

employer) who can in some way be held liable in tort. Third parties targeted in such suits may include independent contractors who are responsible for the exposure or manufacturers of the hazardous materials, who are sued on a defective products strict liability theory. In *Chevron Chem. Co. v. Ferebee*, 736 F.2d 1529 (D.C. Cir. 1984), discussed above in note 2, Ferebee's claim against Chevron was based on a Maryland products liability law that requires warnings to consumers of a product's known dangers. On a failure to warn theory, the plaintiff must prove all of the causal links from exposure to injury, i.e., that "paraquat proximately caused Mr. Ferebee's illness and death" and also that "the inadequacy of the warning proximately caused Mr. Ferebee's illness and death." Warnings were on the package, but they did not refer to dermal absorption and lung disease. The warning, "CAN KILL IF SWALLOWED, HARMFUL TO THE EYES AND SKIN," certainly indicated that Paraquat isn't an appropriate tonic or skin lotion, but the warning did not specifically mention adverse consequences of dermal contact other than skin irritation. This failure in the warning to disclose consequences of exposure known to Chevron was deemed sufficient by the appellate court to support the jury finding in Ferebee's favor.

#### **Landrigan v. Celotex Corporation**

Supreme Court of New Jersey, 1992

127 N.J. 404, 605 A.2d 1079

POLLACK, J. Plaintiff, Angelina Landrigan, sued defendants Owens-Corning Fiberglass Corporation and Owens Illinois, Inc. for the personal injuries and death of her husband, Thomas Landrigan, claiming that exposure to defendants' asbestos had caused his death from colon cancer...

Decedent worked as a maintenance man and pipe insulator at the Bayonne Terminal Warehouse from 1956 until December 1981, when he was diagnosed as suffering from colon cancer. From 1956 until 1972, he allegedly worked with insulation containing asbestos supplied by defendants. In January 1982, he underwent surgery but the cancer spread, and he died in December 1982. The cause of his death was adenocarcinoma, "a malignant adenoma arising from a glandular organ," the most common type of colon cancer. Generally speaking, colorectal cancer is the second most common cancer in the United States, striking 140,000 persons and causing 60,000 deaths annually. In 1984, plaintiff filed this survivorship and wrongful death action, asserting that exposure to asbestos had caused decedent's death.

At the trial in 1989, plaintiff relied on two experts, Dr. Joseph Sokolowski, Jr., a physician who is board certified in both internal medicine and pulmonary medicine, and Dr. Joseph K. Wagoner, an epidemiologist and biostatistician but not a physician. Dr. Sokolowski never treated or examined decedent. He based his conclusions on a review of decedent's history of exposure to asbestos, the absence of other risk factors in decedent's history, and on various epidemiological, animal, and in vitro studies. Stating that physicians regularly rely on epidemiological studies, Dr. Sokolowski testified that asbestos can cause colon cancer in humans. He also described the path asbestos fibers take from inhalation to the gastrointestinal tract. Dr. Sokolowski testified that exposure to asbestos was the cause of decedent's colon cancer...[and] further that decedent would not have contracted colon cancer if he had not been exposed to asbestos.

Plaintiff also offered Dr. Wagoner to testify that asbestos exposure had caused decedent's colon cancer. After conducting a hearing pursuant to Evidence Rule 8, the trial court ruled that

as an epidemiologist and not a physician, Dr. Wagoner was not qualified to testify that asbestos had caused decedent's cancer. The court, however, permitted the witness to testify about epidemiological methods and studies linking colon cancer to asbestos exposure. It also allowed Dr. Wagoner to state his opinion that asbestos causes colon cancer in humans.... At the close of plaintiff's case, the trial court granted defendants' motions for a directed verdict.

In recent years, we have sought to accommodate the requirements for the admission of expert testimony with the need for that testimony. Nowhere is that accommodation more compelling than on the issue of causation in toxic-tort litigation concerning diseases of indeterminate origin. Many such injuries remain latent for years, are associated with diverse risk factors, and occur without any apparent cause. Steve Gold, Note, Causation in Toxic Torts: Burdens of Proof, Standards of Persuasion, and Statistical Evidence, 96 Yale L.J. 376, 376 (1986). In that context, proof that a defendant's conduct caused decedent's injuries is more subtle and sophisticated than proof in cases concerned with more traditional torts....

