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PROVING CAUSATION IN TOXIC TORT CASES:
T-CELL STUDIES AS EPIDEMIOLOGICAL AND
PARTICULARISTIC EVIDENCE

Elizabeth A. Stundtner*

I. INTRODUCTION

The element of causation is difficult to establish in toxic tort cases because the concept of legal causation is much different than scientific causation.¹ To find legal causation, a defendant must have breached a duty to the plaintiff that resulted in an injury to the plaintiff,² while medical causation is the probability that the suspected source did cause the plaintiff’s injury.³ Because toxic torts can best be understood with the use of probabilistic evidence, at present, courts generally rely upon epidemiological evidence as well as particularistic evidence to establish causation in toxic tort cases.⁴

Epidemiological evidence represents one of the most promising scientific methods in toxic tort causation analysis.⁵ An epidemiological study assesses whether a relationship exists between a single factor in a particular environment and the presence of a particular disease within that environment’s population.⁶ There are limitations,
however, to using epidemiological evidence to prove causation. The most difficult problem is that an epidemiological study can never prove causation with regard to a single individual; it can only provide an estimate of risk for a given population.

Particularistic evidence is another method litigants use in presenting evidence regarding causation. In contrast to probabilistic evidence, particularistic evidence deals only with that specific plaintiff. A doctor can provide particularistic evidence by diagnosing a specific injury in a plaintiff and subsequently testifying as an expert that the defendant’s conduct caused the plaintiff’s injury. Particularistic evidence, however, also poses problems in proving legal causation. For example, because the etiology of many diseases is unknown, courts carefully scrutinize the scientific foundation of the conclusions of the treating physician/expert witness.

Although there are a number of scientific means available to develop both epidemiological and particularistic evidence in toxic tort cases, one of the most promising means is the T-cell study. T-cells are one of the components of the immune system which protect the body from foreign matter. An epidemiological T-cell study can show a strong correlation between a disease and its cause within a specific population. In addition, by analyzing T-cells for particularistic evidence, physicians can evaluate the status and strength of an individual’s immune system: an assessment that determines whether a toxic substance has damaged it.

Although epidemiological evidence and particularistic evidence alone have limitations in proving causation, this Comment argues that the use of T-cell studies in toxic tort cases can produce both

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9 Callahan, supra note 1, at 619.
10 See id.
11 Id.
12 Id. at 630-40.
13 DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 587 (27th ed. 1988). Etiology is defined as "[t]he study or theory of the factors that cause disease and the method of their introduction to the host; the cause(s) or origin of a disease or disorder." Id.
14 Callahan, supra note 1, at 630-31.
15 See Alan Levin & Vera Byers, Environmental Illness: A Disorder of Immune Regulation, 2 OCCUPATIONAL MED. 669, 676 (1987).
18 Levin & Byers, supra note 15, at 671-72.
epidemiological and particularistic evidence, and thereby overcome the limitations of only one kind of evidence. More specifically, T-cell studies are a valuable method of assessing correlations between the incidence of an injury or disease and the presence of a toxic substance within a given population. Furthermore, an individual T-cell study can provide specific evidence concerning the injury and its cause for an individual plaintiff. Section II of this Comment considers the difference between scientific and legal causation and the role of epidemiological and particularistic evidence in meeting the demands of legal causation. Section III reviews T-cells' function in the immune system, how "clinical ecology" has developed T-cell studies, and how the legal system has used them. Section IV examines how courts use epidemiological evidence to prove causation in toxic tort cases. In addition, this section discusses what epidemiology is and the value and limits of epidemiological evidence. In Section V, this Comment examines how courts use particularistic evidence to prove causation in toxic tort cases. This section also discusses what particularistic evidence is, as well as the value and limits of particularistic evidence. Section VI examines how courts use both particularistic evidence and epidemiological evidence to determine causation. Finally, Section VII reviews the value of T-cell studies in present and future toxic tort litigation.

Because particularistic evidence and epidemiological evidence each present limitations in proving or disproving causation, both types of evidence are important to determine legal causation. This Comment concludes that T-cell studies are an innovative scientific method that litigants should consider and develop as a new means of providing a basis for both kinds of evidence in toxic tort cases.

II. LEGAL CAUSATION IN TOXIC TORTS AND THE USE OF SCIENTIFIC EVIDENCE TO PROVE CAUSATION

Legal causation is usually difficult to establish in toxic tort litigation. To prove legal causation, a plaintiff must prove that the

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19 See infra notes 27-62 and accompanying text.
20 See infra notes 63-109 and accompanying text.
21 See infra notes 110-11, 146-66 and accompanying text.
22 See infra notes 112-45 and accompanying text.
23 See infra notes 167-69, 186-223 and accompanying text.
24 See infra notes 170-85 and accompanying text.
25 See infra notes 224-308 and accompanying text.
26 See infra notes 309-74 and accompanying text.
defendant breached a duty to the plaintiff and that this breach led directly to the plaintiff’s injury. 28 In toxic tort litigation, the plaintiff must prove that the toxic substance was both the cause-in-fact and the proximate cause of the plaintiff’s disease. 29 A plaintiff establishes cause-in-fact by proving that without the defendant’s act, the injury would not have occurred. 30 To establish proximate cause, which is a policy judgment, the plaintiff must show that the defendant’s breach of duty was so closely connected to the plaintiff’s injury that a court should invoke liability. 31 Although courts generally do not rely on this concept, another legal term of causation is “causal tendency.” 32 Causal tendency is a finding that the defendant’s breach of duty increased the risk that the plaintiff would incur an injury. 33

Courts require the plaintiff to establish by a preponderance of the evidence that the toxic substance was the cause of his or her injury. 34 This means that the plaintiff must prove that the defendant more likely than not caused the plaintiff’s injury. 35 Traditionally, courts have derived a decision on whether causation exists from particular facts and evidence through the process of deduction. 36

Plaintiffs in toxic tort cases, however, face a difficult task in attempting to prove that a particular toxic substance caused their particular injury. 37 The etiology of many diseases is unknown and hazardous substance illnesses often lack a physical trauma that marks their onset. 38 Factors other than the toxic substance may be the actual cause of the disease. 39 This is the indeterminancy problem. 40 There are two facets to the indeterminancy problem, one involving the defendant and one involving the plaintiff. 41 First, a

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28 See W. KEETON ET AL., supra note 2, at 263.
29 See id. at 263–64.
30 Id. at 265.
31 Id. at 264; see Firak, supra note 8, at 311.
33 Id. at 405.
34 W. KEETON ET AL., supra note 2, at 263.
35 Id.
37 Callahan, supra note 1, 616–17.
38 Id. at 617.
40 Callahan, supra note 1, at 612–18; Firak, supra note 8, at 316.
41 Callahan, supra note 1, at 612, 616.
plaintiff may be unable to determine who the defendant is.\textsuperscript{42} If the plaintiff was exposed to more than one toxin, he or she must establish which toxin caused his or her illness.\textsuperscript{43} Second, the plaintiff must show that exposure to the toxin was the direct cause of the injury.\textsuperscript{44} Accordingly, the traditional legal standard of causation, which requires direct proof linking the defendant to the plaintiff, can rarely be met in toxic tort cases.\textsuperscript{45}

The legal standard of causation, however, is much different than the standard of scientific causation.\textsuperscript{46} Scientific evidence merely demonstrates a probability that a given chemical caused a particular individual's disease.\textsuperscript{47} Using inductive reasoning, scientists attempt to determine causation through the process of hypothesis building.\textsuperscript{48} Scientists then test the generated hypothesis repeatedly to establish a statistic or probability.\textsuperscript{49} Epidemiological studies, for example, can establish strong correlations between exposure to a chemical and the occurrence of disease within a certain population.\textsuperscript{50}

Because it is difficult for toxic tort plaintiffs to meet the legal causation standard with scientific evidence, some courts have relaxed the strict legal standard of causation.\textsuperscript{51} Some courts have more liberally construed the Federal Rules of Evidence to allow statistical evidence and novel medical evidence.\textsuperscript{52} The courts, however, have

\begin{footnotes}
\item[43] Callahan, \textit{supra} note 1, at 612.
\item[44] \textit{Id.} at 616–17.
\item[45] See generally, \textit{Developments, supra} note 42, at 1624–25.
\item[46] Brennan, \textit{supra} note 1, 478–83. Troyen Brennan explains that the history of legal causation developed from corpuscularian science, which defined causation as impacts that follow from the physical laws of mathematics and Newton’s concepts of physics. \textit{Id.} at 478. Scientific causation, however, derives from the development of quantum mechanics and the theory of relativity. \textit{Id.}
\item[47] See Brennan, \textit{supra} note 1, at 475. Because the etiology of cancer is still unknown to a great extent, one cannot prove that a specific carcinogen caused a specific individual’s cancer. \textit{Id.}
\item[48] \textit{Id.} at 481.
\item[49] \textit{Id.} at 482–83.
\end{footnotes}
not agreed upon a unified interpretation of rules such as FRE 403,\textsuperscript{53} 702,\textsuperscript{54} or 703\textsuperscript{55} concerning the admission of scientific evidence.\textsuperscript{56}

Another method by which courts have lessened plaintiffs' burden of proving legal causation is the application of the substantial factor test.\textsuperscript{57} With this test, a court would find a defendant liable if he or she contributed to a great extent to the harmful result, even if the harm may have occurred without the defendant's actions.\textsuperscript{58} Thus, the court would find liability even if the evidence only showed that the defendant significantly increased the risk that the plaintiff would suffer an injury.\textsuperscript{59}

Finally, some courts have allowed the use of a "weak" version of the preponderance rule.\textsuperscript{60} This standard allows plaintiffs to prove through statistical proof that the defendant more likely than not caused the plaintiff's injury.\textsuperscript{61} Because these new causation standards are so novel, however, courts have not utilized them on a wide basis. Thus, courts still expect plaintiffs to present causation evidence that meets the conventional standard of legal causation.\textsuperscript{62}

\textsuperscript{53} FED. R. EVID. 403 states that, "although relevant, evidence may be excluded if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence."

