REPORTED

IN THE COURT OF SPECIAL APPEALS

OF MARYLAND

No. 536

September Term, 2011

BERNARD DIXON, ETC., ET AL.

v.

FORD MOTOR COMPANY

Matricciani,
Berger,
Eyler, James R.
(Retired, Specially Assigned),

JJ.

Opinion by Matricciani, J.

Filed: June 29, 2012

On July 1, 2008, Joan Dixon and her husband, Bernard Dixon, brought suit in the Circuit Court for Baltimore City against corporations involved in the manufacturing and distribution of products containing asbestos, including Ford Motor Company, the Georgia-Pacific Corporation ("GP"), Honeywell International, Inc., and the Union Carbide Corporation ("UCC"). Following Mrs. Dixon's death from pleural mesothelioma, Mr. Dixon pursued her claims as representative of her estate, and the Dixons' four adult daughters joined their father as plaintiffs, who are now appellants.

Prior to trial, appellants settled with GP, Honeywell, and UCC, but Ford's cross-claims against those defendants remained for adjudication as potential joint tortfeasors.

Ford moved *in limine* for a hearing to challenge appellants' proffered expert on the issue of causation, as well as to exclude the expert's testimony. Trial commenced on April 15, 2010, and the court denied Ford's motions, along with certain objections Ford raised during the expert's testimony.

On April 27, 2010, the jury returned a verdict awarding appellants a total of $15,000,000 in compensatory damages, which the court reduced to $6,065,000 in accordance with the non-economic damages cap of Maryland Code (2006), § 11-108 of the Courts and Judicial Proceedings Article ("CJ"). Ford subsequently filed post-trial motions requesting a new trial and revisions or judgments notwithstanding the verdict ("JNOV") on both its own cross-claims and appellants' direct claims. The court denied Ford's motions for new trial and JNOV, but ruled that the jury's verdict was inconsistent and revised the judgments against Ford and GP to adjust for the latter's contribution as a

joint tortfeasor. The court entered its revised judgment in favor of appellants in the collective amount of $3,032,500.00, from which both appellants and Ford filed timely appeals.

QUESTIONS PRESENTED

The parties' briefs present a total of eight questions between them,[1] but our

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[1] The remaining questions presented were as follows. Appellants asked:

I. Whether the trial court erred in revising the judgment pursuant to Maryland Rule 2-535(a) to affirmatively hold Georgia-Pacific, LLC liable on Ford Motor Company's cross claim for Mrs. Dixon's mesothelioma[,] contrary to the jury's verdict.

II. Whether portions of the statutory cap on non-economic damages found at Md. Code, § 11-108 of the Courts and Judicial Proceedings Article as applied in this case violates [sic] the equal protection clause of the U.S. Constitution and Article 24 of the Maryland Declaration of Rights.

In addition to the question presented, above, Ford asked:

II. Whether the trial court abused its discretion by failing to exclude the specific causation opinion of Dr. Laura Welch that Mrs. Dixon's alleged secondary exposure to asbestos from her husband's work with Ford brake linings was a substantial contributing factor in causing her mesothelioma, where that opinion was offered in response to a hypothetical question that assumed facts not adduced in evidence?

III. Whether the trial court erred in denying Ford's Motion for New Trial given that (a) the jury's verdict was inconsistent and against the great weight of evidence, (b) Plaintiff's closing arguments were highly improper and prejudicial, and (c) the verdict was shocking.

IV. Whether the trial court erred in failing to enter judgment against the remaining Cross-Defendants in the matter, given the uncontradicted

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opinion need only address the first of Ford's questions presented:

> I. Did the trial court err when it denied Ford's motion to exclude appellants' expert epidemiological opinion on "substantial contributing factor causation" where the expert's testimony did not quantify the probability of causation?

For the reasons that follow, we answer yes and remand the case for a new trial consistent with this opinion.

FACTUAL AND PROCEDURAL HISTORY

Joan Dixon died of pleural mesothelioma on February 28, 2009, having initiated a suit against Ford and various other entities involved in the asbestos market, including GP, Honeywell, and UCC. The complaint alleged that Mr. and Mrs. Dixon "participated in home improvement and maintenance projects throughout the 1960s and 1970s during with [sic] they worked with and around Defendants' asbestos products," and that "[t]hroughout the 1950s, 1960s and 1970s, Mrs. Dixon was exposed to asbestos dust created by Mr. Dixon's work with and around asbestos-containing automobiles and asbestos-containing replacement parts for those automobiles including . . . brakes[.]" Appellants further alleged that Mrs. Dixon's "exposure to Defendants' asbestos containing products and asbestos containing vehicles and the inhalation of asbestos fibers from the products and vehicles caused her disease and eventual death."

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[1](...continued)
evidence presented with respect to their liability at trial?

