Epidemiology and the Law: Courts and Confidence Intervals

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ABSTRACT

Beginning with the swine flu litigation of the early 1980s, epidemiological evidence has played an increasingly prominent role in helping the nation's courts deal with alleged causal connections between plaintiffs' diseases or other harm and exposure to specific noxious agents (such as asbestos, toxic waste, radiation, and pharmaceuticals). Judicial reliance on epidemiology has highlighted the contrast between the nature of scientific proof and of legal proof. Epidemiologists need to recognize and understand the growing involvement of their profession in complex tort litigation. (*Am J Public Health.* 1991;81:1661–1666)

The law, wrote Oliver Wendell Holmes, Jr, "is forever adopting new principles from life at one end, and it always retains old ones from history at the other."[1] The common law (or case law), which was Holmes' subject, is continually recreated as existing legal principles are applied or modified to fit new fact patterns. And because the law deals with real-world facts, the legal system must keep appropriately abreast with new ways of seeing and understanding the world. This means that, as science develops increasingly more sophisticated and precise means of measurement and analysis, the nation's courts must struggle to decide how much legal weight to afford the never-ending stream of new scientific insights and techniques.

Earlier in this century, courts had to decide whether polygraph readings[2] and paternity test results[3] should be admitted as evidence in legal proceedings. Today's legal controversies include the admissibility of such new types of scientific evidence as DNA fingerprinting.[4] In each case, the judicial concern is one of determining if a particular area of science offers results that are valid and reliable enough to meet accepted legal standards of proof.

Epidemiology provides another example of this interaction of law and science. With the swine flu litigation of the early 1980s, epidemiological evidence began to play an increasingly prominent role in helping courts determine whether a plaintiff's disease or other harm was caused by some activity of the defendant. The increasing judicial reliance on epidemiology is dramatic. A computerized search of all reported federal and state judicial opinions found the words "epidemiology," "epidemiological," or "epidemiologist" appearing in three or fewer cases for each of the years 1970 to 1973. In 1990, 86 cases mentioned epidemiology. Figure 1 illustrates the steady and rapid increase in judicial attention to epidemiology. In 1984, D. H. Kaye did a similar search using the keywords "statistically significant" and "statistical significance." He found 519 cases, nearly two thirds of which were from the 4 years immediately preceding the search; only 7 were dated before 1970.[5]

There is a similar, more qualitative indicator of the legal system's increased attention to epidemiology. As judged by the number of conference announcements[6] and articles in legal journals[7–14] and trade papers[15] over the past year or two, the world created by the overlap of epidemiology and the law has indeed come into its own.

The Basics of Tort Law

The main force driving the increased use of epidemiology in the courtroom has been tort litigation. The law of torts determines when one person (or groups of persons, or corporation or government) must pay compensation for civil, noncontractual wrongs caused to others. The injuries addressed by tort law include specific types of intentionally inflicted wrongs (such as assault and battery, defamation, and invasion of privacy), as well as injuries inflicted unintentionally through fail-

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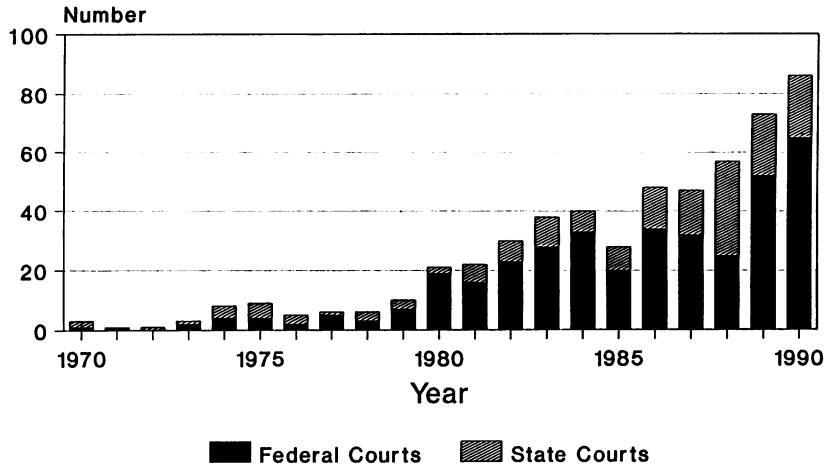

FIGURE 1—Number of times a word beginning with "epidemiol. . ." appears in a court opinion.

ure to exercise the care that could be expected of an ordinarily prudent person.

