Hydroquinone-Induced Leukemias Are Scientifically Valid, Provable, and Important Toxic Injury Claims

By Greg Coolidge, Esq.1

I. Introduction

Workers and consumers exposed to hydroquinone-containing chemical products who tragically suffer from leukemia constitute a previously unappreciated source of toxic injury actions against the manufacturers and suppliers of these products. Workers at risk include chiropractors, x-ray technicians, photograph developers, and others who have been chronically exposed to x-ray developing and photographic developing chemical products containing hydroquinone. Indeed, this author’s firm is presently litigating the wrongful death action of a chiropractor with no demonstrable exposures to benzene, ionization radiation, or other known causes of leukemia, who developed Acute Myelogenous Leukemia with associated chromosome abnormalities as a result of his nearly twenty-year exposure to hydroquinone-containing x-ray developing products while

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Moreover, in July 2006, a case report entitled “Hydroquinone and/or Glutaraldehyde Induced Acute Myeloid Leukemia” was published in the Journal of Occupational Medical Toxicology, which concerned two x-ray developing technicians who developed Acute Myelogenous Leukemia. The authors concluded that the most likely cause of the technicians’ AML was their chronic exposure to hydroquinone contained in the x-ray developing products they used.

Consumers at risk include persons who have regularly used hydroquinone-containing skin whitening products and cosmetics. As a result of such leukemia concerns, the FDA has moved to effectively prevent the future sale of hydroquinone-containing skin whitening products and cosmetics in the United States, and the European Union has officially banned the use of hydroquinone in skin whitening products and cosmetics.

The scientific proof that hydroquinone causes leukemia in humans is found in abundant published scientific literature, including mechanist studies indicating that hydroquinone is the toxic metabolite of benzene which causes leukemia; published epidemiologic studies concerning benzene exposed workers and leukemia; studies finding that hydroquinone causes leukemia in experimental animals; and human in vitro studies finding that hydroquinone is, itself, genotoxic (damages DNA); is associated with damage to blood cells leading to the development of human leukemia; and causes specific types of chromosome abnormalities in human blood cells exposed to it also regularly found in benzene exposed workers suffering from Acute Myelogenous Leukemia and other leukemias.

The purpose of this article is to therefore inform toxic injury attorneys that hydroquinone-induced leukemias are scientifically valid, provable, and important sources of toxic injury actions for workers and consumers suffering from leukemia. This article also addresses the exposure and causation standards of proof which apply to hydroquinone-leukemia cases in California.
II. An Overview of Hydroquinone, It’s Uses, and Persons At Risk From Exposure to Hydroquinone

Hydroquinone, also known as benzene-1,4-diol or quinol, is an aromatic organic compound which is a type of phenol, having the chemical formula C₆H₄(OH)₂. Its chemical structure has two hydroxyl groups bonded to a benzene ring in a para position. It is a white granular solid at room temperature, and is highly soluble in water.

Hydroquinone is produced industrially in several countries, including the United States. Hydroquinone is used as a reducing agent, as an ingredient in photographic and x-ray developers, as a chemical intermediate for the production of antioxidants, antiozonants, agro-chemicals, and polymers, and as an ingredient in skin whitening products, cosmetics, hair dyes, and various medical preparations.

Consumers who have regularly used hydroquinone-containing skin whitening products and cosmetics, and who have thereby been regularly exposed to hydroquinone through dermal absorption, are at an increased risk of developing leukemia in the future. Hydroquinone has therefore been banned in the European Union since 2001 for use in skin whitening products and other cosmetics as a result of serious concerns over its health hazards, including the development of leukemia and other cancers, resulting from dermal exposure to hydroquinone. Moreover, in August 2006, the U.S. Food and Drug Administration (FDA) proposed a ban on the sale of over-the-counter skin whitening products containing hydroquinone, in part, as a result of the FDA’s concern that hydroquinone is a potential cause of leukemia and other cancers in humans based on existing animal studies. Thereafter, the FDA proposed a rule that, if finalized, would establish that over-the-counter skin bleaching products containing hydroquinone are not generally recognized as safe and are therefore misbranded under FDA regulations. Under this rule, the FDA would also consider all skin bleaching products, whether currently marketed on a prescription or over-the-counter basis, to be new drugs requiring an
approved new drug application (NDA) for continued marketing in the United States. The FDA is therefore proposing to effectively eliminate the sale of hydroquinone-containing skin whitening products in the United States due, in part, to concerns about hydroquinone causing leukemia and other cancers in humans.³

Workers with a leukemia risk from hydroquinone exposure include chiropractors, x-ray technicians, photograph developers, and others who have been chronically exposed to x-ray developing and photographic developing chemical products containing hydroquinone. In these workers, exposure to hydroquinone can occur by the inhalation of airborne hydroquinone vapors airing from developing trays or escaping from x-ray processing machines; by the inhalation of airborne hydroquinone dusts released from dried developer spilled on x-ray processing machines, the floor, or other surfaces of the developing room; and by dermal absorption of hydroquinone resulting from skin contact with spilled developers, from skin contact wet photographs or x-rays, or from skin contact with wet parts of x-ray developing machines during the repair of the machines or other contact with the machines. Indeed, in the case presently being litigated by this author’s firm, the chiropractor who died as a result of Acute Myelogenous Leukemia was chronically exposed to hydroquinone through each of the foregoing routes and sources of exposure for nearly twenty years.

In such cases involving x-ray developers exposed to hydroquinone-containing developing products, it is necessary, if the evidence permits such, to rule out overexposure to ionizing radiation as a cause of the x-ray developer’s leukemia. This is so because overexposure to ionizing radiation is a well-established cause of leukemia, and

³ The California Office of Environmental Health Hazard Assessment (OEHHA) is also currently undergoing an evaluation of hydroquinone to determine whether hydroquinone should by a chemical placed on the Proposition 65 list as a chemical known to the State of California to cause cancer in humans.
because persons who engage in x-ray developing, such as chiropractors, are often the same persons who regularly take the x-rays which will thereafter be developed. This ruling out of ionizing radiation can be accomplished through the introduction of evidence that the x-ray technician always stood behind proper lead shielding when taking x-rays, personal and historical dosimetry readings showing that no overexposures to ionizing radiation occurred, and reports from appropriate regulatory agencies indicating that no overexposures to ionizing were measured during periodic inspections by the agencies.

III. Hydroquinone Is a Cause of Leukemia in Humans

Companies defending hydroquinone-leukemia cases will almost certainly argue that hydroquinone does not cause leukemia in humans, because the three existing published epidemiologic studies of workers in hydroquinone manufacturing facilities and workers involved in motion picture film developing did not find an increased incidence of leukemia among these exposed workers. However, each of these studies lacked sufficient statistical power to detect an increased incidence of leukemia as a result of hydroquinone exposure due to the small populations of exposed workers used in the studies, and the small number of expected leukemias in these groups. The present published epidemiologic studies therefore provide no scientifically valid information for determining whether exposure to hydroquinone causes leukemia in humans.

The scientific proof that hydroquinone is a cause of leukemia is therefore found in other scientific literature, including studies of the biologic mechanism through which benzene causes leukemia in humans; epidemiologic studies of benzene-exposed workers finding an increased incidence of leukemia; studies of animals exposed to hydroquinone which developed leukemia; and human in vitro studies concerning exposure to hydroquinone and the resulting genetic and cellular damage.

