Legal vs. Medical Criteria for Determining Causation in Occupational Disease Claims

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Both of the Nation's legal mechanisms for compensating the victims of workplace exposure to hazardous substances are close to foundering on the same rocks—the difficulties of proving workplace causation of harm in occupational disease claims.

These difficulties have rendered the nonfault workers' compensation system as litigation-bound and transaction cost-intensive as the fault-based common law of torts. They have also spawned new bases for compensation plainly aimed at finessing causation issues by permitting recovery in the absence of demonstrable harm. Twenty-seven states' workers' compensation laws now recognize claims for disabling mental stress unaccompanied by physical injury, and, in a handful of states, tort damages may be sought for such ephemera as "cancerphobia," statistically increased risk of disease, medical surveillance, and loss of the quality of life.

My thesis is that the integrity of both workers' compensation and the common law of torts is being severely compromised by the absence of empirical *medical* and *scientific* data essential to the fulfillment of the traditional *legal* requirement of proof of causation to a reasonable certainty by a preponderance of the relevant evidence.

Unless we can fashion a workable surrogate for the missing medical and scientific data needed to make rational compensation decisions, we risk surrendering our entire legal system to the crackpots and charlatans who are already making a mockery of both science and law in their zeal to find "causation" where no empirical basis for it exists.[1]

Put another way, we must find some means of fashioning *legal certainty* of just compensation in an environment of *medical* and *scientific uncertainty*. If we do not, we risk crippling the capacity of workers' compensation to deal with occupational disease claims and making our tort system so speculative and unpredictable that it becomes uninsurable at any price.

A leading scholar in the occupational health field, Dr. Leslie I. Boden, has rightly observed that many occupational diseases:

> . . . may be caused by both occupational and nonoccupational factors. It is often difficult or impossible to determine which of these factors caused the disease in a specific case, or even to determine their relative contribution. . . . Even when epidemiological studies are able to determine very accurately excess risks of disease in *populations*, they are not able to determine which *individuals* in those populations would not have developed the disease without occupational exposure. In many cases, this uncertainty cannot be resolved.[2]

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Boden argues that disease causation presumptions and medical panels will not solve the problem of proving causation in occupational disease claims. He urges instead, "strengthening the evidentiary status of epidemiological and toxicological studies,"[3] on the grounds that "[t]he use of experts in clinical medicine to offer opinions about whether a particular person's lung cancer was caused by workplace exposure is a form of dishonesty required by inappropriate evidentiary requirements."[4] Boden suggests that:

> . . .[a] way of further strengthening the position of claimants is to design presumptions that shift the burden of persuasion based on reasonable inferences from scientific and statistical studies, without the development of the more specific types of evidence normally required to demonstrate individual cause. . . . Here, as with specific disease presumptions and medical panels, the problem is one of design and implementation, not a conceptual one. Clearly, both the quality of studies entered as evidence and the levels of excess risk that are implied by such studies should be important determinants of the weight given to them.[5]

Although some have argued, and in absolute terms may be correct, that statistical evidence of disease incidence among an exposed population is "of little use in judging the condition of a specific worker,"[6] epidemiologic studies, properly conducted and properly used, can provide meaningful guidance as to the *probability* that a particular worker's disease was caused by chemical exposure.

The crucial phase in this formulation is "properly conducted and properly used," for, as an abstract matter, in many chronic diseases, including most cancers, it is impossible to be absolutely sure that *any* individual's disease was caused by chemical exposure:

> . . . At least three factors make certainty unlikely: (1) no symptom of the disease will be peculiar to the exposure (the exceptions including diseases such as asbestosis whose peculiar symptoms label them as asbestos-caused); (2) factors other than exposure to the chemical from any manufacturer's [or employer's] activities, including genetics, other than exposures such as diet, and even exposures to the same chemical from natural background sources, may have caused disease; and (3) it will be the cumulative exposure to the chemical of interest, and not any particular day's [or employer's] dose, that is likely to be related to disease.[7]

Thus, given the uncertainty surrounding the harmful potential of the 50,000 or so chemicals in industrial use, the issue in many occupational disease and toxic tort claims is not only the traditional one of whether the *claimant's* injury was the result of a chemical exposure, but also the more fundamental one of whether exposure to the suspect chemical *can* cause the injury of which the claimant complains.

