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Environmental (Toxic) Torts

Jack W. Snyder*

I want to say first of all that this may be the only time in my career that I stand in for a federal judge, so I'm grateful for the opportunity. I come from the world of medical academia and my venue there is as a pathologist and toxicologist. As a pathologist, I have training very similar to Cyril Wecht's, but Cyril focused his career in the medical examiner's office, and I got into drugs. So I basically tell people that I'm into drugs, and that at least piques their interest. Toxic tort—what is it? Ten years ago if you used the term toxic tort, people thought you were referring to food poisoning. We're not talking about that today. We're talking about a burgeoning arena of litigation that now has its own casebooks, its own conferences, and its own medical and legal scholarship.

What do asbestos, PCB's, Agent Orange, bendectin, swine flu vaccine, IUD's (intrauterine devices), pesticides, paraquat, breast implants, multiple chemical sensitivities, norplant, cigarettes, lead, and accutane have in common? These are the subjects of ongoing, or in some cases, resolved litigation in the arena that has been designated as toxic or environmental torts. A reasonable definition of a toxic tort is "one or more individuals alleging physical injury or other harm due to exposure to environmental agents."

I'm going to talk rather conceptually. My bottom line message is that toxic tort litigation has become a battleground upon which the concept of compensable injury is being inexorably expanded. Indeed, the ever-increasing, ever-expanding definition of compensable injury has become a critical driving force in this

* Associate Professor, Thomas Jefferson University, Philadelphia, Pennsylvania. B.S., Northwestern University; M.D., Northwestern University; J.D., Georgetown University; M.F.S., George Washington University; M.P.H., Johns Hopkins University; Ph.D. (Pharmacology & Toxicology), Medical College of Virginia; Diplomate, American Boards of Toxicology, Medical Toxicology, Toxicological Chemistry, Clinical Chemistry, Preventive (Occupational) Medicine, Legal Medicine, Quality Assurance & Utilization Review, and Anatomic, Clinical, and Chemical Pathology.
sphere of legal activity.

I want to address at least two of the most important issues in toxic tort litigation today; that is, the issue of injury and how we define it, and then, how we prove causation. I will try to show you what’s the same and what’s different about the way scientists and lawyers think about these issues. It’s a real pleasure for somebody from the medical ivory tower to come and talk to a group of jurists because, as I view it, it is the jurist who can make a real difference in how society approaches these problems.

Along the way, I’ll try to put a few things in perspective by introducing you to a bit of the mechanics, the personnel, and the process of the discipline known as toxicology. Let’s paint with a broad brush concerning that discipline before we talk about injury and causation. Toxicology as a scientific discipline has become the tail that wags the dog of pharmacology. In pharmacology, we study the effects of drugs in what we call biologic matrices. We use the term “biologic matrix” because it allows me to talk generally about the effects of environmental agents on anything from subcellular molecules all the way up to an intact human being. We can talk about interactions of drugs or chemicals with something that is part of a cell, one cell, a tissue, an organ, or the entire body. Each of these is a biologic matrix at a different level.

I’m going to explain to you some of the ways that lawyers and scientists think differently about injury and causation, and how those differences play out in toxic tort, product liability, and workers’ compensation litigation. Again, the most important point I want to make today is that we are expanding our notion of compensable injury in this society. We have to decide, and jurists in particular have to decide, to what extent they want to continue that expansion or to put the brakes on it.

The Pennsylvania Supreme Court is one of the few state supreme courts that has actually defined compensable injury. In a workers’ compensation case, the court defined compensable injury as any adverse or hurtful change in the system which would cause lessened facility of the natural use of any bodily activity, or capacity.1 So there’s one definition from a state supreme court.

There are increasing numbers of alternative definitions of injury—compensable or otherwise. To aid your understanding, I

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want to walk you through this conceptual road map. First, let’s walk through it wearing a scientist’s or physician’s hat. Then we’ll put on the jurist’s, or legal professional’s hat, and investigate what’s similar and what’s different about the use of these words in law, science, and medicine.

Let’s look at something you’ve all heard of—benzene. It’s one of the few molecules that the United States Supreme Court has weighed in on. Back in 1980, the Court issued a decision that involved one of the permissible exposure limits set by the Occupational Safety and Health Administration (“OSHA”). The Court viewed the question as whether OSHA had come up with the appropriate evidence to support a reduction of the permissible exposure limit from ten parts per million in the workplace to one part per million.² As I think you’ll recall, the Court said that OSHA had not met its burden of proof, and therefore, OSHA could not lower the permissible exposure limit for benzene from ten parts per million down to one. But let’s start with the benzene molecule and any biologic matrix—anything from a subcellular particle all the way up to an intact human being. And let’s bring the environmental agent and the biologic matrix into the presence of one another. By presence, do we mean geographic proximity, or something more? Hold that question in abeyance and go with me on these words. I hope you’ll begin to see that the different ways people think about these terms often depend on which professional hat they are wearing.

So we bring the molecule and the tissue into the presence of one another. One of two things can happen according to current western biomedical thought. There’s either an impact, and we’ll define that momentarily, or there is no impact. I can bring a benzene molecule into the closest possible proximity with the cell membrane and something may happen or something may not. They may bounce off one another, they may actually have some kind of chemical reaction, or they may not.

Scientists describe varying kinds of impact in physical, chemical, or physicochemical terms. They also have ways to measure this impact. The next question asked by scientists and physicians who study cell injury mechanisms is: “If there’s an impact, does the biologic matrix respond to that impact?” Well, there’s either going to be response or there’s not going to be response. The fact that I bring a chemical into the presence of a biologic matrix and it has an impact that I can measure does not mean

that the biologic matrix will in turn respond. It may or may not. And we can measure that response. When something biologic responds to a chemical in the environment, that response can be viewed either as a beneficial effect or an adverse effect. It's important to understand that contact between a chemical and a human being does not necessarily produce an adverse or undesirable event.

