United States District Court,
D. Massachusetts.

Charles SUTERA, Jr., and Fiorella Sutera, individually and as next friend of Charles Sutera III, Plaintiffs,
v.
THE PERRIER GROUP OF AMERICA INC., Great Waters France, and Source Perrier, Defendants.

Civil Action No. 95-12152-PBS.

Sept. 29, 1997.

Plaintiff who suffered from leukemia sued manufacturer of bottled water, alleging that leukemia was caused by exposure to low levels of benzene found in water. Defendant moved to exclude testimony of physician who was offered as expert witness for plaintiff, and for summary judgment. The District Court, Saris, J., held that: (1) physician's testimony that there was probable causal relationship between leukemia and exposure to benzene was not based on reliable scientific evidence, and thus was inadmissible under *Daubert*, and (2) physician was not qualified to offer expert testimony regarding link between benzene and leukemia.

Motion granted.

West Headnotes

[1] Evidence ⚿555.2
157k555.2

Daubert standard for determining admissibility of scientific evidence establishes duty of trial judge to play role of "gatekeeper," insuring that fact-finding process does not become distorted by what is popularly called "junk science." Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[2] Evidence ⚿508
157k508

[2] Evidence ⚿535
157k535

[2] Evidence ⚿555.2
157k555.2

Under *Daubert* standard for admissibility of scientific evidence, expert testimony must satisfy three requirements in order to survive objection: expert must be qualified, expert's testimony must be reliable, and testimony must "fit" facts of case. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[3] Evidence ⚿536
157k536

To be sufficiently qualified to testify expert, witness needs to have knowledge, skill, experience, training, or education in the specific subject for which his testimony is offered. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[4] Evidence ⚿555.2
157k555.2

[4] Evidence ⚿555.4(2)
157k555.4(2)

Reliability requirement of rule governing admission of expert testimony demands that expert's opinion be based on methods and procedures of science, rather than on subjective belief or unsupported speculation. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[5] Evidence ⚿555.2
157k555.2

Factors that court may use to determine reliability of proffered scientific testimony include (1) whether opinion can be or has been tested,(2) whether theory or technique on which opinion is based has been subjected to peer review and publication, (3) technique's known or potential error rate, (4) existence and maintenance of standards controlling technique's operations, and (5) general acceptance. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[6] Evidence ⚿555.2
157k555.2

It is court's obligation in applying factors used to determine reliability of expert testimony to focus solely on expert's principles and methodology, not on conclusions that they generate. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[7] Evidence ⚿508
157k508

"Fit" requirement of rule governing admissibility of

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expert testimony refers to necessity of a connection between expert's testimony and facts of case. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[8] Evidence ⚿555.5
157k555.5

Where expert seeks to testify regarding cause of injury, it is court's responsibility to assure that opinion is properly grounded in fact. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[9] Evidence ⚿555.5
157k555.5

Expert opinion as to cause of injury must provide more than unsubstantiated conclusions in order to be admissible.

[10] Evidence ⚿555.10
157k555.10

Physician's testimony that there was probable causal relationship between plaintiff's leukemia, and his exposure to benzene through consumption of bottled water in which low levels of benzene were found, was not based on reliable scientific evidence, and thus was inadmissible under *Daubert* in products liability action against bottler of water; no studies were presented which associated plaintiff's leukemia with level of his benzene exposure, mechanistic studies relied on did not explore exposure necessary to trigger cancer, and there was no scientific evidence that no-safe threshold analysis used by physician was acceptable in determining causation. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[11] Evidence ⚿544
157k544

Physician who was oncologist and hematologist, but who had no expertise in epidemiology, toxicology, biostatistics, or risk-assessment, was not qualified to testify as expert witness for plaintiff with respect to causation in products liability action in which plaintiff alleged that his leukemia had been caused by exposure to low levels of benzene contained in bottled water consumed by plaintiff; physician had not done any original research or published any papers about benzene, and his familiarity with literature linking benzene to leukemia was quite limited. Fed.Rules Evid.Rule 702, 28 U.S.C.A.

[12] Food ⚿25
178k25

Plaintiff who suffered from leukemia failed to produce reliable scientific evidence tending to show causal link between leukemia and low levels of benzene found in bottled water consumed by plaintiff, as required to recover on products liability claim against bottler of water.

***656** Evan T. Lawson, Lawson & Lawson, Boston, MA, for Plaintiffs.

Arnold P. Messing, Choate, Hall & Stewart, Boston, MA, Joseph A. D'Avanzo, Michael A. Cerussi, Jr., Curt D. Marshall, Cerussi & Spring, White Plains, NY, James M. Campbell, Pavel Wonsowicz, Campbell, Campbell & Edwards, Boston, MA, Ronald S. Rolfe, Colin Underwood, Cravath, Swaine & Moore, New York City, for defendants.

MEMORANDUM AND ORDER

SARIS, District Judge.

I. *INTRODUCTION*

Plaintiff, Charles Sutera, Jr., [FN1] claims that his regular consumption of Perrier sparkling mineral water caused him to contract acute promyelocytic leukemia ("APL") because it was contaminated with benzene, a known carcinogen.

> FN1. The other plaintiffs are his wife and son. The complaint contains claims based on strict liability, breach of express warranty, breach of implied warranty, negligent infliction of emotional distress and civil conspiracy.

To support Sutera's allegation that the exposure to benzene in the Perrier water caused his disease, plaintiff has offered the opinion of Robert Jacobson, M.D., an oncologist at Good Samaritan Hospital in West Palm Beach, Florida, where he serves as the Medical Director of the Cancer Institute, and a board certified hematologist. Specifically, Dr. Jacobson espouses a "no-threshold" theory of causation. He believes that, because benzene is a known mutagenic carcinogen, ***657** any level of exposure to benzene, including the trace amounts found in Perrier water during the relevant time period, probably did cause Sutera's leukemia.

Defendants, Perrier Group of America, Great Waters of France and Source Perrier (collectively "Perrier"), have filed a motion to exclude plaintiff's

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expert testimony that the exposure to benzene caused plaintiff's leukemia as scientifically unreliable pursuant to Fed.R.Evid. 702. *See Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 592-93, 113 S.Ct. 2786, 2796- 97, 125 L.Ed.2d 469 (1993). Envisioning success on the *Daubert* argument, defendants have styled the motion as one for summary judgment based on the lack of a triable issue of fact in regard to causation.

