

No. A122038

IN THE COURT OF APPEAL OF
THE STATE OF CALIFORNIA

FIRST APPELLATE DISTRICT
DIVISION ONE

GEORGIA-PACIFIC LLC,

Defendant and Appellant,

v.

JOAN MAHONEY AND DANIEL MAHONEY,

Plaintiffs and Respondents.

APPLICATION FOR LEAVE TO FILE BRIEF AMICUS CURIAE

Pursuant to California Rules of Court, rule 29.10, *amici curiae* Bruce Ames, Marcia Angell, John Duffus, Arnold Engel, Sheldon Glashow, Ronald Gots, Leonard Hamilton, Ronald Hart, Steven Lamm, Lawrence Litt, Dennis McBride, A. Alan Moghissi, Rodney Nichols, Malcolm Ross, Emanuel Rubin and Richard Wilson¹, request permission to file the accompanying brief as *amici curiae* in support of defendant and appellant Georgia-Pacific LLC.

Amici have reviewed the briefs on the merits filed in this case and believe that this Court will benefit from additional briefing on the issue of

¹ The credentials of *amici* are set forth in the biographical addendum to the brief.

causation and on the reasoning other courts have employed in analogous cases, expressing the views of neutral experts.

Accordingly, we ask the Court to accept and file the attached brief *amicus curiae* of BRUCE AMES, MARCIA ANGELL, JOHN DUFFUS, ARNOLD ENGEL, SHELDON GLASHOW, RONALD GOTS, LEONARD HAMILTON, RONALD HART, STEVEN LAMM, LAWRENCE LITT, DENNIS MCBRIDE, A. ALAN MOGHISSI, RODNEY NICHOLS, MALCOLM ROSS, EMANUEL RUBIN and RICHARD WILSON.

Dated: Larchmont, New York
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GEORGIA-PACIFIC LLC,

DEFENDANT AND APPELLANT,

v.

JOAN MAHONEY AND DANIEL MAHONEY,

PLAINTIFFS AND RESPONDENTS.

Appeal from the Superior Court of San Francisco County
The Honorable Thomas J. Mellon, Jr.
Case No. CGC-06-458140

**BRIEF *AMICUS CURIAE* OF
BRUCE AMES, MARCIA ANGELL, JOHN DUFFUS,
ARNOLD ENGEL, SHELDON GLASHOW, RONALD GOTS,
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CERTIFICATE OF INTERESTED ENTITIES

(California Rule of Court, Rule 8.208)

No counsel for a party authored this brief in whole or in part and no person or entity, other than *amicus curiae* and their counsel, made a monetary contribution to its preparation or submission. Georgia-Pacific LLC has not made any financial contribution to the preparation of this brief.

Neither Atlantic Legal Foundation nor any of the *amici* has received any compensation or funding from any party to this case.

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IN SUPPORT OF DEFENDANT-APPELLANT**

INTEREST OF *AMICI*

Amici are scientists, who have studied the issue of the role that scientific issues play in public affairs and in particular the way in which they can illuminate disputes between different persons or elements of society in the courts of law. *Amici* include physicians, geologists, physicists, epidemiologists and toxicologists.² Several of the *amici* submitted a brief in the Supreme Court in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, (1993) 509 U.S. 579, the seminal case discussing the federal rule for admissibility of expert scientific evidence, in *General Elec. Co. v. Joiner*, (1997) 522 U.S. 136, and in *Kumho Tire v. Carmichael*, (1999) 526 U.S. 137 and, in California, *San Diego Electric & Gas Co. v. Orange County Superior Court (Covalt)*, (1996) 13 Cal. 4th 893, *In re Lockheed Litig. Cases*, 115 Cal. App. 4th 558 (2004), *Aguilar v. ExxonMobil Corp.*, Court of Appeal, Second Appellate District, Division Three, Case No.: B166347, and *Kennedy v. Southern California Edison Company*, (2000 9th Cir.) 268 F.3d 763.

² The credentials of *amici* are set forth in the biographical addendum to this brief.

Amici are also aware of the significance of asbestos litigation nationally and in California, and they are concerned that the mere utterance of “asbestos” no matter the asbestos fiber-type, or the exposure, and “mesothelioma” can have undue impact on juries. *Amici* believe that based on all the evidence adduced at trial, Respondent did not and could not prove that Mrs. Mahoney’s exposure to asbestos from Georgia-Pacific’s joint compound was a “substantial factor” in causing Mrs. Mahoney’s pleural mesothelioma as required by *Rutherford v. Owens-Illinois, Inc.*, (1997) 16 Cal. 4th 953, 982 (emphasis revised from original; footnote omitted).

INTRODUCTION

Defendant-Appellant Georgia-Pacific LLC. has appealed from a judgment holding Georgia-Pacific Co. liable for Mrs. Mahoney’s asbestos-related disease and death. These injuries were allegedly caused by Mrs. Mahoney’s intermittent exposure to asbestos while helping her husband renovate houses in the 1970s. (4 RT 740, 802-825; 4 RT 744).³

³ Mrs. Mahoney’s main tasks in her husband’s business did not expose her to asbestos – she oversaw the company's books and accounts receivable; she also helped clean the properties prior to site inspections. (3 RT 502-503, 579-580; 4 RT 737-738.) Mrs. Mahoney testified that she helped Mr. Mahoney cut CertainTeed pipe, which contained amphibole asbestos – a type much more likely to cause mesothelioma -- "two or three times" and "occasionally" held the pipe while he cut it. (4 RT 746-749, 759-761; see 3 RT 702.) She estimated that she sanded joint compound more than 50 times and swept up the dust almost 100 times during the 1970s. (4 RT 760.) Her son Sean, who was a young child when the Mahoneys started the construction
(continued...)

The issue on appeal which *amici* address is whether there was evidence that Mrs. Mahoney's exposure to chrysotile asbestos used by Georgia-Pacific LLC in its joint compound ("G-P joint compound") which Mr. Mahoney used in his construction business was the cause of Mrs. Mahoney's mesothelioma, *i.e.*, whether it was a substantial factor in bringing about the disease. *Rutherford v. Owens-Illinois, Inc.*, (1997) 16 Cal. 4th 953, 982 (emphasis added; footnote omitted).

Amici believe, after a review of the trial record, that as a matter of law plaintiffs did not bring forward evidence of a substantial causal connection between asbestos in Georgia-Pacific's joint compound and Mrs. Mahoney's illness.

The verdict in this case should be overturned because Respondents failed to establish that Mrs. Mahoney's exposure to chrysotile asbestos from G-P joint compound caused her mesothelioma, as required by *Rutherford v. Owens-Illinois, Inc.* (1997) 16 Cal.4th 953. Although Respondents' experts asserted that any "special exposure" to any form of asbestos is legally sufficient to cause mesothelioma, they failed to establish the extent of Mrs.

³(...continued)

business, estimated that Mrs. Mahoney used both G-P and Bondex joint compound over 50 times each. (3 RT 571, 600, 608.) Her daughter Deborah testified that Mrs. Mahoney cleaned up "200-300 homes." (3 RT 502503, 508.) Another son, Daniel, testified that the family used Bondex, Kaiser Gypsum and G-P joint compound. (3 RT 707; 4 RT 793.)

