August 15, 2013

Chief Justice Tani Cantil-Sakauye
and Associate Justices
California Supreme Court
350 McAllister Street
San Francisco, CA 94102-4797

Re: *Strickland v. Union Carbide Corporation, et al.*,
Case No. S212424

Dear Chief Justice Cantil-Sakauye and Associate Justices:

Professor Richard Wilson, Dr. Ronald Gots, Professor Arthur M. Langer, Professor Robert P. Nolan, Professor Emanuel Rubin, and Nobel Laureate James Watson ("*amici*") respectfully urge the Court to grant the petition for review in *Strickland v. Union Carbide Corporation, et al.*, Case No. S212424.

Interest of *Amici*

Amici are scientists who have studied 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, chemists, geologists, physicists, epidemiologists and toxicologists. *Amici* are aware of the significance of asbestos litigation nationally and in California, and they are concerned that the mere utterance of "asbestos, "together with "mesothelioma" or "cancer" can have undue impact on juries no matter the asbestos fiber-type, or the level of exposure.*

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* No counsel for any party authored this *amicus* letter in whole or in part, and no counsel or party made a monetary contribution intended to fund the preparation or submission of this letter. No person other than *amici* or their counsel made a monetary contribution to the preparation or submission of this brief.

Amici believe that the decision of the Court of Appeal's decision is incorrect because it does not reflect the consensus of the relevant scientific community regarding causation of peritoneal mesothelioma, and because the opinion of Dr. Samuel Hammar, Plaintiffs' expert on medical causation, is not based on a scientific methodology accepted by the relevant scientific community.

RICHARD WILSON, D.Phil. is Mallinckrodt Research Professor of Physics at Harvard University and the immediate past Director of the Regional Center for Global Environmental Change at Harvard University. Professor Wilson was Chairman of the Department of Physics at Harvard University and a past chairman and currently a member of the Cyclotron Operating Committee at Massachusetts General Hospital, and a member of the visiting committee of the Radiation Medicine Department at Massachusetts General Hospital. He is a founder of the Society for Risk Analysis. He is and has been a consultant to the United States government and the governments of numerous foreign countries on matters of toxicology, epidemiology, public health and safety, nuclear safety, and risk assessment. He is the author of many articles on high energy physics, environmental pollution and risk analysis, including PARTICLES IN OUR AIR, EXPOSURES AND HEALTH EFFECTS (with John Daniel Spengler) (Harvard University Center for Risk Analysis, 1986) and RISK-BENEFIT ANALYSIS (2nd ed., 2001) (with Edmund A. C. Crouch) (Harvard University Center for Risk Analysis). Professor Wilson is the author or co-author of more than 900 published papers on subjects including atomic particles, radioactive particle decay, acute toxicity and carcinogenic risk, carcinogenicity bioassays, statistical distributions of health risks, public health, cancer risk management, shielding of particle accelerators and nuclear reactors, nuclear energy production, health risks of nuclear power plant accidents, health effects of electromagnetic fields, risks and health impacts of radiation, risks of nuclear proliferation, risk-benefit analysis, global energy use and global warming.

RONALD E. GOTS, M.D.,Ph.D., DABT, is CEO of the International Center for Toxicology and Medicine. He is a physician and board certified toxicologist, specializing in toxicology and environmental medicine. He is a member of the Society of Toxicology and the American College of Occupational and Environmental Medicine. Dr. Gots is Adjunct Professor of Pharmacology at Georgetown University School of Medicine. He has been Coordinator of the 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 in the Department of Gastroenterology, Walter Reed Army Institute of Research. Dr. Gots has focused on the scientific methods for assessing causation of diseases allegedly associated with chemical and biological agents, to the causal analysis of chemically-induced illnesses, and to workplace exposures, worker protection

and environmental risk communication. He has provided medical oversight for chemically-exposed individuals. Dr. Gots has chaired two international symposia on "Multiple Chemical Sensitivities: The State of The Science." He was a member of a United Nations committee convened by the International Programme on Chemical Safety (UNEP-ILO-WHO) to evaluate chemical sensitivity. Dr. Gots is the author of six books, and has written chapters in six additional books and has published more than 70 articles on biochemistry and toxicology. Recent book chapters include "Toxic Risks: Science, Regulation, and Perception" and "Risk Analysis and Communication" in *Occupational, Industrial, and Environmental Toxicology,* and "Applying Principles of Science to Daubert Motions in Toxic Tort Claims" in *2000 Wiley Expert Witness Update.*

ARTHUR M. LANGER, Ph.D., is a Professor in the Ph.D. Program in Earth and Environmental Sciences at the Graduate School and University Center of the City University of New York, Director of the Center for Applied Studies of the Environment, Applied Sciences Coordinating Institute, City University of New York, and Research Associate in the Department of Earth and Planetary Sciences of the American Museum of Natural History, New York. He previously was Professor and Director of the Environmental Sciences Laboratory of the Institute of Applied Sciences, Brooklyn College of the City University of New York; Associate Professor at the Center for Polypeptide and Membrane Research, Mount Sinai School of Medicine, New York; Associate Professor of Mineralogy, Department of Community Medicine, Mount Sinai School of Medicine, New York; Science Administrator, Environmental Sciences Laboratory, Mount Sinai School of Medicine; Associate Director of the Environmental Sciences Laboratory, an NIEHS Center, Center for the Study of Biological Effects of Environmental Agents, Mount Sinai School of Medicine; Director of Laboratories, Environmental Sciences Laboratory, Mount Sinai School of Medicine; and Head of the Physical Sciences Section, Environmental Sciences Laboratory, Mount Sinai School of Medicine. He received his doctorate in Geology (Mineralogy) from Columbia University. Dr. Langer has studied the biological activity of chrysotile asbestos in brake pads and linings, and he is the author of *Reduction of the Biological Potential of Chrysotile Asbestos Arising from Conditions of Service on Brake Pads*, 38 Reg. Tox. & Pharm. 71 (2003).

ROBERT P. NOLAN, Ph.D. received a doctorate in chemistry from The City University of New York in 1986. He was been awarded fellowships from the Stony-Wold Herbert Fund, National Research Council, Fulbright and the International Union for Pure and Applied Chemistry. He is the Deputy Director of the Center for Applied Studies of the Environment and a member of the doctoral faculty in Chemistry and Earth and Environmental Sciences at The Graduate School and University Center of The City University of New York. He is the author of more than fifty scientific

papers and is internationally recognized as an expert in the characterization and health hazard evaluation of asbestos and other minerals.

EMANUEL RUBIN, M.D. is Distinguished Professor of Pathology, Anatomy and Cell Biology at Jefferson Medical College in Philadelphia , PA and Chairman Emeritus of the department. He has also served as Chairman of the Department of Pathology at the Mount Sinai School of Medicine and at Drexel University Medical School. He also was Adjunct Professor of Biochemistry and Biophysics at the University of Pennsylvania School of Medicine for 10 years. Dr. Rubin is the author of some 300 papers in the medical and scientific literature and has been continuously funded by NIH for over 45 years, during which time he has served as Principal Investigator on more than $100,000,000 in research grants. As editor-in-chief he founded one of the major textbooks in the field of pathology, RUBIN'S PATHOLOGY, now in its sixth edition. Dr. Rubin is a highly cited expert in Environmental Pathology, and has authored numerous book chapters in that field and in Pulmonary Pathology. He is recognized as an authority on the effects of asbestos on the human body.

