EVIDENTIARY PROBLEMS IN POLLUTION-ENGENDERED TORTS

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This paper reviews the kind of evidence that can be gathered and the utility such evidence can have in bearing upon the central issues of a trial concerning alleged harm resulting from an environmental pollutant.

In order to determine whether harm has resulted from something introduced into the environment, one must answer four questions:

(a) Is there evidence that something was introduced into an environment that normally would not be expected to be there?

(b) Is there evidence that the substance introduced is capable of causing harm?

(c) Is there evidence that this substance causes harm?

(d) Is there evidence that the harm present was related to the substance that was introduced?

The type of evidence available is critical. If direct scientific evidence were available more often, no issue would remain. What one usually has to deal with, however, is objective evidence from which an inference may be drawn. The persuasiveness of the evidence in question usually depends upon the qualifications of the presenter and the type of inference that can be drawn from the available data.

The matter of qualifications of the "expert" may be easily disposed of. Normally a toxicologist or an epidemiologist can supply the needed data. A toxicologist knows what substances can adversely affect animal and vegetable life, and an epidemiologist studies effects on population groups. In both fields, there are variations in quality and limitations inherent in the techniques available.

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Even competent studies seldom answer any of our questions directly; hence there is an opportunity for bias in the inferences drawn.

With these limitations in mind, I'd like to review each of the aforementioned questions and indicate the type of evidence available to deal with each.

Is Pollution Evident?

To answer the first question, we must first determine the presence of the substance, and then decide whether or not it was normally expected to be there.

The detection of the presence of a chemical substance used to be a simple matter, using a standard test. Today, however, technology permits detection of substances in infinitesimal amounts. One must often wonder whether the detection of a substance considered to be an environmental pollutant is not due simply to our increased technical capability to detect it, rather than to its being newly introduced.

There is an unquestioned bias on the part of some laboratories in reporting negative results. If one submits a specimen for a chemical analysis, seldom, if ever, does one get a "negative" report. The usual report reads "below detectable limits," implying that there is a minute quantity present but current technology is insufficient to reveal its presence.

This is important for two reasons: There is a body of opinion in our society that suggests that the presence of any quantity of certain substances is intolerable; and, in spite of the fact that we have had the current technology available for only a short time, and we really don't know what the levels of certain substances were in the environment before they were detectable, it is presumed that if detectable now, the substance must have been newly introduced.

In examining the validity of such a view we need to define a pollutant. Except for meteorites, everything on this earth came from this earth. We may concentrate substances, reduce them to component parts, combine them with other substances, or separate them. But every substance is matter that is part of the chemistry of our planet.

Pollution, then, is not necessarily the result of a new substance being created, but may be due to the collection at one point on this earth of substances normally found elsewhere. In their original habitat, they were not considered pollutants, but resources. As we develop new chemical combinations having commercial applica-

tion, it is surprising to find that some creature has produced a similar substance. Hence, with pollution we often are not dealing with exotic substances, but with an abnormal concentration in time and space.

Quantity and location are important because of the effects on our renewable resources. We constantly reuse the air we breathe and the water we drink. We require a symbiotic relationship with the vegetation on our planet, not only as it contributes to making our air rebreathable or our water redrinkable, but in its role in the food chain. Pollution exists when sufficient quantities of a substance are present to threaten the integrity of the renewable resources or the health of the people living in the area.

Renewable resources are threatened because of the time required to reduce the concentration at a specific place or to reconvert a synthetic substance to nontoxic component parts. While all substances are ultimately degradable, too much in too limited a space may prevent the ecosystem's recycling of the more elementary components.

Thus, pollution is a matter of judgment, depending upon how much of a substance exists in a specific location. When the quantity is great enough to temporarily stress the ecological system, it is agreed that pollution is present.

So the evidentiary questions relate to identifying and quantifying the allegedly polluting substance. Is the amount more than what would normally be expected to be there? Unless data from previous analysis is available, one must look to comparable geographic areas to determine the range of "natural occurrence," to use as a gauge. Unfortunately, too few studies have been done showing the distribution of polluting substances. Usually extensive sampling is necessary in order to produce comparative data to assess the significance of the level found.

Is the Pollutant Potentially Harmful?

The second question, "Is the substance introduced capable of causing harm?" must be dealt with in a similar fashion. There is no such thing as a totally innocuous substance. Every substance, under the right conditions, can result in injury to some biological unit.

Application of large quantities of a substance to a species of experimental animals is considered to be persuasive evidence of the potential harm of minute quantities to other life forms. This is postulated even if the animals metabolize the substances in significant-

ly different ways. I'm not suggesting that animal studies do not have value. I am suggesting that extrapolation from animal studies must be done with caution and considered as only part of the evidence concerning the potential effect of substances present in our environment.

We have a particularly difficult problem with studies in species other than man when dealing with substances that are used because they are selectively lethal to specific life forms. Insecticides and herbicides are used precisely because they interfere with the metabolism of the target organisms. If they weren't effective killers of specific animals or plants, we wouldn't use them. The problem with these substances is the quantity that is tolerable by other life forms.

It is because various species metabolize some substances differently that we can have insecticides and herbicides with minimal effects on other life forms in normal quantities. Yet we tend to forget or disregard interspecies differences when we do studies on mammals. In such studies the assumption is that, moving up the evolutionary ladder, metabolic processes are similar to those in parallel species, and that an adverse effect on one species of mammal signals a comparable effect on another.

This is not true. Instead, what's required is an examination of the effect of a substance on the human organism itself. This is what epidemiology is about. The word is derived from the Greek and literally means "the study of what is upon the people."

In addressing the question of the harmfulness of a substance, we often study the experience of an accessible segment of the population that has had contact with similar amounts of the substance. This is usually a group of workers who have been engaged in the preparation or application of the substance, often over a long period of time.

Table I is an example of the findings of such a study.[1] The data analyzes the number of deaths among all individuals who had worked three months or more in the one plant in the United States that made a chlorinated hydrocarbon insecticide, gathered over the 34-year lifespan of the plant. The table shows that the death rate of this employee group compared favorably with the U.S. population as a whole during the same period of time.

Prior to this study, there was concern over the possibility of an in-

[1] Sidney Shindell, I. Slack Ulrich and Eldred E. Giefer, "The Epidemiology of Chlorinated Hydrocarbon Insecticides," in M.A.Q. Khan, ed., *Halogenated Hydrocarbons* (New York: Pergamon Press, 1981).

