strong one, to rise with increasing levels of exposure.” He mentioned other literature as well that was consistent with the Chinese study.

In addition to this specific testimony supporting a threshold of approximately 10 ppm-years (well below the 24 ppm-years to which Shultz was exposed), at one point in his deposition Dr. Gore was asked whether there was an acknowledged threshold level below which one could say with scientific certainty that benzene exposure would *not* cause AML. Dr. Gore essentially said no. He explained “it is my belief that there is no threshold risk of safe exposure to Benzene. Biologically, it doesn’t make sense that there would be a threshold. Because, Benzene is a genotoxic agent. Any molecules of Benzene interacting with your DNA can cause damage to DNA.” But in any event, he said, “forty ppm-years, . . . is way out of the mainstream industrial epidemiologic literature to my reading.” And he identified a lower limit below which he was prepared to find that a person’s risk of contracting AML would no longer be enhanced. First, he noted that the risk of contracting AML from benzene exposure decreases with time following exposure. Second, he pointed out that according to the studies he relied on, someone who was exposed to less

than 6 ppm-years would no longer be at an elevated risk for AML after a 15-year latency period. On the other hand, Dr. Gore noted that these studies also show that people exposed to more than 10 ppm-years still face approximately an eight-times greater risk of developing AML than those in the general population, even after a 15-year latency period.

In granting Akzo's motion for summary judgment, the district court seized on the portion of Dr. Gore's testimony in which he discussed the "no threshold" idea, and on that basis, it found the entirety of Dr. Gore's opinion to be scientifically unreliable because it thought that the "no threshold" theory is "merely a hypothesis." The district court also faulted Dr. Gore for failing to rule out other potential causes of Schultz's AML, particularly his history of smoking.

## II

We review *de novo* whether a district court properly followed the framework for determining the admissibility of expert testimony under Rule 702 of the Federal Rules of Evidence, which largely codified *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993). *United States v. Brumley*, 217 F.3d 905, 911 (7th Cir. 2000). If the court properly applied the rule, we review its decision to admit or exclude expert testimony only for an abuse of discretion. *Id.* The ultimate decision to grant summary judgment is subject to *de novo* review. *Myers v. Illinois Cent. R.R. Co.*, 629 F.3d 639, 641 (7th Cir. 2010). Here, as we have already observed, these issues collapse into one:



if the district court correctly excluded Dr. Gore's testimony, then Akzo was entitled to prevail as a matter of law; if it did not, then it was error to grant summary judgment and Schultz is entitled to a trial.

Rule 702 permits a qualified expert witness to offer an opinion if the following criteria are met:

- (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;
- (b) the testimony is based on sufficient facts or data;
- (c) the testimony is the product of reliable principles and methods; and
- (d) the expert has reliably applied the principles and methods to the facts of the case.

FED. R. EVID. 702. As *Daubert* explained, the reliability of the expert's principles and methods can be examined by looking at factors such as (1) whether the scientific theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether a particular technique has a known potential rate of error; and (4) whether the theory or technique is generally accepted in the relevant scientific community. *Daubert*, 509 U.S. at 593-94. (This assumes that we are speaking of a scientific expert, such as the ones in this case; experiential experts are also permissible, see *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999), but a somewhat different threshold inquiry is necessary for them.)

Although this places the judge in the role of gatekeeper for expert testimony, the key to the gate is not the ultimate correctness of the expert's conclusions. Instead, it is the soundness and care with which the expert arrived at her opinion: the inquiry must "focus . . . solely on principles and methodology, not on the conclusions they generate." *Daubert*, 509 U.S. at 595. So long as the principles and methodology reflect reliable scientific practice, "[v]igorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence." *Id.* at 596. As we have noted before,

the question . . . whether the expert is credible or whether his or her theories are correct given the circumstances of a particular case is a factual one that is left for the jury to determine after opposing counsel has been provided the opportunity to cross-examine the expert regarding his conclusions and the facts on which they are based. It is not the trial court's role to decide whether an expert's opinion is correct. The trial court is limited to determining whether expert testimony is pertinent to an issue in the case and whether the methodology underlying that testimony is sound.

*Smith v. Ford Motor Co.*, 215 F.3d 713, 719 (7th Cir. 2000) (internal citations omitted); see also *Ortiz v. City of Chicago*, 656 F.3d 523, 536 (7th Cir. 2011). When a district court excludes an expert's testimony, it "must provide more than just conclusory statements about admissibility to

show that it properly performed its gatekeeping function.” *Oritz*, 656 F.3d at 536 (internal quotations omitted).