JAMES D. WATSON is a Nobel Laureate in Medicine (1962) (with F.H.C. Crick and M.H.F. Wilkins), and co-discoverer of the structure of DNA. Dr. Watson has also been awarded the John Collins Warren Prize of the Massachusetts General Hospital, the Albert Lasker Prize of the Public Health Association, the John J. Carty Gold Medal of the National Academy of Sciences, and the Presidential Medal of Freedom. He was director and president of the Cold Spring Harbor Laboratory of the National Institutes of Health, and is currently Chancellor *emeritus* of the Cold Spring Harbor Laboratory. He earned his Ph.D. in Zoology, and has been awarded numerous honorary degrees.

The Underlying Case

The decedent, plaintiffs' husband and father, worked in construction for approximately 45 years with wall board and "joint compound" (a paste that is applied to the joints where two sections of wall board meet to create a smooth surface). Joint compounds contained asbestos. Union Carbide's joint compound product, Calidria, contained only the chrysotile form of asbestos. In his work, Mr. Strickland was also exposed to many asbestos-containing products from other manufacturers, including some which contained the amphibole form of asbestos. Mr. Strickland died of peritoneal mesothelioma.

At issue is whether plaintiffs' expert's testimony that Strickland's exposure to the chrysotile asbestos that Union Carbide mined and marketed under the name "Calidria" in the 1960s and 1970s (which

contain no detectable tremolite asbestos) was a "substantial cause" of Mr. Strickland's peritoneal mesothelioma was probative.

Plaintiffs produced no evidence to show how frequently Mr. Strickland was exposed to Union Carbide's chrysotile product. Plaintiffs' industrial hygiene expert estimated the momentary levels of a person's exposure to asbestos fibers during use of the various products that Strickland encountered, including joint compounds, but he did not attempt to characterize Strickland's overall exposures to chrysotile asbestos generally, or to Union Carbide's product specifically. A biopsy of Strickland's lung tissues after he died did show, however, that he had been exposed to very high concentrations of amphibole asbestos fibers.[1]

Plaintiffs' expert on medical causation was Dr. Samuel Hammar, who testified that in his opinion Strickland's exposure to Union Carbide's product was a "substantial" cause of Strickland's mesothelioma.

It is generally accepted that chrysotile asbestos is far less potent as a carcinogen than amphibole asbestos. It is also generally accepted that both amphibole asbestos and chrysotile asbestos can cause pleural mesothelioma, a cancer of the lining of the lung. There is consensus that amphibole asbestos can cause peritoneal mesothelioma, a cancer of the lining of the abdomen, but most experts do not believe that chrysotile asbestos has been shown to cause peritoneal mesothelioma.

At trial the jury found for plaintiffs, and a judgment was entered against Union Carbide.

The Court of Appeal Decision

Union Carbide appealed, arguing there was inadequate proof of medical causation because Dr. Hammar never testified that exposure to Calidria in and of itself contributed substantially to Strickland's risk of developing peritoneal mesothelioma. (Typed opn. 10).

The Court of Appeal affirmed. While the Court of Appeal accepted Union Carbide's assertion that Dr. Hammar "did not specifically link Strickland's exposure to Calidria to an increased risk of developing [peritoneal mesothelioma], opining instead as to the cumulative impact of his exposure to asbestos." (Typed opn. 12), the court held that "There need not be testimony specifically linking the defendant's product in isolation to the plaintiffs increased risk of developing cancer." (Typed opn. 10), citing the recent decision in *Hernandez v. Amcord, Inc.*, (2013) 215 Cal.App.4th 659. The Court

[1] As is common in asbestos cases, plaintiff sued many companies that made products containing various types of asbestos. By the time of trial Union Carbide was the only defendant.

of Appeal also said that there was a jury question on "substantial factor" causation under *Rutherford v. Owens-Illinois, Inc.* (1997) 16 Ca1.4th 953, 975-979, 982 ("*Rutherford"*) because plaintiffs presented expert testimony that (1) there is a reasonable medical probability the defendant's product *can cause* the type of cancer at issue and (2) the decedent's cumulative exposure to asbestos contributed to his or her disease. (Typed opn. 10). The court concluded that Dr. Hammar's testimony concerning the "cumulative impact" of Strickland's exposures to multiple asbestos-containing products from numerous suppliers was sufficient to permit the jury to find causation under *Rutherford* when the testimony was "coupled with [his] unequivocal opinion [that] chrysotile causes peritoneal mesothelioma." (Typed opn. 12-13) and it was "then up to the jury to determine . . . to what extent this defendant's product, rather than the other asbestos-containing products to which the plaintiff (or decedent) was exposed, was a factor contributing to the disease." (Typed opn. 10). It dismissed Union Carbide's appeal as "in essence, simply challeng[ing] the proper weight to be given this scientific evidence." (Typed opn. 13).

Amici believe that the Court of Appeal confused "weight of evidence" with admissibility of expert testimony that was not based on appropriate scientific methodology.

Mr. Strickland's Exposure to Asbestos

Mr. Strickland began his 45-year career in construction in the early 1960s. (10 RT 1232). He started as a latherer, installing metal studs and lathing (10 RT 1234), which support plaster, but he did not apply the plaster himself. (11 RT 1987-1988). When the construction industry drywall replaced lathe and plaster, Strickland began installing drywall, which included "applying and sanding 'joint compound' to mask the seams between the boards." (*See* 11 RT 1957-1966). It was common for Strickland to work on job sites where other tradespeople used products that often produced dust. (11 RT 1953-1954).

Strickland's exposure to chrysotile asbestos

Some of the drywall joint compounds that Strickland encountered on the job had chrysotile asbestos as an ingredient. (Typed opn. 2) At issue here is Strickland's exposure to the pure form of chrysotile asbestos (*i.e.*, not contaminated with amphiboles) that Union Carbide mined and marketed in the 1960's and 1970's under the name "Calidria."(Typed opn. 2; 12 RT 2166-2167). However, plaintiffs produced no evidence to show how frequently Mr. Strickland was exposed to Calidria, or even to other chrysotile asbestos products. (10 RT 1240; 11 RT 1840, 1959-1966, 1970-1971; 13 RT 2586-2590, 2605-2610).

While plaintiffs' industrial hygienist expert witness, John Templin, estimated the momentary levels of an individual's exposure to asbestos fibers of the types in the various products that Strickland was exposed to, including joint compounds, he did not attempt to calculate or characterize Strickland's overall exposures to the chrysotile form of asbestos generally, or to Union Carbide's product specifically. (10 RT 1277; 12 RT 2127-2140, 2155, 2159-2160, 2164-2165, 2168-2174, 2202-2205, 2215; 16 RT 3465-3467).