TABLE 1

DEATHS AMONG FORMER EMPLOYEES: 1 JANUARY 1946—31 DECEMBER 1979
VELSICOL CHEMICAL CORPORATION — MARSHALL, ILLINOIS PLANT
COMPARED WITH U.S. POPULATION BY CAUSE AND JOB/PRODUCT CLASSIFICATION

Employee Group	All Causes (all ICDA)		Malignant Neoplasms (cancers) (140-209)		Cardio-vascular (heart disease) (390-429, 440-458)		Cerebro-vascular (stroke) (430-438)		Trauma (external causes) (E800-E999)		Other & Unknown (000-136, 210-389, 460-999)	
	Vels.	U.S.	Vels.	U.S.	Vels.	U.S.	Vels.	U.S.	Vels.	U.S.	Vels.	U.S.
White Males												
Opr. Pesticide	8	8.10	1	1.59	5	3.29	—	0.39	1	1.23	1	1.60
Opr. Resins	15	15.84	1	3.05	8	6.42	1	0.84	3	2.45	2	3.80
Opr. Multiple	5	5.21	2	0.98	—	2.32	1	0.30	1	0.51	1	1.00
Opr. Raw Matl.	5	9.25	1	1.76	3	4.00	—	0.61	1	1.11	—	1.77
Shipping	13	18.20	2	3.53	7	8.31	1	1.33	2	1.59	1	3.44
Maintenance	25	31.44	7	6.34	14	14.21	1	2.04	1	2.84	2	6.01
Laborer	19	19.68	3	3.73	8	8.88	—	1.45	4	1.91	4	3.71
Laboratory	11	15.49	2	2.99	8	6.69	1	0.98	—	1.84	—	2.99
Engineering	7	8.33	1	1.66	5	3.61	—	0.49	1	0.97	—	1.60
Mgmt/Clerical	12	11.00	2	2.19	6	4.96	—	0.74	—	1.02	4	2.09
Total White Males	120**	142.54	22	27.92	64	62.69	5	9.17	14	15.47	15*	27.29
Nonwhite Males	—	*0.05*	—	*0.01*	—	*0.00*	—	*0.00*	—	*0.03*	—	*0.01*
Females	4	2.95	—	1.00	—	0.63	1	0.18	—	0.41	3*	0.73
TOTAL	124	145.54	22	28.93	64	63.32	6	9.35	14	15.91	18*	28.03

*Statistically significant difference ($p < 0.05$) **Statistically significant difference ($p < 0.01$)

Numbers shown in parentheses under specific disease headings are the ICDA, 8th Revision categories included.

crease in deaths from cancer associated with the use of this insecticide. The study shows no excess cancer risk. This was true even in the presence of other data demonstrating that workers engaged in pesticide manufacture had an amount of the substance in their bloodstream far in excess of that of the general population.

In a study of some 6,000 pesticide applicators conducted by an independent investigator, a similar finding was reported.[2] Thus, corroborative evidence is available to bring to bear on the issue of the potential harm to the general populace or to individuals exposed under specific circumstances. These data have been used in product liability actions to ascertain whether the substance in question is capable of causing harm in the amounts encountered by a claimant.

Analogous to the situation with laboratory determinations, the epidemiology study also reports findings essentially "below detectable limits." In this instance, however, it is not because there is a presumption that a more sensitive technique might detect a hazard, but it is an acknowledgement that in spite of the most sensitive technique available, the hazard, if it exists, is of such low magnitude that it cannot be demonstrated.

The author of the study from which these data are derived states: "There is, thus, no evidence of any long-term, latent effect on health related in any way to employment at the Velsicol plant in Marshall, Illinois, for the thirty-four year period in which it has been engaged in the production of chlorinated hydrocarbon insecticides."

It is not suggested that under all circumstances the insecticide is innocuous, any more than either plain table salt or plain water is innocuous under all conditions. Whether a substance is harmful depends upon the type of harm alleged or suspected and how much of the substance is present.

The inference is, then, that if under certain conditions no hazard can be demonstrated, this should be true under comparable conditions. Or, as is more usually the case, if in circumstances where there is more exposure and no hazard is demonstrable, there should be no hazard with less exposure.

The issue of whether a specific quantity of a substance is harmful may take years to resolve. The classic example of a substance not known to be harmful until after a considerable period of time is asbestos. High concentrations of asbestos dust were known in the

[2] H.H. Wang and B. MacMahon, "Mortality of Pesticide Applicators," *Journal of Occupational Medicine* 21 (1979): 741-744.

1930s to be hazardous, and the occupational health field had generally agreed upon a safe limit for asbestos workers. However, in 1965 it was shown that very low concentrations of asbestos fiber in the air breathed by a worker handling products containing asbestos may result in disease 20 or more years after exposure.

We recognize that conventional wisdom may turn out to be in error. The presumption was that incorporation of asbestos in a material at 15 percent so bound the asbestos that it was then safe to handle. It was also believed that once a person was removed from exposure to asbestos dust, it would eliminate any further risk. Ex-smokers over time tend to return to the level of risk of non-smokers, so why not other substances? Moreover, before the mid-1960s, it was difficult to identify a large enough group with a specific exposure who could be followed long enough for the hazard to become evident.

So, at a specific time, we may not know whether a substance newly introduced into the environment can be harmful. Often all we have are either indications of a potential hazard, or subpopulation studies that do not reveal the presence of a hazard under the specific conditions of exposure. In most cases, harm can only be determined when we can find evidence in the specific environmental situation that the presence of the substance is harmful.

Has Harm Occurred?

In answering my third query, I will focus only on harm as it relates to human health. In order to do this, we must consider the question of how to measure health.

Health can be a sense of well-being, as well as physical functioning or freedom from disease. Health is seldom measured; ill health is easier to detect. Ill health may be measured by reduced productivity, lost time from work, measurable aberrations in laboratory studies of specific organ systems, incidence of disease, and alteration in life expectancy.

Generally, ill health is viewed as the interaction of one or more disease-producing agents with a specific host under specific environmental conditions. One or more body systems is stressed beyond the point of normal functioning and reflected in recognizable physiologic abnormalities.

The causative agents of many diseases are still unknown. We have no idea in most cases what causes arthritis, why certain people handle emotional stress the way they do, why cells become

cancerous, why certain neurological diseases occur, or why mental processes sometime slow down with age.

The best information we have about some diseases is that they are associated with certain events. An increased incidence of heart disease is related to high blood cholesterol. Down's syndrome is more frequent as a mother's age increases. Diabetes seems to occur in certain families. Breast cancer is more prevalent among affluent whites.

