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

JUSTICE GUZMAN, concurring.

Over the last several decades, asbestos litigation has become ubiquitous in our federal and state courts. In Texas, the Court has decided a handful of seminal cases articulating a legal framework for toxic torts in the context of asbestos litigation. Here, though the Court correctly deems the evidence of causation legally insufficient, I write separately because my approach is more nuanced in that I believe proving an occasional exposure mesothelioma case with epidemiological studies is not an impossible task. I also write to note my belief that the asbestos litigation framework proposed by the dissent fails to adhere to our well-settled precedents as they relate to the preponderance of evidence standard. In short, I am concerned that both writings do not faithfully interpret the preponderance of the evidence standard that stands as the lodestar of civil liability in

Texas. A plaintiff must always prove his toxic tort claim by this standard: Nothing less will suffice, but nothing more is required.

When we allowed scientific rather than direct proof for toxic torts in *Havner*, we interpreted the preponderance standard to mean that a plaintiff must prove he was exposed to a dose of the toxin that more than doubled his risk of injury. In *Flores* and here, the preponderance standard demands that if the plaintiff was exposed to toxins from multiple defendants, he must nonetheless prove he was exposed to a dose of the defendant's toxin that more than doubled his risk of injury. Any standard above or below this threshold fails to comport with the preponderance standard as articulated by this Court.

This matter requires us to apply the preponderance of the evidence standard to mesothelioma cases, and I fear that while the Court may demand too much, the dissent misconstrues our precedents to require too little. The Court holds here that the plaintiff's epidemiological studies were insufficient because they were not "the occasional exposure of a son helping his father on building renovation projects which were not the primary occupation of either father or son, and which included drywall work as well as other construction activities." __ S.W.3d __, __. But we have only required substantially similar—not completely identical—epidemiological studies. Plaintiffs must resolve any differences between the studies and the plaintiff's pattern of exposure through reliable scientific evidence. Here, the plaintiff offered epidemiological studies of occupational exposure that were extrapolated to purportedly measure risk from occasional exposure. But the plaintiff never substantiated those extrapolations, yielding an analytical gap in his proof of causation. Nonetheless,

I agree with the Court that the plaintiff failed to prove his approximate dose of exposure to the defendant's asbestos. Thus, I join the Court's opinion except for parts II.A.3 and II.B.

If the Court arrives at the correct result by potentially setting the evidentiary bar too high for future claimants, the dissent reaches an implausible conclusion by neglecting the preponderance standard as established by our precedents. Not requiring quantifiable evidence that a defendant's asbestos product more than doubled the risk of harm, as the dissent proposes, eases the required burden of proof to something subaltern to a preponderance of the evidence. While mesothelioma is a unique disease in that relatively limited exposure can induce illness, this does not change the burden of proof. It simply permits the plaintiff to present lesser dosage evidence (*i.e.*, epidemiological studies for mesothelioma will show more than a doubling of the risk at a lower dose, and plaintiffs need only show exposure comparable to this dose). The pathological peculiarities of mesothelioma should not render a plaintiff's claim almost impossible to prove or almost impossible to lose. Therefore, I respectfully concur in the Court's judgment.

I. Legal and Factual Background

This Court's foundational case for proving causation in toxic torts matters is *Merrell Dow Pharmaceuticals, Inc. v. Havner*, 953 S.W.2d 706, 708 (Tex. 1997). *Havner* addressed litigation surrounding a drug for pregnant mothers that was alleged to have caused birth defects.[1] *Id*. In *Havner*, we held that where direct, scientifically reliable proof of causation was unavailable, epidemiological studies can prove causation, provided they comply with burden of proof

[1] Causation can be general (whether a substance is capable of causing a particular injury or condition in the general population) or specific (whether a substance caused a particular individual's injury). *Havner*, 953 S.W.2d at 714.

requirements. *Id*. at 715. After a comprehensive review of the applicable academic literature, we established that the burden of proof is satisfied when properly-conducted studies establish more than a "doubling of the risk" caused by the toxic tortfeasor, as this strikes "a balance between the needs of our legal system and the limits of science."[2] *Id*. at 717–18.

