John Crane, Inc. v. James Scribner, et al., No. 92, Sept. Term, 2001
John Crane, Inc. and Garlock, Inc. v. James Scribner, et al., No. 99, Sept. Term, 2001

In action for personal injury based on exposure to asbestos, cause of action "arises," for purposes of Courts and Judicial Proceedings Article, § 11-108(b)(1) when plaintiff suffered cellular change, as result of exposure to defendant's product, that led to the disease constituting the injury.

Circuit Court for Baltimore City
Case No. 24X97353701/CX2561

IN THE COURT OF APPEALS OF MARYLAND

September Term, 2001

No. 92

JOHN CRANE, INC.

v.

JAMES SCRIBNER, et al.

No. 99

JOHN CRANE, INC. AND GARLOCK, INC.

v.

JAMES SCRIBNER, et al.

Bell, C.J.
Eldridge
Raker
Wilner
Cathell
Harrell
Battaglia,

JJ.

Opinion by Wilner, J.

Filed: June 11, 2002

These appeals are from judgments entered by the Circuit Court for Baltimore City in actions for personal injury and wrongful death arising from the exposure of James Scribner to asbestos-containing products manufactured by petitioners, John Crane, Inc. and Garlock, Inc. The issues presented to us principally concern (1) whether Scribner's cause of action against petitioners arose prior to July 1, 1986, the effective date of a statutory limit, or "cap," on the amount of non-economic damages recoverable in a personal injury action, and (2) the procedure used by the Circuit Court to address and resolve that question.

We shall, in this case, set the proper standard for determining when, for purposes of Maryland Code, § 11-108(b) of the Courts and Judicial Proceedings Article – the cap statute – a cause of action for cancer or other disease based on exposure to asbestos arises. We shall also conclude that, if there is a genuine dispute of fact as to whether the plaintiff's cause of action arose prior to July 1, 1986, the plaintiff has the burden of establishing that the cause of action arose prior to that date, and the issue is for the trier of fact to resolve. Our resolution of these issues will result in an affirmance of the judgments entered by the Circuit Court.

BACKGROUND

(1) Procedural Background

In 1995, Mr. Scribner filed suit against Crane, Garlock, Owens-Corning Fiberglas, Inc. (OCF), Flexitallic, Inc., and several other defendants. When Scribner died shortly thereafter, in November, 1995, his widow continued his action as personal representative of

his estate and, along with Scribner's two children, commenced a wrongful death action.[1] The Scribners' case was consolidated with several other asbestos-related cases in a cluster that included five sets of plaintiffs and more than 35 defendants, many of which filed cross-claims and third-party claims against each other. Prior to submission of the case to the jury, the other four plaintiffs settled, the Scribners settled with OCF and one other defendant, and many of the cross-claims and third-party claims were resolved, leaving the jury to consider only the Scribners' case against petitioners Crane and Garlock and petitioners' cross-claims or third-party claims against OCF, Flexitallic, and two other former defendants.

The issues at trial concerned whether Mr. Scribner's exposure to gaskets manufactured by Garlock and gaskets and packing material manufactured by Crane was a proximate cause of the mesothelioma that became manifest years later and from which he eventually died, whether those defendants should be held negligent and strictly liable, and the appropriate amount of compensation in the event the jury found liability on the part of the defendants. At the close of evidence, Crane and Garlock moved for judgment on the ground that there was insufficient evidence regarding Scribner's exposure to their products, of Garlock's failure to warn of the danger of asbestos, and of a respirable release of asbestos fibers from Crane's products. The Scribners also moved for judgment on the issue of whether Scribner developed his mesothelioma prior to July 1, 1986. Those motions were

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[1] In the initial wrongful death action, a third child was joined as a plaintiff, but the verdict was in favor of only the widow and two children.

denied. The court, at the time, was of the belief that the issue of when the cause of action arose, for purposes of the cap statute, was for it, and not the jury, to determine.

On a special verdict sheet, the jury determined that Scribner's exposure to asbestos-containing products manufactured, sold, or supplied by Crane and Garlock was a substantial contributing factor in the development of the mesothelioma that caused his death, and that those defendants were both negligent in and strictly liable for the manufacture, sale, supply, or distribution of asbestos-containing products. The jury assessed damages in the survival action in the amount of $3,500 for funeral expenses, $43,000 for medical expenses, $5,000 for economic loss, and $2,000,000 for pain and suffering. In the wrongful death action, it assessed damages to Mrs. Scribner of $1,000,000 for economic loss and $1,000,000 for pain and suffering, and to each of the two children an unitemized $370,000. It awarded Mrs. Scribner, in addition, $450,000 for loss of consortium, making the total judgment $5,241,500. On the cross- and third-party claims, the jury found that Scribner's exposure to asbestos-containing products manufactured, supplied, installed, or distributed by Flexatillic and OCF was a substantial contributing factor in the development of his mesothelioma and that both of those companies were negligent in and strictly liable for the manufacture, sale, supply, or distribution of those products.

Following the return of those verdicts and the discharge of the jury, but before entry of final judgment on the verdicts, the Court of Special Appeals rendered two decisions – *Owens Corning v. Bauman*, 125 Md. App. 454, 726 A.2d 745, *cert. denied sub nom. Owens*

Corning v. Hammond, 354 Md. 572, 731 A.2d 970 (1999) and *Owens-Corning v. Walatka*, 125 Md. App. 313, 725 A.2d 579, *cert. denied*, 354 Md. 573, 731 A.2d 971 (1999) – in which it concluded, among other things, that, in asbestos-related litigation, the plaintiff had the burden of proving that his or her cause of action arose prior to the effective date of the statutory caps on non-economic damages and that, if there was a genuine dispute on that issue, it was for the trier of fact – in a jury case, the jury – to determine. Because an essential element of a wrongful death action is the death of the person, and it was undisputed that Mr. Scribner died after October 1, 1994 – the effective date of the cap on non-economic damages awarded in a wrongful death action – there was little disagreement that the cap applied to the wrongful death action filed by Mrs. Scribner and the children and that the non-economic damages awarded in that action would have to be reduced from a total of $1,740,000 to $772,500.[2]

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[2] In *United States v. Streidel*, 329 Md. 533, 537, 620 A.2d 905, 907 (1993), we held that the limitation on non-economic damages imposed by Maryland Code, § 11-108, as it then existed, did not apply in an action for wrongful death. In its next (1994) session, however, the General Assembly enacted amendments to § 11-108 that made the limitation applicable to wrongful death claims. *See* 1994 Md. Laws, ch. 477. With the 1994 amendments, § 11-108 sets a limit of $500,000 on non-economic damages with respect to causes of action arising on or after October 1, 1994, subject to the caveats that (1) the cap (continued...)

