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Kansas Journal of Law & Public Policy

Fall, 2012

Kansas Journal of Law & Public Policy

22 Kan. J.L. & Pub. Pol'y 1

LENGTH: 25594 words

ARTICLE: THE "ANY EXPOSURE" THEORY ROUND II -- COURT REVIEW OF MINIMAL EXPOSURE EXPERT TESTIMONY IN ASBESTOS AND TOXIC TORT LITIGATION SINCE 2008

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TEXT:

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To members of the asbestos litigation bar, the any exposure theory of causation has become a familiar part of the landscape over the last decade. Plaintiff experts routinely testify that "each and every exposure" to asbestos in occupational settings or during para-occupational activities (e.g., home repair or backyard brake jobs), no matter how small, is a substantial contributor to asbestos disease. The dose n2 does not matter - virtually every individual [*2] exposure, no matter how limited, is considered causative. As a result, these experts forego any sort of assessment of the overall dose received from any particular defendant's product.

If courts permit this testimony to be presented to a jury, defendants rarely escape the risk of an asbestos jury trial, assuming there is evidence sufficient to support usage of or exposure to the product. In reliance on this theory, asbestos cases have targeted increasingly de minimis exposure scenarios, including not only minimal workplace exposures, but also "bystander" and "take-home" cases where the already miniscule exposures from the product or work activity are reduced even further to near obscurity. n3

Starting in 2005 courts began to realize that the any exposure theory as applied in low-dose asbestos cases made no sense and had no scientific support. The history of decisions rejecting that theory up through 2008 is set forth in a previous article, The "Any Exposure" Theory: An Unsound Basis for Asbestos Causation and Expert Testimony. n4 That article explained the rationale behind these courts' rejection of the theory and recited the specifics of more than a dozen

decisions at that time concluding that the any exposure theory was not scientifically sound. These courts included the highest courts in two significant states (Texas and Pennsylvania), the Sixth Circuit Court of Appeals, and an array of lower state and federal court decisions. n5 This set of rulings constituted a strong movement, virtually without opposing opinions, against the any exposure theory - the curtain had been pulled back, exposing the theory as nothing more than an unproven and unscientific litigation tool.

Since 2008, however, the battle over this theory has hardly resolved itself. Instead, several asbestos courts (although by no means all) have decided to let any exposure theorists testify. n6 Plaintiffs' attorneys and experts have also [*3] made several attempts to export this theory into other toxic tort litigation, especially in benzene cases. For the most part, those efforts have not succeeded - outside of asbestos, the theory has not found significant support - but the effort to export it will undoubtedly continue. n7

The any exposure theory is unquestionably one of the most important scientific issues on the table today in the world of asbestos and toxic torts. Can plaintiffs prove a toxic tort case merely by proving or assuming any exposure (no matter how minimal) and thereby avoid identifying an actual toxic dose of the substance? If the answer is yes, the world of asbestos litigation will expand and extend indefinitely because of the ongoing reality of idiopathic mesothelioma, n8 coupled with ubiquitous asbestos exposure in the modern industrialized world. Many idiopathic cases - more than 300 of them every year in the U.S. alone - are believed to be naturally occurring or otherwise not caused by asbestos exposure. Nevertheless, the any exposure theorists frequently attribute even idiopathic cases to minute amounts of asbestos included in thousands of products up to and after the 1980s. n9 If the any [*4] exposure theory is also sufficient for other toxic torts, then that type of litigation would extend potentially to a large number of other substances known or alleged to be harmful at high doses but not demonstrated in epidemiology study to cause harm at lower doses. This would be a shattering paradigm shift in the legal landscape - the burden of proof would shift to defendants, who would be required to disprove causation after plaintiff demonstrated only mere exposure to get to a jury.

This article picks up where the 2008 article left off and extends the discussion to non-asbestos cases. Based on a wealth of scientific information, the any exposure theory is wholly unsuitable as a basis for asbestos or tort litigation. That conclusion is supported today by close to thirty court opinions and many experts inside and outside of litigation who have critiqued the theory or have testified against it in court. n10 The most recent state appellate court to address the any exposure theory - the Supreme Court of Pennsylvania in May 2012 - thoroughly and unanimously rejected the theory under that state's Frye standard. n11 The few courts that have permitted experts to testify to causation without a dose assessment in recent years typically have one thing in common - they have declined to examine the underpinnings and scientific support for this testimony and have largely accepted what the experts say at face value. This is a serious flaw under any version of expert gatekeeping responsibility.

Ironically enough, based on the last four years of experience, the fate of [*5] the any exposure theory turns more on the court's methodology than it does on the experts'. These experts do not change their approach from case to case, and the theory is no less flawed in the jurisdictions allowing these experts to testify than it is in the many jurisdictions that have rejected such testimony. The difference is that some courts simply choose to conduct only a limited review of the experts' conclusions and approach, and do not look closely enough at the theory's lack of scientific underpinning and legal significance. The review of complex science is often difficult, but imperative for good judicial gatekeeping. The court must read the studies, analyze the expert's thinking, and pierce the veil if in fact the experts are not fairly reporting their work and the literature. Courts that engage in the proper level of review have repeatedly found that the any exposure theory is not supported by published, peer-reviewed articles and is at best litigation-driven speculation.

This article begins with a description of the any exposure theory, how it relates to fundamental principles of dose and causation, and how it is being used today in asbestos and non-asbestos toxic tort cases. Section II addresses the principles under which courts should conduct an analysis of scientific testimony, with a focus on Daubert n12 and the rigor required for a genuine Daubert examination of scientific evidence. Section III, surveying developments in asbestos law regarding the any exposure theory since 2008, demonstrates that courts accepting this evidence are not looking past the experts' own self-serving statements. Section IV then turns to non-asbestos litigation since 2008 and illustrates that in this context, where courts apply a more meaningful review and standard tort causation rules, the any exposure theory is almost universally rejected. Finally, if courts engage in the proper level of review - framing the question correctly, reading and assessing the studies, requiring more than qualitative expression such as "significant," and measuring the theory against the legal yardstick of substantial factor causation - the any exposure theory cannot survive review or support litigation.

I. The Any Exposure Theory in Asbestos and Other Litigation

A. Dose and Asbestos Toxicology

To understand the flaws involved in the any exposure theory, some background on asbestos and toxicology is necessary. "Asbestos" is not an actual substance - it is a word used to describe a group of separate and distinguishable minerals that sometimes form in the shape of a fiber. n13 The most important distinction among those different minerals is the one between amphibole fibers like amosite or crocidolite - long, fairly rigid fibers that the [*6] body cannot get rid of easily - and serpentine fibers such as chrysotile that are easily broken down and removed by the body. n14 This distinction is critical for toxicity purposes. n15 Most studies demonstrating mesothelioma and other asbestos diseases arise in the context of amphibole exposure at what today would be considered high doses, i.e., well above today's regulatory limits on asbestos exposure in the workplace. n16 Chrysotile, however, even in high doses, is only rarely a source of mesothelioma, as demonstrated in multiple studies. n17 [*7] At low doses of chrysotile, n18 epidemiology studies have found the occurrence of mesothelioma does not differ from professions with little or no opportunity for asbestos exposure, such as teachers, accountants, or farmers. n19

The lack of toxicity at low levels is not surprising. Asbestos, like virtually all toxins, only presents a real risk of causing disease if the dose is high enough. n20 The human body has an amazing capacity to deal with a wide variety of toxic substances present in our environment, many of them natural (e.g., radiation from sunlight, carcinogens in food, or even dangerous metals like arsenic or zinc that our body requires in small amounts). n21

Disease results when those exposures reach a level that overwhelms the body's defenses, a dose known as the "threshold" point. Aspirin, alcohol, sunlight, or even known "poisons" are harmless or beneficial at lower doses; however, they can be poisonous if the dose is high enough to make them so. For this reason, since the time of Paracelsus, toxicology has rested on the [*8] bedrock principle that "the dose makes the poison." n22 Thresholds are not always easy to identify, but that does not mean they do not exist. Regulators may take the conservative route of stating there is no "known" safe dose of a substance (as they often do), but that does not excuse courts and experts from the necessity of determining whether an exposure exceeds the approximate threshold of a demonstrable increase in disease in exposed populations. n23

This principle is explained with impressive clarity in the leading scientific article on toxic substance causation by Professor David Eaton at the University of Washington. n24 As Professor Eaton states, "dose is the single most important factor to consider in evaluating whether an alleged exposure caused [*9] a specific adverse effect... . If [the ability of a chemical to cause the disease in question] has been established ... then it must be established that the individual's dose over a defined period of time was sufficient to cause the alleged health effect ... It is not adequate to simply establish that "some" exposure occurred." n25 This dose principle holds true for carcinogens like asbestos just as much as it does for any other toxin:

Most chemicals that have been identified to have "cancer-causing" potential (carcinogens) do so only following long-term, repeated exposure for many years. Single exposures or even repeated exposures for relatively short periods of time (e.g., weeks or months) generally have little effect on the risk of cancer, unless the exposure was remarkably high and associated with other toxic effects. n26

At one time, the one-hit theory posited that carcinogens could cause cancer with a single exposure, but as the quote above demonstrates, that theory has long since been debunked. n27 Today, it is well understood that even carcinogens require significant and multiple exposures beyond harmless levels to produce disease. n28

The same is true in the courtroom - a proper causation assessment of any job-related exposure, and any litigation claim of disease from such an exposure, should include a reasonable assessment of the likely range of dose received by the worker and a determination as to whether that dose is comparable to amounts known (not speculated) to cause disease. n29 Case law throughout the country "overwhelmingly" n30 supports the rule that the plaintiff [*10] in a toxic tort/latent disease case "must prove the levels of exposure that are hazardous to human beings generally as well as the plaintiff's actual level of exposure to the defendant's toxic substance before he or she may recover." n31 This principle is not complex or particularly open to reasonable discussion - no one would conclude that taking aspirin caused someone's death without first at least asking the question how many aspirin are involved.

The foundations of toxicology and realities of varying asbestos potency and dose demonstrate why it is so important in asbestos cases to identify how much of a dose an individual plaintiff incurred and whether that dose has been shown to cause disease in the medical literature. In legal terms, this is known as specific causation. n32 Such proof is mandatory in most non-asbestos litigation. n33 It is not enough to describe the dose in purely qualitative terms like "substantial" or "significant" - those words can (and often do) mean nothing because they are not measured against any

sort of yardstick. n34 The necessity of assessing and estimating the historical dose from a particular product or exposure and comparing that dose to demonstrable levels inducing disease is thus a critical part of a scientifically supported causation assessment.

[*11] The necessity of requiring a dose assessment for asbestos also derives from the combined effect of two other fundamental truths. First, everybody is exposed to asbestos. Certain forms of asbestos are both naturally occurring in the environment and ubiquitous at very low levels due to the widespread use of the product in urban areas. n35 These levels - called "background" exposures - are not considered harmful. n36 Second, like all other known cancers, mesothelioma occurs from causes in addition to asbestos n37 and from natural causes, without any involvement of asbestos - hundreds of such cases appear annually in the U.S. alone and there is no indication that these cases will disappear. n38 These two realities taken together create the following scenario - every person who has mesothelioma has also been exposed to at least some asbestos, and yet twenty percent or so of those cases are believed to have nothing to do with asbestos exposures. n39 Therefore, there is such a thing as inconsequential asbestos exposure even in persons who have mesothelioma.

Thus, it is critical to distinguish these spontaneous or idiopathic cases with incidental but unimportant asbestos exposures from those cases where the dose and type of asbestos are sufficient to conclude the mesothelioma is not idiopathic but an occupationally-derived, asbestos-induced disease. It is not enough to reason that a person who has mesothelioma was also exposed to occupational asbestos, and therefore the exposure must be the cause of the disease. Such statements - regular components of any exposure testimony - represent classic circular reasoning. n40

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B. The Any Exposure Theory in Asbestos Litigation

The any exposure theory contradicts every fundamental principle set forth above. It is usually articulated, in expert reports and testimony, in the broadest possible terms: Each and every exposure to any kind of asbestos that is above (or different from) background exposures is a substantial factor in causing disease. n41 Removal of a single gasket, changing a brake in the back yard, handling a brake pad in a warehouse - virtually any contact with an asbestos-containing product that produces "dust" (and sometimes not even that) is sufficient under the any exposure theory to put the defendant in front of a jury.

One would think, given the extreme nature of this kind of opinion, that the experts would be wary of stating it so boldly to avoid the appearance of overreaching. That is not the case. The experts are unabashedly direct in their claim that every occupational breath is a cause: "[The Experts] do not rely, in any respect, upon any actual quantity or quality of exposure suffered by any specific plaintiff, but rather, conclude that if the evidence supports a single exposure, then causation can be opined and asserted." n42

These theorists support their opinions by asserting that asbestos is a dose-response disease that depends on the cumulative impact of fibers entering the lung. n43 No one can tell which of those fibers actually promoted the tumor (usually mesothelioma in today's litigation). Thus, while ignoring their own inability to prove which exposure caused the disease, the any exposure experts state that all such fibers entering the lung from a workplace or product exposure must be considered causative. n44 Many regulators and some scientific articles have stated the proposition that there is no known safe dose of asbestos. n45 The any exposure supporters turn this proposition into the [*13] affirmative statement that all such exposures must therefore be considered causative. n46 They will agree that mere background exposures to asbestos are not a cause of asbestos disease, but they fail to accept the necessary corollary - that dose must then be important and they must identify an actual causative dose, even in occupational settings. n47 These experts simply assume that all occupational exposures (regardless of dose) are different from background or added to normal background, and are therefore causative even though background exposures (regardless of dose) are not. n48

The scientific flaws in this theory are significant. Several court opinions have thoroughly deconstructed the reasoning behind the any exposure theory, stripping away the pretence and finding it to be at best an unproven hypothesis designed to drive litigation. n49 Those flaws are set forth in detail in the earlier article and court opinions cited therein and will not be restated here. The primary issue remains one of dose - the fundamental principle of toxicology that substances, including carcinogens, are not harmful via "any" exposure but necessarily require a sufficient dose. This is not only blackletter science, it is Toxic Tort Law 101. A plaintiff must prove not only an exposure to a toxin from the defendant's product or activity, but that the exposure created enough of a dose to cause the alleged disease. n50

In asbestos litigation, the battle over this theory continues to rage, with opinions going both ways. n51 Section III reviews a number of recent opinions [*14] rejecting or accepting this testimony. Court acceptance/rejection of the theory continues to dominate what kind of and how many asbestos cases can be filed in jurisdictions where these cases are common. Perhaps as important, the decisions result in even more blatant forum shopping than otherwise might occur, because plaintiffs seek out the jurisdictions that allow this testimony to support a low dose case. Asbestos litigation in Texas, for instance, has reduced in scope dramatically in the last five years following the combination of tort reform legislation and the Texas Supreme Court's rejection of any exposure testimony in the seminal 2007 Borg-Warner n52 decision. As a result, several prominent plaintiff asbestos firms in Texas have opened offices or begun to file cases in more favourable states or courts, including those that will allow such testimony to support a case. n53 The viability of the any exposure issue is presently pending before the Virginia Supreme Court n54 and the United States Court of Appeals for the Ninth Circuit. n55 Defendants routinely file similar motions against the any exposure theory in many cases around the country.

C. Attempts to Inject the Any Exposure Theory into Other Toxic Tort Litigation.

For many years courts dealing with toxic tort litigation have routinely required some evidence demonstrating a sufficient dose to cause injury to allow a non-asbestos case to go to a jury. n56 Asbestos litigation, however, often [*15] has developed its own set of rules separate and apart from other toxic tort litigation. n57 The any exposure theory has largely been confined to asbestos cases until fairly recently.

Perhaps buoyed by success in certain jurisdictions that have accepted the any exposure theory in asbestos litigation, plaintiff experts are increasingly attempting to inject that theory into other kinds of toxic tort cases. Benzene litigation is a popular target. n58 Exposure to pure benzene (as in a factory setting) at high doses can cause a form of cancer known as acute myelitic leukemia (AML), as demonstrated in several epidemiology studies. n59 The studies, however, do not document AML or any other cancer at lower doses experienced, e.g., from exposure to gasoline, which typically contains from one to five percent benzene. n60 This reality has not prevented the any exposure experts from asserting causation in cases involving only small amounts of benzene exposure, e.g., for gasoline station attendants. n61 They do so by utilizing vague references to "significant" or "extreme" exposures and contending there is no safe dose of the substance. n62

While couched in terms of "no safe dose" or other testimony designed to avoid assessing a low dose exposure, the any exposure theory has also raised its head in other non-asbestos litigation, including fluoride in denture cream, n63 diesel fumes, n64 diacetyl (popcorn) lung litigation, n65 the PFOA or Teflon litigation, n66 and medical monitoring and groundwater cases involving [*16] substances such as MTBE and atrazine. n67 If courts were to begin to accept it, the any exposure theory would serve as an important vehicle for plaintiffs to expand chemical and product tort litigation immensely. Most importantly, the theory would allow plaintiff experts to avoid the necessity of estimating a plaintiff's actual exposure. Reconstructing such exposures, depending on plaintiff's recollection, can sometimes be challenging but once done it often demonstrates that the exposure was actually well below levels known to cause disease. It is much easier for plaintiffs - and thus attractive for litigation reasons - simply to assert that every exposure is a cause. The theory allows cases to be brought that would otherwise have no merit because of the minimal exposure involved.

The any exposure theory has the additional advantage for plaintiffs of shifting the burden of proof to the defendants. Once plaintiff establishes some exposure to an identified product, it would be up to the defendant to demonstrate that very low exposures do not cause disease. Conclusively proving the negative can be a difficult proposition given the limitations of epidemiology involving background or close to background exposures. n68 A classic example is the apparently unending efforts to claim that cell phones cause brain damage despite multiple studies finding no such link. n69 The advantages of the any exposure theory to plaintiffs are thus obvious, but it is still incumbent on courts to determine whether it is viable under Daubert or Frye in the first place. Section IV addresses the specifics of some of these new non-asbestos opinions.