The evidence is quite persuasive that certain substances are associated with increases in certain diseases. Mercury exposure can cause mental illness, hence the Mad Hatter in *Alice in Wonderland.* Cigarette smoking increases the risk of lung cancer. Exposure to a variety of dusts results in change in pulmonary functions.

In all these examples, the evidence of an adverse influence is demonstrated by a quantitative change in the incidence of a disease in specific population groups. So in order to demonstrate that a substance is harmful to human health, one must be able to show an aberration in the normal state of affairs.

In order to determine what is considered normal, one must either monitor disease and mortality rates or compare population groups. Here is where we have problems. The monitoring of health status in this country has not been completely satisfactory. We have been collecting mortality data by cause in a somewhat uniform fashion since about 1930. The accuracy of data on cause of death, however, is dependent both on the accuracy of physician records and the accuracy with which it is coded for analysis. While the coding is governed by an International Classification of Diseases, this classification scheme has been continually modified so that only with great care can one be assured that trend lines represent the same disease states.

Detailed data suitable for analysis are generally available only for the United States as a whole. Most states are unable to provide similar detail. With cutbacks in the federal budget, this situation will worsen.

The National Center for Health Statistics also has conducted a health interview survey for a number of years. This is conducted on a sample basis, which limits the utility for data referrable to localities. With current concern for the impact of the occupational environment on health, one would hope there would be occupation-specific data, but this is not the case. Similarly, urban-rural differences in varying parts of the country would be useful.

The one noteworthy attempt to provide small-area data was the

National Cancer Institute's *U.S. Cancer Mortality by County*[3] in which sex- and race-specific, age-adjusted death rates were determined for a series of types of cancers for each county in the United States for the period from 1950 to 1969. Since a decade has passed since these data were collected, changes in rates on a small-area basis would be most helpful.

It would be helpful because overall death rates and death rates from nonrespiratory cancer in the white population have been falling over the past several decades. (See Figures 1-4.)[4]

The data represented in these figures are gross, but they provide the backdrop for reviewing environmental effects on health. We are in a period of generally improving mortality. Thus, not only from the legal, but the scientific point of view, the burden is placed on the complainant to show that in fact harm in terms of altered mortality experience is being caused by a substance introduced into the environment.

Adverse environmental influences so far appear to be confined to specific diseases in a very limited geographic area or with a very limited segment of the population. The evidentiary problem is to identify the specific aberration, and compare health status from one area to another. Not all attempts to do so are successful, as illustrated by the following instances.

A few years ago a study was published which purported to show a higher mortality rate for specific types of cancer in those counties of Ohio using surface water (i.e., from rivers and lakes) as their principal source than in those using deep wells.[5] The presumption was that surface water supplies were more susceptible to pollution.

A case was brought against a manufacturer of carbon tetrachloride who allegedly spilled some of the substance into the Kanawha River in excess of that permitted under regulations in effect at the time. It was contended that even the amount permitted was hazardous to the populations along the Ohio River into which the Kanawha empties.

Data to examine cancer mortality rates along the Ohio River were developed by reviewing not only the data for the counties bordering the river but also along Lake Erie and the principal cities in the cen-

[3] U.S. Department of Health, Education and Welfare (NIH), *"U.S. Cancer Mortality by County, 1950-1969"* (Washington, D.C.: U.S. Government Printing Office, 1974), pp. 74-615.

[4] S. Shindell, "The 'Hazardous' Environment — A Commentary," *Journal of Occupational Medicine* 25 (1981): 198-201.

[5] R.J. Kuzma, C.M. Kuzma and C.R. Buncher, "Ohio Drinking Water. Source and Cancer Rates," *American Journal of Public Health* 67 (1977): 725-729.

FIGURE 1
AGE-SPECIFIC DEATH RATES PER 100,000 WHITE MALES AGE 15 AND OVER FROM ALL CAUSES U.S. 1940—1975.
(IN ORDER TO OBTAIN FIVE-YEAR INTERVALS FOR THE EARLIER YEARS, VALUES WERE DERIVED BY INTERPOLATION.)

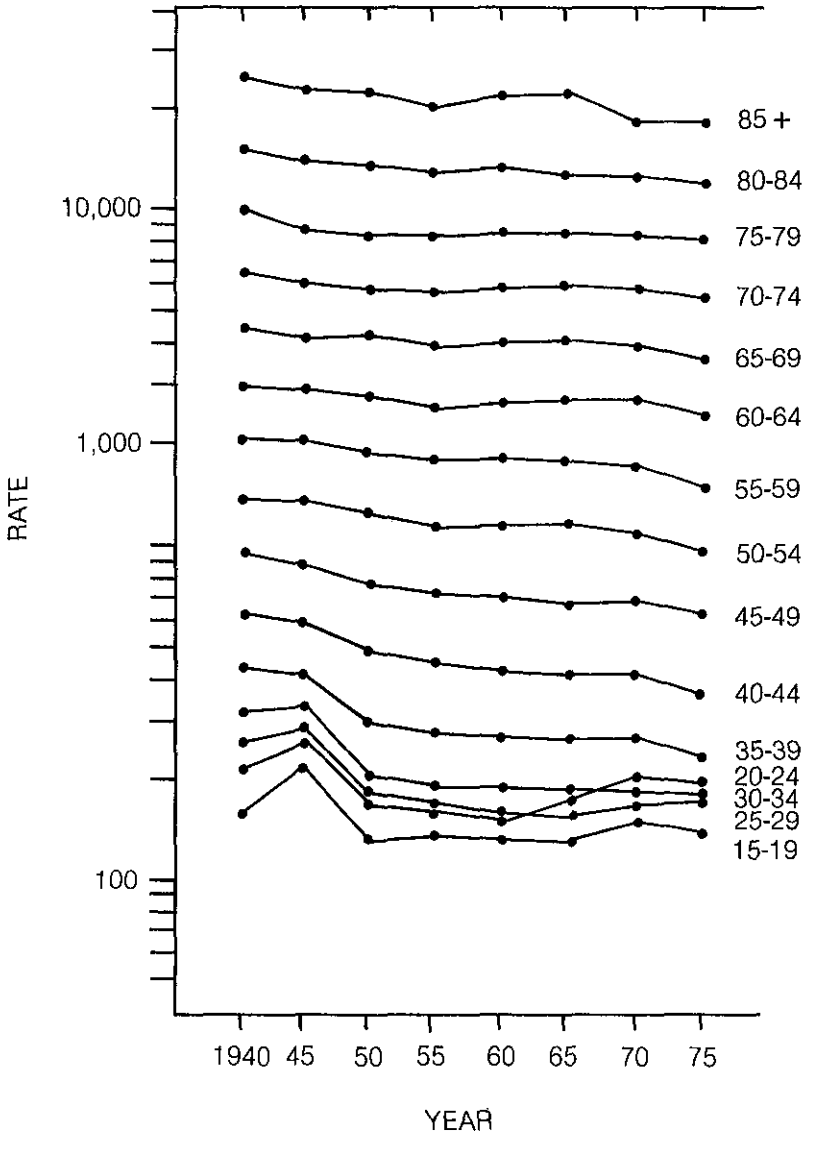


FIGURE 2
AGE-SPECIFIC DEATH RATES PER 100,000 WHITE MALES AGE 15 AND OVER FROM NONRESPIRATORY CANCER U.S. 1940—1975.