Many scientists and physicians will describe the effects of certain chemicals as beneficial. For example, appropriate amounts of vitamins and nutrients are obvious sources of beneficial effects. The science of immunology is full of examples of responses to chemical exposure that produce beneficial effects for an organism or biologic matrix.

But if there is an adverse or undesirable effect that can be defined in different ways that we'll discuss later, that's not the end of the story. In western science and medicine we recognize that the adverse effect leads to one of two events. The matrix either repairs or adapts to the adverse effect or it does not. Failure to adapt or repair can be viewed as a form of permanent injury. Most scientists and physicians, however, characterize irreversible injury at more than one level. Importantly, at least two levels of damage are distinguished by the concept of functionality. That is, some adverse effects that are not repaired in humans or biologic matrices represent a kind of damage that is not accompanied by functional deficit. What's functional deficit? If I knock out a segment of my brain and I can't walk, that's a functional deficit. If I destroy my bone marrow with toxic chemicals so that I can't make blood cells, that's a functional deficit.

But there are many situations where we have damage but it doesn't affect the way we live. It doesn't influence our quality of life or lifestyle in ways that anyone can objectively verify or measure. Scientists, physicians, and some courts call this "de minimis damage."

I don't know how many of you are from the Pennsylvania Commonwealth Court or have adjudicated workers' compensation cases. Some scientific, medical, and workers' compensation professionals say that disease is rampant, that disease is everywhere. The notion, however, that everything is diseased or that all forms of injury to biologic matrices should be defined as disease does not help the jurist who must decide if wealth is to be transferred from one party to another.

In my view, a lot of de minimis damage occurs that is not accompanied by dysfunction. De minimis damage alone cannot and does not define disease. What can and must define disease is quantitatively verifiable, measurable, biologically significant
change that leads to abnormal function that causes a problem in someone’s lifestyle or quality of life.

In the workers’ compensation arena, once we get an expert or a decision-maker to characterize a claimant’s problem as quantitatively significant damage or dysfunction, we still have to decide if the disease or dysfunction is work related. As most of you know, the statutory and caselaw definitions of the scope of work-relatedness vary among the states. But whether we call it an occupational disease or a non-occupational disease, we ultimately get to the concept of compensable injury.

What is driving all of this is the great desire of many in our society to transfer wealth from one party to another. In toxic or environmental tort litigation, you as jurists are the focal point for assuring that those transfers get made appropriately. It’s one thing to talk conceptually about injury, but at some point your major concern is when and how to transfer wealth from one party to another to compensate for that injury.

Now let’s take off the scientific-medical hat and let’s put on the legal hat for a moment. Let’s ask ourselves: “What is the legal system doing with these concepts and terms?” As a point of departure, let’s focus on the terms “presence,” “impact,” “response,” “adverse effect,” “de minimis damage,” and “disease.” I would submit that for each term, there is at least one court, somewhere in this nation, that has equated that term with compensable injury. This is the key concept for the day. Understand that each of these terms can be equated with compensable injury. Why is that of great significance, at least from a toxicologist’s and lawyer’s point of view? Because the quantum of evidence required to prove causation, the numbers and types of experts that counsel must bring to the court, and the nature of the legal argument ultimately depend on how you characterize the compensable injury.

When I tell the scientists and physicians in any forum that the concept of bringing a benzene molecule into the presence of human bone marrow has been equated with compensable injury, they say: “No wonder our legal system is so screwed up.” Many of these professionals simply are not prepared to accept the idea that something less than objectively verifiable and quantitatively significant harm is legally compensable. My colleagues in medicine and science invariably express surprise, chagrin, and occasional disgust when I explain the evolution of at least three new torts in American common law. The first of these torts provides compensation for increased risk of future harm. The second compensates for fear of present or future harm. And the third transfers wealth on the basis of outrage. What are the
elements of proving any of these torts in a particular jurisdic-
tion? That certainly varies, as does the kind of proof required to
overcome the causation hurdle. Some of the plaintiffs' counsel in
the breast implant litigation will tell you that the jury verdicts
for their clients are transferring wealth on the basis of outrage.
The science here is not what is making the day. The bottom line
for many observers of this litigation is that juries are making
these awards because they are outraged by what they perceive
to be the conduct of the manufacturers in going to market before
the products were adequately evaluated. The point for continu-
ing debate, however, is the propriety of imposing a 1995 stan-
dard of care with regard to toxicology testing for pre-market
purposes onto a product that was marketed in the 1940's, 1950's,
1960's, and 1970's. Some juries seem to be imposing a standard
of care in a backwards manner.

So anyone involved with toxic torts should keep these three
relatively new tort concepts in mind. If you ask me what the
elements of those torts are, I think they vary. I don't know what
it takes to prove the tort of outrage, but it's clear that at least a
few of the breast implant juries know what it takes.

Before we leave the concept of injury and move to causation, I
want to bring you up to date conceptually on how the medical
and scientific community conceives the molecular aspects of
injury to a biological matrix. We're not going to get quantitative
here and I'm not going to show you a lot of graphs or charts.

This is a brief digression into biology—just enough science to
try to bring some important points together for you. Let's look at
the fat molecules in all the membranes of the cells in your body.
We call this the lipid bilayer model of cell membranes. This
cartoon depicts the way western science conceives of the outer
barriers to all of your cells as well as some of the barriers found
inside your cells. Embedded in the fat molecules are various
kinds of proteins. The reason you need fat and protein in your
diet is because you have a constant turnover of these molecules.
You're constantly breaking these down and making new
ones—it's a dynamic process. Some proteins span the membrane
channel and provide a canal or mode of entry for various sub-
stances, both wanted and unwanted, into a cell—or into a hu-
man being, if you want to think on a macro level. There are a
number of things that can happen when membranes interact
with environmental agents. Let's discuss a few of them.