II. THE PROPER COURT METHODOLOGY FOR REVIEWING LOW DOSE CAUSATION TESTIMONY

There is a sharp divide between the courts above that have accepted any [*17] exposure testimony and those that have rejected it. That divide is not derived from the methodology of the experts, but, oddly enough, it is the result of highly variant degrees of rigor in the court's methodology. n70 The critical determinant is the degree of scrutiny the court will apply to the experts' opinion before allowing this testimony to carry the day. Courts that examine the studies and other underpinnings of the any exposure theory universally find the theory lacking. Those that largely accept the experts' statements at face value also accept the theory as sufficiently reliable or allow it to support causation.

A. The Standards of Expert Review under Daubert

The tenets of Daubert and its federal predecessor Frye are by now very familiar to any practitioner of law related to science or expert opinion. *Federal Rule of Evidence 702* requires that expert testimony be based on sufficient facts or data, be the product of reliable principles and methods, and that the expert must have reliably applied the principles and methods to the facts. This reliability analysis must be conducted regardless of whether the witness is qualified to give the challenged opinion. n71 The Court must be vigilant in exercising its gatekeeper role to exclude unreliable expert opinions because a jury is likely to give substantial weight to an expert's opinion about which they have no firsthand knowledge. n72 "Scrutiny of expert testimony is especially proper where it consists of "an array of figures conveying a delusive impression of exactness in an area where a jury's common sense is less available than usual to protect it." n73

In *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, the Supreme Court [*18] enumerated several non-exclusive factors calculated to assist trial judges in determining whether scientific evidence is relevant and reliable, and therefore, admissible under *Federal Rule of Evidence 702*. The factors are: (1) whether a theory or technique can be and has been tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) the technique's known or potential rate of error; and (4) the general acceptance of the theory or technique by the relevant scientific community. n74 These factors, however, are not exclusive, and in fact courts use a wide array of approaches to determining the reliability and fit of scientific testimony. The party offering the expert testimony must demonstrate the expert's opinion is based on the methods and procedures of science, not merely subjective beliefs or unsupported speculation. n75

B. The Degree of Court Analysis Required Under Daubert and Frye

The basic tenets of Daubert are well known and frequently quoted in asbestos and other toxic tort cases. The difference in courts that accept any exposure testimony and those that reject it is not the expert's methodology, but it is the court's methodology in applying Daubert. Courts must apply the tenets of Daubert with a certain amount of rigor or those reviews become nothing more than a rubber stamp on what the experts claim is true or accepted.

The United States Court of Appeals for the Ninth Circuit, applying the "gatekeeper" concept, requires courts in that circuit to accept a responsibility that goes beyond acknowledging what the experts themselves say to support their opinions. n76 Instead, Ninth Circuit trial courts are required to delve into the scientific literature the expert claims as support and understand how and why the expert reached the proffered conclusion. n77

In the remanded Ninth Circuit Daubert opinion, *Daubert v. Merrell Dow* [*19] *Pharmaceuticals (Daubert II)*, n78 the court noted that the Supreme Court's then-new Daubert ruling created a "brave new world" for federal courts in which it had become the court's "responsibility to determine whether those experts' proposed testimony amounts to "scientific knowledge,' constitutes "good science,' and was "derived by the scientific method.'" n79 Trial courts could no longer allow disputes simply to go to the jury just because the opposing experts disagree with one another and stated that "our responsibility, then ... is to resolve disputes among respected, well-credentialed scientists about matters squarely within their expertise ... and occasionally to reject such expert testimony because it was not "derived by the scientific method.'" n80 The gatekeeping function requires the trial court to "analyze not what the experts say, but what basis they have for saying it." n81 The role of the court "will require some objective, independent validation of the expert's methodology." n82

The demands of Daubert, as articulated in *Daubert II*, thus go beyond accepting the experts' own recitation of the claimed bases for support. In that case, the Ninth Circuit itself proceeded to "examine carefully" the experts' affidavits and testimony, including reading the underlying literature related to Bendectin and birth defects. That careful analysis, set forth in five full pages of the Court's opinion, determined that the claimed support was in fact not there, even though the expert said it was. n83 The experts' specific claim that Bendectin was a known cause of human birth defects did not in fact appear in any of the studies they cited. Instead, the experts relied on epidemiology studies that actually contradicted the experts' conclusions - they were testifying to causation from literature that at best indicated a possibility of such a link, and that possibility was contradicted by the findings of the epidemiology itself. n84 The Bendectine testifying experts had thus rejected a consistent series of studies that found no such risk while telling the court the studies supported their claims. The court would never have known this if it had not read and dissected the studies.

The seminal Daubert analysis in the Ninth Circuit thus set the standard - trial judges in Daubert matters must review and carefully analyze the bases for the expert's opinion and make their own determination of its reliability. They cannot accept testimony based on possibilities, theories, or unproven hypotheses dressed up as scientific fact based on misused literature.

The United States Supreme Court has articulated this same principle in the second of the trio of Daubert opinions that today form the foundation for federal court expert review. In *General Electric v. Joiner*, the Court rejected an expert's conclusory claims regarding his opinion as the mere "ipse dixit" of [*20] the expert himself. n85 Without foundational support, the unvarnished statements of the expert are not good enough.

One year after its initial Daubert remand ruling, the Ninth Circuit Court of Appeals reiterated in the Rincon case the necessity for rigor in a Daubert analysis. n86 The expert claimed there was a "wealth of research" supporting her approach. n87 Yet when the court looked for that research, it instead found only a single survey of experts on this issue. And even this survey "did not discuss the research in sufficient detail that the district court could determine if the research was scientifically valid." n88 That same year, in *Claar v. Burlington Northern Railroad Co.*, the Ninth Circuit again upheld a trial court's rejection of scientific testimony after the trial court had examined in detail the basis for the expert's opinion. n89 This Court agreed that the trial court was "required" to test the sufficiency of the expert's opinions and "were not mere subjective beliefs or unsupported speculation." n90 The trial court looked closely at the toxicology sections of the proffered affidavits and found that they "failed to discuss the majority of the medical conditions alleged by plaintiffs." n91 The trial court also reviewed a key study relied on by plaintiffs and found that it actually did not support plaintiffs' stated position. n92 This kind of close review of key studies is essential for a Daubert analysis. n93

The Ninth Circuit continues today to require rigor from its courts in reviewing potentially unreliable expert testimony. In *Avila v. Willits Environmental Remediation Trust*, n94 the court last year rejected plaintiffs' expert affidavits intended to support a class action over release of chemicals from a plant. The court noted the gatekeeper standard, Daubert, "obliges a district judge to determine at the outset ... whether an expert's testimony will assist the trier of fact by assessing whether the methodology underlying the testimony is valid and reliable." n95 The affidavits of several experts were [*21] rejected despite claims of "excessively high levels" of exposure due in part to fairly esoteric flaws in the experts' statistical analyses that the judge took the time and initiative to understand (e.g., the experts' decision to convert results of non-detection to a figure half of the detection limit led to an unsupported exposure estimate). n96 The court also:

- . found "no scientific support in the documents mentioned in [the expert's] declaration for his opinion that a combination of chemicals caused the injuries"; n97
- . figured out that the time periods the plaintiffs actually lived in the affected area were much less than claimed by the experts; n98
- . uncovered the fact that the experts relied on a draft public health assessment instead of the final; n99
- . learned that the experts merely "dismissed" confounding factors rather than considering them; n100 and
- . discovered that the articles cited by the expert did not in fact connect either the mix of chemicals at issue or any single one of them with the claimed injury. n101

None of these flaws would have been discovered if the court merely accepted the experts' claims as stated.

The Ninth Circuit is hardly alone in requiring a careful review, but it is beyond the scope of this article to survey other circuits and state law. To be sure, a trial court has a range of discretion to determine whether the testimony is in fact admissible, but the court does not have discretion in whether or not to conduct a rigorous, gatekeeping inquiry into the reliability of the expert testimony. Based on the any exposure and similar medical causation reviews, courts who apply Daubert with some rigor characteristically utilize the following analytical approaches to uncover the errors in the experts' methodologies and reasoning:

- . Framing the issue correctly: Trial courts should carefully scrutinize the expert's statement of the issue to make sure the expert has correctly framed the issue before the court. Experts frequently misconstrue the scientific issue on the table when they realize there is little support for their conclusion in the scientific community. If there is a disagreement over the correct statement of the issue, the court must itself frame the issue in the correct way given the underlying claim of injury and exposure.
- . Identifying key support for the opinion: Trial courts must focus on the studies and other support the experts rely on. Trial judges [*22] who conduct a proper Daubert analysis do not merely accept what the expert claims the literature says - they read the studies, determine whether the expert has properly drawn on the study for relevant conclusions, and look for inconsistencies the expert has hidden or failed to acknowledge. They also determine whether the expert is

relying on trivial evidence or assumptions that are contradicted by significant contrary evidence, usually epidemiology studies, and is thus not fairly assessing the overall science. n102

. Exposing illogical propositions: Many experts draw conclusions in ways that would fail a college freshman logic course. The circular reasoning of the any exposure theory (because plaintiff has mesothelioma and was exposed to asbestos, the asbestos exposure must be the cause) is such an example of logical error, as is these experts' attempts to extrapolate down from high dose causation to speculative low dose causation. n103

. Looking behind generic or qualitative language: Experts whose causation opinions are weak sometimes rely heavily on qualitative words and terms that hide the lack of scientific rigor behind them. Simply calling an exposure "significant" or "severe" or "above ambient" tells the court nothing because these words have no health-or exposure-based benchmark behind them. A reliable expert opinion should compare the exposures to some standard associated with human disease, and offer reliable testimony that plaintiff's exposures exceeded the level known to cause disease.

. Assessing the legal significance of the theory: Apart from the science, courts that reject the theory typically note its inconsistency with a substantial factor or similar causation [*23] standard. Those that permit the testimony either ignore this issue entirely or establish some alternative standard for asbestos cases that would never be accepted in non-asbestos litigation.

With this background, and utilizing these analytical guides to a correct court methodology for causation testimony review, what follows is a survey of the any exposure law as it has developed since 2008. The focus is on the courts' methodologies in reviewing the testimony and evidence.

III. COURT ASBESTOS LITIGATION RULINGS INVOLVING THE ANY EXPOSURE THEORY ISSUED SINCE 2008.

Courts in asbestos litigation have been active since 2008 in addressing the admissibility and sufficiency of any exposure testimony. The following are illustrative cases from important jurisdictions to demonstrate the trend of the law and the approaches courts are taking. The selection of cases is not intended to be fully comprehensive.

A. Federal Courts

The only federal circuit court opinion involving asbestos and the any exposure theory through 2008 was issued by the Sixth Circuit Court of Appeals. n104 In that opinion, the court rejected the theory as insufficient to support causation. n105 The Sixth Circuit has added two more such decisions since, both rejecting the testimony. In 2009, applying Kentucky causation law that the evidence must support a "probability" of causation and not a mere possibility, this court rejected a claim that handling and possibly cutting gaskets over a period of five years sufficed to prevent summary judgment against the plaintiff:

Plaintiff also argues that, because mesothelioma is a progressive disease, any exposure is a substantial cause. This argument would make every incidental exposure to asbestos a substantial factor... . The Sixth Circuit responded to a similar argument in a maritime action by stating that an expert's opinion that "every exposure to asbestos, however slight, was a substantial factor" was insufficient because it would render the substantial factor test "meaningless." n106

In 2011, the Sixth Circuit again rejected expert testimony attempting to assert causation without assessing the dose. n107 This case involved a long-time pipefitter who had both extensive exposure to asbestos insulation for many years through installing and allegedly removing gaskets every single day. n108 [*24] Plaintiffs' experts attempted to claim that the gasket work was a substantial factor in his disease without determining even an approximate dose from the gasket work as compared to the significant and much more dangerous insulation exposures. n109 Instead, the experts relied on the any exposure theory and assumed the gasket exposures were a contributing factor regardless of dose. The appellate court, applying Kentucky's substantial factor standard, rejected this approach, holding:

Where a plaintiff relies on proof of exposure to establish that a product was a substantial factor in causing injury, the plaintiff must show a high enough level of exposure that an inference that the asbestos was a substantial factor in the injury is more than conjectural. n110

The court characterized the exposure to defendant's product as "akin to saying that one who pours a bucket of water into the ocean has substantially contributed to the ocean's volume." n111

Thus, these two opinions are good illustrations of the need for a court to consider the legal significance of the any exposure theory before accepting it. Neither of these appellate opinions could square the experts' assertion that every exposure is substantial with the plaintiff's requirement to prove the exposure was a substantial factor. Essentially, the theory would remove the word "substantial" from the legal test.

The Moeller opinion also illustrates the necessity of looking behind the experts' statements and examining what they rely on for support. The court refused to rely on the parties' briefs or mere expert assertions: "Briefs are no substitute for the record itself, and after conducting our own careful review of the record, including the testimony of each expert, we must conclude that the Plaintiff failed to prove that Garlock's product was a substantial factor in bringing about the harm." n112 This is classic Daubert/Frye analysis and a necessary part of the gatekeeping function.

The Moeller court also addressed the incorrect framing of the issue by pointing out that the plaintiff had experienced amphibole insulation exposure [*25] "thousands of times greater" than any trivial exposure from gaskets. n113 Correctly framed, the question the experts should have asked and answered was whether in the face of such overwhelming, alternative exposures, could minor gasket exposures be a substantial factor? They did not so frame the issue but instead assumed small exposures were causative regardless of other exposures. The court reframed the issue correctly by pointing out that a bucket of water does not substantially contribute to the ocean. n114 Stated correctly in the context of the case, the experts' each and every exposure proposition made no sense, and this court of appeals accordingly rejected it.

No other federal appellate courts have issued any exposure opinions in the asbestos context, although one such case is presently pending before the Ninth Circuit Court of Appeals and is already briefed and argued. n115 The plaintiff in that case claimed some exposure at unknown levels to asbestos woven and bound in dryer felts used in a paper mill, an assertion supported by any exposure testimony from plaintiff expert Dr. Carl Brodtkin. n116 Even though Washington state courts had twice previously rejected any exposure testimony, including that of Dr. Brodtkin himself, n117 the Eastern District of Washington trial court performed a hands-off review and admitted the testimony. n118 The court's final determination, relying primarily on a handful of cases admitting any exposure testimony and Dr. Brodtkin's own statements, illustrates an open-door review not typically acceptable for the gatekeeper role in a toxic tort case:

There is obviously a strong divide among both scientists and courts on whether such expert testimony is relevant to asbestos-related cases. In the interest of allowing each party to try its case to the jury, the Court deems admissible expert testimony that every exposure can cause an asbestos-related disease. n119

The trial judge addressed only two of the many articles Dr. Brodtkin cited and purportedly relied on - rejecting one of them as an unsuitable legal brief and only minimally discussing the other. n120 Whether the Ninth Circuit will support [*26] this sort of analysis under Daubert will likely be determined in the appellate court's upcoming ruling. Given the careful Daubert review the Ninth Circuit requires, as discussed above in section II.B, it would seem appropriate for the appellate court to require greater scrutiny of the any exposure theory in Barabin.

B. Pennsylvania

Until May 2012, Pennsylvania had been a battleground state when it came to the any exposure theory. That state's supreme court settled the issue in May, in the *Betz v. Pneumo-Abex* case, by soundly rejecting the any exposure theory in a unanimous opinion. n121

The seminal opinion Pennsylvania trial court opinion in *In re Toxic Substances* helped jump start the movement against this theory in 2006. n122 Plaintiffs teed up the any exposure theory in what later became the *Betz* case as a test case. Plaintiffs agreed that the exposure of the individual plaintiff in that case (he was a forty-year automobile mechanic) were not relevant because plaintiffs' experts believe all exposures were causation - one brake job was no different than many years of brake work in this regard. n123 More directly, plaintiffs had asserted under the any exposure theory that the plaintiffs, and anyone else who had a single, or "vanishingly small" exposure to asbestos-containing products, could each claim such exposure was responsible for their disease. n124 In response, defendants filed motions challenging the admissibility of the any exposure theory under Pennsylvania's Frye standard. n125 After a three-day hearing, the

trial judge, Robert Colville, excluded the testimony. n126 Judge Colville's opinion remains today one of the best articulated critiques of the logical holes and scientific flaws in the any exposure theory.

Following the Colville opinion, in 2007, the Pennsylvania Supreme Court had commented (in dicta in a different case) that the any exposure theory was a "fiction" that could not substitute for causation evidence. n127 The intermediate appellate court had also previously criticized the notion that a single brake job could be considered causative of anything:

Dr. Gelfand's statement saying every breath is a "substantial contributing factor" is not accurate. If someone walks past a mechanic changing brakes, he or she is exposed to asbestos. If that [*27] person worked for thirty years at an asbestos factory making lagging, it can hardly be said that the one whiff of the asbestos from the brakes is a "substantial" factor in causing disease. n128

Several other Pennsylvania trial courts had both rejected and accepted any exposure testimony. n129 In 2010, however, a majority of the intermediate court of appeals reversed Judge Colville's original order and accepted any exposure testimony. n130 This ruling was in seeming conflict with the Pennsylvania Supreme Court's pronouncement in Gregg that the any exposure theory would not support causation. n131 The appellate court found that the trial judge had abused his discretion by independently analyzing the flaws in the any exposure theory, and cited to numerous "small bridges" relied on by plaintiffs' expert Dr. John Maddox as sufficient to supply sufficient causation evidence to get to a jury in the minimal dose case. n132 The Pennsylvania Supreme Court accepted review and issued its opinion reversing the appellate court (and reinstating Judge Colville's opinion) in May 2012. n133 Because this is the most recent opinion from the highest court in a state, we review it in some detail here.