Two functions are served by recognizing these types of personal harm as a basis for private lawsuits.[16] The most obvious is *compensation:* to make up, through money damages, for harm that injured parties have suffered through no fault of their own. Such harm cannot be undone, but by bringing a negligence action, the victim attempts to shift the cost of the harm to the party responsible for causing it. The second function of tort law is the *prevention of harm* through enforced accountability. By making certain that undesirable and harmful behaviors are costly to the actor, tort law serves to deter such behavior by the defendant and others in the future. Because individuals can rarely secure either compensation or accountability outside the legal system, tort law gives the individual citizen a forum in which to complain on a more equal footing against a potentially mightier wrongdoer. From a social policy perspective, tort liability provides primary prevention of future harm.

For a claimant to succeed in a lawsuit alleging unintentional, negligent harm, four requirements must be met. The plaintiff must prove that (1) the defendant owed the plaintiff a duty to act in a particular way, (2) the defendant failed to fulfill that duty, (3) the plaintiff suffered harm, and (4) the defendant's breach of duty was the *cause* of the plaintiff's harm. The plaintiff bears the burden of demonstrating the existence of all four elements. This need not be proven beyond a reasonable doubt, as in criminal prosecutions, but simply by a preponderance of the evidence. If the plaintiff fails to prove any one of the four required elements by this criterion, the fact that the other elements have been satisfied will not matter; the plaintiff will lose.

Toxic Torts

During most of this century, tort law was concerned predominantly with injuries for which the cause-effect association was clear-cut: a car ran into a pedestrian, a shopper fell on a store's slippery floor, or a baby choked on a toy with small parts. The injury and the facts surrounding it were evident. More recently, however, tort law has been used to seek compensation for injuries in which causation is not provable by mere eyewitness testimony regarding a specific causal event.

At the heart of such litigation has been a new and rapidly growing area of tort law, usually labeled "toxic torts" but perhaps more appropriately referred to as "mass-exposure" or "environmental-injury litigation." Exposure to asbestos, toxic waste, radiation, and pharmaceuticals have led to large numbers of lawsuits in the past 15 years. In a sense, toxic torts could be viewed as one response to the harmful health effects resulting from the careless or irresponsible use of modern technology.

The common element linking these various lawsuits is that some activity or product of the defendant is alleged to be associated with increased rates of a particular type of harm, and the causal relationship between the exposure and the harm is not amendable to eyewitness testimony. Some harmful agents that have been involved in such lawsuits are dioxin, Agent Orange, low-level radiation, contaminated groundwater, lead paint chips, tampons leading to toxic shock syndrome, asbestos, diethylstilbestrol (DES), and various pharmaceuticals (including polio and flu vaccines as well as Bendectin).

These noxious agents have several things in common: (1) all have been alleged to cause harm to humans, (2) this harm has resulted in lawsuits, (3) the causal connection between the agent and the specific harm has been the subject of some specific controversy, and (4) this combination of factors has resulted in epidemiology and epidemiologists being brought into the courtroom. Whether the defendant is selling a pharmaceutical product, is accused of contaminating groundwater, or is responsible for the release of radioactive debris into the atmosphere, epidemiological evidence may be critical to showing that the defendant's actions are causally associated with the plaintiff's damage.

Toxic tort lawsuits do not differ fundamentally from the more familiar motor vehicle injury and product liability lawsuits. There is a victim/plaintiff and an allegedly culpable defendant. The harmful outcome was not sought by the plaintiff. Further, in most cases, the injury was the result of exposure to some form of energy: kinetic, chemical, thermal, electrical, or ionizing radiation.

With the more familiar types of injuries such as those involving motor vehicles, the harm produced is apparent a relatively short period of time after the traumatic event. With toxic tort injuries, on the other hand, there is usually a latency period between exposure and the development of noticeable harm. When harm becomes apparent decades after a toxic exposure, the documentation of a cause-effect relationship must rely on forms of proof that are new to the law. Greatly compounding this difficulty of proof is that few harms are limited to unique single-cause, single-effect connections. Most toxic tort harms can result from several causes, only one of which may involve the defendant. And the plaintiff may have been exposed to more than one noxious agent (e.g., tobacco and asbestos). Thus, it is not enough for toxic tort plaintiffs to show that factor X is capable of causing harm Y. Plaintiffs must also demonstrate that it is more likely than not that factor X caused *their* harm Y. The difficulty here is that, even when it is possible to demonstrate that factor X is responsible for a significant percentage of all cases of harm Y, it can rarely be proven that the harm Y suffered by a particular individual, the plaintiff, was one of the

cases caused by factor X. This means that, even where it can be demonstrated that the defendant is responsible for a significant number of the cases of a particular harm, no plaintiff can prove that he or she is one of these particular cases.

