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CAUSATION AND THE DEFENSE OF TOXIC TORT LITIGATION

MARC STEVEN COLEN

Jones, Day, Reavis & Pogue, 355 So Grand Avenue, Suite 3000, Los Angeles, CA 90071 (U.S.A.)*

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Summary

This paper concerns the nature of the burden of the plaintiff of proving causation in a toxic tort lawsuit, and correlates the techniques available to the defendant to defeat that proof of causation. Causation is shown to be comprised of two components: cause-in-fact, and proximate, or legal cause. Of these, cause-in-fact is far more critical to the defendant in toxic tort litigation, inasmuch as it is more readily refuted. Proximate cause is a policy determined concept and is thus somewhat less amenable to defense attack. In this paper, the factual elements of cause-in-fact that the plaintiff must prove are presented and the requirements for proof are discussed. Challenging proof of cause-in-fact is then extensively considered. Finally, proximate cause and the defense of intervening - superceding causation is discussed.

I. Introduction

In toxic tort litigation, causation is typically the plaintiff's most difficult element of proof and the most critical issue litigated. The plaintiff must establish that there is a causal link between the plaintiff's exposure to a hazardous substance and his subsequent injury, disease, or infirmity. This is true whether the theory under which the plaintiff is desirous of recovering is negligence, strict liability, or some other theory of liability.

For many purposes, causation may be considered as having two distinct components: cause-in-fact and proximate cause. Cause-in-fact (sometimes referred to as actual cause) concerns whether the defendant's actions were in fact or actually connected by some physical events to the plaintiff's injury, disease, or infirmity.

In *Proving Causation in Products Liability Cases* [1], the following definition was proposed:

"Cause-in-fact, on the other hand, is merely the question of whether or not the evidence is sufficient to prove causation." [2]

*The views expressed in this paper are those of the author and necessarily those of Jones, Day, Reavis and Pogue.

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In the vast majority of tort cases, cause-in-fact is not particularly difficult to determine. The traumatic event that is alleged to have caused the injury occurs immediately prior to the occurrence of the injury, and that injury is one which is capable of being caused by such an event. In the case of toxic torts, however, cause-in-fact is more difficult to determine. The injury or disease may not be contemporaneous with the event; there may be little information concerning the natural causation of such injury or disease.

Inasmuch as this is a burden that many courts have apparently found too heavy for a plaintiff to bear, those courts have altered the plaintiff's burden of proof. Inference, circumstantial evidence, and other mechanisms – some rather tenuous – have been permitted.

Historically, the determination of cause-in-fact has been considered a question of fact to be determined by the jury. Policy considerations were not part of a cause-in-fact determination.

Proximate cause (often referred to as legal cause) concerns whether, for the specified physical connection, that connection is sufficiently close to allow compensation to the plaintiff.

In contrast to cause-in-fact, proximate cause has long been recognized as largely a policy determination. In the case of *Palsgraf* v. *Long Island R.R.* [3], Judge Andrews noted in his dissent that:

"What we...mean by the word 'proximate' is that, because of convenience, of public policy, of a rough sense of justice, the law arbitrarily declines to trace a series of events beyond a certain point. This is not logic. It is practical politics...."It is all a question of expediency." [4]

Recently, proximate cause considerations for toxic torts have become increasingly more liberal. Therefore, it often appears that the plaintiff need only seriously consider the cause-in-fact issues; that is, proof of actual causation.

There is ample opportunity for the defense to actively defend a toxic tort lawsuit by attacking the plaintiff's attempts to prove causation – particularly cause-in-fact. Indeed, as medical science progresses, we may expect to find that the ability to defend against toxic tort cases is improved as a result of the increased information available. (Plaintiffs typically rely on animal testing and limited epidemiological studies, whose actual significance, as will be discussed, may be minimal.)

II. The critical elements of the theories of recovery

There are a number of possible theories for recovery in toxic tort litigation, including the following:

1. negligence;
2. strict liability;
3. strict liability for ultrahazardous activity;

4. negligent infliction of emotional distress;
5. intentional infliction of emotional distress;
6. nuisance;
7. misrepresentation;
8. breach of implied warranty; and
9. trespass.

Of these, negligence and strict liability are the most prevalent.

Negligence requires a display of the following elements:

1. Duty of care owed by the defendant to the plaintiff;
2. Negligent conduct on the part of the defendant;
3. Compensationable injury (damages) suffered by the plaintiff;
4. The defendant's conduct as the cause-in-fact of the plaintiff's injury; and
5. The defendant's conduct as the proximate (or legal) cause.