Abundant published scientific literature exists discussing the biologic mechanism through which benzene causes leukemia in humans. After being inhaled or absorbed into
the human body, benzene is first metabolized in the liver to benzene oxide, a large proportion of which is then converted to phenol. Phenol is further metabolized to hydroquinone, which is carried throughout the body in the bloodstream. When transported to the bone marrow, hydroquinone ultimately is converted to benzoquinone, which then causes the cellular and genetic damage resulting in leukemia. Thus, benzoquinone, which is the toxic metabolite of hydroquinone, is the critical leukemogenic metabolite of benzene.

For this reason, the voluminous published epidemiologic studies establishing that leukemia in humans is caused by exposure to benzene is, in actuality, voluminous epidemiologic literature which also establishes that exposure to hydroquinone causes leukemia in humans. This is so because the leukemia in each of the workers in these studies exposed to benzene was, as a mechanistic matter, ultimately caused by hydroquinone. When used in conjunction with the published mechanistic literature, the corpus of published epidemiologic studies concerning benzene-exposed workers and leukemia can therefore be used with equal scientific force to establish that exposure to hydroquinone causes leukemia in exposed workers.

There also exists well more than a hundred published, peer-reviewed studies, including animal studies and human in vitro studies, which show that hydroquinone is genotoxic (damages DNA); is clastogenic (breaks chromosomes and causes the loss of chromosomes); causes leukemia in animals experimentally exposed to it; is associated with the damage to blood cells leading to the development of human leukemia; and causes specific types of chromosome abnormalities in human blood cells exposed to it also regularly found in benzene exposed workers suffering from Acute Myelogenous Leukemia (including monosomy 5, 7, 8, and 21; deletions of chromosomes 5, 7, and 8;
translocations of chromosome 21; and others). Additionally, in July 2006, a case report entitled “Hydroquinone and/or Glutaraldehyde Induced Acute Myeloid Leukemia” was published in the Journal of Occupational Medical Toxicology, which concerned two x-ray developing technicians who developed Acute Myelogenous Leukemia. The authors concluded that the most likely cause of the technicians’ AML was their chronic exposure to hydroquinone contained in the x-ray developing products they used.

The existing scientific literature, taken as a whole, therefore clearly establishes that exposure to hydroquinone causes leukemia in humans. Defendants in hydroquinone-leukemia cases are still likely to argue to trial courts that the absence of positive epidemiologic studies concerning workers exposed directly to hydroquinone renders the opinions of plaintiffs’ experts that hydroquinone causes leukemia inadmissible as lacking in foundation. Putting aside the fact that the existing published epidemiologic studies concerning benzene exposed workers and leukemia are highly relevant to establishing that exposure to hydroquinone causes leukemia for the reasons previously discussed herein, there is not a single published California decision which holds that a medical expert rendering an admissible causation opinion is required to rely on epidemiologic studies. To the contrary, California case law explicitly holds that a medical causation opinion in a toxic injury action is admissible in the absence of supporting epidemiologic studies.

The leading case on this point is Roberti v. Andy’s Termite & Pest Control, Inc. (2003) 113 Cal.App.4th 893. In Roberti, a pesticide manufacturer filed an in limine motion to preclude the plaintiff’s medical experts from opining that plaintiff’s autism, which is a neurologic disorder, was caused by exposure to the pesticide Dursban. In support of his theory that Dursban caused his autism, plaintiff presented expert testimony

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4 Co-panel participant, James Scadden, has provided in his materials a copy of a bibliography prepared by plaintiffs’ expert in the hydroquinone-leukemia case presently being litigated by this author’s firm, which includes citations to the body of published scientific literature generally referenced above.
of several toxicologists and medical doctors in which each stated the opinion, to a reasonable degree of medical probability, that plaintiff’s autism was caused by his exposure to defendant’s Dursban. The experts did not base these opinions on a single epidemiologic study. Instead, these opinions were based entirely on published peer-reviewed animal studies finding that Dursban causes neurologic disorders.

Defendant contended that the expert opinions offered by plaintiff asserted only the possibility of causation because they were unsupported by peer-reviewed epidemiologic studies. Defendant later filed an amended motion in limine, contending that plaintiff’s expert testimony was based on novel methodologies of scientific proof unsupported by peer-reviewed epidemiologic studies (i.e., did not meet the “general consensus” admissibility test set forth in People v. Kelly).

Thereafter the trial court granted defendant’s motion, ruling that "[t]he plaintiff's experts' analysis and causation opinions are not derived from any accepted scientific methodology, are not scientifically valid, and do not possess the evidentiary reliability required by Kelly. . . ." The court further noted that "[t]he consensus in the medical community is that there is no known cause of autism" and further that "[t]here is no consensus among the scientific community that pesticides cause autism." Plaintiff appealed, contending that Kelly’s general acceptance test did not apply to expert medical opinion testimony, and the trial court had no authority to exclude the testimony of Plaintiffs’ medical experts based upon a threshold test of admissibility akin to the Daubert standard used by federal courts.

The Court of Appeal first agreed that Kelly’s general acceptance test did not apply to the expert medical causation opinions of plaintiff’s experts:

Plaintiff's experts based their opinion testimony upon research papers and studies (primarily those conducted on animals) in peer-reviewed journals regarding Dursban and its effects, and to some extent upon physical examination of plaintiff using techniques that are generally accepted in the relevant medical community. They did not rely upon any new scientific technique, device, or procedure that has not gained general acceptance in
the relevant scientific or medical community. Rather, it was the theory of causation, that Dursban caused plaintiff’s autism, that has not gained general acceptance in the relevant medical community. The Kelly test is not applicable even though the proffered evidence presents a new theory of medical causation.

Defendant also argued that the trial court was correct in excluding the testimony of plaintiff’s experts based on lack of an adequate foundation. In making this argument, defendant contended that California trial courts could and should apply the foundational analysis employed in the federal courts to all expert testimony. The Roberti court noted that the federal rule established in Daubert v. Merrell Dow Pharmaceuticals, Inc. (1993) 509 U.S. 579 subjects all expert scientific and technical opinion testimony to a threshold reliability test under Rule 702 of the Federal Rules of Evidence, which superseded the Frye test in federal courts as of 1993. However, the Court of Appeal held that Daubert does not alter California law with regard to admissibility of expert medical opinion testimony and that California trial courts may not “conduct preliminary fact-finding, to make a preliminary assessment of whether the reasoning or methodology underlying the testimony properly can be applied to the facts in issue.” Roberti, at 893.

Defendant next argued that the testimony of the plaintiff’s medical experts could be excluded under Evidence Code § 803, which provides that a trial court "may, and upon objection shall, 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." The Court of Appeal rejected defendant’s misapplication of Evidence Code Sections 801 and 803, which defendant was wrongly equating with a federal court’s authority to determine the admissibility of expert opinions:

Defendant argues that, even if we conclude the Kelly test is not applicable to the expert medical testimony here, the trial court was still correct in excluding it based on its purported lack of an adequate foundation. Defendant contends that we should apply the foundational analysis employed in the federal courts to all expert testimony, an analysis the California Supreme Court has explicitly rejected.
The federal rule established in Daubert v. Merrell Dow Pharmaceuticals, Inc., (Daubert) subjects all expert scientific and technical opinion testimony to a threshold reliability test (under rule 702 of the Federal Rules of Evidence, which superseded the Frye test in federal courts as of 1993). Daubert, however, does not alter California law with regard to admissibility of expert medical opinion testimony.

Under Daubert and the Federal Rules of Evidence it interprets, a district court must first determine whether the reasoning or methodology underlying the testimony is scientifically valid; unlike the Kelly test, however, general acceptance in the scientific community of the underlying methodology is not necessarily required. In addition, the district court must also conduct preliminary fact-finding, to make a preliminary assessment of whether the reasoning or methodology underlying the testimony properly can be applied to the facts in issue. (National Bank of Commerce v. Dow Chemical Co., supra, 965 F.Supp. at pp. 1495-1496, citing Daubert, supra, at pp. 592-595, 113 S.Ct. 2786.)