Havner specifically addressed a single defendant and a non-asbestos tort, but *Borg-Warner Corp. v. Flores* involved multiple defendants in a products liability action involving asbestos. 232 S.W.3d 765, 766 (Tex. 2007). The Court recognized "the proof difficulties accompanying asbestos claims," and accordingly did not demand that causation be proved with "mathematical precision." *Id*. at 772–73. Although the Court only briefly discussed *Havner*, it integrated its reasoning. For instance, while epidemiological studies were not presented in *Flores*, the Court noted that had such studies been introduced, they would have had to show that brake mechanics (the occupational class of the plaintiff) "face at least a doubled risk of asbestosis." *Id*. at 772.[3]

In the wake of *Havner* and *Flores*, then, a plaintiff employing epidemiological studies to prove causation must set forth reliable studies showing exposure to a dosage that more than doubles

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[2] We noted:

> [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.

Id. at 718.

[3] We also cited *Havner* for the proposition that dosage is germane: "We have held that epidemiological studies are without evidentiary significance if the injured person cannot show that 'the exposure or dose levels were comparable to or greater than those in the studies.'" *Flores*, 232 S.W.3d at 771 (quoting *Havner*, 953 S.W.2d at 720–21).

the risk of injury (general causation) and that the plaintiff's exposure to the defendant's toxin was comparable to or greater than the more than doubling of the risk dose in the studies (specific causation). The standard will be the same for both asbestosis and mesothelioma cases, though the epidemiological studies will likely vary considerably depending upon the ailment involved given that different exposure levels are associated with each illness.[4] The studies might differ depending upon the type of the asbestos involved as well.[5]

II. The Court's Methodology

I agree with the Court's ultimate conclusion that Bostic did not produce epidemiological studies that complied with *Havner* and failed to prove his approximate dose of exposure to Georgia-Pacific asbestos. Because I believe the Court's opinion may be interpreted to foreclose recovery in a mesothelioma case based on occasional exposure to asbestos, I expound on this issue.

Here, though the parties strongly contested nearly every issue in this matter, they appear to concede that *Havner* controls. As to *Havner*, the crux of their disagreement centered on its application. At oral argument, Georgia-Pacific argued that *Havner* precludes Bostic from

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[4] The most common diseases that might result from asbestos exposure are (1) Asbestosis: a diffuse, interstitial, nonmalignant, scarring of the lungs; (2) Bronchogenic carcinoma: a malignancy of the interior of the lung; (3) Mesothelioma: a diffuse malignancy of the lining of the chest cavity (pleural mesothelioma), or of the lining of the abdomen (peritoneal mesothelioma); and (4) Cancer of the stomach, colon, and rectum. Consumer Product Safety Commission, 42 Fed. Reg. 38,782, 38,784 (proposed July 29, 1977) (to be codified at 16 C.F.R. pts. 1304 and 1305); *see also Flores*, 232 S.W.3d at 771 ("It is generally accepted that one may develop mesothelioma [in contrast to asbestosis] from low levels of asbestos exposure." (citations omitted)).

[5] "There are six basic varieties of asbestos minerals which are found in fiber form: chrysotile (the most common variety, and that ordinarily found in asbestos-containing products), amosite, crocidolite, actinolite asbestos, tremolite asbestos, and anthophyllite asbestos. Most of the world supply of commercial asbestos is chrysotile, the fibrous form of serpentine." Consumer Product Safety Commission, 42 Fed. Reg. at 38,784.

recovering, because "without dose evidence, you cannot do a *Havner* analysis."[6] Bostic's attorneys countered that they furnished epidemiological studies that measured exposure to a dosage and asbestos type sufficiently analogous to those experienced by Bostic.[7] Thus, despite the interpretative differences, both parties articulated their arguments consistent with the framework we established in *Havner*.

