IN THE SUPREME COURT OF TEXAS

NO. 10-0775

SUSAN ELAINE BOSTIC, INDIVIDUALLY AND AS PERSONAL REPRESENTATIVE OF THE HEIRS AND ESTATE OF TIMOTHY SHAWN BOSTIC, DECEASED; HELEN DONNAHOE; AND KYLE ANTHONY BOSTIC, PETITIONERS,

v.

GEORGIA-PACIFIC CORPORATION, RESPONDENT

ON PETITION FOR REVIEW FROM THE
COURT OF APPEALS FOR THE FIFTH DISTRICT OF TEXAS

Argued September 9, 2013

JUSTICE WILLETT delivered the opinion of the Court, in which CHIEF JUSTICE HECHT, JUSTICE GREEN, JUSTICE JOHNSON, and JUSTICE BROWN joined, and in all but Parts II.A.3 and II.B of which JUSTICE GUZMAN joined.

JUSTICE GUZMAN filed a concurring opinion.

JUSTICE LEHRMANN filed a dissenting opinion, in which JUSTICE BOYD and JUSTICE DEVINE joined.

In *Borg-Warner Corp. v. Flores*,[1] we addressed standards imposed by Texas law for establishing causation in asbestos-disease cases. *Flores* concerned a plaintiff suffering from asbestosis. In today's case, the plaintiffs sued for damages resulting from the suffering and death

[1] 232 S.W.3d 765 (Tex. 2007).

of a family member, Timothy Bostic (Bostic), who succumbed to mesothelioma. We hold that the standard of substantial factor causation recognized in *Flores* applies to mesothelioma cases, and write on the meaning of substantial factor causation in this context. We further hold that the plaintiffs were not required to prove that but for Bostic's exposure to Defendant Georgia-Pacific Corporation's asbestos-containing joint compound, Bostic would not have contracted mesothelioma. In this regard, we disagree with language in the court of appeals' decision. However, we agree with that court that the plaintiffs failed to offer legally sufficient evidence of causation, and accordingly affirm the court of appeals' judgment.

I. Background

In 2002 Bostic was diagnosed with mesothelioma. He was 40 years old, and died of the disease in 2003. Mesothelioma is a rare cancer of a lining of the body's internal organs. There is no dispute that asbestos, when breathed into the lungs, can cause mesothelioma. Bostic's relatives, individually and on behalf of Bostic's estate (Plaintiffs), sued Georgia-Pacific and 39 other defendants, alleging that the defendants' products exposed Bostic to asbestos and caused his disease. Plaintiffs alleged causes of action for negligence and products liability. Plaintiffs claimed that as a child and teenager Bostic had been exposed to asbestos while using Georgia-Pacific drywall joint compound.

The case went to trial in 2006. The jury found Georgia-Pacific liable under negligence and marketing defect theories, and was asked to allocate causation among numerous entities. The jury assessed 25% of the causation to Knox Glass Company, a former employer who had settled with Bostic, and 75% to Georgia-Pacific.

The trial court signed an amended judgment awarding Plaintiffs approximately $6.8 million in compensatory damages and approximately $4.8 million in punitive damages. The court of appeals concluded that the evidence of causation was legally insufficient and rendered a take-nothing judgment.[2]

II. Discussion

A. Proof of Causation in Mesothelioma Cases

The Plaintiffs contend the court of appeals erred in holding that the causation evidence was legally insufficient. In conducting a legal sufficiency review, the final test "must always be whether the evidence at trial would enable reasonable and fair-minded people to reach the verdict under review."[3] "We must view the evidence in the light most favorable to the verdict and 'must credit favorable evidence if reasonable jurors could, and disregard contrary evidence unless reasonable jurors could not.'"[4]

1. *Flores*

Flores concerned proof of causation in a case where Flores, a brake mechanic, allegedly suffering from asbestosis, sued Borg-Warner, a brake pad manufacturer. The jury found that Flores suffered from asbestos-related disease and apportioned to Borg-Warner 37% of the causation.[5] We

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[2] 320 S.W.3d 588, 590, 602.

[3] *Del Lago Partners, Inc. v. Smith*, 307 S.W.3d 762, 770 (Tex. 2010) (quoting *City of Keller v. Wilson*, 168 S.W.3d 802, 827 (Tex. 2005)).

[4] *Id.* (footnote omitted) (quoting *City of Keller*, 168 S.W.3d at 827).

[5] *Flores*, 232 S.W.3d at 768.

concluded that the causation evidence was legally insufficient.[6] We held, consistent with section 431 of the Restatement Second of Torts, that to establish causation in fact the plaintiff must prove that the defendant's product was a substantial factor in causing the disease, and that mere proof that the plaintiff was exposed to "some" respirable fibers traceable to the defendant was insufficient.[7] "The word 'substantial' is used to denote the fact that 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, rather than in the so-called 'philosophic sense,' which includes every one of the great number of events without which any happening would not have occurred."[8] We held the evidence legally insufficient because the record revealed "nothing about how much asbestos Flores might have inhaled."[9] We held that "while some respirable fibers may be released upon grinding some brake pads, the sparse record here contains no evidence of the approximate quantum of Borg-Warner fibers to which Flores was exposed, and whether this sufficiently contributed to the aggregate dose of asbestos Flores inhaled, such that it could be considered a substantial factor in causing his asbestosis."[10]

On further analysis, we held that "proof of mere frequency, regularity, and proximity is necessary but not sufficient, as it provides none of the quantitative information necessary to support

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[6] *Id.* at 774.

[7] *Id.* at 766, 770.

[8] *Id.* at 770 (quoting RESTATEMENT (SECOND) OF TORTS § 431 cmt. a (1965)).

[9] *Id.* at 771.

[10] *Id.* at 772.

causation under Texas law."[11] While the plaintiff was not required to establish causation with "mathematical precision," we required "[d]efendant-specific evidence relating to the approximate dose to which the plaintiff was exposed, coupled with evidence that the dose was a substantial factor in causing the asbestos-related disease."[12] In rejecting a standard that "some" exposure would suffice, the Court recognized: "As one commentator notes, '[i]t is not adequate to simply establish that 'some' exposure occurred. Because most chemically induced adverse health effects clearly demonstrate 'thresholds,' there must be reasonable evidence that the exposure was of sufficient magnitude to exceed the threshold before a likelihood of 'causation' can be inferred."[13]

Plaintiffs urge that the standards established in *Flores* are not fully applicable because today's case is a mesothelioma case and *Flores* was an asbestosis case. They contend that a key factual distinction between the two diseases is that relatively minute quantities of asbestos can result in mesothelioma. In *Flores*, we noted that the development of asbestosis requires a heavy exposure to asbestos, while mesothelioma may result from low levels of exposure.[14] Plaintiffs presented evidence of this same distinction.[15]

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[11] *Id.*

[12] *Id.* at 773.

[13] *Id.*

[14] *Id.* at 771.

[15] For example, one of Plaintiffs' experts, Dr. Brody, testified that "there's no safe level for mesothelioma. In other words, no one's ever been able to show a level that will prevent everyone from getting mesothelioma. Now, you can do that for asbestosis, and you can get pretty close probably for most lung cancer cases, but for mesothelioma, no one's ever shown a safe level."

While *Flores* left open the prospect of treating asbestosis and mesothelioma cases differently, we decline to do so. We believe the *Flores* framework for reviewing the legal sufficiency of causation evidence lends itself to both types of cases. In particular, we hold that even in mesothelioma cases proof of "some exposure" or "any exposure" alone will not suffice to establish causation. While the experts in this case testified that small amounts of asbestos exposure can result in mesothelioma, that fact alone does not merit a different analysis. With both asbestosis and mesothelioma, the likelihood of contracting the disease increases with the dose. As to asbestosis, we noted in *Flores* that this disease "appears to be dose-related, so that the more one is exposed, the more likely the disease is to occur, and the higher the exposure the more severe the disease is likely to be."[16] As to asbestos-related cancer, in *Flores* we discussed the California Supreme Court's decision in *Rutherford v. Owens-Illinois, Inc.*[17] That case described how expert testimony was presented from both sides establishing "that the plaintiffs' asbestos-related disease was 'dose-related'—i.e., that the risk of developing asbestos-related cancer increased as the total occupational dose of inhaled asbestos fibers increased."[18] And in today's case, Plaintiffs' experts consistently testified that all asbestos-related diseases are dose-related.[19] Plaintiffs' experts Brody, Lemen, and Hammar relied in part on the "Helsinki Conference" report,[20] a report stating that "[m]esothelioma

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[16] 232 S.W.3d at 771 (internal quotation marks omitted).

[17] 941 P.2d 1203 (Cal. 1997), *discussed in Flores*, 232 S.W.3d at 772–73.

[18] *Rutherford*, 941 P.2d at 1209.

[19] *See infra* note 96.

[20] *See infra* notes 99–100 an accompanying text.

can occur in cases with low asbestos exposure. However, very low background environmental exposures carry only an extremely low risk."