The Court held an evidentiary hearing on February 28, 1997 during which testimony was given by Dr. Jacobson and Richard Irons, Ph.D., defendant's expert, a board certified toxicologist. Dr. Irons is the Director of the Molecular Toxicology and Environmental Health Sciences Program at the University of Colorado and leader of the University's Cancer Causation Program. As part of the *Daubert* inquiry, the Court also considered materials submitted in connection with the motion for summary judgment. Defendants rely on the written opinions and testimony of Dimitrios Trichopolous, M.D., retired chairman of the Department of Epidemiology at the Harvard University School of Public Health and now a professor of epidemiology and cancer prevention; Ralph O. Wallerstein, M.D., a hematologist, board certified in internal medicine; and Martyn T. Smith, Ph.D., professor of toxicology at the School of Public Health at the University of California at Berkeley. Plaintiffs submitted the testimony of Dr. Richard Clapp, an associate professor in the Department of Environmental Health at Boston University School of Public Health, as a rebuttal witness. This Court ***ALLOWS*** the defendants' summary judgment motion on the ground that plaintiffs submitted no reliable scientific evidence that Sutera's exposure to benzene by drinking Perrier had a causal relationship to his APL.

II. *FACTUAL BACKGROUND*

The Court treats the following facts as undisputed.

A. *Perrier*

Perrier manufactures, advertises, sells and distributes carbonated water for human consumption. Perrier's beverage is marketed as a natural, safe alternative to ordinary tap water. Perrier sparkling mineral water, which is processed both in "regular" (unflavored) and various artificial flavors, can be purchased at retail grocery stores throughout the United States and abroad.

In February 1990, the U.S. Food and Drug Administration ("FDA") authorized a recall of certain flavors of Perrier water when tests revealed that some of the product lines produced between January 1989 and February 1990 contained trace amounts of benzene, a known carcinogen. U.S. Environmental Protection Agency ("EPA") guidelines set the permissible level of benzene in drinking water at five parts per billion ("ppb"). [FN2] Because the agency has estimated that 10 ppb benzene in drinking water causes a risk of one additional cancer per every 100,000 persons, it has also set a goal of 0 ppb for benzene in drinking water. In 1990, some lots of Perrier sparkling mineral water were found to contain up to 28 ppb of benzene. Although the FDA determined that "[a]n acute risk from consumption of the benzene contaminated [Perrier] water does not exist," it did find that "chronic exposure over a lifetime would pose an increase in the risk of cancer." Accordingly, the agency allowed a voluntary "Class II" recall of Perrier water, a recall so classified because it is "a situation in which the use of, or exposure to, a violative product may cause temporary or medically reversible adverse health consequences or *where the probability of serious adverse health consequences is remote.*" [FN3]

FN2. By way of contrast, OSHA sets a guideline of one part per *million* (1 ppm) in air for benzene exposure in the workplace.

FN3. Letter from FDA to Perrier of 2/23/90, at 1 (emphasis added).

***658** Prior to 1989, none of the regular tests that various government agencies performed on Perrier's mineral water had shown the presence of benzene in the water. Moreover, no benzene was detected in the Perrier water that was tested by experts commissioned by plaintiffs in January 1993.

B. *Sutera*

Charles Sutera Jr., a traveling salesman, drank two to three 25-ounce bottles of Perrier sparkling mineral water every day from the spring of 1978 until February 1990. Sutera stopped consuming Perrier early in 1990, when he learned about the nationwide product recall. For the purpose of this motion, the relevant period of Sutera's ingestion of Perrier water is fourteen months-- from January 1989 until February 1990--the period during which tests indicated that some Perrier production lines were benzene-contaminated.

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In September 1992, approximately two and one-half years after he ceased drinking Perrier, Sutera began to experience health problems such as bleeding gums, extreme fatigue, joint pain, flu-like symptoms, severe headaches, spleen aches, and neck palpitations. A routine blood test administered in early October revealed that Sutera had leukemia. He was immediately admitted to the hospital where he was later diagnosed with acute promyelocytic leukemia ("APL").

C. *Leukemia*

Leukemia is a disease that arises in the bone marrow when the blood corpuscles (i.e., cells) undergo a change that makes them malignant. [FN4] Normal bone marrow produces red cells, white cells and platelets in a process called hematopoiesis. Normal hematopoieses begins with a "pluripotent" stem cell [FN5] which divides into lymphoid and myeloid cells. In the marrow, there are feeding support cells, including fat cells and fiberplasts. The stem cell has been likened to a "seed," the growth of which is nurtured by the "soil" of the feeding support cells within the bone marrow environment. Leukemia involves a malignant mutation of stem cells in the bone marrow; accordingly, the disease is sometimes referred to as a hematologic (blood- forming) malignancy.

FN4. A malignant cancer cell is one that is able to "(1) reproduce in defiance of normal restraints, and (2) invade and colonize territories normally reserved for other cells." Bruce Alberts et al., *Molecular Biology: The Cell* 1256 (3rd ed.1994).

FN5. Stem cells that are hemopoietic (blood-forming) are the cells at the very early stages of blood cell development; they are the biological precursors to the formation of blood cells. Alberts at 1161-62. Pluripotent stem cells, which are found in the bone marrow, are the common ancestor of all blood cell types. A pluripotent stem cell gives rise to committed progenitor stem cells, such as lymphoid progenitors and myeloid progenitors, which are cells that will eventually produce certain blood cell types. *Id.*

Two general subtypes of leukemia--myeloid and lymphoid--are differentiated on the basis of the type of precursor cell that undergoes the malignant change. The first general subtype, lymphoblastic or lymphocytic leukemias, are mutations of lymphoid stem cells. The other general subtype, acute myeloid leukemia ("AML"), of which there are seven sub-variants, [FN6] involves malignant transformations at the very early stages of myeloid stem cell development. The latter subtype is the one at issue in this litigation.

FN6. Sometimes AML is called acute non-lymphatic leukemia (ANLL). During the 1980s, the French-American-British ("FAB") classification system was developed, and the seven specific sub-variants of AML--ranging from M1 to M7--were born. Under this system, APL is known as "M3", or "hypergranular promyelocytic" sub-variants of AML. Prior to the 1980s, AML was not specifically subdivided in studies or other scientific literature.