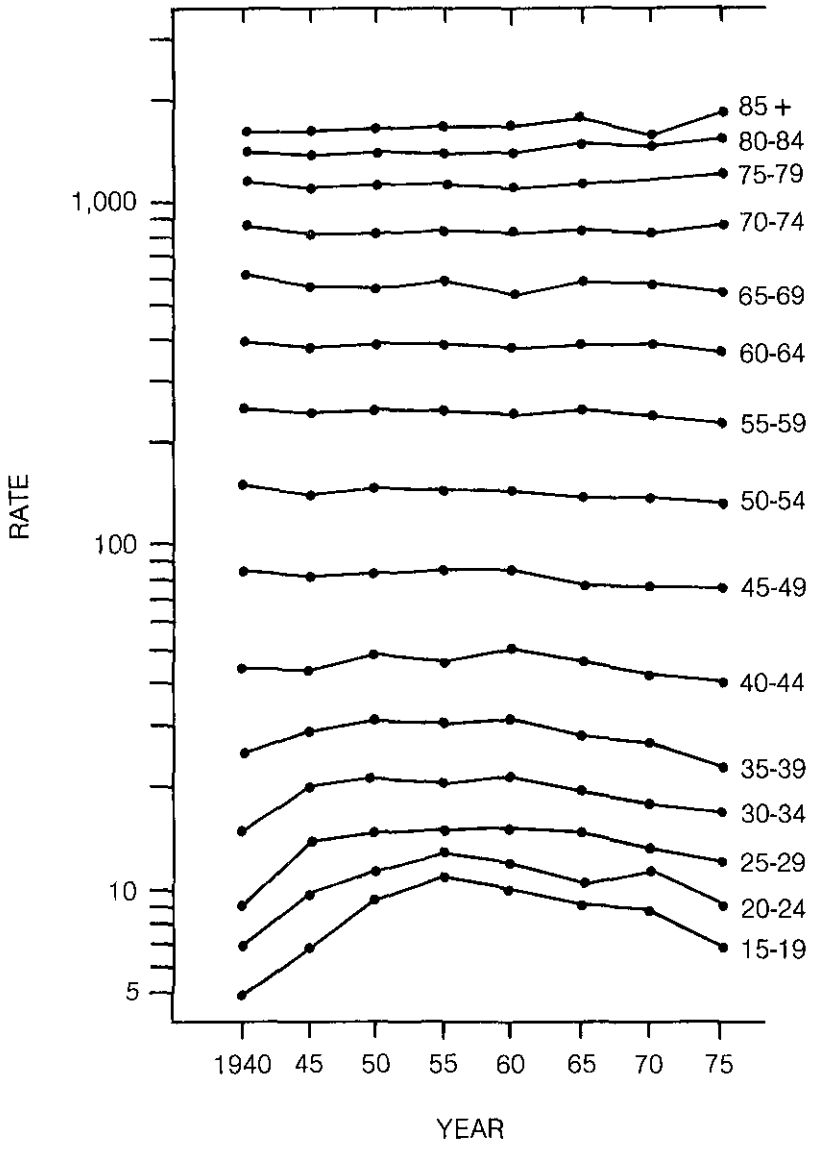


FIGURE 3
AGE-SPECIFIC DEATH RATES PER 100,000 WHITE FEMALES AGE 15 AND OVER FROM ALL CAUSES U.S. 1940—1975.

