

A122449

**IN THE COURT OF APPEAL
OF THE STATE OF CALIFORNIA
FIRST APPELLATE DISTRICT, DIVISION THREE**

MACK SHELBY,
Plaintiff and Respondent,

v.

**SEARIVER MARITIME, INC., FORMERLY KNOWN AS
EXXON SHIPPING COMPANY,**
Defendant and Appellant.

APPEAL FROM SAN FRANCISCO COUNTY SUPERIOR COURT
MARLA J. MILLER, JUDGE • CASE No. CGC-06-449350

BRIEF *AMICUS CURIAE* IN SUPPORT OF APPELLANT

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INTEREST OF AMICI

Amici are scientists with expertise in toxicology and epidemiology. The credentials of *amici* are set out in the Biographical Appendix, *infra*.

Amici are concerned that the court took seriously the testimony of plaintiff's expert in this case, because in our view his testimony should have been excluded under the *Kelly/Frye* standard used by the courts in California.

Amici are also concerned that the contradictory and ambiguous application of the *Kelly/Frye* standard by California appellate courts has created an environment in which juries may be confused by, and swayed by, bare opinion masquerading as "science."

Amici have reviewed the trial testimony ourselves, and find no clear statement by Dr. Avery of the methodology that he used to determine that the plaintiff developed kidney cancer from exposure to benzene. Had he utilized recognized and standard methodology, he would have found that the connection between benzene exposure and renal cell cancers is neither generally accepted in the relevant scientific and medical communities; nor is such a causal connection established scientific knowledge. To elaborate, we shall discuss some of the elements which are required, using proper methodology, to elucidate causal relationships; how Dr. Avery failed to consider these and why, had he done so, he would have necessarily reached

a very different conclusion as to causation. Thus, we submit, Dr. Avery's flawed methodology led to a flawed conclusion which necessarily misled the jury in this case.

FACTUAL BACKGROUND

The parties seem to agree on the essential facts. We set forth below those facts that are pertinent to *amici's* belief that the testimony of plaintiff's expert, Dr. Avery, shows that he did not follow generally accepted methods recognized by the relevant scientific community.

Mr. Shelby's Testimony

Mr. Shelby worked as an able-bodied seaman on SeaRiver Maritime, Inc. (SeaRiver) oil tankers. He claimed that exposure to crude oil and other hydrocarbons containing benzene while he worked at SeaRiver caused cancer, which resulted in the removal of one of his kidneys and has a 5 percent chance of recurring in his remaining kidney.

Mr. Shelby sued Exxon Mobil Corporation, Exxon Shipping Company, and SeaRiver Maritime, his employers, alleging the loss of his right kidney from cancer was caused by occupational exposure to hydrocarbons, including benzene. (1 AA 9-20; 1 AA 56-66, 75-76, 122-123, 124-134.) Mr. Shelby was diagnosed with kidney cancer in 2003. (6 RT 610-611.)

Mr. Shelby claimed that he was exposed to cancer-causing hydrocarbons in excess of Coast Guard regulatory exposure levels, and OSHA and industry standards, and that the owners of the ships on which he served failed to warn him about these risks and failed to provide him with appropriate protective equipment. (1 AA 13-14.)

Mr. Shelby worked loading cargo into and topping off cargo tanks on SeaRiver vessels.¹ (5 RT 578-588.) His work schedule was four (4) hours on, eight (8) hours off. (5 RT 585; 6 RT 656.) Mr. Shelby claimed he might be exposed to benzene during the loading process, when, for 30 to 45 minutes, the crew would pump oil that had dripped from the hoses into a dirt pan and then transferred that into another tank; during loading or while underway, when a vent cap would open and release vapors for from 30 seconds to 30 minutes; during measuring the amount of space in the cargo tank when it had lost oil due to leakage or evaporation (“ullageing”), which would take a total of 4 hours (20 to 30 minutes for each of 15 tanks onboard the vessel). (5 RT 579-588; 6 RT 645-649, 658.)

Mr. Shelby also described being sprayed with “heart-cut reformat”, after which he would immediately shower and change clothes. (6 RT 608-610.)

¹ The vessels on which Mr. Shelby served were owned or operated by SeaRiver and its predecessor companies. We refer to them collectively as

Dr. Avery's Testimony

P{laintiff presented one expert witness, Dr. Nelson Avery, who testified that benzene is capable of causing kidney cancer and in fact caused Shelby's cancer.

Dr. Avery asserted that:

- “Mr. Shelby was exposed to benzene on a regular basis in amounts that exceeded OSHA and industry standards” and that his “15 year history of occupational exposure to benzene [is] sufficient to cause kidney cancer”;
- Solvents, including benzene, are “well recognized as a causation factor for kidney cancer”; and
- Occupational exposures similar to Shelby's have been shown to create an elevated risk of kidney cancer. (1 AA 107-108.)

Dr. Avery testified that benzene was a known cause of a particular type of leukemia, but that there is a “possibility” that other types of leukemia and blood disease, as well as kidney cancer, could result from benzene exposure. (4 RT 234-235.) Dr. Avery hypothesized that the mechanism for benzene-caused kidney cancer theorized was that a substance will pass through the organs with the highest fat content (such as the liver, kidneys, heart and brain) and then move on to other parts of the body like bone marrow and that as metabolized benzene moved through the

kidneys it would have “an opportunity to injure the kidney . . . and to have a carcinogenic effect.” (4 RT 236-237, 243-246.)

Dr. Avery asserted that the fact that Shelby and another worker on the *Wilmington* (one of the ships on which Mr. Shelby served) both had kidney cancer, which is a “rare tumor,” supported his opinion that occupational exposure to benzene causes kidney cancer. (4 RT 252-253.) Dr. Avery acknowledged, however, that the occurrence of these two cases of cancer on the same ship was not statistically significant. (4 RT 253.)

Dr. Avery reviewed approximately 35 articles, many of which found no connection between benzene and kidney cancer or, if they did find a connection, were statistically insignificant. (4 RT 303-321.) Dr. Avery concluded that only six of the 35 articles supported his opinion that benzene exposures in Shelby's line of work could cause kidney cancer. (4 RT 254-264)²

Dr. Avery acknowledged that many of the studies he relied on either did not or could not calculate the levels of exposure experienced by their subjects, which meant he could not make a comparison with Shelby's exposures. (4 RT 286-287.) In the absence of such data, Avery *assumed* that

² None of these six studies found a link between a single chemical and kidney cancer because the occupational exposures examined involved a combination of chemicals. None of the six studies found a causal link

these studies involved some exposures of more than 1 ppm of benzene. (4 RT 287)

ARGUMENT

We shall show that Dr. Avery's methodology does not meet the legal standard in California, and that therefore his testimony should have been excluded.

I. DR. AVERY DID NOT USE A GENERALLY ACCEPTED METHODOLOGY

The generally accepted methodology

Scientists have suggested that in order to establish causation it is convenient to divide the evidence into two groups: General Causation and Specific Causation.³ General causation is satisfied if the assumed cause

between benzene and kidney cancer.

³ A plaintiff must show both general and specific causation. If the chemical does not possess that capacity, then the chemical cannot have been a cause of the plaintiffs claimed harm. But if the chemical does have that capacity, then the causation inquiry becomes whether the plaintiffs exposure to the chemical in question was to a reasonable medical probability a proximate cause of this particular plaintiffs harm (i.e., "specific causation"). (See *In re Hanford Nuclear Reservation Litigation* (9th Cir. 2002) 292 F.3d 1124, 1133. See also *Parker v. Mobil Oil Corp.* (2006) 7 N.Y.3d 434, 448, 857 N.E.2d 1114, 1120 ("It is well established that an opinion on causation should set forth a plaintiff's exposure to a toxin, . . . and that plaintiff was exposed to sufficient levels of the toxin to cause the illness (specific causation)"). Here, Dr. Avery admitted that he did not know, and had not modeled, the benzene exposure or dose Shelby received during his employment; he merely assumed that these exposures

can be shown to cause the medical effect at issue in any group of people. Epidemiologists will naturally choose to study the group of people that is exposed to the highest dose. If they fail to find a statistically significant effect, then it is assumed, using the old principle of Paracelsus “the dose makes the poison”⁴ that anyone exposed at a lower dose will have an even smaller risk and that it is less informative to look at the individual dose and discuss “Specific Causation.”⁵ Most courts have accepted this ordered approach as a useful way of discussing the problem.