A few harmful substances are closely associated with certain signature diseases, such as DES and adenocarcinoma; in such cases, the disease is known to occur rarely, if ever, absent the substance. But these cases are the exception. The trial judge in the Agent Orange litigation explained the causation difficulty in that case:

> Plaintiff's factual case may be briefly summarized. Agent Orange contained small quantities of dioxon. Dioxin is a potent poison which can cause serious harm to humans. Many plaintiffs suffer from diseases that can be caused by dioxin. [Therefore, the plaintiffs' argue,] [d]ioxin caused the diseases. The logical and practical difficulty with their argument is that the diseases referred to may result from causes other than dioxin poisoning.[17]

Swine Flu

Epidemiology often provides the best means of demonstrating a causal connection in toxic tort cases. Yet the earliest such cases found the courts responding inconsistently and often unfavorably to the courtroom use of epidemiological evidence. Even though treating physicians can normally make only limited contributions to the determination of causation, judges and juries were more comfortable with the "eyewitness"—the treating physician who could say, "I took care of this patient and I *know* that her disease Y was caused by X." This is the type of evidence courts have relied on, by necessity, for many years.

Both plaintiffs and defendants in some of the early swine flu cases relied heavily on epidemiological studies conducted by the Centers for Disease Control (CDC), but the results of such reliance varied. *Sulesky v United States,* for example, was a 1982 case brought by a woman who had contracted Guillain-Barŕe syndrome with an onset 14 weeks after she had received a swine flu vaccination. She alleged that the vaccination had caused her neurological disorder. Most epidemiological studies of the 1976 flu immunization program concluded that there was no causative link between the swine flu vaccination and Guillain-Barŕe syndrome with an onset more than 10 weeks after immunization. Still, the court found for the plaintiff. The presiding federal district court judge noted that

> . . . this Court finds that none of the epidemiological studies introduced into evidence may be employed to *establish* the Plaintiff's case by a preponderance of the evidence. . . . [W]hile the Court has found the testimony and documentary evidence of the epidemiologists extremely valuable, and while it is not rejected out of hand, the Court does find the expert epidemiological testimony is not determinative of the issue of causation in this case.
> Rather . . . the Court finds that the resolution of the causation issue turns on the testimony of the treating and evaluating physicians.[18]

But contrast this judicial outlook to that enunciated by a different federal district court judge in another swine flue case decided in the same year. Presented with almost identical facts, the presiding judge in *Cook v United States* reached an opposite finding regarding causation:

> The etiology of GBS is not well understood and not readily demonstrated by clinical or laboratory evidence, particularly after such a latency period. Hence, plaintiff . . . relied on statistical correlation to establish causation, interpreting the CDC data differently than did the doctors who worked with CDC. . . .
> The dispute between the parties in the present actions is how soon the attack rate in the vaccinated population drops below the point where the relative risk is not sufficiently large to assure the Court that a given GBS case was more likely than not caused by swine flu vaccination rather than by some other event.
> . . . [T]he vaccinated attack rates for late onset cases are so close to the range of unvaccinated baseline rates that the statistical evidence does not establish a probability of cause and effect relationship.[21]

Again, presented with comparable evidence and testimony, the *Sulesky* and *Cook* courts reached diametrically opposite conclusions. The primary reason seems to be that the judge in *Sulesky* felt comfortable relying on the eyewitness testimony of treating physicians and uncomfortable with the probabilistic testimony of epidemiologists; the judge in *Cook,* on the other hand, immersed himself fully in the statistical evidence presented by both plaintiff and defendant.