In response to interrogatories, appellants stated that they "believe Joan Dixon may have been exposed to asbestos through her and her husbands' use of and exposure to various building materials, including but not limited to Georgia Pacific pre-mixed drywall joint compound which, upon information and belief, contained Union Carbide Corporation's Calidria brand asbestos." Deposition testimony established that Mrs. Dixon personally sanded joint compound and cleaned up after at least five home construction and renovation projects.[2]

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[2] The trial court summarized Mrs. Dixon's exposure to dry wall joint compound in a post-trial memorandum:

> Mr. Dixon, Mrs. Dixon, and the Dixon's daughters testified that Mrs. Dixon personally sanded joint compound and that she would become so covered in dust that she became "white." Mrs. Dixon testified that they used Georgia Pacific Ready Mix compound while doing textured ceilings at their home in 1966; Mr. and Mrs. Dixon both testified that they used Georgia Pacific joint compound when they built the beauty shop addition in 1971, and that Mrs. Dixon sanded drywall. They also used Georgia Pacific joint compound when they put up drywall and renovated their garage at the same time. The family built a bedroom in the basement of their home in the early 1970s, and Mrs. Dixon used Georgia Pacific joint compound to finish the drywall. Mrs. Dixon was also exposed to the Georgia Pacific compound Mr. Dixon used when he renovated the basement, renovated Mrs. Costello's house, and did construction with Allen and Harry Paul which included putting up and sanding drywall. At Ms. Costello's house, her daughter remembers Mr. Dixon working with the green and white buckets that were identified as Georgia Pacific joint compound.
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> In addition, Mrs. Dixon was exposed to Georgia Pacific joint compound when she and Mr. Dixon built the family meat store and apartments in 1976. Mrs. Dixon testified that she used Georgia Pacific Ready Mix joint compound to finish the drywall work in the apartments from 1976 to 1977. Mr. Dixon testified that she did the drywall work in the same manner as the

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Appellants settled with GP, Honeywell, and UCC prior to trial. Their settlement agreements did not determine whether the defendants were joint tortfeasors, and so they remained in the case nominally as Ford's cross-defendants, unrepresented by counsel.

Appellants sought to introduce Dr. Laura Welch as an expert in asbestos epidemiology and proffered her opinion on causation. Ford did not dispute that Dr. Welch was qualified to render expert testimony on the subject of epidemiology, but instead objected to the methods and substance of her causation opinion. The court denied Ford's motion,[3] and Dr. Welch testified on direct "that mesothelioma in particular is a dose-response disease. Every increasing [asbestos] dose increases the likelihood of getting it, [and] additional doses decrease the time it takes to get the disease as exposure goes up." When asked to assume that Mrs. Dixon was exposed to asbestos in various other ways, Dr. Welch stated that in her opinion, "every exposure to asbestos is a substantial contributing cause and so brake exposure would be a substantial cause even if [Mrs. Dixon] had other exposures." On cross-examination, Dr. Welch further explained her opinion that every exposure to asbestos is a "substantial contributing cause" of mesothelioma:

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[2] (...continued)
work she had previously done, including sanding, cleaning, and doing the laundry. During the relevant time period when Mr. and/or Mrs. Dixon performed home renovation work, Mrs. Dixon did the laundry.

[3] Dr. Welch was admitted as an expert in occupational medicine, internal medicine, epidemiology of asbestos disease, and the diagnosis of asbestos disease.

> Q. Now, with regard to your opinions about asbestos and causation which you have given this jury, it's your opinion, Doctor, that each and every exposure contributes to the development of mesothelioma no matter where it comes from in asbestos exposure; is that right?
>
> A. Right. When somebody has got mesothelioma, all the exposures they have were contributing factors, that's my opinion.
>
> Q. And it doesn't matter to you whether the exposure is on a frequent basis, correct?
>
> A. No. I mean, an exposure can be like one event. On a frequent basis, you would be talking about a task or something like that. But I think each one of those discrete exposures is a contributing factor.
>
> Q. So it can be one event in order to be, in your view, a substantial contributing cause to the development of mesothelioma?
>
> A. Correct. It could be one day of work, for example, or something like that.
>
> Q. So it doesn't have to be a regular course of work? It doesn't have to be something they do occupationally for a period of time; is that true?
>
> A. No, not necessarily. Because we are talking about somebody who has already got the disease. And I think everyone of the exposures that go into making their sum total of exposure to asbestos is a contributing factor.

Against this general causal backdrop, Dr. Welch explained the basis of her opinion that Mrs. Dixon's exposure to automobile brake dust was a substantial contributing factor of Mrs. Dixon's disease:

> The easiest way to do that is to turn back to my paper we kept discussing, the Amicus brief that walks through the information that supports a conclusion that brake mechanics are at an increased risk for mesothelioma. So we have a lot of data that the kind of asbestos that's in brakes, chrysotile, causes mesothelioma. We have a lot of information that brief or low-level exposure to asbestos causes mesothelioma. And

> I think [plaintiffs' counsel] had a number of those papers up there on the slides we put together.
>
> And one other part of it is that, as you and I just talked about, asbestos is, in my opinion, the only recognized cause of mesothelioma.
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> And we know that brake mechanics have exposure to asbestos as has been demonstrated in industrial hygiene studies. There [are] lots of studies that show that exposure occurs. It occurs to [sic] substances known to cause mesothelioma, that people working with friction products get enough asbestos exposure to cause other nonmalignant diseases. And to me, all those studies allow me to say exposure to asbestos by working with brakes can be a substantial contributing cause of mesothelioma.

Dr. Welch admitted that no epidemiological studies had specifically investigated the risk of mesothelioma among wives of brake mechanics. However, Dr. Welch explained that such a study would be practically impossible; if the rate of disease in a group of workers was two-and-a-half cases per hundred thousand—as in the case of mesothelioma—such a study would require thirty-thousand subjects and twenty years to reach a statistically significant conclusion. Instead, Dr. Welch testified that she formed her opinion from the generally accepted fact that asbestos dust left on a worker's clothing can be transmitted to family members in the home environment.