I believe Georgia-Pacific advances the more cogent argument. All but one of the studies Bostic presented were not sufficiently analogous to his situation to meet *Havner* and *Flores* standards; for instance, these studies largely concerned occupational exposure, which measures a much different phenomenon than the occasional exposure Bostic experienced.

The Consumer Product Safety Commission (CPSC) report, however, is based on a much more similar pattern of exposure to that of Bostic.[8] Deriving its statistics from "epidemiological data

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[6] *See* Transcript of Oral Argument at 11.

[7] Counsel for Bostic stated:

> [T]here were studies showing absolutely that chrysotile asbestos causes mesothelioma and then in terms of the claim of Georgia-Pacific, and this is very important, Georgia-Pacific is stating that we need low-dose chrysotile-only studies to survive the causation challenge in this case and that is wrong for two reasons. First, it's not relevant to this Record because this record showed that the Georgia-Pacific joint compound that Timothy Bostic used, while it was trace amounts, still had millions of tremolite fibers in it . . . . [S]econd, pure low-dose chrysotile studies do not exist because people are not exposed solely to chrysotile fibers. People are exposed like Timothy Bostic was to mixed fibers and *Havner* recognized that you're dealing with retrospective exposure analyses. We can't go out and put somebody in a test chamber and say we're going to expose you to this amount of chrysotile asbestos and then that amount of chrysotile asbestos since you're a baby and then wait 40 years and see if that was sufficient.

Id. at 19.

[8] Consumer Product Safety Commission, 42 Fed. Reg. at 38,782.

in the literature," it tied comparatively low levels of asbestos exposure to increased risk of injury.[9] Specifically, it detected increased risk of asbestos-related diseases stemming from exposure to drywall products that were used "six hours a day four times a year"—what it termed "high yet reasonably foreseeable" usage.[10] This is much closer to the exposure allegedly experienced by Bostic in regard to Georgia-Pacific's product. Extrapolating from that dosage, the CPSC concluded that even on the low end, the result would be a ten-fold increase in the risk of illness.[11]

However, I am troubled by two aspects of Bostic's reliance on the CPSC report. First, no evidence supported the extrapolation from the foundational data to the projected risk rates. The underlying epidemiological studies on which the report was based measured occupational exposure. To assess the equivalent risk from occasional exposure such as through consumer products, the CPSC made calculations it retained at its offices based on a published theoretical model. The Court has never held that such extrapolations violate *Havner*, but as we recently held in *Merck & Co. v. Garza*, there must be some "scientific basis" for the extrapolation. 347 S.W.3d 256, 267 (Tex. 2011).

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[9] *Id.* at 38,787. Though not a peer-reviewed academic study, the CPSC report was predicated on a study by Skelikoff of three separate cohorts.

[10] *Id.*

[11] In the relevant part, the report provided:

> For purposes of this assessment, the Commission considered the use of patching compounds by a consumer, for six hours a day four times a year, to be a high yet reasonably foreseeable yearly exposure. The increased risk of death from respiratory cancer induced by this yearly exposure is estimated at between 10 and 2,000 per million. For five years of exposure at these levels, the risk increases geometrically and is estimated at between 1,000 and 12,000 per million. Based on current information, the Commission estimates that the lower estimate of 10 per million is closer to the actual risk for a one year exposure.

Id.

Here, the published study and CPSC calculations form that scientific basis, but Bostic never admitted them into evidence. Thus, the lack of evidence regarding the scientific basis for the extrapolation amounts to an analytical gap in this particular case.

Second, as was the case with the eponymous plaintiff in *Flores*, Bostic failed to prove his dose was comparable to or greater than the dose in the study. He vigorously contests this, citing the accommodating language of *Flores* regarding scientific proof. *See* 232 S.W.3d at 772–73. But even if *Flores* did not require numerically precise dosage, some reasonable approximation is required to satisfy causation. Bostic failed to marshal such an approximation because, as the Court's thorough analysis indicates, testimony only indicated one drywall job where Bostic's father recalled using Georgia-Pacific joint compound, and the father did not recall if Bostic was present during that job.[12] As in *Flores*, this is insufficient evidence of an approximate dose.