Anesthetics and alcohols are two kinds of agents that are well
known to fluidize membranes. What we mean by fluidizing
membranes is perturbing or interfering with the movements and
relationships of fat molecules in cell membranes. Sometimes we
screw up the function of membranes by exposing them to sufficient amounts of alcohol or anesthetics.

Drugs, poisons, and toxins can also interfere with the normal function of protein channels. This is a significant area of pharmacetic and toxicologic research. Many drugs, for example, that are used to treat high blood pressure and other conditions alter the function of these channels by preventing the movement of sodium or calcium through them. These drugs are often designed with the idea of changing cellular function. Sometimes we introduce a little "injury" to overcome or compensate for some other problem. With some drugs, however, the margin for error is quite small. A slight excess can rapidly change a desired perturbation into an unwanted adverse effect that may occasionally be life-threatening.

Most of you know what it means for butter to become rancid. Well, one of the most popular explanations for the aging process involves the idea that our brain, heart, and other tissues gradually rancidify over time. In many health food stores you can find entire shelves of products known as anti-oxidants. With antioxidant research, the pharmaceutical industry is trying to find ways to slow or prevent the rancidification of fat in biologic membranes, a process of cell injury known as lipid peroxidation. Toxicologists believe that lipid peroxidation is one of the undesirable outcomes of oxidative stress, a term that emphasizes the potentially beneficial as well as detrimental role of highly chemically reactive molecular forms of oxygen.

Another mechanism of cell injury involves the cross-linking of cellular proteins. These channels and other proteins that function in signal transduction mechanisms can be disrupted by linking them so that they can no longer do what they are supposed to do. It's like taking ropes and tying them together. That's what we mean by cross-linking.

Before we leave mechanisms, I should remind you of a fundamental principle of toxicology. It's the dose of something that makes it a poison. Remember that a glass of water is probably beneficial for each of us while a bathtub full will most likely kill some of us. Why? If you drink a bathtub full of water, you'll dilute the sodium concentration in your blood. It may fall to the point that seizures develop and death ensues if you're not properly treated. An aspirin or two might be good for you in terms of making your blood a little less clottable. A lot of people out there are taking small amounts of aspirin to prevent strokes. On the other hand, the ingestion of a hundred aspirin will probably kill at least one person in this room. So it's the dose that makes the poison. I wanted to make sure that I reminded you of that fun-
There are several other basic mechanisms of injury that I don’t have time to address. I’ve just given you a flavor of some of the concepts used by scientists and physicians to characterize the effect of an environmental agent on the function of a human being or any biologic matrix.

I won’t spend a lot of time on this, but just so you’re aware, science and medicine have a number of ways to measure injury. We actually believe we can quantitate some aspects of injury. It is these kinds of studies that can provide the basis for opinions expressed by toxicologists, epidemiologists, physicians, and other experts in legal proceedings. When these professionals reach their conclusions, sometimes those opinions are based on mountains of papers they’ve read, but other times there isn’t any data, so they just make it up—they speculate. And it’s your job as jurists to try to figure out—and of course juries have to do this, too—who’s speculating and who isn’t.

Somebody was talking about liver function studies earlier today. How is it that we can take a blood sample and determine that somebody’s liver has been damaged or isn’t functioning as well as it should? Well, we take that sample into the laboratory, such as the one I run, and we measure the release of enzymes into that blood. Physicians also request similar tests of enzyme release into the blood when they try to diagnose a heart attack.

Enzymes are proteins that make a lot things happen in your body. The primary function of enzymes is to catalyze or accelerate the rates of chemical reactions. If you don’t have those enzymes, or if you shut them down, many chemical processes in your body don’t go forward. If you shut down some of the enzymes responsible for production of membrane voltage and energy in the brain or heart, you will kill those cells and that human being. Virtually all cells have power plants known as mitochondria. Why does cyanide kill people within minutes? Because it interferes with the function of a very specific mitochondrial enzyme called cytochrome oxidase. Cyanide shuts that enzyme down and prevents normal function of mitochondria. If your tissues can’t utilize sugar and fat to produce energy because they don’t have functional cytochrome oxidase, you die quickly. Importantly, we can measure losses of enzyme activity, providing yet another marker of cell injury.

Now I want to talk about causation. I want to move from injury to causation on the premise that our definitions of compensable injury ultimately determine what must be brought to the table to prove causation. Our conceptual roadmap of injury can remind us of a fundamental tension in toxic tort litigation.
The plaintiff's counsel typically seeks to push the definition of compensable injury towards "presence" and away from "disease." It's much easier to show mere exposure to a substance, and equate that to compensable injury, than it is to show that a substance has caused disease. On the other hand, defense counsel typically argues in product liability or toxic tort cases that nothing short of quantitatively significant damage is deserving of the transfer of wealth from one party to another in our society.

First we'll discuss cause-and-effect relationships as they are perceived in science and medicine, and then we'll look at causation and its terminology as you know it in American law. My comments derive from many sources, including a valuable piece by Professor Brennan in the Cornell Law Review.3

Isaac Newton, the physicist, and John Locke, the philosopher, articulated some concepts of causation that guided western scientific thought for several hundred years. Quantitative mechanics and the laws of motion and acceleration (as mathematically expressed by Newton and epistemologically explained by Locke) theorized a limited power of one object to produce directional change in another via collision. Thus mechanical contacts between particulate objects provided the basis for a mechanical notion of causation known as corpuscularianism. Along with positivism, which is the belief that scientific knowledge continuously expands, corpuscularian concepts increasingly influenced legal approaches to scientific evidence in the 18th, 19th, and early 20th centuries.