\textsuperscript{54} FED. R. EVID. 702 states that, "if scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise."

\textsuperscript{55} FED. R. EVID. 703 states that, "the facts or data in the particular case upon which an expert bases an opinion or inference may be those perceived by or made known to the expert at or before the hearing. If of a type reasonably relied upon by experts in the particular field in forming opinions or inferences upon the subject, the facts or data need not be admissible in evidence."


\textsuperscript{58} Id. at 173–74. The court determined that the substantial factor test "is particularly suited to injury from chronic exposure to toxic chemicals where the sequent manifestation of biological disease may be the result of a confluence of cases." Id. at 174.

\textsuperscript{59} Id. at 174.

\textsuperscript{60} Callahan, supra note 1, at 611.

\textsuperscript{61} Id.; Developments, supra note 42, at 1619.

\textsuperscript{62} See Callahan, supra note 1, at 611; Parker v. Employers Mutual Liab. Ins. Co., 440 S.W.2d 43, 47 (Tex. 1969) (court required that plaintiff prove his cancer was "more likely than not" caused by his exposure to radiation), construed in Black & Lilienfeld, supra, note 6, at 749.
III. THE FUNCTION OF T-CELLS AND THE USE OF T-CELL STUDIES

A. T-Cells and Their Function

The T-cell study is one epidemiological study or patient exam that plaintiffs or defendants can use to prove or disprove legal causation in toxic tort cases. T-cells are an important component of the immune system. When an "antigen" enters the body, the immune system has one of two immune responses. A "humoral immunity" response mobilizes specific antibodies, which B-cells produce; a "cellular immunity" response mobilizes certain types of white cells, the T-lymphocyte series, T-cells. On the outside of the T-cell is a protein called the "T-cell receptor." Each T-cell has a specific T-cell receptor that binds to a specific type of antigen, similar to a key that fits into a lock. Once the receptor has attached itself to an antigen (which may be within a cell), the T-cell can reproduce to destroy the antigen.

There are four types of T-cells: "cyto-toxic" T-cells, "helper" T-cells, "suppressor" T-cells, and "memory" T-cells. Cyto-toxic T-cells fight against viral infections and other antigens, such as cancerous tumors. Cyto-toxic T-cells are activated when an antigen-bearing cell is covered with major histocompatibility complex (MHC) molecules and antigen peptides lock with the T-cell's receptor. Once the T-cell recognizes the antigen-bearing cell as unhealthy and is then mobilized into a mature T-cell, the cyto-toxic T-cell will reproduce and destroy the unhealthy cell by secreting enzymes.

63 See Marrack & Kappler, supra note 16, at 36.
64 AMERICAN HERITAGE DICTIONARY 115 (2d ed. 1985). The definition of antigen is "a substance that when introduced into the body stimulates the production of an antibody." Id.
66 THE AMERICAN HERITAGE DICTIONARY 114 (2d ed. 1985). The definition of antibody is "any of various proteins in the blood that are generated in reaction to foreign proteins or polysaccharides, neutralize them, and thus produce immunity against certain microorganisms or their toxins." Id.
67 Id.
68 Marrack & Kappler, supra note 16, at 36.
70 von Boehmer & Kisielow, supra note 69, at 75-77.
71 Id. at 74.
72 Id. at 75. MHC molecules carry antigen peptides from within the cell to the surface of the cell. Id.
73 Id. at 74-75.
74 Id.
Helper T-cells assist B-cells (humoral immunity) in producing antibodies to fight toxic bacteria, a type of antigen. Mobilized helper T-cells produce interleukins, which incite B-cells to multiply and send more antibodies into the bloodstream to bind to a toxin and deactivate it. Suppressor T-cells inhibit an immune response by depressing the reaction of the helper T-cell and B-cell immune response.

Finally, memory T-cells, as well as some B-cells, "remember" a first antigen attack. A subsequent attack by an antigen activates the memory cells more quickly which results in a more significant antibody production and a stronger binding action by the T-cells.

B. The Scientific Use of T-Cell Studies

Scientists use studies of T-cells, otherwise known as immune profiles, to show abnormalities in the immune system. T-cell studies arose in the context of the recently developed medical discipline of clinical ecology. Clinical ecology focuses on human chemical hypersensitivity syndrome, also known as multiple chemical sensitivities (MCS). MCS is a disorder involving chronically repeating symptoms that relate to many of the various organ systems and are a response to low-dose chemical exposure. Clinical ecology, however, has yet to understand clearly the etiology and resulting symptoms of MCS. One theory of the immunopathogenesis of MCS is that

75 Id. at 76.
76 Id.
77 Brian McClain, Meet the T-Cell Antigen Receptor, 52 THE AM. BIOLOGY TEACHER 276, 276 (May 1990).
78 SELL, supra note 65, at 217–18.
79 Id. at 218.
80 See Levin & Byers, supra note 15, at 676.
82 Troyen Brennan, Helping Courts with Toxic Torts: Some Proposals Regarding Alternative Methods for Presenting and Assessing Scientific Evidence in Common Law Courts, 51 U. PITTS. L. REV. 1, 59 (1989); Terr, supra note 81, at 684. Multiple Chemical Sensitivities has many synonyms, including environmental illness, chemically induced immune dysregulation, and twentieth century disease. Id. at 684.
84 See Terr, supra note 81, at 690. Once clinical ecologists better understand the etiology and symptoms of MCS, they can attempt to more clearly define, diagnose, and treat MCS. See id. at 684–88.
85 DORLAND’S ILLUSTRATED MEDICAL DICTIONARY 823 (27th ed. 1988). Immunopathogenesis is defined as, "a process in which the course of a disease is altered or affected by an immune response (either the cellular[T-cell] or humoral [B-cell] response) or by the products
toxins cause damage to the T-cells, which results in a damaged immune system. Clinical ecologists therefore perform T-cell studies to determine the presence of such toxin damage.

There are several types of T-cell studies. A T-cell study can calculate the absolute levels of each T-cell population or also determine the balance between the different populations in the form of ratios. Another type of T-cell study can measure the speed of the memory T-cells by exposing a sample of these cells to a small bit of an antigen in order to determine whether they were previously exposed to that particular type of antigen. Yet another type of T-cell measurement is the "mitogen challenge." A researcher withdraws a T-cell sample from a patient's blood and measures its energy per minute. The researcher then exposes the T-cells to an antigen to determine whether the lymphocytes' energy level increases. If the T-cells fail to react quickly, this is an indication that the T-cells have been suppressed or damaged.

People with a healthy immune system have absolute T-cell numbers, reaction rates, and ratios that fall within a normal range. If a person is exposed to a chemical for an extended period of time, however, his or her T-cell numbers and ratios become skewed, indicating immune dysregulation. If an entire population of people suffer from T-cell abnormalities, an epidemiologist would examine the population to find a common factor that would indicate the cause of these abnormalities. For example, epidemiological studies show a significant increase in suppressor cells and/or a decrease in the

of an immune reaction, such as the antigen-antibody-complement complexes deposited in renal glomeruli." Id.

Id. at 691.

See generally id.

See Alan Levin & Vera Byers, Multiple Chemical Sensitivities: A Practicing Clinician's Point of View: Clinical and Immunologic Research Findings, 8 (1991) (unpublished article on file with the authors).

Telephone Interview with Dr. Alan S. Levin, clinical ecologist and immunologist (Nov. 13, 1991); see generally SELL, supra note 65, at 218 (in depth and technical discussion of process).


Id.

Id.

Id.

Dr. Levin's Supplementary Affidavit, cited in, Jan Schlichtmann, Eight Families Sue W.R. Grace and Beatrice Foods for Poisoning City Wells with Solvents and Causing Leukemia, Disease, and Death, 341 Practising Law Inst. 1, 44 (1987).

Id.

Levin & Byers, supra note 15, at 673–74.
helper/suppressor cell ratio in cohort populations that have been exposed to formaldehyde. Thus, T-cell studies could show a causal link between exposure to a chemical and the presence of an illness that was in the past more difficult to prove.

C. The Use of T-Cell Studies in Toxic Tort Litigation

Litigants have used T-cell studies to show causation for cancer, chemically induced immune dysregulation, emotional distress, and increased risk of disease. In litigation, a plaintiff using a T-cell study as evidence essentially argues that exposure to a particular chemical caused the injury or serious abnormality in the immune system. Because T-cell studies were recently developed, litigants have not used them to prove causation in toxic torts on a widespread basis. A number of scientists and doctors do not yet accept clinical ecology, the basis of T-cell studies, as a scientific discipline. Furthermore, judges and juries have difficulty understanding the intricacies of T-cell studies and what the results of T-cell studies actually mean.