It bears noting that even though Bostic failed to prove his case, the preponderance standard does not present an insuperable hurdle for all occasional exposure mesothelioma cases. While the bulk of epidemiological studies appear to focus on occupational exposure, properly substantiated extrapolations can bridge the gap between those studies and the plaintiff who contracted mesothelioma from occasional exposure to asbestos. But such a plaintiff must provide a reliable scientific basis for the extrapolation and exposure to a dose of the defendant's toxin comparable to

[12] The Court does a thorough job of cataloguing Bostic's exposure. As it demonstrates, the only evidence that Bostic was exposed to Georgia-Pacific's joint compound were statements from Bostic and his family, and many of these statements were highly speculative. While there is little doubt that Bostic was exposed to asbestos-containing products, there is significant uncertainty as to the extent that Georgia-Pacific's products were involved.

or greater than the extrapolated dose that more than doubled the risk of injury. Here, Bostic failed to do either.

The Court also seems to improperly apply its own articulated standard governing how closely-tailored an epidemiological study must be to a plaintiff's demonstrated exposure. Interestingly, the Court rightly notes that the exposure measured in the studies and stemming from the plaintiff's own experience need only be "substantially similar," not precisely congruent. __ S.W.3d at __ (quoting *Garza*, 347 S.W.3d at 266). Although the Court advances the proper standard, it seems to misapply it by dismissing all of Bostic's epidemiological studies because they were not "the occasional exposure of a son helping his father on building renovation projects which were not the primary occupation of either father or son, and which included drywall work as well as other construction activities." __ S.W.3d at __. As a practical matter, requiring this level of exactitude may imply that hardly any mesothelioma plaintiff can recover. Indeed, experts have long-acknowledged that asbestos encompasses a broad panoply of constituent types and forms; for instance, there are six types of asbestos fibers, and they may be in either friable or encapsulated form. *See supra* note 5. The CPSC report was predicated on epidemiological studies involving asbestos insulation that contained friable, principally chrysotile fibers. This type of exposure is substantially similar to Bostic's exposure for two reasons. First, both information in the studies and in the record indicate that asbestos exposure from joint compound is more severe than that from insulation.[13]

[13] The underlying data for the CPSC report indicated that insulators were exposed to 15 fibers per cubic centimeters (f/cc) of air for their half day of work, compared to 35.4 to 59.0 f/cc for dry-mixing joint compound, 1.3 to 16.9 f/cc for hand-sanding joint compound, 41.4 f/cc sweeping the dust after applying joint compound, and 26.4 f/cc 35 minutes following sweeping.

Second, the underlying evaluation informing the CPSC report's calculation was a study of predominately friable, chrysotile asbestos, and Georgia-Pacific's joint compound principally contained chrysotile asbestos. The thrust of *Havner* is that we will allow a plaintiff to recover if science can bridge the gap in proof of causation. Requiring perfectly congruent epidemiological studies when science can fill potential analytical gaps undercuts the very purpose of *Havner*.

Lastly, I cannot join Part II.A.3 of the Court's opinion because of the potential conflict between its articulation of substantial factor causation and the Texas comparative fault statute. The Court believes that substantial factor causation means that a defendant whose toxin more than doubled the plaintiff's risk of injury may not be liable if exposure to another defendant's toxin was at a factor 10,000 times more. __ S.W.3d at __. For simplicity's sake, assume a jury found a lesser such defendant 1% at fault and the greater defendant 99% at fault. Applying the Court's view of substantial factor causation to this scenario is problematic. If the Court's interpretation of substantial factor causation requires the defendant found 99% at fault to assume the remaining 1% liability, this runs afoul of the comparative fault statute—the purpose of which is to make each defendant liable for its percentage fault. And if the Court's interpretation requires the plaintiff to assume the remaining liability, this conflicts with our long-standing tradition that a plaintiff can recover the percentage attributable to the defendant after carrying his burden by a preponderance of the evidence. The Court's injection of an ostensibly common sense approach to causation unnecessarily skews the preponderance standard (just in the opposite direction that the dissent's common sense approach does, as addressed below). This deviation from the preponderance standard we have long adhered to is unwarranted, especially in a case where it does not apply.