The district court in National Bank of Commerce applied that test and ruled that plaintiff failed to establish that her exposure to Dursban caused birth defects, where the expert testimony was based on what the court deemed to be studies involving inappropriate protocol and methodology and inadequate exposure and dosage levels, and on animal studies whose applicability to humans was speculative.

"In California evidence is relevant only if it has 'any tendency in reason to prove or disprove any disputed fact' (Evid.Code, § 210). And an expert's testimony must be based on matter 'that is of a type that reasonably may be relied upon by an expert' (id., § 801, subd. (b)). (See People v. Leahy, supra, 8 Cal.4th at pp. 597-598 [34 Cal.Rptr.2d 663, 882 P.2d 321] ....)"

What defendant would have us do, under the guise of determining whether the challenged testimony was supported by the proper foundation, is conduct a Daubert-style analysis, precisely as the court did in National Bank of Commerce. Use of the Daubert threshold reliability test is not, however, in keeping with the law in California.

Defendant's objections are actually to the conclusions plaintiff's experts reached based on the studies available, not with the methodology used in the studies, upon which the experts relied in reaching their conclusions. Defendant's argument in this regard, and the trial court's ruling, instead pertains to the weight of the underlying bases for the expert opinion, not its admissibility.

Roberti, 113 Cal.App.4th at 904-906.
The Roberti court thereafter held that a trial court’s admissibility determination is limited to the general determination under Evidence Code Sections 801 and 803 whether the expert’s opinions are based on matter of the type on which an expert may reasonably rely in rendering such opinions (i.e., whether the studies on which the expert has relied, including animal studies, generally concern the toxins and the diseases at issue in the litigation). The court therefore found that animal studies concerning Dursban and brain and central nervous system disorders were generally of the type on which an expert rendering medical causation opinions concerning Dursban and autism (a type of neurologic disorder) may reasonable rely, because the studies generally concerned the same toxins and same type of injuries at issue in the case. The court thereafter found that the medical causation opinions of plaintiff were admissible, even in the complete absence of supporting epidemiologic studies.

Thus, under California law, even if a trial court were to incorrectly rule that epidemiologic studies concerning benzene exposed workers and leukemia cannot be relied on by a plaintiff’s expert in a hydroquinone-leukemia case, the remaining types of published animal studies, human in vitro studies, and mechanistic studies previously discussed herein provide an adequate foundation to support the admissible opinion that hydroquinone causes leukemia in humans, and caused the particular plaintiff’s leukemia.
IV. Rutherford’s “Substantial Factor Standard” is the Causation Standard in Hydroquinone-Leukemia Cases in California

In 1997, the California Supreme Court issued a landmark toxic tort decision in Rutherford v. Owens-Illinois, Inc. (1997) 16 Cal.4th 953, in which the Supreme Court adopted the extremely liberal “substantial factor standard” for plaintiffs suffering from toxic injuries to prove medical causation in toxic injury actions. Defendants in toxic injury actions regularly argue to trial courts that Rutherford’s substantial factor standard is limited to asbestos lung cancer cases. Defendants are quite wrong.

To fully understand the liberal substantial factor standard created in Rutherford, and why this standard applies to all toxic injury actions it is first necessary to understand the causation issues before the Supreme Court. Rutherford was a lung cancer case brought by a life-long smoker who claimed that his cancer was caused by his exposure to asbestos from the numerous asbestos-containing insulation products of approximately twenty defendants. Prior to trial, Rutherford settled with all of the defendants, except Owens-Illinois. At trial, Rutherford presented evidence of his cumulative exposure to asbestos, including asbestos from Owens-Illinois’ Kaylo product. Owens-Illinois also presented evidence that Mr. Rutherford was exposed to very little asbestos from Owen’s asbestos products, as well as medical evidence that Mr. Rutherford’s 30-year history of cigarette smoking was a substantial factor in causing his lung cancer. Rutherford, 16 Cal.4th at p. 957-962.

In order to lessen Rutherford’s burden of establishing that his exposure to asbestos fibers from Owens-Illinois’ Kaylo product, as opposed to asbestos fibers from settling defendants’ asbestos products, was a substantial factor in causing his lung cancer, the trial court applied a local “burden-shifting instruction.” The jury was instructed that “if the plaintiff has proved that a particular asbestos supplier's product was ‘defective,’ that the plaintiff’s injuries or death were legally caused by asbestos exposure generally, and that
he was exposed to asbestos fibers from the defendant's product, the burden then shifts to the defendant to prove, if it can, that its product was not a legal cause of the plaintiff's injuries or death.” Rutherford, 16 Cal.4th at pp. 960-961. The jury thereafter found for Rutherford, apportioning 1.2% of fault to Owens-Illinois, 2.5% to Rutherford, and 96.3% to asbestos companies with whom Rutherford had settled before trial. Owens-Illinois appealed arguing that the trial court’s “burden-shifting instruction” improperly shifted the burden of proving medical causation, and also plaintiff had failed to prove that he was exposed to Owens’ asbestos products and had failed to prove that any of Owens’ asbestos had caused Rutherford’s lung cancer.

The issues before the Supreme Court were therefore whether the trial court’s “burden-shifting instruction” was proper under California law, and whether Rutherford had introduced sufficient evidence to support the jury’s determination that Owens’ asbestos products contributed to Rutherford’s lung cancer. In determining these issues, the Supreme Court first held that the “burden-shifting instruction” impermissibly shifted the burden of proving medical causation to Owen-Illinois. Rutherford, 16 Cal.4th at pp. 976-983. However, the Court also stated that the trial court’s efforts were quite understandable in attempting to lessen the evidentiary burden of a plaintiff suffering from lung cancer as a result of his exposure to asbestos from the asbestos-containing products of multiple defendants, and where the plaintiff must prove that asbestos fibers from a particular defendant’s products were a substantial factor in causing his lung cancer. For this reason, the second half of the Rutherford decision concerned the creation of the Court’s new “substantial factor standard,” which was designed to lessen a plaintiff’s burden of proving medical causation in such toxic injury actions without impermissibly shifting the burden of proof to defendants. Rutherford, 16 Cal.4th at pp. 979-983.

In adopting its substantial factor standard, the Supreme Court held that to prevail in a toxic injury action involving multiple defendants and numerous toxic products, a
plaintiff need not prove that exposure to a particular defendant’s product actually caused his cancer, but need only show that the exposure to a particular defendant’s product was a substantial factor in increasing his risk of developing cancer. Id., 16 Cal.4th at 982. The Court stated that a plaintiff “cannot be expected to prove the scientifically unknown details of carcinogenesis, or trace the unknowable path of a given asbestos fiber”. Id. at 976. Instead, “we can bridge the gap in the humanly knowable” by requiring a plaintiff to show that his exposure to a defendant’s product was a substantial factor in contributing to the his aggregate exposure, and thus a substantial factor in increasing his risk of developing cancer. Id. A plaintiff need not demonstrate that exposure to the “fibers of the defendant’s particular product were the ones, or among the ones, that actually produced the malignant growth.” Id. at 977. The Court also explained that “the substantial factor standard is a relatively broad one, requiring only that the contribution of the individual cause be more than negligible or theoretical.” Id., 16 Cal.4th at 978. Thus, to establish the element of medical causation as to any particular defendant, the Rutherford Court held that a plaintiff need only submit evidence that his total exposure to asbestos from all sources substantially increased his risk of developing cancer, and that the contribution of asbestos fibers from a particular defendant’s asbestos-containing products was more than a “merely theoretical or infinitesimal” contribution to his total exposure to asbestos.