Hempel summarized the positivism-corpuscularian view of scientific theory as a relationship between a covering law (one set of deductive principles) and an explanandum (the phenomenon that the covering law was to explain). The nature of that relationship (the explanans, or ultimate explanation) was usually expressed in causal language. Thus, according to Hempel, science progresses as deductive reasoning applies ever-expanding covering laws to more and more phenomena, enrolling them into causal chains that in turn connect previously unexplained phenomena. Uncertainty results only when a covering law cannot be applied to a phenomenon. Hempel further argued that deductive reasoning outweighs inductive reasoning as the primary method of causal explanation, of prediction, and of knowledge in general.

The development of calculus, matrix analysis, quantum mechanics, and the theory of relativity in the 20th century undermined Newtonian mechanics and corpuscularian approaches to scientific evidence. Statistical relationships and inductive reasoning, which rely on random sampling and probability calculation, were increasingly accepted as bases for causal propositions. Medicine and science recognized the validity, the importance, indeed, even the indispensability of probabilistic reasoning in the language of scientific explanation. The concept of deductive, either/or, causal chain analysis gave way to emphasis on inductive/probabilistic reasoning and production of evidence to support or deny the validity of a particular hypothesis. Science progresses as some theories and hypotheses provide better causal explanations than others. As Professor Brennan has suggested, if causation is a matter of theory, and if theories are modified over time, then causation is not a simple either/or proposition. The probability that one event caused another can be increased or decreased, depending on how well new evidence fits with the guiding theory.

Thus, concepts of causation have been modified to allow for probabilities. By contrast, the expression or articulation of causation most often has not. The language of causation still centers on deductive causal chains. Confusion arises when a scientific explanation is framed in deductive causal chain terms, but the evidence to support that explanation is summarized by a probability statement. Many scientists and physicians admit that the language of causation they use often expresses inductive/probabilistic reasoning as deductive reasoning or in deductive terms. The bottom line, however, is that the metaphor of science as a constantly growing causal chain is no longer appropriate. Unfortunately, this idea has not gained a strong foothold in American jurisprudence. Current scientific concepts of causation continue to be poorly communicated in current legal articulation of causation.

If causation is, at least in part, a probabilistic concept, then decision makers must deal with degrees, types, and/or levels of uncertainty. This task is typically uncomfortable for jurists and legislators who have not had some formal exposure to probabilistic thinking. In the absence of continuing judicial education, these analytical problems are likely to persist because few attorneys have training in probabilistic reasoning, and the

percentage of young people studying physical and biologic science has been decreasing. Indeed, the medical and scientific establishment has expressed grave concern because the number of American-born individuals pursuing careers in biomedical research is also decreasing.

Does any of this discussion have any real significance for the judiciary? I suppose the answer depends on your view of the role of science and medicine in the legal process. In my experience, crowded dockets and limited resources severely constrain the ability of most courts to deal seriously with scientific evidence and experts. Increasing numbers of judges, however, seek advice on how to improve their performance as Daubert v. Merrell Dow Pharmaceutical, Inc. evidentiary "gatekeepers." Because time is short, I can only discuss one analytical framework that may aid those jurists make serious efforts to separate the wheat from the chaff.

What are the criteria that scientists and physicians typically use, or should be using, when they render an opinion that substance X caused effect Y? In other words, what are the scientific criteria for proof of causation that are out there today? These criteria have been variously known over the years as the Evans-Henle-Koch postulates.

We can, for example, address these causation criteria in the context of silicone breast implants. As we go through these statements, ask yourself: "For a particular adverse effect, disease, or other form of legal cognizable injury, how many of these criteria have been satisfied by the available scientific and medical evidence?" Then ask yourself: "How many of these criteria do I feel have to be fulfilled before I can say that X causes Y?" If you poll the members of any profession or group, and ask each of them how many of these criteria are needed to opine, with a reasonable degree of medical or scientific certainty, that X causes Y, you'll get answers varying anywhere from just one to all nine. Most of you in this room will demand satisfaction of more than one but less than all nine criteria.

First, the current rate of a disorder or injury in a population (prevalence) and the rate of new injuries or disorders in a specific period of time (incidence) should be significantly higher in those exposed to implants than in controls not so exposed. Of course, you can define "disorder" or "injury" at any level along our continuum, and therein lies the problem. People talk past each other in the silicone controversy because they define injury

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differently. Moreover, the increased incidence and prevalence of the injury may result from varying combinations of exposure to the implant and defects in the host response. That is, the relative contribution of environmental and genetic factors may vary among implant recipients.

Second, exposure to implants should be more frequent among those with a defined injury than in controls who do not have the defined injury when all other risk factors are held constant. Of course, this assumes you can control all other risk factors—something that is easier said than done. Third, in the course of time, the injury or disorder that's alleged to have been caused by the implant should follow that exposure. In other words, the exposure ought to come first—you have to prove that someone was exposed to silicone before an injury ensued.

Fourth, a spectrum of adverse effects or injuries should follow exposure to implants along a logical biologic gradient from mild to severe. This is the concept of dose-response that many of you have heard about. With a little bit of exposure, you ought to get a little bit of an effect. With a more intense or lengthier exposure, you ought to get a little more effect. It's amazing how many fact-finders do not understand this concept, and it's clearly reflected in their decisions, because if they understood that fundamental concept, there's no way they could have logically reached their conclusions based on science or medicine. And of course this gets us back to the idea of using science or medicine as a ruse or prop to accomplish something much more insidious, much more under the table, namely, the imposition of 1995 standards of care for activities that went on 20, 30 or 40 years ago.

Fifth, there must be a measurable injury following exposure to implants. This injury should have a high probability of appearing in those lacking the response before exposure, and the injury or disorder should increase in magnitude if it was present prior to exposure to implants. This response pattern should occur infrequently, if at all, in persons not so exposed. Sixth, an experimental reproduction of the injury or disorder should occur more frequently in animals (or man) appropriately exposed to implants than in those not so exposed; this exposure may be deliberate in volunteers or experimentally induced in the laboratory. This means there ought to be an "animal model." Many toxicologists like to hang their hats on this one. Of course, there are many courts that say they don't care what the animal data shows or that a substance may cause injury to animals.