Strict liability is comprised of three separate forms of liability, according to the *Restatement of Torts (Second)* [5], as follows:

1. Manufacturing defect, e.g., where a well-designed product reaches a plaintiff in an unreasonably dangerous, defective condition;
2. Design defect, e.g., where a product is unreasonably and dangerously defective in its design; and
3. Inadequate warnings, where a product is unavoidably dangerous.

Of these, inadequacy of warnings is the aspect of product liability that comprises the preponderance of toxic tort litigation. The elements that must be proven by the plaintiff for this theory are as follows:

1. The defendant had a reason to expect that danger could arise from the use of its product;
2. The defendant failed to give adequate warning of the dangers of using the product;
3. Compensationable injury (damages) was suffered by the plaintiff (as for negligence); and
4. There exists a causal relationship between the defendant's failure to warn and the injury suffered by the plaintiff (as for negligence). [6]

The advantage of a strict liability theory to the plaintiff is clear: it is not necessary to establish a duty to the plaintiff by the defendant and it is not necessary to establish proximate cause, both of which are presumed under the theory.

The remaining theories of recovery for toxic torts have their own elements that must all be proved. The theories all have, however, two elements in common: first, the existence of a compensationable injury, and second, the existence of a causal relationship between some action by the defendant and the injury suffered by the plaintiff. Thus, successful denial of proof of the causal relationship between the plaintiff's injury and the defendant's action will defeat *any* toxic tort theory.

III. Proof of cause in fact

A Introduction

In *Proving Causation in Toxic Tort Litigation: Factual Problems* [7], Messrs. Zeiger and Jones state that there are four primary factual elements to proof of cause-in-fact in toxic tort litigation, which are as follows:

1. Isolation of the toxic substance to which the plaintiff was allegedly exposed;
2. Identification of the injury or disease allegedly caused by the exposure;
3. Establishment of the causative relationship between the injury or disease and the toxic substance; and
4. Accumulation of precise and specific evidence (often termed particularistic evidence) [8].

B Isolation of substance

Isolation of the toxic substance may be very easy or very difficult. In the case of DES or asbestos litigation, identification is simple. Such is not the situation if the plaintiff has been injured after exposure to various agricultural products, for example, each of which is comprised of numerous chemicals, many or all of which may be potentially harmful.

C Identification of disease

Identification of the injury or disease may also be easy or difficult to determine, depending on the circumstances. A well-documented specific diagnosis may assist the plaintiff in accurately ascertaining causation. For example, certain forms of cancer have been linked to asbestos, while others have not. A specific diagnosis of mesothelioma, one of the forms of cancer so linked, will be far more beneficial than a more generalized diagnosis. Of course, in the event that the actual diagnosis is one which has no established connection with the substances which allegedly caused the disease, then the plaintiff's case has minimal value.

D Causative relationship

Establishing the causative relationship between the injury or disease and the toxic substance is frequently difficult. This is typically done using epidemiologic evidence and animal studies, although other methods exist; one of these, *in vitro* testing, will be discussed below.

1 Epidemiological studies

Epidemiological studies evaluate human disease patterns using statistical and other analytic techniques in order to determine whether a statistically significant, and therefore valid, causal relationship exists between the disease being studied and the factor suspected of being related to that disease.

It is important to note that epidemiology is a study of populations, not of

individuals. It is, however, often used for purposes other than originally intended; viz, to prove causation.

There are a number of different types of epidemiologic study methodologies which vary in their significance, including the following:

a. Cohort or prospective studies, which involve the study over time of two groups, one of which has been exposed to the factor being analyzed;
b. Case-control studies, which are retrospective, comparing a group of people who have a disease and a group of people who do not, but who are as similar as possible to the test group;
c. Cross-sectional studies, which are surveys based upon reports by individuals concerning past exposures and diseases;
d. Correlation studies, which compare demographic data regarding two or more populations; and
e. Case reports, which are merely reports on individuals and do not refer to any other (control) group [9].

Of these methodologies, the first is the strongest, the second less so, and the remaining three less reliable, although of some use to a plaintiff attempting to establish a causal relationship.

Analysis of the study data by epidemiologists involves first determining that a statistically significant relationship exists between the factor or agent being studied and the disease that is alleged to be related to such factor or agent. As discussed by Black and Lilienfeld in *Epidemiologic Proof in Toxic Tort Litigation* [10]:

> "[T]he epidemiologist's next task is to estimate the magnitude of the association. The accepted means of measuring such an association is the calculation of relative risk, which is the ratio of the incidence rate of the disease in the exposed group divided by that rate in the non-exposed 'control' group." [11]

Even if an epidemiologic study has been conducted in accordance with all of the accepted principles, the only results which the study can demonstrate is that the risk associated with exposure to a given agent exceeds that which is found in a control group. The ratio of the observed cases to the expected cases is called the relative risk.