In its defense, Ford called epidemiologist and risk assessment expert Herman J. Gibb, Ph.D., who testified that none of eighteen epidemiological studies in evidence showed "any evidence that vehicle mechanics have an increased risk of mesothelioma." By contrast, Dr. Gibb testified that epidemiological studies showed that the risk of mesothelioma among construction workers exposed to asbestos dust is up to seven times

greater than the risk of mesothelioma among the general population. Dr. Gibb admitted on cross-examination that only three of the studies that Ford relied upon categorically examined "brake mechanics," as opposed to "auto workers" or generic "mechanics."[4]

After the close of evidence, the jury deliberated and returned a verdict against Ford, awarding $5,000,000 to Mrs. Dixon's estate, $4,000,000 to Bernard Dixon, and $1,500,000 to each of the Dixons' four daughters, for a total of $15,000,000. After applying the non-economic damages cap of CJ § 11-108, the court entered judgment awarding $5,000,000 to Mrs. Dixon's estate, $426,000 to Bernard Dixon, and $159,750 to each of the Dixons' daughters, for a total of $6,065,000. Ford filed a series of post-trial motions seeking a new trial, JNOV, and revised judgments. On March 10, 2011, the court issued a memorandum opinion and order that granted Ford's request to revise the judgments so as to account for GP as a joint tortfeasor under CJ § 3-1404. Accordingly, the trial court halved the awards to each of the appellants and, on April 21, 2011, entered

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[4] Dr. Welch had earlier given her assessment of various epidemiological studies finding no evidence of increased mesothelioma risk among auto workers:

> I think the group of studies have such significant limitations, they don't -- they conclude -- or an overall analysis of them concludes that asbestos exposure in brakes does not cause mesothelioma, but a study that doesn't find something doesn't mean there is no relationship.
>
> You have to look at the studies and see whether they are capable of answering the question. I think the limitations of those studies is so significant that they don't really answer the question. They don't give us any good information on the question.

judgment awarding $2,500,000 to Mrs. Dixon's estate, $213,000 to Bernard Dixon, and $79,875 to each of the Dixons' daughters, for a total of $3,032,500 against Ford. Ford's remaining motions were denied. Appellants and Ford then filed timely appeals, bringing the case before us.

DISCUSSION

Ford argues that the trial court erred when it admitted Dr. Welch's testimony on the issue of causation and supports its argument with three premises. First, Ford contends that Dr. Welch's testimony was inadmissible under the holdings of *Reed v. State*, 283 Md. 374 (1978), and *Blackwell v. Wyeth*, 408 Md. 575 (2009). Second, Ford argues that Dr. Welch's opinion deserves special scrutiny under *Frye-Reed* "in the face of epidemiological evidence to the contrary," which Ford introduced in its defense. Third, Ford argues that "Dr. Welch's causation opinions fundamentally are at odds with 'the frequency, regularity and proximity' requirements for substantial factor causation" set forth in *Eagle-Picher Industries, Inc. v. Balbos*, 326 Md. 179 (1992). In response, appellants argue first that Dr. Welch's testimony was not subject to the *Frye-Reed* test because it was based upon her specialized knowledge, training, and skill in a relevant field of inquiry. Second, appellants argue that Ford's epidemiological studies are not conclusive. Third, appellants argue that the *Balbos* standard "is a test of legal causation," and that Dr. Welch's ultimate opinion was admissible because it addressed only "medical or epidemiological causation." For the following reasons, we hold that the trial court

abused its discretion under Maryland Rule 5-702 when it admitted Dr. Welch's testimony. *See Exxon Mobil Corp. v. Ford*, 204 Md. App. 1, 27 n.20 (Md. Ct. Spec. App. 2012) (Zarnoch, J. concurring and dissenting) (whereas *Frye-Reed* determinations are reviewed *de novo*, Rule 5-702 decisions are reviewed for abuse of discretion).

In *Reed*, the Court of Appeals adopted the test first articulated in *Frye v. United States*, 293 F. 1013, 1014 (D.C. Cir. 1923), now known in Maryland as the *Frye-Reed* standard: "[B]efore a scientific opinion will be received as evidence at trial, the basis of that opinion must be shown to be generally accepted as reliable within the expert's particular scientific field." 283 Md. at 381. In *Blackwell v. Wyeth*, the Court applied *Frye-Reed* to a novel theory of causation. Drawing from the Supreme Court's decision in *GE v. Joiner*, 522 U.S. 136, 146 (1997), the *Blackwell* Court held that where an expert derives an untested hypothesis from generally accepted theories and research methods, the trial court must weigh the "analytical gap" between the established theories and methods on one figurative side, and the expert's opinion on the other. 408 Md. at 605-08 ("Generally accepted methodology, therefore, must be coupled with generally accepted analysis in order to avoid the pitfalls of an 'analytical gap.'").