Scientists believe that the leukemic changes in the stem cells that are characteristic of all leukemias are the result of DNA instability. When the DNA in a cell becomes unstable, it can lead to the uncontrolled cell growth with chromosomal instability that is characteristic of malignancy. One type of DNA transformation that has been observed in humans with leukemia, particularly APL, Sutera's specific medical condition, is a chromosomal translocation. A translocation is the switching of two different sections of genetic material between two DNA strands.

***659** APL is one of the seven sub-variants of AML. APL is an early stem cell mutation that affects granulocyte blood cells. It is characterized by prominent granules in immature cells, not seen in other AMLs, and by a unique translocation that takes place between chromosomes 15 and 17. Other sub-variants of AML affect the production of other types of blood cells that are produced by the myeloid chain. APL is an extremely rare form of leukemia that comprises a small percentage of the overall incidence of AMLs--only ten percent of all acute myeloid leukemias are categorized as APL.

While the vast majority of the DNA changes that occur in myeloid stem cells occur spontaneously and are idiopathic [FN7] (cause unknown), there are three specific environmental situations in which such DNA changes have been found to occur: (1) when a person has been exposed to ionizing radiation; (2) when a person has received certain therapies, such as chemotherapy; and (3) when a person has been exposed to high concentrations of certain industrial chemicals, such as benzene.

FN7. Dr. Jacobson estimates that 60 percent of all leukemias are idiopathic.

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D. *Benzene*

The chemical benzene is a colorless liquid that is found in the environment (air, water, soil) both as a result of human activity and due to natural processes. Many industries use benzene to produce other chemicals and to manufacture products such as styrofoam, synthetic fibers, rubber, dyes and detergents. Because benzene is made from petroleum, it is also a component of crude oil and gasoline, and it can be derived from natural sources such as forest fires and volcanoes. [FN8]

FN8. *See* U.S. Dep't of Health and Human Servs., *Toxicological Profile for Benzene* 1.1 (1993) [hereinafter, *Toxicological Profile].*

The major sources of benzene exposure for humans are tobacco smoke, exhaust from motor vehicles and industrial emissions. [FN9] People are also exposed to benzene through food, beverages or drinking water. [FN10] Benzene exposure through air is typically measured in parts of benzene per million parts of air ("ppm") or parts of benzene per billion parts of air ("ppb"). [FN11] The same is true of benzene in water. [FN12] Background levels of benzene to which humans are exposed daily through the air or otherwise range from 2.8 ppb to 20 ppb. [FN13]

FN9. Most people are exposed to benzene daily through the air. "Auto exhaust and industrial emissions account for about 20 percent of the entire nationwide exposure to benzene results from smoking tobacco or from exposure to tobacco smoke." *Toxicological Profile,* at 1.3.

FN10. Exposure through food and drink is typically at a lower level than exposure through the air. For example, typical drinking water contains less than 0.1 ppb of benzene.

FN11. One ppm is 1,000 times greater than one ppb.

FN12. Exposure levels measured in ppm or ppb represent the *percentage* of the total amount of the ingested air/water that is comprised of the toxic agent. For example, the measure 28 ppb means that for every billion parts of water there are 28 parts of benzene. Measurements in parts per billion should not be confused with measuring the actual micrograms of benzene that are present in the air/water. The former is a percentage which does not change no matter how much water/air is being consumed; the latter is cumulative.

FN13. It must be noted that the average smoker takes in about 10 times the amount of benzene of the average nonsmoker.

Whether it is inhaled or ingested, [FN14] benzene is a known human carcinogen and an established cause of AML. After the chemical is absorbed by the body, it is converted to products called metabolites. Although many benzene metabolites leave the body in urine, some may be stored in bone marrow and fat. Phenol and guinones, two benzene metabolites made in the liver, are concentrated in the bone marrow. Some studies suggest that an enzyme in the bone marrow has the capability of converting these metabolites into toxic agents in the cell. Scientists have found that, so stored, benzene metabolites can damage the chromosomes in cells in bone marrow, ***660** causing a host of health problems, including leukemia.

FN14. The rate of absorption of inhaled benzene is approximately 50 percent--half of the benzene taken in through the air is exhaled. According to Dr. Jacobson, gastro-intestinal absorption of ingested benzene is at a rate of more than 90 percent.

Although studies have shown both that chronic exposure to high levels of benzene (*e.g.,* through workplace air) may cause cancer and that brief exposure to very high levels of benzene in air (*e.g.,* 10,000--20,000 ppm) can cause death, lower levels (100--3,000 ppm) that are inhaled briefly have only temporary health effects such as drowsiness, dizziness and headaches. "The health effects that may result from eating or drinking foods containing lower levels of benzene are not known." [FN15]

FN15. *Toxicological Profile,* at 1.5.

The quantitative literature for exposure to high concentrations of benzene in an occupational setting suggests a mean latency in individuals so exposed of 13.2 years. Latency means the period between first exposure and development of the disease. The evidence concerning latency of only a few years is sparse. However, some experts, like Dr. Smith, believe the latency period can be as low as six months.

E. *Scientific Thresholds*

A scientific "threshold" is a figure that represents the exposure level at which a potentially toxic agent is

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likely to have an adverse health effect. Exposure thresholds are often expressed as the product of the exposure level (ppm or ppb) and the duration of such exposure. For example, if scientists set a benzene threshold at 10 ppm-years, a person is likely to experience adverse health problems if she is exposed to 1 ppm of benzene for 10 years, or 2 ppm of benzene for 5 years, or 10 ppm of benzene for one year. Each scenario is equivalent (and all represent a 10 ppm-year threshold), since the product of the exposure level and the duration in each case equals 10.

An important corollary to the concept of a threshold level beyond which a substance causes appreciable adverse health effects is the "dose-response" theory of exposure. Simply stated, many toxicologists believe that the dose (amount) of a substance is directly related to the biological effect that the substance has on the human body, and that every substance can be toxic at a sufficiently high dose. The dose-response theory is at the heart of the concept of an exposure threshold for benzene because the theory assumes that there is a level of benzene to which a person can be exposed below which no health problems will occur.