If any exposure at all were sufficient to cause mesothelioma, everyone would suffer from it or at least be at risk of contracting the disease. In *Flores* we noted that one of the plaintiff's experts acknowledged that "everyone is exposed to asbestos in the ambient air" and that "it's very plentiful in the environment, if you're a typical urban dweller."[21] In today's case, one of Plaintiffs' experts, Dr. Brody, confirmed that "[w]e all have some asbestos" in our lungs. He then explained that background levels are sufficiently low that they do not cause disease,[22] and that "multiples of fibers many times over" were required to cause mesothelioma.[23] Acceptance of an any exposure theory

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[21] 232 S.W.3d at 767.

[22] Brody testified:

> Well, so when we're talking about background, we're talking about what we all have. And it's just a fact of modern society as materials that contain asbestos break down or if you live in an area where there's naturally occurring asbestos, that asbestos will accumulate in the lung to some level, which does not produce disease. That's not a level that anyone can measure disease.

[23] Brody testified:

> Q: Can one fiber of chrysotile [asbestos] or one fiber of amosite [asbestos] cause mesothelioma?
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> A: No.
>
> Q: Okay, Do you have to have more than one?
>
> A: Yeah, of course. I mean a single fiber can cause a genetic error, but I told you that that's not enough to cause disease.
>
> Q: Okay. You have to have more than one, some number greater than one to actually cause these mutations that actually . . . cause the uncontrolled cell growth that you talked about?
>
> A: Oh, yes, you have to have many—
>
> Q: Okay.

would contradict the testimony of plaintiffs' own expert, ignore the importance of dose in determining a causative link, and impose liability even where, for all the jury can tell, the plaintiff might have become ill from his exposure to background levels of asbestos or for some other reason.[24]

More fundamentally, if we were to adopt a less demanding standard for mesothelioma cases and accept that any exposure to asbestos is sufficient to establish liability, the result essentially would be not just strict liability but absolute liability against any company whose asbestos-containing product crossed paths with the plaintiff throughout his entire lifetime. However, "[w]e have recognized that '[e]xposure to asbestos, a known carcinogen, is never healthy but fortunately does not always result in disease.'"[25] And we have never embraced the concept of industry-wide liability on grounds that proof of causation might be difficult. Instead, we have rejected such thinking and held firm to the principle that liability in tort must be based on proof of causation by a preponderance of the evidence. In a mesothelioma case, we rejected theories of collective liability—alternative liability, concert of action, enterprise liability, and market share liability—and held instead: "A fundamental principle of traditional products liability law is that the plaintiff must prove that the defendants supplied the product that caused the injury."[26] *Merrell Dow Pharmaceuticals, Inc. v. Havner*, another toxic tort case, further explains:

A: — multiples of fibers many times over to get those kinds of changes.

[24] *See Baker v. Chevron USA, Inc.*, 680 F. Supp. 2d 865, 878 n.9 (S.D. Ohio 2010) ("[S]ince benzene is ubiquitous, causation under the one-hit theory could not be established because it would be just as likely that ambient benzene was the cause of Plaintiffs' illnesses."), *aff'd*, 533 F. App'x 509 (6th Cir. 2013).

[25] *Flores*, 232 S.W.3d at 770–71.

[26] *Gaulding v. Celotex Corp.*, 772 S.W.2d 66, 68 (Tex. 1989).

> Others have argued that liability should not be allocated only on the basis of reliable proof of fault because legal rules should have the goals of "risk spreading, deterrence, allocating costs to the cheapest cost-avoider, and encouraging socially favored activities," and because "consumers of American justice want people compensated." It has been contended that "[f]or some cases that very well may mean creating a compensatory mechanism even in the absence of clear scientific proof of cause and effect" . . . . We expressly reject these views. Our legal system requires that claimants prove their cases by a preponderance of the evidence. . . . As Judge Posner has said, "[l]aw lags science; it does not lead it."[27]

If an "any exposure" theory of liability is accepted for mesothelioma cases because science has been unable to establish a dose below which the risk of disease disappears, the same theory would arguably apply to *all* carcinogens. Dr. Lemen, Plaintiffs' epidemiologist and a former Assistant Surgeon General, testified that for all carcinogens the threshold at which the risk falls to zero is unknown.[28]

The any exposure theory effectively accepts that a failure of science to determine the maximum safe dose of a toxin necessarily means that every exposure, regardless of amount, is a

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[27] 953 S.W.2d 706, 728 (Tex. 1997) (citations omitted).

[28] Lemen testified:

> Q: And isn't it true that this principle that we don't know of any safe level of exposure is true for any carcinogen?
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> A: At the present time, we aren't able to identify the carcinogenic compounds, what is safe and what is not safe. And that is true pretty much across the board for things that cause cancer.
>
> Q: So for anything on this list of carcinogens that we'll talk about later, your answer is true that if it is on the list of carcinogens, it's not just asbestos, it's the entire list that you would say we know of no safe level of exposure to it, correct?
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> A: Basically that's correct.
>
> Q: Even if it's used even today day-in and day-out in industrial and consumer products?
>
> A: That's correct. . . .

substantial factor in causing the plaintiff's illness. This approach negates the plaintiff's burden to prove causation by a preponderance of the evidence. As a federal district court reasoned in excluding the testimony of Dr. Hammar, Plaintiffs' expert on specific causation in today's case:

> Rule 702 and *Daubert* recognize above all else that to be useful to a jury an expert's opinion must be based on sufficient facts and data. The every exposure theory is based on the opposite: a lack of facts and data. . . . It seeks to avoid not only the rules of evidence but more importantly the burden of proof. . . . Dr. Hammar wants to be allowed to tell a jury that all of the plaintiff's *possible* exposures to asbestos during his entire life were contributing causes of the plaintiff's cancer, and, therefore, sufficient to support a finding of legal liability as to the manufacturer of each asbestos containing product, without regard to dosage or how long ago the exposure occurred. Just because we cannot rule anything out does not mean we can rule everything in.[29]

Further, there are cases where a plaintiff's exposure to asbestos can be tied to a defendant, but that exposure is minuscule as compared to the exposure resulting from other sources. Proof of any exposure at all from a defendant should not end the inquiry and result in automatic liability. The Restatement Third of Torts provides that "[w]hen an actor's negligent conduct constitutes only a trivial contribution to a causal set that is a factual cause of harm under § 27 [addressing multiple sufficient causes], the harm is not within the scope of the actor's liability."[30] In *Flores* we held the causation evidence legally insufficient because the record revealed "nothing about how much asbestos Flores might have inhaled" but also because Flores did not "introduce evidence regarding what percentage of that indeterminate amount may have originated in Borg-Warner products."[31] And

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[29] *Smith v. Ford Motor Co.*, 2013 WL 214378, at *2–3 (D. Utah Jan. 18, 2013) (emphasis in original).

[30] RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 36 (2010).

[31] 232 S.W.3d at 771–72.

in *Havner* we held that "if there are other plausible causes of the injury or condition that could be negated, the plaintiff must offer evidence excluding those causes with reasonable certainty."[32] That statement requires some explication in cases involving multiple exposures to the same toxin, as we discuss below, but here it properly stands for the proposition that, even in mesothelioma cases, liability cannot be imposed on every conceivable defendant whose product exposed the plaintiff to some unquantified amount of asbestos, without proof of something more. "The recent, increasingly strict exposure cases . . . reflect a welcome realization by state courts that holding defendants liable for causing asbestos-related disease when their products were responsible for only de minimis exposure to asbestos, and other parties were responsible for far greater exposure, is not just . . . ."[33]

The any exposure theory is also illogical in mesothelioma cases, where a small exposure can result in disease, because it posits that any exposure from a defendant above background levels should impose liability, while the background level of asbestos should be ignored. But the expert testimony in this case was undisputed that the background level varies considerably from location to location. We fail to see how the theory can, as a matter of logic, exclude higher than normal background levels as the cause of the plaintiff's disease, but accept that any exposure from an individual defendant, no matter how small, should be accepted as a cause in fact of the disease. Under the any exposure theory a background dose of 20 does not cause cancer, but a defendant's dose of 2 plus a background dose of 5 does.

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[32] *Havner*, 953 S.W.2d at 720.

[33] David E. Bernstein, *Getting to Causation in Toxic Tort Cases*, 74 BROOK. L. REV. 51, 59 (2008).

For these reasons, we extend the reasoning and holdings of *Flores* to mesothelioma cases, including our rejection of the "any exposure" theory of liability, with the clarifications discussed below.

2. But For Causation

Plaintiffs complain that the court of appeals erred in requiring them to prove but for causation in addition to substantial factor causation. The term "but for causation" may encompass several meanings. As we attempt to clarify, "but for" and "substantial factor" are overlapping concepts and, to the extent they embody different tests, application of those tests usually lead to the same result. But here we are concerned that the court of appeals' decision might be read to require satisfying a proof requirement that but for Bostic's exposure to Georgia-Pacific's products, he would not have contracted mesothelioma. We agree with Plaintiffs that language in the court of appeals' decision appears to require such proof. The court stated that "[b]oth producing and proximate cause contain the cause-in-fact element, which requires that the defendant's act be a substantial factor in bringing about the injury and without which the harm would not have occurred."[34] It stated, "'In asbestos cases, then, we must determine whether the asbestos in the defendant's product was a substantial factor in bringing about the plaintiff's injuries,' and without which the injuries would not have occurred."[35] In doing so, the court of appeals quoted from *Flores* but appended but for language to the end of its sentence. The court expressly disagreed with Plaintiffs' assertion that *Flores* did not

[34] 320 S.W.3d at 596 (quoting *Metro Allied Ins. Agency, Inc. v. Lin*, 304 S.W.3d 830, 835 (Tex. 2009) (internal quotation marks omitted)).

