NO. 04-99-00924-CV

IN THE COURT OF APPEALS
FOR THE FOURTH SUPREME JUDICIAL DISTRICT OF TEXAS
AT SAN ANTONIO, TEXAS

MISSOURI PACIFIC RAILROAD COMPANY d/b/a UNION PACIFIC RAILROAD COMPANY

Appellant

vs.

CENOBIO E. NAVARRO
Representative of the Estate of Manuela Navarro

Appellee

BRIEF OF AMICI CURIAE

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Appellant: Missouri Pacific Railroad Company d/b/a Union Pacific Railroad Company

Appellee: Cenobio E. Navarro, Representative of the Estate of Manuela Navarro (Mrs. Navarro, the plaintiff in this case, died during the trial of this case; Cenobio Navarro is her widower)

Amici Curiae in Support of Appellant: Marcia Angell, Philippe Baveye, Louis Anthony Cox, Jr., Leonard D. Hamilton, Ronald Hart, Clark W. Heath, Dudley Herschbach, Steven H. Lamm, Lee Loevinger, Rodney Nichols, Sally L. Satel, Barry H. Smith, James D. Watson and Richard Wilson

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Appellee

AMICUS DISCLOSURE

This brief is filed on behalf of *amici curiae* Marcia Angell, Philippe Baveye, Louis Anthony Cox, Jr., Leonard D. Hamilton, Ronald Hart, Clark W. Heath, Dudley Herschbach, Steven H. Lamm, Lee Loevinger, Rodney Nichols, Sally L. Satel, Barry H. Smith, James D. Watson and Richard Wilson, and was prepared by Atlantic Legal Foundation. Atlantic Legal Foundation is a non-profit, public interest law firm. None of the parties provided any funding or other thing of value to the *amici* or to Atlantic Legal Foundation. None of the *amici* provided funds for the preparation of the brief.

REQUISITES OF BRIEF

Pursuant to Rule 38 of the Texas Rules of Appellate Procedure, *amici curiae* rely on the Statement of the Case, the Statement of the Issues Presented, and the Statement of Facts contained in Appellants' Brief.

RECORD REFERENCES

Reporter's Record: Record references are designated "1RR," "2RR," etc., for the trial volumes. The clerk's record was not available to *amici* during the preparation of this brief.

ISSUES PRESENTED

1) **Did the Trial Court err in admitting the opinion testimony of the Plaintiff's expert witnesses?**

2) **Was the evidence at trial insufficient as a matter of law to sustain the jury's verdict?**

This brief *amicus curiae* will address only Issue 1.

INTEREST OF *AMICI*

Amici have studied the issue of the role that scientific issues play in public affairs and in particular the way in which they can illuminate disputes between different persons or elements of society in the courts of law. *Amici* include physicians, chemists, physicists, epidemiologists, toxicologists, including two Nobel laureates in Medicine and Chemistry, and a former justice of the Minnesota Supreme Court who has actively followed and written extensively about the interface between law and science.[1] Several of the *amici* submitted a brief in the Supreme Court in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), the seminal case discussing the rule for admissibility of expert scientific evidence. *Amici* support the principles enunciated by the Supreme Court in that case, and by the Supreme Court of Texas in *E.I. du Pont de Nemours & Co. v. Robinson*, 923 S.W.2d 549

[1] The credentials of *amici* are set forth in the biographical addendum to this brief.

(Tex. 1995) and in *Merrell Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706 (Tex. 1997). They believe that those principles should have wide applicability at the interface between science and law and policy. *Amici* believe that the trial judge in this case allowed clearly incompetent and unreliable evidence as to causation proffered by plaintiff to go to the jury, thus abdicating the trial court's critical gatekeeping role.

SUMMARY OF ARGUMENT

The primary disputed issue in this case is whether there was any scientifically reliable evidence to support a finding that Manuela Navarro's bone marrow cancer was caused by any chemical components of diesel exhaust in the air at the Laredo rail yard and bridge facility.

In this case the plaintiff's experts concluded that diesel exhaust causes multiple myeloma. No reputable scientist has to our knowledge come to that conclusion or made that assertion in any published article, monograph, study or textbook. The fact is that the scientific community does not know what causes multiple myeloma. The notion that judicial liability can diverge from scientific knowledge was condemned in *Daubert v. Merrell Dow Phar., Inc.*, 509 U.S. 579 (1993) and in *Merrell Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706 (Tex. 1997), and other recent cases.

Amici submit that the methodology used by the plaintiff's experts in this case is not scientifically sound.

The problem starts with Frank Parker's speculative opinion as to Mrs. Navarro's exposure to diesel exhaust. Mr. Parker's estimate of exposure forms the underlying basis of

the opinions of the other plaintiff's experts that Mrs. Navarro's multiple myeloma was caused by diesel exhaust. Mr. Parker's estimate is contradicted by actual data collected at the Laredo rail yard; that data showed that railroad workers at that yard were not exposed to elevated levels of harmful chemicals in diesel exhaust (13RR 50). Mr. Parker's testimony that Mrs. Navarro was exposed to 128 to 233 micrograms per cubic meter ($\mu g/m^{3)}$ of diesel exhaust was a radical departure from his original estimate of exposure -- 7.2 $\mu g/m^{3-}$ which is close to the amount actually measured at the Laredo rail yard, and was arrived at by using exposure estimates for "hostlers" and "brakers," (who work outdoors in the rail yard) not clerks like Mrs. Navarro, who work primarily indoors, based solely on one table in a 1988 study of railroad workers, and by ignoring numerous other factors that would tend to reduce Mrs. Navarro's actual exposure.

The opinions of Dr. Dayal, an epidemiologist, Dr. Frank Gardner, an oncologist, and Dr. Marvin Legator, a toxicologist, should not have been admitted in evidence because: (1) they were based upon Frank Parker's scientifically inaccurate exposure estimate; (2) they were not supported by scientifically reliable methodology; (3) they were not supported by independent scientific research; and (4) they were not supported by any scientifically sound data, theory, or methodology.

Plaintiff did not satisfy his burden of meeting the admissibility standards set forth in *Robinson* and *Havner*. If the trial court had applied the governing Texas standards for admissibility of expert testimony, it would have excluded plaintiff's unreliable causation testimony.

FACTS

Amici will not extensively recite the facts, and respectfully refer the Court to the brief of appellant for a detailed recitation of the factual background.

Manuela Navarro performed different functions -- as messenger, way bill clerk, yard clerk, janitor, demurrage clerk, etc. for Union Pacific for twenty years as a part-time employee; she worked part of the time outside, either in the Laredo rail yard or as a messenger, and part of the time in the freight office.

In April, 1994, Mrs. Navarro was diagnosed with multiple myeloma, a rare cancer of the bone marrow. Mrs. Navarro's had a family history of and she was diagnosed with diabetes in 1988.

After her diagnosis of multiple myeloma, Mrs. Navarro sued, alleging that her cancer had been caused either by unknown hazardous chemical residues which originated in empty tank cars or else by diesel exhaust in the air at the Laredo rail yard, or both. No evidence was offered as to exposure to hazardous chemicals, and this theory was not submitted to the jury.

Plaintiff's "experts" -- Frank Parker, an industrial hygienist, Dr. Hari Dayal, an epidemiologist, Dr. Frank Gardner, an oncologist, and Dr. Marvin Legator, a toxicologist -- never had done any research about diesel exhaust or its relationship to multiple myeloma before they were retained for this lawsuit.

ARGUMENT AND AUTHORITIES

ISSUE NUMBER 1

The Trial Court Erred in Admitting the Opinion Testimony of Plaintiff's Expert Witnesses Because Their Methodologies and Data Were Scientifically Unreliable

A. The Criteria for Admissibility of Scientific Evidence on Medical Causation

The courts of Texas and other jurisdictions have, over the last several years, focussed on the need for scientifically reliable expert testimony; this concern has been expressed in *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993) and its progeny in the federal courts and in *E.I. du Pont de Nemours & Co. v. Robinson*, 923 S.W.2d 549 (Tex. 1995) and *Merrell Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706 (Tex. 1997) in this state's highest court. This case, like *Daubert*, *Robinson*, and *Havner* concerns a "novel" toxic tort theory[2], and thus the trial judge's gatekeeping role is especially critical. The trial judge in this case did not properly perform that gatekeeping role by critically evaluating the methods and opinions offered by plaintiff's experts.