Strickland's exposure to amphibole asbestos

Dr. Hammar testified that the large numbers of amphibole asbestos bodies found in Mr. Strickland's lung suggest that he "probably had very high exposure during his life to one or more amphibole types of asbestos." (13 RT 2466) and that Strickland could have been exposed to amphibole asbestos on many occasions.

Mr. Strickland's father worked in a naval shipyard (11 RT 1971-72), where workers were frequently exposed to amphibole asbestos. (12 RT 2219). These workers often took home asbestos-containing dust and exposed their family members (like Strickland) to "second-hand" asbestos. (12 RT 2161-2162).

During his career in construction, Mr. Strickland worked in the vicinity of amphibole asbestos many times. He worked alongside insulators and fireproofers (10 RT 1255-1268), who used materials that often contained amphibole asbestos. (13 RT 3166-3168). When Strickland worked with drywall, he frequently had to chip away at the fireproofing, releasing an amphibole asbestos-containing dust that he could inhale. (11 RT 1978-1979). Mr. Strickland was also exposed to clouds of dust that were created when other workers used pipe cement which "could have contained the amphibole "crocidolite" or "amosite" asbestos fibers." (11 RT 1953).

Most significant, Strickland was exposed to "Limpet," an amphibole asbestos product used in the construction of the Cinerama Dome movie theater in 1962. (10 RT 1269-1270). Plaintiff's industrial hygiene expert testified that Strickland's work on the Cinerama Dome was "one of the worse [*sic*], if not the worst, exposure situation of Mr. Strickland's career." (12 RT 2205).

Significantly, a post-mortem biopsy of Strickland's lung tissues showed that he had most likely been exposed to very high concentrations of amphibole asbestos fibers. (Typed opn. 3).

Plaintiffs' causation evidence.

It is accepted that exposure to amphibole asbestos through inhalation is a potent cause of peritoneal mesothelioma. (13 RT 2422, 2459-2460). Dr. Samuel Hammar testified that even a single day's exposure to amphiboles could be enough to cause the disease decades later. (13 RT 2460, 2463).

Dr. Hammar agreed with plaintiffs' attorney that Mr. Strickland was exposed to asbestos "in the course of doing . . . drywall work and other work that was related to the construction industry" and that at "one of the types" to which Mr. Strickland was exposed was chrysotile. (13 RT 2414). Counsel asked Dr. Hammar "whether those exposures that Mr. Strickland suffered as a result of doing drywall work *and other work* where he was exposed to asbestos . . . were a substantial factor that ultimately contributed to his risk of getting mesothelioma." (13 RT 2414-2415, emphasis added).[2]

Dr. Hammar testified that he believes that chrysotile can cause peritoneal mesothelioma, (11 RT 2029-2030, 2044; 13 RT 2414) in part because he has detected the presence of chrysotile fibers in abdominal tissues he has examined (11 RT 2035-2038, 2041-2042, 2051; 13 RT 2413-2416, 2421-2422) – but NOT in Mr. Strickland. He saw no reason why such fibers would cause mesothelioma in the pleura but not in the peritoneum. (13 RT 2415-2416, 2420-2421).[3]

Dr. Hammar said to a reasonable medical certainty that "they were" a substantial factor. (13 RT 2415; *see also* typed opn. 12).[4, 5]

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[2] To put this testimony in context, plaintiffs' attorney had previously asked Dr. Hammar to assume that counsel's use of the word "asbestos" in his questions meant all types of asbestos, including amphiboles. (11 RT 2029, 2044).

[3] Until the late 1980's, Dr. Hammar believed that chrysotile does *not* cause peritoneal mesothelioma. (13 RT 2420, 2439-2440) and he conceded at trial there is "relatively little evidence" and "relatively little proof" that it does. (13 RT 2442). He also agreed that other respected experts continue to believe that no link has been shown between exposure to chrysotile asbestos and peritoneal mesothelioma. (13 RT 2441-2445; *see also* 13 RT 2554, 2564-2566) and that the absence of a link remains the "predominant position" in current medical literature. (13 RT 2444-2445).

[4] In context, "they" refers to the totality of Mr. Strickland's exposure to all types of asbestos. Dr. Hammar addressed the significance of Mr. Strickland's overall asbestos exposures during his working career to the risk that he would develop peritoneal mesothelioma, but it appears that Dr. Hammar did not opine whether Strickland's exposure to any particular type of asbestos, including

(continued...)

In essence, Hammar's reasoning was as follows:

– Hammar found chrysotile fibers in abdominal cavities of some patients who died from peritoneal mesothelioma, but not Mr. Stirckland's. He did not examine Strickland's peritoneal tissue because he was not asked to do so.

– Chrysotile asbestos can cause pleural mesothelioma.

– The tissue in the peritoneum is similar to pleural tissue.[6]

– There is no reason why such chrysotile fibers should cause mesothelioma in the lungs and not in the peritoneum.[7]

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[4](...continued)
chrysotile, was a substantial factor contributing to the risk of developing peritoneal mesothelioma.

[5] Plaintiffs' other expert, industrial hygienist John Templin, did not testify about the medical cause of Strickland's mesothelioma. (12 RT 2156.) Templin testified only that because asbestos is a carcinogen, each exposure results in an increased "body burden of the material," and thereby increases the risk of developing an illness. (10 RT 1277; 12 RT 2144-2145; see also 15 RT 3204). He did not say, however, that the increased risk of an illness from each exposure was *substantial*, and he did not say that the increased risk of developing peritoneal mesothelioma was substantial. Templin agreed he could not tell from the scientific literature at what level of exposure to chrysotile one would see an increased risk of developing peritoneal mesothelioma. (12 RT 2163-2164).

[6]
A: But there's no inherent reason why you would think that if chrysotile can cause pleural mesothelioma, and we know that it gets into the abdominal cavity, why it would also not cause peritoneal mesothelioma. You're talking about the same cells. You're talking about the same type of carcinogens. You're talking about the same mechanisms by which asbestos causes cancer.
(13 RT 2421).

[7]
Q: When you tell us your opinion that chrysotile causes peritoneal mesothelioma, specifically, what do you base that on?
A: It's based on the evidence that chrysotile, as a carcinogen, can cause cancer including mesothelioma, and it's based on the fact that I think that chrysotile can reach the target organ, which in the case of peritoneal mesothelioma is the place where the peritoneal mesotheliomas occur. And I think that Dodson and I have shown that chrysotile can reach that location, and if it can reach that
(continued...)

– Ergo, it is likely that chrysotile fibers *can* cause peritoneal mesothelioma.

Dr. Hammar's conclusion that chrysotile can cause peritoneal mesothelioma has a number of defects:

– It seems to be nothing more than a "hunch" based on a negative inference (if chrysotile can cause pleural mesothelioma, there is no reason it can't cause peritoneal mesothelioma) not supported by, and indeed refuted by, epidemiological studies.