The dilemma arose with respect to the survival action. Having concluded that the issue could not be resolved as a matter of law and having already discharged the trial jury, the court impaneled a new jury to consider only the question of whether Mr. Scribner's cause of action arose against Crane and Garlock prior to July 1, 1986, the effective date of the cap on non-economic damages in a personal injury action. Crane and Garlock, contending that the issues of liability and when the cause of action arose were intertwined, objected to that approach and moved, unsuccessfully, for a new trial on all issues.

In what the parties refer to as Phase II, they presented to the new jury much of the same evidence regarding the disease of mesothelioma and how it grows in the body that was presented to the first jury. That evidence was supplemented by new testimony regarding doubling time, post-operative growth theory, and explosive growth theory in an effort to

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[2](...continued)
amount increases by $15,000 on October 1 of each year beginning in 1995, and (2) in a wrongful death action in which there are two or more claimants, the total award of non-economic damages may not exceed 150% of the basic limitation. Mr. Scribner died in November, 1995, making the basic effective cap $515,000 per plaintiff. As there were multiple claimants, however, the 150% gross limitation applied, so the court multiplied $515,000 by 150%, which produced a maximum award for non-economic damages in the wrongful death action of $772,500. There does not appear to be any dispute in this appeal over that determination.

determine the time when Scribner's mesothelioma first developed. On that evidence, the jury determined, in a special verdict, that (1) the first cellular changes which led to the existence of Scribner's mesothelioma began prior to July 1, 1986, and (2) the mesothelioma itself arose in Scribner prior to July 1, 1986. Upon those findings, the court concluded that the cap did not apply to the survival action and, after deducting the *pro rata* shares of the two settling defendants also found liable (OCF and Flexitallic), entered joint and several judgments against Crane and Garlock for a total of $2,137,000 ($1,025,750 in the survival action, $225,000 for loss of consortium, and $886,250 in the wrongful death action).

Crane and Garlock appealed to the Court of Special Appeals, raising a multitude of evidentiary, substantive, and procedural issues, including some that went to the issue of the cap. They argued that the trial court erred in refusing to apply the cap to the survival action as a matter of law, that the bifurcation allowed the Scribners to present evidence to the second jury that was inconsistent with the evidence presented to the first jury, and that the court erred in including on the second verdict sheet a question that focused on when the cellular changes that led to Scribner's mesothelioma first occurred. The intermediate appellate court initially found reversible error on that last issue but then, in a corrected opinion, concluded that the error was essentially harmless and affirmed the judgments. We granted *certiorari* to consider (1) the proper standard to be applied in determining, for purposes of § 11-108(b), when a cause of action for cancer or other disease based on the alleged exposure to asbestos products arises; (2) who has the burden of proof on that issue;

(3) if there is a genuine dispute of fact bearing on the issue, who decides the issue; (4) whether, in this case, the trial court erred in not deciding the issue as a matter of law; and (5) whether the court erred in submitting the issue to a second jury.[3]

(2) Factual Background

James Scribner enlisted in the Navy in March, 1971. After boot camp and advanced training at nuclear power schools in Maryland and New York, he was assigned, in late 1972, to work on the submarine, *U.S.S. Sturgeon.* He remained with the *Sturgeon* until 1975, when he returned to the nuclear training facility in New York. He left the Navy in 1978 and then worked for Potomac Electric Power Company (PEPCO) until 1995.

Scribner stated in deposition testimony that he was exposed to asbestos throughout his Navy career. While at the training center in New York and while working on the *Sturgeon* in drydock for a six-month period in 1973-74, he worked in areas where outside contractors removed old asbestos pipeline insulation and measured, cut, and installed new insulation. He recalled that the insulation they used was that of OCF and Johns Manville, that he worked around those contractors seven days a week on 12-hour shifts, and that, from the sawing and shaping of the insulation, it was always "very dusty."

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[3] We have rephrased the issues presented by petitioners. Their articulation of some of them incorporated assumptions that we are not prepared to accept.

In addition to that exposure to the dust emanating from the pipe insulation, Scribner said that from 1972 until he left the Navy, he himself cut and fitted Crane and Garlock gasket material and Crane packing material for use on steam and water pipe valves. He said that, upon discovering a leaking valve, he would cut away a small section of pipe insulation, remove the old gasket, cut a new one from the material that came in sheets, and install the new gasket. Another worker would then replace the pipe insulation. Gary Dolese, who worked with Scribner from 1976-78, added that, if the old gasket did not come off easily, it would have to be scraped off and that residue material would be removed with a wire brush. Some dust was created from that operation, although not nearly as much as from the cutting and fitting of pipe insulation. Although Scribner was unable to tell which gaskets contained asbestos and which did not, Dolese testified that the Crane and Garlock gaskets *did* contain asbestos, a fact that was confirmed through other testimony.

Following his discharge from the Navy in 1978, Scribner worked as an analyst and power plant operator for PEPCO until 1995. In December, 1994, he became ill, suffering from what he thought was a persistent cold. In March, 1995, however, a needle biopsy confirmed that he had mesothelioma. In May, Scribner underwent surgery. The plan initially was to remove one lung, but, after making the incision and evaluating the situation, the surgeon determined that the disease had progressed too far for a lung removal to be effective. He instead removed about eight pounds of tumor and tissue, but was required to leave intact some of the tumor on the diaphragm. After surgery, chemotherapy was attempted, but

Scribner was unable to tolerate the side effects. From then until his death in November, 1995, the basic therapy was pain management and assistance with basic life functions.