In *Robinson*, the court interpreted Texas Rule of Evidence 702 to require the proponent to show that an expert's testimony "is based upon a reliable foundation." *Robinson*, 923 S.W.2d at 556. *Robinson*'s reliability requirement for "scientific knowledge," the Texas Supreme Court follows the United States Supreme Court's approach in *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993) and its progeny. The *Robinson* court, like the U.S. Supreme Court, required that expert witnesses use the same methods and

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[2] It is novel because the theory of causation proposed by plaintiff's expert witnesses has not been advanced in the scientific literature, and has not been tested (or, in *Daubert*'s terminology subject to "falsification" by independent experts in the relevant fields.

procedures in arriving at their conclusions in the context of litigation as scientists do in their academic and clinical work:

> Scientific evidence which is not grounded "in the methods and procedures of science" is no more than "subjective belief or unsupported speculation." *Daubert*, 509 U.S. at 590, 113 S.Ct. at 2795. Unreliable evidence is of no assistance to the trier of fact and is therefore inadmissible under Rule 702.

Robinson, 923 S.W.2d at 557.

The *Robinson* court listed several factors for a trial court to consider in evaluating the scientific reliability of the proffered expert testimony: 1) the extent to which the theory has been or can be tested; 2) the extent to which the technique relies upon the subjective interpretation of the expert; 3) whether the theory has been subjected to peer review and/or publication; 4) the technique's potential rate of error; 5) whether the underlying theory or technique has been generally accepted as valid by the relevant scientific community; 6) the nonjudicial uses which have been made of the theory or technique. *Id.* at 557. The *Robinson* court made clear that "the proponent [of the testimony] bears the burden of demonstrating its admissibility." *Robinson*, 923 S.W.2d at 557. The court held that the trial court in that case properly excluded the causation expert's testimony because it was not scientifically reliable.

In *Merrell Dow Pharmaceuticals, Inc. v. Havner*, 953 S.W.2d 706 (Tex. 1997), the Texas Supreme Court reiterated several key points from *Robinson*: Under Rule 702, which governs the admissibility of experts, "[r]eliability is determined by looking at numerous factors including those set forth in *Robinson* and *Daubert*." *Id.* at 712. A trial court may not simply accept the "bare opinion" of the expert, but must consider "[t]he substance of the

testimony." *Id.* at 711; "[t]here must be objective, independent validation of the expert's methodology." *Id.* at 712. *Havner* requires that "[t]he underlying data should be independently evaluated in determining if the opinion itself is reliable." *Id.* at 713 and "[i]f the foundational data underlying opinion testimony are unreliable, an expert will not be permitted to base an opinion on that data because any opinion drawn from that data is likewise unreliable." *Id.* at 713-714. Moreover,

> [A]n expert's testimony is unreliable even when the underlying data are sound if the expert draws conclusions from that data based on flawed methodology. A flaw in the expert's reasoning from the data may render reliance on a study unreasonable and render the inferences drawn therefrom dubious. Under that circumstance, the expert's scientific testimony is unreliable and, legally, no evidence.

Id.

In toxic tort cases the plaintiff must show both general and specific causation. *See Havner*, 953 S.W.2d at 714-18. "General causation is whether a substance is capable of causing a particular injury or condition in the general population, while specific causation is whether a substance caused a particular individual's injury." *Id.* at 714. "Specific causation" is proof that the substance in fact caused a disease in this instance; in considering specific causation, it is essential to know the level and duration of the individual's exposure to the specific chemical. *Havner*, 953 S.W.2d at 720.

While a plaintiff need not prove to the trial court that his or her experts' opinions were credible or their conclusions correct, the plaintiff is required to prove that the experts' methodology and data were scientifically reliable and relevant. *See Gen. Elec. Co. v. Joiner*,

522 U.S. 136 (1997), *Robinson*, 923 S.W.2d at 556-57; *Austin v. Kerr-McGee Refining Corp.*, 25 S.W.3d 280, 288 (Tex. App. – Texarkana 2000).

Plaintiff's expert testimony in this case was scientifically unreliable and therefore inadmissible as to all three inquiries: exposure levels and duration, general causation and specific causation. Plaintiff did not demonstrate, as required by applicable Texas Supreme Court teaching, that hypothesis proffered by plaintiff of a causal connection between diesel exhaust and multiple myeloma is based on reliable scientific data or methodology.

B. Causation

1. The Probability of Causation

The issue of assigning causation does not differ, in principle, whether it is medical causation or engineering causation. If one sees a car with a damaged fender with a dead body in the road just in front of it, it is usual to state that the cause of death was a car accident, although exceptions occur such as when the accident was contrived to cover up a murder). But when the effect follows the postulated cause by a year or more, there is great difficulty in making the connection. If the medical outcome was known to have a unique cause, again assigning causation would be easy. At issue in this case, however, is how we assign causality when the medical outcome (disease) in question can be caused by numerous possible alternative events. It is then not possible merely by diagnosis of the disease, however well done, to assign the origin of the disease to any particular cause. It is necessary to know the exposure to the pollutant that is the postulated cause and the effect exposure of the particular magnitude is known to cause the condition.

One of the main ways of determining causation is to study a group of people with the same disease, and endeavor to discover a common link between them that might explain the disease. This is the field called epidemiology. But it is not enough that there be a statistical association between the hypothesized cause and the disease. There are well defined scientific principles that are used to evaluate whether a statistical "association" that is found should be considered to be "causal." In epidemiological terminology, if the relative risk, or "Risk Ratio," is very large, there is a greater likelihood that a particular exposure causes a particular disease. *See* M. D. Green, D. M. Freedman, L. Gordis, *Reference Guide On Epidemiology*, in Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2ND ED. 2000) (HEREAFTER "REFERENCE MANUAL") at 394-395.

It would be inappropriate for a court to allow the introduction of "scientific" evidence on medical causation without evidence also being proffered on the principles themselves, the logic behind them, and the degree to which the proffered testimony satisfies the criteria of epidemiology with regard to causation.

Even if it is accepted that a particular agent or mixture of agents can cause a particular disease, it does not follow that the inverse is true: that the particular disease is always caused by that particular agent. In deciding upon causation, therefore, it is necessary to consider the relative roles of all possible causes of the disease in question, even if some of those causes are unknown. This procedure can be bypassed logically only if there is evidence that the only possible cause of the disease is the one being considered, and no cases of the disease have ever appeared in the absence of this specific cause.

In the absence of such evidence of specificity, it is well accepted that some estimate of the relative probabilities must be made. The Probability of Causation can be defined as the probability that the medical outcome (disease) in question is caused by exposure to the particular substance. Then:[3]

$$POC = \frac{\text{The risk that the exposure (Es) in question can cause the disease}}{\text{The sum of the risks that anything, including unknown causes, caused the disease}}$$ [4]

From this simple formula it can be seen at once that POC is only greater than 50% if the substance of concern at least doubles the risk (Risk Ratio > 2). If the "background' risk is a sum of independent risks of exposures, E_t, to many substances t, then this follows at once from the theorem, attributed to the Reverend Bayes in the 18th century, that describes how to change a probability when new information is available. Inserting E_s and O instead of the variables used in the textbooks:

$$P(E_s|O) = \frac{P(E_s)P(O|E_s)}{\sum P(E_t)P(O|E_t)]}$$

(where the sum is taken from i=1 to k is over all possible substances (t))

The denominator in the POC equation can often be taken as the incidence of the disease in question in a population that is similar in all respects except the exposure, E_s, to

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[3] Risk analysts often calculate a different but related quantity: the "Risk" that a person can contract the disease from the exposure (or probability that a previously healthy person will contract the disease).

[4] Mettler, F.A. and Upton, A.C., eds., MEDICAL EFFECTS OF IONIZING RADIATION 350-372 (2nd ed. 1995), W.B. Saunders, "Report of the National Institutes of Health Working Group to Develop Radioepidemiological Tables," NIH Publication No. 85-274, (Washington, D.C., U.S., Gov't Printing Office). This follows at once from "Bayes' rule" which is in Chapter 1 of several statistics texts, e.g., Theorem 1.17, in R.E. Walpole and R.H. Meyers, PROBABILITY AND STATISTICS FOR ENGINEERS AND SCIENTISTS (4th ed.).

the individual of concern.[5] Then this can usually be reduced to the equation:

$$POC = R/(P + R)$$

where P in the above equation is then is the *incidence* of myeloma in the population of concern. There are a few caveats: statisticians have pointed out that when the calculated risk of developing cancer is zero or negative (*i.e.* the "pollutant" has a beneficial effect on health), the right hand side of the equation becomes negative, and conceptually one cannot have a negative probability. Thus the equation must be limited to situations, such as this case, where the risk is assumed to be positive. Secondly, a problem arises when the risk of the developing the disease is proportional to the product of the exposures to two different substances -- such as asbestos and cigarettes. In such a case, the sum of POC for asbestos and POC for cigarettes can exceed unity for asbestos workers who smoke cigarettes. Each POC is clearly too high. Many scientists prefer to use the expression "Assigned Share" to the above equation to emphasize that its derivation as a Probability of Causation involves these approximations and limitations.[6]

2. General and Specific Causation

Two additional concepts to aid in estimating POC: the concepts of general causation and specific causation. As stated by the Texas Supreme Court: "General causation is whether a substance is capable of causing a particular injury or condition in the general

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[5] For example POC for an asbestos worker will be different for smokers and non smokers. The appropriate incidence must be chosen.