F. *No-Threshold Model*

Some scientists and medical professionals eschew the concept of dose- response (*i.e,* of threshold), arguing instead that any level of exposure to a toxic agent such as benzene is sufficient to cause leukemia. This model is premised on a "no safe level" assumption. It is sometimes called the "no- threshold" or "linear model."

Applying the "linear no-threshold" model of causation to benzene toxicity leads to the conclusion that virtually any exposure to benzene could cause chromosomal damage and increase the chance of cancer. In this view, any dose could have the risk of yielding negative health consequences.

III. *DISCUSSION*

A. *Legal Standard*

[1] The Supreme Court's decision in *Daubert,* 509 U.S. at 594-95, 113 S.Ct. at 2797-98, which interprets Fed.R.Evid. 702, "establishes the duty of a trial judge to play the role of a gatekeeper' insuring that the fact-finding process does not become distorted by what is popularly called 'junk science.' " *Whiting v. Boston Edison Co.,* 891 F.Supp. 12, 24 (D.Mass.1995)(citing *Wilson v. City of Chicago,* 6 F.3d 1233, 1238 (7th Cir.1993), *cert. denied,* 511 U.S. 1088, 114 S.Ct. 1844, 128 L.Ed.2d 470 (1994)). "This role is especially sensitive in cases 'where the plaintiff claims that exposure to a toxic substance caused his injury, [because a] jury may blindly accept an expert's opinion that conforms with their underlying fears of toxic substances without carefully understanding or examining the basis for that opinion.' " *Whiting,* 891 F.Supp. at 24 (quoting *O'Conner v. Commonwealth Edison Co.,* 807 F.Supp. 1376, 1391 (C.D.Ill.1992), *aff'd,* 13 ***661** F.3d 1090 (7th Cir.1994), *cert. denied,* 512 U.S. 1222, 114 S.Ct. 2711, 129 L.Ed.2d 838 (1994)) (alteration in original)

[2] "After *Daubert,* expert testimony must satisfy three requirements in order to survive a Rule 702 objection: first, the expert must be qualified; second, the expert's testimony must be reliable; and third, it must 'fit' the facts of the case." *Grimes v. Hoffmann-LaRoche, Inc.,* 907 F.Supp. 33, 34 (D.N.H.1995) (citing *United States v. Shay,* 57 F.3d 126, 132 (1st Cir.1995)).

[3] To be sufficiently qualified to testify as an expert, a witness needs to have "knowledge, skill, experience, training, or education," Fed.R.Evid. 702 , "in the specific subject for which his testimony is offered." *Whiting,* 891 F.Supp. at 24. "Just as a lawyer is not by general education and experience qualified to give an expert opinion on every subject of the law, so too a scientist or medical doctor is not presumed to have expert knowledge about every conceivable scientific principle or disease." *Id.*

[4][5][6] "Rule 702's reliability requirement demands that the expert's opinion be based on the methods and procedures of science' rather than on subjective belief or unsupported speculation.' " *Grimes,* 907 F.Supp. at 35 (quoting *In re Paoli R.R. Yard PCB Litig.,* 35 F.3d 717, 742 (3rd Cir.1994), *cert. denied,* 513 U.S. 1190, 115 S.Ct. 1253, 131 L.Ed.2d 134 (1995)) (internal quotation marks omitted). Factors that a court may use to determine the reliability of scientific testimony include:

> (1) whether the opinion can be or has been tested; (2) whether the theory or technique on which the opinion is based has been subjected to peer review and publication; (3) the technique's known or potential error rate; (4) the existence and maintenance of standards controlling the technique's operations; and (5) "general acceptance."

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Grimes, 907 F.Supp. at 35 (citing *Daubert,* 509 U.S. at 593-94, 113 S.Ct. at 2796-97). It is the court's obligation in applying these factors to focus "solely on [an expert's] principles and methodology, not on the conclusions that they generate." *Daubert,* 509 U.S. at 595, 113 S.Ct. at 2797.

[7][8][9] Finally, Rule 702's "fit" requirement "refers to the necessity of a connection between the expert's testimony and the facts of the case." *Grimes,* 907 F.Supp. at 35. Where, as here, an expert seeks to testify regarding the cause of an injury, it is the court's responsibility to assure that the opinion is properly grounded in fact. *See Vadala v. Teledyne Indus., Inc.,* 44 F.3d 36, 39 (1st Cir.1995); *accord Fedorczyk v. Caribbean Cruise Lines, Ltd.,* 82 F.3d 69, 75 (3rd Cir.1996) (indicating that, in order to transcend guesswork, expert testimony needs to be based on direct or circumstantial evidence of the relevant facts). An expert opinion as to the cause of injury must provide "more than unsubstantiated conclusions." *Hayes v. Douglas Dynamics, Inc.,* 8 F.3d 88, 94 (1st Cir.1993), *cert. denied,* 511 U.S. 1126, 114 S.Ct. 2133, 128 L.Ed.2d 863 (1994).

B. *Dr. Jacobson's Expert opinion*

Based on the facts of Sutera's case and six cited references, Dr. Jacobson has rendered an expert opinion that Sutera's "type of leukemia, the amount and duration of exposure and the latency period ... are all consistent with benzene having an etiologic role." He states: "[i]t is my considered opinion, from the information provided, that there is sufficient evidence to conclude there was a probable causal relationship between Mr. Sutera's benzene exposure and his developing acute promyelocytic leukemia." His expert opinion contained four numbered observations to support causation:

First, citing writings by Aksoy [FN16] and Mehlman, [FN17] Dr. Jacobson states: "Acute promyelocytic leukemia has been reported to occur in individuals exposed to benzene. Acute non-lymphocytic leukemias arising from myeloid (granulocyte) stem cells are the commonest types of leukemias reported to ***662** develop in benzene exposed workers." (References omitted.)

> FN16. Muzaffer Aksoy et al., *Leukemia in Shoe-Workers Exposed Chronically to Benzene,* 44 Blood 837 (1974).

> FN17. Myron A. Mehlman, *Benzene Health Effects: Unanswered Questions Still Not Addressed,* 20 Am. J. Indus. Med. 707 (1991).