Despite their perceived differences, the parties in fact agree on the fundamental scientific principles undergirding the expert testimony on both sides. Ford's *amici*,

among them several eminent researchers and professors,[5] note that "[m]any scientists would argue that each exposure adds, not to the disease, but to the probability of the disease," and that "[t]he risk or probability of developing a disease is proportional to the extent of the exposure or cumulative exposure." The *amici* do not quantify how "many" scientists hold this view, but the U.S. Department of Health and Human Services ("DHHS") has reported that there is "general agreement among scientists and health agencies" that "[e]xposure to any asbestos type (*i.e.*, serpentine or amphibole) can increase the likelihood of lung cancer, mesothelioma, and nonmalignant lung and pleural disorders." *Toxicological Profile for Asbestos*, DHHS Public Health Service, Agency for Toxic Substances and Disease Registry, 20 app. F (2001), available at http://www.atsdr.cdc.gov/ToxProfiles/tp61.pdf. *See also Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution*, 23 Scand. J. Work Environ. Health 311, 314 (1997) ("Cumulative exposure, on a probability basis, should thus be considered the main criterion for attribution of a substantial contribution by asbestos to lung cancer risk.").

The question open to scientific debate is the *extent* to which various asbestos

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[5] Among other well-credentialed scientists, the *amici* include James D. Watson, Ph.D., the Nobel laureate best known as a co-discoverer of the structure of DNA, Dudley Herschbach, a Nobel laureate in chemistry and professor of science at Harvard University, Richard Wilson, D.Phil., research professor of physics at Harvard University, and Patricia Buffler, Ph.D., professor of epidemiology at the School of Public Health of the University of California at Berkeley.

exposures increase the risk of mesothelioma. Notably, calculations by the United States Environmental Protection Agency indicate that even ambient air levels of asbestos increase one's risk of cancer and mesothelioma.[6] *Toxicological Profile for Asbestos* at 24 app. F. This finding comports with studies appellants cite for the proposition that there is no perfectly "safe" amount of asbestos exposure; rather, decreasing exposure implies only decreasing risk, and vice-versa. *See* John T. Hodgson & Andrew Darnton, *The quantitiative risks of mesothelioma and lung cancer in relation to asbestos exposure*, 44 Ann. Occup. Hyg. 565-601 (2000) (it is likely that "the exposure response curve simply starts to descend more steeply from some point on the cumulative exposure scale."); Y. Iwatsubo *et al.*, *Pleural mesothelioma: dose-response relation at low levels of asbestos*

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[6] The DHHS reported that one of several "unresolved issues" is "the actual risks for malignant or nonmalignant respiratory disease that may exist at exposure levels below air concentrations (0.1–0.2 fiber/mL) established as recent occupational exposure limits," and noted the following findings:

> Lung Cancer and Mesothelioma: Based on an analysis of data from epidemiologic studies of workers who were exposed to asbestos before modern occupational exposure limits were established, EPA (1986) calculated by extrapolation that lifetime exposure to asbestos air concentrations of 0.0001 fiber/mL could result in up to 2 to 4 excess cancer deaths (lung cancer or mesothelioma) per 100,000 people. This air concentration is within reported ranges of ambient air levels (0.00001 to 0.0001 fiber/mL). The EPA analysis has been extensively discussed and reviewed in the scientific literature (Camus et al. 1998; Hodgson and Darnton 2000; Hughes 1994; Landrigan 1998; Lash et al. 1997). EPA is in the process of reviewing and possibly updating their [sic] cancer risk estimates for asbestos.

Toxicological Profile for Asbestos at 24 app. F.

exposure in a French population-based case-control study, 148 Am. J. Epidemiol. 133-42 (1998) (estimating an odds ratio of 1.1 for exposures less than .5f/ml-years); *Asbestos*, 23 Scand. J. Work Environ. Health at 313 ("Mesothelioma can occur in cases with low asbestos exposure. However, very low background environmental exposures carry only an extremely low risk.").

We therefore have a settled scientific theory of causation, and it is captured by what philosophers of science call "probabilistic causation," a logical model that accounts for the inherent uncertainty in counterfactual truth.[7] *See* Christopher Hitchcock, *Probabilistic Causation*, The Stanford Encyclopedia of Philosophy (Winter 2011 Edition), Edward N. Zalta (ed.). The probabilistic model of causation incorporates the fundamental limitations on human knowledge that prevent absolute certainty about any given inference, including counterfactual causal assertions.[8] Interestingly—and not surprisingly—the motivation for probabilistic causation is the same as the justification that Justice Harlan ascribed to the legal burden of proof in his dissent from *In Re Winship*:

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[7] The other motivation for probabilistic causation—which lies well beyond the scope of this discussion—is the possibility that the physical universe is physically indeterminate, which remains a topic of debate among some of the world's greatest minds. We need not resort to any assumption about determinism, however, for the necessary presence of uncertain factual inquiries in law justifies our use of the probabilistic model.

[8] The model can also describe a non-deterministic universe, but that is certainly beyond the scope of this discussion. *See Probabilistic Causation*, cited above.

> [I]n a judicial proceeding in which there is a dispute about the facts of some earlier event, the factfinder cannot acquire unassailably accurate knowledge of what happened. Instead, all the factfinder can acquire is a belief of what probably happened. The intensity of this belief—the degree to which a factfinder is convinced that a given act actually occurred—can, of course, vary. In this regard, a standard of proof represents an attempt to instruct the factfinder concerning the degree of confidence our society thinks he should have in the correctness of factual conclusions for a particular type of adjudication. Although the phrases "preponderance of the evidence" and "proof beyond a reasonable doubt" are quantitatively imprecise, they do communicate to the finder of fact different notions concerning the degree of confidence he is expected to have in the correctness of his factual conclusions.