[6] These assumptions are discussed in a formal statistical manner by Lagatos, S.W, and Mosteller, F. "Assigned Shares in Compensation for Radiation Related Cancer" *Risk Analysis* 6:345-380 (1986); Cox, L.A., "Statistical Issues in the Estimation of Assigned Shares for Carcinogenesis Liability," *Risk Analysis* 7:71-80 (1987); Robins, J., and Greenland, S., "The Probability of Causation Under a Stochastic Model for Individual Risk," *Biometrics* 45:1125-1138 (1989).

population, while specific causation is whether a substance caused a particular individual's injury."[7] General causation can be seen as a statement that the numerator in this equation, the risk, can be shown to be other than zero.

Specific causation is a statement that the numerator in the equation (exposure of the particular plaintiff) is large enough to be of importance (in legal situations usually greater than the denominator).

a. General Causation

It is not possible to discuss the attributes of causality without first showing a clear association between exposure and symptoms. There are many ways of discussing the attributes of an association that lead scientists to assign causality. The most well known of these, and the ones particularly often quoted in the courtroom, are from the address of Sir Austen Bradford Hill to the Royal Society of Statistical Medicine in 1965, A.B. Hill, "The Environment and Diseases: Association and Causation," 58 *Proc. Royal Soc. Med., Sec. Occup. Med.* 295 (1965)[8]. It is not possible to discuss the attributes of causality without first showing a clear association between exposure and symptoms. Moreover, even if it is accepted that a particular agent or mixture of agents can cause a particular disease, and thereby satisfies the medical requirement of *general* causation, it does not follow that the

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[7] *See Havner*, in which the Supreme Court of Texas reversed a judgment in favor of the Plaintiff and Court found that the Plaintiff had presented no reliable evidence of causation. The Court extended Daubert and Robinson to the "no evidence" review process in order to reach its result.

[8] Hill proposed a list of "attributes" of the association to be considered in evaluating causation: 1. Strength; 2. Consistency; 3. Specificity; 4. Temporality; 5. Biological gradient or dose response relationship; 6. Plausibility; 7. Coherence; 8. Experiment; and 9. Analogy. Hill emphasized that no one principle should be governing, but all should be considered. Earlier, Koch and Henle had proposed a similar series of criteria for making an epidemiological assessment of causation, known as "Koch's postulates": 1. Strength of association; 2. Temporal relationship; 3. Consistency of association; 4. Biologic plausibility (coherence with existing knowledge); 5. Alternative explanations; 6. Specificity of the association; 7. Dose-response relationship. Any person who claims to be an "expert" on medical causation should be familiar with these principles and should be able to demonstrate how his claim of causality fits each of them. Plaintiff's experts were not able to justify their claims of causality under these criteria.

inverse is true: that the particular disease is always caused by that particular agent. *Specific* causation may be lacking. In this case, even if chemical exposure were known to sometimes cause certain neurological symptoms, it would not necessarily follow that observation of these symptoms in a particular patient implies that they were caused by any particular chemical exposure. In deciding upon causation, therefore, it is necessary to consider the relative roles of all possible causes of the disease in question, even if some of those causes are unknown. This procedure can be bypassed logically only if there is evidence that the only possible cause of the disease is the one being considered, and no cases of the disease have ever appeared in the absence of this specific cause. That is manifestly not the case with Mrs. Navarro's disease, multiple myeloma, which is "associated" with many factors, including some medical conditions from which Mrs. Navarro's was known to suffer, such as diabetes.[9]

In the context of this case, the issue is: "has diesel exhaust been shown to cause multiple myeloma in any cohort or group in the past?" *Amici* believe that the answer is "No," and the case for the plaintiff must fail.

The hazards of diesel exhaust have been studied intensively over a period of many years. No recognized independent expert or scientific group has concluded that diesel exhaust causes multiple myeloma or leukemia. For example, the United States

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[9] Mrs. Navarro had a history of diabetes, which Boffetta (Boffetta, P., *A Case-Control Study of Multiple Myeloma Nested in the American Cancer Society Prospective Study* (1989) (hereafter "Boffetta") and Flodin, U., *et al.*, "Multiple Myeloma and Engine Exhausts, Fresh Wood, and Creosote: A Case-Referent Study," *Am. J. Ind. Med.* 12:519-529 (1987) (hereafter "Flodin") state (in studies plaintiff relies upon) is a risk factor associated with multiple myeloma (T. 339, 390) and there was a family history of diabetes (T. 252; 1999 Depo. 99; Tula Depo. 39). Plaintiff's experts made no attempt to rule out diabetes as the cause of Mrs. Navarro's cancer. *See Robinson*, 923 S.W.2d at 558 (expert must rule out other possible causes in toxic tort or his testimony is inadmissible); *see also Austin v. Kerr-McGee Refining Corp.*, 25 S.W.3d 280, 292 (Tex. App. – Texarkana 2000, n.p.h.).

Environmental Protection Agency, an government organization that has a mandate to protect public health, and which would be very unlikely to ignore such information, produced a draft "Health Assessment Document for Diesel Exhaust" EPA/600/8-90/057E, July 2000.[10] Chapter 7 of this draft discusses the epidemiological studies of the carcinogenicity of exposure to diesel exhaust and has "a detailed review of lung cancer studies." Although EPA reports that "Flodin, *et al.* (1987) observed an increased risk for multiple myeloma," the EPA study goes on to state that "because evidence for . . . lymphopohemapoetic cancer (a term that includes multiple myeloma) was found to be equivocal, details of these studies are not presented here." Thus, it is clear that EPA is not unaware of the relevant studies. Those studies, (including Boffetta, on which Dr. Dayal relies) were reviewed in the earlier 1994 edition of the EPA Health Assessment Document. The EPA did not include diesel exhaust as a potential cause of multiple myeloma.

Amici also note that the International Agency for Research on Cancer (IARC) reviewed diesel exhaust in 1989 and did not identify diesel exhaust as a cause of multiple myeloma.[11] This volume also mentions the Boffetta study. Further, the most recent issue of "CA - A Cancer Journal for Physicians" contains an article on diesel exhaust[12]. While not mentioning diesel exhaust as a possible cause of multiple myeloma, this article refers to the Boffetta study cited by plaintiff's experts, and to a quite recent paper by Boffetta and

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[10] One component of air pollution - benzene - has been shown to cause leukemia and small amounts of benzene are undoubtedly present in diesel, as well as other exhausts. But the small amount makes the risk (and the POC) very small.

[11] International Agency for Research on Cancer: *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 45. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels.* International Agency for Research on Cancer (Lyon, France 1989) and International Agency for Research on Cancer: *IARC Mongraphs on the Evaluation of Carcinogenic Risks to Humans. Volume 46. Diesel and Gasoline Engine Exhaust and Some Nitroarenes.* International Agency for Research on Cancer (Lyon, France 1989).

[12] Frumkin, H. and Thun, M.J., "Diesel Exhaust," *Cancer Journal for Physicians* 51(3):193-198 (2001).

Silverman,[13] which discusses the possible association of diesel exhaust and bladder cancer, but not with multiple myeloma. Thus it is clear that it is not generally accepted that diesel exhaust causes multiple myeloma. While "general acceptance" is not a definitive requirement under *Daubert*, *Robinson* or *Havner*, it is one of several criteria that a court may use in establishing admissibility. In this case there not only is there no general acceptance, but there is no recognized scientific body, such as the IARC or the U.S. EPA that finds an association; indeed there is general belief that there is no statistically significant association.[14]

In general, any good scientist discussing a subject, in this case causation, will look at the reviews by other experts in the field and use these reviews as a starting point: Does he or she have a different opinion from other scientists? What is that difference? What, if any, are the new data that warrant the difference of opinion? However, that is not the procedure adopted by plaintiff's principal causation witness, Dr. Dayal. Dr. Dayal did not begin by discussing the general scientific opinion. Instead Dr. Dayal relied on several studies, most of which did not directly associate diesel exhaust and myeloma, but only associated the occupations of railroad workers and truck drivers with myeloma. He failed to discuss the possible confounding association with the many other factors in the occupations examined. In one of these, Boffetta, the authors of the study explicitly measured diesel exhaust and found no association of myeloma with diesel exhaust. Dr. Dayal had a contrived explanation for this, which was only extracted under cross- examination. Only in one study, Flodin, *et al.*, which was mentioned, but discounted, by the United States Environmental Protection

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[13] Boffetta, P., and Silverman, D.T., "A Meta Analysis of Bladder Cancer and Diesel Exhaust Exposure," *Epidemiology* 12:125-130 (2001).