Mahoney's exposure to G-P joint compound, and failed to take into account the overwhelming number of recent studies that show that *chrysotile* asbestos much greater than Mrs. Mahoney's rarely, if ever, increases the risk of developing mesothelioma. The age-adjusted rates of pleural mesothelioma among women in the United States recently have been reported as more or less constant at about 2.5 per million person-years over the period 1973–2005, indicating women have not experienced the observable increase in pleural mesothelioma seen among men.

Appellants' experts were unable to estimate the extent, frequency or intensity of Mrs. Mahoney's exposure to chrysotile fibers from Georgia-Pacific's product. (*See, e.g.*, 2 RT 352-356)⁴ They merely "assumed" that Mrs. Mahoney's exposure to G-P joint compound was "special" enough to have caused her disease.⁵ Respondents' "special exposure" theory is based on the hypothesis that virtually *any* exposure to *any* asbestos-containing product causes mesothelioma. This conflicts with current studies of asbestos exposure and with *Rutherford's* rejection of the theory that exposure to any asbestos,

⁴ Dr. Mark testified "It's the asbestos in these various dusts that causes the cancer." (2 RT 307; emphasis supplied.)

⁵ Plaintiffs' expert, Richard Hatfield, estimated that Mrs. Mahoney's exposure to chrysotile asbestos fibers from G-P products was less than 1/35th of her exposure to a mixture of amphibole and chrysotile fibers from cutting CertainTeed pipe. (3 RT 632-633, 636, 638.)

alone, is enough to establish causation. Respondents' expert testimony was therefor insufficient to establish causation.

Moreover, Respondents' experts based their testimony on high-dose studies involving different types of asbestos that are indisputably more toxic than the chrysotile fibers contained in Appellant's product. No epidemiological study has shown that low-dose exposure to chrysotile increases the risk of mesothelioma in humans.

The causation testimony of Respondents' experts did not establish either general nor specific causation, should not have been admitted, and the verdict should be reversed.⁶

⁶ Plaintiffs proffered the testimony of Dr. Eugene Mark to establish that Mrs. Mahoney's exposure to G-P joint compound caused her mesothelioma. Dr. Mark testified that any "special exposure" to any asbestos-containing product can contribute to the risk of developing an asbestos-related cancer (2 RT 357). Dr. Mark defined "special exposure" as the kind of exposure that might occur in an occupational setting, where a worker is repeatedly exposed to asbestos dust. Dr. Mark did not identify the threshold between "special" and non-"special" exposures, nor did he place Mrs. Mahoney's exposure to asbestos from G-P joint compound along the spectrum he described. Dr. Mark admitted that he knew nothing about Mrs. Mahoney's exposure to G-P joint compound, and his information derived from reviewing a "work history sheet" prepared by Plaintiffs' counsel and conferring with Mrs. Mahoney the day he testified (2 RT 351-352). Dr. Mark acknowledging that some forms of asbestos to which Mrs. Mahoney was exposed are more potent than the type of asbestos found in G-P joint compound (2 RT 374), but he was unable to compare Mrs. Mahoney's exposure to G-P joint compound with her exposures to other products containing more potent type of asbestos (2 RT 352-356). Dr. Mark conceded that there are no epidemiological studies which show an increased incidence of mesothelioma among career drywall workers (2 RT 394).

(continued...)

ARGUMENT

I. Plaintiffs Must Quantify Exposure of Sufficient Frequency, Regularity and Proximity to Asbestos Fibers Released from Defendant's Product.

Under *Rutherford*, a plaintiff must show that "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." (16 Cal.4th at pp. 969, 977-978 & fn. 11; emphasis

⁶(...continued)

Respondents' epidemiologist, Dr. Richard Lemen, did not consider any evidence of Mrs. Mahoney's particular exposure to G-P joint compound or to any other asbestos-containing product (3 RT 468). Although Dr. Lemen testified that all types of asbestos cause lung cancer and mesothelioma in humans (3 RT 426), he admitted that he was not qualified to offer any opinion as to whether Mrs. Mahoney's cancer was caused by her exposure to G-P joint compound (3 RT 409-410). He limited his testimony to whether chrysotile asbestos found in G-P joint compound could cause mesothelioma more generally (3 RT 407-409). Dr. Lemen acknowledged that mesothelioma is a dose-response disease, so that as the cumulative dose increases, the risk of disease increases. (3 RT 431, 448.) He testified that there is no known concentration of asbestos exposure below which some individuals will not be at risk for mesothelioma (3 RT 433), but admitted that science is currently incapable of analyzing the effects of low levels of exposure to asbestos (3 RT 476.) He acknowledged that amphibole asbestos fibers have a different physical chemistry and are significantly more potent than the chrysotile fibers in G-P joint compound. (3 RT 478-479.) Dr. Lemen estimated that amphibole fibers are two to four times more carcinogenic than chrysotile fibers. (3 RT 408-409.) While Dr. Lemen testified that mesothelioma has been described in case reports involving as little as one day of workplace exposure, he did not identify the form of asbestos involved in those cases (3 RT 433-434). He also conceded that no epidemiological studies show an increased incidence of mesothelioma among long time drywall workers (3 RT 482).

in original.) While the plaintiff need not prove "with medical exactitude that fibers from a particular defendant's asbestos-containing product or products were those, or among those, that actually began the cellular process of malignancy," it does not permit a plaintiff to establish causation by showing nothing more than the injury was caused by exposure to asbestos fibers in general or some exposure to the defendant's product. (*Id.* at p. 958.) A plaintiff cannot meet this standard by offering expert testimony that a defendant's product played a "theoretical" role in bringing about the injury: "a force which plays only an 'infinitesimal' or 'theoretical' part in bringing about injury, damages, or loss is not a substantial factor". (*Rutherford* at 969)

Because asbestos-related cancers are dose-response diseases⁷, the plaintiff must show that exposure to the defendant's product (and not merely to similar products) contributed to the aggregate dose of asbestos sufficient to increase the risk of cancer. (*Rutherford* at 976.)

[A]t a level of abstraction somewhere between the historical question of exposure and the unknown biology of carcinogenesis, the question arises whether the risk of cancer created by a plaintiff's exposure to a *particular* asbestos-containing product was significant enough to be considered a legal cause of the disease. Taking into account the length, frequency, proximity and intensity of exposure, the *peculiar properties of the individual product*, any other potential causes to which the disease could be attributed (e.g., other

⁷ Dr. Lemen acknowledged that mesothelioma is a dose-response disease, so that as the cumulative dose increases, the risk of disease increases (3 RT 431, 448).

asbestos products, cigarette smoking), and perhaps other factors affecting the assessment of comparative risk, should inhalation of fibers from the particular product be deemed a 'substantial factor' in causing the cancer?

Id. at p. 975 (emphasis supplied).