The Supreme Court in Rutherford therefore adopted a substantial factor standard which consists of two distinct prongs, both of which must be proven by expert opinion rendered to a reasonable degree of scientific/medical probability: (1) The plaintiff’s cumulative or total exposure to asbestos from all sources, including all of defendants’ products, substantially increased his risk of developing his asbestos-induced cancer; and (2) The contribution of asbestos from a particular defendant’s products was more than a “merely theoretical or infinitesimal” contribution to the plaintiff’s cumulative/total exposure to asbestos. When both prongs are established through expert opinion rendered
to a reasonable degree of scientific/medical probability, a particular defendant is thereby deemed a “substantial factor” in causing the plaintiff’s lung cancer, because asbestos fibers from this defendant’s products were more than a “merely theoretical or infinitesimal” contribution to the plaintiff’s cumulative/total exposure to the asbestos which had substantially “increased the plaintiff’s risk” of developing lung cancer.

However, in adopting its liberal substantial factor standard in Rutherford, the Supreme Court did not state whether the standard would apply in toxic injury actions other than asbestos lung cancer cases. Subsequently, in Bockrath v. Aldrich Chemical Company (1999) 21 Cal.4th 71, 86, a multi-defendant toxic injury action involving a plaintiff suffering from benzene-induced multiple myeloma (not asbestos-induced cancer), the Supreme Court clarified that Rutherford’s liberal substantial factor standard applies in all toxic injury actions involving a single disease caused by exposure to the numerous toxic products of multiple defendants. The Court noted that “[i]n Rutherford . . . we addressed the question of proof of causation in ‘the context of products liability actions.’” Bockrath at 79 (citing Rutherford at 968). The Court then stated that the purpose of the Court’s decision in Bockrath was to set forth how a toxic injury action must be plead to satisfy the elements of proof required under Rutherford’s “substantial factor standard.” The Court held that Rutherford’s “substantial factor standard” applied to plaintiff’s toxic injury action because Mr. Bockrath’s case, as with the asbestos-induced cancer in Rutherford, involved “complicated and possibly esoteric medical causation issues,” i.e., the determination of medical causation when a single disease (e.g., multiple myeloma) is alleged to have been by caused by a plaintiff’s cumulative exposure to toxins (e.g., benzene) from the numerous chemical products (e.g., benzene-containing solvents) of numerous defendants (e.g., the 55 manufacturers of said solvents). Id.

According to the Supreme Court in Bockrath, Rutherford’s substantial factor standard therefore applies in any toxic injury action in which a plaintiff alleges that his
cumulative exposure to toxins from the numerous products of multiple defendants caused him to develop a disease, such as asbestos-induced lung cancer, benzene-induced multiple myeloma, or hydroquinone-induced leukemia. As adopted in Rutherford and extended in Bockrath, this substantial factor standard therefore consists of two distinct prongs, both of which must be proven by expert opinion rendered to a reasonable degree of scientific/medical probability: (1) The plaintiff’s cumulative/total exposure to the toxin(s) at issue (e.g., hydroquinone) from all sources substantially increased his risk of developing his disease (e.g., leukemia); and (2) The contribution of the particular toxin(s) at issue (e.g., hydroquinone) from a particular defendant’s product (e.g., x-ray developer) was more than a “merely theoretical or infinitesimal” contribution to the plaintiff’s cumulative/total exposure to the toxin(s) (e.g. hydroquinone). When both prongs are established through expert opinion rendered to a reasonable degree of scientific/medical probability, a particular defendant is thereby deemed a “substantial factor” in causing the plaintiff’s disease.

Defendants in toxic injury cases nevertheless regularly argue to trial courts that Rutherford’s substantial factor only applies in asbestos lung cancer cases, because the standard was only adopted by the Supreme Court in Rutherford due to the fact that there was no scientific dispute as to the ability of asbestos to cause lung cancer, and Owens-Illinois had conceded that Mr. Rutherford’s lung cancer was only caused by his exposure to asbestos. This argument flies in the face of logic and the facts of Rutherford because the Supreme Court would not have been required to create the first prong of its substantial factor standard (i.e., plaintiff’s cumulative exposure to asbestos “increased his risk” of developing lung cancer), if asbestos was the only known cause of the type of lung cancer from which Mr. Rutherford suffered. In such a case, Mr. Rutherford could have certainly provided medical evidence that his lung cancer was actually caused by his cumulative exposure to asbestos, because only asbestos causes such lung cancer.
“Increasing the risk” is therefore required as a medical causation standard when a disease can be caused by more than one toxic factor which has or may have been encountered by a particular plaintiff, each of which is capable of causing the disease.

Indeed, the Supreme Court in Rutherford specifically noted that Owens-Illinois had also introduced evidence at trial that Mr. Rutherford’s cigarette smoking was a substantial factor in causing his lung cancer. Moreover, the Court noted that the jury was instructed to consider Mr. Rutherford’s smoking for purposes of comparative fault, and the jury thereafter found Mr. Rutherford partially at fault for his lung cancer:

Evidence was also presented that Rutherford had smoked approximately a pack of cigarettes a day over a period of 30 or more years until he quit smoking in 1977. As will be explained, this evidence took on heightened relevance at the second "liability" phase of trial.

[Rutherford, 16 Cal.4th at p. 960]

...Owens-Illinois was also permitted to present evidence that smoking was a "negligent" contributing factor to each plaintiff's condition. Undisputed evidence indicated that smoking sharply increases the risk of lung disease, including lung cancer, and works "synergistically" with asbestos exposure to enhance the severity of resulting damage to the lungs. The trial court's instructions made clear that each plaintiff's entire recovery must be reduced to the extent of his own comparative "negligence" contributing to his condition, because each had continued to smoke tobacco long after he had notice that smoking was hazardous to health, and that the long-term consumption of tobacco products could be a contributing cause of lung disease.

[Rutherford, 16 Cal.4th at p. 962]

...The liability phase jury was instructed to assign percentages of fault for each injury, adding up to a total of 100 percent, among (1) the plaintiff himself (here, plaintiffs' decedent); (2) Owens-Illinois; (3) other manufacturers of asbestos to which the plaintiff or decedent was exposed; and (4) each employer that contributed to the exposure. In Rutherford's case, the jury apportioned fault as follows: 1.2 percent to Owens-Illinois, 2.5 percent to Rutherford himself, and 96.3 percent to the remaining entities to which the jury was allowed to assign fault.

Rutherford, 16 Cal.4th at p. 962.
The Court also stated that cigarette smoking is also a substantial factor in increasing the risk of developing lung cancer, even among asbestos exposed workers, and such indicates that both smoking and asbestos can constitute substantial factors in increasing a particular plaintiff’s risk of developing lung cancer:

[A]t a level of abstraction somewhere between the historical question of exposure and the unknown biology of carcinogenesis, the question arises whether the risk of cancer created by a plaintiff's exposure to a particular asbestos-containing product was significant enough to be considered a legal cause of the disease. Taking into account the length, frequency, proximity and intensity of exposure, the peculiar properties of the individual product, any other potential causes to which the disease could be attributed (e.g., other asbestos products, cigarette smoking), and perhaps other factors affecting the assessment of comparative risk, should inhalation of fibers from the particular product be deemed a "substantial factor" in causing the cancer?

...  