(3) Medical and Exposure Evidence

Much of the evidence bearing on Scribner's exposure to Crane and Garlock products and on the nature and development of mesothelioma was technical in nature and in dispute. Given the issues raised in this appeal, which focus largely on the sufficiency of the evidence presented by Scribner, we shall view the evidence in a light most favorable to him, but we shall summarize much of it and not dwell on some of the technical details. We note that there was considerable evidence presented by the defendants, directly and through cross-examination of Scribner's witnesses, that supported conclusions contrary to those reached by the two juries, but for purposes of this appeal that evidence is of little relevance.

The disease of mesothelioma was described as a malignant tumor that forms in the body cavities, predominantly the thoracic and abdominal cavities. In the thoracic cavity, it directly invades and encases the pleura – the outside lining of the lung – and eventually occupies and eradicates the pleural space. It frequently will grow into the lung and, over time, can metastasize to other structures, including the diaphragm and the abdominal cavity. Although there is a background rate of mesothelioma in the general population that is not asbestos-related, it is very low. Dr. Samuel Hammar, a pathologist, estimated that there were about 2,000 to 2,500 cases of mesothelioma reported in the United States each year and that,

in men, 80% or more were the result of occupational exposure to asbestos. Dr. Rudiger Breitenecker, who performed the autopsy on Mr. Scribner, testified that the cause of death was malignant mesothelioma and that, without a doubt in his mind, the cancer was related to his asbestos exposure. That opinion was shared as well by Dr. Hammar.

Some of the evidence relating to Scribner's exposure to the asbestos-containing products of Crane and Garlock has been summarized above. Dr. James Millette, an environmental scientist, examined the Crane and Garlock gaskets and the Crane packing and testified that about 80% of the content of the gaskets and about 85% of the content of the packing was asbestos. Although evidence was presented tending to show that the actual exposure encountered by Scribner in working with those materials did not exceed certain eight-hour weighted threshold standards established by the American Conference of Governmental and Industrial Hygienists and OSHA, other evidence was to the effect that there was no established threshold risk with respect to mesothelioma and that the risk was really determined by the total amount of asbestos fibers inhaled by a person over his or her working lifetime – that any increase in the amount inhaled or the total duration of exposure will increase the risk. Based on that evidence – the nature of the gaskets and the extent and duration of Scribner's exposure to asbestos in them – Dr. Hammar opined that Scribner's exposure to the Crane and Garlock gaskets was a substantial contributing factor to the development of his mesothelioma.

Much of the evidence, relevant both to whether Scribner's mesothelioma arose from

exposure to Crane and Garlock products and to when the injury arose, dealt with how mesothelioma develops. Dr. Arnold Brody, a pathologist, explained that asbestos fibers cause injury to cells with which they come into contact. An isolated exposure may be successfully dealt with by various bodily defenses, but if too many toxic particles get into the area, "you cannot clear enough of them to prevent disease." Asbestos exposure causes cells to divide and, according to Dr. Brody, a cell is more likely to become a cancer cell when it is dividing. The reason, he said, is that, when cells divide, they lose a protective membrane that protects the genetic material in the nucleus, which exposes that genetic material to foreign elements capable of causing genetic errors. Cancer is a loss of control over cell growth, which "gets back to the issue of the genes that control cell growth." Thus, he testified, "when you have errors in the gene[s] that control cell growth, that can lead to a cancer." Dr. Brody pointed out that, when genetic errors occur in the cells, the cells are programmed to die, but that not all of them do die, and that "all that [a] person needs is a single cell with enough of the right kind of errors to sneak through over the decades and [end] up a cancer." Because of the substantial bodily defenses, however, "[a]n individual must have repeated exposures, must have repeated errors, must have multiple errors in their mesothelial cells for them to go on and be a cancer." He added that any asbestos fiber that reaches the mesothelial cells is capable of becoming a cancer, but that "[w]hether they will or not, obviously I can't tell you and nobody is going to tell you which fiber causes injury, but the more fibers that reach that area, that critical area of the lung and those mesothelial

cells, the more likely you are to develop the disease."

A good bit of Dr. Brody's testimony was confirmed by Dr. Hammar, who testified as well about the latency of the disease. Dr. Hammar defined latency generally as the period between the time "when a person was first exposed to the agent that caused the disease and the time when [he or she was] first diagnosed with the disease." In the case of asbestos, Hammar said, "it would be when they were first exposed to asbestos, and when they were first diagnosed with an asbestos-related disease." With respect to mesothelioma, Dr. Hammar stated that about 90 to 95% of the cases fall within a 20 to 50 year range, with the average being 30 to 40 years. He explained that carcinogens, such as asbestos, act over many years to cause cellular changes that lead to the development of a malignant cell, and that once a cancer cell, about 10 micrometers in diameter, is formed, it may take 10 to 15, or as many as 30, years for that cell to proliferate and form a tumor the size of a golf ball. The more asbestos that gets into the lung, he added, the shorter the latency period is likely to be. In that regard, he said that subsequent exposures – exposures beyond the first – are contributory to the development of mesothelioma, that "all of the exposures . . . contribute to the development of the tumor up until when the first cancer cell is formed."

It was, presumably, upon this evidence that the first jury concluded that Scribner's exposure to the asbestos-containing products of Crane and Garlock was a substantial contributing factor to the development of his mesothelioma.

In Phase II, dealing more specifically with the cap statute, the parties presented much

of the same evidence they offered to the first jury. Scribner's chief witness was Dr. Hammar, who iterated his testimony regarding latency, both generally and with respect to mesothelioma. During the latency period of mesothelioma two things occur: a cancer cell develops, and it grows. Much of his testimony concerned "doubling time" – the time it takes a cancer to double its cells. Dr. Hammar said that most of the solid cancers that form spherical masses go through 20 doublings to produce a tumor one millimeter in diameter, with about a half million cells, and about 30 doublings to produce a tumor the size of a marble, containing about one billion cells. A tumor of one millimeter diameter, he said, would be beyond detection other than with a microscope; with the best CT and MRI scans, one might detect a tumor as small as five millimeters.