– It is based on case reports only.[8] While case reports may lead to enough evidence to warrant a more definitive epidemiological study (as Dr. Hammar admitted), they are not reliable proof of causation. Among other reasons, there is no control to be certain that the patients in the case studies were not also exposed to amphibole.[9] Hammar relied on several case

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[7](...continued)
location and is a carcinogen and is a carcinogen that can cause mesothelioma, there's no reason that I would say that it can cause pleural mesothelioma and not peritoneal mesothelioma. (13 RT 2415-16).

[8]
Q: The articles that you rely on in there, are any of those epidemiology studies?
A: No.

Q: What kind of studies do you have that you rely on for this concept of chrysotile causing peritoneal mesothelioma?
A: Case records or case control type studies where you have an individual or maybe several individuals who have a disease like peritoneal mesothelioma and that they were exposed to asbestos.

Q: Okay. Case reports. So we have a doctor gets a patient who comes in and has peritoneal mesothelioma ... is that kind of how that works?
A: Yes. Yes. Exactly. Say a patient comes in and has peritoneal mesothelioma and the evidence would show that the only type of asbestos that he or she was exposed to was chrysotile asbestos and because it's relatively uncommon, they might write that up.

13 RT 2417-2418.

[9]
Q: Right. I'm just saying rather than going through all the case reports one by one, you would agree with me that if we look at them, you're going to see evidence in most cases that there was also
(continued...)

studies to argue that chrysotile asbestos can cause peritoneal mesothelioma, as well as a number of articles in the medical literature that support this hypothesis.

Dr. Hammar admitted that there are no epidemiological studies that show chrysotile-peritoneal mesothelioma causation (13 RT 2445-2447) and that there is "relatively little proof that chrysotile causes peritoneal mesothelioma." In fact, he cited only one recent case report in which the patient was supposedly exposed only to chrysotile asbestos, and Hammar

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[9](...continued)
amphibole exposure?
A: Right, that's always going to be the study even like in the Yenarco (phonetic) study, is that you're always going to have that problem. I don't think it's possible to really get away from it.

13 RT 2458-2459.

characterized it as a "Black Swan case" because it is "incredibly rare."[10] As noted above, Hammar also admitted that he did not examine Strickland's peritoneal tissue.

– It is of particular note that Dr. Hammar bases his conjecture on a single case reported at a conference in 2010, and one which he isn't certain even supports his theory, because, as he testified, "Now, I don't know if that's [the only fiber type involved was chrysotile] true or not." (13 RT 2451). He seems to concede that epidemiological studies are needed to confirm a theory of causation based on case studies when he testified that the case report "[is] going to lead potentially to an epidemiology study." To the best of that knowledge, no such study has been published.

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[10]

Q: But what they didn't find in that study were any primary peritoneal mesotheliomas among any of the workers they studied?
A: Not until about, what, a couple of months ago when David Egilman recorded a case of peritoneal mesothelioma in a person who worked as a worker for Carey Canada where the only type of asbestos they claimed was there was chrysotile only. Now, I don't know if that's true or not.

Q: Let's talk about -- that's sort of the Black Swan case?
A: That was the Black Swan case, yes.

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A: It was David Egilman actually presented that case in San Francisco on September 15th, 2010.

Q: And why he called it a Black Swan case was the idea that "Wow, this is so incredibly rare," right?
A: Yes, uh-huh, that's correct.

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Q: Again, that's a case report, not an epidemiological study?
A: **It is, but it's going to lead potentially to an epidemiology study**, because if you look at what David Egilman said at the San Francisco meeting where he presented that, is that he's actually advertising in papers to see if there are other people like that Black Swan case.

13 RT 2451-2454 (emphasis added).

– To the extent that Hammar's hypothesis has been tested by epidemiological research, it has not been confirmed and has largely been refuted.

– Hammar admits that his hypothesis is not "generally accepted."

– Even under the more "liberal" *Daubert* tests (*Daubert v. Merrell Dow Pharmaceuticals*, 509 U.S. 579 (1993), Dr. Hammar's testimony is deficient because (1) his hypothesis has not been tested; (2) it has not, as far as the record shows, been published in peer reviewed journals, (3) it seems to have been formulated for purposes of litigation, (4) it is not generally accepted, (5) there has been no attempt to calculate an error rate.

In short, Dr. Hammar's opinion is nothing more than conjecture based on a single unconfirmed case report, the bona fides of which Dr. Hammar was unsure.

The Flaws in Dr. Hammar's Methodology

Dr. Hammar ignored the generally accepted distinction between general causation and specific causation. His methodology does not even establish general causation of peritoneal mesothelioma for chrysotile asbestos, and the scientific consensus is that general causation of peritoneal mesothelioma for chrysotile asbestos is very doubtful.

Dr. Hammar ignored the large body of toxicological studies by official government studies and disinterested investigators that shows that chrysotile asbestos has a very small potential for causing mesothelioma.

Dr. Hammar eschewed the need to consider the dose or level of exposure of the individual patient. Determining the minimum threshold of fiber levels is critical to any consideration of medical causation.

Dr. Hammar appears to have ignored or minimized the much greater risk to Mr. Strickland of developing peritoneal mesothelioma posed by his significant exposure to amphibole asbestos.

In sum, neither Dr. Hammar's methods nor his conclusions satisfy either the scientific or the legal criteria for "general acceptance."

Courts should not accept an unproven and untestable theory as evidence.

General Causation and Specific Causation

General causation addresses the question of whether exposure to the agent of concern has ever caused the disease in question. This is usually discussed by showing that a group of people with high

levels of exposure have developed the adverse outcome, significantly more frequently than among a similar unexposed group. If general causation cannot be proven, then it is superfluous to ask the specific causation question. If the general causation is established than specific causation can be addressed for the exposure history specific to the case.

If the response is affirmative, one goes on to the causal question specific to the individual. Specific causation asks whether a particular individual developed his or her disease as a result of his or her specific exposure to the agent. It is obvious that this requires knowledge of the individual's exposure level to the suspected causal agent. Dr. Hammar acknowledged that he had no evidence that chrysotile fibers had found their way to Mr. Strickland's peritoneum. This implies a complete rejection by Dr. Hammar of the generally accepted distinction between general causation and specific causation. In essence, he testified that because "asbestos" can cause "mesothelioma," anyone with any exposure to any type of asbestos must have developed his or her mesothelioma as a result of that exposure.

Asbestos Type is Crucial

It has long been known, and has become generally accepted, that we must distinguish between six different minerals called "asbestos." These different types of asbestiform fibers behave very differently. Two major groupings are important, those which form "amphibole asbestos" (actinolite, amosite, anthophyllite, crocidolite, and tremolite) and "serpentine asbestos" (chrysotile). The types of asbestos can be easily distinguished using analytical transmission electron microscopy.

Chrysotile asbestos has historically been the dominant type of asbestos used commercially and is the only type of asbestos still in commerce in the United States. The asbestos in Union Carbide's product was pure chrysotile asbestos.