Over 4000 swine flu claims were eventually filed against the US government. The Department of Justice made a pragmatic policy decision not to contest the causation issue for claims in which Guillain-Barŕe syndrome had appeared within 6 weeks of swine flu vaccination. Of the 4000-plus claims, 2813 were denied and 1604 led to lawsuits, nearly two thirds of which resulted in dispositions favorable to the government.[20]

"Reasonably Exclusive Factual Connection"

The legal system has attempted to fit toxic torts into a standard tort framework, but that has proven difficult to do. Even if it can be shown that a defendant is responsible for a doubling or tripling of the number of cases of a particular disease or other harm, it is hard for individual plaintiffs suffering from that harm to demonstrate that theirs is one of the excess cases, rather than one of the cases that would have occurred absent the defendant. A classic example of this difficulty is provided by the case of *Allen v United States.*[21]

The US government conducted nuclear weapons tests at its Southern Nevada test site from 1951 through 1962. Weapons with yields of up to 104 kilotons were detonated, with 118 such weapons releasing radioactivity into the atmosphere. Radioactive debris descended to earth, subjecting the populated areas downwind to high levels of potentially hazardous ionizing radiation. A quarter of a century later, individuals who had lived in this area and who subsequently developed cancer or leukemia brought lawsuits against the US government under the Federal Tort Claims Act.

The federal trial judge in *Allen,* Judge Jenkins, found that "there appears to be no question whether or not ionizing radiation causes cancer and leukemia. It does." The number of cases of cancer and leukemia occurring in this population was indeed significantly higher than would normally have been expected. The epidemiological evidence at the trial provided strong support for the allegation that bomb fallout had produced additional cancers and leukemias in the local population. But epidemiology could not prove that any *individual* plaintiff's disease was the result of fallout. In such a situation, how—if at all—can causation be demonstrated? What can the courts do when the connection between a noxious source and some harm is only a statistical probability?

Well-established criteria, such as the Evans-Henle-Koch postulates,[22] can be used to evaluate the likelihood that an association is causal rather than spurious or artificial. But there are several types of direct causal associations. Proving that the defendant had contributed a factor that

is directly associated with the type of harm suffered by the plaintiff does not complete the plaintiff's case unless it can also be shown that the factor is both a necessary and sufficient cause of such harm. If the direct association is one in which the defendant's factor is (1) a sufficient but not necessary cause, (2) a necessary but not sufficient cause, or even (3) neither a necessary nor sufficient—but still a possible—cause,[23] the problem for the court is how to deal fairly with both plaintiff and defendant.

In *Allen,* Judge Jenkins' solution was to adopt what he termed a "reasonably exclusive factual connection test," under which the plaintiffs had a burden to demonstrate the existence of "substantial, appropriate, persuasive and connecting factors" between their injuries and the defendant's conduct. To judge the validity of each plaintiff's claim, Judge Jenkins looked for specific evidentiary factors.

> Where it appears from a preponderance of the evidence that the conduct of the defendant significantly increased or augmented the risk of somatic injury to a plaintiff and that the risk has taken effect in the form of a biologically and statistically consistent somatic injury, i.e., cancer or leukemia, the inference may rationally be drawn that defendant's conduct was a substantial factor contributing to plaintiff's injury. Unless the facts are proven otherwise by sufficient evidence, the inference provides a rational basis for imposing liability. . . .
>
> In this case, such factors shall include, among others: (1) the probability that plaintiff was exposed to ionizing radiation due to nuclear fallout from atmospheric testing at the Nevada Test Site at rates in excess of natural background radiation; (2) that plaintiff's injury is of a type consistent with those known to be caused by exposure to radiation; and (3) that plaintiff resided in geographic proximity to the Nevada Test Site for some time between 1951 and 1962. Other factual connections may include . . . time and extent of exposure to fallout, radiation sensitivity factors such as age or special sensitivities of the afflicted organ or tissue, retroactive internal or external dose estimation by current researchers, a latency period consistent with a radiation etiology, or an observed statistical incidence of the alleged injury greater than the expected incidence in the same population.

In seeking to distinguish causal from noncausal associations, Judge Jenkins was using criteria similar to those used by many epidemiologists to infer causation. But it was the judge himself who was adopting and applying such criteria rather than relying on epidemiologists to make these policy determinations for him.

However, although the *Allen* decision provides a good model for toxic tort litigation, it has no value as precedent because Judge Jenkins was reversed, albeit on other grounds. A recent change in the law by the US Supreme Court.[24] has made it exceedingly difficult to succeed in suing the US government for negligence, regardless of the merits of a plaintiff's case. In light of this decision, the appellate court held that, even if causation were proven, the government would not be liable in the *Allen* litigation.[25]

The Courts Demand Sound Epidemiological Evidence: Brock v Merrell Dow

Of course, the fact that epidemiological evidence is accepted into evidence by a court does not necessarily mean the plaintiff will prevail. In the swine flu cases, for example, epidemiological evidence was used most effectively by the government to demonstrate that a link between swine flu vaccination and Guillain-Barre syndrome was unlikely.