In a unanimous decision, the Pennsylvania Supreme Court agreed with the criticisms set forth by Judge Colville. The court concluded a Frye analysis was appropriate because of the unusual nature of Dr. Maddox's opinion: "We conclude that a Frye hearing is warranted when a trial judge has articulable grounds to believe that an expert witness has not applied accepted scientific methodology in a conventional fashion in reaching his or her conclusions." n134 It was appropriate for Judge Colville to rigorously scrutinize the illogical suggestion that even the smallest of occupational exposures could cause disease, yet asbestos fibers in the ambient air are not causative, regardless of overall lifetime dose. n135 The court noted that Dr. Maddox's reliance on case reports, animal studies, and regulatory pronouncements provided an unreliable basis for a causation opinion. n136 Further inconsistencies in the any exposure theory included Dr. Maddox's admission that individual exposures differ in the potency of fiber types, intensity of exposures, and the duration of exposures. The any exposure opinion fails to consider the different nature of these exposures, even though Dr. Maddox admitted these factors need to be considered when estimating the effects of different exposures. n137

[*28] The court also dissected one of the primary grounds asserted by plaintiff experts to justify their any exposure opinions - the dose-response curve. Dr. Maddox testified that asbestos disease is based on such a curve, under which high exposures are more likely to cause disease than low exposures. n138 The court took issue, however, with the inconsistency between Dr. Maddox's admission that not all doses pose the same risk and his refusal to identify how much dose would pose so little risk as to be inconsequential in causation: "Simply put, one cannot simultaneously maintain that a single fiber among millions is substantially causative, while also conceding that a disease is dose responsive." n139 The court restated its position in Gregg, that the any exposure theory is a "fiction" that would subject defendants to full joint and several liability for injuries, even in cases where exposure to a defendant's product could be classified as minimal in relation to other exposures. n140

As a result, the Pennsylvania Supreme Court has cleared up the confusion created by the Superior Court. The any exposure theory is no longer viable in Pennsylvania courts. The Pennsylvania decision is extremely significant. It continues the trend established by numerous other federal and state courts in rejecting this testimony, and resolved the conflict in Pennsylvania created by the intermediate court Betz opinion.

C. Texas

In 2007, the Texas Supreme Court rejected testimony in an asbestos case in which the plaintiff experts failed to assess the dose received over a lifetime of brake mechanic work. n141 In reality, the Borg-Warner court did nothing more than apply standard toxic tort causation principles to an asbestos case. Asbestos litigants, however, had become so inured to

lax asbestos rules before some Texas courts that the Borg-Warner ruling represented a dramatic change in Texas asbestos law.

Since Borg-Warner, Texas appellate courts have applied that court's reasoning three times to reject any exposure testimony in related cases. The first, *Georgia-Pacific Corp. v. Stephens*, extended the asbestosis ruling in Borg-Warner to mesothelioma cases and, in the process, rejected the notion that mesothelioma is a doseless disease that is somehow exempt from the dose rule of toxicology. n142 Plaintiffs must still demonstrate a dose sufficient to cause mesothelioma. The most recent Texas decision, *Georgia-Pacific v. Bostic*, n143 essentially applied the rule of Borg-Warner to a ten-year potential joint compound exposure, holding that Borg-Warner rejected the any exposure [*29] approach or other failure to assess the dose. n144

In between these two opinions is a more significant ruling. In *Smith v. Kelly-Moore Paint Company, Inc.*, the Texas Court of Appeals, like the Stephens court, first rejected the contention that mesothelioma cases are different and should not require any proof of dose. n145 After clearing this first hurdle, this court engaged in one of the most careful examinations of the plaintiffs' best attempt to prove that mesothelioma is caused by low doses of chrysotile asbestos - the kind utilized in the joint compound, gaskets, and brakes that make up the bulk of asbestos litigation today. Even though plaintiff experts made all the usual assertions, the court correctly determined that none of these statements or articles relied on by the experts actually identified what dose of chrysotile asbestos would be required to cause mesothelioma because there is no known safe dose of asbestos, even chrysotile is known to cause asbestos, and mesothelioma is a signature and cumulative disease. n146

The key to the Smith opinion is that the court first framed the issue properly - whether low doses of chrysotile are known to cause mesothelioma - and then reviewed the studies to see if they supported the correct proposition. When the court reviewed the studies it found that two of the key studies cited by the expert (Iwatsubo and Rodelsperger) did not even address chrysotile exposures and thus were useless in determining a chrysotile threshold:

The literature upon which Dr. Maddox relied is inconclusive regarding the effect of exposure to only chrysotile fibers ... The studies showing an increased incidence of mesothelioma in these populations did not attempt to extrapolate any minimum dose of chrysotile to which these populations were exposed... . And a study by Iwatsubo showing a four-fold increase of mesothelioma at an exposure level of .5 fibers/cc, and a study by Rodelsperger showing a 7.9 odds ratio of an increased risk of mesothelioma at cumulative exposures between 0.0 and .15 fibers/cc year, both fail to provide the minimum dose evidence required under Borg-Warner: neither study differentiates among fiber types. n147

By framing the issue properly, requiring evidence of chrysotile causation at low doses and then determining whether the cited studies supported that proposition, the Smith court did exactly what is required under any kind of appropriate court review of scientific evidence. n148 The contrast between this approach and that of the federal MDL and other opinions admitting any exposure testimony (discussed below) could not be more stark. The contrast is one of court methodology and rigor, not the quality of the science.

[*30]

D. Georgia

Georgia has joined Texas and the Sixth Circuit in rejecting any exposure testimony in asbestos litigation. In *Butler v. Union Carbide Corporation*, the Court of Appeals of Georgia affirmed the trial court's decision to discount the plaintiffs' experts' opinions by finding them scientifically unsound. n149 Mr. Butler worked with asbestos-containing phenolic molding materials at Union Carbide for about eight years, but only about eight days of that time involved the use of Union Carbide's product. n150 Plaintiffs nevertheless argued that any exposure to this material was causative. n151 They contended that Dr. Maddox's any exposure opinion was "based on reliable science" and is "widely accepted," and that it was "premised upon his reliance upon scientific literature." n152 In several of the cases admitting this expert testimony, this is all it took - the courts accepted such representations at face value.

In *Butler*, however, the appellate court did not accept these propositions at face value and instead found, as the Texas court did in *Smith*, that Dr. Maddox was attempting to rely on studies that were not even relevant to the proposition in the case - whether minimal doses of chrysotile asbestos could cause mesothelioma. n153 This court, like the *Smith* court, rejected the Iwatsubo study because it did not address chrysotile exposures. n154 The court also rejected reliance on the other critical foundation of the any exposure lynchpin, the so-called Helsinki "criteria," because that

document did not address which components of a cumulative exposure could be considered causative. n155 Thus, the Helsinki guidance at best presents the bucket in the ocean problem - it is not enough to say that everything is cumulative, without these experts identifying which exposures are sufficient to be considered a substantial factor in causing disease. n156

Expert testimony will nearly always be admitted if the courts simply accept the experts' view of their own literature and testimony. At least in the any exposure world, however, the recent Texas and Georgia opinions demonstrate that these studies or articles do not support the proffered testimony.

E. Other Trial Court Opinions Rejecting the Any Exposure Theory

Several trial courts around the country have rejected any exposure [*31] testimony in the last three years, but those opinions have not reached the appellate courts. In Miami, a circuit court judge granted a directed verdict to the defense in a brake exposure case after plaintiffs' expert, Dr. Arthur Frank, relied on the any exposure theory in lieu of a careful identification of defendant's products and degree of exposure from them n157

Dr. Frank's testimony was insufficient as a matter of law, because his theory that "any exposure above background" could cause mesothelioma would eviscerate the standard established by Florida law, to wit, a substantial contributing factor. Dr. Frank's testimony appears to disregard the Legislature's specific inclusion of the word "substantial" and treats all exposures as the same. n158

Likewise, in Jones County, Mississippi, the magistrate and trial judge in a recent case rejected the testimony of the medical causation experts who relied on the any exposure theory instead of assessing and proving a harmful dose. n159 The court correctly noted that "to fulfill the Court's role as a gatekeeper, there must be an analysis of the proof underlying each Defendants' exposure history ... It is not enough to take the affidavits and deposition testimony at face value; instead one must examine the factual basis for the opinion." n160 For purposes of asbestos causation, Mississippi is a "Lohrmann" state requiring proof of "frequent, regular, and proximate" exposure to defendant's product. n161 The magistrate in the Nix case contrasted the any exposure theory with this standard:

In contrast to the language of the rule in [Lohrmann], Plaintiff's experts have articulated their own theory of causation, based on two (2) factors or theories: (a) the "any exposure over background" theory, and (b) the "cumulative dose" theory. The two go hand-in-hand. Paraphrasing, Plaintiff's experts' theory goes like this: if Plaintiff was exposed to a product of a particular defendant, and that experience resulted in exposure to fibers from the defendants' asbestos-containing products (ACPs) - in any amount above an average background level - that exposure contributed to the Plaintiff's cumulative dose of asbestos. n162

The court recognized that there is a fundamental conflict between the "frequent, regular, and proximate" standard and testimony that every single [*32] workplace exposure is causative:

I find that Plaintiffs' experts Raterman, Haber and Hammar should be precluded from espousing the "any exposure above background" theory at trial, as it is contrary to the "frequency, regularity and proximity" test established by our Supreme Court. n163

These opinions thus illustrate both the need to examine the literature and expert claims and also to consider the legal sufficiency of the any exposure theory. The any exposure theory, by capturing in its net irregular and infrequent exposures, is no more consistent with the Lohrmann standard than it is with a substantial factor test.

Three trial courts in Louisiana rejected any exposure testimony in 2009 and 2010. In *Degrass v. Anco Insulations*, the Civil District Court for the Parish of Orleans granted a motion to exclude the any exposure opinions of Dr. Jacques Legier, but offered no analysis. n164 In 2011, in *Robertson v. Ashby*, the Parish of East Baton Rouge excluded frequent plaintiffs' expert Dr. Eugene Mark from testifying to the any exposure theory. n165 In granting the defendant's motion, the Court stated,

There's no foundation for this expert to offer such an opinion. He has no epidemiology study to rely upon, he does not know what the dose would have been as to any particular defendant. n166

Finally, in a take-home peritoneal mesothelioma case, *Bello v. ANCO Insulations*, the trial court was dissatisfied with plaintiff experts' failure to understand the actual work experience of plaintiff's husband or attempt to assess how exposures below the OSHA standard would have caused a take-home disease. n167 The court ruled for defendants after a bench trial.

The effect of this trio of decisions is somewhat uncertain after the *Robertson* case went up on appeal - the appellate court reversed the decision for defendants, largely because the defendants did not challenge the any exposure theory in that case on summary judgment but only offered a "product identification" defense. n168 The appellate court also reversed a later decision by the trial judge to strike the any exposure testimony of Dr. Eugene Mark, but [*33] held only that the court did not conduct a proper examination under *Daubert* pursuant to Louisiana statutory requirements. n169 In particular, it appears the defendant did not submit "testimony, affidavits, or other admissible evidence to contradict or question the reliability of any of the statements contained in Dr. Mark's affidavit." n170 *Robertson* was thus reversed, but on grounds that permit the issue to be revisited on remand and in future cases. n171

F. Courts Declining to Reject Any Exposure Testimony

After a string of resounding rejections between 2005 and 2008, the any exposure theory or other low-dose speculative testimony has rebounded, somewhat, in certain jurisdictions, although the ultimate outcome in some of these is still uncertain. These opinions are consistently flawed - they regularly accept the ipse dixit of the experts in substitution for a critical examination of the logic and support for the theory.

1. The Federal Multi-District Litigation Docket

The any exposure theory has gained a foothold in the federal asbestos MDL proceedings before the United States District Court for the Eastern District of Pennsylvania. The first such opinion, in November 2010, n172 consisted of five pages in which the court repeatedly cited only to the experts' own self-serving statements to support the court's determination that any exposure testimony was reliable (e.g., Dr. Maddox will testify that his conclusions represent those "generally accepted by the medical community.") n173 There was no examination whatsoever as to whether these statements were accurate or had any scientific support. n174 This is classic error under *Joiner*, which expressly forbids courts from admitting testimony based on the ipse dixit of the expert.

Nevertheless, this initial opinion set the stage for several cases that followed, each of them resulting in opinions permitting any exposure experts [*34] to testify or support causation. n175 In the subsequent *Schumacher* opinion, for instance, the magistrate again referenced mere citations to the experts' own descriptions of their methodology, as if such citation satisfied the tenets of *Daubert*. In both of these prior cases, Judge Robreno's decisions emphasized that the experts had relied on a "variety of peer-reviewed studies and reports to form [their] ultimate opinions ... Dr. Maddox relied on numerous published studies and reports, drawing from the fields of pathology, radiology, epidemiology, and industrial hygienics." n176 The court in the second case, *Larson*, actually called the any exposure theory "shaky," but still deemed "shaky" evidence sufficient under *Daubert*. n177 This opinion, like the previous ones, engaged in a series of citations to plaintiffs' claimed testimony without any examination of the legitimacy of the testimony. The thinness of review is illustrated by the following:

. The court justified its decision because "Dr. Brody relied on one hundred and forty-six peer-reviewed publications of which he is an author or co-author." This a common technique of these experts - citing to a long list of articles to create the impression of scientific rigor, when the studies do not actually support the proposition at issue in the case. The judge apparently did not review or investigate any of the studies to determine their "fit" under *Daubert* and *Joiner*. n178

. The court found that the opinion was admissible because one expert relied on "epidemiology studies" such as the Helsinki Criteria and a review of peritoneal mesothelioma. n179 The Helsinki Criteria not only does not state that every exposure is a cause, n180 it is not an epidemiology study at all, as even the most cursory of examinations would have shown. The court provided no comment whatsoever regarding the review article and whether it supported the experts' opinion - it was enough that the expert "relied on" epidemiology studies. n181

The most extended discussion of the theory is in the third in the series, *Anderson v. Saberhagen Holdings*, by the magistrate, who again relied on the [*35] trial judge's prior rulings to admit any exposure testimony. n182 Yet even this opinion fails to include any critical analysis of the experts' point, instead merely citing to them without serious examination. As examples:

. The court allowed Dr. Hammar to cite to two amphibole studies, Iwatsubo and Rodelsberger, for the proposition that low exposures to chrysotile fibers would cause disease. n183 This is classic framing error - both studies address the effect of amphibole fiber exposure but explicitly disavow that their findings address chrysotile exposures. The case was not about amphibole exposures (the alleged exposure involved chrysotile), and these studies thus do not satisfy the fit standard of Daubert. n184 The courts of Texas and Georgia, among others, who have actually examined these studies, have found them irrelevant to low dose chrysotile cases. n185

. Dr. Hammar opined that he can legitimately contend that each exposure is a cause because every individual is different, with different susceptibilities. Thus, no one could ever exclude the possibility that small amounts might cause disease in certain persons. n186 Dr. Hammar cited to no published articles stating that susceptibility meant even the smallest exposure is causative, and the court cited to no other opinions allowing limitless causation testimony based on individual susceptibility. n187

[*36] . The court admitted the any exposure testimony because asbestos disease is cumulative. n188 But the court failed to ask the pertinent question - should all exposures then be considered part of the cumulative dose, or only those that actually contributed something significant? The bucket in the ocean analysis of the Sixth Circuit in the Moeller opinion above illustrates the flaw in this argument and the court's holding.

. The court cited to the typical any exposure fall-back - the "no known safe dose" statements of a number of government organizations as proof that any exposure should be considered causative. n189 This is flawed logic - the lack of certainty about where the threshold lies does not mean there is no level that is not causative. Plaintiffs still must prove a causative dose. The reliance on conservative regulatory statements is also not suitable for court causation testimony. n190

As this set of MDL opinions demonstrates, plaintiffs repeatedly attempt to prove their any exposure theory with a great deal of non-proof. Because no one knows what the safe dose is, their argument is that all doses must be causative. No one knows which fiber causes the tumor so all fibers breathed in (except, oddly, background fibers) must be considered causative. The theory is hypothetical, and no peer-reviewed article actually makes such a claim, but the expert will say it is supported by the scientific community. The experts have no epidemiology studies showing excess mesothelioma from low doses of chrysotile, but that does not matter because epidemiology, according to these experts, is not required. The opinions include statements such as "there is a debate in the scientific community," or "it is the jury's province to weigh the evidence," or "the defendant's arguments are fodder for cross-examination" - all of which are evidence of a court failure to tackle the evidence.

These types of statements by courts are signals reflecting an approach inconsistent with the standards articulated by the Ninth Circuit and other courts. These propositions are those of courts who are not using the correct methodology because they are stepping away from the hard, but mandatory, brave new world of Daubert and allowing the experts, not the courts, to dictate what evidence goes to a jury.

It is fascinating that none of the now five written opinions coming out of the federal MDL cite to or analyze a single court opinion rejecting the any exposure theory in asbestos litigation (more than twenty of them in the last six years). n191 One would think that this federal court, at a minimum, would [*37] closely examine the three opinions of the only federal circuit court to rule on any exposure testimony - the Sixth Circuit - and question why its own opinions are so out of sync with that appellate court's views. Nor do any of the federal MDL opinions address how an each and every exposure approach could be consistent with the applicable substantial factor test, the lynchpin of the Sixth Circuit rejections of the theory. The federal MDL opinions to date have not resulted in any appeals to the Third Circuit so it remains to be seen whether that court's acceptance of the theory will survive appellate review.

2. California

California continues to produce lower court asbestos causation opinions that distort the standard enunciated in the landmark California Supreme Court decision of *Rutherford v. Owens-Illinois, Inc.* n192 The Rutherford court stated the usual "substantial factor" test, but then proceeded to articulate that standard in two different ways. The first articulation is fairly prosaic and similar to other states: Plaintiff must "establish some threshold exposure to the defendant's defec-

tive asbestos-containing products, and must further establish in reasonable medical probability that a particular exposure or series of exposures was a "legal cause" of his injury, i.e. a substantial factor in bringing about the injury." n193 In the second articulation, however, the court interjected the notion of risk: asbestos plaintiffs must prove the defendants' product "was a substantial factor contributing to ... [the] risk of developing cancer." n194 Risk is not the same thing as causation. This unfortunate phrasing has allowed trial and appellate courts to approve of any exposure testimony on the ground that each and every exposure theoretically increases the risk of disease in some small and unquantified increment.