Dr. Hammar testified that it would be impossible to place an exact date on when a tumor first arises but that, by using doubling times, it was possible to give a theoretical estimate. He made clear that this was not an exact science. For one thing, many cancers are not truly spherical, and, for another, doubling times may not be constant throughout the process. Nonetheless, he said that the shortest doubling time for a biphasic mesothelioma – the kind that Mr. Scribner had – was 200 days, and, assuming the need for 30 doublings to produce a detectable tumor, it would take 6,000 days, or about 16 years, *from the formation of the first cancer cell* to a detectable size tumor. The total latency period for Mr. Scribner was about 23 years from his first exposure to asbestos in 1972 to detection in 1995. Scribner's last exposure, he said, was in 1979.

Using this approach, and acknowledging again that he could not give an exact date when the first cancer cell formed, Dr. Hammar opined, within a reasonable degree of medical probability, that the first cancer cell appeared sometime during 1980-84, eight to eleven years after Scribner's first exposure to asbestos, and that the tumor growth period was 12 to 15 years. Hammar stated expressly his opinion that the first cancer cell developed in Scribner's body prior to July 1, 1986. On this evidence, and that produced by petitioners, the jury concluded, in response to the two questions presented to it on a special verdict sheet, that (1) the first cellular changes which led to the existence of Scribner's mesothelioma began before July 1, 1986, and (2) the mesothelioma arose in Scribner before that date.

DISCUSSION

The Appropriate Test

Subject to other provisions that inflate or limit the amount for actions that arose on or after October 1, 1994, § 11-108(b)(1) of the Courts and Judicial Proceedings Article provides that, "[i]n any action for damages for personal injury in which the cause of action *arises* on or after July 1, 1986, an award for noneconomic damages may not exceed $350,000." (Emphasis added). Whenever an action is filed any significant time after July 1, 1986, and is based upon a disease with a long latency period, as all of the current asbestos-exposure cases are, the predominant question that arises under that statute is when the cause of action "arose," and the answer to that question is largely dependent on the test or standard

to be applied in making that determination.

We first dealt with that issue in *Owens-Illinois v. Armstrong*, 326 Md. 107, 604 A.2d 47, *cert. denied*, 506 U.S. 871, 113 S. Ct. 204, 121 L. Ed. 2d 145 (1992) (*Armstrong*), although our decision in that case was driven to some extent by our holding in *Mitchell v. Maryland Casualty*, 324 Md. 44, 595 A.2d 469 (1991). *Mitchell* was a dispute over insurance coverage that reached us in the context of a declaratory judgment action. The insured, a mechanical contractor, sold and installed products containing asbestos. From 1955 through at least 1977, it had in place comprehensive liability insurance policies issued by Maryland Casualty Company – policies that, among other things, obligated the insurer to defend Mitchell in actions seeking damages for "bodily injury" caused by an "occurrence." Following the expiration of the last policy, Mitchell was sued by a number of people for personal injuries allegedly sustained by reason of their exposure to Mitchell's asbestos products during the period that the policies were in force. Taking the position that the bodily injuries claimed in those actions did not take place until the injuries were first discovered, which was after the expiration of the last policy, the insurer disclaimed coverage and declined to defend Mitchell. Mitchell asserted that the bodily injury occurred when the personal injury plaintiff was exposed to its asbestos product, not when the disease emanating from that exposure became manifest.

The medical evidence produced at trial was somewhat, although not entirely, in conflict. A pathologist testifying for Mitchell defined "injury" as the alteration of the

structure of a cell, tissue, or organ, including a physical or chemical change that might be detectable only at a subclinical or microscopic level. A clinician testifying for Maryland Casualty opined that injury did not occur until the disease was manifest, because, as a result of the body's own defenses, an exposure may never progress to a disease. Adopting what we regarded as the majority rule around the country, we rejected the insurer's manifestation theory and held that, for purposes of insurance coverage, "'bodily injury' occurs when asbestos is inhaled and retained in the lungs." *Id*. at 62, 595 A.2d at 478. Because we were construing a term in an insurance policy, we did not regard as important the disagreement between the two medical experts "as to the time when the changes in the lungs may be classified as a disease." *Id*.

Unlike *Mitchell*, *Armstrong* directly presented the cap issue. The plaintiff, Armstrong, was diagnosed with asbestosis in 1987. He sued, claiming that the disease resulted from his exposure to the defendant's products during the period of 1962-63. Owens-Illinois, advancing the "manifestation" theory, argued that Armstrong's cause of action did not arise until he was diagnosed with asbestosis. We rejected that approach, as we had done in *Mitchell*.

In terms of pure statutory construction, we noted that the Legislature had cast the statute in terms of when the cause of action "arises," not when, for statute of limitations purposes, the cause of action "accrues." Noting that, in a typical tort action, the injury is usually the last of the elements of the tort to occur, we concluded that the action "arises," and

the statute is thus triggered, when the injury first comes into existence. We pointed out that, when there is a latency period between the exposure or event that ultimately produces the injury and the manifestation or discovery of that injury, the injury will almost necessarily occur before it is, or as a practical matter can be, discovered. Indeed, that is implicit from the "discovery rule" itself, which is founded on the premise that a period of time may elapse between the point at which an injury occurs and hence a cause of action based on that injury arises and the point at which the injured person reasonably may discover that injury. We recognized that, "[d]ue to the latent nature of asbestos-related disease, experts and courts alike have had difficulty in pinpointing its onset," but, with the benefit of hindsight, we found that that difficulty did not present a problem in the particular case. *Id*. at 122, 604 A.2d at 54. Given the 15- to 20-year latency period for the development of asbestosis and the fact that the disease was first diagnosed in 1987, it was clear that Armstrong had asbestosis prior to July 1, 1986, and that his action was not subject to the cap. *Id*. at 123-24, 604 A.2d at 55.