397 U.S. 358, 370 (1970).

Probabilistic causation is, of course, different in kind from the legal burden of proof. The former is a mental model that states causal assertions as *continuous conditional probabilities*, whereas the latter is a *discrete threshold* for legal action set at some level of belief in specific causation. In spite of this difference, both doctrines rest on the premise that empirical inquiry can only ever give a certain *degree of certainty* in an inference of fact from evidence, including conditional and counterfactual assertions of causation.[9] And in both scenarios, a decision maker must weigh the costs of inaction against the expected costs of acting wrongly; we shall return to this point, below.

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[9] For an extended discussion of "subjective probability," see Vern R. Walker, *Preponderance, Probability, and Warranted Factfinding*, 62 Brooklyn L. Rev. 1075, 1080-92 (1996).

Despite the parties' (unrecognized) agreement on the science of mesothelioma causation and the conceptual framework that it entails, Ford attacks Dr. Welch's methodology on the grounds that it is not generally accepted. Specifically, Ford argues that Dr. Welch's opinion testimony violated the tenets of *Blackwell* and *Balbos* by using "downward extrapolation" to arrive at the conclusion that every asbestos exposure is a "substantial contributing factor" in causing eventual mesothelioma. As we now explain, Ford's argument conflates scientific causation and legal causation in attacking Dr. Welch's methodology. However, Dr. Welch *herself* also conflated those concepts, and so her testimony did not "assist the trier of fact to understand the evidence or to determine a fact in issue," as required by Maryland Rule 5-702.

Like the closely-related concept "proximate cause," the term "substantial contributing factor" goes beyond the logically simple question of whether the defendant's action (or inaction) was a "necessary" or "sufficient" condition of harm to the plaintiff. Both proximity and substantiality describe whether "the defendant's conduct has such an effect in producing the harm as to lead reasonable men to regard it as a cause, using that word in the popular sense, in which there always lurks the idea of responsibility[.]" Restatement (Second) of Torts § 431 (1965). Where the question of causation is probabilistic, "substantiality" and "responsibility" necessarily imply some test of

magnitude,[10] *viz*., how much must exposure have *increased* one's *risk* of harm in order to hold the responsible party liable? *See* William M. Landes & Richard A. Posner, *Causation in Tort Law: An Economic Approach*, 12 J. Legal Stud. 109, 133 (1983) ("probability of injury and administrative cost are . . . the two factors that we have argued determine the willingness of the courts to invoke the concept of legal or proximate causation to excuse injurers from liability for certain accidents."); Guido Calabresi, *Concerning Cause and the Law of Torts: An Essay for Harry Kalven, Jr.*, 43 U. Chi. L. Rev. 69, 78 (1975) ("The judgment involved in choosing to forbid or restrict particular behavior is precisely the kind of judgment that depends on the existence of a causal link, for there is no reason to prohibit or restrict behavior that we do not believe will increase the chances of injury in the future.").

If risk is our measure of causation, and substantiality is a threshold for risk, then it follows—as intimated above—that "substantiality" is essentially a burden of proof. Moreover, we can explicitly derive the probability of causation from the statistical measure known as "relative risk," as did the U.S. Court of Appeals for the Third Circuit in *DeLuca v. Merrell Dow Pharmaceuticals, Inc.*, 911 F.2d 941, 958 (3d Cir. 1990), in a

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[10] It also appears that, in this context, "substantiality" is an analogue of "proximity" in that it asks what factors must be considered in assessing the risks of exposure: must we account for the fact that a plaintiff smoked, was exposed to other sources of asbestos, or is biologically susceptible to cancer? Stated in terms of probabilistic causation, proximate causation would determine the set of "constants" to be used in testing and calculating the conditional probabilities of harm with and without exposure.

holding later adopted by several courts.[11] For reasons we need not explore in detail, it is not prudent to set a singular minimum "relative risk" value as a legal standard.[12] But even

[11] *See, e.g.*, *Daubert v. Merrell Dow Pharms., Inc.*, 43 F.3d 1311, 1320 (9th Cir. 1995) (opinion on remand from *Daubert v. Merrell Dow Pharms.*, 509 U.S. 579 (1993)), *cited with approval in In re Hanford Nuclear Reservation Litig. v. E. I. Dupont*, 292 F.3d 1124, 1137 (9th Cir. 2002) (adding that, "when available, known individual risk factors are also relevant"); *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1225-1226 (D. Colo. 1998) ("Plaintiffs must present expert testimony demonstrating that exposure to breast implants more than doubled the risk of their alleged injuries."); *In re W.R. Grace & Co.*, 355 B.R. 462, 483 (Bankr. D. Del. 2006) ("We accept Grace's position that Claimants must establish causation by a 2.0 relative risk rate."); *Marder v. G.D. Searle & Co.*, 630 F. Supp. 1087, 1092 (D. Md. 1986) ("In epidemiological terms, a two-fold increased risk is an important showing for plaintiffs to make because it is the equivalent of the required legal burden of proof -- a showing of causation by the preponderance of the evidence or, in other words, a probability of greater than 50%."), *aff'd without op. sub nom. Wheelahan v. G.D. Searle & Co.*, 814 F.2d 655 (4th Cir. 1987); *Manko v. United States*, 636 F. Supp. 1419, 1434 (W.D. Mo. 1986) (swine flu vaccine allegedly caused Guillain-Barré syndrome), *aff'd in part*, 830 F.2d 831 (8th Cir. 1987) ("A relative risk greater than '2' means that the disease more likely than not was caused by the event."); *In re "Agent Orange" Prod. Liab. Litig.*, 597 F. Supp. 740, 834-37 (E.D.N.Y. 1984) ("Conventional application of the 'weak' version of the preponderance rule would dictate that, if the toxic substance caused the incidence of the injury to rise more than 100% above the 'background' level, each plaintiff exposed to the substance could recover if he or she is suffering from that type of injury."), *aff'd*, 818 F.2d 145 (2d Cir. 1987); *Sanderson v. International Flavors & Fragrances, Inc.*, 950 F. Supp. 981, 1000 (C.D. Cal. 1996) (acknowledging a relative risk of 2 as a threshold for plaintiff to prove specific causation); *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 718 (Tex. 1997) ("The use of scientifically reliable epidemiological studies and the requirement of more than a doubling of the risk strikes a balance between the needs of our legal system and the limits of science.").