After this ruling, plaintiffs have succeeded in presenting any exposure testimony in several California appellate rulings. n195 These courts are allowing cases to go forward without any proof that the dose was sufficient to be causative. Thus, currently in California it is extremely difficult to escape the threat of a jury trial if plaintiff worked with or around an asbestos-containing product, no matter how minimal the exposure. n196 This is precisely the type of [*38] scientific evidence the Daubert court - and, ironically, the Ninth Circuit Court of Appeals which hears California federal cases - tried to prevent in outlining the factors to consider when evaluating evidence.

The California appellate courts in the above decisions have apparently not understood or acknowledged the inherent conflict between Rutherford and any exposure testimony. Rutherford rejected that plaintiff's attempt to shift the burden of proof of substantial factor causation to defendants. But that is exactly what the any exposure theory does. Under Rutherford, plaintiff must take two steps to prove a case - the first step is exposure, proving that plaintiff encountered some amount of defendants' product; the second step is proving that the exposure produced by each defendant's product reached some level of substantiality to suffice as a "legal" cause of the disease. n197 The any exposure theory allows plaintiffs to skip the second step. Several statements in Rutherford make it clear that an any exposure approach is a violation of the Court's proscribed standard:

. Plaintiff must show that "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 disease." n198 This language presumes that some occupational exposures are not significant enough to be the cause of disease and requires plaintiff to distinguish between them.

. The court noted the need to consider "frequency, proximity and intensity of exposure" and "the peculiar properties of the individual product." n199 Also "asbestos products have widely divergent toxicities... all asbestos suppliers did not fire the same shot." n200 Under this language, plaintiff experts cannot ignore the much lower potency of chrysotile and assume that all exposures are equally causative.

. The fundamental disagreement in Rutherford on burden of proof is "which exposures to asbestos-containing products contributed significantly enough to the total occupational dose to be considered "substantial factors" in causing the disease." n201 The Rutherford court held that the burden stays with plaintiff - plaintiff must distinguish which are significant and which are not.

. The any exposure theory creates the exact situation the court criticized:

. "[The burden shifting approach] would require every joined [*39] defendant to exonerate itself upon nothing more than plaintiffs' showing of exposure to defendants' asbestos products, some of which may have caused harm." n202

Resolution of the conflict between Rutherford and any exposure testimony will at some point require an appeal to the California Supreme Court. In the interim, some trial courts have been granting such motions but most have not. n203

3. New Jersey

The New Jersey intermediate appellate court accepted the equivalent of any exposure testimony in *Buttitta v. Allied Signal, Inc.*, but did so without examining the fundamental underpinning of the expert testimony supporting the court's ruling. *Buttitta* was another brake case, but one in which the plaintiff never repaired anything - he merely handled boxes with parts in a warehouse for three summers. n204 The court nevertheless did not require the experts to determine whether handling parts produced enough a dose to be a real cause of disease. n205 Instead, the court relied on the testimony of plaintiff medical experts that mesothelioma (unlike asbestosis or lung cancer) can "develop after only minor exposures to asbestos fibers." n206 Nowhere does the court challenge that statement, or even examine on what basis the expert derived that opinion - the court accepted the statement as fact, even though it is nothing more than the *ipse dixit* of this expert. n207 The *Buttitta* opinion is lengthy and contains substantial discussion, but the ruling ultimately turns on nothing more the determination that the courts of New Jersey will not require dose evidence in a mesothelioma case if a

plaintiff expert contends that the disease only requires minor exposures. n208 This opinion seems to be heavily colored by the tendency of some courts to create different causation rules for [*40] asbestos.

The future of the any exposure theory in asbestos litigation appears to be headed toward a docket-by-docket resolution, with a strong tilt so far toward rejection of the theory in most jurisdictions. Presumably, as asbestos litigation extends into more and more extreme exposure allegations (e.g., the mere presence in a building containing asbestos insulation), the theory will become even more difficult to accept even in jurisdictions favoring lax asbestos rules. If courts can be persuaded to perform a properly rigorous review, the theory should die out altogether.

4. Other State Appellate Courts:

The Supreme Court of Kentucky recently affirmed the use of the any exposure theory, but in a context that actually favored defendants. n209 The ruling was the result of a series of trials and appeals focusing on the question of what proof is necessary to include an "empty chair" defendant in the verdict. Ultimately, the Kentucky court held defendants to the same low bar to which plaintiffs were held in that case: the any exposure bar. The result was that the verdict was overturned because the jury should have attributed some fault to each company in the same position as the remaining defendants - if any exposure is good enough for plaintiffs, it should also be for defendants. n210 Apparently in this case, the defense did not challenge or controvert the any exposure approach. It remains to be seen whether this theory would survive a serious challenge under Kentucky law in a case where defendants challenge the theory. The Sixth Circuit Court of Appeals has already held, twice, under Kentucky law that the theory cannot suffice for substantial factor causation. n211

Maryland likewise has seen rulings in recent months that tend to support, if not true any exposure testimony, at least low dose testimony without the need to assess an actual causative dose. Maryland, however, adheres to the Lohrmann test and thus these appeals were challenges to whether the exposures were sufficiently proximate, regular and frequent to merit trial. n212 The intermediate appellate court in John Crane concluded that testimony about dust in the air when using the products at issue, combined with general medical expert testimony that every fiber above background was sufficient to be a substantial factor in the cause of mesothelioma, was sufficient to prove causation. In the Scapa case, the intermediate appellate court affirmed expert testimony that one year's worth of work around potentially asbestos-containing dryer felts was sufficient evidence, without assessing what the dose from that work might have been. n213 The Maryland Court of Appeals (the highest court) affirmed Scapa, but in less compelling language, by stating "our holding on [*41] this sufficiency of evidence question is not as emphatically stated as the Court of Special Appeals' holding because we conclude that the evidence was sufficient to survive the motions, but decline to state that the evidence "conclusively established' proximity as a matter of law." n214 The Court's approach is very ad hoc:

"There is more evidence in the instant case than there was in Reiter, that Scapa's asbestos-containing dryer felt [produced regular and frequent exposures] The inferences that were found too speculative in Reiter n215 do not arise in this case because of the amount of testimonial and circumstantial evidence placing the asbestos-containing dryer felts within arm's length of Mr. Saville's work-site." n216

Thus, the any exposure theory was not directly at issue in these appeals and they do not serve as acceptances of that theory. Maryland will apparently continue to take a case-by-case, "we'll know it when we see it" approach to the sufficiency of exposure evidence in asbestos cases, and will compare the exposure testimony to the Lohrmann standard rather than substantial factor causation. Arguably, any exposure should not suffice even under Lohrmann - a case brought solely on the basis of any exposure testimony, for an exposure that is not regular and frequent, should be rejected under Scapa.

IV. COURT TREATMENT OF THE "ANY EXPOSURE" THEORY IN OTHER AREAS OF TOXIC TORTS

While the battle rages over the any exposure theory in asbestos litigation, it has made virtually no inroads into other toxic tort litigation despite increasing efforts of plaintiff testifying experts to export it there. This is nevertheless a very important front in tort jurisprudence. The attempts to eliminate the need for any kind of dose assessment in toxic tort cases would represent a radical departure from current law. If successful, these efforts would open the doors to an invasion of an unscientific and litigation-drive theory into heretofore untapped geography.

The any exposure theory is not by logic limited only to asbestos exposures. Asbestos is a carcinogen, one that causes disease through the accumulation of dose over the years and develops into cancer only after a long latency. This makes asbestos pretty much the same as dozens of other known carcinogens like tobacco smoke, benzene, vinyl chloride, and radiation.

The exact mechanism under which asbestos fibers induce a cell to go haywire is also not dramatically different from other carcinogens, although that mechanism is not entirely understood. It is believed to be either the result of inflammation or disruption of the cell genetics or division, both common mechanisms for many carcinogens. And, also like other carcinogens, asbestos [*42] is not the only cause of the tumors it induces. Lung cancer is a known result of high exposure to asbestos. But smoking is by far the leading cause of lung cancers, and approximately ten percent of lung cancers occur as a result of other causes, or are idiopathic or have no known cause. n217 Mesothelioma, likewise, is caused by exposure to asbestos. But numerous studies have documented an increased incidence of pleural mesothelioma after childhood or early exposures to high levels of radiation, and there are other suspected causes. n218

More critically, upwards of 300 cases of mesothelioma occur each year that have no known association with significant asbestos exposures - these cases are considered idiopathic. n219 Many of these idiopathic cases are believed to be the result of the body's own production of aberrant cells, compounded by aging and decline of our defense system against cancerous cells. They are thus spontaneously-induced and have nothing to do with asbestos or any other outside cause. These idiopathic cases are an increasingly active target of litigation, however, through the magic of the any exposure theory - even cases that would never have been attributed to asbestos in the past are today called, in circular fashion, asbestos-induced mesotheliomas as long as the testifying plaintiff experts can find some modicum of asbestos contact in the person's life.

Since asbestos is not much different than any other carcinogen, one would think the any exposure theory - if it had any scientific validity - would have enveloped the world of toxic tort litigation much as it has asbestos litigation. That is most certainly not the case. Only recently have the asbestos and other experts made any concerted effort to assert this theory in non-asbestos litigation. Almost without exception, those attempts have been rebuffed. Thus, it appears, at least for the moment, that the any exposure theory is largely an artifact and earmark of asbestos litigation. Courts nevertheless need to understand this theory, as it has developed in asbestos, in order to address its inevitable assertion in other contexts.

A. The Non-Asbestos Litigation Targets for the Any Exposure Theory

Plaintiffs have chiefly targeted benzene litigation for early attempts at expanding the any exposure theory beyond asbestos litigation. As noted above, benzene is a known human carcinogen; however, epidemiology studies have firmly linked only one kind of cancer (AML) to benzene exposures and primarily in the context typically of high exposures to pure benzene in factory [*43] settings. Benzene at low levels is in fact ubiquitous, because it can be found in most urban air samples and is part of vehicle exhaust and cigarette smoke. Anyone who pumps self-service gasoline inhales small amounts of benzene. n220 The studies that do document AML from benzene exposures find these diseases at higher levels of exposure, between fifty and five hundred ppm-years. The studies are less conclusive when considering minor exposures, but none of the studies support the no safe level or no threshold model for showing unreasonable risk. n221

Nevertheless, much recent litigation has focused on low and unquantified levels of benzene exposure, typically through the use of cleaning solvents or as a result of exposure to gasoline, which contains small amounts of benzene. In *Parker v. Mobil Oil Co.*, plaintiff experts attributed a form of leukemia to a gas station attendant's handling of gasoline products over several years. In lieu of any attempt to identify the dose of benzene and whether it reached levels sufficient to cause AML, these experts opted for "qualitative" assessments - his exposures were "frequent", "excessive," or "extensive." n222 In *Pluck v. BP Oil Pipeline Co.*, benzene found in well water at levels well below the EPA drinking water standard was blamed as the cause for a non-hodgkins lymphoma, which is not even a type of cancer clearly associated with benzene. n223 In *Baker v. Chevron*, plaintiffs claimed that several diseases, including one AML, were caused by air emissions from a nearby refinery producing, at the most, "extremely low doses." n224 In *Blanchard v. Goodyear Tire and Rubber Co.*, plaintiff blamed his non-Hodgkin's lymphoma on playing on a field near a plant that may have released benzene, again with no attempt to identify or define any dose. n225 In *Henricksen v. ConocoPhillips Co.*, a plaintiff truck driver claimed that his work loading and unloading gasoline caused his AML, again without any attempt to assess the dose or compare it to known hazardous levels. n226

Most of the above cases have in common the unwillingness of plaintiffs' experts to engage in any dose assessment. As a substitute, they assert that benzene is a carcinogen and it only takes "one hit" to cause cancer (Baker), that there is "no safe level of benzene in terms of causing cancer (Pluck), or that the mere presence of benzene is sufficient to cause cancer (Blanchard). These cases also have another feature in common - the experts are more reluctant than in asbestos litigation to declare that they are relying on an every exposure theory, even though that is clearly what they are doing. They seem to recognize that outside of certain asbestos courts, the theory is unpalatable on its face.

[*44] Some experts have attempted to bring the any exposure theory into cases other than benzene litigation, but those instances are still fairly rare. In one unique case, the plaintiff in a Florida matter claimed that using fluo-ride-treated denture cream for eight years caused an array of neurological symptoms. n227 Plaintiffs conceded that the cream used in modest amounts was probably safe, much as the asbestos experts agree background exposures do not cause mesothelioma. n228 The court recognized the inconsistency in agreeing that some doses are not causative, but then refusing to estimate or establish that plaintiff received a causative dose: "Neither Plaintiffs' experts nor the articles on which they rely determine how much Fixodent must be used for how long to increase the risk of a copper-deficiency" n229 Likewise, the Arkansas Supreme Court recently faced a claim that diesel fumes and other railroad exposures caused a worker's multiple myeloma. n230 The experts declined to assess the dose other than to claim, in qualitative terms, that it was "substantial and extreme" and "excessive." n231 The Eastern District of Washington federal court dealt with an attempt by a well-known asbestos expert to assert that virtually any exposure to diacetyl in popcorn would be sufficient to cause a bronchial disease known as bronchiolitis obliterans. n232 This is one of the "popcorn lung" cases that were filed a few years ago. As in asbestos cases, the expert claimed that background exposures to diacetyl were not harmful, but that there was no known safe dose and therefore plaintiff's home exposures to popcorn were causative. n233

Groundwater and medical monitoring cases are also an attractive target for the any exposure theorists. Many substances are found in groundwater (and often drinking water as a result) at very low levels not known to be causative and usually well below environmental standards. n234 To turn these situations into litigation, the experts must assert that such low levels are causative, usually without attempting to quantify or assess any actual dose. By adding medical monitoring, the plaintiffs can extend such litigation to cases where no injury has even occurred - they insist that unquantified exposures to such substances, regardless of amounts, should justify medical monitoring for the future risk of disease.

B. Court Rejection of the Any Exposure Theory in Non-Asbestos Cases

Courts have almost uniformly rejected the above cases, typically on motions for summary judgment or to exclude the expert evidence. In the benzene world, for instance, most opinions have rejected any exposure [*45] testimony. The New York Court of Appeals decision in *Parker v. Mobil Oil* remains the seminal appellate opinion rejecting benzene causation absent a dose assessment. n235 In doing so, this court explicitly criticized plaintiffs' experts use of unquantified and rootless phrases like "excessive" or "frequent:"

Dr. Goldstein's general, subjective and conclusory assertion ... that Parker had "far more exposure to benzene than did the refinery workers in the epidemiological studies" is plainly insufficient to establish causation. It neither states the level of the refinery workers' exposure, nor specifies how Parker's exposure exceeded it, thus lacking in epidemiologic evidence to support the claim. n236

Phrases like this should trigger a court's radar - if used without a benchmark, they are often ways of hiding the lack of real scientific assessment behind the words. n237

The Sixth Circuit Court of Appeals in *Pluck* also rejected benzene any exposure testimony, much as it has similar asbestos testimony. n238 Without conducting any dose assessment, the expert opined nevertheless that benzene in well water was the cause of plaintiff's non-Hodgkin's Lymphoma. n239 The Sixth Circuit held, "it is well-settled that the mere existence of a toxin in the environment is insufficient to establish causation without proof that the level of exposure could cause the plaintiff's symptoms." n240 The Court rejected the expert's reliance on a "no safe dose theory" that "had been discredited by other courts as a basis for establishing specific causation." n241

The Washington federal district court's opinion in *Henricksen*, in which a truck driver was unloading gasoline, is probably the best analysis of why the theory is not viable in benzene litigation. n242 Plaintiffs tried to frame the issue as a benzene exposure case in order to bring into play the factory-setting epidemiology studies. n243 The district court,

however, recognized this as error, and correctly re-framed the issue as whether gasoline containing small amounts of benzene is a cause of the alleged disease. n244 The court then performed an extensive review of the literature rather than just citing to the experts' claims about the literature. The Washington court held that experts who "opine on specific causation must pay careful attention to the dose-response relationship" and the "amount of exposure the plaintiff allegedly suffered." n245 One of the experts in this case opined that even small exposures to benzene should be considered substantial risk factors for cancer. n246 The [*46] judge in excluding this line of testimony found that "the use of the no safe level [methodology] ... "flies in the face of the toxicological law of dose response, that is, that "the dose makes the poison'" n247 The judge ultimately found that such an opinion was "a hypothesis rather than science sufficiently reliable for causation ...," and therefore fails the Daubert test. n248

In another benzene case, the Southern District of Ohio focused on the lack of logic behind the notion that background exposures are not causative but small amounts of occupational exposure are. n249 This argument is a regular feature of asbestos any exposure testimony. The court found no logical distinction between background benzene and small occupational exposures: "Since benzene is ubiquitous, causation under the one-hit theory could not be established because it would be just as likely that ambient benzene was the cause of Plaintiffs' illnesses." n250 While an expert's opinions need not be unequivocally supported by epidemiological studies in order to be admissible under Daubert, in this case, the expert's opinions were based on a "scattershot of studies and articles which superficially touch on each of the illnesses at issue" and "no depth of opinion is developed in any of the selected references to any of the Plaintiffs' illnesses." n251 The Court further held that, most importantly, "none of [the] studies supports an opinion that benzene can cause the illnesses from which Plaintiffs suffer at the extremely low doses or exposures experienced in this case." n252 The only way a reviewing court can make such a determination is to conduct the review with sufficient rigor, including reading and understanding the studies and whether they support the experts' contentions.

One of the few benzene cases in which an any exposure theorist survived review is the Nebraska Supreme Court's ruling in *King v. Burlington Northern Santa Fe Railway Co.* n253 Both the trial and intermediate appellate courts had agreed on the exclusion of a well-known plaintiff asbestos expert who was attempting to introduce any exposure concepts into benzene litigation. The state supreme court reversed but never reached the any exposure theory itself - the court instead held that the trial court improperly required conclusive epidemiology studies to support the expert's opinion. n254 It remains to be seen whether this court would in fact support any exposure testimony if rejected by the trial court under what the Supreme Court considers a proper review.