We have examined Dr. Avery’s testimony in court and in his pretrial deposition to ascertain whether or not he followed this, or any other accepted, methodology.

were comparable with those shown in the company-wide air monitoring.

⁴ A discussion of more modern formulations of this principle, which was articulated by Paracelsus in the sixteenth century, can be found in Silbergeld, E.K., (1991) “The Role of Toxicology in Causation: A Scientific Perspective, 1 Cts. Health Sci. & L. 374, 378.

⁵ There are three central tenets of toxicology. First, “the dose makes the poison”-- this implies that all chemical agents are intrinsically hazardous, and whether they cause harm is only a question of dose. Even water, if consumed in large quantities, can be toxic. Second, each chemical agent tends to produce a specific pattern of biological effects that can be used to establish disease causation.(Some substances, such as central nervous system toxicants, can produce complex and nonspecific symptoms, such as headaches, nausea, and fatigue) Third, the toxic responses in laboratory animals are useful predictors of toxic responses in humans. *See* Federal Judicial Center, (2000) REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 403

(2nd ed.).

Dr. Avery's methodology

The studies on which Avery relied for his opinion that benzene caused Shelby's kidney cancer involved occupations with mixed exposures to a variety of chemicals besides benzene, exposures which were not demonstrably comparable to those experienced by Shelby, or showed an insufficient confidence level and Avery admittedly had little information about Shelby's exposure to benzene at work, and therefore relied on the potential or possibility of exposure in rendering his opinion. (1 AA 85-86, 91-93.)

Nevertheless, Dr. Avery thought that there was enough research to allow him to assume an "association" between benzene and kidney cancer. (4 RT 312)

Dr. Avery did not see any air monitoring data for Shelby himself and he had made no effort to calculate Shelby's average level of exposure to benzene while employed at SeaRiver and he had done no dose estimate for Shelby individually. He simply assumed that Shelby's benzene exposures were equivalent to the total exposures reported in the industrial hygiene monitoring data from SeaRiver conducted in 1990.

Avery concluded that these exposure levels were sufficient to cause Shelby's kidney cancer (4 RT 283-285; *see also* 4 AA 852, 865) and opined that benzene was the "most likely" cause of Shelby's cancer because

benzene was the “most likely thing that’s the carcinogen on those ships.” (4 RT 267)

The Flaws in Dr. Avery’s Methodology

Dr. Avery stated that he has examined various internationally accepted compendia for carcinogenic substances and finds that benzene is labeled a Class I carcinogen. We have no quarrel with this. The U.S. EPA’s IRIS database (Benzene (CASRN 71-43-2, <http://www.epa.gov/NCEA/iris/subst/0276.htm>)) and the International Agency for Research on Cancer (IARC), 45 IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels, § 5.5, <http://monographs.iarc.fr/ENG/Monographs/vol45/volume45.pdf> (last updated 01/21/1998, last accessed 05/25/2010) so state. Dr. Avery failed to go further and to examine the work of these agencies and the reason that they assign benzene to be a class I carcinogen.

General Causation

Amici agree with these agencies that benzene in high concentrations doses can cause, and has caused, Acute Myelogenous Leukemia (“AML”). The definitive evidence came, initially, from a small number (less than 10) of leukemias at the Pliofilm plant in Texas, and about 20 cases among Turkish shoemakers. It has been supported by the work of Otto Wong and

others studying tens of thousands of chemical and oil refinery workers in the U.S. and China (Theis, R.P., *et al.* “Smoking, environmental tobacco smoke, and risk of renal cell cancer: a population-based case-control study,” 8 *BMC Cancer*. 387 (2008) (“Theis”); Wong, O. “An industry wide mortality study of chemical workers occupationally exposed to benzene. II. Dose response analyses,” (1987) 44 *Br. J. Ind. Med.* 382-395 (“Wong I”); Wong, O. and Raabe G.K., “Critical review of cancer epidemiology in petroleum industry employees, with a quantitative meta-analysis by cancer site,” (1989) 15 *Am. J. Ind. Med.* 283-310 (“Wong II”); Wong, O. Trent, L., Harris, F., “Nested case-control study of leukemia, multiple myeloma, and kidney cancer in a cohort of petroleum workers exposed to gasoline,” (1999) 56 *J. Occup. Environ. Med.* 217-221 (“Wong III”); Wong, O., Harris, F., Rosamilia, K., Raabe, G.K., “Updated mortality study of workers at a petroleum refinery in Torrance, California, 1959 to 1997,” (2001) 43 *J. Occup. Environ. Med.* 1089-1102 (“Wong IV”); Wong, O., Harris, F., Rosamilia, K., Raabe, G.K., “An updated mortality study of workers at a petroleum refinery in Beaumont, Texas, 1945 to 1996,” (2001) 43 *J. Occup. Environ. Med.* 384-401 (“Wong V”). Although these workers had multiple chemical exposures besides benzene, it has been assumed that benzene is the likely cause of their leukemias. They have not been found to have an excess risk of kidney cancers. None of these international

consensus publications or very large studies found that benzene causes kidney cancer or, indeed, any other solid tumor cancers. Thus, while General Causation for benzene-caused leukemia is well established, it is not established for any solid tumors. Although not clearly stated by Dr. Avery, it appears he assumed that if benzene causes Acute Myelogenous Leukemia then it causes solid cancers. This is far from “established methodology.” On the contrary it is, in the view of *amici*, a fundamental violation of causation methodology to make such an assumption.

While some substances cause cancers at multiple sites and of varying types, many do not. The majority of substances that cause cancer act only to cause or contribute to the development of one or a few specific cancers. Indeed, Sir Austin Bradford Hill in his seminal list of “attributes” of a statistical association that would lead one to infer a causal nature to the association, noted exactly this specificity. See Hill, A.B., “The Environment and Diseases: Association and Causation,” (1965) 58 *Proc. Royal Soc. Med., Sec. Occup. Med.* 295. Hill’s proposed list of “attributes” of the association to be considered in evaluating causation consisted of: 1. Strength; 2. Consistency; 3. Specificity; 4. Temporality; 5. Biological gradient or dose response relationship; 6. Plausibility; 7. Coherence; 8. Experiment; and 9. Analogy. Only a few agents or other risk factors influence the development of cancers at many sites: smoking, obesity and

radiation exposure, for example, although even here there is specificity. For example, although radiation can cause cancer at numerous sites, it is not known to cause kidney cancer. Chow, W.H., Dong L.M., Devesa, S.S., “Epidemiology and risk factors for kidney cancer,” (2010) 7 *Nat. Rev. Urol.* 245-257 (“Chow”).

In some instances of cancer causation, we have a good understanding of the mechanism by which an agent initiates or promotes cancer development. In most cases, associated factors are designated as “risk factors,” because they have been associated with cancer outcomes in human epidemiologic studies, but the mechanistic understanding of their role has not been delineated. For example, there is reasonable indication of how components of cigarette smoke lead to lung cancer. By contrast, obesity is an identified risk factor for breast cancer, but the exact mechanisms are unclear. In the case of benzene and the specific cancer (AML) which has been linked to it, there is both a strong mechanistic hypothesis as to how it leads to leukemia and epidemiologic evidence showing that it does increase the risk of leukemia in humans. When it comes to kidney cancer, the opposite is true. There is a good mechanistic reason to believe it would not cause kidney cancer and there is ample epidemiologic data that it is not a likely risk factor.