Elimination or modification of the implant or a component part should decrease the incidence of injuries or disorders. Pre-
vention or modification of the host's response on exposure to the implant should decrease or eliminate the injury or disorder. And don't forget the ninth (last) criterion. An expert's opinion, including her interpretations of relationships and findings in the literature, should make biologic, toxicologic, and epidemiologic sense. If the expert's opinion or his evidence doesn't make sense to you, stop and ask yourself: "What's wrong here? Does this person's testimony comport with my own knowledge and/or experience?"

I think Daubert creates more work for judges, but I think rightly so, because I don't believe in science courts. We don't ultimately want scientists and physicians making these momentous decisions; we just want their input. I believe the Third Circuit has it right. I believe Judge Becker has it right. The In re Paoli Railroad Yard PCB Litigation decision is one of the best opinions to read in this area; an attempt to understand Judge Becker's approach is worth an investment of your time.

I also like the decision in United States v. Downing. The United States Supreme Court cited Downing in Daubert several times with approval. I was on several panels the year before the Daubert decision, and I don't mind saying that I was typically the only person who predicted that the Supreme Court would go with the Third Circuit. As I often say: "I'm from the Third Circuit, so I have to like what the Third Circuit does."

Let's turn our attention to legal concepts of causation. Let's remind ourselves of the ways that lawyers and judges traditionally think and communicate about causation. By the way, legal reasoning and terminology regarding causation are quite foreign to scientists and physicians. No matter how hard you try, many of them just don't get it. Enough do, however, that it becomes worthwhile to try to explain it to them.

Distinguish causation-in-fact from proximate causation. Remember there are a number of ways to describe cause in fact. If I tip this pitcher of water over, I caused the spill. That's fine—that's everyday language. The legal system, however, has developed some terms of art, and all of you are familiar with the "but for" concept of causation. But for the presence of an environmental agent this injury would never have happened. But for the collision this accident would never have happened. But for this or that, an event would never have occurred. You are also familiar with the "sine qua non" approach to legal causation, at least when it comes to factual causation. A helpful scenario for

6. 35 F.3d 717 (3d Cir. 1994).
7. 753 F.2d 1224 (3d Cir. 1985).
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introducing physicians and scientists to the distinction between factual causation and proximate causation is the “strike a match” scenario. In Philadelphia, if I strike a match at Independence Hall and the building burns down, and then the adjacent building is consumed, and everything burns between Independence Hall and the Spectrum or Veteran’s stadium, it’s obvious that the person who struck the match caused-in-fact the conflagration all the way to Veteran’s stadium.

To introduce the idea of proximate cause, I ask: “Are there any other factors or ‘causes’ that may have contributed to such an extensive fire? And should we hold the matchstriker liable for all, or just part, of the damage done by the fire? Was it foreseeable that striking a match in Independence Hall would lead to the destruction of Veteran’s stadium?” When the concept of foreseeability was introduced into American law, the question for the courts became: “At what point do we draw a line and say that cause-in-fact is no longer ‘cause’ for purposes of awarding compensation for injuries?” Foreseeability in the law is often a new concept for physicians and scientists. They are typically surprised to learn that the term “proximate cause” means more than cause-in-fact. With proximate causation, we’re incorporating policymaking into the identification of the causal agent(s) that the law will hold ultimately responsible for the foreseeable aspects of harm.

A third notion of causation—probabilistic causation—has also evolved over the last century. Probabilistic causation relies on probabilistic reasoning rather than on simple, deductively derived causal chains. Problems have arisen however because, as explained below, probabilistic reasoning can serve two analytically distinct purposes in legal proceedings.

Let’s return to basic civil procedure and basic burdens of proof. Traditionally, a plaintiff has two tasks, or burdens of proof. First, the plaintiff must produce evidence or facts for each element of a particular cause-of-action. Second, the plaintiff must persuade the factfinder that the plaintiff’s version of the facts is worthy of their collective belief with a minimum level of certainty, as defined by a standard of persuasion. The four commonly used standards are: a) “beyond a reasonable doubt” in criminal cases; b) “by clear and convincing evidence” in some civil cases; c) “more likely than not;” or d) “by a preponderance of the evidence” in most civil cases, including toxic tort and

occupational disease claims.

Qualitative concepts of probability (as embodied in the above standards) have long and explicitly influenced jury deliberations as to whether or not the plaintiff has met the burden of persuasion. By contrast, in conventional personal injury litigation, probability and inductive reasoning have not explicitly played a role in factfinding per se. That is, the facts themselves, defined as elements on which one party has the burden of production, are generally deemed true or false—with a probability of either 0 or 1. For example, the light was either red or green, the brakes either did or didn’t work, or the pedestrian either did or didn’t fall.

Among the elements of a case which the plaintiff has the burden of proving is causation-in-fact. This element is common to toxic tort, hazardous waste, occupational disease, and conventional traumatic injury claims. As noted above, causation-in-fact probability is not an issue in most conventional injury cases. The jury simply decides which version of the facts it believes in an all-or-none, yes-or-no fashion, with no room for intermediate probabilities. Causation evidence is not expressed probabilistically.

This is not so in late 20th century environmental claims where, given the frequent impossibility of proving individual causation, statistical causation evidence (expressed probabilistically) is required as a factual estimate of a defendant’s contribution to the plaintiff’s risk. For example, the issue in a typical trauma case may be whether or not a car could have stopped at a red light. Evidence might be heard on speed, braking ability, and driver reaction time for that particular vehicle (car $X$). The jury then finds that car $X$ either could or could not have stopped. However, in the absence of facts concerning the individual car, undisputed evidence may show that of 100 cars chosen at random, 55 would have been able to stop. As to whether or not the plaintiff has met the burden of proof, the jury could find either way, depending on how it responds to probabilistic (statistical) evidence. Jury response is, in turn, likely to be influenced by judicial instructions on inferences to be drawn from group-based information.