For the most part, these courts are merely applying standard toxicology and causation principles regarding dose to causation analysis. As the Arkansas court held in the case involving diesel fumes: "Causation requires more than mere proof of exposure to above-ambient levels of the alleged toxin, and [*47] instead requires evidence of the levels of exposure that are hazardous to human beings generally, as well as the plaintiff's actual level of exposure to the defendant's toxic substance." n255 The Washington court that rejected the popcorn lung expert's speculative testimony similarly stated that "Dr. Egilman cites no other authority for the analytical step he takes from observing that there is no accepted 'safe' level of diacetyl exposure to concluding that even concentrations as low as 0.02 ppm are harmful. There is, then, no reliable methodology supporting Dr. Egilman's opinion" n256 Arbitrary selection of "causative" levels, as this expert engaged in, are usually smokescreens. The expert should be required to cite to epidemiology studies demonstrating that such levels are causative, or at least to other compelling evidence supporting the expert's claimed threshold exceedance.

Courts in groundwater and medical monitoring cases have also not reacted well to attempts to prove these cases using elements of the any exposure approach. In *Emerald Coast Utilities Auth. v. 3M Co.*, for instance, the plaintiff expert concluded that since there was "no safe level of exposure" to PFOA and related chemicals, she did not need to assess the actual dose or exposure but asserted instead that the mere presence of this substance in groundwater was harmful. n257 The expert acknowledged, however, that "both PFOA and PFOS are ubiquitous, and thus most people in this country have been exposed to them" n258 The court rejected this record as insufficient to support standing: "Notably missing from the Burns affidavit is any discussion of the concentrations of PFOA or PFOS in water or in the body that lead to the identified harmful effects." n259

Similarly, plaintiff experts attempted to rely on the "no known safe dose" approach as a substitute for a dose assessment in a case in Kentucky federal court alleging various diseases from exposure to TCE, vinyl chloride, and dioxins in groundwater and air. n260 The court rejected this approach, in part citing to and relying on the Eaton article discussed above:

The plaintiffs have presented neither supporting scientific evidence nor cases holding that the "no-safe-dose" theory is reliable. To the contrary, courts have opined that this principle is not an appropriate one on which to ground a specific causation opinion. See *McClain*, 401 F.3d 1233, 1242-43 ("O'Donnell offers no opinion about the dose of Metabolife that caused ischemic strokes in three plaintiffs and a heart attack in the other. He only said that any amount of Metabolife is too much, which clearly contradicts the principles of reliable methodology...."); *Cano v. Everest Mineral Corp.*, 362 F. Supp. 2d 814, 849 (W.D. Tex. 2005) ("Several courts have [*48] considered and rejected the use of the linear no-threshold model in the litigation context.") (citing cases); see also Eaton, at 34 ("Procedures commonly used in 'risk assessment' for the purposes of establishing public health guidelines that represent 'acceptable' exposure levels for large populations are often, in this author's opinion, of marginal relevance to estimating 'causation' in an individual."). n261

Reliance on the no-safe-dose assumption did not constitute reliable science: "The court finds that the 'no-safe-dose' theory is not a reliable methodology, and it rejects the plaintiffs' claim that said theory entitled their specific causation experts to pay so little attention to the level of exposure in the bellwether plaintiffs." n262 These rulings are consistent with commonly-accepted principles of dose and causation in non-asbestos litigation. n263

V. CONCLUSION

The low dose cases being filed in toxic tort and asbestos litigation today are emblematic of an increased scrutiny of any kind of chemical exposure in our society and an increasing and often irrational fear of even the smallest amounts of exposure to such materials. Good science has repeatedly demonstrated that most of those fears are unfounded, but they often persist in common mythology anyway. The any exposure theory, an outgrowth of this trend, has to date found affirmative acceptance only in a few asbestos jurisdictions. Yet it still presents a major hurdle to any future resolution of asbestos tort crisis. The theory is also by no means a dead issue in other toxic tort litigation. Defendants and American companies can expect to see these experts and others appearing in future matters involving a range of chemicals and product exposures.

Defense counsel need to do their homework to understand the experts' approach and dissect that approach and the literature it purportedly stands on. Courts, in turn, must do their jobs to ensure reliability by examining the theory closely, in the context of the asserted exposures in a particular case. No one is served by allowing meritless, minimal exposure cases to proceed to a jury when real and genuine gatekeeping would dictate otherwise. The any exposure theory does not need or deserve special favors or treatment by the courts, in asbestos cases or otherwise. On its own merits, and reviewed appropriately, the theory cannot satisfy the same standards of reliability and fit as applied to causation testimony in any toxic tort case.

Legal Topics:

For related research and practice materials, see the following legal topics:
 Environmental Law
 Hazardous Wastes & Toxic Substances
 Asbestos
 General Overview
 Environmental Law
 Litigation & Administrative Proceedings
 Toxic Torts
 Evidence
 Scientific Evidence
 Daubert Standard

FOOTNOTES:

n1. Research support for this article was provided in part by funding from the Coalition for Litigation Justice, Inc. The views expressed herein are solely those of the authors.

n2. Dose and exposure are not the same thing, and need to be distinguished to understand the any exposure theory. Exposure is a measure of the amount of material available for uptake by the human body (e.g., through breathing) at a given point in time. Dose measures the total impact of all exposures over time, and thus takes into account the frequency, duration, and extent of all exposures. See Joseph V. Rodricks, Reference Guide on Exposure Science, in Federal Judicial Center, Reference Manual on Scientific Evidence, 507 (West Group 3d ed. 2011) (1994). Unless a substance is an acute toxin, like cyanide gas, an exposure by itself usually does not have toxicological consequences. The cumulative effect of repeated, significant exposures to some substances over time, however, can produce disease if the overall dose is high enough. As a simple example, a single drink of

beer exposes the individual to a certain amount of alcohol that is not likely to produce any effect, but continuous drinking through an evening could result in a less salubrious outcome.

n3. The so-called "bystander" cases involve mesothelioma plaintiffs who may have been present in the vicinity of asbestos but did not touch or work with those products themselves. "Take-home" cases involve family members who allegedly incurred their disease from fibers carried home by family members (e.g., spouses or fathers) who worked with or around asbestos-containing products that produced only minimal exposures at the workplace. Both bystander and take-home instances of asbestos-caused mesothelioma are documented in the literature, but almost exclusively in association with high dose, amphibole workplace exposures such as in old asbestos factories. In instances where the workplace exposures are minimal to begin with, the assertion that such exposure is the cause of passer-by or take-home mesothelioma becomes an exercise in extreme speculation without epidemiological support. Some cases today even allege that the disease was caused through take-home exposure cases resulting from workplace bystander exposure - a double reduction of an already low dose to begin with.

n4. Mark A. Behrens & William L. Anderson, The "Any Exposure" Theory: An Unsound Basis for Asbestos Causation and Expert Testimony, *37 Sw. U. L. Rev.* 479 (2008).

n5. *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 607-08 (N.D. Ohio 2004), aff'd sub nom. *Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488 (6th Cir. 2005); *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 773 (Tex. 2007), reh'g denied, (Oct. 12, 2007); *Gregg v. V-J. Auto Parts, Co.*, 943 A.2d 216, 225 (Pa. 2007); remaining cases cited in Behrens & Anderson, supra note 4, at 6-19.

n6. See, e.g., *Buttitta v. Allied Signal, Inc. No. L-9592-02, 2010 WL 1427273*, at 10 (N.J. Super. Ct. App. Div. Apr. 5, 2010). This and other opinions are discussed infra section III.

n7. See discussion infra section IV.

n8. Idiopathic simply refers to "unknown cause." All cancers have unknown causes, and mesothelioma is no exception. The body itself produces dozens of cancerous cells daily that must be killed, fixed, or eliminated by the body's defenses or they can produce a spontaneous cancer. See generally Stanley Venitt, Mechanisms of Spontaneous Human Cancers, 104 *Env'tl. Health Perspectives* 633 (1996). The existence of idiopathic mesotheliomas, not attributable to asbestos, is well-recognized in the literature and typically admitted by most any exposure theorists. See, e.g., Victor Roggli et al., eds., *Asbestos-Associated Diseases* 108 (Springer 2d ed. 2004); C. Rake et al., Occupational, Domestic and Environmental Mesothelioma Risks in the British Population: A Case-Control Study, 100 *Brit. J. Cancer* 1175, 1181 (2009) (unexplained cases accounted for fourteen percent of male and sixty-two percent of female mesotheliomas in Britain); Mary Jane Teta et al., U.S. Mesothelioma Patterns 1973-2002: Indicators of Change and Insights Into Background Rates, 17 *Eur. J. Cancer Prevention* 525, 534 (2008) (stating that upwards of 300 cases of mesothelioma every year "may be unrelated to asbestos exposure" and may "reflect spontaneous causes"); Lawrence G. Cetrulo, *Asbestos Litigation & Tort Reform: Health Hazards and Diseases*, 4 *Toxic Torts Litig. Guide* § 33:3 (updated Oct. 2011) ("Asbestos exposure is the dominant cause of mesothelioma, and accounts for seventy to eighty percent of all mesothelioma cases."); B.T. Mossman et al., *Asbestos: Scientific Developments and Implications for Public Policy*, Science, vol. 247, issue 4940, Jan. 19, 1990, at 294 ("approximately 20 to 30% of mesotheliomas occur in the general population in adults not exposed occupationally to asbestos"); Lester Brickman, On the Theory Class's Theories of Asbestos Litigation: The Disconnect Between Scholarship and Reality, 31 *Pepp. L. Rev.* 33, 44 n.19 (2003) (stating that approximately twenty percent of malignant mesotheliomas have been attributed to causes other than exposure to asbestos). The reality of idiopathic cases is recognized in case law as well. See, e.g., *Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 551 (Ga. Ct. App. 2011) ("Dr. Maddox stated that there are idiopathic causes of mesothelioma."); *In re Toxic Substance Cases, No. A.D. 03-319, 2006 WL 2404008*, at 12 (Pa. Ct. Com. Pl., Aug. 17, 2006), rev'd sub nom. *Betz v. Pneumo Abex L.L.C.*, 998 A.2d 962 (Pa. Super. Ct. 2010) cert. granted, 9 A.3d

1134 (Pa. 2010), rev'd 44 A.3d 27 (Pa. 2012) ("All of the witnesses, including Drs. Maddox and Laman acknowledge that a certain percentage of mesotheliomas are idiopathic.").

n9. Almost all of these minor asbestos-containing products (e.g., brakes, gaskets, insulating wire, dental tape, floor tiles, caulking and the like) are bound up in resins or other materials and thus do not release their fibers easily. Numerous epidemiology studies have failed to document any increased incidence of mesothelioma in automobile mechanics, who work almost exclusively with bound, chrysotile asbestos products (brake pads, clutches, gaskets). The automotive studies are summarized and discussed in Francine Laden et al., Lung Cancer and Mesothelioma Among Male Automobile Mechanics: A Review, 19 Revs. on Envtl. Health 39 (2004); Michael Goodman et al., Mesothelioma and Lung Cancer Among Motor Vehicle Mechanics: A Meta-analysis, 48 Annals Occupational Hygiene 309, 309 (2004). Exposures to most of these products are typically well below even today's regulatory standard (0.1 f/cc per 8-hr time weighted average). See, e.g., Dennis J. Paustenbach et al., An Evaluation of the Historical Exposures of Mechanics to Asbestos in Brake Dust, 18 Applied Occupational & Envtl. Hygiene, 786, 786-804 (2003) (average lifetime mechanic exposures calculated at 0.04 f/cc or less, below OSHA standard of 0.1 f/cc); Brent Finley et al., Cumulative Asbestos Exposure for U.S. Automobile Mechanics Involved in Brake Repair (circa 1950s-2000), 17 J. Exposure Science & Envtl. Epidemiology, 641 (2007) (cumulative lifetime average exposures for automobile mechanics "are all substantially lower than the cumulative exposure of 4.5 f/cm³ year associated with occupational exposure to 0.1 f/cm³ of asbestos for 45 years that is currently permitted under the current occupational exposure limits in the US."). The any exposure theory, if it is not rejected, likely will extend asbestos litigation out for another 30-40 years by attributing causation for the ongoing flow of idiopathic cases to exposures well below even today's OSHA standard.

n10. See cases discussed in Behrens & Anderson, supra note 4, at 18-26; infra section III; Brief of Amici Curiae Richard Wilson, Patricia Buffler, John Henderson Duffus, Kenneth R. Foster, Ronald E. Gots, Thomas A. Kubic, Steven Lamm, A. Alan Moghissi, Robert Nolan, Malcolm Ross, and Emanuel Rubin in Support of Appellants, *Betz v. Pneumo Abex L.L.C.*, No. 38 WAP 2010 (Pa. filed Apr. 25, 2011), 2010 Pa S. Ct. Briefs 820 [hereinafter "ALF Scientist Brief"].

n11. See generally *Betz v. Pneumo-Abex L.L.C.*, 44 A.3d 27 (Pa. 2012) (holding that the any exposure theory was novel scientific evidence and, subject to a Frye hearing to determine its admissibility, before determining it inadmissible).

n12. *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 592 (1993) ("Faced with a proffer of expert scientific testimony ... the trial judge must determine ... whether the expert is proposing to testify to (1) scientific knowledge that (2) will assist the trier of fact to understand or determine a fact in issue.").

n13. See Roggli, supra note 8, at 1.

n14. Id. at 262-64.

n15. See *Gideon v. Johns-Manville Sales Corp.*, 761 F.2d 1129, 1145 (5th Cir. 1985) ("All asbestos-containing products cannot be lumped together in determining their dangerousness."); *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 606 (N.D. Ohio 2004) ("Prevailing scientific and medical view" supports lower chrysotile potency); *Becker v. Baron Bros., Coliseum Auto Parts, Inc.*, 649 A.2d 613, 620, 623 (N.J. 1994) (holding that the trial court erred in instructing jury that all asbestos-containing friction products without warnings are defective as a matter of law: "Our courts have acknowledged that asbestos-containing products are not uniformly dangerous and thus that courts should not treat them all alike."); *Celotex Corp. v. Copeland*, 471 So. 2d 533, 538 (Fla. 1985) ("Asbestos products ... have widely divergent toxicities, with some asbestos products presenting a much greater risk of harm than others.").

n16. See generally Charles M. Yarborough, *Chrysotile as a Cause of Mesothelioma: An Assessment Based on Epidemiology*, 36 *Critical Revs. Toxicology* 165 (2006), available at <http://www.chrysotile.com/data/Yarborough%202006%20Chrysotile%20as%20a%20Cause%20of%20Mesothelioma%20An%20Assessment%20Based%20on%20Epidemiology.pdf>; Eastern Research Group, Inc., *Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk*, at viii (2003), available at http://www.epa.gov/oswer/riskassessment/asbestos/pdfs/asbestos_report.pdf; Andrew Churg, *Nonneoplastic Disease Caused by Asbestos*, in *Pathology of Occupational Lung Disease* 277, 314 (Andrew Churg & Francis H.Y. Green eds., 2d ed. 1998); B.T. Mossman et al., *Asbestos: Scientific Developments and Implications for Public Policy*, 247 *Science* 294, 296, 299 (1990).

n17. Many studies of populations exposed to chrysotile have found only a few mesothelioma cases, if any, and those at very high doses. See, e.g., David Rees et al., *Case-Control Study of Mesothelioma in South Africa*, 35 *Am. J. Indus. Med.* 213, 220 (1999), available at <http://www.ehrn.co.za/publications/download/27.pdf> (no reports of mesothelioma from chrysotile exposure found despite substantial numbers of miners in chrysotile mines from the 1930s to 1980s exposed to intense concentrations of dust); H.F. Thomas et al., *Further Follow-Up Study of Workers from an Asbestos Cement Factory*, 39 *Brit. J. Indus. Med.* 273, 275 (1982), available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1009023/pdf/brjindmed00059-0065.pdf> (study of 1261 workers at asbestos cement plant using only chrysotile asbestos after 1936 found only two cases of mesothelioma, both in employees who worked at the plant prior to 1936 when the plant was using amphibole asbestos); M. Neuberger & M. Kundi, *Individual Asbestos Exposure: Smoking and Mortality - A Cohort Study in the Asbestos Cement Industry*, 47 *Brit. J. Indus. Med.* 615, 619 (1990), available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1035247/pdf/brjindmed00045-0039.pdf> (finding no incidence of mesothelioma among 2861 cement plant employees exposed only to chrysotile, some with exposures in excess of 50 f/ml); Misty Hein et al., *Follow-Up Study of Chrysotile Textile Workers: Cohort Mortality and Exposure-Response*, 64 *Occup. Environ. Med.* 616, 618, 620 (2007), abstract available at <http://oem.bmj.com/content/64/9/616.abstract> (finding only three mesotheliomas in workers employed in higher exposure jobs out of a cohort of 3,072 workers exposed to chrysotile of up to 700 fibre-years/ml years in an asbestos textile plant); see also John M. Dement et al., *Follow-Up Study of Chrysotile Textile Workers: Cohort Mortality and Case-Control Analyses*, 26 *Am. J. Indus. Med.* 431, 437-38 (1994), abstract available at <http://www.ncbi.nlm.nih.gov/pubmed/7810543>.

n18. For purposes of this article, "low" doses refer to those that are typically below today's Occupational Safety and Health Administration ("OSHA") standard for asbestos fiber exposures - 0.1 fibers per cubic centimeter as measured by an eight-hour time-weighted average (f/cc 8 hr TWA). OSHA considers such exposures to present an acceptable risk even if experienced every day for a forty year work career. The OSHA standard in the era when most exposures took place relevant to today's litigation - the 1970s and early 1980s - was considerably higher, first at 12 f/cc, then 5 f/cc in 1971, 2 f/cc in 1976, and 0.2 f/cc in 1986. J.F. Martonik et al., *The History of OSHA's Asbestos Rulemakings and Some Distinctive Approaches That They Introduced for Regulating Occupational Exposure to Toxic Substances*, 62 *Am. Indus. Hygiene Ass'n J.* 208, 211-12 (2001). The OSHA standard, however, was not established as a known threshold of disease, which must be determined instead from epidemiology studies documenting (or not) increased incidence of mesothelioma among populations with particular fiber type exposures and lifetime doses. For chrysotile, at least, such studies are typically limited to populations with extreme exposures, such as chrysotile miners, textile workers, and asbestos factory workers. See *supra* note 8, at 1.