III. The Dissent's Methodology

If the Court impliedly requires too much of a mesothelioma plaintiff in requiring overly congruent epidemiological studies, the dissent errs in the opposite direction—significantly and errantly easing the burden of proof requirement to something below a preponderance of the evidence. First, it misapprehends *Havner*, suggesting that the case need not apply because Bostic has offered sufficient direct evidence, and therefore alternative methods of proving causation are unnecessary. I disagree. While Bostic introduced evidence of exposure to Georgia-Pacific products containing asbestos, the evidence lacked sufficient specificity. As previously addressed, Bostic's father cited eight discrete examples of drywall jobs, but only recalled using Georgia-Pacific's product in one such job and did not remember whether his son was present for that project. Moreover, Bostic cannot tie a specific manufacturer's asbestos fiber to his ailment. While this is an admittedly formidable evidentiary task, it does not automatically mean, as the dissent suggests, that traditional notions of causation can be relaxed. As the Court rightly notes, a mesothelioma plaintiff asserting a claim stemming from occasional exposure from multiple asbestos sources has a more challenging task than a plaintiff with well-documented occupational exposure from a single source. __ S.W.3d at __. The former plaintiff may still recover, but he must prove causation with evidence that comports with a preponderance of the evidence standard. Here, there is no direct evidence that Georgia-Pacific asbestos fibers caused Bostic's mesothelioma, so Bostic must rely on an alternative method of proving causation. While the dissent found the expert testimony offered by Bostic's witnesses sufficiently specific to prove causation, I believe that the burden of proof demands more closely-tailored evidence.

Second, the dissent overstates the scientific hurdles confronting a mesothelioma plaintiff attempting to prove *Havner* causation. While it contends that "no epidemiological study has established the threshold of exposure over which the risk of developing mesothelioma is doubled" for intermittent exposure, the epidemiological studies in the CPSC report cited previously may serve as a baseline for future mesothelioma plaintiffs with occasional exposure (provided that they substantiate the extrapolation and their approximate dose). __ S.W.3d __, __ (Lehrmann, J., dissenting). In short, then, *Havner* permits a mesothelioma plaintiff to prove causation and recover in tort, and at least one scientific study may exist as a benchmark. Bostic merely failed to sufficiently relate the epidemiological studies in the CPSC report to his own case or submit proper evidence that his dose was comparable to or greater than that in the study. Thus, his failure to prove specific causation renders his claim unrecoverable.

More generally, I fear that the dissent's failure to pronounce a clear standard risks instilling confusion in our courts, where future asbestos litigation will inevitably occur. A plaintiff may recover by direct evidence of causation, or may attempt to prove alternative causation consonant with *Havner*'s framework. Regardless of the litigation path trod, causation must be proved by a preponderance of the evidence. This standard is satisfied differently depending upon whether direct or *Havner* evidence is involved, but under either approach, the plaintiff faces the same burden of proof. We must not dilute the preponderance of the evidence standard that has stood as a hallmark of toxic tort litigation in order to make mesothelioma cases easier to prove. We have declined prior invitations from tort claimants to weaken the preponderance of the evidence standard as it relates to

scientific proof of causation. *See Havner*, 953 S.W.2d at 730; *Flores*, 232 S.W.3d at 774. I join the Court in declining to do so today.

Accordingly, I join all but Parts II.A.3 and II.B of the Court's opinion and concur in the judgment.

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Eva M. Guzman
Justice

OPINION DELIVERED: July 11, 2014