The differences in carcinogenic potency for mesothelioma causation between the major commercial asbestos types has been known and generally accepted since at least 1965. Among workers with high exposure to chrysotile, small numbers, or even zero, mesothelioma cases were generally reported. J. C. Gilson, Wyers Memorial Lecture 1965, *Health Hazards of Asbestos, Recent Studies on its Biological Effects*, 16 Trans. Soc. Occup. Med. 62 (2006). Hodgson and Darnton estimated the mesothelioma causing potency of chrysotile, amosite, and crocidolite as 1:100:500, meaning amosite and crocidolite are, respectively, 100 and 500 times more potent in causing mesothelioma than chrysotile. J. T. Hodgson and A. Darnton, *The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos Exposure*, 44 Ann. Occup. Hyg. 565 (2000) ("Hodgson and Darnton 2000"). In a later paper, Hodgson, *et al.* showed that amphibole asbestos types (amosite and crocidolite) explain the mesothelioma distribution and that chrysotile has zero incidence, indicating

that chrysotile is unlikely to be responsible for any of the mesothelioma cases in the cohort they studied. J. T. Hodgson, D. M. McElvenny, A. J. Darnton, M. J. Price and J. Peto, *The Expected Burden of Mesothelioma Mortality in Great Britain from 2002 to 2050*. 92 Brit. J. Cancer 587, at 590, Fig 5A (2005).

The United States Environmental Protection Agency commissioned a study by Berman and Crump which reached a similar conclusion. D. W. Berman, K. S. Crump, *Update of Potency Factors for Asbestos-Related Lung Cancer and Mesothelioma*, 38 Crit. Rev. Tox. (supp 1) 1 (2008) and D. W. Berman and K. S. Crump, *A Meta-Analysis of Asbestos-Related Cancer Risk That Addresses Fiber Size and Mineral type*. 38 Crit. Rev. Tox. (supp 1) 49 (2008).

These authors would seem to be free of the selection bias of experts who frequently appear as expert witnesses for one side or another in litigation, and they did not use the methodology chosen by Dr. Hammar – *i.e.* that any asbestos exposure is important – but rather considered both asbestos type and cumulative exposure in establishing causation for mesothelioma and lung cancer.[11]

Subsequent to the Hodgson and Darnton and the Berman and Crump meta-analyses of the epidemiological literature, Health Canada, an agency of the Canadian government, convened a Chrysotile Asbestos Expert Panel to develop a consensus statement and summary on the risks of lung cancer and of mesothelioma with asbestos exposure. The panel issued its report, *Chrysotile Asbestos Expert Panel*, *2008 Chrysotile Asbestos Consensus Statement and Summary* (available on request from panel@hc-sc.gc.ca). Most of the Health Canada panel members held that the relative carcinogenic potency of amphibole asbestos potency for mesothelioma was approximately 500-fold that of chrysotile, with a 95% confidence range of 20 to 1000. Chrysotile Asbestos Expert Panel, *2008 Chrysotile Asbestos Consensus Statement and Summary* at 14. As the risk was proportional to the cumulative exposure, the likelihood that any individual's mesothelioma was attributable to their asbestos exposure was also related to the magnitude and nature of their exposure.

Other scientists have recently concluded that chrysotile exposure does not cause mesothelioma to any appreciable extent. *See, e.g.*, C. Yarborough, *Chrysotile as a Cause of Mesothelioma: an*

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[11] It is illogical for courts to accept as "generally accepted" a methodology which acknowledged expert investigators did not utilize and would not accept. Peer-reviewed, published, scientific literature not prepared for purposes of litigation is an accepted source of evidence. *See Daubert v. Merrell Dow Pharmaceuticals*, 509 U.S. 579 (1993); *see also* J. Ziman, *Reliable Knowledge: An Exploration of the Grounds for Belief in Science* 130 (1978); A. S. Relman and M. Angell, *How Good Is Peer Review?*, 321 New Eng. J. Med. 827 (1989).

Assessment based on Epidemiology, 36 Crit. Rev. Tox. 165 (2006) and J. S. Pierce, M. A. McKinley, D. J. Paustenbach, B. L. Finley, *An Evaluation of Reported No-Effect Chrysotile Asbestos Exposures for Lung Cancer and Mesothelioma*, 38 Crit. Rev. Tox. 191 (2008). Yarborough found that "The review of 71 asbestos cohorts exposed to free asbestos fibers does not support the hypothesis that chrysotile, uncontaminated by fibrous amphiboles, causes mesothelioma." Pierce, *et al.*, *supra*, summarized the cumulative exposure-response data for predominantly chrysotile-exposed cohorts in the published literature and found that the predominance of the cumulative "no-effects" exposure levels for mesothelioma fall in the range of approximately 15-500 fiber per milliliter x years. These studies would seem to be free of the selection bias that affects reports of expert witnesses for a party to litigation.

Recent research, of great importance for determining what occupations are at risk, has focused mainly on two questions: (1) whether long asbestos fibers, generally greater than 5 micrometers (μm), are principally responsible for asbestos disease, and (2) whether chrysotile asbestos (which breaks down easily into short fibrils – short slender fibers) is less potent than other forms. An EPA panel, after reviewing the extensive literature, concluded, by consensus, that chrysotile asbestos fibers are far less likely to cause disease than amphiboles, by a factor of at least two orders of magnitude. U.S. EPA, *Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk,* viii (May 30, 2003), *available at* http://www.epa.gov/oswer/riskassessment/asbestos/pdfs/ asbestos_report.pdf (last accessed February 17, 2013): "The panelists also agreed that the available data suggest that the risk for fibers less than 5 μm in length is very low and could be zero" (*id.* at vii-viii) and that "[f]or *mesothelioma*, the panelists supported the use of different relative carcinogenic potencies for different fiber types. The panelists unanimously agreed that the available epidemiology studies provide compelling evidence that the carcinogenic potency of amphibole fibers is two orders of magnitude greater than that for chrysotile fibers." (*id.* at viii). Similarly, a panel of the Agency for Toxic Substances Disease Research ("ATSDR"), part of the Centers for Disease Control, concluded by consensus that "there is a strong weight of evidence that asbestos [fibers] shorter than 5 μm are unlikely to cause cancer in humans." ATSDR, *Report on the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibers: The Influence of Fiber Length,* vi (Mar. 17, 2003), available at http://www.atsdr.cdc.gov/HAC/asbestospanel/asbestostoc.html (last accessed August 12, 2013).

These findings are consistent with the conclusion that there is substantially different carcinogenic potential for different forms of asbestos.[12]

Dose

Moreover, Dr. Hammar's reasoning, based on finding "some" chrysotile fibers in patients' abdominal cavities, is inconsistent with a "central tenet" of toxicology (the study of the adverse effects of chemical substances on living organisms) that determining the dose-response relationship is "essential in evaluating a causal connection between an alleged exposure and a particular disease." Eaton, "Scientific Judgment," *supra*, at 18. In order for an opinion on causation to be reliable, it must be premised on three criteria, each of which depends on a dose-response relationship:

> 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.