Although in *Armstrong* we confirmed our rejection of the "manifestation" test for determining the onset of a latent disease, we did not expressly adopt any alternative test, including the "exposure" test adopted in *Mitchell*, as there was no reason in that case for us to do so. It fell, then, to the Court of Special Appeals to struggle with that issue.

The intermediate appellate court first addressed the issue in *Anchor Packing v. Grimshaw*, 115 Md. App. 134, 692 A.2d 5 (1997), *vacated on other grounds sub nom. Porter Hayden Co. v. Bullinger*, 350 Md. 452, 713 A.2d 962 (1998) (*Grimshaw)*. Several of the

plaintiffs in that case contracted mesothelioma from exposure to the defendants' products and were awarded by the jury non-economic damages in excess of the cap. The defendants moved to reduce the awards in conformance with the statute and complained on appeal about the denial of their motions. Although the court clearly recognized that the causes of action arose prior to the actual manifestation or discovery of the mesothelioma, it was unwilling to conclude that they arose at the time of the exposure to the asbestos. Mere exposure to asbestos, it noted, does not always result in asbestos-related disease, even when the individual's body undergoes cellular changes as a result of the exposure, and, on that basis, the court concluded that "[m]ere exposure to asbestos and cellular changes resulting from asbestos exposure, such as pleural plaques and thickening, alone is not a functional impairment or harm, and therefore, do not constitute a legally compensable injury." *Grimshaw, supra*, 115 Md. App. at 159, 692 A.2d at 17. Indeed, it read our *Armstrong* decision as "obviously look[ing] beyond the date when plaintiff was exposed to asbestos and determin[ing] instead, when the earliest date of asbestosis would arise." *Id*. at 163, 692 A.2d at 20.

The test initially stated by the court was that "an injury occurs in an asbestos-related injury case when the inhalation of asbestos fibers causes a legally compensable harm" and that "[h]arm results when the cellular changes develop into an injury or disease, such as asbestosis or cancer." *Id*. at 160, 692 A.2d at 18. Later in its opinion, the court seemed to re-articulate the test as being when the cellular changes caused by exposure become

permanent and cause "functional impairment." *Id*. at 163, 692 A.2d at 19-20. Under either articulation, this was essentially a "middle ground" test; the critical time, for purposes of § 11-108 (b)(1) would always be some considerable time *after* exposure but nearly always *before* manifestation and discovery. In the particular case, the court noted that the plaintiffs' mesotheliomas were diagnosed in 1993 and 1994 and, relying on medical testimony that mesothelioma typically comes into existence ten years prior to diagnosis, concluded that it must have commenced before July 1, 1986.

Although the Court of Special Appeals has declared its continued allegiance to that test, it has, in subsequent cases, attempted to redefine or apply it in ways that have engendered some confusion. In *AC and S v. Abate*, 121 Md. App. 590, 710 A.2d 944 (1998), one of the plaintiffs, suffering from "pleural disease," which apparently involved pleural plaques that became symptomatic, received an award of non-economic damages in excess of the cap. Notwithstanding historical evidence that his exposure to asbestos occurred between 1950 and 1956 and medical evidence that his condition could have become manifest by 1960, the court held that the cap applied because of the plaintiff's testimony that it was not until 1990 "that he began experiencing the shortness of breath that curtailed his normal activities," and thus it was not until then that he "experienced any functional impairment as a result of that condition." *Id*. at 695, 710 A.2d at 996. In adopting 1990 as the critical date when the cause of action arose, the court, though supposedly applying *Grimshaw*, essentially applied the manifestation test.

In *Ford Motor Co. v. Wood*, 119 Md. App. 1, 703 A.2d 1315 (1998) (which, despite appearing in an earlier volume of the *Maryland Appellate Reports*, was filed a day *after* *Abate*), the plaintiff first began experiencing symptoms of mesothelioma in 1992 and was diagnosed with the disease in 1993. He attributed the disease to his exposure to asbestos-containing brake linings beginning in 1957. Rejecting Ford's entreaty to overrule *Grimshaw* and adopt a manifestation of harm approach, the court, as in *Grimshaw*, relied on medical testimony that the plaintiff's mesothelioma began to develop ten years prior to diagnosis, and held that the cause of action therefore arose prior to July, 1986. In a footnote, the court stated that, unlike certain other conditions, "a condition such as cancer is a compensable injury when it comes into existence even without symptomatology." *Id*. at 45 n.11, 703 A.2d at 1336 n.11.

In *Owens Corning v. Bauman*, 125 Md. App. 454, 726 A.2d 745 (1999), which involved a plaintiff who attributed his mesothelioma, diagnosed in 1995, to exposure to asbestos in the 1970's, the court distinguished *Abate* on the ground that, when the alleged injury at issue arises from pleural plaques, as it did in *Abate*, some manifestation of harm is required. *Abate*, it said, did not change the reasoning applied in *Grimshaw*. Once more, the court rejected the defendants' urging to overturn *Grimshaw* and to adopt a manifestation of harm test for purposes of § 11-108(b)(1). Unfortunately, in its effort to distinguish between the contraction of a *disease*, such as asbestosis or cancer, and the contraction of a non-harmful *condition*, such as pleural plaques, it was somewhat less clear in restating the

applicable test. At one point, it declared that "[w]hen a plaintiff actually contracts an asbestos-related *disease*, the legally compensable harm *may be retraced to the first moment of cellular change*; however, when a plaintiff contracts the *condition* of pleural plaques, the legally compensable harm only arises with the onset of a symptom." *Id*. at 482, 726 A.2d at 759 (second emphasis added). That statement suggests that, where cancer is the injury sued upon, the action arises upon the cellular change, which, almost concededly, occurs shortly after exposure and long before any noticeable symptoms. A paragraph later, however, the court declared:

> "In sum, mere exposure, without cellular change, does not constitute an injury or harm for which one may maintain a cause of action. *Furthermore, cellular change without accompanying injury does not constitute harm or functional impairment that would give rise to a cause of action.* For purposes of the statutory cap, the crucial distinction is whether a plaintiff's cellular change develops into an asbestos-related *disease* or simply into an asbestos-related *condition*.
>
> When cellular change later results in an asbestos-related disease, the harm was irreversible from the time of contraction, *and the 'injury' as well as the cause of action arose when the disease came into existence*. Consequently, the presence or absence of symptomatology is irrelevant for purposes of the statutory cap, because the cause of action arose when the disease was contracted. On the other hand, when a plaintiff becomes afflicted with an asbestos-related condition, such as pleural plaques, it is not until symptomatology is present that any functional impairment occurs."