Second, referring to the *Toxicological Profile* and a work by Infante, [FN18] Jacobson reports that animal and human studies provide "abundant evidence to support that benzene is toxic to the bone marrow."

> FN18. Peter F. Infante et al., *Benzene in Petrol: A Continuing Hazard,* 336 Lancet 814 (1990).

Third, Jacobson explains that benzene is "readily absorbed" by the body when ingested. He cites an animal study by Sabourin [FN19] in which approximately 97 percent of the benzene dose administered to mice and rats was absorbed by their gastrointestinal tracts.

> FN19. Patrick J. Sabourin et al., *Effect of Dose on the Absorption and Excretion of Benzene Administered Orally or by Inhalation in Rats and Mice,* 87 Toxicology & Applied Pharmacology 325 (1987).

Fourth, Jacobson points to studies like the Sabourin study which suggest that mice and rats exposed to benzene exhibit chromosomal damage in bone marrow cells after fairly brief periods of exposure (ranging from four hours to nine days) at "fairly low doses" of benzene (ranging from 6 ppm to 28 ppm).

C. *Daubert*

In applying the *Daubert* factors, pursuant to Fed.R.Evid. 702, I conclude that Dr. Jacobson's testimony must be excluded on the grounds [FN20] that (1) Dr. Jacobson's opinion on causation is not based on reliable scientific evidence; and (2) Dr. Jacobson is not qualified to render an expert opinion regarding whether the plaintiff's exposure to low doses of benzene caused Sutera's illness.

> FN20. In light of this conclusion, I need not address Perrier's other arguments that Dr. Jacobson's opinion does not fit the facts of this case because he did not consider other exposures like cigar smoke.

1. *Scientific Evidence*

[10] Rather than addressing Dr. Jacobson's four numbered observations in turn, the Court finds it more useful to the issue of reliability to consider the three different types of evidence he offered to support

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his theory.

a. *Epidemiological Studies*

Plaintiffs have produced no scientific peer-reviewed epidemiological studies which would associate APL, the specific disease from which Sutera suffers, and benzene exposure at the 5 to 28 ppb level (the level of benzene Sutera was exposed to by drinking contaminated Perrier). At the hearing, Dr. Jacobson specifically denied knowledge of any study--"an epidemiological study, an animal study, a meta analysis, [or] a mechanistic study"--in existence today "that demonstrates an increased or elevated risk of a blood malignancy, any blood malignancy, including APL, to a human being or to a population of human beings from an exposure of five parts per billion in drinking water." (Tr. 64.)

The principal studies to which Dr. Jacobson referred simply do not demonstrate any scientific support for the proposition that Sutera's ingestion of benzene at low levels in Perrier drinking water caused his APL. The Aksoy study involved exposures by shoe workers to benzene by inhalation in the range of 210 ppm to 650 ppm for durations ranging from one to fifteen years. The duration of exposure to the single patient with APL was 2.5 years. Similarly, the dosage and duration of exposure of the pliofilm workers in the Infante and White [FN21] study were also "substantially greater in orders of magnitude" than Sutera's exposure. (Tr. 148.) The levels of benzene inhalation estimated by Infante and White were no lower than 10 ppm and as high as 210 ppm for at least eight hours a day over a period of years. This epidemiological study contained no instances of APL. At the hearing, Dr. Jacobson cited a case study by a group of Italian doctors headed by E.C. Vigliani. (Tr. 86.) These scientists found that leukemia occurred among factory workers who were exposed to concentrations of benzene in air ranging from 25 ppm (25,000 ppb) to 600 ***663** ppm (600,000 ppb) for a period of one to 46 years. Only one patient out of 24 arguably had APL.

FN21. Patrick F. Infante & Mary C. White, *Projections of Leukemia Risk Associated with Occupational Exposure to Benzene,* 7 Am. J. Indus. Med. 403 (1985).

By contrast, defendants' experts soundly established the dearth of any epidemiological evidence supporting a causal relation between exposures to benzene at low levels and APL. Dr. Irons, defendants' expert, testified that there is no evidence in the literature that exposure to 28 ppb of benzene for a *lifetime* is associated with any adverse health affect; that there is no quantitative evidence of an increased incidence of APL in benzene-exposed populations; [FN22] and that the evidence concerning the adverse carcinogenic effects of benzene involves exposures that are "thousands of fold higher" than those that are at issue in this case. He testified that there is a consensus among scientists that a threshold of 100 ppm-years (or greater than 100,000 ppb-years) of chronic exposure to benzene in air will cause leukemogenesis, and that scientists reasonably disagree over the carcinogenic effects of doses between 25 and 50 ppm-years. [FN23] (Tr. 196-97.)

FN22. Dr. Smith agrees that there are no published epidemiological studies which contain a dose response estimate showing that benzene causes APL. Although plaintiff attempts to equate APL with AML because one is the sub-variant of the other, he points to no scientific basis for doing so.

FN23. The peer-reviewed studies demonstrate the range of disagreement. Dr. Paxton wrote: "this is consistent with the hypothesis that exposure in excess of some threshold value, here suggested to be greater than 50 ppm-years is necessary for leukemogenesis." Mary B. Paxton et al., *Leukemia Risk Associated with Benzene Exposure in the Pliofilm Cohort,* 14 Risk Analysis 147, 153 (1994). Dr. Wong, who would apparently disagree with Irons' view of the consensus, wrote: "The estimated threshold specific to AML was found to be at least 200 ppm-years based on one set of exposure estimates; and much higher, had other exposure estimates (most likely more accurate) been used." Otto Wong, *Risk of Acute Myeloid Leukemia and Multiple Myeloma in Workers Exposed to Benzene,* 52 Occupational & Environmental Med. 380, 383 (1995).

Defendant's expert epidemiologist Dr. Trichopolous reviewed the scientific literature on leukemia contracted after prolonged exposure to high concentrations of benzene in the air and found that, "although there is evidence for an increased occurrence of certain types of leukemia, particularly non-lymphocytic leukemias (which includes, in several classification systems, acute promyelocytic leukemia), there is no empirical evidence that the

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likelihood of acute promyelocytic leukemia per se increases following exposure to benzene." (Trichopolous Aff. ¶ 1.)