[12] In *Daubert II*, 43 F.3d at 1320-21, the U.S. Court of Appeals for the Ninth Circuit held that causation under a preponderance standard is equivalent to a "relative risk" of two. In other words, the *Daubert* court held that a plaintiff's risk of injury must have at least doubled in order to hold that the defendant's action was "more likely than not" the actual cause of the plaintiff's injury. The problem with this holding is that relative risk does not behave like a "binary" hypothesis that can be deemed "true" or

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if there were some legal threshold, Dr. Welch provided no information that could help the finder of fact to decide whether the elevated risk in this case was "substantial."

Dr. Welch testified that, in her opinion, "*every exposure* to asbestos is a *substantial contributing cause* and so brake exposure would be a substantial cause even if [Mrs. Dixon] had other exposures." And while we have no doubt that Dr. Welch is well-qualified to render some opinion as to the *likely* intensity of Mrs. Dixon's exposure and the *likely* effect it had on her risk of mesothelioma, Dr. Welch's testimony implied only that both were "more than nothing." For obvious reasons an infinitesimal change in risk cannot suffice to maintain a cause of action in tort. Thus, whatever information Dr.

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[12] (...continued)
"false" with some degree of confidence; instead, the uncertainty inherent in any statistical measure means that relative risk does not resolve to a certain probability of *specific* causation. In order for a study of relative risk to truly fulfill the preponderance standard, it would have to result in 100% confidence that the relative risk exceeds two, which is a statistical impossibility. In short, the *Daubert* approach to relative risk fails to account for the twin statistical uncertainty inherent in any scientific estimation of causation. *See* Steve Gold, Note, *Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence*, 96 Yale L. J. 376, 384 (1986) (failure to distinguish between "fact probability" and "belief probability" "works hidden doctrinal changes and encourages the application of simple-minded quantitative rules and narrow limits on evidence").

All of this is not to say, however, that any and all attempts to establish a burden of proof of causation using relative risk will fail. Decisions can be—and in science or medicine are—premised on the *lower limit* of the relative risk ratio *at a requisite confidence level*. The point of this minor discussion is that one cannot apply the usual, singular "preponderance" burden to the probability of causation when the only estimate of that probability is statistical relative risk. Instead, a statistical burden of proof of causation *must* consist of two interdependent parts: a requisite *confidence* of some *minimum relative risk*. As we explain in the body of our discussion, the flaws in Dr. Welch's testimony mean we need not explore this issue any further.

Welch conveyed could not possibly have helped the jurors to weigh the substantiality of Ford's contribution to Mrs. Dixon's disease. *See Moeller v. Garlock Sealing Techs., LLC*, 660 F.3d 950, 955 (6th Cir. 2011) (explaining that if an opinion that every exposure is substantial "is sufficient for plaintiff to meet his burden, the Sixth Circuit's 'substantial factor' test would be meaningless" (citing *Lindstrom v. A-C Product Liability Trust*, 424 F.3d 488, 493 (6th Cir. 2005))); *Gregg v. V-J Auto Parts Co.*, 596 Pa. 274, 291 (Pa. 2007) ("expert affidavits attesting that any exposure to asbestos, no matter how minimal, is a substantial contributing factor in asbestos disease . . . do not suffice to create a jury question in a case where exposure to the defendant's product is *de minim[i]s* . . .").

Dr. Welch's conclusion that the exposure and risk in this case were "substantial" simply was not a scientific conclusion, and without it her testimony did not provide information for the jury to use in reaching its conclusion as to substantial factor causation. For these reasons and in these circumstances (*i.e.*, where probabilistic causation is the generally accepted scientific theory of causation and scientific expert testimony is required), we join with several other courts in requiring quantitative epidemiological evidence.[13] *See, e.g., Bland v. Verizon Wireless, L.L.C.*, 538 F.3d 893, 898 (8th Cir.

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[13] Appellants cite *ACandS, Inc. v. Abate*, 121 Md. App. 590 (1998), for the proposition that "Maryland jurisprudence does not require proof of an individual's dose of asbestos in order to prove substantial factor causation as articulated by *Balbos*." But appellants overstate the conclusion of *AcandS*, in which we said that "[w]e *shall not hold* that a plaintiff in any asbestos case must present expert testimony as to the amount of respirable asbestos fibers emitted by a particular product." *Id*. at 671 (emphasis added).