Id. at 482-83, 726 A.2d at 759 (emphasis added).

Although the court noted, and did not dispute, the concern posited by the defendants

that this latter articulation, which is consistent with the test adopted in *Grimshaw*, was a difficult one to apply and tended to generate a great deal of disputed medical testimony, the court believed that it was mandated by *Armstrong* and was consistent with the legislative intent behind § 11-108(b)(1). It further held that, when in dispute, the issue of when the injury comes into existence was for the trier of fact, and not the court *qua* court, to decide. In the particular case, because the jury awarded non-economic damages exceeding the cap without specifically making a determination on that issue, the court remanded the matter for submission to a new jury. *Id*. at 522, 726 A.2d at 778-79.

The confusion underlying these two articulations was exacerbated in *Owens-Corning v. Walatka*, 125 Md. App. 313, 319, 725 A.2d 579, 581-82 (1999), in which the court, in a parenthetical reference, regarded *Bauman* as holding that, for purposes of § 11-108(b)(1), disease comes into existence "when, based on expert testimony, the carcinogen caused cellular changes *which led to* an irreversible, fatal, or disabling disease rather than the point in time when the plaintiff inhaled the asbestos, or when the plaintiff was diagnosed or manifested symptoms of such disease." (Emphasis added). In *Hollingsworth v. Connor*, 136 Md. App. 91, 764 A.2d 318 (2000), however, the court expressly disavowed the statements from *Bauman* regarding the retracing to the first moment of cellular change and confirmed its statement that "'[w]hen cellular change later results in an asbestos-related disease, the harm was irreversible from the time of contraction, and the "injury" as well as the cause of action arose when the disease came into existence.'" *Hollingsworth*, 136 Md. App. at 128,

764 A.2d at 338 (quoting *Bauman*, *supra*, 125 Md. App. at 482, 726 A.2d at 759). The court held that "the critical point in time . . . should not have been whether [the plaintiff] had experienced *cellular change* before July 1, 1986; the question should instead have pertained to whether [the plaintiff] had *contracted mesothelioma* before July 1, 1986." *Id*. at 130-31, 764 A.2d at 339.

The parties in this appeal seem to agree on only one thing in this regard – that we should not follow *Grimshaw*. Garlock urges that we adopt the manifestation of harm test in latent disease cases and hold that a cause of action arises, for purposes of § 11-108(b)(1), when the plaintiff either experiences symptoms of the disease or the disease is diagnosed. Crane waffles somewhat on what test should apply, but does suggest that the plaintiff must show that he or she had an "irreversible, fatal, or disabling disease" prior to July 1, 1986. The Scribners contend that we essentially set the standard in *Armstrong* and asks that we simply confirm it. As they construe *Armstrong*, an injury occurs and the cause of action arises when the plaintiff incurs cellular changes that lead to the disease, which, according to the expert evidence, occurs shortly after exposure. *Grimshaw* and its progeny, they argue, are *not* consistent with *Armstrong*.

Before us, in essence, are three possible approaches for determining when a cause of action arises for purposes of § 11-108(b)(1): (1) the manifestation approach, which is the latest in time and looks to when the disease sued upon first becomes either symptomatic or diagnosed, (2) the exposure approach, which is the earliest in time and looks to when the

plaintiff first inhaled asbestos fibers that caused cellular changes leading to the disease, and (3) the *Grimshaw* approach, which, as to disease, looks to when the disease itself first arose in the body. None of these approaches are problem-free, but the one that presents the fewest significant problems and is most consistent with the statutory language is the second.

The manifestation approach has, as its only assets, simplicity and certainty. It is much easier to establish when a disease was diagnosed or became symptomatic than to establish when cellular changes have progressed into a disease that is not, at the time, detectable. If we were to adopt that approach, much of the medical evidence now elicited from pathologists and other experts concerning when the disease first came into existence would not be necessary. The problem with the approach is that it flatly ignores the distinction made by the Legislature between when an action arises and when it accrues, and is therefore wholly inconsistent with the statute. We explained this quite clearly in *Armstrong* and nothing offered by the petitioners has persuaded us that our construction of the statute in that case was erroneous or is in need of modification. It is virtually conceded, even by asbestos-action defendants, that diseases such as cancer and asbestosis exist in the body before they become symptomatic and before they are capable of clinical diagnosis. The manifestation approach would nonetheless apply the cap even when it is clear that the disease existed, and thus the cause of action based on that disease arose, prior to July 1, 1986. We confirm our rejection of that approach.

The *Grimshaw* approach, as the Court of Special Appeals initially and most recently

articulated it, has some conceptual plausibility, but it suffers from the fact that it is impossible to apply in any uniform and rational way and necessarily engenders competing expert testimony as to the timing of an event that no one can precisely define. It first draws a distinction between "conditions" that become symptomatic and "diseases," notwithstanding that both clearly constitute injuries, and treats them differently. In the case of the former, the action does not arise until the condition becomes symptomatic; with respect to the latter, the action arises when the disease first commences, which is likely to be long before it becomes symptomatic. More important, in cancer cases it requires the evidence to focus on when the first cell turned cancerous, which everyone seems to agree cannot be ascertained with any precision under the technology now available. The parties are thus put to proving, or disproving, that which cannot be proved or disproved on a clinical basis, and they must rely instead on theoretical approximations based on assumptions that even the experts who present them concede are not wholly accurate.[4] It is not a workable approach.

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[4] The testimony in this case alone, much less a comparison of it with testimony in other cases, illustrates the problem. Dr. Hammar testified that it was not possible to place an exact date on when a tumor first occurs, much less when the first cell turns cancerous. The latency period for mesothelioma, he said, ranged from 20 to 50 years. He knew of one case with a latency period of five years and another with a period of 72 years. Measurement of onset through doubling times was fraught with uncertainty. One assumption was that the

(continued...)