n19. See, e.g., Kay Teschke et al., *Mesothelioma Surveillance to Locate Sources of Exposure to Asbestos*, 88 *Can. J. Pub. Health* 163, 165 (1997), available at <http://journal.cpha.ca/index.php/cjph/article/view/945/945>; Alison D. McDonald & J. Corbett McDonald, *Malignant Mesothelioma in North America*, 46 *Cancer* 1650, 1653-54 (1980), available at [http://onlinelibrary.wiley.com/store/10.1002/1097-0142\(19801001\)46:7%3C1650::AID-CNCR2820460726%3E3.0.CO;2-Y/asset/2820460726_ft.pdf?v=1&t=h69nb6zo&s=14f20cdc56342ce2ec5a2b86e88f771beb24324d&systemMessage=Wiley+Online+Library+will+be+disrupted+on+25+August+from+13%3A00-15%3A00+BST+%2808%3A00-10%3A00+EDT%29+for+essential+maintenance](http://onlinelibrary.wiley.com/store/10.1002/1097-0142(19801001)46:7%3C1650::AID-CNCR2820460726%3E3.0.CO;2-Y/asset/2820460726_ft.pdf?v=1&t=h69nb6zo&s=14f20cdc56342ce2ec5a2b86e88f771beb24324d&systemMessage=Wiley+Online+Library+will+be+disrupted+on+25+August+from+13%3A00-15%3A00+BST+%2808%3A00-10%3A00+EDT%29+for+essential+maintenance). See also Julian Peto et al., *Occupational, Domestic and Environmental Mesothelioma Risks in Britain: A Case-Control Study*, *UK Health and*

Safety Exec. (2009), available at <http://www.hse.gov.uk/research/rpdf/rr696.pdf>; Rake, *supra* note 8, at 1181-82.

n20. *Wintz v. Northrop Corp.*, 110 F.3d 508, 513 (7th Cir. 1997) (citing Reference Manual on Scientific Evidence); *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996).

n21. See, e.g., Office of Dietary Supplements, Nat'l Insts. of Health, Dietary Supplement Fact Sheet: Vitamin D, available at <http://ods.od.nih.gov/factsheets/VitaminD-QuickFacts/> (last updated October 2, 2011); Casarett & Doull's Toxicology, the Basic Science of Poisons 1073-74 (Curtis Klaasen ed. 6th ed. 2001) (carcinogens in food including arsenic); Office of Dietary Supplements, Nat'l Insts. of Health, Dietary Supplement Fact Sheet: Zinc, available at <http://ods.od.nih.gov/factsheets/Zinc-QuickFacts/> (last updated June 24, 2011).

n22. Bernard D. Goldstein & Mary Sue Henifin, Reference Guide on Toxicology, in Federal Judicial Center, Reference Manual on Scientific Evidence, 633, 636 (West Group 3d ed. 2011) (1994) (the "fundamental tenet" of toxicology). The "father of toxicology," physician and philosopher Paracelsus, first articulated this principle in the 16th century, stating: "All substances are poisonous - there is none which is not; the dose differentiates a poison from a remedy."

n23. "Whether an agent is 'capable of causing' a disease for purposes of determining whether tort liability should be imposed on someone is still a wholly different proposition than whether the agent should be considered carcinogenic as a regulatory matter." *In re Hanford Nuclear Reservation Litig.*, No. CV-91-3015-AAM, 1998 WL 775340, at 141 (E.D. Wash. Aug. 21, 1998); *rev'd on other grounds*, 292 F.3d 1124 (9th Cir. 2002). Advisory classifications are formulated using an explicitly lower proof threshold than that required for causation in a tort case. As the Fifth Circuit explained, courts apply a "more likely than not" or "preponderance" standard, while "regulatory and advisory bodies such as IARC, ... " NTP, and "EPA utilize a [lesser] "weight of the evidence" method to assess the carcinogenicity of various substances in human beings and suggest or make prophylactic rules governing human exposures." *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 198 (5th Cir. 1996). This "threshold of proof is reasonably lower than that appropriate in tort law." *Id.* See also *Mitchell v. Gencorp., Inc.*, 165 F.3d 778, 783 n.3 (10th Cir. 1999) (quoting Allen); *Cano v. Everset Minerals Corp.*, 362 F. Supp. 2d 814, 852 (W.D. Tex. 2005) (refusing to admit IARC Monograph on uranium exposure to support allegation that uranium exposure caused plaintiff's cancer because IARC classification was based on "inadequate" evidence in humans); *In re Agent Orange Prod. Liab. Litig.*, 597 F. Supp. 740, 785 (E.D.N.Y. 1985) (while an "agency may regulate ... toxic substances through rulemaking, despite a very low probability of any causal relationship," a court cannot infer causation from similar proof); *Lofgren v. Motorola*, No. CV 93-05521, 1998 WL 299925, at 21 (Ariz. Super. Ct. June 1, 1998) (stating that "it is appropriate public policy for health organizations such as IARC and the EPA to make judgments concerning the health and safety of the population based on evidence which would be less than satisfactory to support a specific plaintiff's tort claim for damages in a court of law").

n24. David L. Eaton, Scientific Judgment and Toxic Torts - A Primer in Toxicology for Judges and Lawyers, 12 J.L. & Pol'y 5 (2003). The Eaton article has been cited repeatedly in the last five years by courts that have taken a critical view of the any exposure theory. See, e.g., *Adams v. Cooper Indus.*, No. 03-476-JBC, 2007 WL 2219212, at 3 (E.D. Ky. July 30, 2007); *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1242-43 (11th Cir. 2005) (plaintiff expert's testimony that "any amount of [the drug at issue] is too much ... clearly contradicts the principles of reliable methodology delineated by Eaton"); *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1165 (E.D. Wash. 2009) (holding that the "use of the no safe level or linear "no threshold" model for showing unreasonable risk "flies in the face of the toxicological law of dose-response, that is, the "dose makes the poison"") (citing similar opinions); *In re Denture Cream Prods. Liab. Litig.*, 795 F. Supp. 2d 1345, 1351-52 (S.D. Fla. 2011). Those courts that have permitted such testimony do not cite Eaton at all, because the principles Eaton describes are incompatible with the theory.

n25. Eaton, *supra* note 24, at 11, 39.

n26. Id. at 13.

n27. See also *Baker v. Chevron USA, Inc.*, 680 F. Supp. 2d 865, 878 n.9 (S.D. Ohio 2010); Eaton, supra note 25, at 16.

n28. Eaton, supra note 24, at 13.

n29. *Wintz v. Northrop Corp.*, 110 F.3d 508, 513 (7th Cir. 1997) (citing Federal Judicial Center, Reference Manual on Scientific Evidence 1 (1994)) (illustrating that courts routinely require plaintiffs to demonstrate not just some exposure, but "evidence from which the trier of fact could conclude that the plaintiff was exposed to levels of toxins sufficient to cause the harm complained of); *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996) (demonstrating that this is as true for asbestos as for any other potentially toxic substance); see also *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 611 (N.D. Ohio 2004) (rejecting "one-fiber" asbestos theory as not supported by medical literature).

A scientific dose assessment does not necessarily require precise or mathematical calculation of the worker's exact dose, as several courts have held. See, e.g., *Parker v. Mobil Oil Corp.*, 857 N.E. 2d 1114, 1120-1121 (N.Y. 2006); *Henricksen*, 605 F. Supp. 2d at 1157 (stating plaintiffs sometimes argue that the difficulty of calculating an exact dose from long-ago exposures justifies an any exposure approach as the only way to prove a case). A lot of ground remains, however, between precise quantification and assuming that every exposure is a cause. In between, there is plenty of room for a professional and scientific assessment of the likely range of historic dose from a given product and whether that dose would compare to those known to produce disease. See *Parker*, 857 N.E.2d at 1121.

n30. *White v. Dow Chem. Co.*, Civ. Act. No. 2:05-cv-00247, 2007 WL 6948824, at 5 (S.D. W. Va. Nov. 29, 2007), aff'd, 321 F. App'x 266 (4th Cir. 2009).

n31. *Wright*, 91 F.3d at 1106.

n32. *Adams v. Cooper Indus.*, No. 03-476-JBC, 2007 WL 2219212, at 5 (E.D. Ky. July 30, 2007) (citing Federal Judicial Center, Reference Manual on Scientific Evidence 396 (2d ed.2000)).

n33. See *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1241 (11th Cir. 2005) (quoting *Wright*, 91 F.3d at 1106); *Mitchell v. Gencorp Inc.*, 165 F.3d 778, 781 (10th Cir. 1999) (quoting *Wright*, 91 F.3d at 1106); *Nelson v. Tenn. Gas Pipeline Co.*, 243 F.3d 244, 252-53 (6th Cir. 2001) (upholding exclusion of expert witness who "made no attempt to determine what amount of PCB exposure" the plaintiffs received); *Moore v. Ashland Chem. Inc.*, 151 F.3d 269, 278 (5th Cir. 1998), cert. denied, 526 U.S. 1064 (1999) ("Because he had no accurate information on the level of Moore's exposure to the fumes, Dr. Jenkins necessarily had no support for the theory that the level of chemicals to which Moore was exposed caused RADS"); *Baker*, 680 F. Supp. 2d at 887 (expert's testimony excluded where none of the studies he cited supported the opinion "that benzene can cause the illnesses from which plaintiffs suffer at the extremely low doses or exposures experienced in this case," as the subjects of those studies "generally had much higher exposures"); *Rose v. Matrixx Initiatives, Inc.*, No. 07-2404-JPM, 2009 WL 902311, at 13 (W.D. Tenn. Mar. 31, 2009) (excluding testimony of expert whose opinion was "not supported by any studies of the dose-response relationship, which is the hallmark of basic toxicology") (internal quotes and citation omitted); *In re Bextra and Celebrex Mktg. Sales Practices and Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1174 (N.D. Cal. 2007) ("The court finds that dose matters."); *Louderback v. Orkin Exterminating Co.*, 26 F. Supp. 2d 1298, 1305 (D. Kan. 1998) (quoting *Wright*, 91 F.3d at 1106); *Mancuso v. Consol. Edison Co. of N.Y.*, 967 F. Supp. 1437, 1453 (S.D. N.Y. 1997) (expert's testimony that plaintiffs' ailments were caused by exposure to PCBs was inadmissible because, inter alia, expert "did not make a sufficient effort to determine the levels of PCBs to which plaintiffs were exposed"); *Nat'l Bank of Commerce v. Dow Chem. Co.*, 965 F. Supp. 1490, 1524 (E.D. Ark. 1996) ("To establish specific causation in the case it was incumbent upon plaintiffs to provide evidence from which a jury could responsibly assess the level of the exposure

of Mrs. Smits to Dursban while she worked at the bank ... Then the plaintiffs must provide evidence from which the jury could determine whether the levels of exposure and dose experienced by Mrs. Smits and the fetus were likely to produce birth defects of the type experienced by Ashley.").

n34. See, e.g., *Parker*, 857 N.E.2d at 1121-22 (rejecting use of qualitative terms where no assessment of dose took place).

n35. Brickman, *supra* note 8, at 49.

n36. See *Bartel*, 316 F. Supp. 2d at 607-08, *aff'd sub nom. Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488, 498 (6th Cir. 2005); *Borg-Warner Corp.*, 232 S.W.3d at 773 (expert acknowledged background fibers but did not suggest they were a cause of asbestosis); *Georgia-Pac. Corp. v. Stephens*, 239 S.W.3d 304, 315 (Tex. App. 2004) (Dr. Hammar testified that the "level of exposure it takes to cause mesothelioma" could be any level above what is considered to be background"); *In re Toxic Substances Cases*, 2006 WL 2404008 at 3 ("background or ambient exposure is simply not sufficient to allow experts to causally attribute asbestos-related disease to it. Everyone, including the plaintiff's experts, agrees that something greater is required.").

n37. David C. Hodgson et al., Long-Term Solid Cancer Risk Among 5-Year Survivors of Hodgkin's Lymphoma, 25 J. Clinical Oncology, 1489, 1489-97 (2007); M. Jane Teta, DrPH, MPH et al., Therapeutic Radiation for Lymphoma: Risk of Malignant Mesothelioma, 109 Cancer 1432, 1433 (2007); Jonathan D. Tward et al., The Risk of Secondary Malignancies Over 30 Years After the Treatment of Non-Hodgkin's Lymphoma, 107 Cancer 108, 108-15 (2006).

n38. See *supra* note 8.

n39. Roggli, *supra* note 8, at 108.

n40. *Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 551 (Ga. Ct. App. 2011) ("It is improper for an expert to presume that the plaintiff "must have somehow been exposed to a high enough dose to exceed the threshold (necessary to cause the illness), thereby justifying his initial diagnosis.' This is circular reasoning.") (citing *Mancuso v. Consol. Edison Co. of N.Y.*, 967 F. Supp. 1437, 1450 (S.D.N.Y. 1997)); *Nelson v. Tenn. Gas Pipeline Co.*, 243 F.3d 244, 254 (6th Cir. 2001) (rejecting circular reasoning that disease must have been caused by exposure to PCBs, without any assessment of actual PCB exposure); *Lofgren v. Motorola*, No. CV 93-05521, 1998 WL 299925, at 18 (Ariz. Super. Ct. June 1, 1998) ("Throughout these proceedings, it has also been a matter of great concern to the Court that the experts retained by plaintiffs were apparently willing to "assume' the presence of a "significant' dose of TCE to each of the plaintiffs. It appears uncontroverted that each of the plaintiffs' experts was willing to give an opinion in the absence of any accurate information about dosage ... This is circular reasoning.") (quoting *Mancuso*, 967 F. Supp. at 1450).

n41. See, e.g., *Butler*, 712 S.E.2d at 540 ("Dr. Maddox testified that each and every exposure to asbestos above background levels contributes to the development of mesothelioma."); *Bartel v. 316 F. Supp. 2d at 611* ("[The plaintiff's expert] opines that there is no safe level of asbestos exposure, and that every exposure to asbestos, however slight, was a substantial factor in causing Lindstrom's disease."); *Georgia-Pacific Corp. v. Stephens*, 239 S.W.3d 304, 315 (Tex. App. 2004) ("[Expert] expressed an opinion that each and every exposure that an individual has in a bystander occupational setting causes their mesothelioma.").

n42. *In re Toxic Substances Cases*, No. A.D. 03-319, 2006 WL 2404008, at 6 (Pa. Ct. Com. Pl., Aug. 17, 2006), *rev'd sub nom. Betz v. Pneumo Abex L.L.C.*, 998 A.2d 962 (Pa. Super. Ct. 2010) cert. granted, 9 A.3d 1134 (Pa. 2010), *rev'd 44 A.3d 27* (Pa. 2012) (emphasis added). As the trial judge noted, "Nowhere, however, do they even remotely attempt to quantify the actual exposure that they believe would be required" *Id.* at 6.

n43. See, e.g., *Betz v. Pneumo Abex L.L.C.*, 44 A.3d 27, 34-37 (Pa. 2012).

n44. Id.

n45. See, e.g., World Health Org. Int'l Agency for Research on Cancer, Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42, 106-16 (Supp. 7, 1987), available at <http://monographs.iarc.fr/ENG/Monographs/suppl7/Suppl7.pdf>; World Health Org., Elimination of asbestos-related diseases 2 (2006), available at http://whqlibdoc.who.int/hq/2006/WHO_SDE_OEH_06.03_eng.pdf; World Health Org. Env'tl. Health Criteria 53: Asbestos and Other Natural Mineral Fibers, §§9.3.1.3 to 9.3.2 (2006), available at <http://www.inchem.org/documents/ehc/ehc/ehc53.htm#SectionNumber:9.3>.

n46. *In re Toxic Substances Cases*, 2006 WL 2404008, at 13; see also *Free v. Ametek*, No. 07-2-04091-9 SEA, 2008 WL 728387, at 3-4 (Wash. Super. Ct. Feb. 28, 2008) (Plaintiffs' experts Dr. Samuel Hammar and Dr. Carl Brodtkin opined that there is no safe threshold to asbestos exposure and therefore, every biologically significant exposure is causative).

n47. See *In re Toxic Substances Cases*, 2006 WL 2404008, at 13.

n48. Id. at 3. A handful of these experts will at least identify a specific dose below which they would conclude even occupational exposures would not be causative. The dose they select, however, is typically whatever they believe "background" exposures to be - usually extremely low numbers that would not eliminate many occupational exposures, at least on a single exposure basis. Id.

n49. See *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 773 (Tex. 2007) (noting that the prevalence of asbestos in the environment means some exposure "threshold" must be demonstrated before a claimant can prove causation) (quoting David L. Eaton, *Scientific Judgment and Toxic Torts - A Primer in Toxicology for Judges and Lawyers*, 12 J.L. & Pol'y 5, 39 (2003)); see also *Free*, 2008 WL 728387, at 4 (granting motion to preclude expert testimony).

n50. See Eaton, *supra* note 25, at 11 ("All substances are poisonous - there is none which is not; the dose differentiates a poison from a remedy.") (emphasis omitted) (quoting Louis J. Casarett et al., *Casarett and Doull's Toxicology: The Basic Science of Poisons* chs. 1 & 4 (Curtis D. Klaassen ed., McGraw Hill 6th ed. 2001) (internal quotation marks omitted)); See *Wintz v. Northrop Corp.*, 110 F.3d 508, 513 (7th Cir. 1997); *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996); *Parker v. Mobil Oil*, 857 N.E.2d 1114, 1120-21 (N.Y. 2006); *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 607-08 (N.D. Ohio 2004); *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1155 (E.D. Wash. 2009).