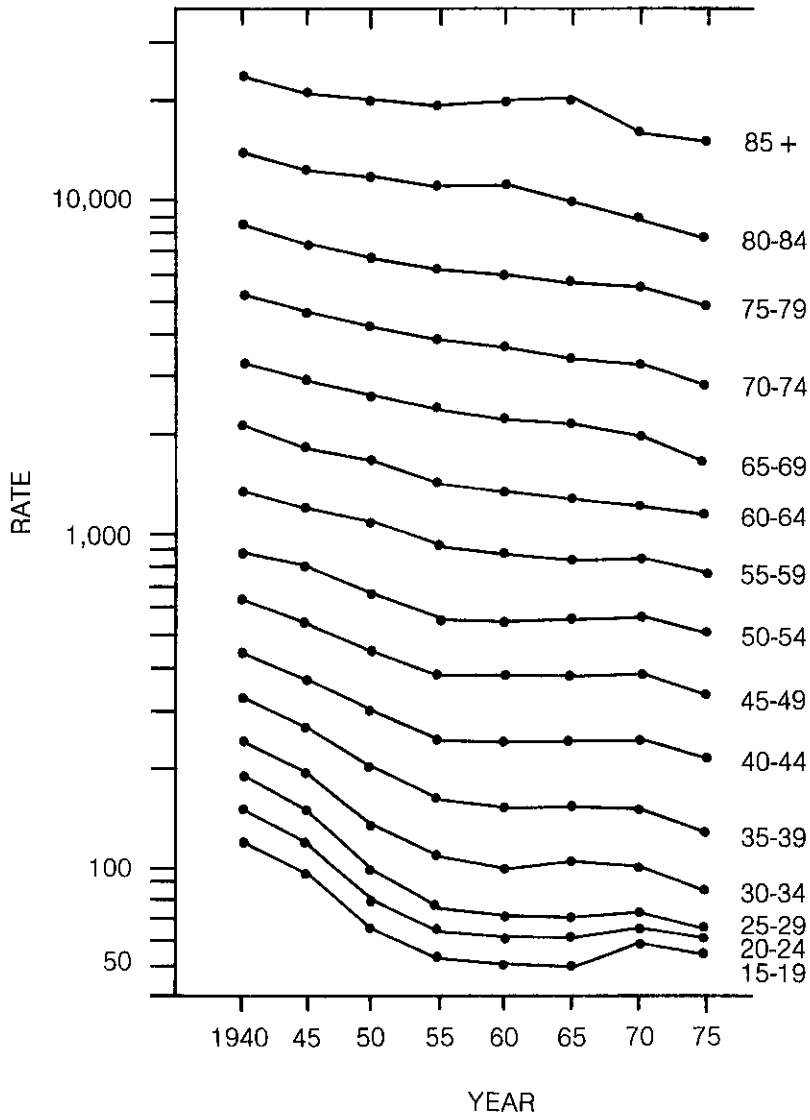
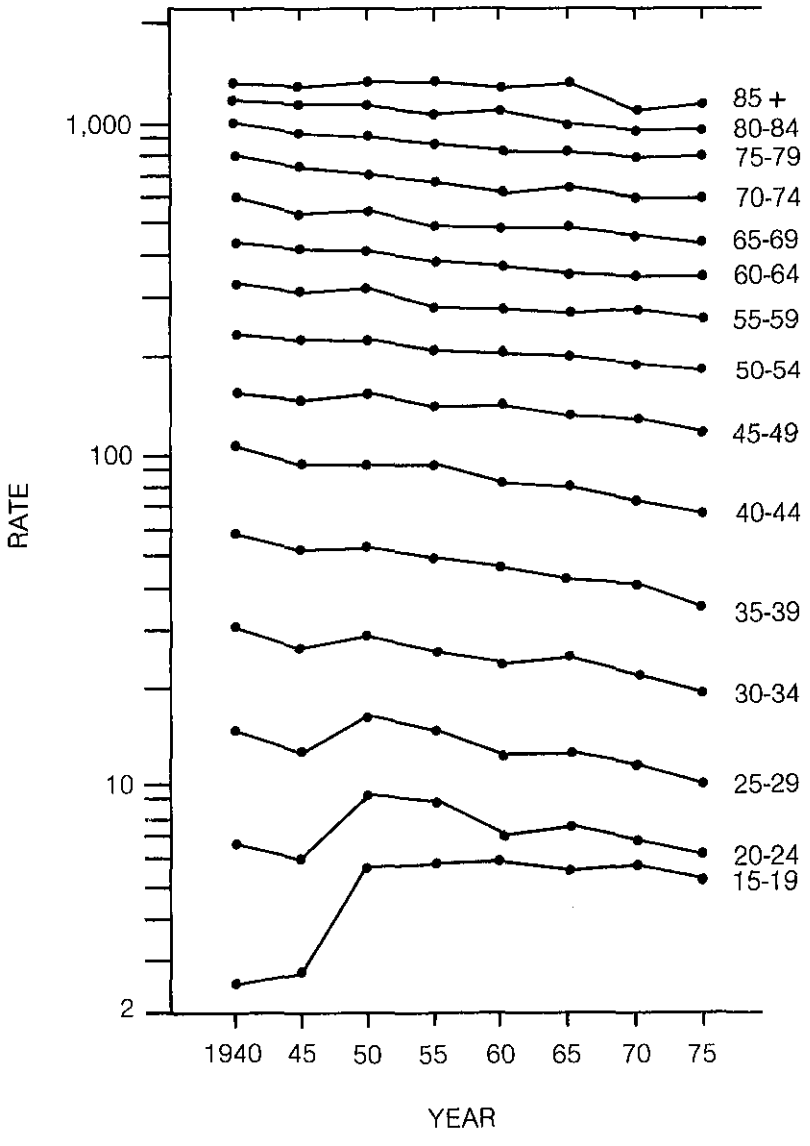


FIGURE 4
AGE-SPECIFIC DEATH RATES PER 100,000 WHITE FEMALES AGE 15 AND OVER FROM NONRESPIRATORY CANCER U.S. *1940—1975.*



tral part of the state.[6]

As Table 2 shows, the Ohio River counties have a generally lower cancer mortality rate than either of the other two groups. Steubenville, which has the second highest overall cancer rate of the Ohio River group, is actually upstream from the point where the Kanawha joins the Ohio, and could not have been affected by its waters. The county with the highest rate is the one in which Cincinatti is located. Located the farthest downstream, the substance would be more diluted than anywhere upstream.

Another instance involves a hearing on air pollution in West Virginia.[7] The contention was that the concentration of chemical plants in the area around Charleston was responsible for an adverse cancer death rate. To examine this question, the NCI study of cancer death rates by county was consulted, and a list of cities with an appreciably greater level of cancer mortality was prepared. (See Table 3.)

This table shows that at least for the 20-year period covered in the NCI report, for white males the age-adjusted cancer mortality rate in Charleston was below the national average and considerably below a number of cities without any heavy industry.

In neither of the examples was the question of the presence of harm in a specific circumstance answered directly. As we know, this is often very difficult to do. In both examples, inferences had to be drawn from comparative data.

We face increasing concern over pollution within a community and must look for variations of health measurements in specific population groups in a limited geographic area. Data are seldom readily available and are time-consuming and costly to develop. Yet the techniques for doing so have been known for a long time. A classic example is Dr. John Snow's study of the cholera mortality rate among users of different water supplies in London in 1854. (See Table 4.)[8] To obtain his figures, Snow not only had to allocate death registrations to specific addresses but also had to identify the source of water in each of the houses in the specific areas studied, primarily by canvassing the households.

[6]S. Shindell, unpublished data. (Presented in Federal Court, Prakersburg, W.Va., September 1977.)

[7]S. Shindell, unpublished data. (Presented at hearing of West Virginia Air Pollution Control Commission, 1 June 1979.)

[8]J. Snow, *On the Mode of Communication of Cholera,* 2d ed. (1854). Excerpts reprinted in S. Shindell, J.C. Salloway and C.M. Oberembt, *A Coursebook in Health Care Delivery* (Appleton-Century-Crofts, 1976), p. 456.