These issues -- mechanisms and identified risk factors -- are methodologically key components in the assessment of causation. Failure to consider them represents a failure of generally accepted causation assessment for cancer causation. Dr. Avery did not consider properly these elements. Had he done so, he would have found no basis for asserting a causal relationship or an increased risk between benzene exposure and renal cancer.

Increased risk is demonstrated through epidemiologic studies.⁶ In the case of specific types of cancer and putative causes or risk factors, one can approach the issue from two directions. For kidney cancers and benzene, one can look at either risk factors for kidney cancers or the incidence of kidney cancers in those exposed to benzene. Both types of studies have been done. Neither the studies, nor consensus publications, find consistent evidence of an association of benzene with renal cancer using either

⁶ “Epidemiology is the field of public health and medicine that studies the incidence, distribution, and etiology of disease in human populations. The purpose of epidemiology is to better understand disease causation and to prevent disease in groups of individuals * * * * Epidemiologic evidence identifies agents that are associated with an increased risk of disease in groups of individuals, quantifies the amount of excess disease that is associated with an agent, and provides a profile of the type of individual who is likely to contract a disease after being exposed to an agent. Epidemiology focuses on the question of general causation (i.e., is the agent capable of causing disease?) rather than that of specific causation (i.e., did it cause disease in a particular individual?).” Federal Judicial Center, (2000) REFERENCE MANUAL ON SCIENTIFIC EVIDENCE, REFERENCE GUIDE ON

approach. In other words, benzene is not a known cause of, or contributor to kidney cancers, and kidney cancers have not been found to be connected to benzene. *See* Theis, *supra*; Wong I-V, *supra*; Chow, *supra*.

A “risk factor” is anything (diet, genetics, medications, smoking, chemicals, etc.) which may predispose someone to develop a malignancy, as evidenced by a higher frequency of such diseases in a population which has that risk factor versus one which does not.. Risk factors for kidney cancer have been identified in many studies: recent reviews were published by researchers at the National Cancer Institute and they represent current thinking (generally-accepted knowledge) on this subject. *See* Theiss, *supra* and Chow, *supra*. There is no mention of benzene in these major reviews.

Known Risk Factors for Kidney Cancer

Studies have found the following risk factors for kidney cancer:

Lifestyle-related risks:

- Smoking: Cigarette smoking is a major risk factor. Cigarette smokers are twice as likely as nonsmokers to develop kidney cancer. Cigar smoking also may increase the risk of this disease.
- Obesity: People who are obese have an increased risk of kidney cancer.

- High blood pressure: High blood pressure increases the risk of kidney cancer.
- Gender. Renal cell carcinoma is about twice as common in men as in women.
- Race. African-Americans have a slightly higher rate of renal cell cancer. The reasons for this are not clear.

Chemical exposure:

- A very few chemicals have been identified as potential risk factors for kidney cancer. These include trichloroethylene, cadmium, arsenic. Benzene is not among them.
- Workplace exposures: Many studies have suggested that workplace exposure to certain substances increases the risk for renal cell carcinoma. Some of these are asbestos, cadmium (a metal), some herbicides, and certain organic solvents, particularly trichloroethylene.

Genetic, hereditary and medical history risks:

- Genetic and hereditary risk factors. Some people inherit a tendency to develop certain types of cancer. The DNA that you inherit from your parents may have certain changes that account for this tendency to develop cancer. Some rare inherited conditions can cause kidney cancer. People who have the conditions listed here have a much higher risk for

getting kidney cancer, although they account for only a small portion of cases overall:

- von Hippel-Lindau disease. People with this condition often develop several kinds of tumors and cysts (fluid-filled sacs) in different parts of the body. They have an increased risk for developing clear cell renal cell carcinoma, especially at a younger age. They may also have benign tumors in their eyes, brain, spinal cord, pancreas and other organs, and a type of adrenal gland tumor called pheochromocytoma. This condition is caused by mutations (changes) in the VHL gene.

- Hereditary papillary renal cell carcinoma. People with this condition have inherited a tendency to develop one or more papillary renal cell carcinomas, but they do not have tumors in other parts of the body, as is the case with the other inherited conditions listed here. This disorder is thought to be caused by changes in the MET gene.

- Hereditary leiomyomatosis and renal cell carcinoma. People with this syndrome develop smooth muscle tumors called leiomyomas or fibroids of the skin and uterus (in women) and have a higher risk for developing papillary renal cell cancers. It has been linked to changes in the fumarate hydratase (FH) gene.

- Birt-Hogg-Dube syndrome. People with this syndrome, which is characterized by the development of small benign skin tumors, have an

increased risk of developing different kinds of renal cell cancers. They may also have benign or malignant tumors of several other tissues. The gene linked to this condition is known as BHD.

- Hereditary renal oncocytoma. Some people inherit the tendency to develop a kidney tumor called oncocytoma, which has a very low potential for being malignant.

- A family history of kidney cancer. People with a strong family history of renal cell cancer (without one of the known inherited conditions listed previously) also have a higher chance of developing this cancer. This risk is even higher in siblings (brothers or sisters) of those affected. It's not clear if this is due to genetics, a shared environmental exposure, or some combination of these.

- High blood pressure. The risk of kidney cancer is higher in people with high blood pressure. Some studies have suggested that certain medicines used to treat high blood pressure may raise the risk of kidney cancer, but it is hard to tell whether the condition or the medicine (or both) may be the cause of the increased risk.

- Advanced kidney disease. Persons with advanced kidney disease, especially those needing dialysis, have a higher risk of renal cell carcinoma. Dialysis is a treatment used to remove toxins from the body if the kidneys do not work properly.

- Long-term dialysis. Dialysis is a treatment for people whose kidneys do not work well. It removes wastes from the blood. Being on dialysis for many years is a risk factor for kidney cancer.

- Certain medicines, such as Phenacetin, once a popular non-prescription pain reliever, have been linked to renal cell cancer in the past. Because this medicine has not been available in the United States for over 20 years, this no longer appears to be a major risk factor.

A “risk factor” is not necessarily an established cause. Not everything which has been associated in a study with an increased risk is an established risk factor. This is so because simple inter-study variations are such that one study may identify slightly increased risks of one cancer or another associated with an exposure; whereas other studies do not. It is only when the majority of well designed and powerful studies (those with a large sample, *i.e.*, enough subjects) find a consistent relationship that a factor becomes generally accepted as a known or recognized risk factor. Thus, while there are a few studies which have found increases in kidney cancers associated with benzene exposure, most studies and the largest studies have not.

Indeed the largest studies of benzene and cancer risks have found no relationship between benzene and kidney cancer, even though there was a relationship to Acute Myelogenous Leukemia. (*See* Poole, C., Dreyer, N.A.,

Satterfield, M.H., Levin, L., Rothman, K.J., “Kidney cancer and hydrocarbon exposures among petroleum refinery workers,” (1993) 101 *Environ. Health Perspect. Suppl.* 6 at 53-62; Theis, *supra*; Wong I-V, *supra*). These data support the contention that General Causation has not been established for benzene and solid cancer tumors and in particular kidney cancer. These studies, taken together are “robust” because collectively they cover approximately 40,000 test subjects.⁷

There have been a number of non-specific studies on exposure to solvents in general, often with other confounding exposures.⁸ Some of these appear to show an increase in solid cancers. In none of these was benzene the only possible agent. Although not specifically stated by Dr. Avery, these may have been the data upon which he relies. An

⁷ The National Cancer Institute lists smoking, obesity, high blood pressure, long-term dialysis, Von Hippel-Lindau (VHL) syndrome, occupational exposure to steel-making coke, asbestos or cadmium; and gender as known risks. See National Cancer Institute, “What You Need to Know About Kidney Cancer, Kidney Cancer: Who's at Risk?” available at <http://www.cancer.gov/cancertopics/wyntk/kidney/page4> (last accessed 05-25-2010) See also Schottenfeld, D. and Fraumeni, Jr., J.F. (eds.), (1996) *CANCER EPIDEMIOLOGY AND PREVENTION* (3rd ed.) 1149-1151.

