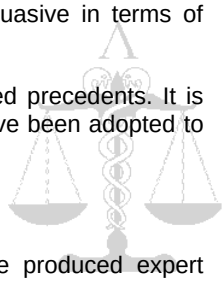
Epidemiology in Court

Epidemiological evidence may form part of the justification of an expert opinion. However, it is not often recognised as evidence in its own right.

English courts have always been reluctant to specify what they have found persuasive in terms of epidemiological evidence.



Express recognition, would only serve to reduce judicial flexibility and set unwanted precedents. It is therefore a practice which appears be avoided, even when the tests themselves have been adopted to reach the decision.

An exception may be made for the dose response relationship.

Whilst equally reluctant to be bound by precedent, the courts in the USA have produced expert guidance on the purpose, methods, capabilities and assessment of epidemiological evidence. "Reference Manual on Scientific Evidence". Federal Judicial Center.... 2000 2nd Edition. The manual will allow courts to understand the basics.

Some of the salient points are:

- "In the absence of an understanding of the biological and pathological mechanisms by which disease develops, epidemiological evidence is the most valid type of scientific evidence of toxic causation."

  Richardson v. Richardson-Merrell, Inc., 857 F.2d 823, 830 (D.C. Cir. 1988) (epidemiology more probative than other forms of scientific studies), cert. denied , 493 U.S. 882 (1989);
  Conde v. Velsicol Chem.Corp., 804 F. Supp. 972, 1025–26 (S.D. Ohio 1992) ("Epidemiologic studies are the primary generally accepted methodology for demonstrating a causal relation between a chemical compound and a set of symptoms or a disease.")

The Manual discusses causation in some depth:

> "These factors guide the epidemiologist in making a judgment about causation. They are:
> 1. strength of the association;
> 2. temporal relationship;
> 3. consistency of the association;
> 4. biologic plausibility (coherence with existing knowledge);
> 5. consideration of alternative explanations;
> 6. specificity of the association; and
> 7.dose-response relationship."

Whilst avoiding the implication that the courts should be swayed by the same list of considerations, the manual explains each aspect in some depth, sufficient to allow clarification of expert evidence.

- "Strength of association is usually measured by relative risk or by odds ratio, depending on the type of research being reported.

  "In general, the relative risk can be interpreted as follows:
  •If the relative risk equals 1.0, the risk in exposed individuals is the same as the risk in unexposed individuals. There is no association between exposure to the agent and disease.
  •If the relative risk is greater than 1.0,the risk in exposed individuals is greater than the risk in unexposed individuals. There is a positive association between exposure to the agent and the disease, which could be causal.
  •If the relative risk is less than 1.0,the risk in exposed individuals is less than the risk in unexposed individuals. There is a negative association, which could reflect a protective or curative effect of the agent on risk of disease. For example, immunizations lower the risk of disease.

  "The higher the relative risk, the greater the likelihood that the relationship is causal."

  "A relative risk of 9 to 10 is so high that it is extremely difficult to imagine any kind of error in the study that would have produced it. The higher the relative risk, the stronger the association, and the more likely an epidemiologist will consider it causal."

The following rule for causation has been derived by mathematical proof and widely accepted by the courts. However it refers to the idealised experiment where there are no biases or confounding factors.

> "When the relative risk reaches 2.0, the agent is responsible for an equal number of cases of disease as all other background causes. Thus, a relative risk of 2.0 implies a 50% likelihood that an exposed individual's disease was caused by the agent. A relative risk greater than 2.0 would permit an inference that an individual plaintiff's disease was more likely than not caused by the implicated agent. A substantial number of courts in a variety of toxic substances cases have accepted this reasoning."
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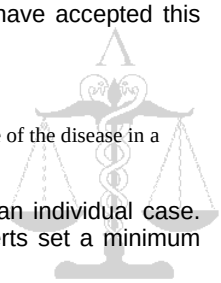
> Landrigan v. Celotex Corp., 605 A.2d 1079, 1087 (N.J. 1992)
> (relative risk greater than 2.0 "support[s] an inference that the exposure was the probable cause of the disease in a specific member of the exposed population").



In practice, a single study with a RR of 2.1 will not be persuasive of causation in an individual case. There are many methodological grounds for doubting the reported RR. Many experts set a minimum threshold of 3.0 before attaching significance to a single study.

However, smaller relative risks cannot be discounted out of hand. But findings of small relative risks are much more susceptible to these [methodological] errors. See Cook v. United States, 545 F. Supp. 306, 316 n.4 (N.D. Cal. 1982).

The Manual also describes the statistical tests that are applied to epidemiological findings.

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