[14] Although some epidemiological investigations of diesel fumes and disease in indoor air settings purport to show an association between inhaled diesel fumes and health effects, none has had sufficient data or experimental design to support plaintiff's claim that diesel fumes caused Mrs. Navarro's multiple myeloma.

Agency, was there a suggestion of a direct association between multiple myeloma and diesel exhaust. In the view of *amici*, Dr. Dayal's testimony not only fails to demonstrate general causation, but flies in the face of established procedures for doing so.

Multiple myeloma is a relatively rare form of bone marrow cancer "with an elusive etiology." *See, e.g.* Peter H. Wiernik, NEOPLASTIC DISEASES, ch. 24 (T. 537). Although the etiology of multiple myeloma is unknown[15], epidemiological studies have identified potential risk factors for multiple myeloma, such as viral infections, a history of diabetes, a history of muskelosketetal conditions, and cigarette smoking.[16] Most multiple myeloma cases are idiopathic, *i.e.*, with no known causes. Numerous attempts to find a scientifically reliable risk association between multiple myeloma and various substances, including diesel exhaust, have been unsuccessful (T. 341, 401-407, 470-477, 598, 603).[17]

There was no scientific evidence in this case (and no scientific evidence has been published since) that any research scientist has ever concluded that diesel exhaust causes multiple myeloma. Plaintiff's experts had no opinions as to what levels of diesel exhaust exposure causes multiple myeloma (8RR 92-93; 10RR 39-40, 221),[18] and for that reason their

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[15] Wyngaarden, J.B., Smith, L.H. and Bennett, J.C., CECIL TEXTBOOK OF MEDICINE (19th ed. 1992); Wilson, J.D., Braunwald, E., Isselbacher, K.J., Peterdorf, R.G., Martin, J.B., Fauci, A.S. and Root, R.K., HARRISON'S PRINCIPLES OF INTERNAL MEDICINE (12th ed. 1991).

[16] *See* Blattner, W.A., "Multiple Myeloma and Cacroglobulinemia," in CANCER EPIDEMIOLOGY AND PREVENTION (Schottenfeld D. and Fraumeni J.F., eds.(1982)); Flodin, U., Fredriksson, M. and Persson, B., "Multiple Myeloma and Engine Exhausts, Fresh Wood, and Creosote: a Case-Referent Study," *Am. J. Ind. Med.* 12:519-529 (1987); Doody, M.M., Linet, M.S., Glass, A.G., Friedman, G.D., Pottern, L.M., Boice, J.D. and Fraumeni, J.F., "Leukemia, Lymphoma and Multiple Myeloma Following Selected Medical Conditions, *Cancer Causes & Control* 3:449-456 (1992).

[17] Finding a scientifically reliable risk association through epidemiological research is only the starting point of establishing the scientific cause of a disease. *Havner*, 953 S.W.2d at 717-19.

[18] None of the plaintiff's experts in this case examined or treated Mrs. Navarro. None had ever conducted research into or investigated any association between diesel exhaust and multiple myeloma (6RR 166-167; 7RR 26-27; 8RR 86-87; 10RR 150). Their knowledge of the subject was obtained for litigation purposes, after they were retained as experts in this case. Thus their methodology and objectivity should be carefully scrutinized. *See Robinson*, 923 S.W.2d at 559 (the fact that research was conducted and the expert's opinion was formed for the purpose of litigation weighs against

opinions as to causation are not helpful. *See Mitchell v. Gencorp.Inc.*,165 F.3d 778, 781 (10th Cir. 1999); *National Bank of Commerce v. Assoc. Milk Producers, Inc.*, 22 F. Supp.2d 942, 983 (E.D. Ark. 1998).

Dr. Dayal relied on several studies, most of which did not directly associate diesel exhaust and myeloma, but only associated the occupations of railroad workers and truck drivers with myeloma. He failed to discuss the possible confounding association with the many other factors in the occupation. In one of these, (Boffetta) the authors of the study had explicitly measured diesel exhaust and found no association with diesel exhaust. Dr. Dayal had a contrived explanation for this which was only extracted under cross- examination. In only one study, Flodin, *et al.*, mentioned, and discounted, by the U.S. Environmental Protection Agency, was there a suggestion of a direct association between multiple myeloma and diesel exhaust. In the view of *amici*, Dr. Dayal's testimony not only fails to demonstrate general causation but flies in the face of established procedures for doing so.

It is well-established that the plaintiff in a toxic tort case must provide scientifically sound expert testimony that a specific chemical to which the plaintiff was exposed can cause the plaintiff's specific disease. *See Havner*, 953 S.W.2d at 714-717 (discussing general causation in toxic torts); *Austin*, 25 S.W.3d at 288-90; *Castellow v. Chevron USA*, 97 F. Supp.2d 780, 796-97 (S.D. Tex. 2000). In the present case, none of the plaintiff's experts identified a specific chemical component of diesel exhaust which has been found to cause multiple myeloma.

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objectively and admissibility); *Mitchell v. Gencorp*, 968 F. Supp. 592, 600 (D. Kan. 1997) (It is "significant that all of the plaintiff's experts developed their opinions expressly for the purpose of testifying. None of the witnesses has done any research on his theories outside the context of this lawsuit").

As noted previously, even if there were a statistical association between the hypothesized cause and the disease, that alone insufficient to find causation and there are well defined scientific principles that are used to evaluate whether a statistical "association" that is found should be considered to be "causal."

It would be inappropriate for a court to allow the introduction of "scientific" evidence on medical causation without evidence also being proffered on the principles themselves, the logic behind them, and the degree to which the proffered testimony satisfies the criteria of epidemiology with regard to causation.

b. Specific Causation

Even if it were demonstrated that a particular agent or mixture of agents can cause a particular disease (by demonstrating that it has done so in the past), and thereby satisfies the requirement of general causation, it does not follow that the inverse is true: that the particular disease is always caused by that particular agent. Specific causation may be lacking. In this case, even if diesel exhaust sometimes causes multiple myeloma (presumably at high exposures), it would not necessarily follow that observation of multiple myeloma in a particular patient implies causation in that particular case by diesel exhaust. In deciding upon causation, it is necessary to consider the relative roles of all possible causes of the disease in question, and to consider that there are causes which have not been identified. This procedure can be bypassed logically only if there is evidence that the only possible cause of the disease is the one being considered, and no cases of the disease have ever appeared in the absence of this specific one cause.

If general causation has been demonstrated, then, and only then, will evidence for exposure of the individual patient be significant. This evidence must not merely be evidence that the plaintiff has had some exposure, but must also include evidence that the exposure has been sufficient to make the Probability of Causation greater than 50%. Evidence that an exposure is greater than that allowed by some regulatory body is interesting, but by itself not conclusive. Regulatory bodies try to reduce risks to the point that the calculated risk of developing a disease such as cancer from an environmental exposure is less than 10^{-6} in a person's lifetime and to the point that the calculated risk of developing cancer from occupational exposures is about 10^{-4}. These risks of one in a million and one in ten thousand, respectively, are very much smaller than the overall risks of developing cancer from all causes, which are about one in four.[19]

Amici have examined the report and trial testimony of Dr. Dayal and find no evidence that the exposure was high enough to cause any disease. Thus any statement by Dr. Dayal that diesel exhaust was a cause of the plaintiff's myeloma is invalid and inadmissible.

There is no scientifically reliable evidence in this record of specific causation -- that Mrs. Navarro did, in fact, contract her multiple myeloma from exposure to components of diesel exhaust. As the court in *Havner* stated, when direct evidence of specific causation is not available in toxic tort cases:

> The finder of fact is asked to infer that because the risk is demonstrably greater in the general population due to exposure to the substance, the claimant's injury was more likely than not caused by that substance. Such a theory concedes that science cannot tell us what caused a particular plaintiff's injury. It is

[19] None of the regulatory agency levels referred to by Dr. Dayal was set to protect against multiple myeloma. The level was set for smell (odor threshold) and for other environmental effects.