Most recently, epidemiological evidence has come to be viewed not simply as a useful adjunct to a plaintiff's toxic tort case but as an almost necessary element. As much as any other case, the recent decision of *Brock v Merrell Dow Pharmaceuticals, Inc.*[26] illustrates the importance of epidemiological evidence and the extent to which the courts have become sophisticated in using—and in this instance, demanding—high-quality epidemiological data.

During the period from 1956 through 1983, as many as 33 million pregnant women used the morning sickness drug Bendectin. Out of so many pregnancies, many birth defects will be found and their relationship, if any, to ingestion of the drug may be difficult to determine. Such seems to be the case with Bendectin, for which the data on a possible teratogenic effect is, at best, equivocal. Still, between 1000 and 2000 lawsuits have been brought against the maker of the drug, and as lawsuits and insurance premiums escalated, the drug was voluntarily taken off the market in 1983.[27]

In *Brock,* plaintiffs had filed suit in federal court to recover damages for birth defects allegedly resulting from Mrs Brock's ingestion of the antinausea drug Bendectin during her pregnancy. The jury awarded the Brocks $1 100 000 and the award was approved by the federal trial judge. But the Fifth Circuit US Court of Appeals reversed the judgment.

This is an unusual action by a reviewing court. Appellate courts look at the evidence presented to a jury in the light most favorable to the party successful in the lower court, giving that party the benefit of all reasonable inferences from the evidence. A reversal of a judgment based on a jury's verdict is appropriate when there can be only one reasonable conclusion drawn from the evidence, and it is a conclusion at odds with that actually reached by the trial court.

After observing that "medical science is now unable, and will undoubtedly remain unable for the foreseeable future, to trace a known birth defect back to its precipitating cause," the appellate court in *Brock* went on to state: "Undoubtedly, the most useful and conclusive type of evidence in a case such as this is epidemiological studies." The court then carefully analyzed the relevant epidemiological evidence. Epidemiologists should note the level of the court's sophistication regarding epidemiological data. After describing the nature of relative risk and of confidence intervals, the court stated:

> [Plaintiffs relied on an analysis of data that] found a relative risk of 1.49. However, [plaintiffs' expert] admits that the confidence interval was from 0.17 to 3; this renders the study statistically insignificant. The plaintiffs did not offer one statistically significant (one whose confidence interval did not include 1.0) study that concludes that Bendectin is a human teratogen. No published epidemiological study has found a statistically significant increased risk between exposure to Bendectin and birth defects. . . .
>
> Although we find [plaintiff's expert's] results inconclusive due to the fact that the confidence intervals include 1.0, we further note that [he] has not published his study or conclusions for the purposes of peer review. . . . [C]ourts must . . . be especially skeptical of medical and other scientific evidence that has not been subjected to thorough peer review. . . .
>
> While we do not hold that epidemiologic proof is a necessary element in all toxic tort cases, it is certainly a very important element. This is especially true when the only other evidence is in the form of animal studies of questionable applicability to humans.[28]

The appellate court was asked to reconsider its decision in *Brock* but, by an eight-to-six vote, declined to do so.[29] Those judges voting to reconsider were primarily troubled by the extreme weight accorded to the role of epidemiology in

this type of case. "In the absence of expert consensus," they asked, "must we now always await population studies before a jury verdict may be based upon medical opinion?"

DeLuca v Merrell Dow[27] is yet another Bendectin decision by a federal court of appeals. As in *Brock,* the court in *DeLuca* displayed a sophisticated understanding of epidemiological methodology. The *DeLuca* court noted that

> the confidence level or "significance" of a statistical analysis is but a part of a meaningful evaluation of its reliability. . . . [A] poorly conceived or conducted study that disproves the null hypotheses at a .01 level of significance may be far less reliable than a well conceived and conducted study that is significant at a .1 level. . . .

In *DeLuca,* the appellate court devoted several pages of its decision to a discussion of Kenneth Rothman's criticism of traditional significance testing.[30] The court explained in some detail the argument for using collective data in the context of confidence intervals, adjusting the confidence level depending on the context in which a decision is required.