[35] *Id.* (quoting *Flores*, 232 S.W.3d at 770).

require proof of but for causation.[36] It then concluded that the testimony of Dr. Hammar was wanting because "he could not opine that Timothy would not have developed mesothelioma absent exposure to Georgia-Pacific asbestos-containing joint compound."[37]

To a point, we agree with Georgia-Pacific that but for causation is a recognized standard for proof of producing cause, also known as causation in fact,[38] applicable to this products liability case.[39] We have often recognized but for causation, alone or in combination with substantial factor causation, as the standard for establishing causation in fact.[40] Indeed, "to say of a cause of an injury

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[36] *Id.* ("[Plaintiffs] assert that *Flores* does not require 'but-for' causation in proving specific causation and that *Flores* requires only that [Plaintiffs] prove Timothy's exposure to Georgia-Pacific asbestos-containing joint compound was a 'substantial factor' in contributing to his risk of mesothelioma. We disagree.").

[37] *Id.*

[38] *See Transcon. Ins. Co. v. Crump*, 330 S.W.3d 211, 223 (Tex. 2010) (recognizing that "the producing cause inquiry is conceptually identical to that of cause in fact").

[39] Producing cause is the level of causation applicable to products liability cases. *See, e.g.*, *Rourke v. Garza*, 530 S.W.2d 794, 801 (Tex. 1975). Plaintiffs sued under theories of negligence and products liability, the latter being based on a marketing defect theory. However, Plaintiffs concede that but for causation was required under their negligence theory of liability because the jury was instructed that proximate cause, a necessary element of negligence liability, required proof of but for causation. The jury was instructed that proximate cause "means that cause which, in a natural and continuous sequence, produces an event, and without which cause such event would not have occurred." *See City of Fort Worth v. Zimlich*, 29 S.W.3d 62, 71 (Tex. 2000) ("Since neither party objected to this instruction, we are bound to review the evidence in light of this definition.").

[40] *E.g.*, *Crump*, 330 S.W.3d at 222–23 ("Cause in fact is established when the act or omission was a substantial factor in bringing about the injuries, and without it, the harm would not have occurred.") (quoting *IHS Cedars Treatment Ctr. v. Mason*, 143 S.W.3d 794, 799 (Tex. 2004)); *Marathon Corp. v. Pitzner*, 106 S.W.3d 724, 727 (Tex. 2003) ("The test for cause in fact, or 'but for causation,' is whether the act or omission was a substantial factor in causing the injury 'without which the harm would not have occurred.'"); *Union Pump Co. v. Allbritton*, 898 S.W.2d 773, 775 (Tex. 1995) ("Cause in fact means that the defendant's act or omission was a substantial factor in bringing about the injury which would not otherwise have occurred."); *Gen. Motors Corp. v. Saenz*, 873 S.W.2d 353, 357 (Tex. 1993) (holding that to establish causation in fact element common to both negligence and products liability causes of action, "plaintiffs must show that but for GM's omission the accident would not have occurred").

that it is one 'but for which the injury would not have happened' is to repeat something already included in the usual and ordinary meaning of the word 'cause.'"[41]

Nor is there anything unusual in our recognizing but for causation as the causation standard in tort cases. The Restatement Second of Torts in section 431 generally recognizes that an "actor's negligent conduct is a legal cause of harm to another if [] his conduct is a substantial factor in bringing about the harm."[42] Comment *a* to this section makes clear that, as a general proposition, substantial factor causation incorporates the concept of but for causation: "In order to be a legal cause of another's harm, it is not enough that the harm would not have occurred had the actor not been negligent. Except as stated in § 432(2), this is necessary, but it is not of itself sufficient."[43] Hence, the comment indicates that but for causation is generally a component of substantial factor causation.

The Restatement Third of Torts likewise embraces but for causation as the general causation standard in tort cases. Section 26 of the subtitle on Liability for Physical and Emotional Harm

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[41] *Tex. Indem. Ins. Co. v. Staggs*, 134 S.W.2d 1026, 1030 (Tex. 1940) (quoting *Tex. & Pac. Ry. v. Short*, 62 S.W.2d 995, 999 (Tex. App.—Eastland 1933, writ ref'd)).

[42] RESTATEMENT (SECOND) OF TORTS § 431 (1965). This provision addresses negligence liability, and as noted today's case is, for our purposes, a products liability case. *See supra* note 39. However, the element of causation in fact is the same under the two theories of liability. To recover under a negligence theory, the plaintiff must establish proximate causation, while recovery under a products liability theory requires proof of producing causation. Proximate cause and producing cause share the common element of causation in fact, with proximate cause including the additional element of foreseeability. *See Crump*, 330 S.W.3d at 222–23; *Flores*, 232 S.W.3d at 770; *Union Pump*, 898 S.W.2d at 775; *Saenz*, 873 S.W.2d at 356; *see also* RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIABILITY § 15 (1998) ("Whether a product defect caused harm to persons or property is determined by the prevailing rules and principles governing causation in tort."); RESTATEMENT (SECOND) OF TORTS § 431 cmt. e (1965) ("Although the rules stated in this Section are stated in terms of the actor's negligent conduct, they are equally applicable where the conduct is intended to cause harm, or where it is such as to result in strict liability.").

[43] RESTATEMENT (SECOND) OF TORTS § 431 cmt. a (1965).

provides: "Tortious conduct must be a factual cause of harm for liability to be imposed. Conduct is a factual cause of harm when the harm would not have occurred absent the conduct."[44] The Restatement Third not only embraces but for causation, but includes some criticism of the substantial factor test.[45]

However, we follow *Flores* and conclude that in products liability cases where the plaintiff was exposed to multiple sources of asbestos, substantial factor causation is the appropriate basic standard of causation without including as a separate requirement that the plaintiff meet a strict but for causation test. Due to the nature of the disease process, which can occur over decades and involve multiple sources of exposure, establishing which fibers from which defendant actually caused the disease is not always humanly possible. Even if the exposure from a particular defendant was by itself sufficient to cause the disease, in multiple-exposure cases the plaintiff may find it impossible to show that he would not have become ill but for the exposure from that defendant.

In *Flores* we recognized "the proof difficulties accompanying asbestos claims. The long latency period for asbestos-related diseases, coupled with the inability to trace precisely which fibers caused disease and from whose product they emanated, make this process inexact."[46] Along similar lines, the Virginia Supreme Court recently observed that "if the traditional but-for definition of proximate cause was invoked, the injured party would virtually never be able to recover for damages

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[44] RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 26 (2010).

[45] *See id.*§ 26 cmt. j.

[46] 232 S.W.3d at 772.

arising from mesothelioma in the context of multiple exposures . . . ."[47] Further, in *Flores* we quoted from *Rutherford*:

> Plaintiffs cannot be expected to prove the scientifically unknown details of carcinogenesis, or trace the unknowable path of a given asbestos fiber. . . . Instead, we can bridge this gap in the humanly knowable by holding that plaintiffs may prove causation in asbestos-related cancer cases by demonstrating that the plaintiff's exposure to defendant's asbestos-containing product in reasonable medical probability was a substantial factor in contributing to the aggregate *dose* of asbestos the plaintiff or decedent inhaled or ingested, and hence to the *risk* of developing asbestos-related cancer, without the need to demonstrate that fibers from the defendant's particular product were the ones, or among the ones, that *actually* produced the malignant growth.[48]

This language is inconsistent with a strict requirement of proving that but for the particular fibers traceable to the sued defendant, the plaintiff would not have become ill. In *Flores* we keyed on substantial factor causation, and did not require proof of but for causation. The absence of but for language in *Flores* was not inadvertent.

Again, our approach did not break new ground. While but for causation is a core concept in tort law, it yields to the more general substantial factor causation in situations where proof of but for causation is not practically possible or such proof otherwise should not be required. A leading treatise has observed that the substantial factor approach "in the great majority of cases . . . produces the same legal conclusion as the but-for test," but "was developed primarily for cases in which application of the but-for rule would allow each defendant to escape responsibility because the

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[47] *Boomer v. Ford Motor Co.*,736 S.E.2d 724, 729 (Va. 2013).

[48] 941 P.2d at 1219 (footnote omitted) (emphasis in original), *quoted in Flores*, 232 S.W.3d at 772–73.

conduct of one or more others would have been sufficient to produce the same result."[49] Likewise, *Rutherford* reasoned that "[t]he substantial factor standard generally produces the same results as does the 'but for' rule of causation," but the substantial factor test "has been embraced as a clearer rule of causation—one which subsumes the 'but for' test while reaching beyond it to satisfactorily address other situations, such as those involving independent or concurrent causes in fact."[50] This problem arises in toxic tort cases such as *Flores*, *Boomer*, *Rutherford*, and today's case, where the plaintiff has suffered exposure from multiple sources.