Rutherford requires that the jury have some basis for evaluating causation in light of the plaintiff's claimed dose of asbestos from the defendant's product. In this case, the jury had no basis for concluding that Mrs. Mahoney's exposure to chrysotile asbestos from G-P joint compound was "significant enough" to constitute a substantial factor in causing her cancer. (*Id.*) Plaintiffs' experts failed to consider the length, frequency, proximity and intensity of Mrs. Mahoney's exposure to G-P joint compound, or the significant potency differences among the various asbestos products to which Mrs. Mahoney was exposed during her life.⁸

⁸ While the jury heard evidence of the frequency, duration and intensity of Mrs. Mahoney's exposure to G-P joint compound and other asbestos products from other witnesses, they had no basis for evaluating that evidence to determine whether Mrs. Mahoney's exposure to G-P joint compound was "substantial" enough to increase her risk of injury. Dr. Mark, as noted above, admitted that there were exposures to chrysotile asbestos that were not sufficient to cause cancer, but he offered no opinion as to where on the spectrum between his category of "special" exposures that cause cancer and non-"special" exposures that he believes do not cause cancer Mrs. Mahoney's exposures to G-P joint compound fell. He had no data on the frequency, duration, intensity or proximity of her exposures, if any. The hypothetical questions posed to Dr. Mark about sanding and sweeping G-P joint compound did not specify the frequency, duration, or intensity of the sanding and sweeping activity. Dr. Mark simply assumed that the frequency and duration of the exposures on which the hypotheticals were based was substantial (continued...)

Amici submit that the *Rutherford* decision, coupled with well-reasoned authority from other jurisdictions, suggest that it is not enough simply to show some minimal exposure to some asbestos fibers from a defendant's product.

It 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.

Eaton, D.L., *Scientific Judgment & Toxic Torts: A Primer in Toxicology for Judges and Lawyers*, (2003) 12 J.L. & POL 5, 39 (quoted in *Borg-Warner Corp. v. Flores*, (2007 Tex.) 232 S.W.3d 765, 773, 2007 WL 1650574).

The "length, frequency, proximity and intensity of exposure" test articulated in *Rutherford*, 16 Cal.4th at 975, is a straightforward application of the requirement that plaintiffs prove substantial-factor causation as an essential element of a negligence or strict liability claim. See *Lohrmann v. Pittsburgh Corning Corp.*, (4th Cir. 1986) 782 F.2d 1156, 1162, 1163. The frequency, regularity, proximity test has been adopted in a majority of jurisdictions in

⁸(...continued)

enough to qualify as "special exposures." He also "made the assumption that the asbestos to which [Mrs. Mahoney] was exposed caused her disease." (See 2 RT 353). Thus he assumed the conclusion plaintiffs were required to prove through his testimony, and his reasoning was circular. Dr. Mark's assumptions do not constitute an evidentiary basis for permitting a jury to conclude that G-P joint compound was in fact a legal cause of Mrs. Mahoney's cancer. See *In re Lockheed Litigation Cases* (2004) 115 Cal.App.4th 558, 563 ("[w]here an expert bases his conclusion . . . upon factors which are speculative, remote or conjectural, then his conclusion has no evidentiary value").

evaluating whether there is sufficient proof to support a reasonable inference of substantial factor causation in an asbestos product liability case. *See also Slaughter v. Southern Talc Co.*, (5th Cir. 1991) 949 F.2d 167, 171 (“The most frequently used test for causation in asbestos cases is the ‘frequency-regularity-proximity’ test announced in *Lohrmann*”); *Chavers v. General Motors Corp.*, (Ark. 2002) 79 S.W.3d 361. “In toxic tort cases, ‘scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that plaintiff was exposed to such quantities are minimal facts necessary to sustain the plaintiff’s burden’”] (quoting *Allen v. Pennsylvania Engineering Corp.* (5th Cir. 1996) 102 F.3d 194, 199).⁹ *See also* Faigman, D.L., *et al.*, MODERN SCIENTIFIC EVIDENCE: THE LAW & SCIENCE OF EXPERT TESTIMONY 28:5 (2005-2006 ed.)

While it may be common for plaintiffs to submit expert testimony claiming that *any* exposure to asbestos, no matter how minimal, is a substantial contributing factor in asbestos disease, such generalized opinions do not suffice to create a jury question in a case where exposure to the defendant’s product is *de minimis*, particularly in the absence of evidence excluding other possible sources of exposure or in the face of evidence of substantial exposure from other sources. *Gregg v. V-J Auto Parts Co.* (Pa. 2007) 943 A.2d 216,

⁹ Footnote 9 intentionally omitted.

226-227 ([it is “a fiction that each and every exposure to asbestos, no matter how minimal in relation to other exposures, implicates a fact issue concerning substantial-factor causation”]; *Lindstrom v. AC Products Liability Trust* (N.D. Ohio 2003) 264 F.Supp.2d 583, 588, *aff’d* (6th Cir. 2005) 424 F.3d 488 (“If an opinion [that there is no safe level of asbestos exposure] would be sufficient for plaintiff to meet his burden, the . . . ‘substantial factor’ test would be meaningless”). One of the difficulties courts face in the mass tort cases arises from the willingness of some experts to offer opinions that are not grounded in the underlying facts or to offer opinions that are not grounded in accepted scientific methodology. *See Gregg v. V-J Auto Parts Company, supra.*

Amici believe that it is appropriate for courts to make a reasoned assessment concerning whether, in light of the evidence concerning frequency, regularity, and proximity of a plaintiff’s/decendent’s asserted exposure, a jury could make the necessary inference of a sufficient causal connection between the defendant’s product and the asserted injury. A court, in determining whether there is a reasonable basis for an expert opinion under Evidence Code section 801, subdivision (b), must examine the method that the expert relied on in forming his or her opinion in order to determine for itself whether it can provide a reasonable basis for the expert's opinion. *See Evid. Code, § 801, subd. (b).* Application of Evidence Code sections 801 and 803 ensures that trial courts will use their statutory authority to assess the adequacy of the

foundation for an expert's opinion and the reliability of an expert's testimony. Section 801(b) of the Evidence Code provides that expert testimony must be “of a type that reasonably may be relied upon by an expert in forming an opinion upon the subject to which his testimony relates.”¹⁰ Reliability is a fundamental requirement for testimony to be admissible. *See, e.g., People v. Bui*, (2001) 86 Cal.App.4th 1187, *Korsak v. Atlas Hotels, Inc.*, (1992) 2 Cal.App.4th 1516, 1524, *Pacific Gas & Elec. v. Zuckerman*, (1987) 189 Cal.App.3d 1113.

Amici understand that under California case law Plaintiff does not need to prove with “medical exactitude” that fibers from a particular defendant's asbestos containing products were those, or among those, that actually began the cellular process of malignancy. (*Rutherford*, 16 Cal.4th at 958.) and that *Rutherford* does not require that the defendant's product independently caused plaintiff's injury, or that, but for that exposure, the plaintiff would not have contracted the injury. (*See Jones v. John Crane, Inc.*, 132 Cal.App.4th 990, 998 fn. 3.) Respondents' experts attempted to draw conclusions about *chrysotile* asbestos from studies involving completely different, more toxic, forms of asbestos. (*See* 2 RT 374, 3 RT 408-409.) They also relied on studies

¹⁰ *See People v. Gardeley* (1996) 14 Cal.4th 605, 618.) Section 801, subdivision (b) limits expert testimony "to such an opinion as is [b]ased on matter . . . that is of a type that reasonably may be relied upon by an expert in forming an opinion upon the subject to which his testimony relates" (Emphasis supplied)

involving a different disease – lung cancer – which is not the disease diagnosed in this case. (3 RT 455.) Respondents’ repeated reliance of evidence that some types of asbestos cause cancer, without any analysis or differentiation of the different effects of specific types of asbestos, is a fatal flaw.¹¹

In this case, we submit, given the paucity of evidence of any frequent, regular or sustained exposure of Mrs. Mahoney to asbestos in defendant’s products, the jury could not logically make such an inference. *See Gregg v. V-J Auto Parts Company, supra.*

Rutherford teaches that California courts should take into account the length, frequency, proximity and intensity of exposure, the properties of the individual product, any other potential causes to which the disease could be attributed (*e.g.*, other asbestos products, other occupational or environmental exposures, or other direct exposure such as cigarette smoking), and perhaps other factors affecting the assessment of comparative risk. *Borg-Warner Corp. v. Flores*, (Tex. 2007) 232 S.W.3d 765, 2007 WL 1650574, at *1 , *7. *See Rutherford* at 16 Cal. 4th at 975, *citing Lineaweaver v. Plant Insulation Co.*,

¹¹ Respondents assert that Appellant's expert, Dr. Hughson, admitted that even the chrysotile form of asbestos can cause malignant mesothelioma. (5 RT 1244.) They fail to inform the court that Dr. Hughson testified, consistent with current studies, that "very heavy exposure to chrysotile contaminated with tremolite [an amphibole type of asbestos]" is required to be causally linked to mesothelioma.(5 RT 1288; emphasis supplied).