⁸ Confounding exposures are agents besides the one being studied which might give rise to the observed effect. For example, since a few chemicals such as trichlorethylene are associated with kidney cancer, if workers who were exposed to both benzene and trichloroethylene had excesses of kidney cancers, one would not be able to exclude the trichlorethylene as the potential cause. Trichloroethylene would be a “confounder.”

examination of these studies shows that although the comparison between the cancers in the solvent-exposed cohort and the general background often appeared to be statistically significant, no account was taken of the uncertainty of comparing two groups which may have little in common. Even if these data were statistically significant, with all uncertainties included, it would still be necessary to ask, as did Austin Bradford Hill in his famous list of “attributes,” whether it was logically permitted to consider the “association” as “causal.” This is the fundamental issue noted above.

A few positive associations do not equal causation or a known, accepted, relationship.⁹ There is no hint in the trial record that Dr. Avery ever considered the difference between such scattered, inconclusive information, and the conclusions drawn from more extensive and robust data.

⁹ It is a basic principle of epidemiology that “*an association is not equivalent to causation*. An association identified in an epidemiologic study may or may not be causal. Assessing whether an association is causal requires an understanding of the strengths and weaknesses of the study’s design and implementation, as well as a judgment about how the study findings fit with other scientific knowledge.” (Federal Judicial Center, (2000) REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2d ed.) at 336-337) (Emphasis in original)

Specific Causation

Most scientists would accept that if General Causation is not established then it is unnecessary to consider Specific Causation, *e.g.*, whether the dose to which Mr. Shelby was exposed was large enough to cause the specific cancer. However, when the evidence as to dose is examined, we find no evidence that Mr. Shelby's exposure was large enough.

We examine the data on the cohorts which have been used by the regulatory or advisory agencies to assign the classification I to benzene as a cause of leukemia. In particular, we look at the data on the Pliofilm cohort. *See, e.g.*, Paxton, M.B., "Leukemia Risk Associated with Benzene Exposure in the Pliofilm Cohort," (1996) 104 Environ. Health Perspect. (Supp. 6) 1431-1436; Crump, K.S., "Risk of benzene-induced leukemia predicted from the Pliofilm cohort," (1996) 104 Environ. Health Perspect. 1437-1441.

While it is sometimes difficult to obtain data from "bad old days," when there was high occupational exposure, these older data were examined in several scientific papers, which concluded that the average dose was probably higher than the 50 ppm, and often up to 300 ppm, benzene in the air and this dose was sustained for long periods.

There are no data that show that benzene causes leukemia at any dose below 20 ppm (*See* Rinsky, R.A., Hornung, R.W., Silver, S.R., Tseng,

C.Y., "Benzene Exposure and Hematopoietic Mortality: A Long-Term Epidemiological Risk Assessment," (2002) 42 *Am J. Ind. Med.* 474-480; Crump, K.S., "Risk Assessments for Benzene and Leukemia -- A review," in (1992) ONCOGENE AND TRANSGENICS CORRELATES OF CANCER RISK ASSESSMENT), although it is often assumed for regulatory purposes, as opposed to established, with reasonable medical/toxicological probability, that it can do so at lower levels, but with a proportionately smaller probability. This is often called the assumption of "low dose linearity." If a person is exposed at this lower dose or less, then it is likely that any tumor would not be caused by benzene but by some other agent. Any calculation of the "Probability of Causation" would be considerably below 50% and then any statement that "it is more likely than not" that the cancer was caused by benzene would have no logical foundation whatsoever. We find no testimony by Dr. Avery that he ever calculated a "Probability of Causation."

The recommended exposure to a substance to which a person can safely be exposed is known as the "Permissible Exposure Limit" ("PEL"). It takes into consideration the concentration of the substance and the duration of exposure. OSHA regulations, 29 C.F.R. 1910.1028, stipulate that the PEL for benzene in air is 1 part per million (ppm) based on time-weighted exposures of 8 hours a day, 40 hours a week. (5 RT 517-519;

4 AA 824) There is also a Short-Term Exposure Limit (S.T.E.L.) of 5 ppm for any 15 minute period during the work day, not to exceed 4 times per day.” (4 AA 824) It follows that levels occasionally exceeding the PEL by small factors do not alter the conclusion that the risk is small, even miniscule.

Dr. Avery provided no evidence of what Shelby’s exposures were, or could have been.¹⁰ Dr. Avery referred to the PEL established by the OSHA as 1 ppm in the air weighted average over an 8 hour period. Dr. Avery claims that this was frequently exceeded on the ship(s) on which Mr. Shelby served, but he provided no evidence, and there is none in the record, that the exposures experienced by Mr. Shelby frequently exceeded the 1ppm level, let alone by 50 fold.

The only other fact Dr. Avery relied on to support his opinion, that another seaman on the ship on which Shelby served also developed kidney

¹⁰ Air monitoring samples taken for Mr. Shelby on board ship in April 1990 and in September 1992 showed benzene exposure of 0.051, 0.04, 0.67, and 0.1309 ppm for exposure times ranging from 205 to 247 minutes. Air monitoring samples for Charles Pollard, who also served on SeaRiver tankers, taken at SeaRiver in March 1990 and in February 1992, showed benzene exposure of 0.2 ppm for 189 minutes, 0.185 ppm for 62 minutes, and 0.02 ppm for 455 minutes. (5 RT 480-483) Benzene exposure readings for other seamen who served on some of the same ships as Shelby showed much higher exposures: 1.086 ppm benzene for 245 minutes while loading; 1.687 ppm benzene for 25 minutes while doing final gauging; 1.109 ppm benzene for 1,206 minutes while loading (6 RT 622-625).

cancer, is insufficient to support a general causation opinion. A second occurrence of kidney cancer aboard a SeaRiver vessel does not establish a causal link between benzene and kidney cancer; at most it supports an association, which is not proof of causation. *See* Federal Judicial Center, (2000) REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2d ed.) at 348 An association between exposure to an agent and disease exists when they occur together more frequently than one would expect by chance. Although a causal relationship is one possible explanation for an observed association between an exposure and a disease, an association does not necessarily mean that there is a cause and effect relationship.

Dr. Avery did not go back, as *amici* have done, to examine the record that led to the establishment of 1 ppm as the recommended limit. It is noteworthy that the OSHA regulations specifically refer to risk of leukemia, not to kidney or other solid tumor cancers. *See* 29 CFR Part 1910.¹¹ The regulatory record clearly shows that the exposure is 1 ppm

There was no incidence of kidney cancer among the other crew members.

¹¹ OSHA regulations, 29 CFR 1910.1028, Supp. C state:

III. Signs and Symptoms

The detrimental effect on the blood-forming system of prolonged exposure to small quantities of benzene vapor is of extreme importance. The hematopoietic system is the chief target for benzene's toxic effects which are manifested by

alterations in the levels of formed elements in the peripheral blood. These effects have occurred at concentrations of benzene which may not cause irritation of mucous membranes, or any unpleasant sensory effects. Early signs and symptoms of benzene morbidity are varied, often not readily noticed and non-specific. Subjective complaints of headache, dizziness, and loss of appetite may precede or follow clinical signs. Rapid pulse and low blood pressure, in addition to a physical appearance of anemia, may accompany a subjective complaint of shortness of breath and excessive tiredness. Bleeding from the nose, gums, or mucous membranes, and the development of purpuric spots (small bruises) may occur as the condition progresses. Clinical evidence of leukopenia, anemia, and thrombocytopenia, singly or in combination, has been frequently reported among the first signs.

Bone marrow may appear normal, aplastic, or hyperplastic, and may not, in all situations, correlate with peripheral blood forming tissues. Because of variations in the susceptibility to benzene morbidity, there is no "typical" blood picture. The onset of effects of prolonged benzene exposure may be delayed for many months or years after the actual exposure has ceased and identification or correlation with benzene exposure must be sought out in the occupational history.