The Restatement Second of Torts likewise recognizes an alternative to strict but for causation in certain cases involving multiple causes of injury. While, as noted, section 431 and its comment *a* generally require but for causation, comment *a* further notes that this rule applies "[e]xcept as stated in § 432(2)." Section 432(2) addresses cases involving multiple causation: "If two forces are actively operating, one because of the actor's negligence, the other not because of any misconduct on his part, and each of itself is sufficient to bring about harm to another, the actor's negligence may be found to be a substantial factor in bringing it about."[51] Section 432(2) recognizes a scenario where the actor's conduct is not, strictly speaking, a but for cause, because another force would have caused the harm anyway.

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[49] W. PAGE KEETON ET AL., PROSSER AND KEETON ON THE LAW OF TORTS § 41 (5th ed. 1984).

[50] 941 P.2d at 1214.

[51] RESTATEMENT (SECOND) OF TORTS § 432(2) (1965).

Likewise, while the Restatement Third generally embraces but for causation in section 26,[52] as noted above, it elsewhere still recognizes substantial factor causation in some products liability cases[53] and in a sense recognizes the converse of substantial factor causation, by providing in section 36 that "[w]hen an actor's negligent conduct constitutes only a trivial contribution to a causal set that is a factual cause of harm under § 27, the harm is not within the scope of the actor's liability."[54] So while not requiring substantial factor causation in section 26, which sets out the general causation standard, it recognizes in the negative that a trivial contribution to causation will not suffice. This rule hardly represents a sea change, as section 433(a) of the Restatement and Restatement Second have long stated that, in making a substantial factor determination, an important consideration is "the number of other factors which contribute in producing the harm and the extent of the effect which they have in producing it."[55] Further, while section 26 moves away from the substantial factor standard, comment *j* to that section explains its particular usefulness in certain multiple-causation cases.[56]

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[52] RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 26 (2010).

[53] *See* RESTATEMENT (THIRD) OF TORTS: PRODUCTS LIABILITY § 16(a) (1998) ("When a product is defective at the time of commercial sale or other distribution and the defect is a substantial factor in increasing the plaintiff's harm beyond that which would have resulted from other causes, the product seller is subject to liability for the increased harm.").

[54] RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 36 (2010).

[55] RESTATEMENT (SECOND) OF TORTS § 433(a) (1965); RESTATEMENT OF TORTS § 433(a) (1934).

[56] RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 26 cmt. j (2010) (stating that the "primary function" of the substantial-factor test "was to permit the factfinder to decide that factual cause existed when there were multiple sufficient causes—each of two separate causal chains sufficient to bring about the plaintiff's harm, thereby rendering neither a but-for cause").

Moreover, the Restatement Third, like the earlier Restatements, does not require strict but for causation in a toxic tort multiple-exposure case like today's case. Section 26 generally requires but for causation, by stating that "[c]onduct is a factual cause of harm when the harm would not have occurred absent the conduct." However, section 26 ends by stating that "[t]ortious conduct may also be a factual cause of harm under section 27." Section 27 addresses cases of multiple causation and states: "If multiple acts occur, each of which under § 26 alone would have been a factual cause of the physical harm at the same time in the absence of the other act(s), each act is regarded as a factual cause of the harm."[57] Read in a vacuum, sections 26 and 27 might appear to require strict but for causation for each defendant in a multiple-exposure case where the exposures did not occur "at the same time," the position we understand Georgia-Pacific to take. These sections are fraught with complexities and what if scenarios, set out in many comments and illustrations. Comment *f* to section 27 states:

> In some cases, tortious conduct by one actor is insufficient, even with other background causes, to cause the plaintiff's harm. Nevertheless, when combined with conduct by other persons, the conduct overdetermines the harm, i.e., is more than sufficient to cause the harm. . . . The fact that an actor's conduct requires other conduct to be sufficient to cause another's harm does not obviate the applicability of this Section.[58]

And comment *g* posits the scenario closest to our case:

> [T]he situation addressed in Comment *f* has occurred most frequently in cases in which persons have been exposed to multiple doses of a toxic agent. When a person contracts a disease such as cancer, and sues multiple actors claiming that each provided some dose of a toxic substance that caused the disease, the question of the

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[57] *Id.* § 27.

[58] *Id.* § 27 cmt. f.

> causal role of each defendant's toxic substance arises. Assuming that there is some threshold dose sufficient to cause the disease, the person may have been exposed to doses in excess of the threshold before contracting the disease. Thus, some or all of the person's exposures may not have been but-for causes of the disease. Nevertheless, each of the exposures prior to the person's contracting the disease . . . is a factual cause of the person's disease under the rule in this Section.[59]

In short, we do not think the Restatements, in their attempts to synthesize many decades of tort law, would require the plaintiffs to meet a strict but for causation test in a case like today's case. More importantly, our controlling decision in *Flores* does not impose this requirement. Accordingly, we hold that Plaintiffs were required to establish substantial factor causation, but were not required to prove that but for Bostic's exposure to Georgia-Pacific's products, he would not have contracted mesothelioma. The court of appeals erred insofar as it stated otherwise.

3. Further Analysis, under *Havner*, of Substantial Causation in Asbestos Cases

We write further on the meaning of substantial factor causation in asbestos cases. First, we note that for all the refinements *Flores* places on the substantial causation standard, we also believe that some discretion must be ceded to the trier of fact in determining whether the plaintiff met that standard. One respected treatise has opined that it is "neither possible nor desirable to reduce [substantial factor] to any lower terms."[60]

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[59] *Id.* § 27 cmt. g.

[60] W. PAGE KEETON ET AL., PROSSER AND KEETON ON THE LAW OF TORTS § 41 (5th ed. 1984); *see also id.* § 41, at n.30 ("Hart and Honoré . . . object strongly to the phrase as undefinable. So, Green suggests is 'reasonable,' but that does not prevent its use to pose an issue for the jury.").

We recognized a quantitative approach to causation in *Merrell Dow Pharmaceuticals, Inc. v. Havner*,[61] and Georgia-Pacific urges use of that approach in today's case. *Havner* provides useful insights that should be integrated with our analysis here.

In *Havner*, the plaintiffs sued on behalf of a child born with birth defects allegedly caused by a drug, Bendectin, taken by the mother while she was pregnant. The Court held that the expert testimony, which relied in part on epidemiological studies, was legally insufficient to establish causation.[62] We recognized that epidemiological studies showing that the population exposed to a toxin faced more than double the risk of injury facing the unexposed or general population could be used to establish causation.[63]

While recognizing that causation might be established through epidemiological studies showing more than a doubling of the risk, also described as a relative risk of more than 2.0,[64] we recognized that this requirement was not a "litmus test" or "bright-line boundary" and that a single study would not suffice to establish legal causation.[65] We discussed several other indicia of scientific validity. A claimant must show that his circumstances are similar to the group analyzed in the study.[66] We observed that scientific studies also consider the "significance level" or "confidence

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[61] 953 S.W.2d 706 (Tex. 1997).

[62] *Id.* at 708, 730.

[63] *Id.* at 717–18.

[64] *See id.* at 718; *see also id.* at 721 ("For the result to indicate a doubling of the risk, the relative risk must be greater than 2.0.").

[65] *Id.* at 718–19, 727.

[66] *Id.* at 720.

level," that the generally accepted confidence level is 95%,[67] and that statistical significance also requires a "confidence interval" that does not include the number 1.[68] We noted that "[t]here are many other factors to consider in evaluating the reliability of a scientific study including, but certainly not limited to, the sample size of the study, the power of the study, confounding variables, and whether there was selection bias."[69] We also noted that courts must be "especially skeptical of scientific evidence that has not been published or subjected to peer review,"[70] and that "[a] related factor . . . is whether the study was prepared only for litigation."[71]

Havner is a foundational part of our jurisprudence. We have never held that it applies universally to all tort cases where causation is an issue.[72] It offers an alternative method of establishing causation "[i]n the absence of direct, scientifically reliable proof of causation."[73] To some extent *Havner*'s discussion of the use of scientific studies addressed whether those studies

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[67] *Id.* at 723–24.

[68] *Id.* at 723.

[69] *Id.* at 724.

[70] *Id.* at 727 (internal quotation marks omitted); *see also E.I. du Pont de Nemours & Co. v. Robinson*, 923 S.W.2d 549, 557 (Tex. 1995) (holding that one factor in deciding reliability of expert testimony is "whether the theory has been subjected to peer review and/or publication").

[71] *Havner*, 953 S.W.2d at 726.

[72] For example, we noted in *Flores* that epidemiological studies discussed in *Havner* "are not necessary to prove causation" though properly designed studies can serve as part of the evidence establishing causation. *Flores*, 232 S.W.3d at 772.