As noted, the appellate courts in both *Brock* and *DeLuca* demonstrated sophistication in dealing with epidemiological evidence, the judges distinguishing between good and bad epidemiology. The *DeLuca* court, however, was more troubled by what threshold standard to apply, realizing that the arbitrary and subjective confidence levels of traditional hypothesis testing may not serve legal decision-making needs. Some defense lawyers argue that plaintiffs in toxic tort cases should be held to the same standards as prevail in peer-reviewed scientific journals. But if this is the standard, toxic tort plaintiffs will be forced to meet a heavier burden of proof than is normally the case in tort litigation.[5,31]

Ironically, although Bendectin plaintiffs have met with very little success, both "escalating insurance and litigation costs resulting from these cases, and decreased use of Bendectin flowing from the controversy surround[ing] its safety"[27] (rather than any action on the part of the Food and Drug Administration) led the manufacturer to cease production.

What Does This Mean?

The expanding role of epidemiology in tort litigation serves to highlight an important and interesting contrast between the nature of scientific proof and of legal proof. Science is a matter of probabilities in a universe of randomness and uncertainty. From the scientist's point of view, it is clear that no amount of empirical evidence can conclusively verify the type of universal propositions known as scientific laws. To demand certainty would be to misunderstand the nature of scientific knowledge. The legal system, on the other hand, seeks finality in the resolution of disputes. Without such finality, the legal process would be one of continual litigation and relitigation. For this reason, concepts of legal causation have favored single-cause explanations. Tort law posits a direct chain of causation, and a tort defendant's conduct is held to be a cause of a particular event if the event would not have occurred "but for" that conduct or if the conduct was a "substantial factor" in bringing the event about.

It is important to keep in mind that the degree of certainty that must be present for a position to be considered "proven" varies according to the forum in which proof is being evaluated. In a criminal court, proof beyond a reasonable doubt is required; in a civil court, a preponderance of proof or enough to tip the balance (i.e., greater than 50%) is required. For many scientists, proof—if it exists at all—is achieved at the *P* value of .05 or .02.

Rothman, in *Modern Epidemiology,* criticizes the importance placed on statistical significance:

> The motivation for the development of statistical hypothesis testing was to provide a basis for decision making in agricultural and quality-control experiments. These experiments were designed to answer questions that called for specific actions, so that the results had to be classified, if possible, into qualitatively discrete categories. Thus arose the practice of declaring associations in data as "statistically significant" or "nonsignificant," using arbitrary criteria that became conventional. The notion of statistical significance has come to pervade epidemiologic thinking as well as that of other disciplines.[32]

Perhaps litigation has some similarities to agriculture in this respect. The job of the court is to come to the peaceful resolution of disputes. The court does not have the luxury of awaiting further scientific studies to approach the truth; it must come to a timely decision for the benefit of the litigants and the judicial system. Certainly the court would like its decision to be based on what it understands to be the truth, but what the true facts are is often exactly what is being contested. In the end, the court must act on uncertainties to resolve the dispute.

The idea of acting on uncertainty may cause discomfort to scientists, whose discipline allows them to admit that they have not yet achieved a complete understanding of the truth and that further investigation is necessary. When the work of scientists is being used as proof in court—for example, the use of epidemiological evidence in toxic tort cases—scientists may complain that undue weight is being attributed to inconclusive findings. The misperception, however, is in thinking that the conclusion sought by the court is the same conclusion sought by the scientist. The scientist's conclusion is achieved when truth is illuminated, and the level of certainty or proof required for this is very high. The court's conclusion is achieved when the best decision, given the weight of the evidence, is made for that case and the litigants' dispute has been resolved in a socially acceptable fashion. For this, the level of certainty need not be that of the scientist. In this context, it would be well to remember the words of Sir Austin Bradford Hill, writing 25 years ago:

> All scientific work is incomplete—whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time.[33]

Conclusion

Epidemiologists need to recognize the growing involvement of their profession in complex tort litigation. The epidemiological profession must consider the implications of this involvement on such things as graduate training programs. What do epidemiologists, as well as other public health professionals, need to know about law and public policy? How can they best acquire this knowledge?

As a simple first step, epidemiology and the law should become a standard part of health law courses. On a more complex level, if one or two schools of public health established enough of a reputation in some of the areas being confronted by the courts in toxic tort litigation, these institutions could serve as valuable resource centers to the courts. Judges are free to pick court-appointed experts, but in the toxic tort area they most often do not know where to turn. The result is one of "hired guns" providing expertise for one or both sides of the litigation.

The epidemiological profession cannot make this situation go away by ignor-

ing it. The profession needs to come to grips with expert testimony and other legal policy issues. It also needs to confront the ethical questions surrounding this area.[34] Whatever course is ultimately charted, it is clear that epidemiology and the law will be working closely together for some time to come. □

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