* * * *

V. Surveillance and Preventive Consideration

A. General

The principal effects of benzene exposure which form the basis for this regulation are pathological changes in the hematopoietic system, reflected by changes in the peripheral blood and manifesting clinically as pancytopenia, aplastic anemia, and leukemia. Consequently, the medical surveillance program is designed to observe, on a regular basis, blood indices for early signs of these effects, and although early signs of leukemia are not usually available, emerging diagnostic technology and innovative regimes make

averaged over a long period (such as a year), the risk of developing Acute Myelogenous Leukemia is less than 1 in 10,000. Moreover, this is purely a theoretical risk, established for regulatory purposes. No studies have ever shown that levels this low actually increase the risk of either leukemias or renal cell carcinoma.

The studies upon which Dr. Avery relied for his opinion that benzene caused Mr. Shelby's kidney cancer involved occupations with mixed exposures to a variety of chemicals in addition to benzene, exposures which were not demonstrably comparable to those experienced by Mr. Shelby, or showed an insufficient confidence level and Dr. Avery admittedly had little information about Mr. Shelby's exposure to benzene at work, and therefore relied on a mere potential or possibility of exposure in rendering his opinion. (1 AA 85-86, 91-93.)

Nevertheless, Dr. Avery thought that there was enough research to allow him to find an "association" between benzene and kidney cancer. (4 RT 312)

consistent surveillance for leukemia, as well as other hematopoietic effects, essential.

The Centers for Disease Control, National Institute for Occupational Health and Safety, likewise notes the risk to humans of chronic benzene exposure for irreversible injury to the blood-forming organs; effects include aplastic anemia and leukemia." See "Occupational Health and Safety Guideline for Benzene, Potential Human Carcinogen" at <http://www.cdc.gov/niosh/docs/>

As noted above, Dr. Avery did not see any air monitoring data for Mr. Shelby himself and he had made no effort to calculate Mr. Shelby's average amount of exposure to benzene while employed at SeaRiver and he had done no dose estimate for Shelby individually. As we have shown, the measured exposures were significantly lower than those on which the regulatory agencies based their standards.

Moreover, had Dr. Avery examined the scientific literature, he would have found three critical issues which would have, of necessity, altered his opinion: First, there is no known or generally-accepted relationship between kidney cancers and benzene; second, even if there were a generally-accepted relationship, without a dose estimation, one cannot not make such a causal link; and third, numerous alternative factors (including unknown or idiopathic causation) cannot be ruled out. Dr. Avery's failure to consider these critical issues rendered his testimony methodologically unsound and should have led the trial court to exclude his testimony as an expert witness on causation.

Thus, Dr. Avery had no foundation for his opinions. His opinion that benzene could cause kidney cancer was based on studies which neither linked any increased kidney cancer risk to benzene nor concluded there was a causal link between benzene and Mr. Shelby's type of cancer. Dr. Avery

admitted that he did not know what level of benzene Mr. Shelby was exposed to, and without a known level of exposure it is not possible to reach a conclusion as to causation.

In conclusion, *amici* submit that Dr. Avery completely failed to explain his methodology in any logical way. His conclusion is, therefore, mere *ipse dixit*, without support in the scientific literature. Accordingly, Dr. Avery's opinion does not constitute substantial evidence.

II. THE LEGAL STANDARD FOR ADMISSIBILITY OF EXPERT TESTIMONY

Evidence Code section 801 establishes a “threshold requirement of reliability” for expert testimony. (*People v. Gardeley* (1996) 14 Cal.4th 605, 618) A trial court is required to “exclude testimony in the form of an opinion that is based in whole or in significant part on matter that is not a proper basis for such an opinion.” (Evid. Code, § 803)

An expert opinion is “no better than the facts on which it is based.” *Gardeley* 14 Cal.4th at 618; *see also People v. Bassett* (1968) 69 Cal.2d 122, 141 (“Expert evidence is . . . valuable only in regard to the proof of the *facts* and the validity of the *reasons* advanced for the conclusions” (emphasis in original)); *Kelley v. Trunk* (1998) 66 Cal.App.4th 519, 523, 78 Cal.Rptr.2d 122 (“An expert's opinion, even if uncontradicted, may be rejected if the reasons given for it are unsound.”)

Where, experts, like Dr. Avery in this case, base their conclusions on “findings” or theories that are speculative, remote, or conjectural, their opinions do not constitute substantial evidence sufficient to support a judgment. *See Lockheed Martin Corp. v. Superior Court* (2003) 29 Ca1.4th 1096, 1110, 63 P.3d 913, 131 Cal.Rptr.2d 1 (“*Lockheed Martin*”) (“[O]ur settled understanding [is] that an expert's opinion which rests upon guess, surmise or conjecture, rather than relevant, probative facts, cannot constitute substantial evidence.”); *Saelzler v. Advanced Group 400* (2001) 25 Ca1.4th 763, 776-777, 23 P.3d 1143, 107 Cal.Rptr.2d 617 (proof of causation cannot be based on an expert's opinion based on inferences, speculation and conjecture); *Lockheed Litigation Cases*, (2004) 115 Cal.App.4th 558 at 564 126 Cal.App.4th 271, 23 Cal.Rptr.3d 762 (“*Lockheed*”) (“An expert opinion has no value if its basis is unsound **** the matter relied on [by an expert] must provide a reasonable basis for the particular opinion offered and. . .an expert opinion based on speculation or conjecture is inadmissible.”)

An expert must provide the court with the bases for his or her opinion, and offer more than bare conclusions. *See Kelley v. Trunk* (1998) 66 Cal.App.4th 519, 524. The value of opinion evidence rests not in the conclusion reached but in the factors considered and the reasoning employed," and where, as here, “an expert bases his conclusion upon

assumptions which are not supported by the record . . . or upon factors which are speculative, remote or conjectural, then his conclusion has no evidentiary value.” *Pacific Gas & Electric Co. v. Zuckerman* (1987) 189 Cal.App.3d 1113, 1135. “[A]n expert's opinion that something *could* be true if certain assumed facts are true, without any foundation for concluding those assumed facts exist in the case before the jury, does not provide assistance to the jury because the jury is charged with determining what occurred in the case before it, not hypothetical possibilities.” *Jennings v. Palomar Pomerado Health Systems, Inc.* (2003) 114 Cal.App.4th 1108, 1117, 8 Cal.Rptr.3d 363

The test in *People v. Kelly*, (1976) 17 Cal.3d 24 (“*Kelly*”), which is derived from the test in *Frye v. United States* (D.C. Cir. 1923) 293 F. 1013, 54 App.D.C. 46 (“*Frye*”) and once applied in federal courts, requires that “new” types of scientific evidence be subject to judicial investigation to ensure that the evidence is “generally accepted in the relevant scientific community.” *Kelly, id.* at 30.

Dr. Avery, Mr. Shelby's sole causation expert, did not discuss or present any articles showing that benzene alone, as opposed to a myriad of other occupational chemical exposures could cause kidney cancer. Dr. Avery testified only that benzene was "associated" with certain types of cancer, including the "possibility" that benzene might be linked to kidney

cancer. (See 4 Augmented RT 292.) Mere association, or an unsupported "possibility" is nothing more than speculation, and is not substantial evidence to support a judgment.

Amici submit that Dr. Avery's methods were not permissible because they are not "generally accepted." "Cherry picking" or selective use of studies on which to rely, and excluding the overwhelming number of studies that do not support his thesis, is misleading and considered to be improper scientific methodology.