[73] *Havner*, 953 S.W.2d at 715.

supported general causation—the issue of whether Bendectin was capable of causing birth defects.[74] Epidemiological studies by their nature address general causation by analyzing a cohort of individuals,[75] rather than specific causation—the jury issue of whether the defendant's product caused the specific injury in issue,[76] but these studies are sometimes used effectively by experts to help establish specific causation, as *Havner* recognized.[77] In today's case, general causation is not an issue. Georgia-Pacific does not dispute, for purposes of this appeal, that exposure to asbestos fibers can cause mesothelioma.[78]

Despite differences between *Havner* and today's case, *Havner*'s focus on proof of more than a doubling of risk, as established by scientifically reliable studies, is premised on fundamental principles of tort law that have application here. *Havner*'s discussion of epidemiological studies was based on the tenet in our law that expert testimony on causation must be scientifically reliable. "If

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[74] *Id.* ("The Havners rely to a considerable extent on epidemiological studies for proof of general causation."); *see also Merck & Co. v. Garza*, 347 S.W.3d 256, 265 (Tex. 2011) ("*Havner* holds . . . that when parties attempt to prove general causation using epidemiological evidence, a threshold requirement of reliability is that the evidence demonstrate a statistically significant doubling of the risk.").

[75] *See Havner*, 953 S.W.2d at 715 ("Epidemiological studies examine existing populations to attempt to determine if there is an association between a disease or condition and a factor suspected of causing that disease or condition.").

[76] *Id.* at 714 ("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.").

[77] *Id.* at 715.

[78] Some of the Georgia-Pacific drywall compound to which Bostic was allegedly exposed contained chrysotile asbestos fibers. Plaintiffs' experts testified that the prevailing scientific consensus is that chrysotile fibers can cause mesothelioma. While Georgia-Pacific contends that a scientific debate continues as to whether inhalation of chrysotile fibers causes mesothelioma, it states in its principal brief that it is not challenging "the assumption that exposure to chrysotile can cause mesothelioma."

the expert's scientific testimony is not reliable, it is not evidence."[79] We discussed our decision in *E.I. du Pont de Nemours and Co. v. Robinson*,[80] where we analyzed the issue of expert reliability.[81] As recognized in *Robinson,* "In addition to being relevant, the underlying scientific technique or principle must be reliable. Scientific evidence which is not grounded in the methods and procedures of science is no more than subjective belief or unsupported speculation. Unreliable evidence is of no assistance to the trier of fact and is therefore inadmissible under Rule 702."[82]

Havner also held that, notwithstanding competing policies of deterrence, risk-avoidance, or compensating innocent injured parties, "[o]ur legal system requires that claimants prove their cases by a preponderance of the evidence," and we rejected all rationales for adopting a lesser burden of proof.[83] In concluding that studies showing more than a doubling of the risk may be supportive of legal causation, provided that other indicia of reliability are met, we explained that this standard corresponds to the legal requirement that the plaintiff prove his case by a preponderance of the evidence:

> Recognizing that epidemiological studies cannot establish the actual cause of an individual's injury or condition, a difficult question for the courts is how a plaintiff faced with this conundrum can raise a fact issue on causation and meet the "more likely than not" burden of proof.
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[79] *Id.* at 713.

[80] 923 S.W.2d 549 (Tex. 1995).

[81] *Havner*, 953 S.W.2d at 712, 714.

[82] *Robinson*, 923 S.W.2d at 557 (citation, internal quotation marks omitted).

[83] 953 S.W.2d at 728.

> Other courts have likewise found that the requirement of a more than 50% probability means that epidemiological evidence must show that the risk of an injury or condition in the exposed population was more than double the risk in the unexposed or control population.
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> * * *
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> Although we recognize that there is not a precise fit between science and legal burdens of proof, we are persuaded that properly designed and executed epidemiological studies may be part of the evidence supporting causation in a toxic tort case and that there is a rational basis for relating the requirement that there be more than a "doubling of the risk" to our no evidence standard of review and to the more likely than not burden of proof.
>
> Assume that a condition naturally occurs in six out of 1,000 people even when they are not exposed to a certain drug. If studies of people who *did* take the drug show that nine out of 1,000 contracted the disease, it is still more likely than not that causes other than the drug were responsible for any given occurrence of the disease . . . . However, if more than twelve out of 1,000 who take the drug contract the disease, then it may be *statistically* more likely than not that a given individual's disease was caused by the drug.
>
> This is an oversimplification of statistical evidence relating to general causation . . . but it illustrates the thinking behind the doubling of the risk requirement.
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> * * *
>
> [T]he law must balance the need to compensate those who have been injured by the wrongful actions of another with the concept deeply imbedded in our jurisprudence that a defendant cannot be found liable for an injury unless the preponderance of the evidence supports cause in fact. 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.[84]

In sum, *Havner* enunciated principles in toxic tort cases that (1) expert testimony of causation must be scientifically reliable, (2) the plaintiff must establish the elements of his claim by a preponderance of the evidence, and (3) where direct evidence of causation is lacking, scientifically reliable evidence in the form of epidemiological studies showing that the defendant's product more than doubled the plaintiff's risk of injury appropriately corresponds to the legal standard of proof by

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[84] *Id*. at 715–18 (citations omitted) (emphasis in original).

a preponderance of the evidence. These principles should apply to asbestos cases. As to the availability of scientific studies, asbestos-related disease has been researched for many decades and the population of potentially affected persons numbers in the millions. Dr. Lemen, one of Plaintiffs' experts, testified that many millions of people have been exposed to chrysotile asbestos from manmade sources, that a scientific consensus that asbestos causes serious illness has existed since 1930, that a statistically significant link between asbestos and mesothelioma was shown in 1963, and that by 1965 over a thousand publications discussed asbestos disease. We observed over 15 years ago that "[a]sbestos litigation, particularly asbestos products cases, has achieved maturity."[85] We therefore conclude that in the absence of direct proof of causation, establishing causation in fact against a defendant in an asbestos-related disease case requires scientifically reliable proof that the plaintiff's exposure to the defendant's product more than doubled his risk of contracting the disease. A more than doubling of the risk must be shown through reliable expert testimony that is based on epidemiological studies or similarly reliable scientific testimony.

Multiple-exposure cases raise the issues of how the finder of fact should consider exposure from sources other than the defendant, what proof might be required as to those other sources, and who has the burden of proof regarding those other sources. These are difficult questions.

We recognized in *Havner*, generally, that "if there are other plausible causes of the injury or condition that could be negated, the plaintiff must offer evidence excluding those causes with reasonable certainty."[86] We think this statement in *Havner* is correct but cannot be applied without

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[85] *In re Ethyl Corp.*, 975 S.W.2d 606, 610 (Tex. 1998).

[86] 953 S.W.2d at 720.

qualification to cases involving multiple sources of exposure to the same toxin. We think the plaintiff should be required to establish more than a doubling of the risk attributable to the defendant's product, for the reasons discussed, but do not think it necessary or fair to require a plaintiff to track down every possible source of asbestos exposure and disprove that those other exposures caused the disease. Strict application of *Havner*'s requirement of ruling out all other possible causes of disease would in effect re-introduce a strict but for requirement, which for reasons already discussed is not appropriate in a multiple-exposure case like today's case. Our law accepts that in cases of multiple exposure multiple defendants may be held liable for causing the plaintiff's disease. And in multiple-exposure cases few if any plaintiffs could ever establish which particular fibers from which particular defendant caused the disease, and we do not believe the plaintiff should be required to quantify the exposure from every other conceivable source, occurring perhaps over a period of decades.

However, when evidence is introduced of exposure from other defendants or other sources, proof of more than a doubling of the risk may not suffice to establish substantial factor causation. In the Restatement Second of Torts, and as quoted by our Court in *Flores*, substantial factor causation "denote[s] the fact that 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, rather than in the so-called 'philosophic sense,' which includes every one of the great number of events without which any happening would not have

occurred."[87] The law should retain this concept. Along the same lines, the Restatement Third recognizes that a defendant's trivial contribution to multiple causes will not result in liability.[88]

Suppose a plaintiff shows that his exposure to a defendant's product more than doubled his chances of contracting a disease, but the evidence at trial also established that another source of the toxin increased the chances by a factor of 10,000. In this circumstance, a trier of fact or a court reviewing the sufficiency of the evidence should be allowed to conclude that the defendant's product was not a substantial factor in causing the disease.

JUSTICE LEHRMANN presents a thorough and thought-provoking dissent, but we cannot agree with its ultimate conclusion that the evidence of causation was legally sufficient in this case. The dissent contends that *Havner* primarily focused on general causation. As noted above, *Havner* was concerned with general causation while today's case is not. But *Havner* was also concerned with specific causation. General causation is never the ultimate issue of causation tried to the finder of fact in a toxic tort case. The ultimate issue is always specific causation—whether the defendant's product caused the plaintiff's injury. General causation as established through epidemiological studies is relevant only insofar as it informs specific causation. In *Havner*, we held that where direct evidence of specific causation is unavailable, specific causation may be established through an alternative two-step process whereby the plaintiff establishes general causation through reliable

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[87] *Flores*, 232 S.W.3d at 770 (quoting RESTATEMENT (SECOND) OF TORTS § 431 cmt. a (1965)).

[88] RESTATEMENT (THIRD) OF TORTS: LIABILITY FOR PHYSICAL AND EMOTIONAL HARM § 36 (2010).

studies, and then demonstrates that his circumstances are similar to the subjects of the studies.[89] By meeting these requirements, the plaintiff shows that his exposure to the defendant's product more than doubled his individual risk and thereby establishes specific causation. *Havner* is, therefore, relevant to our analysis today. Its recognition that every plaintiff must prove his case by a preponderance of the evidence has application here.