However, epidemiology alone does not provide a probability that a given case is related to particular exposure. The concept of probability of causation was developed to meet this need. To determine the proportion of all cases of a disease in the total population attributable to a specific exposure, the following formula may be used:

$$\text{Percentage probability} = \frac{\text{Relative risk} - 1}{\text{Relative risk}}$$

This equation also reflects the change that any given individual in a particular

population developed his disease as a result of exposure to a particular agent. It must be noted, however, that this simplistic analysis ignores the complex interrelation of factors that may cause a disease. Further, the most that can be said about this analysis is that as a matter of probabilistic analysis, the chances that the individual acquired the disease being studied due to the agent are as shown. If the relative risk is greater than 2, then the percentage probability is in excess of fifty percent. However, that percentage does not substantiate, by any means, that a particular individual could not be the victim of another causative agent instead.

2 Animal studies

Animal studies involve the use of animal models to assess the potential health risks to humans arising from chemical exposure. Inasmuch as these are models that require extraordinary extrapolation to humans and to typical human dosages, such studies are largely a matter of opinion and speculation. Although they have been relied on extensively by the government in assessments of health risks, their actual reliability at proving causation is minimal.

3 Other studies

Among the other methods of demonstrating the causative relationship are *in vitro* tests. Although such testing is still not prevalent, it is expected to expand dramatically in its usage. The statistical inferences of epidemiology and the extrapolation from weak models in animal studies are avoided. Only *in vitro* testing permits the low dosage and high volume testing that is requisite to accurate determination of the true propensity for substance to have an adverse reaction on human tissue.

E Particularistic evidence

As previously discussed, particularistic evidence is usually required by the courts. It is also, for the plaintiff, a more cost effective method for obtaining evidence than through epidemiologic or animal studies. Particularistic evidence includes any evidence tending to prove causation in the *particular* plaintiff.

IV. Requirements for proof of causation in fact

A Standard of proof

The plaintiff's burden as to the standard of proof is the same as for all of the elements of the theory of recovery which the plaintiff is advancing; viz, a preponderance of the evidence. Although the courts have continued to decrease the burden on the plaintiff, this requirement remains intact. In *Epidemiologic Proof in Toxic Tort Litigation* [12], Black and Lilienfeld noted that:

"Even commentators who have advocated changes to make it easier for plaintiffs to recover in toxic tort cases have explicitly recognized that the more-likely-than-not test is the present rule." [13]

Mathematically, preponderance of the evidence (or the more-likely-than-not test) translates to a variable in excess of fifty percent; indeed, this is how the courts have so construed it [14]. Thus, expert testimony that a toxic substance "possibly" caused the plaintiff's injury is typically deemed insufficient, while testimony that a toxic substance "probably" caused the plaintiff's injury is generally sufficient [15]. As stated in *Parker* v. *Employers Mutual Liability Ins. Co. of Wisconsin* [16]:

"A possible cause only becomes a probable cause when in the absence of other reasonable causal explanations it becomes more likely than not that the injury was a result of its action. This is the outer limit of inference upon which an issue can be submitted to the jury." [17]

This traditional standard of proof of causation was delineated in 1959 by the United States Supreme Court in the case of *Sentilles* v. *Inter-Caribbean Corporation* [18], where the Supreme Court held that a jury could properly infer that a particular accident had caused a plaintiff's injury, despite the fact that there was an absence of any direct medical testimony that the accident had in fact caused the injury.

In the *Sentilles* case, a seaman brought a claim under the Jones Act to recover for a tubercular-type disease which he alleged was caused by an accident at sea and for which the owner of the ship was liable. His theory was simply that the accident either activated or aggravated a previous tubercular condition.

At trial, three medical experts testified and gave the following three opinions:

1. Acute dissemination of the tuberculosis *might be* a consequence of the accident;
2. The trauma and the pre-existing injury were the *most likely* causes of the aggravation of the tuberculosis (although he was unable to identify which of the two was more likely to be responsible); and
3. The accident *probably* aggravated the condition.

The jury returned a verdict in favor of the plaintiff.