The jury may believe that 55% of cars could have stopped, but have no idea whether car $X$ is among that group. Thus, the jury would say that the plaintiff had not met the burden of proof.

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Alternatively, the jury may believe that 55% of cars could have stopped and infer that car X (assuming it is not atypical) more likely than not would have stopped since most cars would have. This finding, however, incorporates an inference from established fact about a population to a conclusion about a particular car.

The propriety of this kind of inference is one of the most controversial aspects of toxic tort and occupational disease cases, where causation often cannot be properly formulated as a yes-or-no fact. Instead, parties rely on evidence of increased risk or enhanced probability of disease which may or may not be attributable to the defendant's conduct. The inquiry becomes one of the existence and magnitude of a fact probability. Therefore, understanding the dual nature of probability, as both a factual statistical quantity (fact probability) and a measure of strength of belief (belief probability), becomes important. Unfortunately, fact probability and belief probability have not been kept analytically distinct. Courts have "collapsed" the requirements for burden of proof and burden of persuasion into one test which blurs the plaintiff's two-fold task of defining not only the facts or elements to be proven but also the amount of credence to be accorded a fact in support of a finding. When a judge tells a jury that "the plaintiff must show that causation is more likely than not," the judge risks confusion. Does the judge mean that the fact of causation which the plaintiff must prove (burden of proof) is not traditional true-or-false (100% vs. 0%) causation but only the existence of a statistical probability of causation greater than 50%? Or does the judge refer to the burden of persuasion guided by a standard of belief that causation is "more likely than not" true; that is, does the jury believe a knowable fact with more than 50% confidence?

Concern over haphazard and unrecognized transfer of "preponderance of evidence" or "more likely than not" standards from the burden of persuasion to the burden of factual proof involves more than idle semantics. The adverse effects of failure to undertake a deliberate, two-step probabilistic analysis include: a) undue preference for particular probabilities of causation found in one epidemiological study, especially when meta-analysis of multiple studies is not possible or available; b) unrecognized lowering of the burden of proof with concomitant stiffening of the burden (standard) of persuasion; c) inappropriate fixation on simplistic quantitative rules such as the ">50% likelihood" rule; and d) poorly reasoned opinions because courts fail to explain exactly how they apply the >50%, "more-likely-than-not" rule.
Courts that apply the rule only to fact probabilities essentially seek a yes-or-no belief in a >50% fact probability. By contrast, traditional courts that apply the rule only to belief probabilities seek a >50% belief in a yes-or-no fact. In toxic tort/occupational disease claims where both fact probability and belief probability are issues, there are at least two other approaches. Courts could apply the “more-likely-than-not” standard jointly, reducing alleged fact probability by a factor reflecting the jury’s doubt about its truth. By contrast, the rule could be applied sequentially to require only a >50% belief in a fact probability which itself may barely exceed the >50% threshold. It is important to see that joint application stiffens the causation burden-of-production/burden-of-persuasion, while sequential application substantially lessens the causation production/persuasion requirements. The point here is that, regardless of approach, a court that deals with causal indeterminacy characteristic of toxic tort/occupational disease claims should be explicit about what it is doing, especially if the defendant’s culpability of conduct or duty to prevent risk is factored into determination of the causation issue.

You may be thinking: “Judges and juries don’t operate at this level; we let the chips fall where they may and we can’t get into a juror’s mind.” But as jurists, you ought to at least develop a framework for thinking about what kind of evidence you’re going to allow and what kind of evidence you’re going to exclude. The kind of analysis reviewed here is offered to help you clarify your own thought processes.

Let’s talk about the use of experts. In toxic tort and product liability litigation, where do experts get used? It typically depends on what the plaintiff believes has to be proven. Most likely, you’re going to have experts testify for one or more of five purposes. First, the plaintiff must prove exposure. Toxicologists aren’t necessarily the people that prove exposure. Industrial hygienists, safety officers, environmental scientists, geologists, and others get involved at this stage of the litigation. Next, you bring somebody in to prove a causal relationship between the alleged toxic substance and the harm. We’ve talked about the burden of production, the burden of persuasion, and the standard of persuasion. Again, remember that the definition of compensable injury is ultimately going to drive the quantum and nature of the proof offered to establish a relationship between the substance and the harm.

Third, diagnosis or proof of harm is typically required. Again, the characterization of the injury is crucial. It’s much easier to prove “presence” or “impact” than it is to prove “adverse effect,”
"disease," or "disorder." And many courts do not require the testimony of a physician toxicologist in tort cases involving toxic chemicals. There are about 6,000 individuals in the U.S. who hold themselves out as toxicologists. Analytical toxicologists, who are typically responsible for laboratory testing and its proper interpretation, are most often associated with crime labs, clinical labs, or independent operations. Some analytical toxicologists participate in the legal process; others do not. Those that do are frequently called "forensic toxicologists." Regulatory toxicologists, by contrast, work in state or federal agencies or in the pharmaceutical industry where they undertake risk assessments or assure the safety of drugs or devices before marketing. Veterinary toxicologists usually confine their practices to the care of animals, while physicians trained in various medical specialties practice as medical or clinical toxicologists treating poisoned patients and consulting with poison control centers or other physicians. Finally, there's a group of several thousand scientists who are interested in the fundamental biochemical mechanisms by which chemicals injure cells. These professionals are known as research, biochemical, or mechanistic toxicologists.

There are several certifying boards for practitioners of toxicology. The American Board of Toxicology has certified over 1500 Ph.D. or other doctoral level individuals practicing in government, industry, or academia. The American Board of Clinical Chemistry has certified approximately 100 Ph.D.'s as toxicological chemists. The American Board of Forensic Toxicology has certified several hundred forensic professionals. The American Board of Veterinary Toxicology has certified about 100 veterinarians. Finally, the American Board of Medical Specialties has designated Medical Toxicology as a subspecialty with a joint certification process sponsored by the American Boards of Emergency Medicine, Pediatrics, and Preventive Medicine.