This problem has often been characterized as the "Can, Does, Did Triad"[8]: *Can* substance *X* cause disease *Y*? *Does* the claimant have disease *Y*? *Did* substance *X* cause the claimant's disease *Y*? Assuming proper diagnosis, the "Does" question can almost always be answered. But except in a handful of known cause-and-effect relationships, the "Did" question, as we have seen, can almost never be answered with certainty. What about the "Can" question? What can be learned about it that could help answer the "Did" question?

Here is where epidemiology, properly conducted and properly used, can play an important role. Generally speaking, results of epidemiologic studies can be expressed in terms of the *relative risk* of disease associated with exposure. If exposure is not associated with disease, the relative risk is 1. That is, the exposed person is no more likely to have the disease than is a nonexposed person. But if

the relative risk is, for example, 2, an exposed person is approximately twice as likely to become diseased.[9]

It is a basic characteristic of statistics that relative certainty is directly related to sample size. In a properly designed epidemiologic study, the chance factor in small samples can be quantified through the calculation of levels of significance designed to eliminate the random or stochastic nature of cancer.[10]

It is also important in using epidemiology on disease causation issues that the relative risk demonstrated by a particular study show a strong association between the suspect chemical and the disease at issue. For example, if epidemiologic studies indicate that the relative risk of developing cancer *X* from exposure to 100 units of chemical *Y* is 4, and worker A had 100 units of exposure to chemical *Y*, there will be a three out of four (or 75 %) chance that worker A's cancer *X* is due to chemical *Y*. However, if the relative risk from 100 units of chemical *Y* is only 1.1, then there will be only a 1 out of 11 (or 9%) chance that the cancer is due to the chemical exposure.

Yet even properly performed population and epidemiologic studies cannot by themselves serve as the basis of a biologic inference that a causal relationship exists: "[T]he epidemiologist must integrate additional scientific information. The derivation of such an inference requires rigorous consideration of laboratory, experimental, demographic and epidemiologic data,"[11] widely shorthanded as the Henle-Koch-Evans Postulates.[12]

How can these *scientific* principles be integrated with traditional *legal* principles of causation, which essentially require a claimant plaintiff to prove, by a preponderance of the evidence, that it is more likely than not that the defendant's conduct caused his harm?

The essentially *qualitative* legal test can be, and has been, expressed in *quantitative* terms. An extensive 1950's study proposing how courts should decide cases concerning radiation-induced leukemias concluded:

> . . . If as little as 2.5 rems exposure of a fetus and from 25 to 50 rems exposure of an adult doubles the incidence of leukemia, then a person so exposed could claim . . . that if he should develop leukemia . . . the chances are better than fifty-fifty that his leukemia resulted from the radiation exposure, rather than from all other causes together. Therefore "more probably than not" his leukemia was caused by the radiation to which he was exposed.[13]

But a purely *quantitative* scientific analysis, even one based on rigorous adherence to the Henle-Koch-Evans Postulates, cannot serve by itself as a surrogate for the *qualitative* legal standard of "more likely than not" on disease causation issues:

> The Henle-Koch-Evans Postulates do not, by themselves, provide a complete legal standard because the determination of legal causation requires consideration of the degree of certainty required to meet the plaintiff's burden of proof. This deficiency can be remedied, however, by requiring in addition that the attributable risk for the factor at issue be greater than .50. . . . If, in an exposed population, more than half the cases of a disease can be attributed to the exposure, and if the postulates are satisfied, then absent other information about a diseased individual, it is more likely than not that his or her illness was caused by the exposure.[14]

A correct *qualitative legal* standard thus would be one in which both the *quantitative*, scientifically determined *increased* risk and *attributable* risk exceeded the 51% or "more likely than not" test. And in cases in which exposure in sufficient amounts and durations is certain, "any relative risk greater than 2 would lead to an attributable risk of more than .50."[15]

If it is thus possible to scientifically quantify the qualitative legal test of "more likely than not" through rigorous adherence to the Henle-Koch-Evans Postulates and attributable risk analysis, what weight should be given to what even the advocates of statistical evidence concede is nothing more than a biologic inference that a causal relation exists between a particular chemical exposure and a particular disease?