Plaintiff might argue that *Kelly* limits the application of the "general acceptance" test to "new scientific principles." The language of *Kelly* indicates a much broader concern with the admissibility of junk scientific evidence, lauding *Frye*'s "conservative nature" and the importance of "interpos[ing] a substantial obstacle to the unrestrained admission of evidence." *Id.* The court's main rationale for applying *Frye* was that scientific evidence tends to disproportionately influence the jury and should be withheld from the jury unless the trustworthiness of that evidence justifies the importance the jury will place upon it. *Id.*

In *People v. Leahy*, (1994) 8 Cal.4th 587, 882 P.2d 321, 34 Cal.Rptr.2d 663 ("*Leahy*") the Supreme Court noted that scientific evidence based upon new and experimental methods might be prejudicial, since "jurors tend to give considerable weight to "scientific evidence" and there is

“a misleading aura of certainty which envelopes a new scientific process, obscuring its currently experimental nature.” *Id.* at 595. The *Kelly/Leahy* language regarding the gullibility of juries when presented with scientific evidence is cited to this day. For example, it was recently quoted at length in *Harrison v. Smith*, 2008 WL 2673831 (Cal. App. 1st Dist. 2008). See also *People v. Venegas*, (1998) 18 Cal.4th 47, 954 P.2d 525, 74 Cal.Rptr.2d 262 (the *Kelly* test is necessary to “forestall the jury's uncritical acceptance of scientific evidence or technology that is so foreign to everyday experience as to be unusually difficult for laypersons to evaluate.” *Id.* at 80.

This case might be seen as presenting a somewhat different issue: Whether expert scientific evidence that derives from established scientific techniques, but extrapolates from those techniques in an unreasonable manner to reach a conclusion that is completely unsupported by the underlying data, is admissible. *Amici* submit that such “evidence” based on improper extrapolation and incomplete data is not admissible.

It is widely held at all levels of California courts that conjectural or unreasonable scientific evidence poses a substantial threat to the jury’s fact-finding ability, *Kelly* and *Leahy* and other cases on would support a “gatekeeper” role for the court. Expert opinion that has no foundation in the underlying data or scientific learning, such as Dr. Avery’s opinions in the case at bar, seem to be precisely the sort of evidence that the *Kelly* court

would want to withhold from the jury because the jury will give more credit to the evidence than it deserves because of its purported “scientific origins.”

The *Leahy* court thought that by rejecting the *Daubert* rule (*Daubert v. Merrell Dow Pharmaceuticals, Inc.* (1993) 509 U.S. 579)) (“*Daubert*”) and preserving *Frye*, it was maintaining a higher bar against “junk science” by preserving what it described as a “conservative test.” When *Leahy* was decided, in the early post-*Daubert* period, the prevailing view was that *Daubert* had liberalized, not restricted, the admission of expert evidence in the federal courts. *See Leahy*, 8 Cal.4th 587 at 596, 597. Thus, the *Leahy* court seemed to think that an adoption of *Daubert* would lower the bar for admission of evidence based upon new and unproven scientific procedures. The court stated that *Frye* was “an essential check” in the criminal process which would be eliminated if *Daubert* was adopted. *Leahy* at 603.

What would the *Leahy* court make of the current climate, where *Kelly* and *Leahy*, as interpreted by some trial and intermediate appellate courts, have made California a haven for “junk science” in civil cases? We believe a proper interpretation of Evidence Rule 801 would cure that problem.

Some often-cited cases in the California Courts of Appeal have excluded expert testimony which is not supported by the underlying data without referring to the risk of undue influence upon the jury. These

courts, applying section 801(b) of the California Evidence Code (discussed below), hold that conjectural scientific testimony has no evidentiary value and must be excluded.

In *Pacific Gas and Electric Company v. Zuckerman*, (1987) 189 Cal.App.3d 1113, 234 Cal.Rptr. 630, for example, the court held that when an expert (in financial valuation) gave a valuation that was underlain by “faulty assumptions, [that] undermine the validity of the expert's opinion to such an extent that his opinion cannot be deemed to constitute substantial evidence of value.” *Id* at 1129. Since the court held that expert testimony with no basis in fact had *zero* evidentiary value, it did not need to invoke the special impact that scientific evidence could have on a jury. Completely valueless evidence can traditionally be excluded for more prosaic reasons, like waste of time.

Similarly, in *Lockheed Litigation Cases*, (2004) 115 Cal.App.4th 558, 23 Cal.Rptr.3d 762, another court reached a virtually identical conclusion. In *Lockheed*, the trial court was faced with a very similar situation as the trial court in *Shelby*. The plaintiff's expert was arguing that chemicals the plaintiff was exposed to while working for Lockheed caused his cancer. The expert based this conclusion upon a study which showed that exposure to a “cocktail” of dozens of chemicals, many of which (1) the plaintiff had not been exposed to at Lockheed and (2) were known carcinogens, could

create an increased risk of cancer. Lockheed argued (and the trial court agreed) that the study was not indicative of increased risk, since the observed increase in cancer rates could be wholly caused by the chemicals in the study to which the plaintiff had not been exposed. The court cited *Pacific Gas*, holding that the expert's assertions "ha[d] no evidentiary value" and were properly excluded. (23 Cal.Rptr.3d at 771 (citation omitted)). It took a clear position: "an expert opinion has no value if its basis is unsound." *Id.*

The *Lockheed* court also grounded its decision on its interpretation of California Rule of Evidence 801(b), determining that the trial court was empowered under California law to exclude scientific evidence that was purely conjectural. *Id.* at 771-772.

While *Kelly* and *Leahy* demonstrate a policy concern with all unsupported scientific evidence, the actual test that those cases created, the *Kelly* test, is only applied to new evidence. Thus, the obvious question: why is *Kelly* only limited to new scientific methods? A proper reading of *Kelly* does not, we submit, support only applying the "general acceptance" test to new methodologies.

The *Kelly* court reasoned that *Frye's* "general acceptance" by the scientific community test was necessary to promote uniformity. 17 Cal.3d 24, 32. If each piece of scientific evidence was to be investigated by every

trial court *de novo* to determine if it had suitable reliability, the differing opinions of reliability from judge to judge would result in inconsistent admission of types of evidence. Instead, the *Kelly* court believed that using “consensus in the scientific community” as the arbiter of scientific reliability would “promote a degree of uniformity of decision.” *Id.* The *Kelly* Court noted that judges have no special skill as scientists and that “the requirement of general acceptance in the scientific community assures that *those most qualified to assess the general validity of a scientific method will have the determinative voice.*” *Id.* at 31 (emphasis in original).

Later decisions that follow *Kelly* repeat this argument, mostly quoting *Kelly*. *Leahy* both paraphrases *Kelly* and enlarges upon the benefits of a general acceptance requirement. The *Leahy* court noted that the general acceptance rule will create a lag between the creation of a new type of scientific evidence and its validation as admissible evidence in California courts, but that this lag will ensure that scientific evidence of “experimental or of dubious validity” (8 Cal.4th 587, 602) does not enter the California courts, as it might in certain cases if judges could make individual ad hoc determinations.

The policy considerations enumerated in *Kelly* and *Leahy* point strongly in favor of excluding scientific testimony with no basis in the underlying data methods. Nevertheless, a line of cases culminating in

Roberti v. Andy's Termite and Pest Control, (2003) 113 Cal.App.4th 893, 6 Cal.Rptr.3d 827, have expressly refused to allow judicial investigation in the reliability of scientific evidence based upon established methods, no matter how unreliable that evidence might be.

In *Roberti*, the trial court had applied *Kelly* to questionable medical expert testimony that derived speculative conjectures from well-accepted scientific papers. The court drew a explicit distinction between new methods or procedures, which were subject to the *Kelly* test, and new opinions or theories drawn from preexisting methods, which were not. *Id* at 832-833. The court synthesized an extensive list of cases in which the *Kelly* test was not applied to new opinions from established methods and concluded that (1) a literal interpretation of *Kelly* required that the test only be applied to new procedures and (2) that “juries do not view the subjective thought processes of an expert as having the aura of infallibility they tend to attribute to scientific devices, techniques, or procedures.” *Id* at 833-834. In other words, procedures -- the generation of data -- carry the risk of undue influence, but the interpretation of that data does not. *People v. McDonald*, (1984) 37 Cal.3d 351, 208 Cal.Rptr. 236, 690 P.2d 709, a case cited by *Roberti*, reaches an almost identical conclusion, holding that, “[w]hen a witness gives his personal opinion on the stand-even if he qualifies as an expert-the jurors may temper their acceptance of his

testimony with a healthy skepticism born of their knowledge that all human beings are fallible.” (37 Cal.3d 351, 372) Also citing *McDonald*, the court in *People v. Stoll*, (1989) 49 Cal.3d 1136, 265 Cal.Rptr. 111,783 P.2d 698 held that, “absent some special feature which effectively blindsides the jury, expert opinion testimony is not subject to *Kelly/Frye*.” *Id.* at 1157.