TABLE 2

CANCER MORTALITY (AGE-ADJUSTED RATES) IN SELECTED OHIO COUNTIES, 1950—1969

		All Sites (white male)	Stomach (white male)	Bladder (white male)	Stomach (white female)
Lake Erie Counties					
Cuyahoga (Cleveland)		211.9	20.7	8.4	10.3
Lucas (Toledo)		196.4	15.8	8.8	6.7
Lorain		189.7	20.2	7.5	9.8
Erie (Sandusky)		189.0	13.5	6.7	6.2
Lake		185.9	18.4	6.6	8.2
Ottawa		179.1	17.7	10.8	6.6
Ashtabula		178.5	21.8	6.9	10.4
Sandusky		158.3	10.8	5.8	6.5
	Median	187.5	18.1	7.2	8.0
Ohio River Counties					
Hamilton (Cincinnati)		203.8	12.6	8.8	6.3
Jefferson (Steubenville)		185.7	19.3	5.8	8.6
Lawrence (Ironton)		175.8	15.0	7.3	7.3
Scioto (Portsmouth)		173.7	15.6	4.9	7.8
Clermont		170.3	11.0	6.3	5.4
Brown		142.6	12.5	5.2	4.5
Gallia		140.9	9.3	3.8	4.2
Adams		130.4	10.2	5.0	4.7
	Median	172.0	12.6	5.5	5.8
Other Counties with Major Cities					
Mahoning (Youngstown)		203.0	23.1	6.5	11.0
Trumball (Warren)		186.6	19.1	7.0	9.0
Franklin (Columbus)		186.4	12.3	6.7	5.9
Summit (Akron)		185.5	16.2	8.0	8.2
Montgomery (Dayton)		175.9	11.7	6.9	6.2
Stark (Canton)		173.7	15.8	7.2	6.9
Clark (Springfield)		171.1	11.5	7.0	5.4
Butler (Hamilton)		163.7	9.6	5.6	5.7
	Median	180.3	14.0	7.0	6.6

SOURCE: *U.S. Cancer Mortality by County, 1950-1969.*

TABLE 3

AGE-ADJUSTED CANCER DEATH RATES IN WHITE MALES (PER 100,000 POP.) IN SELECTED CITIES AND COUNTIES (1950—1969)

CITY	COUNTY	STATE	
Baltimore	—	Maryland	233.3
Boston	Suffolk	Massachusetts	223.3
Philadelphia	Philadelphia	Pennsylvania	221.1
New Orleans	10 parishes	Louisiana	220.2
St. Louis	—	Missouri	220.1
New York	6 counties	New York	215.3
Charleston	Charleston	South Carolina	214.5
San Francisco	San Francisco	California	212.0
Cleveland	Cuyahoga	Ohio	211.9
Detroit	Wayne	Michigan	209.2
Buffalo	Erie	New York	207.0
Chicago	Cook	Illinois	205.9
Providence	Kent & Providence	Rhode Island	205.7
Cincinnati	3 counties	Ohio & Kentucky	204.1
District of Columbia			203.7
New Haven	New Haven	Connecticut	203.6
Milwaukee	Milwaukee	Wisconsin	203.2
Wheeling	Ohio	West Virginia	203.1
Youngstown	Mahoning	Ohio	203.0
Pittsburgh	Allegheny	Pennsylvania	202.1
Weirton	Hancock	West Virginia	200.7
Charleston	Kanawha	West Virginia	172.3
United States			174.0

SOURCE: *U.S. Cancer Mortality by County, 1950-1969.*

Snow was concerned with only one disease. Today we are concerned with deaths from a variety of causes as well as other measures of harm, such as the influence of a possible pollutant on mental functioning, on the ability to reproduce, and on the frequency of congenital malformations.

Since data are limited we must approach each problem almost the same way John Snow did over a century ago. It is possible to develop monitoring systems, but the increasing difficulty of gaining access to the needed information will make our task harder.

TABLE 4

"PROPORTION OF DEATHS TO 10,000 HOUSES, DURING THE FIRST SEVEN WEEKS OF THE EPIDEMIC, IN THE POPULATION SUPPLIED BY THE SOUTHWARK AND VAUXHALL COMPANY, IN THAT SUPPLIED BY THE LAMBETH COMPANY, AND IN THE REST OF LONDON."

	Number of Houses	Deaths from Cholera	Deaths In Each 10,000 Houses
Southwark and Vauxhall Company	40,046	1,263	315
Lambeth Company	26,107	98	37
Rest of London	256,423	1,422	59

Did the Pollutant Cause the Harm?

Basically, if the first three questions have been answered affirmatively, the last question -- "Is there evidence that the harm is the result of the substance that was introduced?" -- is a matter of presumption. If we had shown that a substance had been introduced into a population subgroup and that the substance is capable of producing harm, and in fact such harm is present in the subgroup, we may conclude that we have shown the harm present to be due to the substance that was introduced.

The matter may not be quite so simple, however. Often we may demonstrate that the presence of pollution and of harm are associated geographically or in time, but this does not necessarily mean that one has caused the other.

The major problem in determining whether there is a causal relationship between a hazard and a health effect is that most health effects occur to some degree whether or not the hazard is present. A health effect is only causally related if:

(a) it would not be expected to occur in the absence of the hazard; or

(b) it would not have occurred with the frequency experienced had it not been for the presence of the hazard.

We are fairly comfortable in suggesting causal relationships in the case of unusual diseases. The observations that angiosarcoma occurred in vinyl chloride workers and mesothelioma in asbestos workers led to the conclusion that the disease and the exposure

were related because of the extreme rarity of these conditions in the general population.

It is also easier to assume a causal relationship when a health effect is dramatic, such as in the smog experiences in London, England and Donora, Pennsylvania. There people died from "ordinary" diseases, but in obviously excessive numbers.

In the absence of these conditions, however, it is much more difficult to demonstrate that a negative effect is caused by pollution. The best we can do is monitor and analyze indices of ill health on a continuing basis and watch for change. This will allow us to associate changes in pollution with measures of ill health, and to evaluate the effectiveness of our abatement technology.

It is interesting that while many individuals presume pollution, per se, is hazardous and should be abolished, others note that its elimination is neither technically possible nor efficient. We could never achieve a perfectly clean environment, nor would it be efficient to aim at such a policy goal. Resources for pollution abatement are scarce and subject to the principle of diminishing returns.

In the limited environment of the workplace, we have been able to determine threshold limit values for the more common substances. Individual workers may be monitored for acute manifestations of toxicity, but industries rarely maintain a formal monitoring system that systematically aggregates the data concerning the individual workers. The absence of such a system means we shall continue to be unable to resolve the evidentiary problems posed in this paper.

Techniques for precise monitoring are available.[9] If done appropriately, an employee health surveillance system can be cost-effective not only in minimizing on-the-job injury and illness but also in increasing productivity and reducing costs of Workers' Compensation and health insurance. A health surveillance system can also provide data for defense of unwarranted compensation and product liability claims.