Certainly, such evidence, even if impeccably prepared, cannot serve as the foundation for a presumption, conclusive or otherwise, that a worker's or a tort plaintiff's exposure to chemical *X* caused his or her disease *Y*. Presumptions, either medically or legally speaking, exist to reflect reality, not create it.

If responsibly conducted epidemiologic studies, however, can demonstrate that there is a greater than twofold higher risk of contracting a particular disease as a member of a workplace or other population exposed to a particular chemical in specified concentrations and durations, than as a member of a general population shouldn't that demonstration have some "added value" in the adjudicative process?

My answer is "Yes," quickly followed by the question, "How much added value?" Returning to the "Can, Does, Did Triad," it seems to me that if responsibly conducted epidemiologic studies can establish that there is a greater than twofold increased risk (over that of the general population) of contracting a particular disease as a result of being a member of a particular population exposed in requisite duration to a particular concentration of a particular chemical, then a member of that exposed population who can present such evidence should be deemed to have met the burden of proving the first element of the "Can, Does, Did Triad": Chemical *X*, in sufficient concentrations and durations, can cause disease *Y*.

It would then be up to the claimant to establish the two other elements of the triad—that he in fact does have disease *Y*, and that given the duration and concentration of his exposure to chemical *X*, it is more likely than not that *X* did cause his particular *Y*.

The employer or defendant in this formulation could contest the bases on which the increased risk and attributable risk analyses were made.[16] In addition, the employer or defendant would be free, as they now are, to argue that notwithstanding the increased or attributable risk occasioned by the exposure, factors other than the exposure were predominant in the disease etiology.

Moreover, as in other fields of expertise, there is "room for responsible epidemiologists to differ significantly on many of the key choices and assumptions to be made in analyzing [a] causal relationship,"[17] and expert witnesses would be needed to explore the complexities of detailed application of the Henle-Koch-Evans Postulates.[18]

Thus, even an epidemiologically derived standard for establishing inferences of causation would not eliminate the contentiousness in occupational disease or toxic tort claims. But among members of a similarly exposed group, it would go a long way toward eliminating the individual expense of having to establish, on a case-by-case basis, that exposure to chemical *X* can cause disease *Y*.

It would also avoid the adoption of other surrogates that would do greater damage to both science and law, such as presumptions of causation based on exposure in excess of regulatory limits or presumptions based on animal or tissue culture studies.

Strengthening the evidentiary status of epidemiologic evidence in the adjudicatory process will mean little to claimants or plaintiffs, however, unless epidemiologic evidence exists. What can be done to assure the availability of the needed studies?

In my judgment, H.R. 162 and S. 79 of the current Congress, the proposed High Risk Occupational Disease Notification and Prevention Act, should be promptly enacted. The enactment of these bills would encourage insurers and self-insured employers to undertake clinical and epidemiologic studies of exposed worker populations to determine what is and, equally important, what is not dangerous to workers' health.

If these studies show that particular concentrations or durations of exposure, or both, do not create increased risks of work-related disease, then it follows that they can be used not only to forestall unnecessary notifications under the Act, but also to controvert spurious occupational disease and toxic tort claims.

By the same token, if these studies show that there are increased risks in such exposures, then they can be used not only for insurance underwriting, rating, and loss prevention purposes, but also as compensability guidelines by workers' compensation tribunals and courts.

The proposed Act offers the business community as a whole—and insurers in particular—the opportunity to help the proposed Risk Assessment Board do for the prevention of occupational disease and the rationalization of occupational disease compensation what Underwriters Laboratories, ANSI, ASTM, and, most recently, OSHA have done for the prevention of traumatic injury by countless products and materials both inside and outside the workplace.

The reason is that the medicoscientific and procedural framework within which the Risk Assessment Board would operate would enable the Board, over time, to establish a rational basis not only for preventing occupational disease, but also for determining, for compensation purposes, what conditions do or do not increase the risk of occupational disease.