(continued...)

2008); *Lindstrom*, 424 F.3d at 498; *Mitchell v. Gencorp Inc.*, 165 F.3d 778, 781 (10th Cir. 1999); *Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 278 (5th Cir. 1998).

Before we conclude our discussion, we address Ford's remaining arguments so as to avoid four possible points of confusion. First, Ford complains that "downward extrapolation" is not a reliable method to establish substantial factor causation, but that argument is only trivially true. As we have seen, "substantiality" is a legal concept and not an objective property testable by the scientific method. The contributions of science end at quantitative estimates of exposure and risk. Thus, it is primarily Dr. Welch's *conclusion*, and not her *methodology*, with which we take issue.

Second, we agree with Ford's contention that "case reports" and other anecdotal evidence are not probative of either general or actual causation,[14] but we must point out

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[13] (...continued)
Our decision did not address whether exposure—or risk—must be quantified because, "[u]nder the peculiar circumstances of this particular case, the evidence against [the defendant] was sufficient without such testimony." *Id*. The circumstances of this case are "peculiar" in a different way: they present such attenuated evidence of exposure and risk that the jury could not possibly weigh substantial factor causation without the aid of expert testimony. And as our discussion has shown, one can draw no conclusions about probabilistic causation without estimated quantities of exposure and risk.

[14] *See* Federal Judicial Center, *Reference Manual on Scientific Evidence* 91 (2d ed. 2000) ("'Anecdotal evidence' means reports of one kind of event following another. Typically, the reports are obtained haphazardly or selectively, and the logic of 'post hoc, ergo propter hoc' does not suffice to demonstrate that the first event causes the second."). *See also Haggerty v. Upjohn Co.*, 950 F. Supp. 1160, 1163–64 (S.D. Fla. 1996) ("anecdotal case reports appearing in medical literature . . . can be used to generate hypotheses about causation, but not causation conclusions" because "scientifically valid cause and effect determinations depend on controlled clinical trials and epidemiological
(continued...)

that Ford severely overstates the strength of its own epidemiological evidence. As Dr. Welch rightly explained, fifteen of Ford's eighteen proffered studies measured mesothelioma incidence in "vehicle mechanics" without quantifying or controlling for varying exposures within that broad industrial category. Furthermore, the leading epidemiological report cited by Ford and its *amici* that specifically studied "brake mechanics," P. A. Hessel *et al*., *Mesothelioma Among Brake Mechanics: An Expanded Analysis of a Case-control Study*, 24 Risk Analysis 547 (2004), does not at all dispel the notion that this population faced an increased risk of mesothelioma due to their industrial asbestos exposure. In cases of rare diseases, a measure known as the "odds ratio" provides an estimate of the "relative risk," which is the "times" the default risk without exposure increases with exposure (a relative risk of two implies a doubled risk, three implies a tripled risk, and so forth). Federal Judicial Center, *Reference Manual on Scientific Evidence* 348-50 (2d ed. 2000). When calculated at the 95% confidence level, Hessel *et al*. estimated that the odds ratio of mesothelioma could have been as low as 0.01 or as high as 4.71, implying a nearly *quintupled risk* of mesothelioma among the population of brake mechanics. 24 Risk Analysis at 550-51.

Third, we would not use language so strong to describe Ford's arguments about the "analytical gap" in this case, but Professor Vern R. Walker dispelled similar notions in his

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[14] (...continued)
studies").

comprehensive article "Restoring the Individual Plaintiff to Tort Law by Rejecting 'Junk Logic' About Specific Causation," 56 Ala. L. Rev. 381 (2004). In it, Professor Walker explains that *any* opinion rendered as to the likelihood of *actual* causation in a specific case must bridge the "analytical gap"[15] between epidemiological research and the plaintiff's situation:

> The first [is] the problem of . . . finding that a reference group adequately represents the specific individual—that is, that it adequately matches the plaintiff in all causally relevant variables, such as being a woman, being age forty, having no history of cancer in the immediate family, and so forth. The second [is] the uncertainty in assigning a particular probability to the individual case, even when the reference group adequately represents the specific individual. These two major sources of uncertainty can be called, respectively, uncertainty about plaintiff-representativeness and uncertainty about assigning a probability to the individual plaintiff.

Id. at 384-85.

Thus, it could be said that while epidemiology can provide an accurate estimate of general causation among a population that controls for as many variables as possible, actual causation lies outside the bounds of epidemiological inquiry *per se*. See *Reference Manual on Scientific Evidence* at 381. Epidemiology *informs* an opinion of actual causation by establishing general causation, but the link between the two will always require some degree of untested inference and uncertainty, which brings us to our third

[15] Professor Walker does not invoke this legal phrase, but the "inferential gaps" addressed in his article could have no other meaning.

point of clarification. It would be folly to require an expert to testify with *absolute certainty* that a plaintiff was exposed to a *specific* dose or suffered a *specific* risk. Dose and risk fall on a spectrum and are not "true or false." As such, any scientific estimate of those values must be expressed as one or more possible *intervals* and, for each interval, a corresponding *confidence* that the true value is within that interval. *See id*. at 117-21 (discussing the statistical "confidence interval"). Importantly—and as we discussed above—there is no logical reason to discriminate between uncertainty in the limits of epidemiological or medical knowledge and uncertainty in the particular facts of the case.[16] We rely on experts to bridge the analytical gap between general and actual causation by applying the facts of the case to existing epidemiological research about general causation. If the facts of the case must be proven with reasonable certainty (*i.e*., certainty