Finally, defendant advanced the Tsai [FN24] peer-reviewed epidemiological study as most reflective of the exposure in this case. The Tsai study of refinery workers found that no workers developed leukemia after exposure to low levels of benzene. [FN25] The exposure in this study ranged from 140 ppb to 530 ppb and the duration of the exposure was for periods of one to twenty-one years. The contrast of the figures in defendants' studies to this case is striking, since the maximum level of exposure Sutera experienced was 28 ppb for slightly over one year.

FN24. Shan P. Tsai, *Retrospective Mortality and Medical Surveillance Studies of Workers in Benzene Areas of Refineries,* 25 J. Occupational Med. 685 (1983).

FN25. *Id.* at 690.

b. *Mechanistic Evidence*

Plaintiff also relies on mechanistic peer-reviewed studies to support his causation analysis, but such evidence does not support plaintiff's no-threshold theory because it does not explore the exposure necessary to trigger the cancer-producing mechanism. The mechanistic evidence fails for at least three reasons. First, the levels in the mechanistic studies which plaintiff introduced into evidence are significantly higher than the 28 ppb contained in the Perrier water. For example, the mouse study by Kolachana [FN26] was an "extremely high" dose study. (Tr. 199.) The equivalent for a human being would be a one-day exposure to inhalation of 466 ppm. Second, the Levay study [FN27] is of ***664** little direct relevance to whether benzene causes APL at low doses because it involves a human APL cell line (HL-60), *i.e.*, the cells were already cancerous at the time the studies were performed. (Tr. 200.) The Levay study involved high doses of benzene metabolites administered to a cancer cell line and was designed to address the mechanism of leukemogenesis by studying the effect of combinations of benzene metabolites in binding directly to DNA. (Tr. 204-05.) In other words, the study does not address the degree of benzene exposure necessary to *create* the genotoxic effects in bone marrow. Third, the studies fail to prove the mechanics of absorption relevant to this case. The Sabourin study concluded that "the total metabolite formation was exponentially related to the benzene exposure concentration" in experiments with rats and mice. Dr. Jacobson believes this study is significant because it demonstrates that 97% of certain benzene doses in rats and mice was absorbed in their gastrointestinal tracts. [FN28] However, the record is unclear as to how to relate the dosages administered to these species to plaintiff's exposure.

FN26. Prema Kolachana et al., *Benzene and Its Phenolic Metabolites Produce Oxidative DNA Damage in HL 60 Cells in Vitro and in the Bone Marrow in Vivo,* 53 Cancer Res. 1023 (1993).

FN27. Gyorgy Levay & William J. Bodell, *Potentation of DNA Adduct Formation in HL-60 Cells by Combinations of Benzene Metabolites,* 89 Proc. Nat'l Acad. Sci. U.S.A. 7105 (1992).

FN28. This is consistent with Dr. Smith's opinion that a person would absorb about 90% of benzene consumed in water. (Dep. Tr. 19.)

Along these lines, plaintiff further argues that the evidence of Sutera's 15/17 chromosomal translocation mechanistically supports his theory of causation because benzene metabolites are known to cause specific chromosomal translocations, including the 15/17 translocation. Dr. Smith testified that "it's very likely that benzene metabolites could cause a translocation between chromosomes 15 and 17." (Smith Dep. 62.) Dr. Wallerstein testified that the 15/17 translocation is common in virtually all patients with promyelocytic leukemias, including both those who have been exposed to chemical solvents and metals and those who have not. (Wallerstein Dep. 89). While Dr. Wallerstein and Dr. Smith agreed that benzene metabolites can cause a chromosomal translocation between chromosomes 15 and 17, both agreed that the mere fact that Sutera has this translocation is insufficient to prove that it was caused by the exposure to the Perrier water. (Smith Dep. 62-63; Wallerstein Dep. 98.) As with all of plaintiff's mechanistic evidence, the translocation of chromosomes is insufficient to support causation.

c. *Risk Assessment Approach*

Dr. Jacobson embraces the no-safe level model for benzene exposure based on a risk assessment approach. He states: "I do not believe there is a threshold level of risk for highly toxic leukemogenic agents such as benzene. Indeed, the toxic history of benzene has been to repeatedly adjust the levels that

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were considered safe downward." He relies on four pieces of evidence as reliable scientific evidence of the no-threshold approach.

First, Dr. Jacobson relies on the EPA's regulation which sets the permissible level of benzene in drinking water at 5 ppb, and its goal of 0 ppb for benzene in drinking water. However, a regulatory standard, rather than being a measure of causation, is a public-health exposure level that an agency determines pursuant to statutory standards set by Congress. *See, e.g., Industrial Union Dep't, AFL-CIO v. American Petroleum Inst.*, 448 U.S. 607, 630-32, 100 S.Ct. 2844, 2858-59, 65 L.Ed.2d 1010 (1980) (rejecting OSHA regulatory standards that reduced exposure from 10 ppm to 1 ppm on the ground that there was insufficient evidence of a significant risk of harm). The Fifth Circuit helpfully distinguished between OSHA regulations, on the one hand, and causation in a particular case, on the other, when it noted that a regulator's purpose is to "suggest or make prophylactic rules governing human exposure ... from the preventive perspective that agencies adopt in order to reduce public exposure to harmful substances." *Allen v. Pennsylvania Eng'g. Corp.*, 102 F.3d 194, 198 (5th Cir.1996). In doing so, the "agencies' threshold of proof is reasonably lower than that in tort law, which 'traditionally make[s] more particularized inquiries into cause and effect' and requires a plaintiff to prove that it is more likely than not that another individual has caused him or her harm.' " *Id.* (quoting *Wright v. Willamette Indus., Inc.*, 91 F.3d ***665** 1105, 1107 (8th Cir.1996)) (alteration in original). In other words, the fact that an agency, ex ante, sets an exposure standard of 5 ppb for drinking water does not compel, or even necessarily support, the ex post conclusion that Sutera's leukemia was caused by Perrier.