Two statements in Dr. Gore’s testimony require discussion: (1) Even after a 15-year latency period, a person exposed to 11 ppm-years of benzene or more would be at an eight-times greater risk for developing AML, and thus (because Dr. Stewart ascertained that Schultz had been exposed to more than double that amount) this meant that Schultz’s benzene exposure was a significant risk factor; and (2) With carcinogens like benzene, which cause permanent DNA mutations, it is theoretically possible that any amount of exposure could damage the DNA in a human cell. It is important to understand the difference between these two statements. The first says, in essence, that scientific studies confirm the danger of exposure to more than 10 ppm-years of benzene. The second says that no one is sure whether 10 ppm-years is the floor for risk, or 5 ppm-years, or 1 ppm-year, or nothing. There is nothing inconsistent between these two assertions. The latter may have been an unnecessary observation in Schultz’s case, since his exposure was 24 ppm-years, but there is no rule requiring the exclusion of expert testimony just because the expert digresses into a collateral issue to explain where the frontier of research lies. Our system relies on cross-examination to alert the jury to the difference between good data and speculation. Akzo’s counsel was also free to argue, based on its own expert’s submission, that 11 ppm-years was too low, and that risk does not arise until the 40 ppm-year level is reached.

In striking Dr. Gore's findings because the "basic thrust" of his opinion was that "the amount of benzene exposure is irrelevant," the district court overlooked Dr. Gore's unambiguous conclusion that Schultz had been exposed to a level of benzene that has been shown in studies to be a "very toxic and dangerous level." Had Dr. Stewart calculated that Schultz's exposure was only 5 ppm-years, we would have a different case, in which the district court's concern about an ill-defined floor for safety would have been justified. But we do not. Far from limiting his testimony to the proposition that the amount of exposure may be "irrelevant," Dr. Gore focused specifically on the amount of benzene to which Schultz had been exposed and related this amount to the scientific literature. He stated that, given a 15-year latency period, exposures of less than 6 ppm-years are *unlikely* to cause AML, but exposures of 11-ppm years or more put one at an eight-times greater risk of AML (as compared to the general population). Had Schultz been exposed to less than 6 ppm-years, Akzo would have been entitled to point out to the district court that Schultz's own expert was unwilling to point to benzene exposure as a likely cause of Schultz's AML. In short, Dr. Gore not only identified 11 ppm-years as a level that has been proven to be toxic, but he also suggested that 6 ppm-years might be a lower limit given current knowledge (while as a careful scientist reserving the possibility that even less exposure might be dangerous). There was no need for him to do more for purposes of Rule 702.

In finding Dr. Gore's testimony unreliable, the district court also emphasized that Dr. Gore's conclusion

diverged from a different study in the record in which the authors found that benzene has carcinogenic effects only at exposures greater than 40 ppm-years. But the competing study appears to rely on the identical methodology—observing AML rates in populations exposed to benzene over time—as the studies that Dr. Gore cited in support of his opinion that greater than 10 ppm-years exposure increases the risk of AML, even after 15 years. Indeed, as we noted earlier, Dr. Gore explained that the study finding a 40 ppm-year threshold was conducted with an extremely small sample size (only six cases of AML), unlike (for example) the Chinese study he submitted, which found that more than 10 ppm-years’ exposure was a significant risk factor based on observations of more than 30 cases of AML. Rule 702 did not require, or even permit, the district court to choose between those two studies at the gatekeeping stage. Both experts were entitled to present their views, and the merits and demerits of each study can be explored at trial.

The district court also suggested that Dr. Gore’s opinion was unreliable because he failed to rule out other potential causes of Schultz’s AML, including Schultz’s weight and smoking history. While the district court’s decision rather curiously says nothing about the legal standard for Schultz’s toxic tort claim, we presume that Wisconsin law applies. (The court was exercising diversity jurisdiction on the ground that some of the events took place in Wisconsin, and the default rule is to apply the law of the state where the district court sits unless the parties contend otherwise, which they

have not done here.) In Wisconsin, a strict products liability action requires a plaintiff to show that the product “was a cause (a substantial factor) of the plaintiff’s injuries or damages.” *Zielinski v. A.P. Green Indus., Inc.*, 661 N.W.2d 491, 494 (Wis. Ct. App. 2003) (quoting *Cook v. Gran-Aire, Inc.*, 513 N.W.2d 652, 654 (Wis. Ct. App. 1994)). In order to show that a toxin is “a cause” or “a substantial factor,” Schultz was not required to demonstrate that benzene exposure was the *sole* cause of his disease, so long as he showed that benzene contributed substantially to the disease’s development or significantly increased his risk of developing AML.