B. D. Goldstein & M. S. Henifin, "*Reference Guide on Toxicology,*" Reference Manual on Scientific Evidence 661 (Fed. Judicial Center 3d ed. 2011) ("Toxicology Guide").

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[12] Dr. Hammar relied in part on the "Helsinki Criteria," (13 RT 2558-60), more formally the "Consensus Report, *Asbestos, Asbestosis, and Cancer: the Helsinki Criteria for Diagnosis and Attribution,"* 23 Scand. J. Work Environ. Health 311 (1997)). The Helsinki Criteria is a useful document, but it is not an authoritative, scholarly treatise on mesothelioma causation. Many statements in it on important matters are not supported with references to the literature. Less than one page of the seven-page document addresses mesothelioma. Much of the Helsinki Criteria supports our discussion of mesothelioma and asbestos. The Helsinki Criteria recommends that efforts should be made to quantify asbestos exposures to determine cumulative asbestos exposure in fiber/per milliliter x years when possible). The Helsinki Criteria does not support Dr. Hammar's conclusion that chrysotile asbestos exposure causes peritoneal mesothelioma. Other experts conclude that the environmental asbestos exposures causing mesothelioma are limited to the more carcinogenic amphibole asbestos types. This conclusion and the facts in this case – *i.e.*, that Mr. Strickland had very high exposure to amphibole asbestos – are consistent.

Causation "is based on an assessment of the individual's exposure, including the amount, the temporal relationship between the exposure and disease, and other disease-causing factors.[13] This information is then compared with scientific data on the relationship between exposure and disease." (Toxicology Guide at 665) and "When an exposure to a chemical is less than that known to produce a toxic response, scientific data cannot, as a rule, support a claim of a causal connection." (Gots, *Toxic Risks*, at 163).

While precise dosage information may not be needed if it is established that harm can be caused by cumulative exposure to a substance, California requires that a plaintiff prove that the exposure was a "substantial factor" contributing to the risk of developing the asbestos-related disease, by showing the contribution to the risk of disease in question was itself substantial. *Rutherford,* (1997) 16 Ca1.4th 953, 975-979, 982 ("*Rutherford"*).

In asbestos cases, the plaintiff must both establish some threshold exposure to the defendant's defective asbestos-containing products and that "in reasonable medical probability [that exposure] was a substantial factor in contributing to the aggregate *dose* of asbestos the [worker] inhaled or ingested, and hence to the *risk* of developing asbestos-related cancer, without the need to demonstrate that fibers from [the] product were the ones, or among the ones, that *actually* produced the malignant growth." *(Rutherford, supra,* 16 Ca1.4th 953 at 975-977. This requires a plaintiff to show that the risk of cancer created by a worker's exposure to a particular asbestos-containing product *"was significant enough* to be considered a legal cause of the disease." *(Id.* at p. 975, emphasis added.)

While *Rutherford* relieved asbestos-injury plaintiffs of the burden of showing that exposure to a defendant's product *actually* contributed to a worker's injury, a natural constraint on who may be liable inherent in the "but for" test,' and substituted the requirement that plaintiffs prove the exposure made a *substantial* contribution to the worker's *risk* of injury, in the context of all of the injured party's other exposures. The Court balanced a plaintiffs interest in recovering for an injury, despite

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[13] The relevant question is "Is any **meaningful** amount [of a toxin] present?" R.E. Gots, Toxic Risks: Science, Regulation, & Perception, at 108 (CRC Press 1993) ("Toxic Risks"). (emphasis supplied). The dose-response relationship, which "describes the relationship between the dose and the severity of the effects of a substance's dose," D. L. Eaton, *Scientific Judgment & Toxic Torts – A Primer in Toxicology for Judges & Lawyers*, 12 J.L. & Pol'y 5, 15 (2003), is fundamental to the science of toxicology. *See* Casarett and Doull's Toxicology, *supra* and E. K. Silbergeld, *The Role of Toxicology in Causation: A Scientific Perspective*, 1 Cts. Health Sci. & L. 374, 378 (1991).

an inability to show its precise origin, against the defendant's right to avoid responsibility unless its participation in the events leading to the injury was significant enough to be treated as a legal cause.

Rutherford reinforced the importance of particularized proof that the defendant's product contributed significantly to the risk of harm by noting that there is a "wide variation in form and toxicity of asbestos products . . . ." *(Rutherford, supra,* 16 Ca1.4th at 972; *see also id.* at 979 and "In some industries" – including the construction industry where Strickland worked – "many different asbestos-containing products have been used, often including several similar products at the same time periods and worksites." *(Id.* at 975.) "The probability that *any one* defendant is responsible for plaintiffs injury *decreases* with an increase in the number of possible tortfeasors.'" *(Id.* at p. 979, emphases added.)

The issue, the *Rutherford* Court explained, is whether, "[t]aking into account the length, frequency, proximity and intensity of exposure . . .[and] any other potential causes to which the disease could be attributed, 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?" *(Rutherford,* 16 Ca1.4th at 975.). Moreover, a jury must be instructed that an exposure to asbestos "is deemed to be a substantial factor in bringing about the injury *if its contribution* to the plaintiff or decedent's *risk or probability* of developing cancer *was substantial*." *(Id.* at 977, first and third emphases added, second emphasis in original.).

With respect to the likelihood that chrysotile asbestos causes peritoneal mesothelioma, the epidemiological evidence negates Dr. Hammar's hypothesis. The Surveillance Epidemiology and End Results (SEER) program found one death from pleural mesothelioma in every 555 US males (0.18%) and one death from peritoneal mesothelioma in every 7,142 US males (0.016%) from 1973 to 2002. B. Price, R. Wilson, "Trends in incidence of mesothelioma and evaluation of exposure asbestos" in R. P. Nolan, A. M. Langer, M. Ross, F. J. Wicks, R. F. Martin (eds.), The Health Effects of Chrysotile-Asbestos: Contribution of Science to Risk Management Decisions, The Canadian Mineralogist, Special Publication: 2001; 5: 53-61; M. J. Teta, P. J. Mink, E. Lau, B. K. Sceurman, E. D. Foster, "US Mesothelioma Patterns 1973-2002: Indicators of Change and Insight to Background Rate," European J. Cancer Prevention 2008: 17; 525-34. In the general U.S. male population, only 8% of the mesothelioma cases occur in the peritoneum. M. J. Teta, *et al.*, *supra*,; S. H. Moolgavkar, R. Meza, J. Turim, "Pleural and Peritoneal Mesothelioma" in SEER: Age Effects and Temporal Trends, 1973-2005: Cancer Causes Control 2009: 20: 935-44.