Thus, many toxicologists who provide expert testimony are not physicians; indeed, a significant number of those who testify have not acquired any doctoral credentials. Their testimony, nevertheless, is valued and desirable and has been admitted into evidence for decades. Importantly, toxicology is one of the few arenas in which courts allow non-physicians to render both diagnostic and causation opinions. You need these experts to prove the fourth and fifth elements of the plaintiff's toxic tort case, namely, that the cognizable harm is consistent with the exposure, and that the defendant was responsible for the harm. Of course, other types of expertise may be required to prove that the defendant's product, activity, or release contributed to the plaintiff's injuries.
Regarding the silicone breast implant controversy, let me alert you to the concerns that led the FDA to severely limit the market for these devices. The agency issued a report in the early 1990's detailing what it believed to be the biologic risks associated with implantation of devices containing polydimethylsiloxane. The FDA said there were immunologic risks, sensitization risks, risks from leakage, risks from infection, capsular contraction, and calcification, and risks from migration or degradation of silicone in other parts of the body. Concern was also expressed about the possibility of autoimmune disorders, carcinogenicity, teratogenicity, and interference with interpretation of mammography. To this day, however, the FDA has never stated that silicone implants have been shown to "cause" systemic injury or illness.

In my view, the silicone breast implant litigation could redefine legal concepts of soft tissue injury.10 I previously suggested that plaintiffs' verdicts primarily reflect jury outrage over the failure of manufacturers to employ 1995 standards in safety evaluations undertaken during the sixties and seventies. However, the nature of the scientific evidence that's being presented to enable plaintiffs to withstand motions for summary judgment also merits scrutiny. To support a causal connection between exposure to implants and the development of novel or previously unrecognized connective tissue or autoimmune disorders, plaintiffs with unremarkable physical examinations have relied on subjective reporting of symptoms plus the results of laboratory tests that indicate the presence of antibodies to silicone or other foreign substances, and/or the presence of autoantibodies. Plaintiffs' counsel have sought to equate the combination of unverifiable symptomatology and antibodies to silicone with legally compensable injury. I want to emphasize the novelty and potentially far-reaching ramifications of this characterization of injury for law, science, and medicine.

The production of antibodies is a normal and expected response to the presence of foreign substances. And the regulated production of autoantibodies to human proteins or antigens is also a normal, expected, required event. Only when these required normal events get out of control do you have the potential for injury or disease. The mere presence of autoantibodies is not sufficient for a designation of autoimmune injury or disease. Let me emphasize that loss of immune tolerance or loss of immune

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regulatory control has yet been shown to be the result of implantation of silicone devices. Indeed, the American Rheumatology Association has stated that evidence of injury or harm to the immune system has not been shown in silicone breast implant recipients. Therefore, the ground-breaking question for law, science, and medicine is whether or not to redefine or significantly expand traditional concepts of diseased or injured persons to include those who report symptoms and demonstrate expected, required, normal responses or results during laboratory testing.

Q. Many times we've seen that 15-20 years later in asbestosis or silicosis, where people said there's no causal relationship and these people were otherwise healthy, we have nothing else to explain it and there is a latency. I can remember dealing with a case many years ago where a woman complained of something and was denied social security disability benefits, and fourteen years later it appeared in medical literature, and nine years later in psychiatric literature. She reported the same symptomatology for thirteen years and was called a liar. Women are not believed but a man saying it is important, it has a different perspective. How do you factor that?

A. Questions about latency arise frequently in many toxicologic contexts. At least two of the pneumoconioses have been linked in the epidemiologic literature to one or more tumors. There is, however, to my knowledge, no evidence that the incidence or prevalence of cancer is increased in women with silicone breast implants. The FDA is one of the few groups that I'm aware of that has implied that the risk of tumorigenesis from exposure to silicone implants may be significant. The reason for the fundamental tension that you are raising is that we don't know what to do with the latency problem. No one knows what to do because, for most exposures, you can't predict it, at least not yet.

Ideally, each decision in mass tort litigation or in individual cases should rise or fall on the merits. The issue of latency may or may not be addressed. It is possible that in ten or twenty years, some women exposed to silicone will develop either a well-known disorder or a heretofore not previously described disorder. Obviously, anything is possible and any expert worth his or her salt will acknowledge that in this or any other forum.

You have to take each environmental agent and each symptom or objective manifestation and ask yourself: “What can science and medicine tell us about possible relationships?” At this point, I do not believe we can causally link silicone, as studied in the test tube, tissues, or elsewhere, with any of the systemic
problems that have been described. The only objective evidence that has been brought to the table is data from laboratory tests. And I have just now suggested to you that the results of those tests are not necessarily abnormal or indicative of harm, injury, or disease.

Q. Don't they have toxicological evidence from other studies?
A. For silicone? Now you've opened another Pandora's box in the breast implant litigation. The silicon atom takes many different forms in nature. Some of these forms cause various biologic effects, but most do not. Although a few reports suggest that some forms of silicon may enhance the immunogenicity of other chemicals in test tube experiments, reproducible evidence of tissue damage has not been shown for the forms and concentrations of silicone found in breast implants. From the toxicology, chemistry, and pathology perspective, let me emphasize that the chemical configuration in which the silicon atom is found in implants is distinctly different from that for the silicon associated with silicosis. Silicon is not silicon is not silicon. You have to know what form of silicon you're dealing with before you render any opinions about its possible biologic effects. For example, the polydimethylsiloxane found in breast implants must be chemically and toxicologically distinguished from silicon dioxide and the silicates.