Techniques to monitor the health of the general population also exist but are not sufficiently utilized. There is a mechanism to monitor mortality, but it has its shortcomings. Age-specific mortality rates by cause for the United States do not become available until three years later. Except for rough figures for rates, there are no trend lines, and data for states, regions, and cities are not produced. Data have been collected to perform such analyses, but no commit-

[9]S. Shindell and H.M. Goldberg, "Surveillance Systems: What to Include and Why," *Occupational Health and Safety* 50 (1981): 34-56.

ment has been made by the National Center for Health Statistics. Their official policy is that they are responsible for collection, not analysis, yet without analysis there is little rationale for data collection.

Conclusion

This paper has investigated the evidentiary problems relating to causation and harm in pollution-engendered torts. I have suggested ways to improve the collection and use of data pertaining to potentially harmful substances. I conjecture that as our monitoring techniques improve, we will find that our past performance against pollution has been tolerably good. Meanwhile, such improved monitoring ought to promote the health of our population, because with early detection significant harm can be avoided from hazardous wastes.

Finally, I suspect that a new source of liability is about to arise. Negligence exists when one knows or should have known of the presence of a hazard. As the technology becomes available to conduct stricter monitoring of the environment, it seems plausible that actions will be brought for failure to institute a surveillance system that could have warned of a potential hazard in a specific environmental circumstance.

FIVE ISSUES OF CAUSATION AND PROOF

Randy Barnett

In any discussion of causation in torts cases in general and pollution-engendered torts cases in particular it is necessary to distinguish two types of inquiry. The first is the nature of causation in torts; that is, what do we mean when we say that one person causes harm to another? Given an answer to this question, the second inquiry is how we demonstrate the causal connection in practice; that is, how do we prove that a particular activity by one person has caused a certain harm to another? It seems fair to say that while causal questions of the first sort are not more vexing in pollution cases than in most other types of torts cases, the second kind of inquiry can present quite serious and sometimes intractable difficulties.

With this distinction in mind, Professor Shindell's paper[1] can be seen as a provocative exploration of some problems faced by those attempting to demonstrate a causal link between a particular pollutant and a subsequent health effect. In my comments I will attempt to put his analysis in the context of five common legal issues of causation and proof. The first is the concept of causation employed in the analysis. The second is the nature of the evidence used by Professor Shindell. The third is the standard of proof that should be applied. The fourth is the question of who should bear the burden of proof. Finally, I shall briefly consider the need for a causal requirement. Though I will not here attempt to conclusively resolve these issues, much confusion will be avoided if they are kept in

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[1]Sidney Shindell, "Evidentiary Problems in Pollution-Engendered Torts" *Cato Journal* 2 (Spring 1982): 137-155.

mind when discussing problems of environmentally engendered torts.

The Nature of Causation

In the course of his excellent discussion, Professor Shindell makes an observation that if taken seriously can only result in causal problems becoming more acute than necessary. Concerning the nature of the causal relationship between a health hazard and a health effect, he says:

> A health effect is only causally related if:
>
> (a) it would not be expected to occur in the absence of the hazard, or
>
> (b) it would not have occurred with the frequency experienced had it not been for the presence of the hazard.[2]

This characterization of the causal issue is a form of the well-known "but for" test of causality.[3] Professor Shindell might have said, "But for the hazard, a health effect would not have occurred as frequently." Notice, as Professor Epstein has pointed out, that this expression "is in counterfactual form and requires an examination of what *would have* been the case if things had been otherwise."[4] It does not ask the more straightforward question of what caused the health effect being considered.

The problem created by such a formulation is that it fails to identify the agent causally responsible for the health effect. On this view any occurrence that was a necessary condition for the health effect is a cause of the effect. Thus, in the case of water pollution, were it not for the consumption of the contaminated water no health effect would have occurred. Is the drinking of contaminated water to be considered a "cause" of the same order as the existence of the contamination? The "but for" test cannot distinguish among necessary conditions, and efforts to identify the "proximate" cause have met with little success.[5]

[2]Ibid., p. 153.

[3]See generally William L. Prosser, *Handbook of the Law of Torts,* 4th ed. (St.Paul, Minn.: West Publishing Co., 1971), pp. 236-244.

[4]Richard A. Epstein, "A Theory of Strict Liability," *Journal of Legal Studies* 2 (1973): 160.

[5]See ibid., pp. 160-189; idem, "Causation and Corrective Justice: A Reply to Two Critics," *Journal of Legal Studies* 8 (1979): 477; see generally H.L.A. Hart and A.M. Honore, *Causation of the Law* (London: Oxford University Press, 1959); Prosser, *Law of Torts,* pp. 244-289.

This type of counterfactual concept of causality is unnecessary. The statement, "asbestos-caused mesothelioma" needs no more linguistic reformulation than "arsenic-caused death" or "the blow caused a broken nose." Though the factual chain of causation could be very long indeed, the causal paradigm operating in pollution cases is simply, "A poisoned B." It should be stressed that such a change in the causal language Professor Shindell has employed would not affect the merit of his analysis. What he is observing in this passage is the fact that the only evidence of a causal link between the hazard and the harmful effect may be a statistical association. It is to this issue that I now turn.

The Nature of Causal Evidence

The causal link between the defendant's conduct and the plaintiff's injury is a fact that must be proved in every action in tort. The evidence offered in proof may be of two kinds: Direct and circumstantial. Direct evidence proves the fact in issue without reference to any intermediate inference. Most often it takes the form of eyewitness testimony to the effect of "I saw A hit B in the nose." This is direct evidence of the identity of B's assailant and the nature of the attack. Of course it may be believed or disbelieved by the trier of fact. If accepted as true, however, these facts are, without more, proved. Circumstantial or indirect evidence is evidence of facts that together with other proof or reasonable inferences can be indicative of a fact in issue — here causation. An example of circumstantial evidence is a fingerprint found at a crime scene that might indicate presence, but not time or circumstances. It should not be assumed that one form of evidence is inherently more reliable than the other. Often circumstantial proof will be preferred to eyewitness testimony.

It is unlikely that causation in cases of poisoning or pollution could ever be other than circumstantial. Even if a witness saw A pour arsenic into B's tea, the fact that B's subsequent death was caused by the arsenic would need to be inferred from an examination of B's remains. And the degree of certainty with which such a conclusion can be made will depend upon the strength of the direct and circumstantial evidence as well as upon the extent of our understanding of the effect of arsenic on the human body that permits us to make inferences from the circumstances. It should not be thought that causal questions are unique in this regard. A person's state of mind must almost always be inferred from that person's

behavior, though factual presumptions are also employed.[6]

The evidence that Professor Shindell advances as proof of causation in pollution cases takes the form of statistical associations, which are (given certain epistemic assumptions) one kind of circumstantial evidence. When should this type of evidence be allowed to supplement or substitute for direct proof of causation? If our answer is *never,* then we are in effect granting immunity to most polluters for whatever harm they may cause. If our answer is that *any* association will always be dispositive, then we will inevitably enjoin industrial activities that are not in fact harmful. Assuming that statistical associations may be a way of discovering the existence of a causal link, and further, that the strength of any such inference will depend on the degree of association, then we must determine what degree is sufficient for a finder of fact to conclude the existence of the causal link. What should be the standard of proof?

