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APPROVAL OF THE APPELLATE DIVISION

SUPERIOR COURT OF NEW JERSEY
APPELLATE DIVISION
DOCKET NO. A-3509-08T3

JEANNETTE LEWIS, Administratrix
of the ESTATE OF NICHOLAS LEWIS,
SR., and JEANNETTE LEWIS,
Individually,

Plaintiff-Appellant/
Cross-Respondent,

v.

AIRCO, INC., as successor in
interest to AIR PRODUCTS AND
CHEMICALS, INC.; ALLSTATE INSURANCE
COMPANY; HONEYWELL INTERNATIONAL,
INC., f/k/a ALLIED SIGNAL, INC.,
individually and as successor in
interest to ALLIED CHEMICAL
CORPORATION; THE AMERICAN CHEMISTRY
COUNCIL, f/k/a/ THE CHEMICAL
MANUFACTURERS ASSOCIATION and THE
MANUFACTURING CHEMISTS ASSOCIATION;
BRIDGESTONE/FIRESTONE, INC.,
individually and as successor in
interest to THE FIRESTONE TIRE &
RUBBER COMPANY and FIRESTONE
PLASTICS COMPANY, a division of
THE FIRESTONE TIRE & RUBBER CO.;
CONDEA VISTA COMPANY, individually
and as successor in interest to
CONOCO, INC., individually and as
successor in interest to the
CONTINENTAL OIL COMPANY and THOMPSON-
APEX COMPANY and CONOCO CHEMICALS;
THE DOW CHEMICAL COMPANY, individually
and as successor to UNION CARBIDE
CORP.; ETHYL CORPORATION; GENCORP,
individually and as successor in
interest to GENERAL TIRE AND RUBBER

COMPANY; POLYONE CORPORATION, f/k/a/
THE GEON COMPANY; GEORGIA GULF CORPORATION,
as successor in interest to GEORGIA
PACIFIC CORPORATION; GOODYEAR TIRE AND
RUBBER COMPANY; GULF OIL CORPORATION,
individually and as successor in
interest to CHEVRON U.S.A., INC.;
PHARMACIA CORPORATION f/k/a MONSANTO
COMPANY;[1] NEW JERSEY MANUFACTURERS
INSURANCE CO.; OCCIDENTAL CHEMICAL
CORPORATION, individually and as
successor in interest to OCCIDENTAL
ELECTROCHEMICALS CORPORATION and
HOOKER CHEMICAL CORPORATION and
DIAMOND SHAMROCK CHEMICALS COMPANY and
DIAMOND CHEMICALS COMPANY and
DIAMOND SHAMROCK CORPORATION and
DIAMOND ALKALI COMPANY; OCCIDENTAL
ELECTROCHEMICALS CORPORATION; OLIN
CORPORATION; PACTIV CORPORATION;
PANTASOTE, INC.; RHONE-POULENC, INC.,
individually and as successor in
interest to STAUFFER CHEMICAL COMPANY;
THE SOCIETY OF THE PLASTICS INDUSTRY, INC.;
TENNECO AUTOMOTIVE, INC.; TENNECO,
INC.; EPEC POLYMERS, INC., as
successors to TENNECO OIL COMPANY;
SENTRY INSURANCE COMPANY; SHINTECH,
INC.; UNION CARBIDE CORPORATION;
UNIROYAL, INC., individually and as
successor in interest to U.S. RUBBER
COMPANY; WHITTAKER CORPORATION,
individually and as successor to
GREAT AMERICAN CHEMICAL CORPORATION;
LIBERTY MUTUAL CORP., and ZENECA, INC.,
f/k/a ICI AMERICAS, INC.,

Defendants,

and

GOODRICH CORPORATION, f/k/a

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[1] Incorrectly designated in the third-amended complaint as Monsanto Corporation.

B.F. GOODRICH COMPANY; PPG INDUSTRIES, INC.; SHELL OIL COMPANY, individually and as successor in interest to SHELL CHEMICAL, INC. and SHELL CHEMICAL COMPANY; and HEXION SPECIALTY CHEMICALS, INC., f/k/a/ BORDEN CHEMICAL, INC.,

Defendants-Respondents/
Cross-Appellants.

Argued October 6, 2010 – Decided July 15, 2011

Before Judges Fuentes, Gilroy and Ashrafi.

On appeal from the Superior Court of New Jersey, Law Division, Essex County, Docket No. L-10503-02.