Despite the difficulties in understanding the biology of T-cells, T-cell studies are basically a means to measure the health of the immune system. A researcher can test the T-cells of an individual

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98 Levin & Byers, supra note 88, at 8.
99 See generally Levin & Byers, supra note 15, 673–74.
102 Anderson, 628 F. Supp. at 1226.
103 Id. at 1231.
105 Brennan, supra note 82, at 61 n.268. The American Academy of Allergists does not accept the scientific basis of clinical ecology. Id.
106 See Sterling, 855 F.2d at 1188 (court rejected T-cell study because it was not traditional clinical test); Compare Telephone Interview with Dr. Alan S. Levin, clinical ecologist and immunologist (October 15, 1991), discussing, Tiderman v. Fleetwood Homes of Washington, 670 P.2d 685 (Wash. Ct. App. 1983), rev’d, 684 P.2d 1302 (Wash. 1984). In this case, the plaintiff’s expert performed a memory T-cell test to determine whether the plaintiff had been exposed previously to formaldehyde. Id. Although, the test showed previous exposure and the trial court found the defendant liable, the Washington Court of Appeals did not find the evidence persuasive. Id. The Supreme Court of Washington held that the plaintiff’s evidence was persuasive and thus reversed the Court of Appeals decision. See Tiderman, 684 P.2d at 1304.
107 See Levin & Byers, supra note 15, at 676.
in a number of ways.\textsuperscript{108} If a researcher decides to study the T-cells of a population of people, that may be the beginning of a epidemiological study.\textsuperscript{109}

IV. THE USE OF EPIDEMIOLOGICAL EVIDENCE TO PROVE CAUSATION IN TOXIC TORTS

Although most courts still require plaintiffs to meet the strong version of the preponderance rule to prove causation, many courts do regard highly epidemiological studies that show strong causal associations between chemical exposure and disease.\textsuperscript{110} If an epidemiological study shows a significant statistical association between an injury and a toxin, and the study was conducted in a manner that follows accepted scientific criteria, it can be strong evidence of a causal association between the plaintiff’s injury and the defendant’s conduct.\textsuperscript{111}

A. Epidemiology as a Science

Epidemiology is the study of the dispersion of disease occurrence within and among human populations and the determination of the possible causes through the use of statistics.\textsuperscript{112} Using a chosen human population,\textsuperscript{113} an epidemiological study can generate evidence that demonstrates whether a probabilistic correlation exists between the defendant’s action and the plaintiff’s injury.\textsuperscript{114} An epidemiological study is the only study that can quantify the magnitude of the studied correlation in humans.\textsuperscript{115}

Although many believe that epidemiological studies are often the best way of determining health risks that a toxin may pose to human health, epidemiology is not a perfect science. The diseases usually involved in toxic torts can be characterized by long latency periods,
uncertain causes and etiology, and the fact that a chemically caused disease is indistinguishable from a disease caused by something else.¹¹⁶ Furthermore, an epidemiological study can never show conclusively that an individual's exposure to a chemical caused a particular injury.¹¹⁷

Because epidemiological studies rely on human populations, researchers cannot exercise the same degree of control that they do in the laboratory.¹¹⁸ In order to ensure the validity of a study's statistical results, researchers must control the elements of chance, bias in subject selection or manner of study, and confounding.¹¹⁹ An epidemiologist also must determine whether the results of the study apply generally beyond the population studied.¹²⁰ As a result, valid and reliable epidemiological correlations are difficult to establish.¹²¹

Indeed, even if an epidemiologist discovers a valid and significant statistical association, that does not necessarily indicate a causal association.¹²² Strict criteria exist that epidemiologists follow in order to establish causation in an epidemiological study.¹²³ First, the strength and significance of association is a ratio measure between a control group and a group exposed; the higher the ratio, the more significant the relationship between the factor and the disease.¹²⁴ Second, findings by separate researchers using different study designs and under different circumstances should show a consistency of association.¹²⁵

A third criterion is specificity.¹²⁶ Specificity requires a showing that a certain type of exposure is associated with a certain type of disease.¹²⁷ Because some diseases, however, may have many possible

¹¹⁶ Wong, supra note 111, at 85.
¹¹⁷ Dangel, supra note 7, at 178.
¹¹⁸ See HENNEKENS & BURING, supra note 109, at 31 (scientists cannot perform direct experiments on humans).
¹¹⁹ See id. Confounding occurs when a correlation, or lack of correlation, between an injury and disease is not causal, but rather, a third factor that is related to the exposure is causing the injury. See id. at 35–36. An example of this problem is that studies of asbestos workers who later become ill with cancer may be confounded by the possible factor that asbestos workers tend to smoke more than the average individual.
¹²⁰ Id. at 37.
¹²¹ Id. at 50.
¹²² Id.
¹²⁴ Id. at 21–22; see supra note 115 and accompanying text.
¹²⁵ Hill, supra note 122, at 22.
¹²⁶ Id.
¹²⁷ Id.
causes, specificity is not a strict requirement. Fourth, a researcher must show temporality. Temporality refers to the determination that the exposure to the possible cause of disease came before the onset of the disease. A fifth criterion is the dose-response relationship, which is a comparison of the strength of the exposure and the severity of disease response. If the severity of the disease increases with the amount of exposure, a strong dose-response relationship exists. The final guideline is biological plausibility. If the causal relationship between exposure and disease is consistent with present scientific knowledge, the argument for causation is stronger.

Once the epidemiological study is completed, an epidemiologist calculates the "attributable risk" to measure the causal relationship between an exposure and a specific individual. The attributable risk is a measure of association, quantifying the probability that the studied exposure did cause the exposed group's disease as compared to some other cause. To calculate the attributable risk, the epidemiologist first determines the relative risk of the population. The "relative risk" measures the probability of an individual in the exposed group becoming ill as compared to an individual in the nonexposed group. An epidemiologist calculates this number by dividing the disease incidence in the exposed group of people in the study over the disease incidence in the non-exposed group of people. The relative risk number is needed in order to calculate the attributable risk. Epidemiologists contend that if the attributable risk is greater than fifty percent, that calculation meets the more-likely-than-not standard of causation.

128 Id.
129 Id.
130 See id. at 49.
131 Id.
132 Id.
133 Id.
134 Id.
135 Id. at 50.
136 HENNEKENS & BURING, supra note 109, at 87.
137 Wong, supra note 5, at 49.
138 HENNEKENS & BURING, supra note 109, at 77.
140 Wong, supra note 5, at 50. Attributable risk is calculated as: AR = P(RR - 1)/(P(RR - 1) + 1). Id. "P" equals the proportion exposed in the study group and "P" equals one when the entire group is exposed. Id.
141 See id.
Consequently, although epidemiological studies can offer strong evidence of levels of risk in a population due to a certain cause, such studies at the most can show only that risk of disease.142 Epidemiological studies can never establish the cause of the disease in a specific individual.143 Furthermore, in attempting to produce valid studies, epidemiologists must be extremely careful to limit the problems of chance, confounding, and bias.144 Finally, to show a causal association, epidemiologists must follow several criteria that are difficult to meet.145

B. The Courts’ Analysis of Epidemiological Evidence

In toxic tort cases, some courts regard epidemiological studies as the only relevant evidence to prove causation.146 Even when it is available, particularistic evidence concerning a plaintiff may not have the same value to the court as studies that show an increased risk across a population.147 Thus, because epidemiological studies may appeal more to judges and juries than testimony of a physician’s examination in the toxic tort context, some plaintiffs need epidemiological evidence in order to prevail.148

In a number of recent cases, courts have carefully analyzed and understood epidemiological evidence.149 For example, In re Joint

142 Dore, supra note 51, at 433.
143 See supra note 117 and accompanying text.
144 See supra notes 105–08 and accompanying text.
145 See supra notes 109–21 and accompanying text; see generally Hennekens & Buring, supra note 59, at 31, 39.
146 See, e.g., In re “Agent Orange” Prod. Liab. Litig., 597 F. Supp. 740, 836 (E.D.N.Y. 1984), aff’d, 818 F.2d 145 (2d Cir. 1987). In the case the District Court for the Eastern District of New York decided to use the weak version of the preponderance rule, which only requires statistical evidence to prove causation. Id. The court reasoned that it is difficult, if not impossible, for plaintiffs to provide particularistic evidence, and that any particularistic evidence is based on probabilities. Id.
148 Christophersen v. Allied-Signal Chem. Corp., 839 F.2d 109, 1115–16 (5th Cir. 1991) (although plaintiff did not offer epidemiological evidence, court noted that even plaintiff’s expert believed that epidemiological evidence is important to establish causation); Heyman v. United States, 506 F. Supp. 1145, 1149 (S.D. Fla. 1981) (court denied plaintiff relief because she did not produce epidemiological evidence to show causation).
149 In a few older cases, courts understood the value and the limits of epidemiological evidence, admitted it as evidence, and cautiously analyzed its worth. See, e.g., Miller v. Nat’l Cabinet Co., 168 N.E.2d 811, 813–14 (N.Y. 1960) (court acknowledged that epidemiological, or statistical, evidence had value in proving causation). This trend has continued. See Brennan, supra note 1, at 494 n.127 (citing Oxendinge v. Merrell Dow Pharmaceuticals, Inc., 506 A.2d 1100 (D.C. Cir. 1986)). The District of Columbia Circuit Court closely examined the four principle methods for identifying toxicity and causation—cluster analyses, animal bioassays,
Eastern and Southern District Asbestos Litigation, the United States District Court for the Southern District of New York considered the importance of relative risk ratios in studying the plaintiff’s epidemiological studies to prove causation and granted the defendants’ motion to dismiss because the plaintiff could not show that her husband’s exposure to asbestos gave him twice the normal risk of developing cancer. Reversing the District Court’s decision, the United States Court of Appeals for the Second Circuit held that the plaintiff did not need to provide a certain level of epidemiological proof to defeat summary judgment because the plaintiff also provided particularistic evidence. The Court of Appeals, however, still expected a plaintiff to present some epidemiological evidence to prove causation.