Instead, we can bridge this gap in the humanly knowable by holding that plaintiffs may prove causation in asbestos-related cancer cases by demonstrating that the plaintiff's exposure to defendant's asbestos-containing product in reasonable medical probability a substantial factor in contributing to the aggregate dose of asbestos the plaintiff or decedent inhaled or ingested, and hence to the risk of developing asbestos-related cancer . . .

Rutherford, 16 Cal.4th at pp. 975-977; See also Cottle v. Superior Court (1992) 3 Cal.App.4th 1367, fn 5. (“Cigarette smoking presents a striking exception to the general rule that the excess risk created by an activity does not exceed the background risk. For example, the incidence of lung cancer among asbestos-exposed workers who smoke is 10 times greater than that among similarly exposed workers who do not smoke.”) (citing Selikoff & Hammond, Asbestos and Smoking, 242 J.A.M.A. 458, 458 (1979) (editorial).

Thus, the medical fact that both cigarette smoking and asbestos, among other factors, can cause lung cancer is why the Supreme Court found it necessary to lessen a plaintiff’s burden of establishing medical causation by only requiring a plaintiff to prove that his cumulative exposure to asbestos substantially increased his risk of developing lung cancer (Prong Number 1 of the “substantial factor standard”). Because cigarette
smoking and asbestos can both cause lung cancer, the Court concluded that it would be practically impossible for a plaintiff to present medical evidence that asbestos, as opposed to cigarette smoking, actually caused the formation of the cancerous cells in his lungs which resulted in lung cancer. At most, a plaintiff can present evidence that both asbestos and smoking substantially increased his risk of developing lung cancer. Afterward, a jury is thereby permitted to conclude, as apparently the jury did in Rutherford by apportioning comparative fault to Mr. Rutherford, that both cigarette smoking and asbestos increased the plaintiff’s risk of developing his lung cancer.

Similarly in non-asbestos cases, such as the multiple myeloma case before the Supreme Court in Bockrath, epidemiologic studies indicate that there exists a significant increased risk of developing multiple myeloma as a result of exposures to benzene, asbestos, pesticides, paints, solvents, engine and diesel exhausts, formaldehyde, vinyl chloride, styrene, ionizing radiation, and other toxins. See e.g., Herrington, et. al., “Multiple Myeloma,” published in Cancer Epidemiology and Prevention (2nd Ed.) 1996, Schottenfeld & Fraumeni (Editors), Oxford University Press, Chapter 43, pages 956-963. Because a variety of toxins can cause multiple myeloma, including benzene, the Bockrath Court correctly concluded that it would be practically medically impossible for a plaintiff, such as Mr. Bockrath, to present medical evidence that benzene, as opposed to other toxins known to cause multiple myeloma to which the plaintiff was also exposed, actually caused the formation of the cancerous cells resulting in his multiple myeloma. At most, such a plaintiff can present evidence that his cumulative exposure to benzene substantially increased his risk of developing multiple myeloma. For this reason, Rutherford’s substantial factor standard was extended by the the Supreme Court in Bockrath to all toxic injury actions, including benzene and hydroquinone actions.

Indeed, in a remarkably forward-looking and eloquent dissent in Cottle v. Superior Court (1992) 3 Cal.App.4th 1367, Justice Johnson anticipated and urged the Supreme
Court’s decisions in Rutherford and Bockrath to adopt “increasing the risk” as the causation standard in all toxic injury actions:

I differ with my colleagues about the proper standard of causation in a toxic tort case. It is true the expert witness declarations the plaintiffs proffered in this proceeding could have been more precise about degrees of probability and the like. Nonetheless, these experts addressed the proper test for causation in a toxics case and were sufficient to survive any legitimate summary judgment motion.

Several of plaintiffs' expert witnesses-toxicologists, neurologists, and the like-furnished declarations offering their opinions to a reasonable medical certainty that: (1) Individual plaintiffs were and are suffering significant diseases and conditions; (2) The chemical soup in this former dump contains a number of individual chemicals and other toxics in substantial amounts; (3) These toxics have been scientifically found to increase the risk those exposed will suffer the same types of diseases and conditions plaintiffs in fact were and are experiencing. That is, persons exposed to these toxics will suffer increased incidences of these diseases and conditions compared to those who are not so exposed. (Significantly, the experts also compared plaintiffs with the general population and found a consistent pattern of damage to the immune system. This renders plaintiffs less resistant to a wide range of serious and not-so-serious diseases now and into the future.)

. . . . Instead what the trial court sought was an impossibility in this as in virtually all toxic tort cases-evidence a given toxic or combination of toxics was the cause in fact of a given disease or other condition in a specific individual. This is not a reasonable causation standard to apply in a toxics tort case such as the one before the court.

. . . [S]cores or hundreds of different environmental factors can cause or contribute to most forms of cancer and other injuries and diseases typically confronted in toxic tort cases. Consequently, when a plaintiff is exposed to a toxic and subsequently suffers some disease or injury no expert honestly can testify the toxic caused that particular individual to experience that particular disease or injury.

What science typically can tell us, on the other hand, is that people exposed to a certain toxic have a heightened risk of developing a certain disease or injury. To put it another way, a higher percentage of them will experience that disease or injury than those who are not exposed to the particular toxic. But even when the plaintiff proves he has both been exposed to the toxic and later developed the disease or other injury, no expert can responsibly testify to a medical certainty the toxic is the cause in fact of the disease or injury. Only in the rare situation where exposure to a particular toxic raises the risk of the disease by over 50 percent can an expert even testify it is more probable than not the toxic is the cause in fact of the disease or injury.
Toxics thus pose a new problem for the law of torts. The old rules of causation simply don't work—because toxics are not automobiles or the other instruments of sudden destruction so familiar to the law. Toxics operate at a microscopic, often submicroscopic, level. They also typically do their damage over the course of months or years. Consequently, there are no witnesses to the "events" linking the toxic to its victim—no one to say "I saw this toxic invade this cell and chemically alter its composition so that a dozen cell generations later it mutated into a cancer that then grew larger and larger until it now threatens the plaintiff's life." Unless the toxic is one of those rare agents, like asbestos, which conveniently causes its own unique disease, there is no way for anyone to testify to the causal path that actually linked the toxic with its injurious effect in the particular case.

As a result, what the trial court sought from plaintiffs' experts in this case—although the traditional causation standard for negligence cases—was not feasible or appropriate in a toxic tort case. The remaining question is whether the law is to deny recovery in the vast majority of toxic tort cases because it is not possible to satisfy a causation test developed in the context of and for the purpose of deciding an entirely different class of cases.


Defendants also regularly argue in toxic injury actions that the limited application of Rutherford's substantial factor standard to asbestos lung cancer cases is established CACI No. 435. CACI No. 435 is labeled "Causation for Asbestos-Related Cancer Claims," and states in its directions for use that an instruction based on Rutherford's substantial factor standard must be given in asbestos-related cancer cases. However, nowhere is there a statement in the directions that Rutherford's substantial factor can only be used in asbestos lung cancer cases. Moreover, merely because the California Judicial Council, which is not a law-making body, has failed to create a recommended jury instruction for all toxic injury actions based on Rutherford's substantial factor standard does not mean that the standard does not apply in such cases. Such failure by California Judicial Council merely means that the Council has yet to fully and properly consider Bockrath's extension of Rutherford's substantial factor standard to all toxic injury actions, including hydroquinone-leukemia cases.