The Standard of Proof

The standard of proof to be adopted in a given type of case will depend on the nature of the interests that would be affected by an incorrect decision. So even if we believe, as I do not, that it is sometimes justified to protect persons from harming themselves by the consumption of some chemical like nicotine or saccharin, we may be unwilling to do so on the basis of a "mere" statistical association between consumption and the incidence of cancer in humans or more attenuated inferences drawn from the results of tests on other animals. In the case of an involuntary ingestion[7] of the same chemical, however, our standard might differ and such evidence be allowed. When the interest affected is individual liberty, as it is when the sanction is imprisonment for a crime, the standard adopted is a high one, but not one that guarantees no error.

The problem posed by pollution cases, indeed all private tort actions, is that a decision in favor of one party will be a decision against the other. Assuming our law is based on individual rights, an erroneous judgment will unjustly restrict the losing party's liberty. To place a greater burden, then, on one party than the other would seem to provide a greater protection for the rights of the party with the lesser burden. All things being equal, we should place

[6] We say, for example, that a person is presumed to intend the natural and ordinary consequences of his or her actions as a matter, perhaps, of introspective insight.
[7] E.g., poisoning cases of which pollution cases may be considered a species.

the same standard of proof on all parties. But all things are not equal. The standard of proof will depend on factors apart from the seriousness of the interests at stake.

Standards of proof that amount to a (rebuttable) presumption in favor or against finding a particular fact to be true will also depend on our common understanding of the world. Hence, if we believe that in most instances chemicals released into the environment will not cause adverse health effects or if we believe that the chances of a particular act of pollution being responsible for a given injury is quite low, we may adopt a presumption to that effect. In doing so we are saying that recovery may be allowed provided the plaintiff demonstrates the truth of his or her claim beyond a certain level of doubt.

This type of analysis can help account for and justify a different standard of proof in judging requests for damages after a harm has been sustained than is used in cases where injunctive or preventive relief is sought. Surely the standard of "substantial risk of imminent harm" applied to pleas for injunctive relief reflects both our uncertainty about whether a harm will in fact occur if the activity is allowed and an even more fundamental doubt about the feasibility of demonstrating such a fact.

Allocation of the Burden of Proof

Yet a fourth issue must be identified in pollution-engendered torts cases and that is: Who shall be required to produce evidence of a certain fact? Put bluntly, must a plaintiff prove that the defendant's emissions caused or will cause a harm or must the defendant prove this is not or will not be the case? Allocation of this burden will involve both factors mentioned in regard to the standard of proof, but will also depend upon the principle of justice that prohibits individuals from being forced to rectify injuries they haven't caused. Before acting a court will need a reason to single out the defendant, and this reason has traditionally been demanded of the requesting party.[8] More will be said on this point in the next section.

[8]Two aspects of "burden of proof" have been distinguished: The burden of going forward with evidence; i.e., which party must raise an issue by competent evidence and the burden of persuasion; i.e., once raised, which party must satisfy the trier of fact by proof beyond a certain level of doubt. It may, for example, be the rule that a plaintiff must present some evidence indicating that the defendant caused an injury and then the defendant must prove beyond a preponderance of the evidence that he did not. An allocational choice must be made here that will involve much the same concerns outlined throughout this comment.

The Doctrine of *Res Ipsa Loquitur* and the Need for a Causal Requirement

Res ipsa loquitur as traditionally formulated is not a doctrine that speaks to *causal* issues but one which specifies certain circumstances where the defendant's conduct is rebuttably presumed to be *negligent*. In these cases where it is shown that the defendant's conduct harmed the plaintiff the burden will shift to the defendant, who then must prove that he was not negligent. A case subject to this doctrine is one of *strict liability*,[9] and if this presumption is held to be legally unrebuttable then the theory is one of *absolute liability*. In neither case are causal barriers lowered except in one sense: If a plaintiff must show that defendant acted negligently, it must also be shown that it was the defendant's negligence that caused the harm to the plaintiff. To illustrate this, suppose that while driving my car at twice the speed limit I run you over. If it can be shown that had I been operating the car at normal speed, (i.e., non-negligently), I still could not have avoided hitting you — because of lighting, your clothing, etc. — then on a negligence theory there is no causal link between my speeding (i.e., the negligence) and your injury even though my actions were concededly the cause-in-fact of your injuries, and you will be denied recovery.[10]

In this way *res ipsa loquitur* may limit the more extensive and complicated causal inquiry required by a theory of negligence, but it does not eliminate the need to prove that the defendant's conduct caused the plaintiff's injury.[11]

Persons should not be held liable for harms that we don't or can't know they caused. The problem of limited knowledge is an inherent quality of human existence. We must always operate on less than perfect information. A free society that recognizes individual rights consistent with individual liberty must adopt certain principles of justice, the most important of which is that a person will be free to use and enjoy his or her person and property unless that use invades the equal rights of his or her neighbors. Any proposal that preventively restricts human action or retroactively penalizes individuals for conduct that has *not* infringed on another's

[9] See Epstein, "A Theory of Strict Liability"; idem, "Defenses and Subsequent Pleas in a System of Strict Liability," *Journal of Legal Studies* 4 (1975): 391.

[10] In theory, that is. The bias in favor of strict liability in such cases is so great that even within a nominally negligence system a defendant would be lucky to escape liability absent serious misconduct by plaintiff.

[11] Contra Robert Best and James Collins, "Legal Issues in Pollution-Engendered Torts," *Cato Journal* 2 (Spring 1982): 101-136.

rights has seriously undermined the very concept of individual liberty. In the absence of proof to the contrary, we must presume that one person's conduct is not invasive of another's rights if we wish to remain a free society. There is no middle course.

Conclusion

It is important to be aware that decisions concerning the five issues of causation and proof raised here must be made and that any decision reached will favor one party at the expense of the other. Ultimately, these issues must be resolved by an appeal to notions of how the world works, how we come to understand it, and where justice lies.