In a case in which the defendants offered strong epidemiological evidence to the court, a group of plaintiffs lost a motion for summary judgment in the case, In re “Agent Orange” Product Liability Litigation. Claiming that exposure to the herbicide Agent Orange caused their health problems, the plaintiffs, Vietnam veterans, sued seven Agent Orange manufacturers. The United States District Court for the Eastern District of New York began its discussion of the worthiness of epidemiological evidence with the statement that those epidemiological studies concerning the link between exposure and disease to Agent Orange were the only useful studies having any bearing on causation. The court found that all the available epidemiological evidence showed no causal link between Agent Orange and the illnesses of the Vietnam veterans. Therefore, according to the court, the plaintiffs could not offer expert testimony because there was no strong epidemiological evidence on which to

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short-term molecular assays, and epidemiological studies. Id. The court also reviewed the expert testimony dealing with epidemiological evidence, the importance of relative risk ratios, and the statistical significance of confidence intervals, to decide that the plaintiff’s evidence did establish a causal link between birth defects and benedictin. Id.

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151 Id. at 202–03.
152 In re Joint Eastern, 964 F.2d at 97.
153 See id.
155 Id. at 1228.
156 Id. at 1231. The court stated that “[i]n a mass tort case such as Agent Orange, epidemiologic studies on causation assume a role of critical importance.” Id. at 1239.
157 Id. at 1231–34. The court cited two epidemiological studies that showed no causal link between Agent Orange exposure and harmful reproductive effects. Id. at 1231–32. Other epidemiological studies have been negative or inconclusive concerning a possible causal link between Agent Orange and the illnesses of the Vietnam veterans. Id. at 1232–34.
base their experts' argument that Agent Orange caused the plaintiffs' diseases.\textsuperscript{158} Furthermore, although one of the plaintiffs' experts submitted affidavits that included the results of T-cell studies performed on the plaintiffs, he did not conclusively state that Agent Orange more likely than not caused the plaintiffs' illnesses.\textsuperscript{159} The court noted that if the plaintiffs' experts could not state that the T-cell studies showed that Agent Orange more likely than not caused their illnesses, the plaintiffs could not prove causation.\textsuperscript{160}

The \textit{In re Agent Orange} court concluded that the plaintiffs failed to show causation for three major reasons.\textsuperscript{161} First, their experts' conclusions did not consider the epidemiological studies that showed no causation.\textsuperscript{162} Secondly, according to the court, the unidentified studies of animals and industrial workers upon which the plaintiffs' experts relied were not relevant.\textsuperscript{163} Finally, the experts did not carefully analyze other possibilities of causation.\textsuperscript{164}

The preceding cases show that more courts are using epidemiological studies to determine if the causation element has been met to prove a toxic tort.\textsuperscript{165} Even with expert testimony concerning the individual plaintiff's illness, plaintiffs may now need a valid epidemiological study to indicate that exposure to a certain chemical will probably result in a rise in the incidence of the illness.\textsuperscript{166}

\section*{V. The Use of Particularistic Evidence to Prove Causation in Toxic Torts}

Particularistic evidence of causation provides a court with direct evidence of the cause of a particular plaintiff's injury.\textsuperscript{167} Particularistic evidence is the only evidence that can specifically analyze the relation between a particular exposure and a particular plaintiff.\textsuperscript{168} Consequently, courts have historically relied on this kind of evidence to prove causation.\textsuperscript{169}

\begin{itemize}
\item \textsuperscript{158} \textit{Id.} at 1234.
\item \textsuperscript{159} \textit{Id.} at 1237.
\item \textsuperscript{160} \textit{See id.} at 1238.
\item \textsuperscript{161} \textit{Id.} at 1250.
\item \textsuperscript{162} \textit{Id.}
\item \textsuperscript{163} \textit{Id.}
\item \textsuperscript{164} \textit{Id.}
\item \textsuperscript{165} \textit{See supra} notes 149–64 and accompanying text.
\item \textsuperscript{166} \textit{See}, e.g., \textit{In re "Agent Orange,"} 611 F. Supp. at 1238.
\item \textsuperscript{167} \textit{See generally} Callahan, \textit{supra} note 1, at 619.
\item \textsuperscript{168} \textit{See id.}
\end{itemize}
A. The Origins of Particularistic Evidence

Particularistic evidence of causation most commonly takes the form of expert testimony by a physician who personally has examined the patient.\textsuperscript{170} As a basis for his or her conclusion, the physician most likely has performed clinical tests gauging the patient’s health.\textsuperscript{171} The purpose of the testimony is then for the medical expert to state his or her determination that the cause of the plaintiff’s injury is the defendant’s conduct.\textsuperscript{172} To meet the standard of legal causation, the expert must testify to a reasonable degree of medical certainty that the defendant’s conduct caused the plaintiff’s injury.\textsuperscript{173}

Thus, the importance of particularistic evidence is that it provides a court with causal evidence concerning a specific plaintiff.\textsuperscript{174} In fact, some courts demand particularistic evidence in order for a plaintiff to meet the demands of the “strong” version of the preponderance rule.\textsuperscript{175} Indeed, other kinds of evidence—\textit{in vivo} studies, \textit{in vitro} studies, case reports, and epidemiological evidence—cannot address the cause of any specific plaintiff’s injury.\textsuperscript{176}

Particularistic evidence, however, does have significant limits in the context of toxic tort cases.\textsuperscript{177} There is usually a long latency period between an exposure and the manifestation of a disease.\textsuperscript{178} Moreover, the etiology of many diseases is still unknown,\textsuperscript{179} and physicians usually are unable to differentiate between when a disease is caused by toxic exposure and when the same disease is caused by another factor.\textsuperscript{180}

\textsuperscript{170} Callahan, \textit{supra} note 1, at 619.
\textsuperscript{171} See Levin & Byers, \textit{supra} note 15, at 673–75.
\textsuperscript{172} See Callahan, \textit{supra} note 1, at 619.
\textsuperscript{173} Id.
\textsuperscript{174} Id. at 608, 639. Callahan contends that there are two prongs of the causation analysis. Id. at 608. The first prong is the determination that the defendant’s behavior did or did not put the plaintiff at risk of incurring an injury. Id. The second prong is the occurrence determination, which assumes that the defendant did put the plaintiff at risk but then analyzes whether the defendant did in fact cause the plaintiff’s injury. Id. Callahan argues that particularistic evidence is the only evidence that goes to proving the occurrence prong of the causation analysis. Id. at 608, 639.
\textsuperscript{175} See Callahan, \textit{supra} note 1, at 610–11, \textit{citing} \textit{In re “Agent Orange” Prod. Liab. Litig.}, 611 F. Supp. 1223, 1261 (E.D.N.Y. 1985), aff’d, 818 F.2d 187 (2d Cir. 1987); \textit{Developments}, \textit{supra} note 42, at 1619.
\textsuperscript{176} See \textit{generally} Callahan, \textit{supra} note 1, at 639; Dangel, \textit{supra} note 5, at 174–78. \textit{In vivo} studies evaluate the result of exposing animals to different doses of a toxin. Id. at 175. \textit{In vitro} studies examine the result of exposing individual animal cells to a toxin. Id.
\textsuperscript{177} Callahan, \textit{supra} note 1, at 630–40.
\textsuperscript{178} Wong, \textit{supra} note 111, at 85.
\textsuperscript{179} Id.
Furthermore, expert testimony regarding particularistic evidence must meet the standards of FRE 403, 702, and 703.\textsuperscript{181} Under FRE 702, the testimony must be in the area of the expert's qualifications, and must aid the court or jury in comprehending the evidence.\textsuperscript{182} If the expert is qualified to give testimony that would help the court, the expert also must meet the provision of FRE 703 requiring a reasonable scientific foundation for the expert's opinion.\textsuperscript{183} FRE 403 demands that the expert testimony not be greatly outweighed by prejudice, confusion, or deception of the jurors.\textsuperscript{184} In a toxic tort case, even when an expert can give testimony that meets the FRE requirements, an expert cannot give causation testimony completely free of any doubt.\textsuperscript{185}

B. The Courts' Analysis of Particularistic Evidence

Some courts have reasoned that because statistical evidence will never meet the legal causation standard, it does not warrant serious consideration.\textsuperscript{186} These courts have relied upon particularistic evidence regarding the plaintiff, no matter how weak, in favor of evidence that can only establish a probability of causation.\textsuperscript{187}

In \textit{Ferebee v. Chevron Chemical Co.},\textsuperscript{188} the trial court awarded the Ferebee family $60,000 for the wrongful death of the plaintiff, who had been exposed to the Chevron herbicide paraquat while he was working at an agricultural center.\textsuperscript{189} The plaintiff's job was to spray the herbicide which came into contact with his skin on several occasions.\textsuperscript{190} Several months after the first exposure, the plaintiff developed pulmonary fibrosis and died.\textsuperscript{191} At trial, the plaintiff's family presented the expert testimony of two pulmonary specialists who had examined the plaintiff.\textsuperscript{192} They concluded that paraquat poisoning was the cause of the plaintiff's injury.\textsuperscript{193}

\begin{footnotesize}
\begin{enumerate}
\item[181] See Callahan, \textit{supra} note 1, at 631.
\item[182] Id.; FED. R. EVID. 702.
\item[183] FED. R. EVID. 703; Callahan, \textit{supra} note 1, at 632–33.
\item[184] FED. R. EVID. 403; Callahan, \textit{supra} note 1, at 633.
\item[185] \textit{See generally} Callahan, \textit{supra} note 1, at 639.
\item[189] \textit{Id.} at 1529, 1531–32.
\item[190] \textit{Id.} at 1532.
\item[191] \textit{Id.} at 1533.
\item[192] \textit{Id.} at 1533.
\item[193] \textit{Id.}
\end{enumerate}
\end{footnotesize}
The defendants appealed the case to the the United States Circuit Court of Appeals for the District of Columbia. The appellate court held that the plaintiffs did not need to establish cause through epidemiological evidence or animal studies because testimony by the two expert witnesses was legally sufficient to show causation. In this case, then, particularistic evidence satisfied the causation standard.