Defendants in toxic injury actions will also often attempt to introduce expert evidence which contravenes Rutherford's substantial factor standard in cases in which the
trial court has determined the standard applies. Specifically, a defendant will individually designate an expert who has quantified/estimated the cumulative amount of the toxin at issue (e.g., hydroquinone) to which the injured plaintiff was exposed only from this particular defendant’s products. This expert will then offer an opinion that plaintiff’s cumulative exposure to this toxin from this particular defendant’s products was below or equal to the ambient level of this toxin to which an average person is exposed in the environment during his lifetime. The expert will then offer the opinion that plaintiff’s exposure to this “infinitesimal” amount of this toxin from this particular defendant’s products was, itself, insufficient to have substantially increased plaintiff’s risk of developing the disease from which he suffers, and therefore such exposure was not a substantial factor in causing the disease.5

The Court of Appeal in Jones v. John Crane, Inc. (2005) 132 Cal.App.4th 990 clearly explained that these types of opinions cannot be used to defeat medical causation in a toxic injury action. This is so because Rutherford’s substantial factor standard only requires an injured plaintiff to prove that his exposure to a toxin from a particular defendant’s products was more than a merely theoretical or infinitesimal contribution to the total exposure to this toxin which substantially increased his risk of developing his disease. A plaintiff is not required and cannot be required to prove that his exposure to this toxin from this particular defendant’s product was, itself, sufficient to have substantially increased his risk of this disease. The Court of Appeal therefore concluded that even if a plaintiff’s exposure to the toxin at issue from a particular defendant’s

5 For example, the expert will opine that the plaintiff’s increased risk of developing leukemia as a result of his cumulative exposure to this toxin from a particular defendant’s products was only 0.00000001% which is itself an infinitesimal increased risk of developing leukemia.
products was below ambient lifetime exposure levels, the plaintiff’s exposure to this particular defendant’s products can still be determined by the jury to be a substantial factor in causing his disease, so long as the jury concludes that this amount of exposure was more than a merely theoretical or infinitesimal contribution to the plaintiff’s total exposure to this toxin. See Jones v. John Crane, Inc. (2005) 132 Cal.App.4th 990, 1000:

Defendant argues that the fibers released from its products were no greater than the ambient level of asbestos in the atmosphere. Its industrial hygienist testified that the general range of fibers released from installation and removal of Crane's products is 0.01 to 0.1 fibers per cubic centimeter of air, and its experts calculated Jones's total asbestos exposure from Crane's products at 10 fiber hours, or .005 fiber years. Defendant's experts considered this amount trivial when compared to the more than 200 fiber years of asbestos exposure Jones suffered during the 7 to 12 years he worked with thermal insulation and his estimated 2.8 fiber years of asbestos exposure from non-occupational ambient asbestos over the course of his lifetime.

Plaintiffs . . . challenged defendant's assertion that Jones's total exposure to asbestos from its products was significantly less than a lifetime of exposure to ambient asbestos. Based on defendant's calculations, the fibers released from defendant's valve packing, 0.01 to 0.1 fibers per cubic centimeter, are tenfold the level of asbestos found in ambient air, i.e., from .001 to .01 fibers per cubic centimeter. Defendant attempts to minimize this disparity by comparing a potential lifetime of ambient exposure to Jones's aggregate occupational exposure to asbestos from its products. But a level of exposure that is the equivalent of that to which one might be exposed in the ambient air over a lifetime is not necessarily insignificant.

Rutherford does not require that each exposure be sufficient to independently cause lung cancer. To the contrary, the exposure need only be “a substantial factor in contributing to the aggregate dose of asbestos the plaintiff ... inhaled.” The mere fact that comparable levels could be found in ambient air does not render the exposure “negligible or theoretical.” As Dr. Hammar recognized, if a person were exposed to six different products, each with a release level similar to the asbestos levels recorded in ambient air, the combined concentration in the total dose would contribute substantially to the increased risk of cancer. We heed the admonition in Rutherford to be wary of the misapplication of the substantial factor test.
See also Rutherford v. Owens-Illinois (1997) 16 Cal.4th 953, 979-980 (“[W]e can bridge this gap in the humanly knowable by holding that plaintiffs may prove causation . . . by demonstrating that the plaintiff’s exposure to defendant’s . . . product was a substantial factor in contributing to the aggregate dose . . . the plaintiff inhaled or ingested, and hence the risk of developing asbestos related cancer, without the need to demonstrate that fibers from a defendant’s particular products were the ones, or among the ones, that actually produced the malignant growth... Without such guidance, a juror might well conclude that the plaintiff needed to prove that fibers from the defendant's product were a substantial factor actually contributing to the development of the plaintiff's or decedent's cancer.”)

A defendant therefore cannot defend a hydroquinone case by having its expert render the opinion that plaintiff’s exposure to hydroquinone from this defendant’s products was, itself, insufficient to have substantially increased plaintiff’s risk of developing the leukemia from which he suffers. Indeed, such an opinion would be properly excluded from a hydroquinone-leukemia case in California on the basis that the opinion violates the applicable causation standard set forth in Rutherford, and would therefore only confuse the jury and unduly prejudice plaintiff. Instead, a defense expert is required to render the opinion in a hydroquinone-leukemia case, and to justify this opinion before the jury, that the plaintiff’s cumulative exposure to less than ambient levels of hydroquinone from a particular’s defendant’s products was not a substantial factor in causing plaintiff’s leukemia, because this amount of hydroquinone was merely a theoretical or infinitesimal contribution to plaintiff’s total exposure to hydroquinone from all sources which had substantially increased his risk of developing leukemia.

Another import principle to take from the Jones decision is this: Once a plaintiff’s expert has rendered the admissible opinion that a plaintiff was exposed to an actual and measurable amount of hydroquinone from a particular defendant’s products (i.e, it was
more than theoretical), no matter how small this exposure may be, and has also rendered
the admissible opinion that this exposure was a substantial factor in causing the
plaintiff’s disease, it will always be a factual issue for the jury whether this exposure was
indeed a substantial factor (i.e., whether this exposure was less than or more than a merely
theoretical or infinitesimal contribution to the plaintiff’s total exposure to hydroquinone).
In the face of such opinions, it will always be improper for a trial court to conclude, as a
matter of law, that the actual and measurable amount of hydroquinone to which the
plaintiff was exposed from a particular defendant’s products was merely a theoretical or
infinitesimal contribution to the plaintiff’s total exposure to hydroquinone, and therefore
this defendant was not a substantial factor in causing the plaintiff’s leukemia. This is so
because only a jury can factually determine the limits of a theoretical or infinitesimal
contribution of hydroquinone.

V. California Law Does Not Require a Plaintiff to Quantify His Exposure to
Hydroquinone to Establish Causation

Defendants in toxic injury cases regularly argue to trial courts that an injured
plaintiff is required to quantify the dose, level, duration, or frequency of his exposure to
the toxin at issue in order to prove that his exposure to this toxin was a substantial factor
in causing his disease. However, there are no published California decisions in which a
court has held that a plaintiff is required to provide expert testimony or other evidence
quantifying the duration, frequency, dose, or level of his exposure to a particular toxin,
either in the aggregate or from a particular defendant’s chemical product, in order to
prove causation in a toxic injury action. To the contrary, California courts addressing the
issue have held that a plaintiff need only introduce direct and/or circumstantial evidence
which provides a reasonable inference that a plaintiff was substantially exposed to the
toxin at issue, and expert medical opinion that this exposure was a substantial factor in
causing the plaintiff’s disease at issue.
In Lineweaver v. Plant Insulation Co. (1995) 31 Cal.App.4th 1409, 1420, a plaintiff suffering from asbestosis introduced circumstantial evidence of exposure, including the prevalence of defendant’s asbestos-containing insulation product at the worksite and plaintiff’s testimony concerning plaintiff’s proximity to dusts when similar asbestos insulation products were removed. Plaintiff also introduced the testimony of an exposure expert, who based on this circumstantial evidence, opined that plaintiff was substantially exposed to asbestos fibers from defendant’s asbestos product. Plaintiff also introduced testimony from a medical causation expert who had estimated the dose of plaintiff’s exposure in “asbestos fiber years.” Based on all of the foregoing evidence, this medical causation expert also opined that plaintiff’s exposure to defendant’s asbestos product was a substantial factor in causing his asbestosis. The jury thereafter found that plaintiff’s exposure to defendant’s asbestos insulation was a substantial factor in causing his asbestosis and awarded a verdict in plaintiff’s favor. Defendant appealed, arguing that plaintiff’s exposure and medical causation evidence was insufficient to support the medical causation opinions of plaintiff’s experts, particularly the “asbestos fiber years” dose estimate of plaintiff’s causation expert, which defendant contended was speculative and lacking in foundation.