Mark R. Cuker argued the cause for appellant/cross-respondent (Williams Cuker Berezofsky and Ron Simon (Simon & Associates) of the Washington D.C. bar, admitted pro hac vice, attorneys; Mr. Cuker, of counsel and on the brief).

Thomas L. Feher (Thompson Hine, LLP) of the Ohio bar, admitted pro hac vice, argued the cause for respondents/cross-appellants Goodrich Corporation, PPG Industries, Inc., and Shell Oil Company (Lowenstein Sandler, PC, and Mr. Feher, attorneys; David W. Field and Priya R. Masilamani, of counsel; Mr. Field, on the brief).

Timothy E. Corriston argued the cause for respondent/cross-appellant Hexion Specialty Chemicals, Inc. (Connell Foley, L.L.P., attorneys; Mr. Corriston, of counsel; Mr. Corriston and Andrew B. Buckman, on the brief).

PER CURIAM

This is an occupational-exposure, toxic-tort products liability action. Plaintiff, Jeannette Lewis, as Administratrix of the Estate of Nicholas Lewis, Sr. (Lewis), and individually, appeals from the December 15, 2008 order that barred the trial testimony of two of her liability experts; and from the February 5, 2009 order that denied her first motion for reconsideration and granted defendants Goodrich Corporation (Goodrich), PPG Industries, Inc. (PPG), Shell Oil Company (Shell), and Hexion Specialty Chemicals, Inc. (Hexion) (collectively, the defendants) summary judgment. Plaintiff also appeals from the March 20, 2009 order that denied her second motion for reconsideration.

Goodrich, PPG, and Shell cross-appeal from: the November 8, 2007 order that denied their motions for summary judgment on plaintiff's inadequate warning and civil conspiracy claims; the May 29, 2008 order that denied their motions for reconsideration; the December 15, 2008 order that denied their motions seeking to bar the testimony of two of plaintiff's liability experts; and the February 5, 2009 order that denied their motions for reconsideration of the order that denied summary judgment on plaintiff's inadequate warning claims based on the state-of-the-art defense. In a separate cross-appeal,

Hexion appeals from the same orders as the other three defendants. We reverse on the appeal, and affirm in part and reverse in part on the cross-appeals.

I.

Prior to his death on December 31, 2000, Lewis had worked at the Pantasote, Inc., manufacturing facility in Passaic from 1961 to 1989. At its facility, Pantasote manufactured polyvinyl chloride (PVC)[2] resins and products. Vinyl chloride monomer[3] (VCM) (also known in the industry as vinyl chloride), an established carcinogen, is used in the production of PVC. Although occupational exposure to VCM has been causally associated with angiosarcoma (ASL), an extremely rare form of liver cancer, Lewis' cause of death was not from ASL, but rather from hepatocellular carcinoma (HCC), the most common form of liver cancer. In June 2003, believing that her husband's death was caused by exposure to VCM at the Pantasote facility, plaintiff filed a third-amended complaint against numerous parties, including those that manufactured and/or supplied VCM to Pantasote. The third amended complaint alleged causes of

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[2] A glossary of the acronyms frequently used throughout this opinion is set forth in the attached appendix.

[3] The term "monomer" means "a simple molecule that can form polymers by combining with identical or similar molecules." Webster's New World Dictionary 878 (3d ed. 1996).

action sounding in negligence, products liability failure-to-warn of the risks caused by exposure to VCM, and civil conspiracy to fraudulently conceal those risks from the public.

On December 8, 2006, defendants filed motions for summary judgment on the fraud, civil conspiracy, and inadequate warning claims. On November 8, 2007, the court entered an order supported by an oral decision of October 25, 2007, granting the motions on the fraud claim, but denying the motions on the civil conspiracy and inadequate warning claims.

Also on December 8, 2006, defendants filed motions to exclude the trial testimony of plaintiff's four experts: Dr. Peter Infante, an epidemiologist; Dr. Howard Kipen, an occupational physician; Dr. David Groth, a pathologist; and James Jones, an industrial hygienist. On December 3, 2008, following an extensive N.J.R.E. 104(a) hearing in January and May 2008, the court granted the motion as to Infante and Kipen, determining that their conclusions that VCM exposure caused HCC were not sufficiently reliable, but denied the motion as to Groth, and as to Jones in part. The court entered a memorializing order on December 15, 2008.

In December 2008, defendants filed a motion for summary judgment contending that plaintiff could not prove general and specific causation without Infante's and Kipen's testimony.