Roberti rejected the argument made in *Pacific Gas* and *Lockheed* that conjectural scientific evidence was inadmissible if it was irrelevant and lacked evidentiary value as an end-run around *Leahy*’s express rejection of the *Daubert* test. If, the court noted, scientific testimony was (1) based upon established methods and (2) contained conclusions that spoke to the causation of the injury in issue, then the only “gap” in the scientific evidence that could justify deeming it irrelevant was the lack of reliability of the conclusions that the expert had drawn, and this was a *sub rosa* application of the *Daubert* test. *See* 6 Cal.Rptr.3d at 835-836.

The policy considerations discussed in *Kelly* and *Leahy* strongly argue in favor of some sort of judicial assessment of the merits of scientific conclusions, as does common sense. The policy arguments advanced in *Roberti* are somewhat strained. The claim that an active role for the judge in reviewing scientific evidence, even if only to keep out junk science, would overrule *Leahy* by bringing *Daubert* into California courts is misplaced. We submit that *Roberti* was wrongly decided.

In *Kelly*, the court held that two sections of the Evidence Code establish the requirements for expert testimony: sections 720, which require that an expert be properly qualified, and 801(b), which states that the “matter” on which the expert’s opinion is based must be “of a type that reasonably may be relied upon by an expert.” The court interpreted 801(b) to require that the scientific “method” used by an expert be reliable. 549 P.2d at 1244. To determine whether a method is reliable, the Court turned to *Frye* and its general acceptance test. Later cases such as *Stoll* and *Roberti* briefly cited and accepted the *Kelly* analysis of 720 and 801(b).

In *Leahy*, the Court acknowledged that there is no language in 720 and 801(b) that explicitly supports a “general acceptance” test.

Sections 720 and 801(b), in combination, seem the functional equivalent of Federal Rules of Evidence, rule 702, as discussed in *Daubert*. Although some language in section 801(b) is broad enough to include a *Frye* standard of general acceptance (matter “of a type that reasonably may be relied upon by an expert”), nothing in these sections expressly establishes general acceptance as an absolute prerequisite to admissibility, and nothing in the legislative history leading to adoption of the Evidence Code indicates that a general acceptance standard was intended.

8 Cal.4th 587 at 598.

Indeed, the *Leahy* Court concluded that since *Kelly* affirmed the application of *Frye* in California after the Evidence Code was enacted, the *Kelly* Court must have been aware that *Frye* had no “real” support in the

Code and since the Code was unchanged since *Kelly*, *stare decisis* applied and the Code's provisions must still be read to justify application of *Frye*. *Id* at 599. Interestingly, the Court noted (and quoted) Cal. Evid. Code §§210 and 350, *id.* at 597, which combine to exclude irrelevant evidence, defined as evidence which has no "tendency in reason to prove or disprove any disputed fact that is of consequence to the determination of the action." Cal. Evid. Code § 210. Nevertheless, the significance of this language is not discussed. Instead, the court implicitly argued that any scientific expert conclusions which, if true, would make the theory of causation or guilt more likely, are inherently relevant. The Court argued that *Kelly* held that the *Frye* test excludes relevant evidence. *Id* at 601. However, on a plain reading of *Kelly* it is unclear where the *Leahy* court finds language to support this assertion.

Many other California courts have interpreted 801(b) quite differently to allow for judicial inquiry into the reliability of an expert's reasoning. Cases in which conjectural expert testimony has been excluded obviously depart from the *Kelly* interpretation of section 801(b). They do so by reinterpreting 801(b) in several different ways.

An example of a reinterpretation of 801(b) is in the recent decision in *Dee v. PCS Property Management*, (2009) 174 Cal.App.4th 390, 94 Cal.Rptr.3d 456. In *Dee*, like in *Shelby*, the court was presented with a

classic case of “junk science” proffered by a tort plaintiff. The plaintiff alleged that mold exposure caused her illness even though there was no evidence she was actually exposed to the mold and, even if she was, the evidence indicated that she was exposed to an amount far less than the minimum amount known to cause harm. Plaintiff’s expert, nevertheless, testified that the mold caused plaintiff’s illness.

The court in *Dee* rejected the expert’s testimony, holding that it violated section 801(b). The court distinguished *Roberti* because (1) Plaintiff’s expert was using new techniques (a classic *Kelly* application) and (2) the expert reached his conclusion without a “reasonable basis” in the underlying method, thus the opinion lacked a “foundation” which was required by section 801(b). *Id.* at 405-406. This second rationale is quite at odds with *Leahy*, which explicitly rejects this argument as de facto *Daubert*. It is, however, no less consistent with the express language of section 801(b) than *Kelly* or *Leahy*.

Jennings v. Palomar Pomerado Health Systems, Inc., (2003) 114 Cal.App.4th 1108, 8 Cal.Rptr.3d 363, articulates a different reinterpretation of section 801(b) to reach a similar result. At first, the reasoning seems nearly identical to that of *Pacific Gas and Lockheed*:

However, even when the witness qualifies as an expert, he or she does not possess a carte blanche to express any opinion within the area of expertise. For example, an expert's opinion

based on assumptions of fact without evidentiary support or on speculative or conjectural factors has no evidentiary value and may be excluded from evidence.

Id at 1117 (citations omitted)

In *Jennings* the court concluded that “an expert's conclusory opinion that something did occur, when unaccompanied by a reasoned explanation illuminating how the expert employed his or her superior knowledge and training to connect the facts with the ultimate conclusion, does not assist the jury.” *Id.* at 1117-1118. Since section 801(b) requires that, “the opinion of [the] expert . . . assist the trier of fact,” the court held that unreasoned expert testimony fails that criterion of section 801(b) and must be excluded. The court in *Jennings* showed that the expert so completely failed to explain his reasoning that his testimony could not conceivably assist the jury (*Id.* at 1118).

An overarching issue in deciding whether judges should review the reasoning employed by an expert is that it is hard to find middle-ground between *Roberti* approach, which would permit no inquiry, and some applications of *Daubert*, in which the judge's role is so active that many argue it intrudes upon the province of the jury. *Jennings* held that the court's examination should be limited to whether the connections between the expert's conclusions and his methodology are sufficiently clear to assist the jury. This permits judges to exclude testimony based on pure

conjecture, while allowing testimony that might reach a questionable conclusion but is, at least, supported by well-reasoned argument.

Lockheed reached a very similar conclusion to *Jennings*, but with another interpretation of section 801(b). The main holding of *Lockheed* was that, “[a]n expert opinion has no value if its basis is unsound . . . the matter relied on must provide a reasonable basis for the particular opinion offered, and that an expert opinion based on speculation or conjecture is inadmissible.” 23 Cal.Rptr.3d 762, 771. The court held that this conclusion arose out of the portion of section 801(b) which states that an expert’s opinion must be based on material “of a type that *reasonably may be relied upon by an expert in forming an opinion upon the subject to which his testimony relates.*” (*Id.*, emphasis in original). The court held that purely conjectural scientific testimony does not demonstrate the required reasonable reliance under section 801(b).