The court thereafter held that plaintiff’s circumstantial exposure evidence was alone sufficient to support the jury’s verdict because a plaintiff, in order to establish exposure to a defendant’s product in a toxic injury action, need only introduce direct and/or circumstantial evidence, such as the prevalence of the product at the work site and the plaintiff’s proximity to noticeable dusts from such products, which provides a reasonable inference that a plaintiff was exposed to a particular defendant’s product:

While there was no direct evidence that Lineaweaver was exposed to Plant-supplied Pabco, the circumstantial evidence was sufficient to support a reasonable inference of exposure. Unlike Dumin v. Owens-Corning Fiberglas Corporation, supra, 28 Cal.App.4th 650, in which we found insufficient evidence of exposure to a particular asbestos product, plaintiff has established that defendant's product was definitely at his work site and
that it was sufficiently prevalent to warrant an inference that plaintiff was exposed to it . . . . (Id. at 420)

Concerning the issue of medical causation, the defendant in Lineweaver thereafter argued that the medical causation opinions of plaintiff’s expert should have been excluded, because the expert’s opinion that plaintiff’s exposure to defendant’s asbestos products was a substantial factor in causing his illness was based on the expert’s speculative “asbestos fiber years” dose estimate of plaintiff’s exposure. The Lineweaver court held that even if the dose estimate of plaintiff’s causation expert was completely disregarded, there was sufficient evidence, in the form of plaintiff’s testimony regarding his exposures to dusts and circumstantial evidence concerning the prevalence of defendant’s products at the worksite, for plaintiff’s medical causation expert to render the admissible opinion that plaintiff’s exposure to defendant’s asbestos products was a substantial factor in causing his asbestosis:

As for biological causation, a physician expert in occupational medicine concluded that Lineweaver's exposure to Pabco products was "a very substantial factor" in causing Lineweaver's asbestosis. The physician, Dr. Richard Cohen, even opined that it is more likely than not that Lineweaver would have developed asbestos-related disease from the exposure to Pabco products alone. [Defendant] disputes the validity of these opinions as based on unsupported quantification in "fiber-years" of Lineweaver's exposure to Pabco. But the opinions of plaintiffs' experts and an inference of Pabco exposure as a substantial factor in contributing to Lineweaver's asbestosis may be drawn from evidence independent of Dr. Cohen's quantification methodology.

As discussed above, Lineweaver presented evidence of exposure to Plant-supplied Pabco on a regular basis over more than 30 years of working with and near asbestos insulation products. Lineweaver was exposed to pipe covering and block insulation which is friable and "very powdery," and created visible dust reminiscent of a "snow storm." While there are other possible sources of Lineweaver's asbestosis given his exposure to many different asbestos products, it is significant that Pabco products were prominent and prevalent at his work site. Viewing this evidence in Lineweaver's favor, it was sufficient to support a jury's inference that exposure to Pabco products was a substantial factor in causing Lineweaver's asbestosis. (Id. at 1420-1421)

Similarly, in Sparks v. Owens-Illinois (1995) 32 Cal.App.4th 461, a plaintiff suffering from mesothelioma caused by his decades-long exposure to asbestos dusts from
asbestos insulation products brought a products liability action against the numerous manufacturers of these products. The jury awarded a verdict in plaintiff’s favor, and in so doing found Owens-Illinois 100% at fault for causing plaintiff’s illness, even though plaintiff was exposed to asbestos dusts from the asbestos products of numerous settling defendants. Owens-Illinois appealed the verdict, and argued, in part, that plaintiff’s exposure and medical causation evidence failed to establish that Owens-Illinois’ “Kaylo” product was the cause of plaintiff’s mesothelioma. Plaintiff’s exposure and medical causation evidence included the opinions of several experts, none of whom calculated plaintiff’s asbestos dose. Rather, these experts opined, based on plaintiff’s testimony, that plaintiff’s exposure to asbestos dusts from Kaylo was “intense,” “substantial,” and sufficient to cause Plaintiff’s illness.

The Court of Appeal held that the opinions of plaintiff’s experts were sufficient to support the jury’s finding that plaintiff’s exposure to Kaylo was the cause of plaintiff’s mesothelioma:

The testimony of plaintiffs' medical experts was clearly sufficient to support a jury finding that Owens-Illinois's product, Kaylo, was more likely than not the source of asbestos fibers that caused Mr. Sparks's mesothelioma. Each of the experts testified that Sparks's exposure to asbestos-containing products during his time aboard the Bremerton was the first, and most intense period of exposure in his lifetime. Each of the medical experts also testified that Sparks's asbestos exposure on the Bremerton was almost certainly sufficient to have caused his mesothelioma. At least one of these experts further stated that Sparks's exposure to Kaylo during the decommissioning was, by itself, sufficient to have caused his disease.


In so ruling, the Sparks court cited Lineweaver’s exposure standard and noted that although the frequency and proximity of exposure are indeed relevant factors when determining exposure, such factors are not necessary to prove exposure in every case:

In a case decided while appellant's petition for rehearing was pending, Division One of this court discussed the burdens of proof on the issues of causation for asbestos-related personal injuries. (Lineweaver v. Plant Insulation Co. (1995) 31 Cal.App.4th 1409, 1416 [37 Cal.Rptr.2d 902].) Writing for the majority, Justice Strankman held that the plaintiff has the
burden of proving that "there [is] a reasonable medical probability based upon competent expert testimony that the defendant's conduct contributed to the plaintiff's injury." The court further observed that many factors are relevant in assessing the medical probability that an asbestos exposure was a "substantial factor" in causing the plaintiff's disease: "Frequency of exposure, regularity of exposure, and proximity of the asbestos product to plaintiff are certainly relevant, although these considerations should not be determinative in every case.


Moreover, in adopting its liberal substantial factor standard in Rutherford v. Owens-Illinois (1997) 16 Cal.4th 953, the Supreme Court explicitly stated that a plaintiff in a toxic injury action need not quantify the dose, level, frequency, or duration of his exposure to a toxin to prove medical causation. Rather, a plaintiff need only submit exposure and causation evidence which permits the jury to reasonably conclude that plaintiff’s exposure to a particular defendant’s product was a substantial factor in causing his disease:

It might also be possible to fashion an instruction that shifted the burden on causation only after the plaintiff had proven, in addition to exposure as such, sufficiently lengthy, intense and frequent exposure as to render the defendant's product a substantial factor contributing to the risk of cancer. As explained earlier, however, there is no need for such a tailored burden shifting instruction; instead, we have determined the jury should simply be told that substantial factor causation can be shown through evidence of exposure to a defendant's product that in reasonable medical probability contributed to the plaintiff or decedent's risk of developing cancer.