Another case that rejected the need for epidemiological evidence to prove causation involved a child with birth defects who brought a products liability action against a pharmaceutical company that manufactured a spermicide. In *Wells v. Ortho Pharmaceutical Corp.*, the trial court awarded the plaintiff over five million dollars even though the defendants presented epidemiological evidence that no causal association existed between birth defects and the spermicide.

Relying on *Ferebee*, the United States Court of Appeals for the Eleventh Circuit affirmed the trial court decision. The court reasoned that the use of epidemiological evidence to refute a causal association between the spermicide and the plaintiff’s birth defects was not of legal worth as it did not relate to that particular plaintiff in that particular case. The court closely examined the plaintiff’s particularistic evidence, and ignored the defendant’s epidemiological study showing that no association existed between birth defects and the use of spermicides. According to the court, the plaintiff’s highly qualified expert witnesses established causation more strongly than the defendant’s epidemiological studies that did not concern this specific plaintiff.

Plaintiffs presented T-cell studies as particularistic evidence to prove causation in *Sterling v. Velsicol Chemical Corp.* Residents who lived near a chemical waste disposal site sued the Velsicol

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194 *Id.* at 1533.
195 *Id.* at 1535.
196 See *id.* at 1535–36.
198 *Id.*
199 *Id.* at 296–98.
200 *Id.* at 292.
201 *Wells v. Ortho Pharmaceutical Corp.*, 788 F.2d 741, 745 (11th Cir. 1986).
202 See *id.* at 745.
203 See *id.* at 744.
204 See *id.* at 744–45.
Chemical Corporation, accusing Velsicol of allowing chemicals that the company had dumped to leak into the plaintiffs' drinking water. \textsuperscript{206} The United States District Court for the Western District of Tennessee held that the defendant was strictly liable, negligent, and liable in both trespass and nuisance. \textsuperscript{207} The court awarded the five representative plaintiffs five million dollars and the entire group of plaintiffs seven and one half million dollars in punitive damages. \textsuperscript{208}

Two immunologists, Dr. Alan S. Levin and Dr. William G. Crook, testified for the plaintiffs to prove injury of the plaintiffs' immune systems. \textsuperscript{209} As a result of his studies of the T-cell levels of five specific plaintiffs, Dr. Levin concluded that their immune systems were permanently and seriously damaged. \textsuperscript{210} He diagnosed these plaintiffs as having "chemically induced immune dysregulation," which only develops because of chronic chemical exposure. \textsuperscript{211} Dr. Levin determined from his studies that the source of the illnesses was the chemical exposure from the Sterling Velsicol Chemical Corporation. \textsuperscript{212} Dr. Crook supported Dr. Levin's statements regarding the extent of the damage to the plaintiffs' immune systems. \textsuperscript{213} Relying on the testimony of the plaintiffs' experts, the court found that the defendant's expert testimony was "against the preponderance of the evidence." \textsuperscript{214}

The United States Court of Appeals for the Sixth Circuit, however, reversed on the issue of compensatory damages for immune dysregulation, reasoning that the plaintiffs' experts did not perform the proper clinical tests or examinations on the plaintiffs. \textsuperscript{215} Furthermore, according to the court, the medical basis \textsuperscript{216} of their testimony did not comport with FRE 702. \textsuperscript{217} Although the court admitted

\textsuperscript{206} Id. at 306.  
\textsuperscript{207} Id. at 307.  
\textsuperscript{208} Id.  
\textsuperscript{209} Id. at 499.  
\textsuperscript{210} Id. at 500-01. Dr. Levin determined that all five plaintiffs had a high percentage of T-cells. Id.  
\textsuperscript{211} Id. at 502, 505.  
\textsuperscript{212} See id. at 504-05.  
\textsuperscript{213} Id. at 505.  
\textsuperscript{214} Id. at 507.  
\textsuperscript{215} Sterling v. Velsicol Chem. Corp., 855 F.2d 1188, 1209 (6th Cir. 1988).  
\textsuperscript{216} Id. at 1208-09 (court contended that prominent medical societies in field of allergy and immunology reject clinical ecology, which was medical basis of Dr. Crook's and Dr. Levin's testimony).  
\textsuperscript{217} Id. the Sixth Circuit applied a four-pronged test to determine whether the testimony is admissible under FRE 702. The expert must be 1) a qualified expert, 2) testifying on an appropriate topic, 3) that coincides with a generally accepted theory, and 4) the probativeness of the testimony must outweigh any prejudicial effect. Id. at 1208.
that numerous medical associations, including the American Medical Association, have not repudiated clinical ecology, the court demanded a more traditional methodology. Thus, because the Sixth Circuit would not accept the validity of T-cell tests nor the methodology of clinical ecology, the plaintiffs lost on their immune dysregulation claims.

Not all courts have considered T-cell studies unable to meet the requirements of FRE 702. In Elam v. Alcolac, Inc., the United States District Court for the Western District of Missouri found that T-cell studies can show the effects of chemical exposure on the immune system. Thus, the Elam court is an example of a court that has accepted the science of clinical ecology, carefully weighed the probativeness of T-cell studies, and accepted the T-cell study results as determining causation. To prove the strong version of the preponderance rule, courts require particularistic evidence, which in the case of Elam, the evidence was T-cell studies.

VI. THE USE OF BOTH EPIDEMIOLOGICAL AND PARTICULARISTIC EVIDENCE TO PROVE CAUSATION IN TOXIC TORTS

Although some courts have criticized the limits of either epidemiological or particularistic evidence, other courts rely on both to prove the strong version of the preponderance rule, or in other words, traditional causation. Even in early toxic tort cases, courts acknowledged the importance of the combination of particularistic and epidemiological evidence to prove causation.

In Allen v. United States, the plaintiffs sued the United States government under the Federal Tort Claims Act to obtain damages for cancer incidence resulting from nuclear bomb testing between 1951 and 1963. The plaintiffs alleged that the radioactive fallout from the testing drifted into their communities and caused them to develop cancers including leukemia. Noting the long latency period

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218 See id. at 1209.
219 See id.
221 Id.
222 See, e.g., id.
223 See generally id.
224 Developments, supra note 42, at 1619.
227 Id. at 247.
228 Id. at 252–58.
and the unknown etiology of cancer, the United States District Court for the District of Utah acknowledged the difficulties the plaintiffs had in proving legal causation.\footnote{Id. at 405–06.}

The court found, however, that the plaintiffs' evidence showed a causal connection between the defendant's conduct and their injuries.\footnote{Id. at 406.} Applying the substantial factor test of causation,\footnote{Id. at 411; see supra notes 51–53 and accompanying text.} the court held that the plaintiffs had established causation by presenting an extensive amount of evidence relating the defendant's conduct to the plaintiffs' injury.\footnote{See id. at 425.} In reaching its conclusion, the court considered particularistic factors such as the fact that the injury, cancer, was consistent with the type of exposure the plaintiffs experienced; the proximity of the plaintiffs to the test site; the extent of their exposure; and the existence of a latency period that was consistent with radiation etiology.\footnote{Id. at 415.}

The Allen court also considered probabilistic factors, such as the probability that the plaintiffs were exposed to radiation and whether the incidence of injury in the community was statistically above the norm.\footnote{Id. at 418.} Although it admitted that statistical evidence, or epidemiological evidence, is the strongest evidence for proving causation in toxic torts,\footnote{See id. at 416.} the Allen court rejected the concept of a 5% limit of chance as a threshold value and other technical rules of statistical significance.\footnote{Id. at 418.} Consequently, the court relied on all evidence that was persuasive, not just epidemiological evidence, in concluding that the United States government was responsible for the plaintiffs' injuries.\footnote{Id. at 416.} The court allocated damages to those plaintiffs who provided both strong statistical evidence of a causal connection and positive expert testimony regarding that specific plaintiff's injury.\footnote{See id. at 437–43.}

In a more recent case, Elam v. Alcolac, Inc.,\footnote{765 S.W.2d 42 (Mo. App. 1988), cert. denied, 493 U.S. 817 (1989).} the plaintiffs also used both epidemiological and particularistic evidence to prove cau-
sation.\(^{240}\) In *Elam*, residents of Sedalia, Missouri, sued Alcolac, Inc., the owner of a chemical plant that had spilled chemicals including epidichlorohydrin, toluene, and allyl alcohol.\(^{241}\) The trial court jury decided the issues of lowered market value of property which sounded in nuisance, and personal injury negligence.\(^{242}\) The jury awarded each plaintiff two hundred thousand dollars for personal injury and over 1.3 million dollars for punitive damages.\(^{243}\) The trial court denied the defendant's motion for a judgment not withstanding the verdict, but granted a new trial as to the issue of damages.\(^{244}\) Consequently, the plaintiffs appealed the court's order to set aside the damage awards and the grant for a new trial.\(^{245}\)