(1995) 31 Cal. App. 4th 1409, 1416-17, *Lohrmann v. Pittsburgh Corning Corp.* (4th Cir. 1986) 782 F.2d 1156, 1162-1163.

The REFERENCE MANUAL ON SCIENTIFIC EVIDENCE published by the Federal Judicial Center and distributed to all federal judges provides the following guidance:

An opinion on causation should be premised on three preliminary assessments. First, the expert should analyze whether the disease can be related to chemical exposure by a biologically plausible theory. Second, the expert should examine if the plaintiff was exposed to the chemical in a manner that can lead to absorption into the body. *Third, the expert should offer an opinion as to whether the dose to which the plaintiff was exposed is sufficient to cause the disease.*

See Goldstein, B.D. and Henifin, M.S., *Reference Manual on Toxicology*, in Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 401, 419 (2d ed. 2000) (emphasis added). Specific causation thus demands a knowledge or reliable estimate of exposure and hence dose. An opinion on causation should set forth a plaintiff's exposure to a toxin, that the toxin is capable of causing the particular illness (general causation) and that plaintiff was exposed to sufficient levels of the toxin to cause the illness (specific causation). See *Parker v. Mobil Oil Corp.*, (2006) 7 N.Y.3d 434, *rearg. den.*, ___ N.Y.3d ___, 2007 N.Y. LEXIS 3. None of plaintiffs' experts provided sufficient information to support an opinion regarding specific causation because they failed to follow the well-established standard scientific approach employed by toxicologists and environmental scientists to determine reliably

whether chemicals have caused a particular disease in an individual, which involves a determination of: (1) whether the individual was exposed to a particular chemical or chemicals plausibly associated with the illness at issue; (2) the quantitative ‘dose’ of the chemical(s) that the person absorbed; and (3) whether that dose is capable of causing the specific illness (also known as ‘dose-response’ and ‘biological plausibility’). *See* Guzelian, P.S., Victoroff, M., Halmes, N.C., James, R.C., Guzelian, C.P., “Evidence-based Toxicology: A Comprehensive Framework for Causation,” (2005) 24 *Human and Experimental Toxicology* 161-201; Guzelian, P.S. and Guzelian, C.P., “Authority-Based Explanation,” (2004) 303 *Science* 1468-1469.

A determination of dose is critical because, as Paracelsus explained as early as the sixteenth century, “All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy.” *See* Doull and Bruce, “Origin and Scope of Toxicology,” in Casarett & Doull’s *TOXICOLOGY: THE BASIC SCIENCE OF POISONS* (3d ed. 1986). Succinctly put, “[t]he dose makes the poison.” *National Bank of Commerce v. Assoc. Milk Producers, Inc.*, (1998 E.D. Ark.) 22 F. Supp.2d 942, 958. “Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff’s burden in a toxic tort case.” *Allen v. Pennsylvania Eng’g*

Corp., (1996 5th Cir.) 102 F.3d 194, 199; *see also Mitchell v. Gencorp. Inc.*, (1999 10th Cir.) 165 F.3d 778, 781-82.

The Supreme Court of Texas, in *Borg-Warner Corp. v. Flores* (Tex. 2007) 232 S.W.3d 765, 773 rejected, as inconsistent with the substantial-factor test, a rule that "if there is sufficient evidence that the defendant supplied *any* of the asbestos to which the plaintiff was exposed, then the plaintiff has met the burden of proof," and also rejected the notion that exposure to some respirable fibers is sufficient to show that a product containing asbestos was a substantial factor in causing asbestosis. In *Georgia-Pacific Corp. v. Stephens*, (Tex. Ct. App. 2007) 239 S.W. 3d 304, 320-321, the Texas Court of Appeals applied the same rule to mesothelioma. ("To prove substantial-factor causation, a plaintiff must show both frequent, regular, and proximate exposure to the product *and* reasonable quantitative evidence that such exposure increased the risk of developing the asbestos-related injury. It is not adequate to simply establish that 'some' exposure occurred." *Id.* at *8.)

II. Plaintiffs Must Establish that the Dose from Defendant's Product Is a Substantial Contributing Factor to the Overall Dose.

Plaintiffs must also establish a reasonable medical probability that a particular exposure or series of exposures was a *substantial factor* in bringing about the injury, *Rutherford*, 16 Cal. 4th at 982. In *Rutherford* the California Supreme Court rejected the argument that the burden of proving causation should shift to the defendants after the plaintiffs had proven some exposure.

While Plaintiffs need not prove that fibers from the defendant product were those (or among those) that began the process of malignant cell growth, they must establish that asbestos supplied by the particular defendant was a substantial factor in contributing to the aggregate dose of asbestos the plaintiff or decedent inhaled or ingested, *id.* at 976.¹² An infinitesimal or theoretical part in bringing about injury is not sufficient, *id.* at 969.

[P]laintiffs 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. (Footnote omitted)

While it is true that Rutherford cautions that “Undue emphasis should not be placed on the term “substantial” and that the substantial factor standard was formulated to aid plaintiffs as a broader rule of causality than the “but for” test, the “undue emphasis” to be avoided is the invocation of “substantial” by “defendants whose conduct is clearly a “but for” cause of plaintiff's injury but is nevertheless urged as an insubstantial contribution to the injury. (*Rutherford*, 16 Cal.4th at 969; citation omitted). *See also Kennedy v. Southern California Edison Company*, (2000 9th Cir.) 268 F.3d 763, 768-769 [applying California

¹² The reasoning of *Rutherford* cries out for evidence as to the total exposure to all likely causes of the injury and exposure to defendant's product. Plaintiffs failed to establish either exposure.

law] (mere possibility that defendant's conduct caused injury, standing alone, is insufficient to establish a prima facie case. As the Texas Supreme Court observed in *Borg-Warner, supra, Rutherford's* requirement of proof that an exposure was a substantial factor in contributing to the aggregate dose of asbestos, together with the requirement that the aggregate exposure be more than infinitesimal, theoretical or negligible, implies *some* quantification of dose. *See also Parker v. Mobil Oil Corp.*, (2006) 7 N.Y.3d 434, *rearg. den.*, (2207) 2007 N.Y. LEXIS 3.