Sixty-four percent of the crocidolite-caused mesotheliomas occur in the peritoneum and about half the mesotheliomas that occur in the amosite-exposed cohorts were peritoneal. These lead to a risk

ratiio ("RR") of 81 and 765, respectively, when compared to the general male population (Table 1). Among the anthophyllite asbestos-exposed cohort there was one peritoneal mesothelioma accounting for 25% of the mesothelioma cases in 503 deaths for a RR of 16, with a large uncertainty. The evidence of increased RR for peritoneal mesothelioma for Finnish workers is statistically weak (*see* Table 1, *infra*).

For the other asbestos fiber types, tremolite-actinolite asbestos and chrysotile, the pattern is different. Each cohort had only one peritoneal mesothelioma (Table 1). In the cohort exposed to tremolite-actinolite asbestos from Libby, Montana there were 767 deaths and the RR for both peritoneal and pleural mesothelioma was 10. For chrysotile, the asbestos cohort with the largest number of deaths, 7,883, no peritoneal mesothelioma deaths occurred, and the RR was less than one. Thus high occupational exposure to chrysotile **did not** increase the risk of developing peritoneal mesothelioma beyond what would be expected in the general population.

Among the cohorts with occupational exposure to chrysotile asbestos mesothelioma mortality is less than 0.5% in two chrysotile mining cohorts (*see* Graham W. Gibbs, Geoffrey Berry, "Mesothelioma and Asbestos," 52 Regulatory Toxicology and Pharmacology S223–S231 (2008) for the amphibole asbestos cohorts). In the two chrysotile cohorts of miners (where the majority of the chrysotile mesothelioma cases occur) the estimated cumulative chrysotile exposure is ***more than*** 500 f/mL x years (Gibbs and Berry 2008; Hodgson and Darnton 2000). In one chrysotile cohort engaged in manufacturing friction products, no mesothelioma deaths were was found among the 1,267 deaths in that cohort (Gibbs and Berry 2008). Friction product workers had cumulative chrysotile exposures significantly in excess of the background and yet none developed mesothelioma.

Historically, high occupational exposure of cohorts of workers exposed to all the amphibole asbestos types report cases of pleural and peritoneal mesothelioma. The chrysotile exposed cohorts present a different pattern: pleural mesothelioma among those with high chrysotile exposures are barely twice that of the general population, while the incidence of peritoneal mesothelioma **is less than the general population**. High occupational exposure to chrysotile asbestos did not increase the risk of developing peritoneal mesothelioma beyond what would be expected in the general population. There is no evidence that occupational exposure to chrysotile asbestos increases the risk of developing peritoneal mesothelioma.

Table 1. Comparison of the incidence of pleural and peritoneal mesothelioma deaths in occupational cohorts exposed to various asbestos types and the calculated Relative Risk of developing these diseases compared with males in the general population.

		Pleural		Peritoneal	
Asbestos Type	**Cohort**	**Mesothelioma Deaths as % of All Deaths**	**Relative Risk**	**Mesothelioma Deaths as % of All Deaths**	**Relative Risk**
Crocidolite§	Mines & Factory	**7.3% (229/3,137)**	**39**		
Crocidolite	US & Canadian			**10.7% (9/84)**	**765**
Amosite§	Factory	**1.2% (12/962)**	**10**	**1.1% (11/962)**	**81**
Tremolite-Actinolite†	Miners	**1.8% (14/767)**	**10**	**0.13% (1/767)**	**9**
Anthophyllite‡	Miners	**0.59% (3/503)**	**3.2**	**0.2% (1/503)**	**16**
Chrysotile§	Quebec & Italy Miners & Millers	**0.44% (35/7883)**	**2**	**0% (0/7,883)**	**0**

§ J. T. Hodgson, A. Darnton, "The quantitative risk of mesothelioma and lung cancer in relation to asbestos exposure," Annals of Occup. Hyg. 2000; 44: 564-601; Graham W. Gibbs, Geoffrey Berry, "Mesothelioma and Asbestos," 52 Regulatory Toxicology and Pharmacology S223–S231 (2008).

† Calcic amphiboles with increased sodium and potassium. *See* P. A. Sullivan, "Vermiculite, Respiratory Disease, and Asbestos Exposure in Libby, Montana: Update of a Cohort Mortality Study,"Environ. Health Perspect, 2007; 115: 579-585.

‡ L. O. Meurman, H. Kiviluoto, M. Hakama, "Mortality and Morbidity among the Working Population of Anthophyllite Asbestos Miners in Finland," Br. J. Ind. Med. 1974: 31**:** 105-112 and L.O Meurman,H. Kiviluoto, M. Hakama, "Mortality and Morbidity among the Working Population of Anthophyllite Asbestos Miners in Finland," Br. J. Ind. Med. 1974; 31: 105-112.

Specific Causation

Plaintiffs' evidence of specific causation is even more problematic. When studying specific causation, it is necessary to have some idea of the exposure of the specific individual and the relationship of that exposure to the exposure of the group for which general causation has been established.

The trial court allowed Dr. Hammar to offer a causation opinion without even discussing, let alone estimating: (1) Mr. Strickland's overall chrysotile asbestos exposure from all defendants' products cumulatively, (2) Mr. Strickland's overall chrysotile asbestos exposure from any product, (3) Mr. Strickland's overall asbestos exposure from any product of any one defendant, (4) Mr. Strickland's exposure from Union Carbide products, or (5) the relative risk of Mr. Strickland's exposure to Union Carbide's chrysotile asbestos products compared to the background risk of mesothelioma in the general population.[14]

Dr. Hammar's Departures From the Scientific Method

The "scientific method is based on generating hypotheses and testing them to see if they can be falsified." *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 593 (1993). This basic method of "falsification" distinguishes science from other fields of human inquiry. Karl R. Popper, THE LOGIC OF SCIENTIFIC DISCOVERY 32, 40-41, 46 (1959) and "the criterion of the scientific status of a theory is its falsifiability, or refutability, or testability." Karl R. Popper, Conjectures and Refutations: The Growth of Scientific Knowledge 37 (5th ed. 1989).[15] The scientific method is a method of research in which a problem is identified, relevant data are gathered, a hypothesis is formulated from these data, and the hypothesis is empirically tested. The scientific method works because the availability of underlying data to other scientists ensures the ability to test or verify a scientific experiment by a parallel experiment or other standard of comparison.

A "methodology" by which an expert replaces the testing of the expert's primary hypothesis through human epidemiological studies with case reports and unscientific assumptions is at odds with the very essence of the scientific method, by which an hypothesis is "formulated" *and then empirically tested*.

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[14] For a recent cogent decision on the need for an expert on causation to quantify exposure, *see Moeller v. Garlock Sealing Technologies LLC*, 660 F.3d 950, 955 (6th Cir. 2011).

[15] We cite Karl Popper both because he is a leading philosopher of science and because he was cited by Dr. Hammar in his testimony explaining Hammar's "black swan" approach. (13 RT 2563).

Dr. Hammar's methodology does not include the generally accepted methodologies used by other scientists when determining the causation of asbestos-related diseases.