Traditionally, plaintiffs have established a connection between tortious conduct and personal injuries through the testimony of medical experts who testify that the defendant's specific conduct was the cause of the plaintiff's injuries. Toxic torts, however, do not readily lend themselves to proof that is so particularized. Developments in the Law-Toxic Waste Litigation, 99 Harv. L. Rev. 1458, 1620 (1986). Plaintiffs in such cases may be compelled to resort to more general evidence, such as that provided by epidemiological studies. A basic understanding of some fundamentals of epidemiology is essential for an assessment of the admissibility of such evidence.

Simply defined, epidemiology is "the study of disease occurrence in human populations." Gary D. Friedman, *Primer of Epidemiology* 1 (3d ed. 1987). Epidemiology studies the relationship between a disease and a factor suspected of causing the disease, using statistical methods to determine the likelihood of causation. Bert Black & David E. Lilienfeld, *Epidemiologic Proof in Toxic Tort Litigation*, 52 Fordham L. Rev. 732, 750 (1984). By comparison to the clinical health sciences, which are directly concerned with diseases in particular patients, epidemiology is concerned with the statistical analysis of disease in groups of patients. The statistical associations may become so compelling, as they did in establishing the correlation between asbestos exposure and mesothelioma, that they raise a legitimate implication of causation. "Statistical associations," however, "do not necessarily imply causation.... It is important, therefore, to have some basis for deciding whether a statistical association derived from an observational study represents a cause-and-effect relationship." Friedman, *supra*, at 182-183. See Austin B. Hill, *The Environment and Disease: Association or Causation?*, 58 Proc. Royal Soc. Med. 295 (1965) (criteria to assess likelihood of causal relationship from statistical associations).

At oral argument, defendants, for example, stressed two criteria, among others, that are crucial in determining whether a statistical association will give rise to an inference that a particular substance causes a certain disease in people who are exposed to it. The two criteria are the strength of the association and the consistency of any such association with other knowledge. The argument is sound. As Professor Friedman explains:

In general, the stronger the association, the more likely it represents a cause-and-effect relationship. Weak associations often turn out to be spurious and explainable by some known, or as yet unknown, confounding variable.... Strength of an association is usually measured by the relative risk or the ratio of the disease rate in those with the factor to the rate in those without. The relative risk of lung cancer in cigarette smokers as compared to nonsmokers is on the order of 10:1, whereas the relative risk of pancreatic cancer is about 2:1. The difference suggests that cigarette smoking is more likely to be a causal factor for lung cancer than for pancreatic cancer.

If the association makes sense in terms of known biological mechanisms or other epidemiologic knowledge, it becomes more plausible as a cause-and-effect relationship. Part of the attractiveness of the hypothesis that a high-saturated fat, high-cholesterol diet predisposes to atherosclerosis is the fact that a biologic mechanism can be invoked. Such a diet increases blood lipids, which may in turn be deposited in arterial walls. A correlation between the number of telephone poles in a country and its coronary heart disease mortality rate lacks plausibility as a cause-and-effect relationship partly because it is difficult to imagine a biologic mechanism whereby telephone poles result in atherosclerosis. [Friedman, *supra*, at 183–184.]

The “attributable risk,” by comparison, is the proportion of the disease that is statistically attributable to the factor. Black & Lilienfeld, *supra*, 52 Fordham L. Rev. at 761. It “is a composite measure that takes into account both the relative risk of disease if exposed and the proportion of the population so exposed.”...

Plaintiff’s medical expert was Dr. Sokolowski. Initially, he explained that he had examined certain literature on colon cancer, including the landmark study by Dr. Irving Selikoff. See Irving Selikoff, et al., *Mortality Experience of Insulation Workers in the United States and Canada*, 330 *Annals N.Y. Acad. Sci.* 91 (1979). The study indicated a relative risk of colon cancer from the exposure to asbestos of 1.55. The attributable risk, which would vary according to the extent and intensity of the exposure, was approximately thirty-five percent. Thus, assuming a causal relationship, the Selikoff study indicates that thirty-five percent of the cases of colon cancer in the population exposed to asbestos can be attributed to that exposure.

Dr. Sokolowski had never treated or examined decedent, but he had reviewed decedent’s medical records and plaintiff’s answers to interrogatories. Those materials indicated that decedent had been exposed to asbestos in his work. They also indicated the absence of other risk factors such as a family history of colon cancer, a high-fat diet, and the undue consumption of alcohol. Dr. Sokolowski acknowledged that “many studies...show no statistically significant increase in colon cancer in workers exposed to asbestos.” Finally, he relied on the results of animal and in vitro studies.

The trial court rejected Dr. Sokolowski’s testimony as a “net opinion” unsupported by any facts. Specifically, the court stated that “epidemiological evidence can only be used to show that a defendant’s conduct increased a plaintiff’s risk of injury to some measurable extent but it cannot be used to answer the critical question did the asbestos cause Mr. Landrigan’s colon cancer.”