Moreover, any scientific analysis must take into account the different toxicity of various types of asbestos. It was partly because of the "widely divergent toxicities" of various asbestos products that *Rutherford* rejected a rule that fails to differentiate among defendants and their respective products. (*Rutherford*, 16 Cal.4th at 972-973 (quoting *Vigiolto v. Johns-Manville Corp.* (W.D. Pa. 1986) 643 F.Supp. 1454, 1463, *aff'd.* (3d Cir. 1987) 826 F.2d 1058); *see also Gideon v. Johns-Manville Sales Corp.* (5th Cir. 1985) 761 F.2d 1129, 1145 ("all asbestos-containing products cannot be lumped together in determining their dangerousness."))

A. There Is No Credible Evidence that Mrs. Mahoney's Disease Can Be Attributed to Georgia-Pacific's Joint Compound.

As noted, in rendering an opinion as to medical causation, a scientist must take into account the specific properties of the asbestos in the G-P joint compound, *Rutherford*, 16 Cal. 4th at 975, *Lineaweaver*, 31 Cal. App. 4th at 1417.

Asbestos is a term used to describe a group of minerals used worldwide for their physical properties.¹³ There is little, if any, disagreement that amphibole asbestos can, and has, caused both pleural and peritoneal mesothelioma. Many scientists incorrectly argue that establishing general causation for amphibole asbestos implies that it must also be true for chrysotile asbestos. This is false logic, and could only be *provisionally* accepted, *with a large uncertainty attached*, if there were only information showing adverse health effects in an amphibole asbestos exposed population.

¹³ Scientifically, asbestos is categorized into two mineral groups: serpentine (chrysotile) and amphibole (crocidolite, amosite, tremolite, actinolite and anthrophyllite) asbestos. Each asbestos type has a distinct chemical formula. Amphiboles occur both as asbestiform (fibrous) and non-asbestiform (massive) structures in nature, and each type (in either form) retains its elemental composition. Chrysotile is a sheet silicate that rolls into nano-sized tubular structures possessing a hollow core, whereas amphiboles are double chain silicates. Given their different properties, it is not surprising that they should behave somewhat differently. The asbestiform (fibrous) type damages the lung and related tissue while the non-asbestiform analog has been shown not to present a similar hazard. *See* Ross M., Langer A.M., Nord G.L., Nolan R.P., Lee R.J., Van Orden D., Addison, J. (2008) The Mineral Nature of Asbestos, 52 *Regulatory Toxicology and Pharmacology* S26-S30.

Asbestos products have widely divergent toxicities. *Rutherford*, 16 Cal. 4th at 979. Scientific studies show that the mesothelioma potency of chrysotile asbestos is at least 20-fold *less* than that of amphibole asbestos, *not taking* exposure differences into account. See Hodgson, J.T. and Darnton, A., (2000) “The Quantitative Risk of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure,” 44 *Annals of Occup. Hyg.* 564-601 (“Hodgson and Darnton 2000”) and Tables 1 and 3, *infra*. Although many scientists claim, on general theoretical grounds, that chrysotile can cause mesothelioma, it’s potency compared with crocidolite and amosite is at least 500-fold and 200-fold *less*, respectively. *Id.* The evidence that chrysotile can cause mesothelioma is therefore not unequivocal or even quite certain.¹⁴

In deciding whether the requirement of general causation is satisfied, the following three questions for the asbestos minerals were asked:

1. Is the Relative Risk (RR) 2 or higher, with a confidence interval that does not include 1?
2. Alternatively, is the Probability of Causation (POC) greater than 50% with a confidence interval that does not include 0%?
3. Are the studies confounded so that, if an increased risk is present, no clear statement can be made about chrysotile asbestos causing mesothelioma?

¹⁴ Some claim that it may not cause mesothelioma at all. Yarborough, C.M., (2006) “Chrysotile as a Cause of Mesothelioma: An Assessment Based on Epidemiology,” 36 *Critical Reviews in Toxicology* 165-187.

These criteria, which are very sensible from a scientific standpoint, are also now accepted by some courts. *See, e.g., Merrell Dow Pharm., Inc. v. Havner*, (Texas 1997) 953 S.W.2d 706.

Amici submit that the evidence for general causation of mesothelioma by chrysotile asbestos is marginal, despite the fact that more than 95% of commercial asbestos ever sold was chrysotile, with many people having experienced high exposures. This is shown by examining two chrysotile-exposed cohorts (Quebec miners and millers and the South Carolina textile workers) to illustrate the risk calculation and the confounding factors. These data are shown in Table 1, which is updated from a table in Hodgson and Darton 2000.

**Table 1. Data for the five chrysotile-exposed cohorts
(all the mesotheliomas are pleural).**

Fiber type	Name and Location	Mesotheliomas / all deaths (%)	Exposure f/ml x yrs	Risk Ratio (RR) Observed/ Background
	<i>Miners & Millers</i>			
Chrysotile	<i>Thetford Mines, Quebec</i>	25/4,125 (0.6%)	600	2.8 (0.6/0.22 [†])
	<i>Asbestos City Quebec</i>	8/3,331 (0.24%)	600	1.1 (0.24/0.22)
	Subtotal Canadian Mines	33/7,456 (0.44%)	600	2.0 (0.44/0.22)
	<i>Manufacturers</i>			
Chrysotile	Charleston, SC <i>Males only</i>	3/1,186 (0.25%) [§]	28	1.6 (0.25/0.16 [*])
Chrysotile	New Orleans, LA	0/259 (0%)	22	0
Chrysotile	Connecticut	0/557 (0%)	46	0
	<i>All Males Manufacturers</i>	3/2,002 (0.15%)		0.9 (0.15/0.16)
	TOTAL all studies	39/10,540 (0.37%)	170	1.5

§ Hein, M.J., Stayner, L., Lehman, E., Dement, J.M., (2007) "Follow-up study of chrysotile textile workers: cohort mortality and exposure-response," 64 *Occupational and Environmental Medicine* 616-625 ("Hein, *et al.* 2007"). The other data are from Hodgson and Darnton, 2000.

† Percentage of death due to mesothelioma in the general male population of Quebec, Canada.

* Percentage of death due to mesothelioma in the general male population of the United States (*see* Table 2).

General causation for exposure to chrysotile asbestos causing pleural mesothelioma is marginal among the Quebec miners and millers. It critically depends upon the estimate of background mesotheliomas. Adding in manufacturing workers makes the evidence weaker.

Mesothelioma most commonly occurs in two anatomical sites: in the pleura and the peritoneum. Pleural mesothelioma develops in the chest, specifically the pleural space around the lungs. This is the type of malignancy with which Mrs. Mahoney was diagnosed, and we limit our discussion to this site among males. If we were to average the data for males and females (not shown here) the already equivocal suggestion that general causation is satisfied would be further weakened.