This language in section 801(b) was interpreted in *Kelly* and the cases that follow it, most recently *Roberti*, as justifying a *Frye* test and only a *Frye* test; in *Dee*, *Lockheed*, and *Jennings*, the same language was applied to justify judicial inquiry into an expert’s reasoning. *Dee* discussed *Roberti* at length because *Roberti*, with such similar facts, if followed by the *Dee* court, would have required admission of the expert testimony. *Dee* ultimately deemed *Roberti*’s analysis to be incorrect, arguing that *Roberti*

focused mistakenly upon a literal and narrow application of *Kelly* and not the language of section 801(b) as written. 174 Cal.App.4th at 404-406. *Dee* also noted *Roberti*'s dismissal of the section 210 relevance argument for excluding the evidence. While *Dee* did not cite section 210 elsewhere, this represents an implicit acknowledgement of the possibility of excluding junk scientific evidence under that provision.

The policy concerns the California Supreme Court expressed in deciding *Kelly* and *Leahy* strongly argue in favor of some kind of judicial inquiry into the reasons underlying an expert's testimony. The recent trend of the California courts, as exemplified by *Lockheed*, supports engaging in such an inquiry. If such an inquiry were made here, the Court should find that even if Dr. Avery's methodology was not "new," his conclusions are not supported by the available data or the weight of scientific evidence, or current scientific knowledge regarding the risks of benzene and the causes of kidney cancer.

CONCLUSION

Amici submit that Dr. Avery's opinion does not constitute substantial evidence and should have been excluded. The judgment of the trial court therefore should be reversed.

Dated: May 26, 2010

Respectfully submitted,

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BIOGRAPHICAL ADDENDUM

PATRICIA A. BUFFLER, Ph.D., M.P.H., C.P.H., is Professor of Epidemiology, University of California, Berkeley School of Public Health

In addition to teaching, Dr. Buffler is the Principal Investigator of the NIH-funded, Northern California Childhood Leukemia Study (NCCLS); an epidemiological investigation of childhood leukemia in Northern and Central California. Other research interests include: evaluating the role of genetic factors and environmental exposures in the etiology of childhood cancers and childhood brain tumors.

Dr. Buffler has served on numerous national and international advisory groups, and was recently appointed to the Advisory Committee for the National Center for Health Statistics. She was elected to the Institute of Medicine/National Academy of Science in 1994 and as a Fellow in the American Association for the Advancement of Science. She has received many distinctions in epidemiology including the prestigious Lilienfield Award from the American College of Epidemiology for her contributions to the field of epidemiology, the James D. Bruce Memorial Award from the American College of Physicians and American Society of Internal Medicine for her work in public health and preventive medicine and the 2001 Visiting Scientist Award from the International Agency for Research on Cancer (IARC).

She has been elected and served as President for numerous epidemiologic professional societies, including the Society for Epidemiologic Research, the American College of Epidemiology and the International Society for Environmental Epidemiology. She is currently a member of the Executive Committee and Treasurer of the International Epidemiological Association and the National Board of Public Health Examiners.

Dr. Buffler is currently a professor of Epidemiology in the School of Public Health at the University of California at Berkeley. After completing her undergraduate degree from Catholic University of America in Washington, D.C. she worked in public health and received her Master's and Ph.D. in epidemiology from the University of California at Berkeley. Her distinguished tenure at Berkeley includes serving as Dean from 1991-1998 and being named to the Kenneth and Marjorie Kaiser Endowed Chair in Cancer Epidemiology in 2004.

Dr. Buffler's research has shaped public health policy in several arenas. Her analysis of the effects of cigarette smoking on lung cancer risk in women helped target more smoking cessation programs towards women. The U.S. and California governments used information from her study of lung cancer in women exposed to second hand smoke during the development of their environmental and workplace tobacco regulations. Dr. Buffler's current research focuses on the genetic, environmental, and infectious exposures associated with childhood leukemia and brain tumors. She is the founding member of the Childhood Leukemia International Consortium (CLIC) involving collaboration with numerous studies of childhood leukemia from across the globe, and an active member of two other international consortia of childhood cancer investigators, the International Study of Embryonal Tumors (ISET) and the Brain Tumor Epidemiology Consortium (BTEC) and the Epidemiology Committee of the U.S.-based Children's Oncology.

RONALD E. GOTS, M.D., Ph.D. specializes in toxicology and environmental medicine, and is board certified in toxicology. He is Principal of the International Center for Toxicology and Medicine and Medical Director and President of the National Medical Advisory Service. He is also Lecturer in and Adjunct Professor of Pharmacology, Department of Pharmacology, Georgetown University School of Medicine. He has been Coordinator, Pharmaceutical Class Labeling Project, of the U.S. Food and Drug Administration, Medical Director and Examining Physician of the Occupational Health Units, Bureau of Economic Analysis, Census Bureau and Immigration and Naturalization Service, Senior Investigator/Chief, Department of Gastroenterology, Walter Reed Army Institute of Research. He was Conference Chair of the conference on "Multiple Chemical Sensitivities: State-of-the-Science Symposium" co-sponsored by the International Society of Regulatory Toxicology and Pharmacology, The Johns Hopkins University/National Institute for Occupational Safety and Health Educational Resource Center in the Occupational Safety & Health and National Medical Advisory Service.

STEVEN 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

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A. ALAN MOGHISSI, Ph.D. is President of the Institute for Regulatory Science (RSI), a non-profit organization whose major activity is conducting scientific peer reviews for government agencies, and dedicated to the idea that societal decisions must be based on the best available scientific information. The activities of RSI include research, scientific assessment, and science education at all levels – particularly the education of minorities.

Dr. Moghissi held positions at the U.S. Public Health Service and the U.S. Environmental Protection Agency (EPA). After his retirement from EPA, Dr. Moghissi joined the University of Maryland at Baltimore as Assistant Vice President for Environmental Health and Safety; subsequently he was Associate Vice President for Environmental Health and Safety at Temple University in Philadelphia, Pennsylvania. He has been a visiting professor at Georgia Tech and at the University of Virginia. Dr. Moghissi's research has dealt with diverse subjects ranging from measurement of pollutants to the biological effects of environmental agents. A major segment of his research has been on scientific information upon which laws,

regulations, and judicial decisions are based -- notably risk assessment. Dr. Moghissi's research has included biological and environmental kinetics, but increasingly has focused on the development and implementation of the concept of Best Available Science (BAS) in societal and regulatory decisions. He has published over 300 papers and several books. He was the editor-in-chief of *Environment International* and *Waste Management* and editor-in-chief of *Technology*, traces its roots to the *Journal of The Franklin Institute*, one of America's oldest continuously published journals of science and technology; Dr. Moghissi is a member of the editorial board of several other scientific journals. He is a member of the Advisory Committee of the Environmental Engineering Division of the American Society of Mechanical Engineers. Dr. Moghissi also serves on the U.S. National Commission for UNESCO, a Federal Advisory Committee to the Department of State that provides expert advice to the State Department on issues of Education, Science, Communications and Culture. Dr. Moghissi received his education at the University of Zurich, Switzerland, and Technical University of Karlsruhe, Germany, from which he received a doctorate in physical chemistry.

RICHARD WILSON, D. Phil., 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 Science and International Affairs and of the Center for Middle Eastern Studies at Harvard University. Professor Wilson was Chairman of the Department of Physics at Harvard University and 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 toxicology, epidemiology, public health and safety, nuclear safety, and risk assessment. Professor Wilson's areas of expertise include elementary particle physics, radiation physics, chemical carcinogens, air pollution, ground water pollution by arsenic, and human rights. He is the author of many articles on high energy physics, environmental pollution and risk analysis, including *PARTICLES IN OUR AIR, EXPOSURES AND HEALTH EFFECTS* (with John Daniel Spengler) (Harvard University Center for Risk Analysis, 1986) and *RISK-BENEFIT ANALYSIS* (2nd ed., 2001) (with Edmund A. C. Crouch) (Harvard University Center for Risk Analysis,). Professor Wilson is the author or co-author of more than 880 published papers on subjects including atomic particles, radioactive particle decay, acute toxicity and carcinogenic risk, carcinogenicity bioassays, statistical distributions of

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I declare under penalty of perjury under the laws of the State of New York that the foregoing is true and correct

Dated: May 26, 2010

Martin S. Kaufman