What I am trying to impart here is that we are on the precipice of doing something unprecedented in American medicine and in American jurisprudence. With the exception of psychiatric diagnoses, we have not yet recognized compensable injuries, diseases, or disorders solely on the basis of subjectively reported symptoms. Those individuals who report symptoms in the context of multiple chemical sensitivities or silicone breast implantation genuinely believe they have a physical (non-psychiatric) problem, and no reasonable physician is going to question that these patients perceive that they have an illness. But in the absence of objective evidence of physical or laboratory abnormalities, how should American medicine and jurisprudence respond to those who say that they have been harmed by various forces in their environment? Jurists will play a key role in determining what kinds of objective evidence, if any, are going to be required or admissible to support subjective reporting of symptoms. In my opinion, jurists could enhance the public confidence in principled legal decisions by more frequently articulating the reasons why they chose to admit or exclude novel or controversial types of expert testimony.

Let me remind you of some of the problems that remain for the jurist post-Daubert. Does Daubert apply to jury as well as
non-jury trials? Does the decision apply to all, or just novel expert testimony? Does Daubert apply only to "scientific" experts? In appellate courts, how much de novo review is warranted? As gatekeepers, you may need to look at several aspects of an expert's testimony, including the theory or reasoning behind it, the methodology or technique employed, the protocols followed, the data generated, the conclusion reached, or the interpretation of the opinion in a legal context. Daubert seems to indicate that the Federal Rules only require the reasoning or methodology underlying the testimony to be scientifically valid. If either of these aspects of an expert's opinion have the indicia of validity, it would appear that testimony governed by the Federal Rules should be admitted. But is the Daubert approach restricted to assessment of the validity of theory or method, or should courts look behind apparently legitimate reasoning or technique and evaluate the legitimacy of the results as embodied in proper following of protocols, generation of data, and reaching of conclusions? In the same vein, is the apparent Daubert distinction between theory and methodology realistic or useful? Perhaps, but courts must recognize that two experts operating under the same generally accepted theory may employ radically different methods, each of which may be generally accepted in one scientific community but not in the other. In those situations, courts must recognize that experts from different disciplines often make certain assumptions that can never by "scientifically" proven, and that these assumptions may lead legitimate experts to equally logical, but clearly opposite conclusions. Regarding the indicia of validity and reliability, how should courts weigh the factors enunciated by the Daubert Court? How do courts factor the importance of peer review, publication, testing, rates of error, the existence or lack of standards, and the notions of widespread or general acceptance? The Court did not state that any one indicator of validity or reliability is essential under the Federal Rules of Evidence. And finally, what is meant by reliability and validity? The scientific and medical literature definition of these terms is quite different from the definitions used by the Supreme Court and legal commentators. In science and medicine, reliability refers to precision or reproducibility, while validity basically refers to accuracy. By contrast, many legal commentators and courts have equated reliability with accuracy or the probability of accuracy, and validity with sound reasoning. Jurists should not be surprised that scientists or physicians may not understand or accept the meanings or connotations that courts and some members of the bar have applied to the terms "reliability” and “accuracy.” Thus, it would appear that uniformi-
ty of approach to the admissibility of scientific evidence will not easily be accomplished on the heels of *Daubert*.

Q. We've heard many times, whether under *Frye v. United States*¹¹ or *Daubert*, that ours is an advocacy system and it is not a magisterial system. Everything you're saying I would rely on the defense attorney to do. If a guy walks into court and is the greatest b.s. artist, I have no way of knowing whether it is the truth. If he's made a great impression and I'm the trier of fact, he may have won me over to his side. I have no other way of knowing anything about him, other than what I hear in the courtroom. I would assume that if you're a total fraud, that the defense attorney in an adversarial role would show that to me. I would rely upon the advocates. I cannot use my own personal opinions, but this is the theme that I've gotten out of this. A group of forensic experts is deeply concerned that false science is taking over the system and clogging the courts. But under an adversarial system, the judges can't get into that other than in each individual courtroom.

A. All I'm suggesting is that counsel meet those burdens and do their jobs with varying levels of sophistication. I guess that's almost stating the obvious, but our job here is to alert you conceptually to what's going on so that you can at least begin to sniff out that which is fraudulent from that which is not and that which is strong from that which is weak. But most of all, it comes back to what you as a jurist and what any fact-finder is going to recognize as compensable injury. The point is, as a society, we have simultaneously broadened both the concept of compensable injury and the cottage industry of expert testimony.

Q. Isn't this going to be a political decision? Isn't Congress or a legislature sooner or later going to say enough is enough?

A. I would say yes. Everything is ultimately a political decision, especially if we're going to work at the societal level.

Q. There are judges who are defense lawyers so you're going to get some rulings that favor one side or the other and that seem to depend on bias.

A. Perhaps an analogy could be made to the percentage of the GDP that is devoted to health care. As a society, we're at the 15% level now, and obviously one of the things we're grappling with is the projection that it could reach 20% to 25% of the GDP within our lifetimes. The question is, to what extent, as a legal system, do we want our courts to be dealing with toxic tort or product liability cases as opposed to any other kind of litigation?

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11. 293 F. 1013 (D.C. Cir. 1923).
We could make 25, 30 or 40% of all the cases brought in some courts this kind of litigation if we chose to do that. As long as the pressure remains to broaden the definition of compensable injury to include risk or latency or fear of harm, courts and society will increasingly have to decide if we want to compensate now for the possibility of what is perceived to be harm in the future. I can't overemphasize what I see as a tremendous push in our society to redistribute wealth in response to smaller and smaller increments of perceived injury. By the way, for those of you wanting to guess who might write future Supreme Court opinions in the toxic tort arena, I suggest you read Justice Breyer's book Breaking the Vicious Circle.12

Q. Isn't it more likely that this is not going to be resolved on a scientific basis? You're very persuasive on what you're saying until the business aspect of practicing law is considered.

A. At some point, as a society, we may decide we can't spend all this money such that a few people get millions and a lot of people get virtually nothing. We've got to figure out better ways to handle the various social costs associated with new inventions and new creations.