The Appellate Division agreed with that assessment, explaining that Dr. Sokolowski had failed to account for other factors that may have caused decedent’s cancer. Although it accepted the validity of the Selikoff study, the court stated that the 1.55 relative risk was insufficient to support Dr. Sokolowski’s opinion that decedent’s exposure had caused the cancer. Without expressly adopting a specific standard, the court cited with approval several cases that adopted a requirement that an epidemiological study show a relative risk in excess of 2.0 to prove that causation in a specific individual was more probable than not. The significance of a relative risk greater than 2.0 representing a true causal relationship is that the ratio evidences an attributable risk of more than fifty percent, which means that more than half of the cases of the studied disease in a comparable population exposed to the substance are attributable to that exposure. This finding could support an inference that the exposure was the probable cause of the disease in a specific member of the exposed population.

Defense counsel urges that the Appellate Division opinion may be read as requiring that an expert may not rely on an epidemiological study to support a finding of individual causation unless the relative risk is greater than 2.0. At oral argument before us, they agreed that such a

requirement may be unnecessary. Counsel acknowledged that under certain circumstances a study with a relative risk of less than 2.0 could support a finding of specific causation. Those circumstances would include, for example, individual clinical data, such as asbestos in or near the tumor or a documented history of extensive asbestos exposure. So viewed, a relative risk of 2.0 is not so much a password to a finding of causation as one piece of evidence, among others, for the court to consider in determining whether the expert has employed a sound methodology in reaching his or her conclusion....

The court must also examine the manner in which experts reason from the studies and other information to a conclusion. As previously indicated, that conclusion must derive from a sound methodology that is supported by some consensus of experts in the field.

In the present case, Dr. Sokolowski began by reviewing the scientific literature to establish both the ability of asbestos to cause colon cancer and the magnitude of the risk that it would cause that result. Next, he assumed that decedent was exposed to asbestos and that his exposure, in both intensity and duration, was comparable to that of the study populations described in the literature. He then assumed that other known risk factors for colon cancer did not apply to decedent. After considering decedent's exposure and the absence of those factors, Dr. Sokolowski concluded that decedent's exposure more likely than not had been the cause of his colon cancer.

Without limiting the trial court on remand, its assessment of Dr. Sokolowski's testimony should include an evaluation of the validity both of the studies on which he relied and of his assumption that the decedent's asbestos exposure was like that of the members of the study populations. The court should also verify Dr. Sokolowski's assumption concerning the absence of other risk factors. Finally, the court should ascertain if the relevant scientific community accepts the process by which Dr. Sokolowski reasoned to the conclusion that the decedent's asbestos exposure had caused his cancer. Thus, to determine the admissibility of the witness's opinion, the court, without substituting its judgment for that of the expert, should examine each step in Dr. Sokolowski's reasoning.

Our decision does not necessarily mean that on remand the trial court must reach a different result. Although the diagnosis of decedent's disease and the cause of his death are not in dispute, the parties vigorously contest the probability that decedent's colon cancer was caused by asbestos exposure. The issue posed to both Dr. Wagoner and Dr. Sokolowski was the likelihood that decedent's colon cancer was caused by asbestos exposure. Dr. Wagoner did not rely exclusively on epidemiological studies in addressing that issue. In addition to relying on such studies, he, like Dr. Sokolowski, reviewed specific evidence about decedent's medical and occupational histories. Both witnesses also excluded certain known risk factors for colon cancer, such as excessive alcohol consumption, a high-fat diet, and a positive family history. From statistical population studies to the conclusion of causation in an individual, however, is a broad leap, particularly for a witness whose training, unlike that of a physician, is oriented toward the study of groups and not of individuals. Nonetheless, proof of causation in toxic-tort cases depends largely on inferences derived from statistics about groups. Gold, *supra*, 96 Yale L.J. at 401....[Reversed and remanded.]