First, the largest number of chrysotile pleural mesotheliomas, 33 cases, occurred among 7,456 deaths in the Quebec chrysotile mines (Table 1 and Hodgson & Darnton 2000). The average exposure for these miners was higher than for any known chrysotile group – 600 fiber/milliliter x years – and therefore can be expected to have the highest risk. (Table 1) Of the two major Quebec mining areas the percentage of deaths from mesothelioma is 0.6% in the Thetford area and 0.2% in the nearby mining complex in Asbestos City. The general causation argument is anchored by the higher mesothelioma mortality in the Thetford area that may be confounded by the presence of fibrous tremolite and/or other amphibole asbestos fiber-types in the chrysotile mine. Churg, A., Wiggs, B., DePaoli, L., Kampe, B., Stevens, B. (1984) “Lung Asbestos Content in Chrysotile Workers with Mesothelioma,” 130 *American Review of Respiratory Disease* 1043-1045; Dufresne, A., Harrigan, M., Masse, S, Begin, R., (1995) “Fibers in Lung Tissues of Mesothelioma Cases among

Miners and Millers of the Township of Asbestos, Quebec,” 27 *American Journal of Industrial Medicine* 581-592; McDonald, J.C. and McDonald, A.D., (1995) “Chrysotile, Tremolite and Mesothelioma,” 267 *Science*, 776-777). A critical issue is the relative risk of mesothelioma in the absence of exposure to chrysotile in the Quebec mines. These additional fibrous types are not present in chrysotile from South Africa. (Rees, D., Goodman, K., Foire, E., Chapman, R., Blignaut, C., Beachmann, M.O., Myers, J., (1999) “Asbestos Exposure and Mesothelioma in South Africa,” 89 *S. Afr. Med. J.* 637-634; Rees, D., Phillips, J.I., Garton, E., Pooley F.D., (2001) “Asbestos Lung Fibre Concentrations in South African Chrysotile Mining Worker,” 45 *Annals of Occupational Hygiene*, 473-477; Nolan, R.P., Ross, M., Nord, G.L., Raskina. M., Phillips, J.I., Murray, J., Gibbs, G.W., (2006) “Asbestos Fiber-Types and Mesothelioma Risk in the Republic of South Africa,” 12 *Clay Science* 223-227) and the Russian Federation, (Shcherbakov, S.V., Kashansky, S.V., Domnin, S.G., Nolan, R.P., (2001) “Health effects associated with mining and milling chrysotile asbestos in the Urals Region of the Russian Federation,” in “The Health Effects of Chrysotile-Asbestos: Contribution of Science to Risk Management Decisions,” 5 *Canadian Mineralogist, Special Publication* 187-198) where increased risk of mesothelioma *have not been reported* among the miners and millers in both countries.

A crucial issue is what would the mesothelioma rate be in the absence of exposure to chrysotile in the mine? Ideally there would be data available for similar people who develop mesothelioma in the time under study. There are no direct data, but we make the assumption that the rate would be the same as the general Quebec population.

Because of the importance of this assumption we present the detail of our calculation. We compare the fraction of male chrysotile miners and millers who develop pleural mesothelioma to the fraction of males in the general population in the same region, and in the same time period, who develop pleural mesothelioma. We estimate first the background pleural mesothelioma mortality in the general population of Quebec, which is the location of some large asbestos mines and which kept good medical records. We do so in three ways:

1. Health Canada reports the Standard Incidence Rates (SIR) for both Quebec and the U.S., which are 100 and 79 respectively, for males and females combined. As noted above, the number of females who develop mesothelioma is smaller, and their inclusion would only dilute the evidence for general causation, so we restrict the calculations to males. In the U.S. 0.1% of all deaths are from mesothelioma that by proportionality would be 0.126% in Quebec – mesothelioma (pleural tumors, pleural and peritoneal) is 26% more common in Quebec than in the U.S. We estimate that 0.17% of all U.S.

male deaths are from mesothelioma (Table 2), then 0.21% of all Quebec males deaths would be from mesothelioma.

2. The incidence rates of mesothelioma per 100,000 living people each year in Canada are:

<u>Site</u>	<u>Males</u>	<u>Females</u>
Pleural	1.7	0.3
Peritoneal	0.06	0.06

Quebec has a population of approximately 7,492,100 and the Canadian death rate is 720 per 100,000. In the recently period about 54,000 deaths occur each year in Quebec and we assume 27,000 are male deaths. If 64 male mesotheliomas were occurring among 27,000 deaths, the percentage of mesothelioma deaths among all male deaths in Quebec would be 0.236%.

3. Health Canada reports 1,210 male mesotheliomas cases in Quebec from 1982 to 2002 or on average about 57 cases per year if we again assume 27,000 deaths each year than 0.213% of all deaths among all males in Quebec are from mesothelioma.

Averaging the three estimates, we get 0.22% of all the deaths among males in Quebec are from mesothelioma. There are 0.44% male mesothelioma deaths among the Quebec chrysotile-mining cohorts making the Relative Risk equal to 2.0 (Table 1). Since the number of cases is 33, and the lower 90th percentile of an “expected” 33 is about 23. This would be a Risk Ratio of 1.4,

which would seem to satisfy the criterion for general causation that the confidence interval should not include one. The Probability of Causation (POC) is $0.22/0.44 \times 100$ or 50%, with a lower 90th percentile of about 30%. Thus, general causation would be marginally satisfied for this group. However, more uncertainties exist than the statistical sampling errors, and there may be confounding with other mineral fibers. Any such confounding would inevitably reduce the relative risk, and the evidence for general causation would then be inadequate.

Many scientists argue that one should compare the proportionate mortality in the high exposure cohort to a calculated background on the hypothetical assumption that the high exposures, to amphibole asbestos, in the period from 60 years ago to the recent period of controlled use, from 1971 forward, had not taken place. This is logically incorrect. They believe, and *amici* tend to agree, that the present background incidence is largely due to these past asbestos exposures. But whatever the cause of the background exposure, the *background incidence* must be subtracted from the total incidence in this cohort to arrive at the mesothelioma incidence from chrysotile alone.

Although manufacturing workers in the United States using chrysotile asbestos are less exposed than the Quebec miners and millers, we also examine the known manufacturing cohorts shown in Table 1. The first cohort of

manufacturing workers exposed to chrysotile in South Carolina has recently been updated and three mesotheliomas have now occurred among 1,841 deaths. These workers had an exposure to chrysotile about ten-fold lower than the miners and millers in Quebec. (Hein, M.J., Stayner, L., Lehman, E., Dement, J.M., (2007) "Follow-up study of chrysotile textile workers: cohort mortality and exposure-response," 64 *Occupational and Environmental Medicine* 616-25). The main thrust of Hein, *et al.* 2007 is that there is a strong exposure-response relationship between cumulative exposure to chrysotile and mortality from asbestosis and lung cancer. *Amici* do not dispute this.

Only three mesothelioma cases occurred among 2,002 male deaths in the U.S. cohort of workers in manufacturing chrysotile containing-products. The complete occupational history in these three mesothelioma cases is not available, nor are there any lung content analyses in these three cases to establish the concentration(s) and asbestos type(s) to which they were exposed. We make the assumption that in the absence of specific chrysotile exposure in the manufacturing process, the mesothelioma rate would be the same as in the general U.S. population in the same time period. Among the general male U.S. population, 0.16% will die from mesothelioma (Table 2). The Risk Ratio (RR) for mesothelioma among the males in the South Carolina cohort alone is 1.6 (too small to satisfy general causation, and if the other two manufacturing cohorts are added, as we should, the probability of causation is 0.9, which is

less than the general male population (shown in Table 1 and Table 2). The Probability of Causation (POC) becomes $-0.01/0.16 \times 100$ or -6.5% . A risk ratio (RR) less than 1 and a negative probability of causation (POC) indicates the risk is *lower* in the exposed group than in the general population. From this, it can be seen that averaging these manufacturing workers with the mining and milling workers would weaken the argument for general causation.

Table 2. The mortality in the United States in 1999 for the general population and the percentage of mesothelioma.