The dissent suggests that our analysis is flawed because specific causation as explicated in *Havner* is different from substantial factor causation. We disagree. "Substantial factor" is a term we use to describe the level of proof required to establish specific causation, which is always an element of the plaintiff's case.

The dissent argues that *Havner* is inapplicable to multiple-exposure cases. We are at a loss to understand why. If exposure from other sources were irrelevant when we decided *Havner*, we would not have stated that other causes of the disease should be excluded,[90] a requirement we actually relax in today's case because of the special difficulties encountered in multiple-exposure cases, as discussed above. But we think *Havner*'s requirement of proof of a more than doubling of

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[89] *See Havner*, 953 S.W.2d at 715 ("[I]n many toxic tort cases . . . there will be no reliable evidence of specific causation. In the absence of direct, scientifically reliable proof of causation, claimants may attempt to demonstrate that exposure to the substance at issue increases the risk of their particular injury. The finder of fact is asked to infer that because the risk is demonstrably greater in the general population due to exposure to the substance, the claimant's injury was more likely than not caused by that substance. Such a theory . . . is based on a policy determination that when the incidence of a disease or injury is sufficiently elevated due to exposure to a substance, someone who was exposed to that substance and exhibits the disease or injury can raise a fact question on causation."); *id.* at 720 ("To raise a fact issue on causation and thus to survive legal sufficiency review, a claimant must do more than simply introduce into evidence epidemiological studies that show a substantially elevated risk. A claimant must show that he or she is similar to those in the studies."); *see also Merck*, 347 S.W.3d at 265 ("*Havner* holds . . . that when parties attempt to prove general causation using epidemiological evidence, a threshold requirement of reliability is that the evidence demonstrate a statistically significant doubling of the risk. In addition, *Havner* requires that a plaintiff show 'that he or she is similar to [the subjects] in the studies . . . .'").

[90] 953 S.W.2d at 720.

the risk is particularly useful in multiple-exposure cases where the alternative is to abdicate resort to scientifically reliable proof and accept that any exposure will suffice.

The dissent also suggests that we would require the application of *Havner* even in cases where the only conceivable source of exposure to a toxin is the defendant. If the plaintiff can establish with reliable expert testimony that (1) his exposure to a particular toxin is the only possible cause of his disease, and (2) the only possible source of that toxin is the defendant's product (or, in another of the dissent's hypotheticals, the products of two defendants whose combined doses established the required threshold dose to cause disease), this proof might amount to direct proof of causation and the alternative approach embraced in *Havner* might be unnecessary. These hypotheticals certainly do not apply to today's case, as discussed further below. Plaintiffs never claimed that Georgia-Pacific was the only source of Bostic's exposure or that combined exposures from multiple defendants were needed to cause his disease.[91] Plaintiffs tried the case in exactly the opposite manner, by insisting that any exposure to asbestos beyond background exposure should be treated as a cause of Bostic's disease. Further, in the real world of complex environments and complex organisms, we think that science is often limited to establishing probabilities. *Havner*'s

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[91] On the latter point, the following exchange occurred at oral argument:

> Q: [Y]ou argue, as I understand it, that if there hadn't been any other exposure, the exposure to Georgia-Pacific product was enough.
>
> A: Correct, Your Honor, which is what—
>
> Q: So you're not making the argument that even though it wasn't enough, if you add it in with everything else, that would have been enough.
>
> A: No. We are not at all, and I want to be very clear on that . . . .

recognition that science must sometimes resort to probabilistic approaches is hardly a valid criticism of that decision. We assume the dissent has no quarrel with quantum mechanics. Establishing the direct proof posited in the dissent's hypotheticals might prove far more difficult than the method of proof sanctioned in *Havner*. Excluding the universe of all other possible causes, which we do not require, might prove more daunting than what we do require. And even in a single-exposure case, we think that proof of dose would be required, because as *Flores* noted, "One of toxicology's central tenets is that the dose makes the poison."[92] As explained below, dose was not established in this case.

4. Recapitulation

We conclude that in all asbestos cases involving multiple sources of exposure, including mesothelioma cases, the standards for proof of causation in fact are the same. In reviewing the legal sufficiency of the evidence:

- proof of "any exposure" to a defendant's product will not suffice and instead the plaintiff must establish the dose of asbestos fibers to which he was exposed by his exposure to the defendant's product;
- the dose must be quantified but need not be established with mathematical precision;
- the plaintiff must establish that the defendant's product was a substantial factor in causing the plaintiff's disease;

[92] *Flores*, 232 S.W.3d at 770 (internal quotation marks omitted).

- the defendant's product is not a substantial factor in causing the plaintiff's disease if, in light of the evidence of the plaintiff's total exposure to asbestos or other toxins, reasonable persons would not regard the defendant's product as a cause of the disease;
- to establish substantial factor causation in the absence of direct evidence of causation, the plaintiff must prove with scientifically reliable expert testimony that the plaintiff's exposure to the defendant's product more than doubled the plaintiff's risk of contracting the disease.

B. Proof of Causation in This Case

Georgia-Pacific manufactured and sold asbestos-containing joint compound from 1965 to 1977. Bostic was born in 1962 and turned 15 in 1977. The joint compound was sold in a dry-mix form, to which water was added to make drywall "mud," and a pre-mixed form. The compound was used to smooth cracks and joints during drywall installation and repair. During the 1965–77 period, the compound contained chrysotile asbestos,[93] the most common form of asbestos used commercially. Asbestos fibers can become airborne when dry compound is sanded, mixed, or swept as part of normal drywall work.

Bostic and his father Harold Bostic (Harold) testified by deposition at trial. Bostic's exposure to asbestos-containing Georgia-Pacific products occurred when, as a child and teenager, he assisted Harold in remodeling projects for friends and family. Plaintiffs contend that Bostic's exposure as a child is particularly significant since several experts agreed that children are especially

[93] As noted above, *see supra* note 78, Georgia-Pacific does not dispute for purposes of this appeal that exposure to chrysotile asbestos fibers can cause mesothelioma. "Chrysotile asbestos is the most abundant type of asbestos fiber and is a serpentine fiber consisting of 'pliable curly fibrils which resemble scrolled tubes.'" *Flores*, 232 S.W.3d at 766 n.4.

vulnerable to exposure to asbestos and carcinogens in general. Bostic helped his father mix and sand drywall compound from the age of five. Plaintiffs contend that Bostic was also exposed to asbestos from exposure to Harold's clothing. Bostic lived with his father until his parents divorced in 1972, when he was 9, and he stayed with his father thereafter on weekends, holidays, and at times during the summer.

Harold testified that he performed drywall work on various projects during the relevant period. He testified that he used Georgia-Pacific drywall compounds "[l]ike 98% of the time." Bostic assisted Harold on projects during the 1967–77 time frame when Georgia-Pacific drywall compound contained asbestos. Harold testified that he and Bostic used Georgia-Pacific compound "[m]any, many, many times." He was able to recall specifically eight projects during the relevant period, although he thought there were other projects he simply could not recall. Of the specific projects he could recall, he specifically identified one where Georgia-Pacific compound was used, a job where he constructed a kit house for a friend. He could not recall whether Bostic was present when drywall work was done on this project. Bostic could not recall with certainty ever using Georgia-Pacific drywall products during the relevant 1967–77 period.

Bostic was exposed to asbestos from Knox Glass Company. Harold was employed at Knox Glass from 1962 until 1984. Bostic lived with his father until his parents divorced and sometimes stayed with his father after 1972 as noted above. He also lived with his father from ages 15 to 18. Bostic worked at Knox Glass in the summers of 1980, 1981, and 1982. While Plaintiffs point to Bostic's testimony that he spent only about three months during these summers in the "hot end" of the plant where asbestos was prevalent, he testified that he frequently worked 16 hours a day as "a

relief hot end worker." Asbestos was used in products extensively at the plant, in cements, fireproofing, asbestos cloth, pumps, packing, valves, furnaces, and other products. Bostic's work included cutting asbestos cloth, cleaning up after asbestos pipe insulation was repaired, removing and replacing asbestos from machines, and wearing asbestos gloves. One of his main jobs was cutting asbestos cloth. He had no respiratory protection. He was exposed to asbestos from the Knox Glass plant due to his own employment and also from exposure to asbestos brought home on his father's clothes. Bostic and Harold participated in a study finding that 27% of workers at the plant had developed asbestos-related illnesses, although the duration of Bostic's employment at the plant was at the low end of the employees studied.

Bostic was exposed to asbestos while employed by another company, Palestine Contractors, in 1977 and 1978, and while working alone and with his father on automobiles with brake pads and other parts that contained asbestos. As an adult Bostic was also exposed to asbestos while doing remodeling work, where he was exposed to shingles, tiles, and other asbestos-containing building materials that were not manufactured by Georgia-Pacific. His primary employment, from 1984 until he stopped working due to his illness at the end of 2002, was as a correctional officer with the Texas Department of Criminal Justice (TDCJ). He did not claim exposure to asbestos from this employment.