The defendant appealed, contending that the evidence could not justify the verdict of the jury concerning causation. The Supreme Court affirmed the jury's verdict, holding that:

"The jury's power to draw the inference that the aggravation of petitioner's tubercular condition, evident so shortly after the accident, was in fact caused by that accident, was not impaired by the failure of any medical witness to testify that it was in fact the cause. Neither can it be impaired by the lack of medical unanimity as to the respective likelihood of the potential causes of the aggravation, or by the fact that other potential causes of the aggravation existed and were not conclusively negated by the proofs." [19]

B Statistical evidence

Although, in theory, a plaintiff could satisfy the preponderance, or more-likely-than-not standards by merely showing that he was exposed to a toxic substance known to cause the type of injury that the plaintiff suffers from more than fifty percent of the time, the courts predominantly refuse to allow judgements which are based on statistical analysis alone [20].

The courts have demonstrated a long-standing antagonism to the use of mere statistical evidence, which antagonism derives from nontoxic tort litigation. The various rationales which underlie the antagonism include the following:

1. A belief that particularistic evidence is more trustworthy than bare statistical evidence;
2. A desire to encourage juries to believe in the truth of their own decisions; and
3. A desire to attain a higher degree of reliability in the decision making process [21].

Notwithstanding the fact that many courts require that a plaintiff present particularistic evidence in toxic tort cases, this requirement is not universal [22]. Further, much of the specific evidence that a plaintiff uses is derived from statistic evidence; that is, expert testimony concerning the cause of the plaintiff's injury on which it is based.

Despite the fact that statistical evidence alone is probably not sufficient, in toxic tort litigation it should not be minimized. In toxic tort cases, epidemiological studies – which are statistical by their very nature – are routinely accepted, and may be the only basis for an expert's testimony concerning causation.

C Quality of evidence

Frequently, a plaintiff in a toxic tort case will allege cause-in-fact of his injury, illness, or other infirmity, by an exposure to some chemical where the published literature offers little support for the causal relationship. Of course, the defense will counter that the plaintiff's proffered expert testimony is based on speculation and not based upon a principle that is accepted in the community. The defense will request that the proffered testimony therefore be excluded.

In order to minimize speculative evidence, the courts frequently require that expert medical testimony be "within a reasonable degree of medical certainty" in order for it to be admissible [23]. The plaintiff's expert (or, indeed, the defendant's expert) is thus only permitted to testify as to a medical conclusion if the principle upon which it is based is a generally accepted medical principle. This was well articulated in the case of *Frye* v. *United States* [24], where the court excluded expert testimony, holding that:

"Just when a scientific principle crosses the line between the experimental and demonstrable

stages is difficult to define. Somewhere in this twilight zone the evidential force of the principle must be recognized, and while courts will go a long way in admitting expert testimony deduced from a well recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the field to which it belongs." [25]

This principle was enunciated in the *Frye* case in 1923; it has been broadly adopted and is still followed. In the context of a personal injury suit, the principle was articulated in *Puhl* v. *Milwaukee Automobile Insurance Company* [26], as follows:

"There is no testimony that the generally recognized medical authorities on Mongolism have agreed what causes this deformity. When scientific or medical theories or explanations have not crossed the line and become an accepted medical fact, opinions based thereon are no stronger or convincing than the theories. While the Court has gone a long way in admitting expert testimony deduced from well-recognized scientific and medical principles or discoveries, nevertheless the facts from which the opinion is made must be sufficiently established to have gained general acceptance in the particular medical field in which they belong." [27]

Puhl also enunciates the principle that the data relied upon must be "of a type reasonably relied upon by experts in the particular field," as stated in the Federal Rules of Evidence [28].

D A substantial factor

Toxic tort litigation is somewhat more difficult than other forms of tort litigation, inasmuch as there is an insufficiency of sound scientific data upon which viable conclusions can be made, and there are almost invariably a myriad of possible causes for the plaintiff's injury or illness. For example, a recent study by epidemiologists at Oxford University concluded that the causes of cancer are extremely varied, and included the following [29]:

Geography	3%
Environmental pollution	2%
Infection	10%
Medical treatment	1%
Sexual factors	7%
Occupation	4%
Industrial products	1%
Food additives	1%
Alcohol consumption	3%
Tobacco	30%
Diet	10% to 70%

It is the plaintiff's burden to prove that the act of the defendant was a "substantial factor" in producing the injury, or that "but for" the plaintiff's act, the defendant would not have suffered his injury.

The term "substantial" can be rather difficult to define in this context.