The Missouri Court of Appeals held that the plaintiffs' evidence was sufficient for the jury to determine that the toxic spills caused the residents' injuries, and that the compensation damages were valid.\(^{246}\) Because of the improper admission of evidence regarding "chemically induced acquired immune deficiency syndrome" (AIDS), the *Elam* court demanded a new trial on the issue of punitive damages.\(^{247}\)

In reviewing the case, the *Elam* court had applied the substantial factor test to determine causation.\(^{248}\) To prove causation, the plaintiffs offered both particularistic and epidemiological evidence.\(^{249}\) To provide particularistic evidence, the plaintiffs retained Dr. Carnow.\(^{250}\) Dr. Carnow performed a patient evaluation of symptoms, laboratory tests, and medical history interviews on each of the plaintiffs.\(^{251}\) In addition, Dr. Arthur C. Zahalsky performed T-cell studies

\(^{240}\) See id. 180–85.
\(^{241}\) Id. at 49, 72.
\(^{242}\) Id. at 49.
\(^{243}\) Id. The award for the nuisance claims varied for the different plaintiffs. *Id.*
\(^{244}\) *Id.*
\(^{245}\) *Id.*
\(^{246}\) See id. at 189–97, 222.
\(^{247}\) See id. at 213–14.
\(^{248}\) Id. at 173–74. The source of the substantial factor test, is the Restatement (Second) of Torts § 432 (2). According to the Restatement, "[i]f two forces are actively operating, one because of the actor's negligence, the other not because of any misconduct on his part, and each of itself is sufficient to bring about harm to another, the actor's negligence may be found to be a substantial factor in bringing it about." *Id.* at 174.
\(^{249}\) *Id.* at 73.
\(^{250}\) See id. at 89. To prove cause-in-fact, Dr. Carnow had to prove all elements of an external cause: a related external event, exposure of the plaintiffs to the event, effect from exposure, relationship of the effect to the external event, evidence that others had been similarly exposed and similarly harmed, and laboratory results that were expected. *Id.*
\(^{251}\) *Id.* at 91.
of the plaintiffs. According to Dr. Zahalsky's testimony, the results of his studies showed that all of the plaintiffs' immune systems were fighting against chronic exposure to toxins. A different laboratory also performed T-cell studies on twenty-four of the plaintiffs and discovered that nineteen of them had an immune dysfunction, which Dr. Zahalsky believed to be overwhelmingly statistically significant. As a result of his findings, Dr. Zahalsky diagnosed all thirty-one plaintiffs with "systemic, progressive chemical intoxication." Dr. Carnow confirmed this diagnosis. For each of the plaintiffs, Dr. Zahalsky also tested a T-cell antibody, which led him to conclude that ten plaintiffs had chemically induced AIDS. Further, because the immunological problems have been progressive, the other plaintiffs that only have moderate immune dysregulation eventually will acquire AIDS.

To show a relationship between exposure to the defendant's chemicals and the plaintiffs' injuries—or in other words, biological causation—the plaintiffs' experts presented epidemiological evidence. A toxicologist for the plaintiffs presented epidemiological and animal studies showing a correlation between exposure to epichlorihydrin and cancer, chromosomal damage, and immune damage. As an epidemiologist and environmental medicine specialist, Dr. Carnow

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252 Id. at 84. Dr. Zahalsky testified that immune dysfunction means that because of chemical exposure or some other antigen attack, one's immune system becomes less protective against foreign agents that enter the body and a harmful substance that does enter the system may escape attack and damage the system further. Id. at 88.

253 Id. Dr. Carnow testified on the importance of B-cells and T-cells in the immune system. Id. T-cells "search and destroy" antigens in the body, remember prior attacks by antigens, live long, and can expand. Id. Because of the plaintiffs' T-cell irregularities, Dr. Carnow concluded that the T-cell study results were consistent with persistent exposure to the Alcolac's toxins. Id. at 90–100.

254 Id. at 85. Specifically, Dr. Zahalsky tested for the number of total lymphocytes (T-cells and B-cells), the speed of the T-cells when attacked, the number of each different kind of T-cells, and the ratios of the different kind of T-cells. Id. at 99.

255 Id. at 86. Even the Alcolac experts acknowledged that immune dysregulation may result from exposure to the Alcolac chemicals. Id. at 99.

256 Id. at 100–01.

257 Id. at 85. By testing the T-cell antibody, HNK-1, Dr. Zahalsky could determine whether there was an abnormal ratio of helper and suppressor cells: an indication of chemically induced AIDS. Id.

258 Id.

259 Id. at 73–74. The court stated that to show biological causation, a plaintiff must offer epidemiological, toxicological, or pharmacological evidence. Id. at 185. Statistically significant studies are solid circumstantial evidence. Id. at 185–87.

260 Id. at 73.

261 Id. at 91.
confirmed these findings.\textsuperscript{262} Even an expert for Alcolac admitted that studies have shown exposure to epichlorohydrin creates a higher risk of cancer and may damage the immune system.\textsuperscript{263}

Consequently, the defendants did not attempt to prove that the chemicals could not cause injuries similar to those of the plaintiffs.\textsuperscript{264} Alcolac did contend, however, that the plaintiffs could not prove that they were in fact exposed to the chemicals, or that chronic systemic chemical intoxication was a valid diagnosis of their condition.\textsuperscript{265} Because the plaintiffs testified to seeing the toxins and Dr. Carnow testified that Alcolac's spills could be the only possible cause of their injuries, however, the \textit{Elam} court held that such evidence was sufficient to prove cause-in-fact.\textsuperscript{266} The court also recognized chronic systemic chemical intoxication as a valid medical diagnosis.\textsuperscript{267} Thus, the court relied on both epidemiological evidence as well as particularistic expert testimony to reach its result.\textsuperscript{268}

Although it never came to a final verdict, a case in which the plaintiffs performed their own epidemiological study and extensively used T-cell studies was \textit{Anderson v. W.R. Grace & Co.}.\textsuperscript{269} Several children in Woburn, Massachusetts, contracted leukemia during the 1970s.\textsuperscript{270} The residents of Woburn later discovered that the city wells were contaminated with the carcinogens, trichlorethylene (TCE) and tetrachloroethylene (PCE).\textsuperscript{271} Consequently, plaintiffs sued W.R. Grace and Beatrice Foods for wrongful death and pain and suffering for the deaths of family members; emotional distress for witnessing the deaths of family members or for suffering from illnesses themselves; compensation for various illnesses; and increased risk of developing future illness.\textsuperscript{272} In the months of preparation before trial, the plaintiffs' investigations disclosed a great amount of dumping

\textsuperscript{262} \textit{Id.} at 73.
\textsuperscript{263} See \textit{id.} at 74. Alcolac also possessed a material safety data sheet indicating that a recent epidemiological study was "highly suggestive" that exposure to epichlorohydrin increases the risk of cancer. \textit{Id.}
\textsuperscript{264} \textit{Id.} at 185.
\textsuperscript{265} \textit{Id.} at 180, 189.
\textsuperscript{266} See \textit{id.} at 180–85.
\textsuperscript{267} \textit{Id.} at 189–90.
\textsuperscript{268} See \textit{id.} 73–74, 86.
\textsuperscript{270} Jan Schlichtmann, \textit{Eight Families Sue W.R. Grace and Beatrice Foods for Poisoning City Wells with Solvents and Causing Leukemia, Disease, and Death}, 341 PRAC. L. INST. 1 (1987).
\textsuperscript{271} \textit{Id.} at 1.
\textsuperscript{272} \textit{Anderson}, 628 F. Supp. at 1222.
near the water wells by Beatrice Corporation, W.R. Grace, and the
Unifirst Corporation.\textsuperscript{273} Unifirst and the plaintiffs, however, soon
reached a settlement for one million dollars.\textsuperscript{274}

In order to understand the extent of the damage the toxins
tallegedly caused to each of the plaintiffs, the plaintiffs retained Dr.
Alan S. Levin.\textsuperscript{275} Dr. Levin conducted T-cell studies, both of the
plaintiffs and their families, that calculated the absolute numbers
and ratios of T-cells.\textsuperscript{276} In his affidavit, Dr. Levin evaluated the
medical histories and test results of individual family members,
which indicated that the subjects had been fighting an unrelenting
carcinogen.\textsuperscript{277} He explained that the carcinogens present in Woburn's
well system could cause this harm to the plaintiffs' immune sys-
tems.\textsuperscript{278} Consequently, in a weakened state, the immune system
cannot fight the carcinogens that are constantly assaulting the
body.\textsuperscript{279}

Regarding epidemiological evidence of causation, the Harvard
School of Public Health and the Centers for Disease Control (CDC)
completed an epidemiological health study of the children in the area.\textsuperscript{280} The study found a strong link between childhood illnesses,
including leukemia, and the children's use of the contaminated wells
in Woburn.\textsuperscript{281} Thus, the Harvard Health Study bolstered Dr. Levin's
conclusions that a causal link existed between the defendants' chem-
icals and the plaintiffs' injuries.\textsuperscript{282}