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[16] Professor David Rosenberg succinctly stated this principle in his article, *The Causal Connection in Mass Exposure Cases: A "Public Law" Vision of the Tort System*, when he wrote:

> The entire notion that "particularistic" evidence differs in some significant qualitative way from statistical evidence must be questioned. The concept of "particularistic" evidence suggests that there exists a form of proof that can provide direct and actual knowledge of the causal relationship between the defendant's tortious conduct and the plaintiff's injury. "Particularistic" evidence, however, is in fact no less probabilistic than is the statistical evidence that courts purport to shun. "Particularistic" evidence offers nothing more than a basis for conclusions about a perceived balance of probabilities.

97 Harv.L.Rev. 849, 870 (1984) (footnotes omitted). *See also* Walker, *Preponderance, Probability, and Warranted Factfinding*, 62 Brooklyn L. Rev. at 1080-92.

that exceeds the burden of proof), it follows that an expert must *estimate* exposure and risk with reasonable scientific or medical certainty. Here, Dr. Welch could have taken appellants' allegations as hypothetical facts and estimated the likelihood that Mrs. Dixon suffered various exposures[17] to asbestos, as well as the likely risks attendant to those possible exposures taken from epidemiological studies.[18] For example, it is a necessary truth of statistics that Dr. Welch would have been *more* confident that Mrs. Dixon suffered *at least* low asbestos exposure, but *less* confident that Mrs. Dixon suffered *at least* some *higher* level of exposure. *Id*. at 119 ("For a given confidence level, a narrower interval indicates a more precise estimate. For a given sample size, increased confidence

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[17] For the sake of simplicity, we refer to exposure in the abstract and need not account for its various relevant dimensions, such as the frequency and duration of exposure. These factors are generally accounted for in scientific literature by measuring exposure as a matter of both time and concentration. *See Toxicological Profile for Asbestos* at 17 ("Inhalation exposure is [] generally regarded as cumulative, and exposures have been expressed in terms of concentration of fibers over time or PCM fiber-years/mL (f-yr/mL)."). Thus, Dr. Welch could have inferred from existing research how likely it was that Mrs. Dixon's exposure fell above, below, or between various fiber-years/mL values.

[18] As noted, above, mesothelioma risk has been studied and estimated for asbestos exposures as low as natural atmospheric levels. Furthermore, the record contains four studies quantifying the asbestos exposure of brake mechanics: Brent L. Finley *et al.*, *Cumulative asbestos exposure for US automobile mechanics involved in brake repair (circa 1950s–2000)*, 17 J. Expo. Sci. Environ. Epidemiol. 644-55 (2007); William V. Lorimer *et al.*, *Asbestos Exposure of Brake Repair Workers in the United States*, 43 Mt. Sinai J. Med. 207-18 (1976); Arthur N. Rohl, *et al.*, *Asbestos Exposure during Brake Lining Maintenance and Repair*, 12 Envir. Research 110–128 (1976); D. E. Hickish & K. L. Knight, *Exposure to Asbestos during Brake Maintenance*, 13 Ann. Occup. Hyg. 17-21 (1970). Dr. Welch would then have to establish Mrs. Dixon's secondary exposure using further studies and estimates.

can be attained only by widening the interval."). The same would be true of risk estimates: Dr. Welch could be *more* certain of *at least* some low risk to Mrs. Dixon and *less* certain of *at least* some *higher* risk. Armed with expert estimates, rendered with reasonable certainty, the jury could then have weighed the evidence to determine whether the factual foundations and methods of the risk estimate were sound *and* whether the resultant risk was "substantial." Instead, Dr. Welch's opinion merely implied that there was some non-zero probability that Mrs. Dixon was exposed to asbestos from Ford's products, and that this resulted in some non-zero increase in her risk of contracting mesothelioma. As such, Dr. Welch's conclusion that the risk and probability of causation was "substantial" provided the jury with nothing more than her subjective opinion of "responsibility," not scientific evidence of causation.

Practical and statistical limitations may have prevented Dr. Welch from providing any particular estimates of Mrs. Dixon's exposure or relative risk, or from opining with any reasonable certainty that the probability of causation was enough that a reasonable person would consider it substantial. But lack of epidemiological data does not give an expert license to state his or her *belief* that exposure and risk—however low they may be—are "substantial." While under Rule 5-704 Dr. Welch's testimony was "not objectionable merely because it embraces an ultimate issue to be decided by the trier of fact," the remainder of her opinion lacked any information that would "assist the trier of fact to understand the evidence or to determine a fact in issue" as required by Rule 5-702.

Having erroneously gained the court's imprimatur of expert testimony, Dr. Welch's unhelpful opinion was prejudicial to Ford and demands a new trial, either without her opinion on substantiality or else with some quantitative testimony that will help the jury fulfill its charge. We therefore vacate the judgments in favor of appellants and remand the case for a new trial consistent with this opinion.

JUDGMENTS VACATED. CASE REMANDED TO THE CIRCUIT COURT FOR BALTIMORE CITY FOR NEW TRIAL CONSISTENT WITH THIS OPINION. COSTS TO BE PAID BY APPELLANTS.