Indeed, in *Multiple Causation in Tort Law: Reflections on the DES Cases* [30], Robinson noted that:

"When confronted with the question of definition, the typical judicial response is that the term 'substantial' cannot be further explained, but is simply a matter for the jury's common sense." [31]

V. Challenging proof of cause in fact

A Introduction

Successful challenge of the plaintiff's proof of cause-in-fact establishes a complete defense. All possible methods for such a challenge must be considered by the defense attorney in light of the evidence determined during the discovery phase of litigation, seeking out the weakest link or, preferably, links. There are many possible methods for challenging the plaintiff's proof, the most important of which will be discussed herein.

B Challenges to medical evidence

1 Epidemiologic studies

Epidemiologic studies are frequently subject to attack as being statistically insignificant. Different researchers may make different value judgments concerning the viability of underlying data or the methodology used to obtain and analyze such data. Subjective analysis and conclusions are suspect; a strong mathematical association is necessary. In one case, the court excluded statistical evidence which fell below the apparent threshold, 95%, confidence level [32].

Related to statistical insignificance is the potential failure to control or account for all of the relevant variables. As stated by Dore in his paper *A Commentary on the Use of the Epidemiological Evidence in Demonstrating Cause-In-Fact* [33]:

"A study should account for all relevant risk factors, standardize for age, sex, and race, and involve significant exposures to the alleged causative factor. The court should exclude any study that does not sufficiently isolate the alleged causative factor as the risk factor." [34]

There may be inconsistent biological or toxicological evidence. As previously discussed, a mere statistical inference is weak; when there is inconsistent scientific evidence, then the epidemiological evidence becomes rather insignificant. The postulates discussed above can be used to determine whether statistical data is actually demonstrative of a causal relationship.

A lack of comparability between the chemical composition of the substance which was the subject of the epidemiological study and the substance to which the plaintiff was allegedly exposed can readily occur. One possibility is where

the chemical composition of some substance has changed over a period of time. For example, the chemical composition of PCBs manufactured in the United States has changed to a significant degree recently, with toxicity being altered. This would largely invalidate the use of earlier epidemiological studies.

In a monograph series on carcinogens, the International Agency for Research of Cancer stated that a study which shows a positive association between an agent and a cancer "may be interpreted as implying causality, to a greater or lesser extent," if the following five criteria are met:

1. There is no identifiable positive bias, where positive bias refers to factors in the design or the execution of the study which would erroneously lead to a more strongly positive association between the agent and the disease than is actually the case;
2. The possibility of positive compounding has been considered where compounding refers to the extraneous influence of factors, other than the one that is being measured in the association with the risk. Positive compounding occurs when the association between the agent and the disease appears to be more strongly positive than is actually the case as a result of any association between the subject agent and another agent which either causes or prevents the disease;
3. The association is unlikely to be a result of chance alone;
4. The association is strong; that is, statistically significant; and
5. There is a defined dose–response relationship [35].

Similarly, the National Academy of Science has defined seven factors for demonstrating that once a statistically significant relationship exists, there is a nexus between the statistical results and the plaintiff's facts, as follows:

1. Strength of statistical relationship;
2. Dose–response relationship;
3. Confirmation of exposure;
4. Confirmation of relationship between exposure and the disease;
5. Temporal sequence;
6. Specificity of response; and
7. Consistency with laboratory data [36].

When the epidemiological data is suspect, it can be challenged. The Federal Rules of Evidence permit experts to base their opinions on facts or data compiled by third parties if the facts and data are of type reasonably relied upon by other experts in the same field [37]. (Note that the facts and data need not be independently admissible.)

Many courts have taken a rather liberal approach to this rule. The courts should not, however, hesitate to preclude expert testimony based on unreliable data, notwithstanding the Federal Rules of Evidence. The relevant Rule, No. 703, must be considered a hurdle, but not conclusive. Experts in a particular field may form opinions for differing reasons and with differing impacts. Sim-

ply because data is suitable for one purpose does not mean that it is necessarily suitable for all purposes.

For example, a company may choose not to market a particular product when the danger is only indicated by a low reliability data base, inasmuch as a risk–benefit analysis may indicate that it would not be beneficial to market the product. That same low reliability data base should not be used by a plaintiff to demonstrate a causative relationship.