Dr. Smith explains why a violation of a safety regulation does not, without more, suffice for reliable scientific evidence of causation with respect to any given individual who has contracted leukemia as follows. There are about 80,000 new cases of leukemia and lymphoma in the United States each year. (Smith Dep. 35.) Using the risk assessment approach, the EPA estimates that environmental benzene exposure only contributes four cases of those 80,000. (*Id.* at 36.) With respect to Sutera's exposure to Benzene in the Perrier, his excess cancer risk above the risk from the background environmental exposure is "of the order of a one-in-a-million excess risk." (Smith Aff. ¶ 5.) Plaintiff's toxicologist, Dr. Diane Silverman, using EPA's risk assessment methodology, agreed that the excess cancer risk from plaintiff's bottled water dose at 28 ppb was in the range of one excess cancer risks over and above the background cancer risk in one million exposed people assuming an exposure of 3.5 years.

Second, Dr. Jacobson refers to the Mehlman Coxmentary, [FN29] which encourages regulatory agencies to take a tougher stand. Because the data concerning the carcinogenity of benzene are not "yet available" and "we do not know of any safe level above zero," Dr. Mehlman urges setting regulatory guidelines at "current recommended maximum levels of exposure of 0.004 to 0.1 ppm." [FN30] Such commentary is not a valid basis for reliability here for the same reason that regulatory standards generally do not support causation in a given case.

FN29. Mehlman, *supra* note 17.

FN30. Id. at 709.

Third, Dr. Jacobson anchors his opinion on an article by Dr. Smith. [FN31] Dr. Jacobson believes this study provides evidence of the growing acceptance within the scientific community of applying a no-threshold linear analysis to determine whether and to what extent benzene causes leukemia. The article is a review of mechanistic research performed by other scientists in which Smith takes the "admittedly controversial" position that "benzene is a genotoxic carcinogen which poses a risk even a low doses." [FN32] Dr. Smith concludes:

FN31. Martyn T. Smith, *Mechanistic Studies of Benzene Toxicity-- Implications for Risk Assessment, in Biological Reactive Intermediates V* 259 (R. Snyder et al. ed., 1996).

FN32. *Id.* at 259.

> This data suggests that background exposure to benzene and its metabolites in the normal human environment contributes to the background level of leukemia. Further, it suggests that additional benzene exposures at the workplace and elsewhere would cause a linear increase in leukemia risk on this background. In conclusion, the most recent mechanistic data on benzene suggest that benzene is a genotoxic carcinogen that poses a risk, albeit small, even at low doses. Further, the most recent data suggest that the risk posed by additional benzene exposures in the low dose region is most

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likely linear. [FN33]

FN33. *Id.* at 263.

However, Dr. Smith also specifically states (in a section on the implications for risk assessment) that "[f]or the development of leukemia, it is clear that *multiple molecules of benzene metabolites will be needed* and that these metabolites must also target the stem cell in the bone marrow." [FN34] He goes further: "This would imply that benzene has a non-linear dose response curve in the low-dose region." [FN35] While Dr. Smith believes that background levels of exposure to benzene may provide enough initial molecules of benzene metabolites to make additional benzene exposure an increased risk, his article does not lead to the conclusion that there is no threshold for exposure to benzene. Indeed, Dr. Smith asserts that, from background exposure alone, "the number of benzene metabolite molecules reaching ***666** the human bone marrow in a typical human is likely to measure in *the tens of thousands,* " and that only when there is this initial build up would additional benzene exposure ("such as occupational exposure") be likely to contribute linearly to an additional leukemia risk. [FN36]

FN34. *Id.* (emphasis added).

FN35. *Id.*

FN36. *Id.*

Plaintiffs' reliance on Dr. Smith's interesting article is flawed because it was written to provide guidance in the area of risk assessment, not causation. As noted above, Dr. Smith himself disagrees with Jacobson's causation analysis because of his failure to factor in dose. Dr. Smith testified at his deposition that, assuming that Sutera was an average non-smoker and took in around 500 micrograms of benzene a day, which is the national average background exposure to benzene, drinking two liters of water per day containing 28 ppb benzene for fourteen months would present a "minimal increase in risk and therefore couldn't be a substantial contributor to his disease." (Smith Dep. 32.)

Finally, plaintiff finds some solace in the testimony of defense expert Dr. Wallerstein, who conceded in his deposition that the no-threshold hypothesis is an acceptable scientific model. Dr. Wallerstein agreed that a linear model-- whereby a scientist extrapolates from levels where adverse effects occur down to a baseline, and assumes that the risk continues in a straight line--is an acceptable scientific technique, although he disagreed with it. (Dep.19). Harvard epidemiologist Dr. Trichopolous also believed this hypothesis was credible, although not proven. He discusses the "no-threshold theory" as follows:

> The hypothesis that any amount of a particular carcinogen, however small, can increase the risk of the corresponding cancer beyond the baseline level by a similarly small amount is credible, although this has not been unequivocally confirmed or refuted. According to a recent estimate advanced by the World Health Organization, an increase of benzene concentration in the drinking water by 10 ug/liter is expected to increase the risk of cancer by 0.00001 (or 10-5 or 0.001 percent). Even if this risk is concentrated in the myeloid leukemia group, which has a cumulative risk among caucasian USA men of 0.39 percent, the incremental risk associated with continuous drinking of Perrier water containing benzene in concentration of 28 ug/liter would be an additional 0.003 percent for a total of 0.393. Therefore, for a man drinking this type of water and developing myeloid leukemia (like Mr. Sutera), there would be less than 1% chance (0.003/ 0.393 = 0.008 or 0.8 percent) that his disease was due to benzene in his drinking water. That occurrence of myeloid leukemia in Mr. Sutera at a relatively young age is balanced by the fact that consumption of water containing 28 ug of benzene per liter was limited to 13 years of the preceding life. [FN37]

FN37. Written Testimony of Dimitrios Trichopolous, at 4.

In sum, he concludes: "Even when theoretical models of no-threshold carcinogenicity of benzene were invoked, the likelihood that the disease of the plaintiff (leukemia) was due to benzene-contaminated Perrier water would be no more than 1 per cent." [FN38]

FN38. Plaintiff's rebuttal expert, Dr. Richard Clapp, an epidemiologist, disagreed with this conclusion, stating: "The scientific literature supports the contention that Mr. Sutera disease was, to a reasonable degree of scientific certainty, substantially contributed to by his benzene exposure from drinking contaminated water." However, he does not point to any literature to support his point, and concedes there are no epidemiological studies which show any increase in the incidence of APL due to benzene.