Asbestos Fiber-Type	Total № of Mesotheliomas/ Deaths (%)[§]	Risk Ratio (RR) Observed/Background
United States General Population	2,485 (0.1%) [†]	
<i>Males</i>	1,995 (0.16%)	
<i>Females</i>	490 (0.04%)	
All Charleston, South Carolina Chrysotile Textile Workers	3 (0.16%)	1.0 (0.16/0.16)
<i>Males</i>	3 (0.25%)	1.6 (0.25/0.16)
<i>Females</i>	0(<0.15%)	0

[§] Mesothelioma as a percentage of all deaths.

[†] National Institute for Occupational Safety and Health (NIOSH), May 2003, Work-Related Lung Disease Surveillance Report 2002, Section 7, Table 7-1, Division of Respiratory Disease Studies.

About 2,485 mesotheliomas occurred in the United States in 1999 where the disease was about 4-fold more common in males than females (National Institute for Occupational Safety and Health (NIOSH), May 2003, Work- Related Lung Disease Surveillance Report 2002, Section 7, Table 7-1,

Division of Respiratory Disease Studies. In total 2,394,871 deaths occurred in the U.S. that year with 1,217,136 in males (National Center for Health Statistics Data Warehouse, File GMWK3_1999, <http://www.cdc.gov/nchs/data/statab/>). Recently in the U.S. general population mesothelioma accounts for 1 death in 1,000 deaths in the general population and 1 in 600 and 2,000 for males and females respectively. Moreover, although the South Carolina factory used a small amount of crocidolite, one ton (0.03%), the balance was chrysotile (Hein, et al. 2007). Yet 12% of the asbestos fibers in the pulmonary tissue of the South Carolina workers was crocidolite (Pooley, F.D., Mirtha, R., 1986. "Fiber Types, Concentrations and Characteristics Found in Lung Tissues of Chrysotile-Exposed Cases and Controls," 1 *Accomplishments in Oncology* 1-10) suggesting that crocidolite was the cause of their mesotheliomas.

In contrast to the failure to find many mesotheliomas among miners and millers of chrysotile asbestos, mesotheliomas were frequently found in cohorts exposed to amphibole asbestos. Wagner, J.C., Sleggs, C.A., Marchand, P., (1960) "Diffuse Pleural Mesothelioma and Asbestos Exposure in North Western Cape Province," 17 *Brit. J. Industr. Med.* 260-271. Wagner, et al. were able readily to identify 33 mesothelioma cases (40% having only non-occupational exposure) from crocidolite exposure, while it took more than 30 years to find a similar number among the Quebec chrysotile miners and

millers, with a paucity of non-occupational cases in Quebec. Although the United States has used at least 24.5 million tons of chrysotile asbestos since 1931, the best epidemiology available indicates an excess of mesothelioma even associated with high cumulative exposure is small and may be due to confounders.

Since any effect of a pollutant will be proportional to Risk Ratio minus 1, chrysotile asbestos is at least 20 times less likely to cause mesothelioma than amphibole asbestos (Table 3). The difference is in fact greater, because the exposures in the chrysotile mines were nearly 10-fold higher than in the amphibole cohorts (Table 1).

Table 3. Mesothelioma mortality in ten epidemiologic cohort studies of individuals exposed to crocidolite, amosite, actinolite asbestos and tremolite asbestos where general causation is well established.

Fiber type	Name and Location	Mesotheliomas /all deaths (%)	Exposure f/ml x years §	Risk Ratio (RR) Observed/ Background
Crocidolite	Miners			
	<i>South Africa(SA)</i>	20/423 (4.7%)	16.4	29 (4.7/0.16)
	<i>Wittenoom, Australia</i>	72/719 (10%)	23	62 (10/0.16)
	Factory Workers			
	<i>Massachusetts</i>	5/28 (17.8%)	120	111 (17.8/0.16)
Summary		97/1170 (8.3%)	53	52 (8.3/0.16)
Amosite	Paterson, NJ			
	<i>Workers</i>	17/740 (2.3%)	65	14 (2.3/0.16)
	<i>Household Neighborhood</i>	4/115 (3.5%)	Unknown	22 (3.5/0.16)
		1/780 (0.13%)	Unknown	0.8 (0.13/0.16)
	Tyler, TX	6/222 (2.7%)		17 (2.7/0.16)
	Uxbridge, UK	5/333 (1.5%)		9 (1.5/0.16)
	South African Miners	4/648 (0.6%)	23.6	4 (0.6/0.16)
Summary		37/2838 (1.3%)	47	8 (1.3/0.16)
Tremolite-Actinolite Asbestos	Miners, Libby, MT [†]	12/286 (4.2%)		26 (4.2/0.16)
Mean for four amphibole asbestos minerals		146/4294 (3.4%)		21 (3.4/0.16)

[†] McDonald, J.C., Harris, J., Armstrong, B., (2004) "Mortality in a Cohort of Vermiculite Miners Exposed to Fibrous Amphiboles in Libby, Montana," 61 *Occup. Environ. Med.* 363-366.

Age-adjusted rates of pleural mesothelioma among men rose from about 7.5 per million person-years in 1973 to about 20 per million person-years in the early 1990s and appear to be stable or declining thereafter, driven largely by birth cohort effects. Age-adjusted rates of pleural mesothelioma among women have remained more or less constant at about 2.5 per million person-years over the period 1973-2005. Age-adjusted rates for peritoneal mesothelioma in both men (1.2 per million person-years) and women (0.8 per million person-years) exhibit no temporal trends. *See* Moolgavkar, S.H., Meza, R. and Turim, J., (2009) Pleural and Peritoneal Mesotheliomas in SEER: Age Effects and Temporal Trends, 1973–2005, *Cancer Causes Control*, DOI 10.1007/s10552-009-9328-9 (Springer Science+Business Media B.V. 2009). Convergence of recent male and female rates in older age groups, except those who are between the age of 60 and above has been observed.

It is agreed that Mrs. Mahoney had some exposure to chrysotile asbestos, but the argument that such exposure leads to increased risk of mesothelioma is unsupported. If women in the general population showed some increased risk of mesothelioma that could be asbestos-related, it would tend to support, but still not prove, Mrs. Mahoney's claim, because one could argue that she is among that excess rate group. But there are no good epidemiology studies showing Mrs. Mahoney is in an increased risk group. The only group with the possibility of such a risk is the chrysotile-exposed

miners in Quebec. They have 600f/ml-years of exposure that barely doubles the risk of mesothelioma. None of the United States manufacturing cohorts with occupational exposure to chrysotile has a risk of mesothelioma above the background in the general population. With no trends of increased mesothelioma risk in the women in the general population, more specific evidence and reasoning is needed, but has not been offered.

Plaintiffs' claim needs to be supported by evidence showing that Mrs. Mahoney had chrysotile exposure similar to others known to have increased risk of mesothelioma. She would have to show an exposure higher than in the United States manufacturing cohorts where the cumulative exposures are estimated to be 28 f/ml-years, 22 f/ml-years, and 46 f/ml-years, respectively for South Carolina, New Orleans and Connecticut, to argue that her mesothelioma is chrysotile asbestos-related.