In the case of *Standard Oil Company of California* v. *Moore* [38], the Ninth Circuit court of appeal held that:

"It is for the trial court to determine, in the exercise of its discretion, whether the expert's sources of information are sufficiently reliable to warrant reception of the opinion." [39]

The case of *Lima* v. *United States* [40] demonstrates the application of the requirement of reasonable reliability in toxic tort litigation. *Lima* was one of the numerous actions that arose out of the swine flu inoculations which allegedly caused Guillan–Barre Syndrome (hereinafter referred to as "GBS"). The cause of GBS is unknown. Both parties were therefore forced to rely heavily on the use of epidemiological evidence, particularly a study undertaken by The Center for Disease Control. That study reached the conclusion that there existed a greater risk of contracting GBS during the first weeks following inoculation, but that there was no relation between the swine flu vaccine and the onset of GBS more than ten weeks after vaccination.

The plaintiff in *Lima* did not develop GBS until four or five months after the inoculation. The plaintiff conducted a survey of GBS cases which occurred in the same month that he was diagnosed as having GBS. He also obtained statistical data concerning the incidence of GBS from the Colorado Department of Health. Based on that data, the plaintiff performed a statistical analysis and concluded that there had been an increase above the norm in the number of GBS cases during the subject month.

The plaintiff's expert testified on the basis of that data, stating that the data was of a type reasonably relied upon by an expert in the field. The defense expert criticized the study; the trial court granted a defense motion to strike the testimony of plaintiff's expert, holding that the opinion did not meet the threshold requirements of Rule No. 703 because the data relied upon were inadmissible hearsay and not of the type reasonably relied upon by experts in epidemiology and neurology. The ruling was affirmed on appeal.

Other types of epidemiological data that may be challenged are the retrospective governmental investigations and studies which may be introduced as evidence under Federal Rules of Evidence, Rule No. 803, which creates, in pertinent part, an exception to the hearsay rule of exclusion for public reports, data compilations, and similar materials.

This type of data has recently been applied in Toxic Shock Syndrome (here-

inafter referred to as "TSS") litigation. The basis for that litigation was several governmental studies conducted by The Center for Disease Control and the state public health departments in several states. The methodology of these studies was to place TSS victims who had the onset of their illness during menstruation in the case group, and then selecting a control group. Center for Disease Control Study No. 1 revealed that all of the cases used tampons, as compared to 86% of the controls. This indicated an association which would not be explained by random chance; however, the study did not prove that tampons caused TSS, nor did the data explain the role that tampons play in TSS.

In *Kehm* v. *Proctor and Gamble* [41], the district court admitted the reports and permitted a former member of The Center for Disease Control task force to testify regarding the studies. The defendant objected, but the court rejected the defendant's argument that the studies were not factual findings. The court noted that government reports setting forth opinions and conclusions are frequently admitted because they are based upon investigations conducted pursuant to lawful authority and are therefore presumptively reliable, with the burden being on the opposing party to prove that such reports are untrustworthy. The court also concluded that the studies employed procedures and methods that were widely accepted in the field of epidemiology, and that the timeliness of the investigations, coupled with the agency's skill and motive to fairly and adequately inform the public, rendered the evidence properly admissible.

2 Toxicological studies

There is a tremendous body of information concerning the failings of animal models in toxicological studies to prove human susceptibility, reaction, or risk. Only the primary deficiencies in this type of study will be discussed herein.

First, humans and animals are separate species; the dissimilarities are so profound that any extrapolation from an animal model is of low reliability. As noted by Sackett in *The Diagnosis of Causation* [42]:

"Both the rates and routes of drug metabolism differ among the various animals, and it is therefore clear that non-human studies may both falsely accuse and acquite drugs of producing adverse affects in men. Accordingly, although hypothesis-forming expeditions may provide an adequate base for making the clinical decision to withdraw a drug from use, they are inadequate as the sole base for making the causal decision that a drug is truly responsible for a given adverse effect." [43]

Similarly, in *The Rat as a Model for Human Toxicological Evaluation* [44], Oser stated that:

"[T]he critical investigator cannot ignore the degree to which qualitative and quantitative differences affect inter-species extrapolation. The choice of a suitable species for toxicological study

is generally decided on the basis either of the sensitivity to the toxicant in question or the similarity of the metabolism to that in humans. However, lip service is often paid to these criteria and the rat is selected merely for pragmatic reasons." [45]

Indeed, as stated by Higginson in *Chronic Toxicology: an Epidemiologists Approach to the Problem of Carcinogencity* [46]:

"[T]here is no rational method available for calculating a non-effect dose in man from animal data, nor can a negative result in an animal be necessarily accepted as guaranteeing safety." [47]

The fact that viral causes of cancer have been isolated in animals, but that no such causative agent has been isolated in humans, is further indication of the failings of animal testing [48].