> based on a policy decision that when the incidence of a disease or injury is sufficiently elevated due to exposure to a substance, someone who was exposed to that substance and exhibits the disease or injury can raise a fact question on causation.

953 S.W.2d at 715. In *Havner*, the Texas Supreme Court held that "epidemiological evidence must show that the risk of an injury or condition in the exposed population was more than double the risk in the unexposed or control population," *id.* at 716, to be a substitute for direct evidence of specific causation.[20]

In the present case, however, none of the plaintiff's experts cited a single epidemiological study in which the author concluded that he had found a scientifically reliable "doubling of the risk" association between diesel exhaust exposure and multiple myeloma with a confidence interval excluding 1.0. As the Texas Supreme Court said in *Havner*, 953 S.W.2d at 725, a confidence level "that included 1.0 or a lower number would be inconclusive and statistically insignificant".

The "doubling of the risk" surrogate for specific causation requires evidence that the level and duration of the plaintiff's exposure was the same as or greater than those in the relevant epidemiological studies. *Havner*, 953 S.W.2d. at 720. Mr. Parker's estimate of possible exposure provides no scientifically reliable evidence of Mrs. Navarro's actual exposure.

All of Mrs. Navarro's causation experts relied solely upon Frank Parker's exposure estimates in forming their opinions that Mrs. Navarro's multiple myeloma was caused by exposure to diesel exhaust (6RR 53). Because Mr. Parker's estimates were not based on

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[20] In *Havner*, the court stated that it was not holding that "a relative risk of more than 2.0 is a litmus test or that a single epidemiological test is legally sufficient evidence of causation. Other factors must be considered." 953 S.W.2d at 718. *See also* Taubes, "Epidemiology Faces Its Limits," *Science* 269:164-169 (1995). However, absent other evidence of causation, plaintiff's expert testimony is not based on reliable data.

sound scientific methodology, their conclusions as to specific causation are inadmissible. *See, e.g. Christopherson v. Allied-Signal Corp*., 939 F.2d 1106, 1114-15 (5th Cir. 1991) (*en banc*), *cert. denied*, 503 U.S. 912 (1992) (court properly excluded expert's causation opinion that was based on insufficient data regarding the dosage and duration of exposure to the harmful substance); *Edwards v. Safety-Kleen Corp.*, 61 F. Supp. 2d 1354, 1358 (S.D. Fla. 1999) (causation expert who relied on scientifically unreliable exposure estimates was "suspect" and testimony was inadmissible).

If there are other possible or plausible causes of the disease, then "the plaintiff must offer evidence excluding those causes with reasonable certainty." *Havner,* 953 S.W.2d at 720. Mrs. Navarro had a long history of diabetes, which Boffetta and Flodin state (in the same studies upon which the plaintiff relies) is a risk factor associated with multiple myeloma (T. 339, 390). Both of her parents and her two daughters also had diabetes and her mother died of lung cancer (T. 252; Tula Depo. 39). There is no evidence that any of the plaintiff's experts had made any attempt to rule out diabetes as the cause of Mrs. Navarro's cancer. *Robinson*, 923 S.W.2d at 558 (expert must rule out other possible causes in toxic tort or his testimony is inadmissible); *Austin*, 25 S.W.3d at 392.

Plaintiff's experts did not offer any direct evidence of specific causation, nor did they offer scientifically sound evidence of epidemiological significance.

C. The Testimony of Plaintiff's Experts Was Scientifically and Legally Deficient

1. Frank Parker

As the famed Swiss physician Paracelsus explained in the sixteenth century: "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy." Doull & Bruce, "Origin and Scope of Toxicology," in CASARETT & DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS (3d ed. 1986). Succinctly put, "[t]he dose makes the poison." *National Bank of Commerce v. Assoc. Milk Producers, Inc.*, 22 F. Supp.2d 942, 958 (E.D. Ark. 1998). As the court in *National Bank* noted, consistent with principles of modern toxicology, all chemical agents are harmful at some level, it is only a question of dose. *Id.* At 958. "Scientific knowledge of the harmful level of exposure to a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiff's burden in a toxic tort case." *Allen v. Pennsylvania Eng'g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996); *see also Mitchell v. Gencorp. Inc.*, 165 F.3d 778, 781-82 (10th Cir. 1999); *Castellow*, 97 F. Supp.2d 780, 782 (S.D. Tex. 2000) ("to be legally sufficient evidence, proof of causation requires a plaintiff to prove, at a minimum, exposure to the allegedly harmful substance at a level shown by scientifically reliable studies [to be] capable of causing the complained of ailment"); *Havner*, 953 S.W.2d at 720 (plaintiff in toxic tort case who relies upon epidemiological studies must show she "was exposed to the same substance, that the exposure or dose levels were comparable to or greater than those in the studies").

The initial problem with plaintiff's scientific "proof" of causation is that Frank Parker's opinion as to Mrs. Navarro's exposure to diesel exhaust was speculative and scientifically unsupported. Because Mr. Parker's estimate of Mrs. Navarro's exposure was seriously flawed, plaintiff's experts on causation did not have a sound basis for their testimony. This flaw is fatal to all of plaintiff's proffered expert testimony, for as plaintiff's trial counsel stated: "All of the medical experts that we have hired are basing their opinions and have so stated in their depositions upon [Mr. Parker's] information" (6RR 53).

Mr. Parker, an industrial hygienist, was the plaintiff's only expert on the dose and duration of Mrs. Navarro exposure to diesel exhaust (6RR 22, 53). He testified that it is essential to conduct air sampling at the work site, and to make calculations from that sampling (6RR 28-29).

Mr. Parker failed to employ the procedure he described and acknowledged was applicable. He did not conduct any air sampling at the Laredo rail yard (6RR 34-35, 6RR 180; 7RR 20) and he performed no calculations of Mrs. Navarro's possible worksite exposure to diesel exhaust (7RR 24). He did not know the conditions at the Laredo yard (7RR 46). In fact, he has never sampled any location for diesel exhaust. His first experience with diesel exhaust was as plaintiff's expert in this lawsuit (6RR 166-67).

Instead of using the actual measured quantity of diesel fumes or its components at the Laredo yard, Mr. Parker used exposure estimates for "hostlers" and "brakers," (who work outdoors in the rail yard) not clerks like Mrs. Navarro, who work primarily indoors, based on one table in an article by S. K. Hammond, *et al.*, "Markers of Exposure to Diesel Exhaust

and Cigarette Smoke in Railroad Workers" published in 1988 (6RR 39). However, Mr. Parker's estimate is contradicted by actual data collected at the Laredo rail yard. That data showed that railroad workers at that yard were not exposed to elevated levels of harmful chemicals in diesel exhaust (13RR 50).

Mr. Parker's testimony that Mrs. Navarro was exposed to 128 to 233 micrograms per cubic meter ($\mu g/m^{3)}$ of diesel exhaust was a radical departure from his original estimate of exposure -- 7.2 $\mu g/m^{3}$ – which is close to the amount actually measured at the Laredo rail yard. Parker conceded that he dramatically increased his estimate of Mrs. Navarro's exposure to diesel exhaust components from significantly less than 150 $\mu g/m^{3}$ or 50 $\mu g/m^{3)}$ to significantly above those levels (7RR 42). Mr. Parker did not perform any testing of the air at the Laredo facility for its diesel exhaust component volume to confirm the accuracy of his estimated exposure levels. Parker increased his exposure estimate based on several arbitrary assumptions.[21]

Parker's opinion is suspect because (a) he did not use the measured exposure level data that was available[22] and (b) he radically revised his estimate of exposure dosage upward.[23] *See Castellow*, 97 F. Supp.2d at 971.

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[21] Mr. Parker manipulated his original estimate, and arrived at a far higher figure. He decided that because Mrs. Navarro spent about one-third of her working time outside, her tasks were more like those of hostlers and brakers in the Hammond study, and he used Hammond's numbers for those full-time job titles instead of those for a clerk who at least sometimes worked part-time (6RR 36-37, 41,180). Hostlers and brakers, according to Hammond's study, spend their entire careers "working on or near operating trains" and have medium exposure to diesel exhaust (T. 5824). Parker did not know, at the time he formed his opinion, that hostlers spend all day moving diesel engines around a yard and shoveling sand into the sand hopper right behind the engine smokestack (6RR 189; 13RR 53-55).