The exposure approach is consistent with our holdings in *Mitchell* and *Murphy v. Edmonds*, 325 Md. 342, 601 A.2d 102 (1992), and, if carefully delineated, is both theoretically supportable and workable. It rests, initially, on the premise that there is, in fact, an injury. If there is no injury, there is no cause of action. Thus, it need not attempt to address the problem of entirely inconsequential exposures or exposures that produce only pleural plaques or other conditions that, absent more, do not constitute injuries, which seems to have plagued the Court of Special Appeals, for, if that is all that the plaintiff has, no cause of action exists and § 11-108(b)(1) never comes into play. We start, then, with the requisite premise that the plaintiff has established to the satisfaction of the trier of fact that he or she has an injury that was proximately caused by exposure to the defendant's asbestos-containing product. Whether the injury sued upon is cancer or asbestosis, the plaintiff must, at the outset, establish that he or she has that disease and that it was caused, in whole or substantial part, by exposure to the defendant's asbestos-containing product. The question, for purposes of § 11-108(b)(1), is when that injury came into existence.

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[4](...continued)
cancer is spherical, which Dr. Hammar acknowledged is not always the case and apparently is not the case with mesothelioma. Another assumption was a constant growth rate, which Drs. Hammar and Edward Gabrielson, another pathologist, both said may not be accurate. The times themselves were approximations – the 200-day cycle used by Dr. Hammar being the "shortest" for a biphasic mesothelioma.

In the case of cancer, the most accurate answer to that question seems to be that, on the basis of our present technology, no one can ever tell. There is no available test and no reasonably reliable methodology to determine when the first cell that turned cancerous did so, or even when the first hundred thousand cells did likewise. Although the medical evidence shows that cancers take time to develop and may remain *in situ* and non-invasive for long periods of time, it has not been seriously urged, and we would not be prepared to accept it if it were urged, that an *in situ* and non-invasive cancer is not an injury; an undetectable malignant tumor *is* an injury.

What the evidence in nearly all of the cases reveals is that, (1) inhalation of asbestos fibers causes cellular damage, (2) the cellular damage occurs shortly after inhalation, (3) with respect to cancer, the exposure of the cells to asbestos fibers causes the cells to divide, (4) the increased cellular division increases the risk of cellular genetic error, and (5) that, in turn, increases the risk of one or more cells turning cancerous. The evidence establishes, as well, that the greater the exposure, at one time or over time, the greater is the cellular damage, the greater is the chance that the ordinary body defenses will be unable to cope with that damage, and the greater is the likelihood of disease formation. The evidence, viewing the process in hindsight, is that, if the plaintiff in fact has a disease that he or she establishes is traced to exposure to asbestos, it developed from the cellular damage caused by the asbestos inhalation. Although it is as impossible to ascertain which fiber ultimately caused which cell, over time, to escape the body's defenses and turn cancerous, as it is to determine when that

occurred, the certainty is that it did occur. In *Mitchell*, we regarded that cellular damage, caused by the inhalation of asbestos fibers, and which later produced the disease, as a bodily injury.

In nearly all of the asbestos cases that have arisen under § 11-108(b)(1), beginning with *Armstrong* and including this one, the plaintiff's last exposure to asbestos (or at least to the defendant's asbestos-containing product) was well before 1986, in most instances in the 1950's, 1960's, or 1970's. That may not always be the case, of course, but so far it has been and most likely will continue to be so. Thus, in all of these cases, the cellular damage that actually led to the onset of the disease occurred prior to July 1, 1986. Given the practical impossibility of ascertaining with any degree of precision when that onset actually occurred, we consider it to be more reasonable to look back to the exposure that ultimately produced the disease, which cannot, of course, be later than the last exposure, than to engage in "guesstimates" of when the first cell became diseased, "guesstimates" based on contradictory expert testimony – the plaintiffs' experts invariably moving the date back and the defendants' experts just as invariably moving it forward – all of which, in any event, seems to be founded upon uncertain assumptions. *See Ins. Co. North America v. Forty-Eight Insulations*, 633 F.2d 1212, 1218 (6th Cir. 1980), *clarified*, 657 F.2d 814, *cert. denied*, 454 U.S. 1109, 102 S. Ct. 686, 70 L. Ed. 2d 650 (1981) (noting that "it is almost impossible for a doctor to look back and testify with any precision as to when the development of asbestosis 'crossed the line' and became a disease" and, as we did in *Mitchell*, adopting the exposure approach to

determine when a bodily injury occurred for insurance coverage purposes).

In *Murphy v. Edmonds, supra*, 325 Md. 342, 601 A.2d 102, we noted that § 11-108 was first enacted in response to a perceived insurance crisis and that the General Assembly's objective was "to assure the availability of sufficient liability insurance, at a reasonable cost, in order to cover claims for personal injuries to members of the public." *Id*. at 369, 601 A.2d at 115. We added that "[a] cap on noneconomic damages may lead to greater ease in calculating premiums, thus making the market more attractive to insurers, and ultimately may lead to reduced premiums for individuals and organizations performing needed services." *Id*. at 369-70, 601 A.2d at 115. The exposure approach that we adopt is in no way inconsistent with that legislative objective. Neither the availability nor the cost of liability insurance now should be affected by whether judgments based on exposures occurring prior to 1986 are subject to the cap. Those claims, if covered at all, would be covered under policies that have long since expired, not under ones being purchased today.

We thus hold that, in actions for personal injury founded on exposure to asbestos, the court, as an initial matter, may look, for purposes of § 11-108(b)(1), to the plaintiff's last exposure to the defendant's asbestos-containing product. If that last exposure undisputedly was *before* July 1, 1986, § 11-108(b)(1) does not apply, as a matter of law. If the only exposure was undisputedly *after* July 1, 1986, then obviously the cap applies as a matter of law. In those hopefully rare instances in which there was exposure both before and after July 1, 1986, and there is a genuine dispute over whether either exposure was sufficient to cause

the kind of cellular change that led to the disease, the trier of fact will have to determine the issue based on evidence as to the nature, extent, and effect of the pre- and post-July 1, 1986 exposures. In this case, it was undisputed that Scribner's last exposure to Crane's and Garlock's products occurred well before 1986. Accordingly, for that reason alone, the judgment of the Circuit Court was not in error.