Further, the variation in actual exposure levels between test animals and humans is profound. Extrapolation from the effects of high doses of some of the substances tested to the possible effect at low doses is routinely done, despite the fact that no data exist for the lower levels, and construction of dose–response curves for the low levels are prepared [49]. These efforts are significantly hampered because it is not known, in the case of carcinogens, to what extent deoxyribonucleic acid (DNA) repairs itself at low levels of exposure [50].

Other challenges to toxicological studies are analogous to those for epidemiologic studies, including questioning any inconsistencies, challenging the statistical significance of data, and challenging the lack of comparability of the chemical composition of the substance tested and the substance to which the plaintiff was allegedly exposed.

3 Credentials of the plaintiff's experts

While it is customary to at least consider challenging the credentials of opposing experts, this challenge is especially valuable in toxic tort litigation. The medical sciences involved are complex; no one person is a specialist in all of them. Examination may reveal highly useful admission that an expert does not claim expertise in any specialty other than his own. This significantly narrows the areas in which he is able to give a credible opinion and may affect the expert's overall credibility with the jury, particularly if he attempts to stray out of his admittedly limited area of expertise.

4 Other considerations

A delineation of considerations that must be made to medically indicate the requisite causal relationship between a suspected factor and a disease was discussed in Ballanfant, *Legal Proof of Causation* [51], and Evans, *Causation and Disease: the Henle-Koch Postulates Revisited* [52]. The considerations, some of which have already been discussed, are as follows:

a. Prevalence of the disease should be significantly higher in the group exposed to the suspected factor than in the control group;

b. Exposure to the suspected factor should be more common in those with the disease than in the control group without the disease;
c. Incidence of the disease should be significantly higher (in a statistical sense) in those exposed to the suspected factor than those not so exposed, as demonstrated in prospective studies;
d. Temporally, the disease must follow exposure to the suspected factor such that the distribution of the incubation period simulates a bell-shaped curve;
e. A spectrum of host responses should follow exposure to the suspected factor along a logical biologic gradient from mild to severe;
f. A measurable host response following exposure to the suspected factor should regularly appear in those lacking this in the past, or should increase in magnitude if present before exposure, which pattern should not occur in persons not so exposed;
g. Experimental reproduction of the disease should occur in higher incidence in animals or humans appropriately exposed;
h. Removal of the instrumentality carrying the suspected factor should decrease the incidence of the disease;
i. Prevention or modification of the host's response on exposure to the suspected factor should decrease or eliminate the disease; and
j. The considerations must make scientific sense according to the known principles of biology and epidemiology [53].

Each of the aforesaid considerations must be considered during the careful scrutiny of the plaintiff's evidence to establish every possible point of attack.

C Challenging the nexus between plaintiff and product

There are two aspects to challenging the nexus between the plaintiff and the product of the defendant. The first is to challenge actual exposure. It may be that evidence such as employment records or results of medical tests (particularly blood and adipose tissue sampling) indicates little or no exposure to or intake of the substance that the plaintiff alleges to be the cause of his injury or disease.

The second aspect is to challenge whether the substance that the plaintiff alleges to have caused his injury or disease was one which was manufactured, distributed, sold, or owned by the defendant. In some cases, the inability on the part of the plaintiff to prove the source of a substance was an adequate defense; in many cases it is not, with the burden shifting to the defendant to prove that it was not the responsible party.

D Challenging the temporal relationship

In the event of acute exposure to a toxic substance, the injury or disease occurs quickly; the temporal relationship is therefore easy to establish. However, in the vast preponderance of toxic tort litigation, the exposure is subacute or chronic, with manifestations of disease occurring only years later.

Frequently, epidemiological studies exist which indicate the range and distribution of the latency period for the substance and disease which are the subject of the litigation. Any time that the actual latency period is not within the expected range, suspicion may be had that the injury or disease was actually caused by other factors.

When considering the temporal relationship, care must be taken to avoid the classic non sequitur best expressed in its original Latin as *post hoc, ergo propter hoc.* The phrase translates to "after this, therefore because of this." While it is easy to surmise that because an event followed another event there was a causative relationship between the two, especially if one suspects that there is such a relationship and is highly motivated by a potential financial recovery in the event that such a relationship exists, mere wanting does not make it so; this is rather fortunate for the party defending a toxic tort lawsuit.

E Challenging the degree and route of exposure

There is a minimum threshold level for most substances (below which exposure is harmless) known as the virtually safe dosage. It is a point on the curve of the dose–response relationship. If the degree of exposure is below that of the known virtually safe dosage, either as determined in the literature or as admitted by the plaintiff's experts, then causation is questionable.