The defendants, however, moved for summary judgment, claiming
that the statute of limitations barred three of plaintiffs' wrongful
death claims, and that the plaintiffs' claims based on emotional distress and increased risk of cancer were insufficient.\footnote{Anderson v. W.R. Grace & Co., 628 F. Supp. 1219, 1222 (D. Mass. 1986), aff'd sub nom., Anderson v. Beatrice Foods Co., 900 F.2d (1st Cir. Mass.), cert. denied, 111 S. Ct. 233 (1990).} The Massachusetts statute of limitations bars wrongful death claims if death occurs more than two years after injury.\footnote{Id. at 1225 (citing M.G.L. CH. 229 § 2).} The United States District Court for the District of Massachusetts held that continued exposure to carcinogens, even after the onset of disease, indicated further injury.\footnote{Id. at 1225.} The Anderson court reasoned that Dr. Levin's evidence regarding T-cell studies proved that the decedents' continuous exposure to toxins from the well water even after the diagnosis of their leukemia caused further injury quickening the deaths of James Anderson and Carl Robbins, children of family members suing for wrongful death.\footnote{See id.} In other words, the later, continuous exposure was the cause of death.\footnote{Id.} Therefore, because James Anderson's last exposure to the defendants' chemicals was within two years of his death, his family could recover, however Carl Robbins's family could not recover because Carl's last exposure was not within two years of his death.\footnote{Id. at 1226.}

W.R. Grace and Beatrice Foods also argued in the same summary judgment motion that the nonleukemic plaintiffs did not have a cause of action for negligent infliction of emotional distress because they could not prove that they suffered a physical injury.\footnote{Id. at 1225.} The defendants argued that T-cell studies are not "objective symptomatology" of harm, as the Massachusetts Supreme Judicial Court requires.\footnote{Objective symptomatology is evidence of physical harm that can be supported by expert medical testimony. See id. at 1226–27.} The Anderson court, however, disagreed and held that the nonleukemic plaintiffs did have a cause of action for emotional distress.\footnote{Id.} Relying on Dr. Levin's testimony that the plaintiffs had suffered T-cell damage, the court concluded that these plaintiffs did suffer a physical injury to their immune systems.\footnote{Id. at 1226.} Furthermore, according to the court, there had been no ruling by the Supreme Judicial Court that the physical harm should be easily detectable as compared to subcellular.\footnote{Id. at 1226–27.} Thus, the court held that T-cell damage could prove...
the physical injury requirement of negligent infliction of emotional distress.\textsuperscript{294}

Another important issue in the defendants' summary judgment motion was whether a claim for increased risk of future illness existed.\textsuperscript{295} The court delayed action on this count, explaining that the plaintiffs could not recover until they showed that certain diseases traditionally resulted from the health problems they presently had.\textsuperscript{296} According to the court, the plaintiffs had to prove that there was a "reasonable probability" that the diseases would occur.\textsuperscript{297}

In the first phase of the trial, the court entered judgment in favor of Beatrice.\textsuperscript{298} The \textit{Anderson} court found W.R. Grace liable in negligence for contaminating the wells.\textsuperscript{299} Consequently, W.R. Grace settled with the plaintiffs for eight million dollars.\textsuperscript{300} Although there was never a complete trial, the court's summary judgment holding shows the court's willingness to examine the value of T-cell studies in proving causation.\textsuperscript{301} Supplemented by physicians' examination of the plaintiffs and the Harvard epidemiological studies, T-cell studies could have established causation for the plaintiffs' injuries and emotional distress.

Some courts require plaintiffs to meet the demands of the strong version of the preponderance rule in order to gain compensation.\textsuperscript{302} In \textit{Allen}, the plaintiffs presented both particularistic and epidemiological evidence.\textsuperscript{303} The court accepted this evidence, realizing its limits, but understanding its importance for showing causation.\textsuperscript{304} In \textit{Elam}, because of the strength of the plaintiffs' particularistic and epidemiological evidence regarding causation, the defendants did not even contest causation.\textsuperscript{305} The evidence helped to establish chronic systemic intoxication as a recoverable injury.\textsuperscript{306} Finally, \textit{Anderson}
demonstrates a court's willingness to accept T-cell studies bolstered by strong epidemiological evidence.\(^{307}\) In all three cases, defendants were found liable or failed to win a summary judgment motion because of the strength of the plaintiffs' epidemiological and particularistic evidence.\(^{308}\)

VII. THE VALUE OF T-CELL STUDIES IN MODERN-DAY TOXIC TORT LITIGATION

The legal standard for causation in toxic tort cases is far from settled.\(^{309}\) Particularistic evidence of causation is usually difficult to establish because of the nature of toxic tort injury.\(^{310}\) Furthermore, scientific and statistical evidence will never meet all the demands of the legal causation standard.\(^{311}\) Consequently, courts have struggled to decide what kind of evidence is relevant in proving causation in toxic tort cases.\(^{312}\) Confusion regarding the standard of causation and the intricacies of scientific evidence, however, have brought about inconsistencies among courts' decisions.\(^{313}\)

Some courts demand particularistic evidence,\(^{314}\) some courts demand epidemiological evidence,\(^{315}\) and some courts demand both.\(^{316}\) Courts that use the strong version of the preponderance rule, which requires both particularistic and epidemiological evidence to determine causation, understand that each type of evidence overcomes some of the other's limitations.\(^{317}\) Because T-cell studies can be the basis for both an epidemiological study and a particularistic test, such studies can thus satisfy the strong version of the preponderance rule. If scientists, immunologists, and litigants develop the potential of T-cell studies, these studies can become a more prevalent form of proof in toxic tort cases.

\(^{307}\) See Schlichtmann, supra note 270, at 4–5.

\(^{308}\) See id.; Allen, 588 F. Supp. at 415; Elam; 765 S.W. 2d at 180–85.

\(^{309}\) See supra notes 27–62 and accompanying text.

\(^{310}\) See supra notes 170–85 and accompanying text.

\(^{311}\) See supra notes 112–45 and accompanying text.

\(^{312}\) See supra notes 146–66, 186–308 and accompanying text.


\(^{314}\) See supra notes 186–223 and accompanying text.

\(^{315}\) See supra notes 146–66 and accompanying text.

\(^{316}\) See supra notes 226–308 and accompanying text.

\(^{317}\) See generally id.
A. The Limits and Potential of T-Cell Studies

The methodology from which T-cell studies developed, clinical ecology, is still a much disputed area of science. The study of MCS in clinical ecology has resisted a standard definition, theory of etiology, symptomatology, diagnosis, and treatment. An undisputed fact, however, is that chemicals in the environment are causing damage to humans—what we do not know is to what extent. Because clinical ecology is still in its formative stages, it is worthy of study and research, in order to understand the significance and etiology of MCS.

B. The Use of T-Cell Studies as Epidemiological Evidence

Because some courts rely on epidemiological evidence for proof of causation, plaintiffs and defendants should be able to offer epidemiological evidence to show that there is or is not a link between exposure to the defendant's chemical and the plaintiff's injury. Even when plaintiffs offer particularistic evidence that they have an injury, and that they were exposed to the defendant's chemical, courts often require evidence of a known biological relationship between the kind of exposure and the kind of injury that the plaintiff has. Therefore, epidemiological studies of T-cell levels in a population are a way of relating chemical exposure to immune damage or other injuries that result from immune damage.

For example, if a plaintiff wanted compensation for immune dysregulation, the plaintiff could present epidemiological studies that show a positive correlation between exposure to the defendant's chemical and chemically induced immune dysregulation. Few epidemiologists have conducted T-cell epidemiological studies to show a correlation between toxin exposure and injury. One study that researchers have conducted, however, is an epidemiological study.

318 See supra notes 94–95 and accompanying text.
319 See Terr, supra note 81, at 684–93; Stephen Mooser, The Epidemiology of Multiple Chemical Sensitivities, 2 OCCUPATIONAL MED. 663, 664 (1987).
320 See generally Mooser, supra note 319, at 663; Cullen, supra note 83, at 655–56.
323 See Terr, supra, note 81, at 689, 692.
determining the risk of acquiring asthma from formaldehyde exposure.\textsuperscript{324}

Because T-cell damage can range from slight immune dysregulation to chemically induced AIDS, T-cell epidemiological studies may have the advantage, over epidemiological studies of a specific disease incidence, by showing distinct dose-response relationships.\textsuperscript{325} A study of specific disease incidence does not show a variance in response.\textsuperscript{326} For example, a person has cancer or does not have cancer.\textsuperscript{327} The frequency of disease incidence determines dose-response.\textsuperscript{328} Therefore, to show a significant dose-response relationship in an epidemiological study of cancer incidence, an epidemiologist must use a very large exposure group covering a wide range of exposures.\textsuperscript{329} Although an epidemiological study of T-cell response also would require a variance of exposure levels, a valid study would not require a huge study population. For an epidemiological study of T-cell damage, the dose-response measurement would be the correlation between the variance in the exposure level to the variance in the T-cell damage; not a correlation between the variance in the exposure level and the number of a yes or no disease incidence.

In many toxic tort cases, courts demand epidemiological evidence because particularistic evidence of causation is minimal.\textsuperscript{330} As a basis for an epidemiological study, T-cell studies can show an existing relationship or lack of relationship between a chemical and an injury, and also demonstrate a dose-response relationship between an exposure level and a kind of injury. In future toxic tort cases, however, plaintiffs and defendants could provide, in addition to epidemiological studies, particularistic evidence of their T-cell health.