Burden of Proof

Crane and Garlock contend that Scribner had the burden of establishing that his cause of action arose prior to July 1, 1986, that, in the main trial, he produced insufficient evidence to establish that fact, and that, as a result, they were entitled, as a matter of law, to a reduction of the non-economic damages in conformance with § 11-108(b)(1). Although Scribner certainly contests the argument that he failed to produce sufficient evidence of when his cause of action arose, he does not take issue with the assertion that he had the burden of proof on the issue. The Court of Special Appeals dealt with this issue in *Owens-Corning v. Walatka, supra*, 125 Md. App. at 322-31, 725 A.2d at 583-88, and, for the reasons well stated by Judge Adkins in that case, we conclude that the burden is on the plaintiff to establish that his or her cause of action arose prior to the effective date of the applicable cap.

Although, in enacting § 11-108, the Legislature made no statement regarding who has the burden with respect to its application, it made clear enough its policy that the statute should apply unless the cause of action arose prior to its enactment. That suggests, for

actions filed and reaching judgment after July 1, 1986, an intended presumption of application, and it is the usual rule that a party who seeks exemption from a statute has the burden of justifying the exemption. *See Newell v. Richards*, 323 Md. 717, 724-26, 594 A.2d 1152, 1156-57 (1991). Moreover, as the Court of Special Appeals pointed out, the issue of when the plaintiff was exposed to the defendant's product is one upon which the plaintiff will normally have superior knowledge, and it is therefore more fair and more practical to place the burden of production and persuasion on the plaintiff. *See Walatka*, *supra*, 125 Md. App. at 326-27, 725 A.2d at 585-86; *see also Winkler Constr. Co. v. Jerome*, 355 Md. 231, 254, 734 A.2d 212, 225 (1999) (following "the general rule in civil actions that, when a particular party has peculiar knowledge of a fact, the burden of alleging and offering evidence of that fact is on that party"); 2 KENNETH S. BROUN, ET AL., MCCORMICK ON EVIDENCE § 337, p. 413 (John W. Strong ed., 5th ed. 1999).

Who Makes the Decision?

Apparently as a result of some language used by the Court of Special Appeals in *Grimshaw*, the trial courts in Baltimore City took the position that the issue of whether the cap applied was for the court, not the jury, to determine, and that included the resolution of any dispute over when the plaintiff's cause of action arose. In *Owens Corning v. Bauman, supra*, 125 Md. App. 454, 726 A.2d 745, which, as noted, was filed *after* the first jury's

verdicts were returned in this case, the court made clear that, if there was any genuine dispute over the matter, it was for the jury to determine. That is what led to the impaneling of the second jury and the submission of that issue to it here.

The problem seemed to be (1) the language in § 11-108(d) that, in a jury trial, the jury may not be informed of the statutory limitation and that, if its award exceeded the cap, "the court shall reduce the amount to conform to the limitation," and (2) language in *Grimshaw* and *Ford Motor Co. v. Wood, supra*, 119 Md. App. 1, 703 A.2d 1315, to the effect that a factual determination of that issue by the court will not be overturned unless clearly erroneous. The *Bauman* court explained that the statutory direction not to reveal the cap to the jury did not remove from it the obligation to determine the factual question of when the plaintiff's cause of action arose, and that the issue of whether that question was for the court or the jury was simply not raised in *Grimshaw* or *Ford*. Relying on *Hill v. Fitzgerald*, 304 Md. 689, 501 A.2d 27 (1985), *Murphy v. Edmonds, supra*, 325 Md. 342, 601 A.2d 102, and *Ethridge v. Medical Ctr. Hosps.*, 376 S.E.2d 525 (Va. 1989), the *Bauman* court concluded that the resolution of any dispute over when the plaintiff's cause of action arose was part of the jury's fact-finding function in its assessment of damages, and that "when parties dispute the date upon which the cause of action arose, the jury must determine this issue in order to complete its function as trier of fact." *Bauman,* 125 Md. App. at 509, 726 A.2d at 772. We agree with that holding and with the reasoning behind it.

The question to be presented to the jury, *if there is a genuine dispute as to the matter*

and the jury decides to find for the plaintiff and award non-economic damages, is whether the plaintiff's cause of action arose prior to July 1, 1986. That question, of course, must be accompanied by appropriate instructions regarding the test the jury is to apply in determining that issue. The court's function, as a legal matter, is to decide whether the cap applies based on the jury's response to that question.

This Case

The case-specific complaints made by Crane and Garlock are that, (1) the § 11-108(b)(1) cap should apply as a matter of law because Scribner failed to produce sufficient evidence before the first jury that his cause of action arose prior to July 1, 1986, (2) presentation of the issue to the second jury was inappropriate because it allowed Scribner to produce evidence to that jury that was inconsistent with the evidence produced to the first jury regarding when the mesothelioma first came into existence, and (3) the verdict sheet presented to the second jury employed an improper exposure test for determining when the cause of action arose.

In light of our holding that the exposure test *is* the appropriate test to use, once the jury found that the plaintiff had, in fact, suffered an injury substantially caused by exposure to the defendant's asbestos-containing products, it is clear that none of these complaints has merit. The first jury concluded that Scribner suffered and died from mesothelioma and that the mesothelioma was substantially caused by exposure to Crane and Garlock products. As

we have indicated, the evidence was undisputed that Scribner's last exposure to those products was well before 1986. Under the test we have adopted, that made § 11-108(b)(1) inapplicable as a matter of law. Submission of the matter to the second jury, under the supposition that, under the *Grimshaw* test, a genuine dispute of fact was generated, was, in light of the jury's determination of the issue, harmless error.

JUDGMENT OF COURT OF SPECIAL APPEALS AFFIRMED, WITH COSTS.