#### COMMENTARY & QUESTIONS

**1. Epidemiology, relative risk, and attributable risk.** Although the *Landrigan* opinion is not very explicit on this point, relative risk (RR) and attributable risk (AR) are, in a sense, reciprocal measures. When the relative risk is 2.0 (sometimes expressed as 2:1), the attributable risk is 50% (.5). What this relative risk of 2.0 or 2:1 means in popular parlance is that a person with that risk factor is twice as likely as someone without the

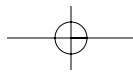
factor to have the correlated harm occur. The precise equation that relates the two values is  $RR-1/RR=AR$ . So, for example, in the cigarette smoking to lung cancer example, where the relative risk is 10, the attributable risk is 90% (.9), i.e., nine of the ten lung cancers that befall smokers are attributable to smoking cigarettes rather than to other factors. In the Selikoff study, the relative risk is 1.55, so the attributable risk is  $(1.55-1)/1.55=.35$ , or 35%. For a good introductory discussion to the forms of epidemiological data and its use in toxic tort litigation, see Dintzer & Mosher, *Epidemiological Evidence in Toxic Tort Cases*, 17 *Nat. Resources & Env't* 222 (2003).

**2. Legal causation and statistical proof: comparing apples and pears.** What makes a relative risk value of 2.0 appear to be particularly important in the legal arena? A relative risk value in excess of 2.0 makes it appear more likely than not that the risk factor is the operative cause of the injury. This linguistic formulation tracks the standard that the plaintiff must meet to carry the burden of proof on the issue of causation-in-fact. That is, the plaintiff has the burden of persuading the trier of fact that defendant's action is more likely than not the cause of plaintiff's injury. Using the term *attributable risk* makes this linguistically even clearer; when the risk attributable to defendant is more than half (>50%) of the total risk, it is easy to conclude that the defendant's act is more likely than not the cause of the plaintiff's injury.

The court in *Landrigan* makes it very clear that failure to establish a relative risk value in excess of 2.0 is not fatal to a plaintiff's case. Why shouldn't it be? The court's answer is that other factors could be proved in the case that would make it more likely in this particular case that the risk factor for which the defendant is responsible is the operative cause of the particular plaintiff's injury. Examples include specific clinical evidence or proof of extraordinary exposures. What about the flip side of the issue — will proof of a relative risk in excess of 2.0 always result in a decision in plaintiff's favor on the issue of cause-in-fact? It should be obvious that the same type of individualized proof that can allow a plaintiff to win despite a relative risk that does not exceed 2.0 can also allow a defendant to prevail in a case of relative risk higher than 2.0.

**3. Legal causation and statistical significance: comparing apples and pear melba.** When the term *significant* appears in legal discourse, it usually has its lay meanings that speak to the importance of a factor or that contrast the central with the peripheral. In relation to scientific studies that find a correlation significant (as between an exposure and the subsequent onset of disease), the word *significant* is a term of art that describes the reliability of the linkage. To be more precise, the significance of a correlation is the likelihood that the data observed are not the product of mere random chance. In a case of radiation exposure, one judge explained statistical significance this way:

Where there is an increase of observed cases of a particular cancer or leukemia over the number statistically "expected" to normally appear, the question arises whether it may be rationally inferred that the increase is causally connected to specific human activity. The scientific papers and reports will often speak of whether a deviation from the expected numbers of cases is "statistically significant," supporting a hypothesis of causation, or whether the perceived increase is attributable to random variation in the studied population, i.e., to chance. The mathematical tests of significance commonly used in research tend to be stringent; for an increase to



be considered “statistically significant,” the probability that it can be attributed to random chance usually must be five percent or less ( $p=.05$ ). In other words, if the level of significance chosen by the researcher is  $p=.05$ , then an observed correlation is “significant” if there is 1 chance in 20 — or less — that the increase resulted from chance. In scientific practice, levels of significance of .01 or .001 are used providing an even more stringent test of a chosen hypothetical relationship. *Allen v. United States*, 588 F. Supp. 247, 416 (D. Utah 1984).

Accordingly, from the legal viewpoint, a causal hypothesis that narrowly fails to satisfy a .05 level of statistical significance is not insignificant. For instance, data for which there is only a 1 in 19 chance of its being random fails a .05 significance test but nevertheless evidences a relationship for which “the probability is 94.73 percent or 18 chances out of 19 that the observed relationship is not a random event...[and] the certainty that the observed increase is related to its hypothetical cause rather than mere chance is still far more likely than not.” *Id.* What does all this mean for the trial of toxic tort cases? First, even causal hypotheses that are not statistically significant at traditional p-values used in scientific research may be significant proof that helps establish causation in fact.

**4. The plaintiff’s dilemma.** Under the *Landrigan* standard for proving causation, how often can plaintiffs win exposure-induced cancer cases where the epidemiological studies do not evidence a relative risk value in excess of 2.0? What will be the usual impact on available proof of the long latency periods between exposure and the onset of disease? Most likely, the delay will make it difficult for plaintiffs to obtain reliable proof of exposure levels and introduce, in the course of living, many confounding variables, such as other exposures or lifestyle choices, thereby reducing the chance of making the individualizing proofs the *Landrigan* court views as so important.