[22] None of the actual measures of diesel exhaust components at the rail yard showed hazardous levels. When an expert's "estimate" varies from actual test results by 1,000 to 3,000 percent, there is a presumption that there is something wrong with the expert's method.

[23] In *Castellow*, the expert tripled his exposure estimate; in this case, Parker raised his exposure estimate from 7.2 to between 128 and 233 $\mu g/m^{3}$, multiples of almost 18 to more than 32.

The trial court abandoned its gatekeeping role in allowing Mr. Parker's speculative exposure estimate to be considered by the jury and to form the basis of the causation testimony of plaintiff's other experts.

2. Dr. Hari Dayal

Dr. Hari Dayal, an epidemiologist, testified that "based on epidemiological evidence" Mrs. Navarro's exposure to diesel exhaust as a railroad worker was a major factor in the causation of her multiple myeloma." (8RR 4-5, 28, 85).[24]

Dr. Dayal conceded that knowing the amount of an exposure is important to determining whether that exposure might have caused a disease (8RR 88), yet he admitted that he had "absolutely no idea" how much diesel exhaust to which Mrs. Navarro was exposed. Dr. Dayal further conceded that he did not know how much exposure to diesel exhaust was necessary to cause multiple myeloma. (8RR 93). This, in itself, is sufficient to impeach Dr. Dayal's causation conclusion.

Dr. Dayal relied primarily on the 1989 study by Boffetta. (8RR 37).[25] In this "cohort study," Boffetta found that <u>both</u> a history of diabetes and various occupations had an elevated association with multiple myeloma (T. 339-344). Boffetta found that a history of diabetes,

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[24] This lawsuit was the first time he had ever examined diesel exhaust exposure (8RR 86-87), and thus his testimony is suspect because his work on this subject was done expressly for purposes of litigation. *See Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 43 F.3d 1311 (9th Cir. 1994), the United States Court of Appeals for the Ninth Circuit, on remand, rejected the analysis of plaintiffs' experts because they "developed their opinions expressly for purposes of testifying." "That an expert testifies based on research he has conducted independent of the litigation provides important, objective proof that the research comports with the dictates of good science.... experts whose findings flow from existing research are less likely to have been biased toward a particular conclusion by the promise of remuneration; when an expert prepares reports and findings before being hired as a witness, that record will limit the degree to which he can tailor his testimony to serve a party's interests. *Id.* at 1317.

[25] Dr. Dayal testified that he based his causation opinions on only two studies (9RR 127). Significantly, he admitted that there are at least 14 other studies involving diesel exhaust and multiple myeloma that show no statistically significant increased risk. (9RR 85).

a disease from which Mrs. Navarro suffered, has an odds ratio of 2.0 to develop multiple myeloma and occupation as a farmer had a 2.7 odds ratio.[26]

Dr. Dayal testified that the deaths of three railroad workers' death from multiple myeloma was statistically significant because the overall study involved a large number of people (8RR 47-48). Boffetta declined to conclude that there was any statistically significant association between multiple myeloma and employment as a railroad worker because his case study included only three railroad workers who died from multiple myeloma. (T. 339; 8RR 115-118, 123). The authors of the study were unwilling to draw any causal conclusion from this finding, but instead urged caution in any interpretation "[g]iven the small number of exposed cases and controls in these occupations." Boffetta, *id.* at 558.

This discrepancy indicates that there is a lack of scientific "fit" between the Boffetta study and Dr. Dayal's conclusions. When the author of a scientific study asserts that his data is not statistically significant, that study does not support a testifying expert's assertion that this same data is statistically significant. *See Joiner*, 522 U.S. at 144 (when the study's authors were unwilling to say PCBs caused cancer, the study did not support the expert witness's opinion that PCBs caused cancer); *National Bank*, 22 F. Supp.2d at 978-79 (articles that declined to find statistical significance in their data could not support expert's assertions that aflatoxin can cause cancer of the larynx); *Savage v. Union Pacific R.R. Co.*, 67 F. Supp. 1021, 1027 (W.D. Ark. 1999).

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[26] The 1989 Boffetta study examined the relationship between multiple myeloma and exposure to diesel exhaust and found no statistically significant association. In Table III, the authors report 14 cases and 43 controls exposed to diesel exhaust leading to an odds ratio of 1.4 and a confidence interval from 0.7 to 2.7. Under generally accepted statistical principles, as recognized in *Havner*, since the confidence interval includes 1.0, this finding is not statistically significant and is inconclusive. *See Havner* at 725. Dr. Dayal admitted that the Boffetta study found that people who reported exposure to diesel exhaust had no statistically significant increased risk of developing multiple myeloma. (8RR 128).

Dr. Dayal also relied on a 1993 study by E. S. Hansen, *et al.*, "A Follow-Up Study on the Mortality of Truck Drivers," *The American Journal of Industrial Medicine* 23:811-821 (1993) (T. 410). Hansen found a statistically significant increase of multiple myeloma in truck drivers, but her findings, like Boffetta's, were based on a small sample -- just five deaths (T. 410; 8RR 148-149). Hansen, also, like Boffetta, acknowledged problems with the data, and declined to find any causal relationship between the occupation of truck driving or exposure to diesel exhaust and multiple myeloma (T. 410; 8RR 145-149).[27] The study by Hansen involved a cohort of 14,225 Danish truck drivers whose mortality from cancer was compared to that of a control cohort of Danish unskilled male laborers. As with the Boffetta study, Hansen was unwilling to reach a conclusion as to causation; she suggested that the result, based on five deaths, "may have been due to chance." (23 *American Journal of Industrial Medicine* at 818). Dr. Dayal provided no explanation for why he ignored that suggestion. The only inference that Hansen was willing to make from the finding was that, "[t]he possible relationship between multiple myeloma and the components of vehicle exhaust seems to be worth some attention in future epidemiological and laboratory investigations." (*id.*). Dr. Dayal admitted that he was disagreeing with Hansen about the meaning and significance of her findings (8RR 151-52) and he provided no explanation as to why he could infer from the Hansen study that there was not only a definite association, but actually a causal relationship. Dr. Dayal's reliance on the Hansen study is unwarranted for many of the same reasons as was his reliance on the Boffetta study.

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[27] Hansen concluded that "the observation of five deaths from multiple myeloma was unexpected, and although this finding is statistically significant, it may have been due to chance." (8RR 149).

Dr. Dayal also relied on the study by Flodin (T. 390; 8RR 70-75). The Flodin study combined occupational exposure to diesel exhaust and gasoline engine exhaust and found that there was about a 2.3 times greater likelihood of contracting multiple myeloma in persons so exposed, less than half the 5.1 increased risk for diabetics on medication) (8RR 72). The 95% confidence level included an interval between 1.0 to 4.9,[28] and Dr. Dayal admitted that it was only "almost" statistically significant (8RR 75).

Dr. Dayal did not even try to rule out alternative causes. He dismissed the possibility that Mrs. Navarro's diabetes caused her multiple myeloma (8RR 49-52), even though the 1989 Boffetta study on which he relied heavily had reported that previous history of diabetes was a risk factor "consistently suggested by the results of the analysis." (*see* Boffetta at 554).

Dr. Dayal acknowledged that epidemiological studies are not evidence that exposure causes the disease, that such studies merely find an association, a relationship (8RR 114)and that scientists must take a step further to investigate whether the relationship is due to chance or "confounding" (the effect of other exposures) (*id.*).[29]

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[28] A confidence interval range including 1.0 is not statistically significant. *See Havner, supra* and *Brock v. Merrell Dow Pharmaceuticals, Inc.*, 874 F.2d 307, 312-13 (5th Cir.1989) for a discussion of the distinction between relative risk and the confidence interval range.

[29] In a case such as this, in which the plaintiff alleges that an ailment was caused by a particular substance, the issue of causation also depends on the field of toxicology. The Federal Judicial Center's REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2nd ed. 2000) (hereafter "REFERENCE MANUAL") at 403 describes toxicology as an "age-old science." As the REFERENCE MANUAL notes, "Toxicological studies, by themselves, rarely offer direct evidence that a disease in an individual was caused by a chemical exposure. However, toxicology can provide scientific information regarding the increased risk of contracting a disease at any given dose and helps rule out other risk factors for the disease." *Id.* at 403 (emphasis supplied). Even if it is accepted that a particular agent or mixture of agents can cause a particular disease, it does not follow that the inverse is true: that the particular disease is always caused by that particular agent. In deciding upon causation, therefore, it is necessary to consider the relative roles of all possible causes of the disease in question. This procedure can be bypassed logically only if there is evidence that the only possible cause of the disease is the one being considered, and no cases of the disease have ever appeared in the absence of this specific cause. That is manifestly not the case with multiple myeloma, which is known to be associated with diabetes, among other conditions.