Similarly, there are various means for a toxic substance to enter a human, including inhalation, ingestion, and absorption. The virtually safe dosage of certain toxic substances is a function of the means of entry into the body. Thus, evidence of the method of entry affects the virtually safe dosage, which in turn affects the viability of the alleged causation.

F Challenging the diagnosis

Many diseases have specific symptoms which serve to differentiate that disease from others. It is therefore necessary to obtain as much information as possible about the plaintiff's condition so that the diagnosis of the plaintiff's expert may be analyzed for speculation on the part of the expert. Although a highly specific diagnosis which incorporates the known symptoms of a particular toxic exposure may be difficult to challenge, frequently the diagnosis is less specific and based upon general symptomatology.

G Alternate causes

For many diseases, there are many alternate causes, such as those discussed above with reference to cancer. Plaintiffs have historically prevailed despite the existence of multiple causes; however, damages are often mitigated [54]. It is necessary for the plaintiff who has been exposed to multiple causative agents to prove that the defendant's act was a substantial factor in producing the injury. In the case of an intervening cause, which will be discussed with respect to proximate cause, a complete defense may be established.

VI. The proximate cause aspect of the case

As discussed earlier, proximate causation is largely a policy decision, to determine whether the defendant should be held legally responsible for whatever it is that the defendant did.

There are three primary tests that the courts use to establish proximate cause. The particular test that will be used depends on the jurisdiction, the circumstances of the case, and the court's particular preference. These tests are as follows:

1. Was the type of injury suffered by the plaintiff a reasonably foreseeable result of the defendant's tortious activity?
2. Was the plaintiff's injury a direct result of the defendant's tortious activity?
3. Was the defendant's tortious activity a substantial factor in producing the plaintiff's injury [55]?

In many toxic tort cases, the proximate cause issue becomes moot from the standpoint of the defense because injuries caused by toxic substances are frequently foreseeable.

There are, however, several defenses, possibly the most important of these being intervening causation, which may negate liability. For example, if a doctor errs in prescribing medication for a plaintiff, the doctor's tortious conduct may be held to supersede that of the original defendant [56]. According to the *Restatement of Torts (Second)* [57],

"A superseding cause is an act of a third person or other force which by its intervention prevents the actor from being liable for harm to another which his antecedent negligence is a substantial factor in bringing about."

The *Restatement* also delineates the factors to be considered in determining whether an intervening force is a superseding force, relieving the original tortfeasor of his liability, as follows:

"The following considerations are of importance in determining whether an intervening force is a superseding cause of harm to another:

"(a) the fact that its intervention brings about harm different from that which would otherwise have resulted from the actors' negligence;

(b) the fact that its operation or that the consequences thereof appear after the event to be extraordinary rather than normal in view of the circumstances existing at the time of its operation;

(c) the fact that the intervening force is operating independently of any situation created by the actor's negligence, or, on the other hand, is or is not a normal result of such a situation;

(d) the fact that the operation of the intervening force is due to a third person's act or to his failure to act;

(e) the fact that the intervening force is due to an act of a third person which is wrongful toward the other and as such subjects the third person to liability to him;

(f) the degree of culpability of a wrongful act of a third person which sets the intervening force in motion." [58]

This analysis was done in *Little* v. *PPG Industries* [59] where the court considered a wrongful death action of a worker in a steel plant who was using a solvent manufactured by defendant PPG Industries, Inc. The court of appeal reversed the lower court decision and held that:

"Where the buyer is notified of the danger, or discovers it for himself, and delivers the product without a warning, it usually has been held that the responsibility has shifted to him, and his negligence supersedes the liability of the seller." [60]

Conclusion

Toxic tort litigation can be one of the most difficult types of litigation to defend, both because of the technical nature of the defense and because public policy has continued to maximize the ease with which a plaintiff may prove his case, despite the ostensible burden of proving each element of his case by a preponderance of the evidence.

Despite the resulting challenge, the defense of toxic tort litigation is certainly feasible. The most effective area of attack is in the causation element of the plaintiff's proof, particularly the cause-in-fact aspect.

It is of some comfort to defendants and potential defendants in toxic tort litigation that, as the scientific base of data upon which causation is proved (particularly *in vitro* testing, which permits the low dosage and high volume testing requisite to accurately ascertain the true propensity for a particular substance to have an adverse reaction on human tissue), defense will become easier, as the plaintiff is precluded from using the vast extrapolations now routinely presented to the courts.

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