**5. The pitfalls of probabilistic proof.** A hot debate in the legal literature surrounds the advisability of allowing proof of probabilities to establish causation in fact. There are numerous good articles on this subject, two of the classics being Tribe, *Trial by Mathematics: Precision and Ritual in the Legal Process*, 84 Harv. L. Rev. 1329 (1971), and Nesson, *Agent Orange Meets the Blue Bus: Factfinding at the Frontier of Knowledge*, 66 B.U. L. Rev. 521 (1986).

Consider the following hypotheticals:<sup>28</sup> Following exposure to defendant’s toxic waste that leached into an aquifer, the cancer rate in a community rises from 10 per year to 19 per year (hypo 1) or 21 per year (hypo 2) and all other possible causes have been ruled out by undisputed expert testimony. If probability is translated uncritically into plaintiffs’ failure or success in carrying the burden of proof on the cause-in-fact issue, then in hypo 1 defendant goes free of liability to any of the 19 victims, and in hypo 2 defendant must compensate all 21 victims. Is such a result absurd? Do the hypotheticals make it clear why the *Landrigan* court is chary of making a relative risk of 2.0 a litmus for recovery?

Should plaintiffs be relieved of their usual burden of proof because of these proof problems? In *Allen v. United States*, 588 F. Supp. 247 (D. Utah 1984), noted above for its discussion of statistical significance, the court fashioned a more lenient standard that

28. The hypotheticals are adapted from Delgado, *Beyond Sindell*, 70 Calif. L. Rev. 881, 885 (1982).

adapted the substantial factor doctrine that was developed in the joint and several liability context to allow for proof of cause-in-fact in toxic exposure cases. *Allen* involved very compelling facts: the unannounced atmospheric testing of atomic weapons that resulted in mass exposure of the general public to ionizing radiation from the fallout released by the test. The government's lack of warning and haphazard monitoring of fallout levels made it impossible for citizens to avoid the exposure and, likewise, impossible for them to reliably prove their level of exposure. Still, the relative risk levels (a term not used in the *Allen* litigation) were far lower than 2.0. Although its precise ruling is elusive and not easy to summarize, the court allowed the plaintiffs to shift the burden of proof on the cause-in-fact issue (which the *Allen* court refers to as the factual connection issue) to the defendant by proving that the defendant's conduct was a substantial risk-increasing factor. Once plaintiff makes that showing, the defendant is to come forward with evidence that tries to weaken the factual connection between its acts and the plaintiff's injuries. In the end, no fixed rule is announced: "Whether any of these factual connections will lead to liability is, as Professor Thode reminds us, 'an issue involving *the scope of the legal system's protection afforded to plaintiff* and not an issue of factual causation.' Thode, *Tort Analysis: Duty-Risk v. Proximate Cause and the Rational Allocation of Functions Between Judge and Jury*, 1977 Utah L. Rev. 1, 6." (Emphasis by the court.) In short, the key issue becomes one of policy regarding the extent of protection offered by the legal system, the very essence of the proximate cause inquiry.

Are there other ways of relaxing the plaintiff's burden on cause-in-fact that are not fraught with unfairness to defendants? Professor Richard Delgado suggests allowing full recovery for the harms caused by defendant (9 persons harmed in hypo 1, 11 in hypo 2) to be shared by the entire class of plaintiffs. This forces defendant to bear its costs, and "probabilities are not used...to establish a causal link between conduct of a certain type and a particular injury." Delgado, *Beyond Sindell*, 70 Cal. L. Rev. 881, 905 (1982).

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## C. LAW AND SCIENCE IN THE TOXIC TORT CONTEXT

### Section 1. COMPETING CONCEPTIONS: LAW, SCIENCE, AND POPULAR PERCEPTION

In a very real sense, law and science are contrasting modes of ascertaining different varieties of truth.

- *Functional Differences.* The major function of the legal system is to resolve disputes efficiently, effectively, and equitably. The major function of science is to make accurate, empirically verifiable predictions about the physical world.
- *Conclusiveness vs. Tentativeness.* Because the primary function of private law (and to a lesser extent public law) is to finally resolve disputes so that they do not fester and threaten social cohesiveness, the legal system places a substantial value on the finality of decisions. Doctrines such as *res judicata*, collateral estoppel, double jeopardy, statutes of limitations, and the presumption against nonretroactivity of legislation support the need for finality of legal decisions. Science, with a goal of accurate prediction, can afford to wait. It proceeds by way