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Accordingly, although there is evidence that one camp of scientists (i.e., Dr. Mehlman and Dr. Smith) believes that a non-linear model is appropriate basis for predicting the risks of low-level exposures to benzene, there is no scientific evidence that the linear no-safe threshold analysis is an acceptable scientific technique used by experts in determining causation in an individual instance. Cf. *City of Greenville v. W.R. Grace & Co.*, 827 F.2d 975, 980 n. 2 (4th Cir.1987) (admitting expert testimony that "in the absence of scientific studies concerning exposure to low levels of asbestos, one technique accepted in the scientific community for predicting the ***667** risks associated with low-level exposures is to extrapolate the risk downward from results obtained in studies involving high level exposures").

In sum, plaintiff has produced no reliable scientific evidence that plaintiff's leukemia was more likely than not caused by his drinking Perrier. Not only does Dr. Jacobson's conclusion fail the general acceptance test, but he offers no testing, peer-reviewed literature or other reliable scientific methodology or basis for his conclusion that the low dose of benzene in Perrier's water was itself a substantial factor in causing Sutera's illness. The linear no-threshold theory of causation so applied "has no known or potential rate of error." *Whiting*, 891 F.Supp. at 25 (finding the non- threshold model unreliable in part because it "cannot be falsified, nor can it be validated"). "It is merely an hypothesis." *Id.* This Court concludes that Dr. Jacobson's analysis flunks all the *Daubert* reliability factors.

2. *Qualifications*

In many cases, resolution of the causation issue turns on the testimony of treating physicians. See *Mason v. Texaco, Inc.*, 741 F.Supp. 1472, 1496 (D.Kan.1990), *aff'd in part*, 948 F.2d 1546 (10th Cir.1991), *cert. denied*, 504 U.S. 910, 112 S.Ct. 1941, 118 L.Ed.2d 547 (1992); *Benedi v. McNeil-P.P.C., Inc.*, 66 F.3d 1378, 1384 (4th Cir.1995) (permitting treating physician to render an opinion that combining alcohol with therapeutic doses of acetaminophen caused liver disease in light of "medical community's daily use of the same methodologies in diagnosing patients").

However, as the *Mason* case correctly pointed out, "individual cases of benzene related leukemia do not lend themselves to the same certainties as do diseases such as asbestosis, whose cause is readily attributable to a particular agent or class of agents." *Mason*, 741 F.Supp. at 1497. As discussed above, there is no scientifically reliable methodology which will "establish an absolute causal link between a given case of leukemia and benzene exposure because there is simply no clinical test or examination that a physician can conduct to make this determination." *Id.* Simply having a medical degree or training is insufficient expertise to establish causation which hinges on factors such as "dosage, duration of dosage and latency periods." *Id.* (pointing out that these parameters "have been established and defined through epidemiological studies"); *see also Whiting*, 891 F.Supp. at 25.

[11] Dr. Jacobson is an oncologist and hematologist who has no expertise in epidemiology, toxicology, biostatistics or risk-assessment. Although he is a clinical physician (mt Mr. Sutera's treating physician) with considerable expertise diagnosing and treating leukemia, the ability to diagnose and to treat a disease is substantially different from the expertise required to assess its genesis to a reasonable degree of scientific certainty. Dr. Jacobson has not done any original research or published any papers about benzene, its properties, or its leukemogenic effects. Moreover, no evidence was presented at the hearing that Dr. Jacobson has ever rendered a scientific opinion regarding whether a toxic agent caused a specific individual's hematologic disorder.

While Dr. Jacobson has reviewed the literature linking benzene to leukemia, his familiarity with it is quite limited. For example, at the hearing he lacked the expertise to compare the dosages of benzene to which animal species were exposed with commensurate dosages to humans. *Cf. Shay*, 57 F.3d at 133 n. 5 (requiring plaintiff to offer reliable expert testimony that results observed in the animal studies are transferable to humans) (citing *Paoli*, 35 F.3d at 743). Moreover, his citation to epidemiological studies did not support his position, and he was unfamiliar with the studies which undercut his opinion.

In sum, Dr. Jacobson lacks the specific knowledge, education, training and experience to render an opinion as to whether the exposures to low levels of benzene in the Perrier for a short time period caused the specific disease at issue in the case.

D. *Defendants' Summary Judgment Motion*

"To succeed [in a motion for summary judgment], the moving party must show that ***668** there is an

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absence of evidence to support the nonmoving party's position." *Rogers v. Fair,* 902 F.2d 140, 143 (1st Cir.1990); *see also Celotex Corp. v. Catrett,* 477 U.S. 317, 325, 106 S.Ct. 2548, 2553-54, 91 L.Ed.2d 265 (1986). "Once the moving party has properly supported its motion for summary judgment, the burden shifts to the non-moving party, who 'must set forth specific facts showing there is a genuine issue for trial.' " *Barbour v. Dynamics Research Corp.,* 63 F.3d 32, 37 (1st Cir.1995) (quoting *Anderson v. Liberty Lobby, Inc.,* 477 U.S. 242, 256, 106 S.Ct. 2505, 2514, 91 L.Ed.2d 202 (1986)), *cert. denied,* 516 U.S. 1113, 116 S.Ct. 914, 133 L.Ed.2d 845 (1996). The Court must "view the facts in the light most favorable to the non-moving party, drawing all reasonable inferences in that party's favor." *Barbour,* 63 F.3d at 36.

[12] The plaintiffs in this case would have the burden of proving at trial that, more likely than not, the benzene in Perrier's bottled water during the relevant time period was a substantial factor in causing Sutera's medical condition. Plaintiffs have offered no reliable scientific evidence tending to show a causal link between Perrier and Sutera's leukemia. *See O'Connor v. Raymark Indus., Inc.,* 401 Mass. 586, 592, 518 N.E.2d 510 (1988) (substantial factor test). There is, thus, no genuine issue of fact as to whether Perrier's water caused plaintiff Sutera's illness.

IV. *ORDER*

Defendants' motion for summary judgment (Docket No. 93) is ***ALLOWED.***

JUDGMENT

In accordance with the Court's Memorandum Order dated September 29, 1997 granting Defendant's motion for summary judgment in the above-entitled action, it is hereby ORDERED:

Judgment for the Defendant.

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