Furthermore, the district court was mistaken if it thought that Dr. Gore had ignored other possible causes altogether. The method of differential diagnosis on which Dr. Gore relied to assess the development of Schultz’s AML routinely identifies multiple causal factors. We have recognized this method of differential diagnosis and differential etiology as a generally accepted means for evaluating the cause of a plaintiff’s injury. *Myers*, 629 F.3d at 644 (“[I]n a differential etiology, the doctor rules in all the potential causes of a patient’s ailment and then by systematically ruling out causes that would not apply to the patient, the physician arrives at what is the likely cause of the ailment. There is nothing controversial about that methodology. The question of whether it is reliable under *Daubert* is made on a case-by-case basis, focused on which potential causes should be ‘ruled in’ and which should be ‘ruled out.’”) (internal citations omitted).

In *Myers*, we found an expert's testimony unreliable because it "did not rule in any causes of Myers's ailment, nor did [it] rule out anything." *Id.* When asked about a prior back surgery that may have contributed to Myers's back problems, Myers's physician responded, "I don't really think that it makes a hell of a lot of difference one way or the other . . . Now, if you are interested in causation, then from your standpoint, it's important." *Id.* at 645 (emphasis in original). This made clear that the physician had not considered potential causes of Myers's injury at all. (This may be a good illustration of the difference in perspective between doctors and lawyers: doctors normally want to treat the patient's ailment, no matter how it may have come about, while lawyers must dig further into causation.) *Myers* illustrates the situation contemplated by the Committee Notes to Rule 702, providing that a court may consider "[w]hether the expert has adequately accounted for obvious alternative explanations." FED. R. EVID. 702 (2000) Committee Note. That consideration should show why a particular alternative explanation is not, in the expert's view, the *sole* cause of the disease. See *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 156 (3d Cir. 1999). Beyond that, while *Myers* and the Committee Notes suggest that a reliable expert should consider alternative causes, they do not require an expert to rule out every alternative cause.

Unlike the expert in *Myers*, Dr. Gore considered which alternative causes should be ruled in, and which could be ruled out. He "determined that [Schultz's] smoking history may have contributed, but [he] found no evidence that any other risk factor played a role." He

further “ruled out, to a reasonable degree of medical certainty, that any other known risk factors for AML contributed to Mr. Schultz’s disease.” In fact, Dr. Gore’s report thoroughly addressed the possibility of alternative causes, explaining that:

[N]o case of cancer truly has only a single cause. Because cancer development is a complex, multi-stage process where many factors work together to contribute to the ultimate emergence of a full blown malignancy, each of those factors . . . must properly be considered a cause of the ultimate cancer and a substantial factor in bringing it about. . . . Thus, critically for the purposes of a specific causation analysis, the mere fact that genetics and/or other environmental risk factors . . . have been identified as probable causes of a particular case of cancer in no way refutes the possibility that chemical exposures being investigated have also played a substantial contributing role at one or more stages of the development of that person’s cancer.

In his deposition, Dr. Gore elaborated on the reasons why he concluded that exposure to benzene was a significant cause for Schultz, despite the history of cigarette smoking: “[T]obacco is one of the strongest causative factors . . . [but] it’s not as strong on a risk basis, per se, as Benzene. . . . As a matter of fact, the Benzene in the smoke is one of the things that is thought, potentially, to contribute to the development of leukemia. . . .”

*Daubert* counseled that courts should focus “solely on principles and methodology, not on the conclusions that



they generate.” 509 U.S. at 595. Because Dr. Gore’s testimony does not suffer from either of the deficiencies that the district court attributed to it, the court erred by excluding it. Moreover, with Dr. Gore’s contribution restored to the case, we conclude that Schultz has presented enough to defeat Akzo’s motion for summary judgment.

### III

Durako is another matter. The only evidence in the record even hinting that Schultz was exposed to Durako products is a document provided by Chrysler entitled “Possible Paint Related Products Active at Kenosha Plants 1985-89” in which one Durako product is mentioned. This document was not authenticated; there appears to be no foundation in the record about who created it or why; and it does not indicate when, how much, or how often the Durako product was used. This falls short, as a matter of law, for the purpose of demonstrating that Schultz used any amount of Durako paint during the course of his work for Chrysler. Therefore, Schultz cannot support a products liability claim against Durako. *Zielinski*, 661 N.W.2d at 494.

### IV

For these reasons, we REVERSE the district court’s decision granting Akzo’s motion for summary judgment, and we AFFIRM the district court’s decision granting Durako’s motion for summary judgment. The case is

REMANDED to the district court for further proceedings consistent with this opinion.