Indeed, in stating that a plaintiff is required to show exposure to asbestos from a particular defendant’s product in order to establish medical causation, the Supreme Court cited several California and foreign decisions employing “lenient” and “stringent” exposure standards, none of which require a plaintiff to quantify the frequency, duration, level, or dose of his asbestos exposure, but rather require a plaintiff to introduce a greater or lesser degree of circumstantial evidence which permits a reasonable inference of substantial exposure to a defendant’s asbestos products:
FN12 We do not here endorse any one particular standard for establishing the requisite exposure to a defendant's asbestos products, as the issue has not been raised or briefed in this case. We note that a number of different formulations have been applied, both in the reported California cases, and in federal and sister-state jurisdictions. (See, e.g., Dumini v. Owens-Corning Fiberglas Corp., supra, 28 Cal.App.4th at p. 655 [applying "the most generous application of a lenient causation standard"]; In re Hawaii Federal Asbestos Cases (9th Cir. 1992) 960 F.2d 806, 816-817; Blackston v. Shook & Fletcher Insulation Co. (11th Cir. 1985) 764 F.2d 1480, 1485 [stringent approach requiring particularized proof that the plaintiff came into contact with the defendant's product]; Lockwood v. AC & S Inc. (1987) 109 Wn.2d 235 [744 P.2d 605, 613] [lenient approach; sufficient if plaintiff proves defendant's product was at his or her work site, but resolution depends on particular circumstances of each case].)


Thus, in a hydroquinone-leukemia case in California a plaintiff is not required to introduce evidence which quantifies his exposure to hydroquinone, whether by cumulative exposure in parts per million years or some other dose/exposure metric, in order to prove that his exposure hydroquinone was a substantial factor in causing his leukemia. Instead, a plaintiff need only introduce direct and/or circumstantial evidence which provides a reasonable inference that a plaintiff was substantially exposed hydroquinone, and expert medical opinion that this exposure to hydroquinone was a substantial factor in causing his leukemia.

Several courts from other jurisdictions agree with California’s position that a plaintiff need not quantify his exposure to prove exposure and medical causation in a toxic injury action. See, e.g., the following:

Amateis v. City of Bridgeport (2000) 2000 Neb.App. Lexis 194 [“We are not prepared to hold that a plaintiff must prove a mathematically precise level of exposure in order to recover in a toxic tort case. Roth-Nelson’s testimony that Anthony was exposed to a sufficiently high concentration to cause his seizures, along with the testimony of the other experts, was sufficient to support the court’s findings regarding proximate cause.”]

Bainbridge v. Boise Cascade Plywood Mill (1986) 111 Idaho 79, 721 P.2d 179 [“Boise Cascade urges that Ms. Bainbridge has not presented a prima facie case because
NIOSH air quality standards permit formaldehyde levels of up to one part per million and the record provides no evidence indicating that the formaldehyde levels at the situs either reached or exceeded that level. However, to rest its case, as Boise Cascade does at this point, does not negate the prima facie case. The NIOSH standard only sets a parameter for measuring the toxicity of formaldehyde fumes alone. It does not purport to establish a safe level for the combination of formaldehyde and wood particulates. Indeed, Boise Cascade never addressed the issue of whether formaldehyde, at whatever level of concentration in the air, would be absorbed into the wood dust particulates thereby increasing either the concentration of formaldehyde or the toxic effect of the combination. Moreover, evidence was presented indicating that highly susceptible individuals could contract such asthma at a level below the NIOSH standard.”

Clausen v. M/V New Carissa (9th Cir. 2003) 2003 WL 22208783 [“The fact that the minimum threshold level of oil necessary to cause harm to shellfish has not yet been established with any degree of certainty does not render Dr. Elston's evaluation mere guesswork, as the shipowners argue. While ‘precise information concerning the exposure necessary to cause specific harm [is] beneficial, such evidence is not always available, or necessary, to demonstrate that a substance is toxic ... and need not invariably provide the basis for an expert's opinion on causation.’ Westberry [v. Gislaved Gummi AB (4th Cir. 1999)] 178 F.3d [257] at 264; Heller [v. Shaw Industries, Inc. (3d Cir. 1999) 167 F.3d [146] at 157 (‘even absent hard evidence of the level of exposure to the chemical in question, a medical expert could offer an opinion that the chemical caused plaintiff's illness’).”]

Donaldson v. Central Illinois Public Service Company (2002) 199 Ill.2d 63, 767 N.E.2d 314, 262 Ill.Dec. 854 [“Additionally, we reject CIPS’s assertion that causation includes a showing of exposure, which must be quantified. A Plaintiff must establish that he or she came into contact with chemicals produced by the defendant. See Mitchell v.
Gencorp, Inc. 165 F.3d 778, 781 (10th Cir. 1999) [In this context, however, Illinois law does not require that plaintiffs quantify the level of exposure. . . . Environmental exposure cases, like the instant case, do not afford litigants the opportunity to specify with such certainty the exact level and dose of exposure. In most instances, the details of exposure, including information of exactly when or where exposure occurred, is not available. Here, plaintiffs were not required to show the exact amount of exposure. See La Salle National Bank v. Malik, 302 Ill.App.3d 236, 235 Ill.Dec. 755, 705 N.E.2d 938 (1999) (the inability to show the level of exposure did not bar an expert’s opinion); Harris [v. Cropmate Co. (1999)] 302 Ill.App.3d [364] at 371 (discussing causation testimony that did not calculate the concentration of exposure, but instead reached the conclusion that exposure occurred based upon their “generalized knowledge and firsthand experience with and observations of the effects of exposure”)]

Elam v. Alcolac, Inc. (1988) 765 S.W.2d 42, 1988 Mo.App. LEXIS 1546 [“A person subjected to chronic, long-term chemical exposures is not expected to foster a study group in order to prove liability for a disease not even anticipated. . . . This lack of detail of exposure quantitation notwithstanding, the evidence agrees that the plaintiffs were exposed on sufficient occasions, for sufficient durations, in sufficient concentrations to toxic Alcolac chemicals to cause them recurrent [and even chronic] irritations and ailments to their eyes, skin and upper respiratory systems. . . . In this state of the evidence, testimony of the ‘total adverse effects produced by the toxicant when administered continuously over a long period of time’ bears as circumstantial proof of causation.”]

Fulmore v. CSX Transportation, Inc. (2001) 252 Ga.App.884, 557 S.E.2d 64 [“While both analyses involve a question of the concentration levels of the toxin to which the plaintiffs were exposed, it does not necessarily follow that plaintiffs must show specific air measurement readings, or that they have not otherwise established causation.”]
Lewis v. FAG Bearings Corp. (Mo.App. 1999) 5 S.W.3d 579 [“Even where the evidence does not identify the particular chemical at a particular exposure, the particular concentration of the chemical, the particular dosage of the chemical taken in bodily, or the particular duration of the exposures, the identity of the toxic substances to which the harm is attributed may be shown by circumstantial evidence.” Id., citing Elam v. Alcolac, Inc., supra, 765 S.W.2d at 178-179.

Rockwell International v. Turnbull (Colo.App. 1990) 802 P.2d 1182 [“Nor do we agree that the record fails to support Dr. Aldrich’s assumption that claimant was exposed to harmful quantities of toxic chemical substances, including perchloroethylene and 1,1,1-trichloroethane. This assumption is supported by the employer’s acknowledgment that 1,1,1-trichloroethane was used daily in the area where claimant worked. In addition, claimant and a co-worker, both testified regarding workplace exposure to solvents prior to 1978, and to claimant’s virtually “continuous” exposure to a “fog” of a refrigerative coolant that was sometimes rancid with bacterial contamination during his last year of employment. Contrary to the petitioner’s suggestion, lay testimony is sufficient to support a determination of injurious exposure.”]