The Act, in my judgment, would eventually assure the availability, to employer and employee representatives alike, of a centralized source of the very best medical and scientific information on how to prevent, treat, and compensate for occupational diseases. The bill's scientific and procedural provisions would give all affected interests a tremendous opportunity to advance their collective knowledge of chronic and latent workplace disease.

Society is likely to find *cures* for most long-latency diseases before it fully understands their *causes*. But until these cures are found, sufferers of diseases caused by exposure to chemicals in the workplace and elsewhere must be compensated on a fair and rational basis.

If those of us with a stake in the survival of the workers' compensation and tort systems fail to devise that fair and rational basis, the courts, administrative tribunals, and legislatures will find other means, perhaps unfair and irrational, of achieving compensation objectives.

Although properly conducted and properly used epidemiologic data cannot provide all the answers, they can nonetheless provide some useful guidance. Every step should be taken to assure its proper integration into the adjudicative process.

Similarly, Congress should encourage the needed clinical and epidemiologic studies by enacting the proposed High Risk Occupational Disease Notification and Prevention Act.

NOTES AND REFERENCES

1. See, for example, *Gicas* v. *United States*, 50 F. Suppl. 217 (E.D. Wis. 1981).
2. "Compensating Victims of Pollution: The Workers' Compensation Experience," statement of Leslie I. Boden, Assistant Professor of Economics, Occupational Health Program and Department of Health Policy and Management, Harvard School

of Public Health, before the Subcommittee on Commerce, Transportation and Tourism of the Committee on Energy and Commerce, U.S. House of Representatives, November 22, 1983, at 6 [emphasis in original].)

3. *Idem* at 12.
4. *Idem* at 12–13.
5. *Idem* at 13.
6. Note, "Compensating Victims of Occupational Disease," 93 Harv. L. Rev. 916, 931, n. 108 (1980).
7. HARDY, T. S. 1982. Determination of Causation in Compensation of Persons Chronically Injured by Toxic Substances Exposures. Unpublished manuscript, at 13 (January 7).
8. See, generally, GOTS, R. E. 1983. Medical/Scientific Decision-Making in Occupational Disease Compensation. Appendix I. Role of the State Workers' Compensation System in Compensating Occupational Disease Victims, Crum and Foster (June).
9. HARDY, op. cit. *supra* n. 7, at 12.
10. *See* HAVENDER, W. R. 1982. Assessing and controlling risks. *In* Social Regulation, Bardach and Kagan, Eds. . . . "[A]mong a group of similarly exposed persons, only some of them—seemingly at random—will actually develop cancer. Not every smoker of high-tar cigarettes contracts lung cancer (in fact, only one in five does), and not every worker who worked with asbestos comes down with mesothelioma." *Idem* at 24–25.
11. BLACK & LILIENFELD. 1985. Epidemiologic proof in toxic tort litigation. 52 Fordham L. Rev. 732, 762.
12. *Idem* at 763.
13. STASON, E. B. *et al.* 1959. Atoms and the law, at 498. See also, note, Tort Action for Cancer: Deterrence, Compensation, and Environmental Carcinogenesis. 1981. 90 Yale L.J. 840, 861.
14. BLACK & LILIENFELD. op. cit. *supra* n. 11 at 767 [footnotes omitted].
15. *Idem* at 768 [footnote omitted].
16. "In using the Henle-Koch-Evans Postulates as constrained by attributable risk, great care must be taken in defining the exposure and the exposed population. In some instances, the focus should be on the total exposure above a certain level; in other cases the extent of exposure at any given time may be more important. The population of interest should be limited to individuals exposed at or beyond the level or extent at issue." *Idem* at 767, n. 143.
17. *O'Gara* v. *United States*, 560 F. Suppl. 786 (E.D. Pa. 1983).
18. "[T]he witnesses through whom the evidence is introduced must be suitably qualified. . . . Precedent . . . supports a rule requiring that a medical expert be qualified as an epidemiologist before testimony on causation is admitted. . . ." Black and Lilienfeld, op. cit. *supra* n. 11 at 769 [footnotes omitted].