n51. Cf. *Norris v. Crane Co.*, No. B196031, 2008 WL 638361, at 14 (Cal. Ct. App. Mar. 11, 2008) (holding there was "substantial evidence [victim]'s exposure to asbestos from materials in Crane valves increased his risk of developing mesothelioma and, therefore, was a substantial factor in causing his injury"); *Jones v. John Crane, Inc.*, 132 Cal. App. 4th 990, 999 (Cal. Ct. App. 2005) (holding cumulative exposure was sufficient to find defendants' exposing victim to asbestos products caused victim's cancer); *Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 544 (Ga. Ct. App. 2011) (holding the trial court's discretion was not abused when the judge granted a motion to strike expert testimony for failing to prove causation); *Georgia-Pac. Corp. v. Bostic*, 320 S.W.3d 588, 601-02 (Tex. App. 2010) (holding evidence existed that deceased employee in wrongful death suit was exposed to an asbestos compound made by the manufacturer, but the evidence was legally insufficient to establish the requisite causation).

n52. 232 S.W.3d 765 (Tex. 2007).

n53. See, e.g., Cortney Fielding, Plaintiff's Lawyers Turn to L.A. Courts for Asbestos Litigation, Daily J. (Los Angeles), Feb. 27, 2009 (Verdicts & Settlements), at 1.

n54. *Honeywell Int'l, Inc. v. Boomer*, No. 120299, 2012 Va. LEXIS 107 (Va. 2012).

n55. *Barabin v. AstenJohnson, Inc.*, No. 10-36142, consolidated with No. 11-35020 (9th Cir. argued Jan. 11, 2012).

n56. See, e.g., *Abuan v. Gen. Elec. Co.*, 3 F.3d 329, 332-34 (9th Cir. 1993) (affirming summary judgment for defendant) (PCBs) ("In cases claiming personal injury from exposure to toxic substances, it is essential that the plaintiff demonstrate that she was, in fact, exposed to harmful levels of such substances.") (emphasis added by Court) (citation and quotation omitted); *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1241 (11th Cir. 2005) (reversing judgment for plaintiff) (ephedrine) ("In toxic tort cases, "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 plaintiffs' burden.") (citing and quoting *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996)); *Wintz v. Northrop Corp.*, 110 F.3d 508, 513 (7th Cir. 1997) (affirming judgment for defendant) (bromide) (holding a plaintiff must offer evidence from which the trier of fact could conclude that "the dose to which the plaintiff was exposed was sufficient to cause the disease" complained of) (citing and quoting the Federal Reference Manual on Scientific Evidence); *Wright v. Willamette Indus., Inc.*, 91 F.3d 1105, 1107 (8th Cir. 1996) (reversing judgment for plaintiff) (formaldehyde) ("At a minimum, we think there must be evidence from which the trier of fact could conclude that the plaintiff was exposed to levels of that agent that are known to cause the kind of harm that the plaintiff claims to have suffered.") (citing *Abuan*, 3 F.3d at 333).

n57. See Victor E. Schwartz & Rochelle M. Tedesco, The Law of Unintended Consequences in Asbestos Litigation: How Efforts to Streamline the Litigation Have Fueled More Claims, 71 *Miss. L.J.* 531, 542-47 (2001); Victor E. Schwartz & Leah Lorber, A Letter to the Nation's Trial Judges: How the Focus on Efficiency Is Hurting You and Innocent Victims in Asbestos Liability Cases, 24 *Am. J. Trial Advoc.* 247, 256-58 (2000).

n58. See, e.g., *Blanchard v. Goodyear Tire & Rubber Co.*, 30 A.3d 1271 (Vt. 2011); *Parker v. Mobil Oil*, 857 N.E.2d 1114 (N.Y. 2006); *Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671 (6th Cir. 2011); *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142 (E.D. Wash. 2009); *King v. Burlington N. Santa Fe Ry. Co.*, 762 N.W.2d 24 (Neb. 2009).

n59. See *Henricksen*, 605 F. Supp. 2d at 1151.

n60. See *Parker*, 857 N.E.2d at 1117.

n61. See *Henricksen*, 605 F. Supp. 2d at 1165-66; *Parker* 857 N.E.2d at 1118-19; *King*, 762 N.W.2d at 31-32.

n62. *Id.*

n63. See *In re Denture Cream Prods. Liab. Litig.*, 795 F. Supp. 2d 1347, 1352-53 (S.D. Fla. 2011).

n64. See *Richardson v. Union Pac. R.R.*, 2011 Ark. App. 562 (Ark. Ct. App. 2011).

n65. See *Newkirk v. Conagra Foods*, 727 F. Supp. 2d 1006 (E.D. Wash. 2010).

n66. *Emerald Coast Utils. Auth. v. 3M Co.*, 746 F. Supp. 2d 1216 (N.D. Fla. 2010); *Rhodes v. E.I. du Pont de Nemours and Co.*, 636 F.3d 88, 95 (4th Cir. 2011) ("The presence of PFOA in the public water supply or in the plaintiffs' blood does not, standing alone, establish harm or injury for purposes of proving a negligence claim ...").

n67. See *Emerald Coast Utils. Auth.* 746 F. Supp. 2d at 1227-28; *City of Greenville v. Syngenta Crop Prot.*, 756 F. Supp. 2d 1001 (S.D. Ill. 2010) (holding plaintiff's argument that "mere presence of atrazine" in water supply sufficed for injury).

n68. In oversimplified terms, epidemiology studies compare an "exposed" population to an "unexposed" one to see if the exposed group has a statistically higher incidence of a particular disease. These studies become problematic when they attempt to assess low exposures that are close to those experienced generally by normal populations - there is no "unexposed" group for comparison. Since substances like asbestos and benzene are ubiquitous, it is difficult to construct a study that "proves" low or background doses do not cause disease. Instead, public health officials typically rely on negative studies to conclude that, absent contrary evidence, such low exposures do not pose an unacceptable risk and need no intervention.

n69. See *Newman v. Motorola, Inc.*, 218 F. Supp. 2d 769, 773-74 (D. Md. 2002) ("substantial body of literature" exists showing no general acceptance of the theory that cell phones cause brain cancer); Stephen Riccardulli et al., *Cellphone Waves May Bring a Litigation Wave*, Forbes Magazine, June 20, 2011, available at <http://www.forbes.com/2011/06/20/cellphone-cancer-litigation.html>; Joshua E. Muscat et al., *Handheld Cellular Telephone Use and Risk of Brain Cancer*, 284 JAMA 3001, 3001-07 (2000) (noting the fact that data suggested use of cellular telephones is not associated with a risk of brain cancer); Elisabeth Cardis, *Brain Tumour Risk in Relation to Mobile Telephone Use: Results of the INTERPHONE International Case-Control Study*, 39 Int'l J. Epidemiology 675, 675-94 (2010) (finding no increase in the risk of glioma or meningioma from the use of mobile phones).

n70. The any exposure theory rests exclusively on expert opinion and thus is usually amenable to a challenge under the federal expert admissibility standard established under *Daubert v. Merrell-Dow Pharmaceuticals*, 509 U.S. 579 (1993), or the older Frye federal standard still used today by many states, *Betz v. Pneumo Abex L.L.C.*, 44 A.3d 27 (Pa. 2012). In many instances, however, these cases turn on a sufficiency of the evidence analysis, either in summary judgment or as a result of trial or post-trial motions.

We analyze the approach under the expert exclusion rules of Daubert and Frye below, but the court analysis is not substantially different if raised in the context of a sufficiency of evidence review. That is not to say that there are no differences between an expert challenge and a challenge to sufficiency of the evidence. The admissibility of evidence under Daubert and Frye is only the first hurdle to supporting a causation case, one that if not typically cleared ends the case by leading to a "no causation evidence" summary judgment ruling. Even if an expert survives an admissibility challenge, however, the testimony technically may still be found insufficient to support substantial factor causation. *Borg-Warner Corp. v. Flores*, 232 S.W.3d 765, 769 (Tex. 2007) (citing *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1162 (4th Cir. 1986); *Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 611 (Ohio 2004)). In reality, it would be unusual for a court to find the any exposure theory passable under Daubert, but nevertheless find it insufficient to support causation - the theory tends to rise or fall on its own and not on the procedural mechanism for challenging it.

n71. See *Berlyn, Inc. v. Gazette Newspapers, Inc.*, 214 F. Supp. 2d 530, 536 (D. Md. 2002).

n72. See *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 148-49 (1999); *Daubert*, 509 U.S. at 595.

n73. See *Tyger Constr. Co. Inc. v. Pensacola Constr. Co.*, 29 F.3d 137, 145 (4th Cir. 1994) (quoting *E. Auto Distribs., Inc. v. Peugeot Motors of Am.*, 795 F.2d 329, 338 (4th Cir. 1986)).

n74. *Daubert*, 509 U.S. at 593-95.

n75. *Id.* at 589-90. Many states continue to utilize the older Frye standard rejected by the Daubert court. Under Frye, the issue is not the reliability of the methodology per se; instead, the inquiry is focused on the general acceptance of the scientific principle involved within the relevant scientific community. Initially, the legal community expected Daubert to result in more frequent admission of expert testimony than Frye because no longer would an opinion need to pass the hurdle of general acceptance to be presented to a jury. Instead, however, Daubert has turned out to be the more stringent test, simply because it is more flexible and allows judges more room to closely examine the expert's opinion, the methodology used, and the science relied on for the opinion. Many states utilize some variant on these two approaches or apply general rules of evidence to keep out unreliable scientific testimony. See Martin S. Kaufman, *The Status of Daubert in State Courts*, Atlantic Legal Foundation (2006), available at, <http://www.atlanticlegal.org/daubertreport.pdf>; *Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114, 1120 (N.Y. 2006) (rejecting expert testimony under general evidentiary rule of reliability rather than Frye).

n76. See e.g., *Daubert v. Merrell Dow Pharm. Inc.*, 43 F.3d 1311 (9th Cir. 1995); *United States v. Rincon*, 28 F.3d 921, 923-26 (9th Cir. 1994).

n77. See *id.*

n78. *Daubert*, 43 F.3d at 1311.

n79. *Id.* at 1316.

n80. *Id.* (emphasis added).

n81. *Id.*

n82. *Id.*

n83. *Id.* at 1322.

n84. *Id.*

n85. 522 U.S. 136, 146 (1997).

n86. *United States v. Rincon*, 28 F.3d 921, 923-26 (9th Cir. 1994).

n87. *Id.* at 924.

n88. *Id.*

n89. 29 F.3d 499, 502 (9th Cir. 1994).

n90. *Id.*

n91. *Id.*

n92. *Id.* at 503; see also *id.* at 505 n.7 ("They also argue that producing specific causation evidence is impossible, citing a study that states, 'it is impossible to define selective differences between the effects of various solvents.' What the study actually stated however was that 'on the basis of this study it is impossible ...' to define such differences.).

n93. The Ninth Circuit again visited the extent of trial court analysis in *Cabrera v. Cordis Corp.*, 134 F.3d 1418 (9th Cir. 1998) (relating to plaintiff's contention that a brain shunt implant injured her as the result of silicone toxicity). Among other things, the experts cited to a number of articles they claimed supported their contention that the shunt's hard silicone content caused the disease in question. *Id.* at 1422-23. But when the court examined the studies, it found that they actually related to a different medical outcome, hypersensitivity reactions to malfunctioning stunts. *Id.* Cabrera thus represents a court looking behind the self-serving testimony of the experts and discovering that the cited studies did not support the proposition before the court.

n94. 633 F.3d 828 (9th Cir. 2011).

n95. *Id.* at 834 (emphasis added).

n96. *Id.* at 837.

n97. *Id.*

n98. *Id.* at 838.

n99. *Id.* at 840.

n100. *Id.*

n101. *Id.*

n102. See, e.g., *Daubert v. Merrell Dow Pharm. Inc.*, 43 F.3d 1311, 1319 (9th Cir. 1995) (holding that expert testimony was contradicted by substantial epidemiology).

n103. *Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 550-51 (2011), cert. denied (Oct. 17, 2011) ("It is improper for an expert to presume that the plaintiff 'must have somehow been exposed to a high enough dose to exceed the threshold (necessary to cause the illness), thereby justifying his initial diagnosis.' This is circular reasoning.") (citing *Mancuso v. Consol. Edison Co. of New York, Inc.*, 967 F. Supp. 1437, 1450 (S.D.N.Y. 1997)); *Nelson v. Tenn. Gas Pipeline Co.*, 243 F.3d 244, 254 (6th Cir. 2001) (rejecting circular reasoning that disease must have been caused by exposure to PCBs, without any assessment of actual PCB exposure); *Lofgren v. Motorola, Inc.*, CV 93-05521, 1998 WL 299925, at 18 (Ariz. Super. Ct. June 1, 1998) ("Throughout these proceedings, it has also been a matter of great concern to the Court that the experts retained by plaintiffs were apparently willing to 'assume' the presence of a 'significant' dose of TCE to each of the plaintiffs. It appears uncontroverted that each of the plaintiffs' experts was willing to give an opinion in the absence of any accurate information about dosage ... This is circular reasoning.") (citing *Mancuso*, 967 F. Supp at 1450); *In re Toxic Substances Cases*, A.D. 03-319, 2006 WL 2404008, at 7 (Pa. Com. Pl. Aug. 17, 2006) (rejecting downward extrapolation); *Free v. Ametek*, No. 07-2-04091-9 SEA, 2008 WL 728387 (Wash. Super. Ct. Feb. 28, 2008).

n104. *Lindstrom v. A-C Prod. Liab. Trust*, 424 F.3d 488 (6th Cir. 2005).

n105. *Id.* at 498.

n106. *Martin v. Cincinnati Gas & Elec. Co.*, 561 F.3d 439, 443 (6th Cir. 2009) (quoting *Lindstrom*, 424 F.3d at 493).

n107. *Moeller v. Garlock Sealing Techs.*, 660 F.3d 950 (6th Cir. 2011).

n108. *Id.* at 955.

n109. *Id.* Most of the companies that made or applied asbestos insulation have gone bankrupt, primarily due to the impact of asbestos litigation. Lawsuits today must these seek additional targets for recovery, in the process stretching to ever more tangential asbestos exposure to do so. See Susan Warren, *Asbestos Quagmire: Plaintiffs Target Companies Whose Premises Contained Any Form of Deadly Material*, Wall St. J., Jan. 27, 2003, at B1, available at 2003 WLNR 3099209 (discussing asbestos-related lawsuits targeting companies with little or no apparent connection to the material).

n110. *Id.* (quoting *Lindstrom*, 424 F.3d at 492). In *Lindstrom*, the court "permitted evidence of substantial exposure for a substantial period of time to provide a basis for the inference that a product was a substantial factor in causing the injury." 424 F.3d at 492. The court, however, also cautioned that when a plaintiff relies on proof of exposure to establish a product was a substantial factor "the plaintiff must show a high enough level of exposure that an inference that the asbestos was a substantial factor in the injury is more than conjectural." *Id.* (internal quotation and citation omitted).

n111. *Moeller*, 660 F.3d at 955 (citing *Gregg v. V-J. Auto Parts, Co.*, 943 A.2d 216, 223 (Pa. 2007)).

n112. *Id.* at 954.

n113. *Id.* at 955.

n114. *Id.*

n115. *Barabin v. AstenJohnson, Inc.*, No. 10-36142 (consolidated with No. 11-35020) (9th Cir. argued Jan. 11, 2012).

n116. *Barabin v. Albany Int'l Corp.*, No. C07-1454RSL, 2009 WL 2578967 (W.D. Wash. Aug. 18, 2009).

n117. Transcript of Record at 144-45, *Anderson v. Asbestos Corp.*, No. 05-2-04551-5SEA (Wash. King Cnty. Super. Ct. Oct. 31, 2006); *Free v. Ametek*, No. 07-2-04091-9 SEA, 2008 WL 728387 (Wash. Super. Ct. Feb. 28, 2008).

n118. *In re Asbestos Prod. Liab. Litig.*, No. 10-CV-61118, 2011 WL 605801 (E.D. Pa. Feb. 16, 2011).

n119. Order on Motions in Limine, *Barabin v. Albany Int'l Corp.*, No. C07-1454RSL at 11 (W.D. Wash. Sept. 18, 2009).

n120. That article is the self-styled "Helsinki criteria," an attempt in 1996 by a selected group of experts to attribute asbestos disease to certain levels and types of exposure. A. Tossavainen, Asbestos, asbestosis, and cancer: The Helsinki Criteria for Diagnosis and Attribution, 23 Scand. J. Work Environ. Health 4, 311-16 (1997). The court opinion does not give any indication the court actually read this article, or considered whether it fit the testimony the expert gave. Helsinki did not consider specific fiber types and toxicities, or establish that every single exposure no matter how small is causative. It instead required "significant" workplace exposures before attributing mesothelioma causation. See *Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 552 (2011); ALF Scientist Brief, supra note 10.

n121. *Betz v. Pneumo Abex L.L.C.*, 44 A.3d 27 (Pa. 2012).

n122. See, e.g., *In re Toxic Substance Cases*, No. A.D. 03-319, 2006 WL 2404008 (Pa. Ct. Com. Pl., Aug. 17, 2006), rev'd sub nom. *Betz v. Pneumo Abex L.L.C.*, 998 A.2d 962 (Pa. Super. Ct. 2010) cert. granted, 9 A.3d 1134 (Pa. 2010), rev'd 44 A.3d 27 (Pa. 2012).

n123. *Betz*, 44 A.3d at 28.

n124. *In re Toxic Substance Cases*, 2006 WL 2404008 at 8.

n125. *Id.* at 1.

n126. *Id.* at 2.

n127. *Gregg v. V-J Auto Parts, Co.* 943 A.2d 216, 226-27 (Pa 2007).

n128. *Summers v. Certainteed Corp.*, 886 A.2d 240, 244 (Pa. Super. Ct. 2005).

n129. See, e.g. *Basile v. American Honda Motor Co., Inc.*, No. 11484 CD 2005, 2007 WL 712049 (Pa. Com. Pl. Feb. 22, 2007); *In re Asbestos Litig.*, No. 0001, 2008 Phila. Ct. Com. Pl. LEXIS 229 (Pa. Com. Pl. Sept. 24 2008).

n130. *Betz v. Pneumo Abex L.L.C.*, 998 A.2d 962, 983 (Pa. Super. Ct. 2010) rev'd, 44 A.3d 27 (Pa. 2012).