Dr. Dayal relied on only three of the 140 studies that he deemed relevant (9RR 85-87). Two of those articles found a heightened risk association between diabetes and multiple myeloma, yet Dr. Dayal never considered diabetes as the possible cause of Mrs. Navarro's cancer (9RR 35). Fourteen studies specifically found that there was no increase of risk relationship between diesel exhaust exposure and multiple myeloma (9RR 85-87). Dr. Dayal conceded that there were over a dozen studies showing no statistically significant association between diesel exhaust and multiple myeloma (9RR 86-87), but he ignored.

Even if there were a statistical association between the hypothesized cause and the disease, that is insufficient to find causation. There are well defined scientific principles that are used to evaluate whether a statistical "association" that is found should be considered to be "causal."[30] In epidemiological terminology, if the "Relative Risk" or "Risk Ratio," is very large, there is a greater likelihood that a particular exposure causes a particular disease.[31]

It is inappropriate for a court to admit "scientific" evidence on medical causation without evidence also being proffered on the principles themselves, the logic behind them, and the degree to which the proffered testimony satisfies the criteria of epidemiology with regard to causation. The trial court thus erred in admitting Dr. Dayal's testimony.

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[30] A.B. Hill, "The Environment and Diseases: Association and Causation," 58 *Proc. Royal Soc. Med., Sec. Occup. Med.* 295-300 (1965). *See* Note 8, *supra*. It appears that Dr. Dayal misunderstood that the Hill criteria should be used to analyze a collective body of epidemiological research, *see Havner* at 719 n. d. Dr. Dayal stated that he applied the Hill criteria to individual studies (9RR 127), but never explained how his incorrect analysis justified his final conclusion. He extrapolated from two isolated findings to reach an opinion as to general causation. Anyone who claims to be an "expert" on medical causation should be able to demonstrate how his claim as to causation fits each of the criteria. Dr. Dayal failed to do so.

[31] For a discussion of "odds ratio," "relative risk" and "attributable risk" *see* REFERENCE MANUAL at 348-352.

3. Dr. Frank Gardner

Dr. Frank Gardner, plaintiff's oncology expert, testified that diesel exhaust can cause multiple myeloma and that Mrs. Navarro's workplace exposure to diesel exhaust caused her to develop that disease (9RR 150). Dr. Gardner agreed that the dose level is important to the severity of health effects (10RR 134), but he admitted that (like Dr. Dayal) he had no knowledge as to what extent or concentration of diesel exhaust would cause multiple myeloma. (10RR 40-41). On this basis alone, his testimony should have been excluded. *See Robinson*, 923 S.W.2d at 559.

Dr. Gardner conducted no research on the carcinogenicity of diesel exhaust, but only purportedly reviewed the literature (10RR 150). He could not testify to any diesel exhaust concentration that is necessary for multiple myeloma to develop, and conceded that no dose-response relationship has been established (10RR 40-42). He did not know of any scientific studies that show that animals can contract multiple myeloma when exposed to diesel exhaust (11RR 131).

Dr. Gardner did not know of the mode of absorption of diesel fumes or its chemical components; he merely hypothesized that it might be possible for diesel exhaust particulates to enter bone marrow through inhalation, swallowing, or dermal absorption (10RR 68-78). He admitted, however, that this theory was "speculation" and that he did not know of any physical or chemical mechanism by which organic portions of diesel exhaust particles might be absorbed through the skin and into the bloodstream (10RR 91, 118, 121).[32]

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[32] The mode of absorption can be critical. Tthe skin, for example, is a remarkably effective barrier. Absorption through the skin is generally slow and readily detoxified.

Havner (953 S.W.2d at 729) made it clear that non-epidemiological evidence such as animal and cell biology, standing alone, cannot prove causation. Dr. Gardner admitted that he was not aware of any animal studies that showed a specific multiple myeloma response from diesel exhaust exposure. (10RR 131).[33] Moreover, to be useful in supporting a finding of causation, the expert must explain his reasons for extrapolating from *in vivo* animal studies or *in vitro* chemistry studies to the effect of a chemical on humans. *Id.* Dr. Gardner failed to explain how animal or *in vitro* studies supported his opinion as to causation in humans.[34]

Dr. Gardner's opinion did not follow from reasonable scientific methodology, it did not satisfy the *Havner* or *Robinson* standards, and it should not have been admitted.

4. Dr. Marvin Legator

Dr. Marvin Legator testified that in his opinion there is no "safe threshold" for any substance which is toxic (10RR 215). He testified that cancer is caused by a single cell mutation and "if you've had exposure to a carcinogenic substance and you come down with

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[33] Dr. Gardner referred to an *in vitro* study involving lymphocyte cells, but he admitted that he could not relate the cell abnormalities in that study to multiple myeloma. (10RR 49-50). He also admitted that he could not extrapolate from the chemical exposures in the study to diesel exhaust generally or to a specific analogous quantity of diesel exhaust involved in this case. (10RR 50). In *Havner*, 953 S.W.2d at 729, the court criticized the testimony of an expert who offered no explanation for extrapolating study dosages to humans.

[34] A test tube is an artificial environment quite unlike a human body. Unlike a test tube, the body has a metabolic process that quickly acts to remove toxic substances. Thus, whereas a toxic substance can linger indefinitely in a test tube, such a substance would be quickly detoxified and expelled from a living body by the body's metabolism. It is widely recognized that *in vitro* evidence is of limited utility of establishing causation in a living body "because the relevant chemical compound does not go through the metabolic process before affecting the culture." Sanders, "Scientific Validity, Admissibility and Mass Torts After Daubert," 78 Minn. L. Rev. 1387, 1409 (1994). It is therefore no surprise that scientists do not generally accept the proposition that *in vitro* test results can be directly extrapolated to a living body. "[I]n vitro animal test data are not relied upon by experts in the field of teratology for extrapolating the results found directly to the human experience." *Wade-Greaux, v. Whitehall Labs., Inc.*, 874 F. Supp. 1441, 1484 (D.V.I.), *aff'd*, 46 F.3d 1120 (3d Cir. 1994). "[D]ifferent species of animals react differently to the same stimuli." *Turpin v. Merrell Dow Pharm.*, Inc., 959 F.2d 1349, 1359 (6th Cir.), *cert. denied*, 506 U.S. 826 (1992). Accordingly, courts have consistently emphasized that animal studies alone do not typically provide a scientifically reasonable basis for concluding that a particular substance causes birth defects in humans. *See, e.g.*, *Brock*, 874 F.2d at 313; *Richardson by Richardson v. Richardson-Merrell, Inc.*, 857 F.2d 823, 830 (D.C. Cir. 1988), *cert. denied*, 493 U.S. 882 (1989); *Wade-Greaux*, 874 F. Supp. at 1483; *Cadarian v. Merrell Dow Pharm.*, Inc., 745 F. Supp. 409, 412 (E.D. Mich. 1989); *In re "Agent Orange" Prod. Liab. Litig.*, 611 F. Supp. 1223, 1241 (E.D.N.Y. 1985), *aff'd*, 818 F.2d 187 (2d Cir. 1987), *cert. denied*, 487 U.S. 1234 (1988).

a neoplasm, then in all probability that exposure caused or significantly contributed to that neoplasm." (10RR 221). This theory of causation has been explicitly rejected as scientifically unsound in *Castellow*, 97 F. Supp.2d at 793 and in *National Bank*, 22 F. Supp. at 946.

Dr. Legator mentioned one or two pieces of scientific evidence without giving any reasoned explanation for how he connected them to his causation opinion. He admitted that he could not extrapolate from any animal studies to quantify the amount of diesel exhaust exposure necessary to cause multiple myeloma in humans. (10RR 223). (*See Havner*, 953 S.W.2d at 729 (requiring expert to explain dosage extrapolations from animal studies)). As with plaintiff's other experts, Dr. Legator did not know what level of exposure to diesel exhaust is necessary to cause multiple myeloma in humans. (16RR 75-76).

Dr. Legator made no attempt to satisfy the *Havner* guidelines of sound scientific methodology based on scientific data and reasoning. Dr. Legator testified that he was not aware of any *in vitro* studies that showed chromosomal changes caused by diesel exhaust. (16RR 79-80). He mentioned a Chinese hamster study also cited by Dr. Gardner, but gave no explanation as to how it supported his opinion. (16RR 88).