Work history sheets provide certain details of Bostic's work history. These were based on information provided by Bostic and reviewed by Plaintiffs' experts. Bostic reported that he had used drywall compounds from seven different manufacturers.

Plaintiffs offered the testimony of several experts. Dr. Richard Lemen, an epidemiologist, testified about the history of research linking asbestos in its various forms to diseases including mesothelioma. Dr. William Longo, a material scientist, testified about the concentrations of asbestos that would be released into the air by workers performing typical drywall work. Dr. Arnold Brody, a pathologist, testified regarding asbestos, including the chrysotile variety used in the drywall compound, as a recognized cause of mesothelioma and other diseases. Dr. Samuel Hammar, a pathologist, was Plaintiffs' expert on specific causation.

Hammar testified that any asbestos exposure above background levels causes mesothelioma. He testified that he had not reviewed the deposition testimony of Bostic and Harold. He reviewed the work history sheets but conceded they did not indicate the duration or intensity of exposure. Hammar, Brody, and Lemen repeatedly testified that "each and every exposure" to asbestos was a cause of Bostic's disease.[94] Longo conceded that his studies did not attempt to "mimic any one

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[94] For example, Hammar testified:

> Q: And is it fair to say then that to a reasonable degree of medical probability, that if somebody has mesothelioma that each and every exposure to asbestos that that person had would be a significant contributing factor to the development of mesothelioma?
>
> A: I believe so, at least potentially a contributing factor, yes.
>
> * * *
>
> Q: And did each and every exposure that Timothy Bostic had to Georgia Pacific joint compounds and wallboard materials increase his risk of mesothelioma?
>
> A: Yes.
>
> * * *
>
> Q: And is that consistent with your opinion that each and every exposure to asbestos is a contributing factor?
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> A: Yes.
>
> * * *
>
> Q: And do you agree that each and every exposure that he had to asbestos, regardless of the source to the extent he had an exposure, that those were significant and contributing factors in the development

person's actual exposure to asbestos," so he made no attempt to measure Bostic's actual aggregate dose assignable to Georgia-Pacific or any other source.[95]

We conclude, under the principles stated above, that the causation evidence was legally insufficient to uphold the verdict. Proof of substantial factor causation requires some quantification of the dose resulting from Bostic's exposure to Georgia-Pacific's products. Plaintiffs did not establish even an approximate dose. Instead, the expert testimony was to the effect that any exposure was sufficient to establish causation, a theory we rejected in *Flores*. Plaintiffs' counsel reinforced this testimony in opening and closing argument by embracing the any exposure theory. In opening counsel argued:

> of his mesothelioma?
>
> A: Yes.

Brody agreed that "each and every exposure that a person has to asbestos contributes to their risk for developing disease," and that "you have to consider that each and every one of those exposures played a role in the development of the disease." He agreed that "each and every one of the asbestos fibers that a person inhales into their lungs has to be considered a cause" of his mesothelioma. Lemen agreed that "each and every exposure that somebody has . . . increase[s] their risk of developing mesothelioma." He agreed that "any exposure" and "each exposure" to asbestos "caused [Bostic's] mesothelioma."

[95] Longo's experiments measured the intensity of exposure a worker might encounter while performing various drywall tasks. He did not attempt to establish Bostic's actual aggregate dose. We think Georgia-Pacific's expert, Dr. William Dyson, correctly explained the difference between intensity of exposure and dose:

> [A]ll diseases, including those associated with asbestos, follow a dose-response relationship. And a dose is the multiplication product of the exposure intensity times the exposure duration. Those are the two components of dose. So measuring the airborne concentration in fibers per cubic centimeter or a million particles per cubic foot is a measure of the intensity of the exposure or the level of exposure in the air. Then you take the duration of that exposure, and those two components give you dose.

Dyson further explained that "dose is a two-component factor. It's the intensity of exposure, which are the measurements that Dr. Longo provides us here but also the duration of exposure." *See also* Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, *in* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 633, 638 n.12 (Fed. Jud. Ctr. 3d ed. 2011) ("Dose is a function of both concentration and duration.").

> [W]e have the burden of proof. . . . And we assume that burden and will prove this case to you by meeting that burden of proof. To prove our case that it is more likely true than not true that Georgia-Pacific sold an asbestos product, that Timothy Bostic was exposed to this asbestos product, and that he died as a result of the exposure to this and other asbestos products.

In closing counsel argued:

> The first part of this [jury] question is proximate cause, and that's what I want to talk to you about first. . . . And in this case, you have seen that Timothy Bostic did have more than just one exposure to asbestos. And at no point in this trial have we ever said that one of those exposures you could just pull out and forget about it. You're [] not going to hear us say that. You didn't hear our experts say that. *Each and every exposure is a contributing factor to the disease.* That's just the science. *But when more than one exposure comes together to cause a disease, they're all responsible.* You can't just separate one out.

Counsel invited the jury to find that any exposure was sufficient to impose liability and that aggregate and relative dose did not matter.

Rather than attempting to quantify the aggregate dose of asbestos attributable to Georgia-Pacific's products, Plaintiffs' experts expressly eschewed this approach in favor of the view that any exposure at all was sufficient to constitute a cause of the disease, even though Hammar, Brody, and Lemen conceded that all asbestos diseases are dose-related,[96] Brody conceded that everyone has

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[96] Hammar testified:

> Q: Now, Doctor, that leads me to the next question, which is: Are asbestos-related diseases what we call dose-related diseases?
>
> A: Yes.
>
> Q: What does that mean?
>
> A: That means that the cumulative dose, at least up to the point at least in cancer where the first cell developed, were all causative or potentially causative of disease. And that basically means that the more asbestos exposure you have the greater the risk of developing asbestos-related disease.

He agreed "if you were to look at it from a probability point of view" that "higher levels may contribute more to the

some asbestos in his lungs, but at levels too low to cause disease,[97] and Lemen conceded that "the only way to adequately study subjects and their risk of developing disease is to study the exposure they have." We agree with the Pennsylvania Supreme Court that an expert opinion embracing the any exposure theory while recognizing that the disease is dose-related "is in irreconcilable conflict with itself. Simply put, one cannot simultaneously maintain that a single fiber among millions is substantially causative, while also conceding that a disease is dose responsive."[98]

Not only did *Flores* reject the any exposure theory, but Plaintiffs' experts purported to rely on studies that contradicted or at least did not confirm a theory that each and every exposure should be treated as a substantial cause of the disease. For example, Brody, Lemen, and Hammar purported to rely on a report[99] of the "Helsinki Conference" on asbestos disease which states that while mesothelioma can occur in cases of low exposure, "very low background environmental exposures carry only an extremely low risk."[100] Brody also relied on an article by Philip Landrigan and others finding it "widely accepted that asbestos fibers, including chrysotile fibers, increase the existing risk

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development of the disease than exposures at much lower levels. . . . I would give you that at least from a probability point of view, the more exposure to asbestos that you have from any given exposure, the more likely that that exposure is to contribute to the development of that mesothelioma." Brody similarly agreed that "[a]ll these [asbestos] diseases are so-called dose response diseases. That means the more you're exposed to, the more likely you are to get [the] disease." He later testified that because Bostic worked only nine months at the Knox Glass plant his risk of disease would be less than the risk of employees who had worked at the plant for 20 years. Lemen agreed "that asbestos-related diseases were dose-response diseases." He stated: "I think the jury should understand that the higher the exposure, the more the risk increases."

[97] *See supra* note 22.

[98] *Betz v. Pneumo Abex, LLC*, 44 A.3d 27, 56 (Pa. 2012).

[99] *Consensus Report, Asbestos, Asbestosis, and Cancer: the Helsinki Criteria for Diagnosis and Attribution*, 23 SCANDINAVIAN J. WORK, ENV'T & HEALTH 311 (1997).

[100] *Id.* at 313.

of developing lung cancer in proportion to the cumulative exposure that occurred up to a time 10 years prior to evaluation."[101] Brody repeatedly testified that minimal exposure to asbestos does not cause mesothelioma.[102]

Hammar and Lemen testified that any exposure to asbestos should be treated as a cause of Bostic's mesothelioma. In reaching this conclusion they relied in part on publications in the Federal Register, including a 1977 report of the Consumer Product Safety Commission[103] (CPSC) proposing to ban asbestos-containing patching compounds. This report was not itself a peer-reviewed epidemiological study, although it cited a number of studies. It concluded, based in part on theoretical arguments, that "[a] 'no effect' level theoretically may exist, but it has not been demonstrated. Therefore, there is no known threshold below which exposure to respirable free-form asbestos would be considered safe."[104] Lemen also discussed 1972 OSHA regulations concerning asbestos exposure standards. This publication recognized "controversy as to the validity of the measuring techniques" and "controversies concerning the relative toxicity of various kinds of asbestos," but concluded that in view of the risk of not acting "it is essential that the exposure be regulated now, on the basis of the best evidence available now, even though it may not be as good as scientifically desirable."[105] These publications are not scientific studies, and while a federal

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[101] Philip J. Landrigan et al., *The Hazards of Chrysotile Asbestos: A Critical Review*, 37 INDUS. HEALTH 271, 273 (1999).

[102] *See supra* notes 22–23.