Dr. Hammar ignored the overwhelming toxicologic and epidemiologic evidence that chrysotile asbestos exposure is not causally associated with peritoneal mesothelioma. His reliance on isolated case reports do not constitute a scientifically sound or generally accepted method. Case reports describe a particular effect in an individual or group of individuals who were exposed to a substance. These reports are often anecdotal or highly selective in nature and **generally are of limited use for hazard assessment**. Specifically, cancer causality can rarely be inferred from case reports alone. *See* United States, Environmental Protection Agency, "Guidelines for Carcinogen Risk Assessment," at 2-6 (March 2005) (emphasis added), available online at http://www.epa.gov/raf/publications/pdfs/CANCER GUIDELINES FINAL 3-25-05.pdf (last visited Aug. 12, 2013). "Anecdotal reports can provide some information, but they are more useful as a stimulus for further inquiry [as Dr. Hammar himself suggested, 13 RT 2452-2454], not as a basis for establishing association. . . . Typically, the reports are obtained haphazardly or selectively, and the logic of "post hoc, ergo propter hoc" does not suffice to demonstrate that the first event causes the second. Consequently, while anecdotal evidence can be suggestive, it can also be quite misleading. David H. Kaye & David A. Freedman, *Reference Guide on Statistics*, Reference Manual on Scientific Evidence, at 90-91 (Fed. Judicial Center 2d ed. 2000) (footnote omitted). In short, anecdotal case reports are incompetent as evidence of causation because their use for this purpose is not "generally accepted."

While California cases hold that "general acceptance" is applicable only to "new scientific techniques," *see, e.g. People v. Leahy*, (1994) 8 Cal.4th 587, 605, and not to the expert's ultimate conclusion, under California's *Kelly/Frye* test, a party that is seeking to introduce a new scientific methodology must show (1) that the reliability of the technique has "gained general acceptance in the relevant scientific community;" (2) that the expert testifying about that technique is qualified to do so; and (3) that "correct scientific procedures were used in the particular case." *See Geffcken v. D'Andrea*, (2006) 137 Cal.App.4th 1298, 1309. We believe that Dr. Hammar's reliance case reports – and, in essence, on a single so-called "Black Swan"case report, and slighting of contrary epidemiological evidence, is a new, and not generally accepted, method.

Moreover, in *Sargon Enter. v. University of Southern California* (2013) 55 Ca1.4th 747, this Court seems to have moved a toward a *Daubert*-like standard of admissibility of expert testimony, holding that under Section 801 of the Evidence Code an expert opinion must be based on matter that provides a reasonable basis for the opinion. *Sargon*, 55 Cal.4th at 770, and that expert opinion based "on conjecture or speculation is inadmissible." *Id.* Further, the opinion "may not be based 'on assumptions of fact without evidentiary support.'" *Id.* Under section 802 of the Evidence Code, a

trial court is to examine the actual reasons for an expert's opinion. If there is "too great an analytical gap between the data and the opinion proffered" then a court may rule the testimony inadmissible. *Id*. at 771. It is the trial court's responsibility to analyze the logic of an expert's conclusion, not the persuasive or probative power of the conclusion. *Id*. If the matter relied on provides a reasonable basis for the opinion, it should be admitted. *Id*. The court's gatekeeping function can be summarized as excluding "'clearly invalid and unreliable' expert opinion." *Id*.

Amici submit that Dr. Hammar's opinion was based on assumptions without evidentiary support and are mere conjecture or speculation.

We respectfully urge this Court to grant review because the Court of Appeal decision warrants review to settle a question of law important to civil litigation. *See* Cal. R. Ct. 8.500(a)(1)..

Respectfully submitted,

Martin S. Kaufman
Counsel for *Amici*

MSK:mbs

Strickland v. Union Carbide Corporation

PROOF OF SERVICE

I am counsel for *amici curiae* Richard Wilson, *et al.* in this matter. I am over the age of 18 and not a party to the within action. My business address is 2039 Palmer Avenue, Suite 104, Larchmont, New York 10538.

On the date stated below, I served in the manner indicated below, the foregoing document described as: AMICUS LETTER BRIEF on the parties indicated below by placing a true copy thereof, enclosed in a sealed envelope addressed as follows:

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by United States Postal Service. I personally deposited envelopes containing copies of the attached *amicus* letter with the United States Postal Service that same day, with the postage thereon fully prepaid, in a postal depository box under the exclusive care of the United States Postal Service in Larchmont, New York and I served the Court of Appeal by electronic mail, as indicated o the Service List.

I declare under the penalty of perjury under the laws of the State of New York that the foregoing is true and correct.

Executed this 15th day of August, 2013.

_____

Martin S. Kaufman

SERVICE LIST

STRICKLAND v. UNION CARBIDE CORPORATION
Case No.: S212424
LASC No. BC379088 -- COA Case No. B234459

Individual / Counsel Served	Party
Ted W. Pelletier, Esq. LAW OFFICES OF TED W. PELLETIER 22 Skyline Road San Anselmo, California 94960 (415) 454-8783 / Fax: (415) 454-1592	Plaintiffs and Respondents LINDA S. STRICKLAND individually and as successor-in-interest to ARTHUR STRICKLAND, deceased; and LINDA S. , HOLLY MORGAN, ROBERT STRICKLAND, as legal heirs of ARTHUR STRICKLAND, deceased
Dean A. Hanley, Esq. PAUL & HANLEY LLP 1442 Grand View Drive Berkeley, California 94705 (510) 685-0117 / Fax: (510) 644-9571	Plaintiffs and Respondents LINDA S. STRICKLAND individually and as successor-in-interest to ARTHUR STRICKLAND, deceased; and LINDA S. , HOLLY MORGAN, ROBERT STRICKLAND, as legal heirs of ARTHUR STRICKLAND, deceased
David M. Axelrad, Esq. Daniel J. Gonzalez, Esq. HORVITZ & LEVY LLP 15760 Ventura Boulevard, 18th Floor Encino, California 91436-3000 (818) 995-0800 / Fax: (818) 995-3157	Defendant and Appellant UNION CARBIDE CORPORATION
Morton D. Dubin, Esq. ORRICK, HERRINGTON & SUTCLIFFE LLP 51 West 52nd Street New York, NY 10019-6142 (212) 506-5000 / Fax: (212) 506-5151	Defendant and Appellant UNION CARBIDE CORPORATION

Catherine Morris Krow, Esq. Thomas A. Harvey, Esq. ORRICK, HERRINGTON & SUTCLIFFE LLP The Orrick Building 405 Howard Street San Francisco, CA 94105 (415) 773-5700 / Fax: (415) 773-5759	Defendant and Appellant UNION CARBIDE CORPORATION
Hon. Charles F. Palmer Los Angeles County Superior Court Central District 111 N. Hill Street - Dept. 33 Los Angeles, California 90012-3014 (213) 974-5641	Trial Court Judge LASC No. BC379088
California Court of Appeal Second Appellate Division Two 300 South Spring Street Second Floor, North Tower Los Angeles, CA 90013 (213) 830-7102	Court of Appeal Case No. B234459 Electronic Service Copy Only http://www.courts.ca.gov/9280.htm
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