C. The Use of T-Cell Studies as Particularistic Evidence

In the past, when plaintiffs using T-cell evidence have lost their cases, courts have claimed that T-cell studies have failed to overcome

\begin{itemize}
  \item \textsuperscript{324} H. Pross et al., \textit{Immunologic Studies of Subjects with Asthma Exposed to Formaldehyde and Urea-Formaldehyde Foam Insulation (UFFI) Off Products}, 79 J. OF ALLERGY & CLINICAL IMMUNOLOGY 797 (1987).
  \item \textsuperscript{325} Interview with Dr. Dean Hashimoto, Asst. Professor, Boston College Law School (Mar. 15, 1992).
  \item \textsuperscript{326} See \textbf{HENNEKENS} \& \textbf{BURING}, supra note 109, at 43.
  \item \textsuperscript{327} See generally id.
  \item \textsuperscript{328} Id.
  \item \textsuperscript{329} See id.
  \item \textsuperscript{330} See \textit{In re “Agent Orange” Prod. Liab. Litig.}, 611 F. Supp. 1239, 1243 (E.D.N.Y. 1985), aff’d, 818 F.2d 187 (2d Cir. 1987).
\end{itemize}
the weight of epidemiological studies. This conclusion is ironic because a plaintiff can only prove legal causation by showing that a particular chemical more likely than not caused his or her particular injury—epidemiological studies can never show causation for a specific individual. An examination of an individual's immune system, however, can at least show that an extended exposure to a chemical harmed the individual. This is an advantage that T-cell studies have over other kinds of studies, including, in vivo studies, in vitro studies, case reports, and epidemiological studies. Furthermore, if a plaintiff can eliminate the possibility that a source other than the defendant's chemical caused the plaintiff's harm, then the plaintiff can prove that the only identifiable chemical to cause his or her injury must have been the defendant's chemical.

Although some courts have contended that most medical societies do not accept the value of clinical ecology, which T-cell studies are derived from, T-cells are undisputably an essential part of the immune system. The immune system protects our bodies from disease and T-cells are an important mechanism in initiating an immune response, attacking antigens, and regulating the immune response. Tests showing abnormal T-cell ratios and numbers, therefore, indicate that an individual's immune system is damaged.

An immunologist can perform a number of T-cell tests to determine an individual's immune system damage. The results of a plaintiff's T-cell tests are then compared to established normal ranges of T-cell numbers. From such a comparison, an immunologist can determine the extent of the T-cell damage that is present.

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331 See id. at 1239, 1250, 1263.
332 Dore, supra note 51, at 434.
335 See Callahan, supra note 1, at 639; Dangel, supra note 7, at 174–78; Brennan, supra note 1, at 507.
336 See Levin & Byers, supra note 15, at 673–76.
338 See Marrack & Kappler, supra note 16, at 36, 45.
340 Id. at 74–76.
341 Id.
342 SELL, supra note 65, at 218–20.
343 See Levin & Byers, supra note 15, at 672.
344 Levin & Byers, supra note 88, at 8.
345 Id.
T-cell damage can vary from chemically induced immune dysregulation, chemically induced AIDS, and cancer. There is also the possibility that present immune dysregulation may get progressively worse and lead to chemically induced AIDS or cancer.

Once T-cell test results show an injury to the plaintiff, the next step is to determine the possible sources of the damage. One way is to test the memory cells to show individual causation. Because memory cells respond more quickly after a first attack by an antigen, scientists can expose a sample of an individual’s memory cells to a small amount of the toxin and examine the reaction rate of the memory cells. If that particular plaintiff has been in contact with the toxin before, the memory cells will react rapidly. If a plaintiff’s memory cells show a fast reaction to a suspected injury-causing toxin, the reaction is analogous to a fingerprint on the smoking gun.

The use of the memory cell test is therefore useful in mitigating the indeterminate plaintiff and the indeterminate defendant problem. The test diminishes the indeterminate plaintiff problem by showing that the plaintiff has been exposed to that chemical before. To lessen the indeterminate defendant problem, an immunologist can test the memory cells with other chemicals. If the plaintiff’s memory cells only react to or react the quickest to the defendant’s chemical, such a result would indicate that the cause of the plaintiff’s injury is the defendant’s chemical. Not only is this evidence useful for a plaintiff, but defendants may also utilize this test to show that no injury is present in the plaintiff or that the plaintiff was not even exposed to the defendant’s chemical.

The T-cells are thus a potential basis for a test to establish particularistic evidence of causation for both plaintiffs and defendants. Plaintiffs can use this test to show that they were exposed to the


347 See Levin & Byers, supra note 88, at 9.
348 See, e.g., Elam, 765 S.W.2d at 83.
349 See id.
350 See generally SELL, supra note 65, at 215–18.
351 See generally id.
352 See id.
353 See generally id.
354 See supra notes 37–45 and accompanying text.
355 See generally Callahan, supra note 1, at 616–17.
356 See id. at 612.
357 See generally id. at 612, 616–17.
defendant's chemical and that they were injured. Defendants can also use the test to prove the opposite conclusion. Because most courts still rely on some form of particularistic evidence and the strong version of the preponderance rule requires it, T-cell tests for the individual plaintiff are a potential test for scientists, plaintiffs, and defendants to develop.

D. The Integrated Use of T-Cell Epidemiological Studies and Individual T-Cell Tests

Some courts require both epidemiological and particularistic evidence to prove causation. This requirement, referred to as the strong version of the preponderance rule demands particularistic evidence regarding the specific plaintiff and epidemiological evidence relating to proximate cause and the level of risk to which the defendant subjected the plaintiff. By providing both kinds of evidence, litigants come close to proving or disproving traditional legal causation. Although scientific evidence will never fully meet the demand of traditional legal causation, the use of both epidemiological and particularistic evidence allows each kind of evidence to overcome some of the limits of the other. The development and use of T-cell studies as a basis for epidemiological and particularistic evidence could provide strong causal evidence for a plaintiff or defendant.

For example, a plaintiff develops cancer after chronic exposure to formaldehyde insulation and sues the manufacturer under a products liability claim. An immunologist could analyze epidemiological studies that compare formaldehyde exposure and immune system damage. The immunologist could then testify that a biologic relationship exists between formaldehyde exposure and immune damage and that this immune damage led to cancer. Admittedly, not many scientists have conducted epidemiological studies comparing a type of exposure and T-cell abnormalities. These kind of studies, however, are a potential for the scientific community to research and develop and litigants to consider and use.

Next, an immunologist could perform a T-cell test to determine the plaintiff's T-cell absolute numbers and ratios. The immunolo-

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358 See supra notes 224-308 and accompanying text.
359 See supra notes 60-62 and accompanying text.
360 See supra notes 224-93 and accompanying text.
361 See Pross, supra note 324, at 797. In the study, only small, significant immunologic changes occurred after short-term exposure to formaldehyde. Id. at 803, 808.
363 See supra notes 80-99 and accompanying text.
gist could then compare the plaintiff’s test results with epidemiological studies or established normal T-cell ranges to show immune system damage. An immunologist could also perform a mitogen challenge test to determine how strongly the plaintiff’s immune system is working. If the immune system’s attack is weak, the immune system is damaged. Finally, a memory cell test could provide evidence of exposure to formaldehyde, thus eliminating the defendant and plaintiff indeterminacy problems.

This example is similar to the evidence that Dr. Levin, the Centers for Disease Control (CDC), and the Harvard School of Public Health produced for the Woburn plaintiffs in Anderson v. W.R. Grace & Co. Although a T-cell epidemiological study was not conducted, Harvard and the CDC conducted an epidemiological study of childhood leukemia incidence in the Woburn area. This evidence complemented Dr. Levin’s survey of immune damage in the plaintiffs’ families. The combined studies showed that PCE and TCE exposure caused immune damage and such immune damage led to leukemia in children. Particularistic and epidemiological evidence of T-cell damage can therefore lead to further inferences of a causal relationship between chemical exposure and diseases other than immune damage.

Although many experts believe that epidemiological studies are the strongest evidence available in proving causation in toxic torts, critics and supporters agree that an epidemiological study alone only can show the probability that the defendant’s chemical caused the plaintiff’s injury, or the degree of risk that the defendant subjected the plaintiff. Litigants also must utilize particularistic evidence to show that this particular defendant did or did not cause this particular plaintiff’s injury. T-cell studies can accomplish both.

364 See Levin & Byers, supra 88, at 8.
365 See Elam, 765 S.W.2d at 99.
366 See id.
367 See supra notes 37–45 and accompanying text.
369 See supra notes 280–82 and accompanying text.
370 See supra notes 276–78 and accompanying text.
371 See generally id.
372 See Callahan, supra note 1, at 626–27; Firak, supra note 8, at 315; McElveen & Eddy, supra note 139, at 43.
373 See Callahan, supra note 1, at 630; Gold, supra note 180, at 379–86. Although in toxic tort cases, courts should utilize statistics to determine the burden of proof, courts mistakenly use statistics to determine whether a plaintiff has met the preponderance standard. Id. Instead, the jury should make this decision by taking all the evidence into consideration. Id.
Although some courts now allow plaintiffs to prove a weak version of the preponderance rule instead of the traditional strong version of the preponderance rule, courts that accept the validity of T-cell studies do not have to replace the strong version of the preponderance rule for a weaker version. T-cell studies can be a means for a plaintiff or a defendant to provide a court with both particularistic and epidemiological evidence of whether or not causation exists.

VIII. CONCLUSION

Although clinical ecology has been criticized for its lack of foundation in science, it is an area of medicine that requires further study. Scientific knowledge has not developed to the extent that one can know how exposure to various chemicals affects the human body. It does make sense, however, that exposure to deleterious chemicals would have an adverse affect on the human immune system. Consequently, the development of T-cell studies is necessary to understand exactly how chemicals can damage the immune system. Once this is determined, T-cell studies could provide powerful causal evidence of a chemical's risk to a population and the source of a particular plaintiff's serious or fatal injury.

374 See Callahan, supra note 1, at 611.