Dr. Legator did not provide any reliable data and and an explanation of his reasoning from that data to his ultimate conclusion on causation. *See Havner*, 953 S.W.2d at 714. Dr. Legator did not use scientific methodology or scientific reasoning to reach his conclusions. He merely presented a "bare opinion" that Mrs. Navarro's multiple myeloma was caused by her occupational exposure to diesel exhaust. However, "an expert's bare opinion will not suffice." *Havner*, 953 S.W.2d at 711.

The opinions of Dr. Dayal, plaintiff's epidemiologist, Dr. Frank Gardner, plaintiff's oncologist, and Dr. Marvin Legator, plaintiff's toxicologist, should not have been admitted as evidence because: (1) they were based upon Frank Parker's incorrect and methodologically flawed exposure estimate; (2) their opinions were not supported by scientifically reliable methodology; (3) their opinions on general and specific causation were not supported by any scientifically sound data, theory, methodology or by independent scientific research.

CONCLUSION

The trial court abused its discretion in admitting the speculative, scientifically unreliable testimony of Frank Parker, Dr. Hari Dayal, Dr. Frank Gardner, and Marvin Legator. All of the testimony of the plaintiff's experts, Frank Parker, Dr. Hari Dayal, Dr. Frank Gardner, and Marvin Legator, as to causation should have been excluded under the applicable cases defining the standards for admissibility of expert testimony. There is no scientifically reliable evidence in the record in this case that diesel exhaust caused Mrs. Navarro's multiple myeloma.

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Respectfully Submitted,

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BIOGRAPHICAL ADDENDUM

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CLARK W. HEATH, M.D. is a medical doctor, specializing in epidemiology and preventive and community medicine. He has been Clinical Professor of Community Health, Emory University School of Public Health since 1990. He has taught community health, preventive medicine, epidemiology and biostatistics at Emory University School of Medicine, and at the University of South Carolina School of Medicine and School of Public Health. In addition to his academic appointment, he was most recently Associate Chief of Research, Radiation Effects Research Foundation of the National Academy of Sciences. From 1988 to 1998 he was Vice President for Epidemiology and Surveillance Research, American Cancer Society. From 1985 to 1988, he was Director, Bureau of Preventive Health Services, South Carolina Department of Health and Environmental Control. He has been a senior scientist at the Center for Disease Control, with responsibility for numerous projects dealing with epidemiology, including cancer and birth defects, viral diseases, and chronic diseases. Dr. Heath has served as an advisor to numerous committees, commissions and panels for the U.S. government, international organizations, and scholarly institutions in the fields of epidemiology, occupational medicine and environmental science. He is a fellow of the American College of Epidemiology and the American Association for the Advancement of Science; he is a member of numerous professional and scholarly organizations, including the American Association for Cancer Research, the American Epidemiological Society, the American Public Health Association, the American Society of Preventive Oncology, the International Epidemiological Association, the International Society for Environmental Epidemiology and the Society for Epidemiologic Research. Dr. Heath has received awards for meritorious service from the United States Public Health Service and the Center for Disease Control.

DUDLEY HERSCHBACH is a Nobel Laureate in Chemistry (1986). He is Baird Professor of Science at Harvard University, where he was previously Professor of Chemistry, Chairman of the Chemistry Department and Chairman of the Chemical Physics program. He is the recipient of the Pure Chemistry Prize of the American Chemical Society, the Linus Pauling Medal, the Michael Polanyi Medal, the Irving Langmuir Prize of the American Physical Society, the National Medal of Science and the Jaroslav Heyrovsky Medal.

STEVEN H. LAMM, M.D., D.T.P.H. is a medical doctor; he also holds a diploma in tropical public health. He is board certified in pediatrics, in occupational medicine and preventive medicine. He is a charter fellow of the American College of Epidemiology, and a winner of the Annual Prize of the Society for Epidemiologic Research. Dr. Lamm also holds a Master of Science degree in biophysics. He is President of Consultants in Epidemiology & Occupational Health, Inc., Associate Professor in the Department of Health Policy and Management at the School of Hygiene and Public Health of The Johns Hopkins University, Adjunct Associate Professor, Preventive Medicine and Biometrics, Uniformed Services University of the Health Sciences, Clinical Assistant Professor of Pediatrics at the Georgetown University Medical School, Washington, DC. He was Senior Epidemiologist in the Epidemiology Branch of the National Institute of Child Health and Human Development of the National Institutes of Health; Epidemic Intelligence Service Officer at the Centers for Disease Control. He has served as a consultant to the U.S. Food and Drug Administration, the Health Resources and Services Administration, the government of Inner Mongolia, the United States Department of Justice, and the National Center for Health Statistics.

LEE LOEVINGER, was a judge of the Supreme Court of Minnesota, Assistant Attorney General in charge of the Antitrust Division of the U.S. Department of Justice, and a commissioner of the Federal Communications Commission. He was a founding member of the American Bar Association Section of Science and Technology, and is on the editorial board of and a frequent contributor of articles on science, technology and law to JURIMETRICS, the journal of the American Bar Association Section of Science and Technology. He is a leader in the study of issues at the intersection of law and science. He is currently of counsel to the Washington, D.C. law firm Hogan & Hartson.

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SALLY L. SATEL, M.D. is resident scholar at the American Enterprise Institute. She is also lecturer at Yale University Medical School, and was assistant professor of Psychiatry at Yale Medical School from 1988 to 1995. She was also a visiting research scientist at the University of Pennsylvania Medical School. Dr. Satel has written numerous monographs and articles on drug treatment, the neurobiology of mental illness, neuropharmacology, the treatment of substance abuse, and depression, schizophrenia and paranoia. She earned her medical degree at Brown University. She is a diplomate of the American Board of Psychiatry and Neurology. She has won numerous awards, including the Menninger Award of the Central Neuropsychiatric Association.

BARRY H. SMITH, M.D., Ph.D. is Director of The Dreyfus Health Foundation; a member of the board of directors and Senior Vice President, The Rogosin Institute at The New York Presbyterian Hospital - Weill Medical College (Cornell) Medical Center, New York, New York and President of the Rogosin Research Foundation. He is also Professor in the Department of Surgery, Cornell University Medical College/New York Hospital. He is a member of the American Association for the Advancement of Science, the Society for Neuroscience, the International Brain Research Organization, the New York Academy of Science, the American Public Health Association, the Global Health Council and the International Organization of Psychophysiology. He is Co-Editor, ENCYCLOPEDIA OF NEUROSCIENCE, Associate Editor, *Journal of Neuro-Oncology*, Reviewer, *Journal of Critical Care Medicine*. He is the author of over 100 publications in the fields of neuroscience, organ transplantation and public health. He is a medical doctor, licensed in New York, Maryland and Massachusetts, specializing in neurosurgery, and holds a Ph.D. in Biology from the Massachusetts Institute of Technology.

JAMES D. WATSON is a Nobel Laureate in Medicine (1962) (with F.H.C. Crick and M.H.F. Wilkins), and co-discoverer of the structure of DNA. Dr. Watson has also been awarded the John Collins Warren Prize of the Massachusetts General Hospital, the Albert Lasker Prize of the Public Health Association, the John J. Carty Gold Medal of the National Academy of Sciences, and the Presidential Medal of Freedom. He earned his Ph.D. in Zoology, and has been awarded numerous honorary degrees. He is director and president of the Cold Spring Harbor Laboratory of the National Institutes of Health.

RICHARD WILSON is Mallinckrodt Research Professor of Physics at Harvard University and immediate past Director of the Regional Center for Global Environmental Change at Harvard University. He is an Affiliate of the Center for Risk Analysis, the Center for Science and International Affairs and the Center for Middle Eastern Studies at Harvard University. He is a past Chairman of the Department of Physics at Harvard University, a past chairman and currently a member of the Cyclotron Operating Committee. He is a founder of the Society for Risk Analysis. He is and has been a consultant to the United States government and the governments of numerous foreign countries on matters of nuclear safety, toxicology, epidemiology, public health and safety and risk assessment. He is the author of many articles on high energy physics, environmental pollution and risk analysis.

CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the Amicus Brief of Marcia Angell, Philippe Baveye, Louis Anthony Cox, Jr., Leonard D. Hamilton, Ronald Hart, Clark W. Heath, Dudley Herschbach, Steven H. Lamm, Lee Loevinger, Rodney Nichols, Sally L. Satel, Barry H. Smith, James D. Watson and Richard Wilson has been provided to the following attorneys of record, by first class mail on this the 29th day of August, 2001:

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