Plaintiffs' expert, Richard Hatfield, testified that sanding for 30 minutes released from 1.4 to 6.6 chrysotile fibers per cc¹⁵; sweeping released from 2.0 to 7.0 chrysotile fibers per cc and cutting CertainTeed pipe released from 170 to 250 crocidolite and chrysotile fibers per cc. (3 RT 632-633, 636, 638.) Hatfield, estimated that Mrs. Mahoney's exposure to chrysotile asbestos fibers from G-P products was less than 1/35th of her exposure to a mixture of

¹⁵ One cubic centimeter (cc) is equivalent to one milliliter (ml), so these numbers can be compared with the data in Table 1.

amphibole and chrysotile fibers from cutting CertainTeed pipe. (3 RT 632-633, 636, 638.) On the basis of these data, the time-weighted average exposure from sanding and sweeping ranged from 0.2 - 0.82 fibers per cc. (3 RT 660-661.) Mrs. Mahoney's exposure to chrysotile asbestos from sanding and sweeping dust from G-P products was far, far less than the exposure to chrysotile shown in Table 1 to be associated with a Risk Ratio above 2.0.

None of Plaintiffs' experts claimed that Mrs. Mahoney had a cumulative exposure remotely similar to a chrysotile miner in the 20th Century and Mrs. Mahoney surely had a lower exposure than did the chrysotile-manufacturing workers and her amphibole asbestos exposure is very significant.

CONCLUSION

Amici believe the claim that exposure to chrysotile asbestos while sanding G-P's joint compound and sweeping the dust from sanding caused an increased risk of mesothelioma in Mrs. Mahoney cannot be maintained. The increased risk is so small that it must be deemed to be *de minimis*. The judgment entered below should be reversed.

Dated: Larchmont, New York
May 29, 2009

Respectfully submitted,

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BIOGRAPHICAL ADDENDUM

BRUCE N. AMES is Professor Emeritus of Biochemistry and Molecular Biology at the University of California in Berkeley, California and was chairman of that department from 1983-1989. He received his B.A. from Cornell University in 1950 and his Ph.D., with a biochemistry major, from the California Institute of Technology in 1953. He is a member of the National Academy of Sciences and he was on its Commission on Life Sciences. He was a member of the board of directors of the National Cancer Institute, the National Cancer Advisory Board, from 1976 to 1982. He was the recipient of the U.S. National Medal of Science (1998), the General Motors Cancer Research Foundation Prize (1983), the Tyler Environmental Prize (1985), the Gold Medal Award of the American Institute of Chemists (1991), the Glenn Foundation Award of the Gerontological Society of America (1992), the Lovelace Institutes Award for Excellence in Environmental Health Research (1995), the Honda Prize of the Honda Foundation, Japan (1996), the Japan Prize, (1997), the Kehoe Award, American College of Occup. and Environ. Med. (1997), the Medal of the City of Paris (1998), the Linus Pauling Institute Prize for Health Research (2001), and the American Society for Microbiology Lifetime Achievement Award (2001). He has received a number of awards, including the Charles F. Mott prize from the GM Cancer research Foundation, the Gold Medal from the American Institute of Chemists, the Society of Toxicology Public Communications Award and the Wadsworth Award, among others. His over 450 publications have resulted in his being among the few hundred most-cited scientists in all fields.

MARCIA ANGELL, M.D., F.A.C.P., is Senior Lecturer in the Department of Social Medicine at Harvard Medical School. A graduate of Boston University School of Medicine, she trained in both internal medicine and anatomic pathology and is a board certified pathologist. She joined the editorial staff of the *New England Journal of Medicine* in 1979, became Executive Editor in 1988, and was Editor-in-Chief in 1999-2000. Dr. Angell is a member of the Association of American Physicians, the Institute of Medicine of the National Academy of the Sciences, the Alpha Omega Alpha National Honor Medical Society, and is a Master of the American College of Physicians. In 1997, *Time* magazine named Marcia Angell one of the 25 most influential Americans. Dr. Angell writes and speaks frequently in professional journals and the popular media on a wide range of topics, particularly medical ethics, health policy, the nature of medical evidence, the interface of medicine and the law, and care at the end of life. Dr. Angell is co-author of the first three editions of the textbook, *BASIC PATHOLOGY*. Her book, *Science on Trial: The Clash of Medical Evidence and the Law in the Breast Implant Case* (1996)

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JOHN DUFFUS, Ph.D., D.Sc., F.R.S.C., is the Director of the Edinburgh Centre for Toxicology, an independent consultancy. He is also the Chair of the International Union of Pure and Applied Chemistry (IUPAC) Division VII, Chemistry and Human Health, Subcommittee on Toxicology and Risk Assessment and adjunct Professor of Toxicology at the Asian Institute of Technology. He is a graduate of the University of Edinburgh and a member of the Royal Society of Chemistry Environment, Health and Safety Committee. He is the author of nearly 200 substantive publications, including books, and many shorter communications on toxicology and related subjects. He has a specialist interest in the role of inorganic substances in toxicology, especially in relation to fundamental processes related to carcinogenicity, and has significant publications in this area. He has acted as a consultant to the United Nations Environmental Programme (UNEP), The International Programme on Chemical Safety (IPCS), the World Health Organization (WHO), and the European Commission (EC) on toxicology and carcinogenesis. He has recently contributed to Environmental Health Criteria Document 234, “Elemental Speciation in Human Health Risk Assessment.”

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RONALD E. GOTS, M.D., Ph.D. specializes in toxicology and environmental medicine. He is Principal of the International Center for Toxicology and Medicine and Medical Director and President of the National Medical Advisory Service. He is also Lecturer in and Adjunct Professor of Pharmacology, Department of Pharmacology, Georgetown University School of Medicine. He has been Coordinator, Pharmaceutical Class Labeling Project, of the U.S. Food and Drug Administration, Medical Director and Examining Physician of the Occupational Health Units, Bureau of Economic Analysis, Census Bureau and Immigration and Naturalization Service, Senior Investigator/Chief, Department of Gastroenterology, Walter Reed Army Institute of Research. He was Conference Chair of a conference on "Multiple Chemical Sensitivities: State-of-the-Science Symposium" co-sponsored by the International Society of Regulatory Toxicology and Pharmacology, The Johns Hopkins University/National Institute for Occupational Safety and Health Educational Resource Center in the Occupational Safety & Health and National Medical Advisory Service.

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RONALD HART, Ph.D., is Director Emeritus of the National Center for Toxicological Research; Distinguished Scientist in Residence, United States Food and Drug Administration (retired). He is currently adjunct professor of Cancer Prevention at the Strang Cancer Research Institute of Rockefeller University. Dr. Hart developed the first direct proof that DNA damage was causal in cancer causation (1974), established much of the modern basis for the role of food intake on aging, degenerative disease occurrence and chaired, among many others, the White House Consensus Policy on Chemical Carcinogenesis.

STEVEN H. LAMM, M.D., D.T.P.H. is a medical doctor; he also holds a diploma in tropical public health. He is board certified in pediatrics, in occupational medicine and preventive medicine. He is a charter fellow of the American College of Epidemiology, and a winner of the Annual Prize of the Society for Epidemiologic Research. Dr. Lamm also holds a Master of Science degree in biophysics. He is President of Consultants in Epidemiology

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Dated: Larchmont, New York
June 4, 2009

Martin S. Kaufman
Counsel for *amici curiae*

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[*Mahoney v. Georgia-Pacific LLC*, Court of Appeal No. A122038]

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IN SUPPORT OF DEFENDANT-APPELLANT

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