n131. *Gregg*, 943 A.2d at 226-27.

n132. *Betz*, 998 A.2d at 967-68.

n133. *Betz*, 44 A.3d at 58.

n134. *Id.* at 53.

n135. *Id.* at 56.

n136. *Id.* at 55.

n137. *Id. at 56-57*. In support of this position, Dr. Maddox offered a number of analogies. For example, he offered that his opinion is akin to the sentiment that "every soldier in the field has a substantial effect on the outcome of a war." *Id. at 57*. The court was highly critical of these analogies concluding that while they may be true in a figurative and honorary fashion, they did not bear any connection to science. *Id.*

n138. *In re Toxic Substance Cases*, 2006 WL 2404008 at 6.

n139. *Betz*, 44 A.3d at 56.

n140. *Id.* (citing *Gregg v. V-J Auto Parts Co.*, 943 A.2d 216, 226-27 (Pa. 2007)).

n141. *Borg-Warner v. Flores*, 232 S.W.3d 765, 771-72 (Tex. 2007).

n142. *Georgia-Pac. Corp. v. Stephens*, 239 S.W.3d 304, 320-21 (Tex. App. 2007).

n143. 320 S.W.3d 588 (Tex. App. 2007).

n144. *Id. at 598*.

n145. 307 S.W.3d 829, 834-35 (Tex. App. 2010).

n146. *Id. at 839*.

n147. *Id. at 837-39*.

n148. *Id. at 839*.

n149. 712 S.E.2d 537, 540 (Ga. Ct. App. 2011) (The Court found one of the plaintiffs' experts, Dr. Maddox, a "quintessential expert for hire.").

n150. *Id. at 539*.

n151. *Id. at 540*.

n152. *Id. at 541-42*.

n153. *Id. at 542*.

n154. *Id.* (noting that the methodology behind the study did not allow the researchers to identify those subjects whose exposure was only to chrysotile fibers).

n155. *Id.*

n156. In *Betz v. Pneumo Abex L.L.C.*, 44 A.3d 27 (Pa. 2012), the Pennsylvania Supreme Court also rejected reliance on the Helsinki criteria because "appellants correctly observed that these do not embody the any-exposure theory." *Id.* at 55 n.35.

n157. *Daly v. Arvinmeritor, Inc.*, No. 07-19211, 2009 WL 4662280 at 4 (Fla. Cir. Ct., Nov. 30, 2009). In another Florida case, *Honeywell Int'l, Inc. v. Guilder*, 23 So.3d 867 (Fla. Dist. Ct. App. 2009), the trial court's acceptance of the any exposure issue was central to the appeal, but the appellate court reversed the verdict on other grounds.

n158. *Daly*, 2009 WL 4662280, at 4 (emphasis added).

n159. Special Master's Report and Recommendation on Defendants' Motions for Summary Judgment and Daubert Motions Regarding Plaintiff's Experts Raterman, Haber and Hammar, *Nix v. AGCO Corp.*, No. 2010-85-CV8 (Miss. Cir. Ct. Jones Cnty. Sept. 9, 2011) (adopted by the court Sept. 21, 2011).

n160. *Id.* at 6.

n161. This phrase refers to the Lohrmann decision by the United States Fourth Circuit Court of Appeals in 1986. Many states adopted this standard thereafter. See *Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1163 (4th Cir. 1986).

n162. Special Master's Report and Recommendation, *supra* note 161 at 2.

n163. *Id.* at 6-7. The magistrate granted summary judgment to one defendant because the alleged exposure "amounts to the type of casual or minimal contact discussed in Lohrmann, which is insufficient to prove causation." Summary judgment was denied, without discussion, as to several other defendants.

n164. Judgment on Motion in Limine as to Dr. Jacques Legier, *Degrasse v. Anco Insulations*, No. 07-12736 (Civ. Dist. Ct., Parish of Orleans June 11, 2009).

n165. Motion Hearings, *Robertson v. Ashby*, No. 532, 769 (Dist. Ct. Parish of E. Baton Rouge, La. Jan. 19, 2010).

n166. *Id.*

n167. Judgment, *Bello v. Anco Insulations*, No. 559, 507 (Dist. Ct. Parish of E. Baton Rouge, La. Oct. 19, 2010).

n168. *Robertson v. Doug Ashy Bldg. Materials, Inc.*, No. 2010 CA 1551, 2011 La. App. LEXIS 1173, at 35 (Oct. 4, 2011). A "product identification" defense is premised on the plaintiffs' failure to identify a particular defendant's product sufficiently to prove exposure to that product. The any exposure theory, in contrast, comes into play only when the product has been identified but the alleged exposure is minor and unlikely to cause disease.

n169. *Id.* at 54.

n170. *Id.* at 48.

n171. The appellate court included several pages of discussion of Dr. Mark's opinions that seem to favorably comment on those opinions, without an explicit finding of reliability. *Doug Ashy Building Materials, Inc., 2011 La. App. LEXIS 1173, at 33-38*. This recitation, if it were indeed an analysis, would constitute a classic failure under Daubert - the court repeats, in great detail, what Dr. Mark said, including his self-serving statements that his opinions are widely accepted, scientific, and supported by the literature. But the court never analyzed whether any of those statements are in fact true.

n172. Order at 3-4, *Schumacher v. Amtico*, No. 5:10-01627 (E.D. Pa. Nov. 2, 2010) ("Dr. Maddox opines that cumulative low-level exposures can result in mesothelioma ... Dr. Maddox relies on [a] variety [of] peer-reviewed studies and reports ... Dr. Maddox is prepared to testify ... Dr. Maddox looks to case studies ... his opinion is based on numerous studies ... Dr. Maddox takes one position, and Defendants' experts take another."). Nowhere in this opinion is there any indication the court read the studies and reports to ascertain whether Dr. Maddox's use of them was reliable and fit the facts of the case.

n173. *Id.* at 3.

n174. *Id.* at 4.

n175. The further opinions are: *Larson v. Bondex Int'l, No. 09-69123, 2010 WL 4676563* (E.D. Pa. Nov. 15, 2010); *In re Asbestos Prod. Liab. Litig.*, No. 10-CV-61118, *2011 WL 605801, at 1* (E.D. Pa. Feb. 16, 2011); Memorandum Opinion, *Rabovsky v. Air & Liquid Sys. Corp.*, No. 10-cv-03202 (E.D. Pa. Jan. 25, 2012) (Strawbridge, Mag.), *aff'd mem.*, *Rabovsky v. Air & Liquid Sys. Corp.*, No. 10-3202 (Mar. 12, 2012) (Robreno, J.).

n176. Order at 3-4, *Schumacher v. Amtico*, No. 5:10-01627 (E.D. Pa. Nov. 2, 2010).

n177. *Larson, 2010 WL 4676563, at 6 n.3.*

n178. The court cited to four studies as examples, all of which are animal or in-vitro studies of mechanisms of chrysotile action. None of these studies even comes close to supporting the notion that every breath of chrysotile is a cause of real disease. *Id.* at 4.

n179. See Paolo Boffetta, *Epidemiology of Peritoneal Mesothelioma: A Review*, 18 *Annals of Oncology* 985 (2007).

n180. See *supra* note 122.

n181. *In re Asbestos Prod. Liab. Litig.*, *2010 WL 4676563, at 6 n.3.*

n182. *In re Asbestos Prod. Liab. Litig.*, No. 10-CV-61118, *2011 WL 605801, at 1.*

n183. *Id.* at 5.

n184. *Id.* at 5-6. The any exposure experts get around this problem with Iwatsubo and Rodelsperger by claiming they were "mixed" exposures and thus any plaintiff with any kind of mixed amphibole and chrysotile exposures would have the same risk of mesothelioma. But this argument entirely ignores the different potency of the two fibers. A person may die from a "mix" of arsenic and Coca-Cola, but that does not prove that low doses of Coca-Cola had any connection to the death. As noted above, the Texas courts engaged in a serious review of

these two studies and pointed out the logical error in relying on them for chrysotile exposure cases. See *Georgia-Pac. Corp. v. Bostic*, 320 S.W.3d 588, 601 (Tex. App. 2010); *Georgia-Pac. Corp. v. Stephens*, 239 S.W.3d 304, 321 (Tex. App. 2007). Both Iwatsubo and Rodelsperger explicitly stated in their published articles that their studies cannot offer any evidence as to chrysotile exposures. See Iwatsubo, et al., Pleural Mesothelioma: Dose Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study, 148 Am. J. of Epidemiol 133, 141 (1998) ("We could not examine mesothelioma risk according to fiber types because our study design ... did not allow us to identify those subjects whose exposure was only to chrysotile fibers."); Rodelsperger, et al., Asbestos and Man-Made Vitreous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results From a German Hospital-Based Case-Control Study, 39 Am. J. of Indus. Med. 262, 263 (2001) (observing no reliable dose-response relationship between chrysotile and the risk of mesothelioma). Experts who are excluded under Daubert often read more into certain articles than is warranted.

n185. See *Butler v. Union Carbide Corp.*, 712 S.E.2d 537, 542-43 (Ga. Ct. App. 2011), cert. denied Oct. 17, 2011.

n186. *In re Asbestos Prod. Liab. Litig.*, 2011 WL 605801, at 4.

n187. See *id.* at 1-3. This "susceptible persons" argument is not new and has been rejected previously in toxic tort litigation. See, e.g., *Pohl v. NGK Metals Corp.*, 936 A.2d 43, 51 (Pa. Super. Ct. 2007). The flaw in the argument is that variations in individual susceptibility are not infinite - they occur within a range and do not extend down to zero exposures. The height of human beings certainly includes considerable individual variation, but no one is one inch tall.

n188. *In re Asbestos Prod. Liab. Litig.*, 2011 WL 605801, at 7.

n189. *In re Asbestos Prod. Liab. Litig.*, 2011 WL 605801, at 11.

n190. See *supra* note 17.

n191. The magistrate in one opinion justifies the ruling by stating that "multiple courts have found [the expert's] opinions to be sufficiently reliable to meet the admissibility standard." *In re Asbestos Products*, 2011 WL 605801, at 7. Yet the magistrate never mentioned or analyzed the steady drumbeat of opposite opinions issuing since 2005.

n192. 941 P.2d 1203 (Cal. 1997).

n193. *Rutherford v. Owens-Ill., Inc.*, 941 P.2d 1203, 1223 (Cal. 1997).

n194. *Id.* at 1220 (emphasis added).

n195. See *Jones v. John Crane, Inc.*, 35 Cal. Rptr. 3d 144, 148 (2005); *Behshid v. Bondex Int'l Inc.*, No. B194789, 2008 WL 2807226 (Cal. Ct. App. July 22, 2008).

n196. Defendants trapped in the any exposure net sometimes have used it to spread responsibility to other low-dose defendants. For example, in *Silvestro v. Kaiser Gypsum Co.*, No. B196906, 2009 WL 976820, at 6-8 (Cal. Ct. App. Apr. 13, 2009), the Court of Appeals reversed a jury award holding the appellant liable for a thirty percent of fifteen million dollar judgment. The plaintiff had proceeded on the theory that every exposure above background can cause disease, and testimony at trial indicated that he had come into contact with at least eight asbestos-containing joint compounds when working in construction (however minor the exposure). The jury,

however, allocated zero percent fault to five of those manufacturers. The court remanded the case for a reallocation of the damage award to include the manufacturers who had been given no fault. See also *CertainTeed Corp. v. Dexter*, 330 S.W.3d 64, 64 (Ky. 2010).

n197. *Rutherford*, 941 P.2d at 1223; see also *Lineaweaver v. Plant Insulation Co.*, 31 Cal. App. 4th 1409, 1415-16 (Cal. Ct. App. 1995).

n198. *Rutherford*, 941 P.2d at 1218 (emphasis added).

n199. *Id.*

n200. *Id.* at 1221.

n201. *Id.* at 1220 (emphasis added).

n202. *Id.* at 1222.

n203. See, e.g., *Behshid v. Bondex Int'l, Inc.*, No. B194789, 2008 WL 2807226 (Cal. Ct. App. July 22, 2008); *Norris v. Crane Co.*, No. BC340413, 2008 WL 638361 (Cal. Ct. App. Mar. 11, 2008); *Hoeffler v. Rockwell Automation, Inc.*, Nos. A107353, A107964, 2006 WL 185479 (Cal. Ct. App. Jan. 26, 2006).

n204. *Buttitta v. Allied Signal, Inc.*, No. L-9592-02, 2010 WL 1427273 (N.J. Super. Ct. App. Div. Apr. 5, 2010).

n205. *Id.* at 9.

n206. *Id.* at 10.

n207. As to chrysotile fibers, the kind found in brakes, the notion that mesothelioma arises from "minor exposures" is manifestly untrue and widely contradicted in the medical literature. See Eastern Research Group, Inc., Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk, viii (2003), available at http://www.epa.gov/oswer/riskassessment/asbestos/pdfs/asbestos_report.pdf ("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."); Rake, *supra* note 8, at 1182.

n208. To be sure, the court stated that this testimony was "uncontradicted." *Buttitta*, 2010 WL 1427273, at 10. It would be surprising if in fact the defense failed to put anything in the record contradicting the notion that low doses of chrysotile fibers cause mesothelioma, as such a defense is standard in brake cases. This statement at least opens the door to a different outcome, if and when defendants mount a full challenge to any exposure testimony in New Jersey.

n209. *CertainTeed Corp. v. Dexter*, 330 S.W.3d 64, 82 (Ky. 2010).

n210. *Id.* at 79.

n211. See *Moeller v. Garlock Sealing Techs.*, 660 F.3d 950, 955 (6th Cir. 2011); *Martin v. Cincinnati Gas & Elec. Co.*, 561 F.3d 439, 446 (6th Cir. 2009).

n212. *John Crane, Inc. v. Linkus*, 988 A.2d 511, 513 (Md. Ct. Spec. App. 2010); *Scapa Dryer Fabrics, Inc. v. Saville*, 988 A.2d 1059, 1066 (Md. Ct. Spec. App. 2010), aff'd in part, rev'd in part, 16 A.3d 159 (Md. 2011).

n213. *Scapa*, 16 A.3d at 167-68.

n214. *Id.* at 168.

n215. See generally *Reiter v. Pneumo Abex, L.L.C.*, 8 A.3d 725 (Md. 2010).

n216. *Scapa*, 16 A.3d at 167.

n217. Kathryn S. Bilello, Epidemiology, Etiology, and Prevention of Lung Cancer, 23 Clinics in Chest Med. 1 (2002).

n218. See e.g., U.S. Mesothelioma Patterns 1973-2002: Indicators of Change and Insights Into Background Rates, 17 Eur. J. Cancer Prevention 525, 534 (2008); see generally Jack T. Peterson et al., Non-asbestos Related Malignant Mesothelioma: A Review, 54 Cancer 951, 955-59 (1984); Y. Izzettin Baris et al., Environmental Mesothelioma in Turkey, 330 Ann. NY Acad. Sci. 423 (2002); see also Roggli, supra note 8 at 109-11.

n219. See Roggli supra note 8.

n220. *Baker v. Chevron USA, Inc.*, 680 F. Supp. 2d 865, 870 (S.D. Ohio 2010).

n221. *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142, 1150-51, 1166 (E.D. Wash. 2009).

n222. 857 N.E.2d 1114, 1122 (N.Y. 2006).

n223. 640 F.3d 671, 674-675 (6th Cir. 2011).

n224. 680 F. Supp. at 887.

n225. 30 A.3d 1271, 1277-78 (Vt. 2011).

n226. 605 F. Supp. 2d at 1165-66.

n227. *In re Denture Cream Prods Liab. Litig.*, 795 F. Supp. 2d 1345 (S.D. Fla. 2011).

n228. *Id.* at n.33.

n229. *Id.* at 1352.

n230. *Richardson v. Union Pac. R.R. Co.*, 2011 Ark. App. 562, at 1, at 15-16.

n231. *Id.* at 18-20.

n232. *Newkirk v. Conagra Foods, Inc.*, 727 F. Supp.2d 1006 (E.D. Wash 2010). The court, in a 32-page opinion, dissected the expert's testimony and reasoning. *Id.*

n233. *Id.* at 1015.

n234. Safe Drinking Water Act, 42 U.S.C. § 300f et seq. (1974)

n235. *Parker v. Mobil Oil Corp.*, 857 N.E.2d 1114 (N.Y. 2006).

n236. *Id.* at 1121-22.

n237. *Id.* at 1122.

n238. *Pluck v. BP Oil Pipeline Co.*, 640 F.3d 671 (6th Cir. 2011).

n239. *Id.* at 675-76.

n240. *Id.* at 679.

n241. *Id.* at 675.

n242. *Henricksen v. ConocoPhillips Co.*, 605 F. Supp. 2d 1142 (E.D. Wash. 2009).

n243. *Id.*

n244. *Id.* at 1156.

n245. *Id.* at 1157.

n246. *Id.* at 1165.

n247. *Id.* at 1165-66.

n248. *Id.* at 1166.

n249. *Baker v. Chevron USA, Inc.*, 680 F. Supp. 2d 865 (S.D. Ohio 2010).

n250. *Id.* at 887 n.9.

n251. *Id.* at 887.

n252. *Id.*

n253. 762 *N.W.2d* 24, 50-51 (*Neb.* 2009).

n254. *Id.* at 51.

n255. *Richardson v. Union Pac. R.R. Co.*, 2011 *Ark. App.* 562, at 1, at 2-3.

n256. *Newkirk v. ConAgra Foods, Inc.*, 727 *F. Supp. 2d* 1006, 1024 (*E.D. Wash.* 2010).

n257. 746 *F. Supp. 2d* 1216, 1223 (*N.D. Fla.* 2010).

n258. *Id.*

n259. *Id.*

n260. *Adams v. Cooper Indus., Inc.*, No. B171606, 2007 *WL* 2219212, at 5, 7 (*E.D. Ky.* 2007).

n261. *Id.* at 7.

n262. *Id.*

n263. See, e.g., *Nelson v. Tenn. Gas Pipeline Co.*, 243 *F.3d* 244, 252-53.