[103] Respirable Free-Form Asbestos, 42 Fed. Reg. 38782 (July 29, 1977).

[104] *Id.* at 38786.

[105] Standard for Exposure to Asbestos Dust, 37 Fed. Reg. 11318, 11318 (June 7, 1972).

agency may be authorized to ban a product based on the lack of proof of its safety,[106] a "fundamental principle" of Texas products liability law "is that the plaintiff must prove that the defendants supplied the product which caused the injury."[107] Because "[o]ur legal system requires that claimants prove their cases by a preponderance of the evidence," our law "lags science; it does not lead it."[108] Like the CPSC, Lemen could not state that "there is not a safe level" of asbestos. Instead, his testimony was that "we don't know really how much exposure it takes to cause mesothelioma," and that "one of the reasons we recommend banning of asbestos, all types of asbestos, is because that level is so low that we have not been able to measure that level." As noted above, Lemen testified that for all carcinogens, the threshold at which the risk of disease falls to zero is unknown. Brody similarly testified that "no one's ever been able to show a level that will prevent everyone from getting mesothelioma." Assuming this testimony is factually correct, the failure of science to isolate a safe level of exposure does not prove specific causation in today's case, but the any exposure theory in effect asks the Court to do so. As noted above, one court, in refusing to admit Hammar's any exposure testimony, observed that "Dr. Hammar wants to be allowed to tell a jury that all of the plaintiff's *possible* exposures to asbestos during his entire life were contributing causes of the plaintiff's cancer, and, therefore, sufficient to support a finding of legal liability . . . . Just because

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[106] For example, the FDA "may make regulatory decisions . . . based on postmarketing evidence that gives rise to only a suspicion of causation." *Matrixx Initiatives, Inc. v. Siracusano*, 131 S. Ct. 1309, 1320 (2011). Hence, "efforts to invoke . . . regulatory standards are also ineffectual in terms of substantial-factor causation, since the most these can do is suggest that there is underlying risk from the defendants' products . . . ." *Betz*, 44 A.3d at 55.

[107] *Gaulding v. Celotex Corp.*, 772 S.W.2d 66, 68 (Tex. 1989).

[108] *Havner*, 953 S.W.2d at 728.

we cannot rule anything out does not mean we can rule everything in."[109] Stated another way, the inability of science to establish a maximum safe dose does not mean that science cannot establish a statistically significant link between a dose and the disease. It seems to us that all other things being equal, the more toxic the substance, the easier it should be to establish a *Havner*-compliant statistical link.

So far as we can tell, none of the peer-reviewed scientific studies on which Plaintiffs' experts relied found a statistically significant link between mesothelioma and occasional exposure to joint compounds comparable to Bostic's exposure, namely the occasional exposure of a son helping his father on building renovation projects that were not the primary occupation of either father or son, and which included drywall work as well as other construction activities. For example, Lemen testified about one of his own published articles[110] which relied on a study of a Chinese asbestos plant where workers were employed at the plant, presumably full-time, for an average of over two decades.[111] While, as Lemen reported, the study of the Chinese plant met standards we recognized in *Havner* (a relative risk of 4.29, with a confidence level of 95% and a confidence interval of 2.17 to 8.46), the cohort studied consisted of individuals whose circumstances were very different from those of Bostic. Lemen also discussed a study by Frank Stern and others[112] of union plasterers and

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[109] *Smith v. Ford Motor Co.*, 2013 WL 214378, at *3 (D. Utah Jan. 18, 2013).

[110] Richard A. Lemem, *Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model*, 10 INT'L J. OCCUPATIONAL & ENVTL. HEALTH 233, 235 (2004).

[111] Eiji Yano et al., *Cancer Mortality Among Workers Exposed to Amphibole-Free Chrysotile Asbestos*, 154 AM J. EPIDEMIOLOGY 538 (2001).

[112] Frank Stern et al., *Mortality Among Unionized Construction Plasterers and Cement Masons*, 39 AM. J. INDUS. MED. 373 (2001).

cement masons where the authors made reference to another study of drywall construction which found asbestos fiber concentrations "similar to those measured in the work environment of asbestos insulation workers who"[113] in yet another study by Irving Selikoff and others[114] "had a seven-fold increased risk of cancer of the lung and of the pleura."[115] However, the Stern and Selikoff studies were of workers employed in the trades studied, not persons like Bostic who performed occasional drywall work outside of their primary employment. Further, the Stern study found that the correlation between employment in the trades studied and mesothelioma was "not statistically significant,"[116] despite special efforts by the authors to manually review death certificates "to obtain a more accurate assessment of mesothelioma-related deaths in this cohort."[117] In *Havner*, we held that the plaintiff "must show that he or she is similar to those in the studies. This would include proof that the injured person was exposed to the same substance, that the exposure or dose levels were comparable to or greater than those in the studies . . . and that the timing of the onset of injury was consistent with that experienced by those in the study."[118] Without such a showing, "epidemiological studies are without evidentiary significance."[119] While the exposure of those in

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[113] *Id.* at 383.

[114] Irving J. Selikoff et al., *Mortality Experience of Insulation Workers in the United States and Canada, 1943–1976*, 330 ANNALS N.Y. ACAD. SCIS. 91 (1979).

[115] Stern, *supra* note 112, at 383.

[116] *Id.* at 376.

[117] *Id.* at 381.

[118] 953 S.W.2d at 720.

[119] *Flores*, 232 S.W.3d at 771.

the study need not exactly match the plaintiff's exposure, "the conditions of the study should be substantially similar to the claimant's circumstances,"[120] a requirement that was not met.

Plaintiffs' experts did not show, through reliance on scientifically reliable evidence, that Bostic's exposure to asbestos from Georgia-Pacific's products more than doubled his risk of contracting mesothelioma.

Evidence was presented of another source of asbestos exposure, namely Bostic's employment at Knox Glass, where he was exposed to asbestos from numerous sources. Hammar testified that Bostic's exposure to asbestos from Knox Glass was minimal as compared to his exposure from construction, but this testimony was conclusory, as it was not based on any scientific studies or any scientific attempt to measure the relative exposures. An expert's testimony that brings no more than "his credentials and a subjective opinion" will not support a judgment.[121] The testimony may also have been based on an incorrect assumption that Bostic's primary occupation was in construction, because the work history sheets Hammar reviewed made no mention of Bostic's employment with the TDCJ.[122] "[C]ourts must look beyond the bare opinions of qualified experts and independently evaluate the foundational data underlying an expert's opinion in order to determine whether the

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[120] *Merck*, 347 S.W.3d at 266.

[121] *Havner*, 953 S.W.2d at 712.

[122] Hammar testified, incorrectly, that from his review of the work history sheets Bostic "actually worked in construction primarily" and "it looked to me like his primary occupational exposure itself was actually in the construction industry." "We are not required . . . to ignore fatal gaps in an expert's analysis or assertions that are simply incorrect." *Volkswagen of Am., Inc. v. Ramirez*, 159 S.W.3d 897, 912 (Tex. 2004).

expert's opinion is reliable."[123] If the testimony is not reliable, it is not evidence.[124] Further, another of Plaintiffs' experts, Dr. Brody, testified that if, as was the case, Bostic worked nine months at the Knox Glass plant and had amosite asbestos in his lungs, that exposure "substantially contributed to his mesothelioma." Without any meaningful and scientific attempt to quantify the exposures from the two sources, the testimony was legally insufficient, for there was no meaningful way for the jury to conclude that Bostic's exposure to Georgia-Pacific's products was a substantial factor in causing his disease, nor was there any basis for the jury to apportion liability between these two sources of asbestos. In *Flores* we found the evidence of causation legally insufficient not only because of the plaintiff's failure to establish his aggregate dose but also his failure to "introduce evidence regarding what percentage of that indeterminate amount may have originated with [Defendant] Borg-Warner's products" as opposed to "other brands of brake pads."[125]

The dissent would hold the causation evidence legally sufficient if an expert testified that exposure to a defendant's product was "significant." In bringing this suit Plaintiffs claimed exposure from 40 defendants, and the case as Plaintiffs tried it to the jury (1) relied on opening and closing arguments and on multiple experts who repeatedly testified[126] that any exposure to asbestos should be considered a cause of Bostic's disease, (2) failed to quantify, even approximately, the aggregate dose, (3) failed to quantify, even approximately, the dose attributable to Georgia-Pacific, and

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[123] *Merck*, 347 S.W.3d at 262.

[124] *Havner*, 953 S.W.2d at 713.

[125] 232 S.W.3d at 772.

[126] *See supra* note 94.

(4) failed to show that the dose fairly assignable to Georgia-Pacific more than doubled Bostic's chances of contracting mesothelioma. The evidence was sufficient only if proof of some exposure is sufficient to establish causation. It is not. The essential teaching of *Flores* is that dose matters, and this requirement applies to mesothelioma cases.

For these reasons, we conclude that the evidence of causation was legally insufficient to sustain the verdict in this case.

III. Conclusion

While we do not agree with all of the language of the court of appeals' decision, that court reached the correct result in reversing the trial court's judgment and rendering a take-nothing judgment. We affirm the court of appeals' judgment.

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Don R. Willett
Justice

OPINION DELIVERED: July 11, 2014