Plaintiff filed a cross-motion for reconsideration of the order barring not only those two experts from testifying at trial, but also from barring Jones' testimony in part. On February 5, 2009, the trial court entered an order supported by an oral decision of February 3, 2009 denying plaintiff's motion for reconsideration and granting defendants' motions for summary judgment dismissing the complaint. On February 20, 2009, plaintiff filed a second motion for reconsideration. The court denied the motion on March 20, 2009.[4]

II.

A. Vinyl Chloride and the Pantasote Facility and the Manufacturing Process.

Under normal pressure, VCM is a colorless flammable gas that has a mild sweet odor detectable at concentrations between 2,000 and 3,000 parts per million (ppm). According to a 1992 United States Environmental Protection Agency (USEPA) publication, inhaled VCM has been shown to increase the risk of developing ASL. Acute short term exposure to high levels of VCM affects the central nervous system. Long-term exposure is linked to liver damage and acroosteolysis, a degenerative

[4] During the course of the action, all other defendants either settled or were dismissed from the action. Plaintiff also dismissed her failure-to-warn claim against Hexion, but not the civil conspiracy claim.

disease causing loss of bone tissue in the hands and sensitivity to cold.

Pantasote manufactured PVC resins and products, including vinyl film and sheeting, plasticized compounds, and thermoformed products. In 1957, Pantasote manufactured PVC resins in plant 1, and in 1960 Pantasote expanded production to include the larger plant 2. Until 1984, the facility used about 145,000 pounds of VCM a day to produce approximately 160,000 pounds of PVC, operating twenty-four hours a day, seven days per week, with three rotating shifts of workers. Goodrich, PPG, and Shell supplied VCM to the plant; Hexion did not. Pantasote ceased production in 1984 and demolished the plants.

Former Pantasote employees described the Pantasote facility and the PVC manufacturing process, in relevant part, as follows. Liquefied VCM was shipped to the facility in 10,000 to 22,000 gallon rail tank cars and unloaded to underground storage tanks. VCM was then piped in a closed system, from the storage tanks to polymerization reactors where, under pressure, it was combined with deionized water and a suspending agent to produce a PVC slurry. The polymerization process took between eight to twelve hours to complete. The reactor building was ventilated by ceiling and side-mounted fans, which forced air through ducts to bag houses and separators, prior to venting into the atmosphere.

After the polymerization process was completed, the PVC slurry was first transferred to outside storage tanks and then to a centrifuge and a dryer. The resultant powdered blend of PVC was air-conveyed through a dust collector, a sifter, and then to a bagging machine. The bags of PVC resin were stored in an adjacent warehouse for shipment, or for manufacturing products at the plant.

In the compounding process, bags of PVC resin were mixed with various additives such as plasticizers, fillers, and stabilizers, to produce PVC compounds. PVC products were manufactured in a semi-closed process in the calendaring buildings, where the compounded product was rolled into thin sheets of plastic. The buildings were ventilated by ceiling and hood exhaust fans.

B. The Workers' Exposure.

Lewis worked at the Pantasote plant as a service operator, chemical operator, or yardman. Although the record indicates that these titles were interchangeably used throughout his employment, Lewis was in charge of unloading VCM from the rail tank cars into the underground storage tanks, regenerating the water and demineralization units in the reactor buildings, transporting bags of PVC resin by forklift to various places

within the plant, and measuring levels of PVC resins in the storage silos.

Lewis' co-workers testified at depositions that VCM had been released into the ambient air in the workplace at levels high enough to have had a detectable odor, either from leaks or from the normal manufacturing process. Joseph Genardi, a co-worker who performed the same job as Lewis, testified that he smelled VCM, which he described as having a "sweet smell," when he unloaded the rail tank cars in the closed pump house. It generally took Genardi three and one-half to five hours to unload the cars. The unloading process involved removing plugs from the tank car valves, attaching lines to the valves, opening the valves, and adjusting the air pressure to force VCM into the underground storage tanks. According to Genardi, there were "always leaks" in the valves, and sometimes in the fittings and gaskets, and when the transfer was completed, the lines to the tank cars were disconnected and any remaining VCM in the line was released and then vented outside the pump house.

Further, Genardi said he always smelled VCM in the reactor rooms, where he and Lewis worked. During a typical shift, Genardi entered the reactor room in plant 1 every hour, and entered the reactor room in plant 2 twice each shift. He was exposed to VCM when the reactors were opened, when slurry was

dumped into the sewers, and when the seals in the reactor rooms were broken and VCM had to be vented out into the atmosphere because otherwise, the whole reactor building would become "filled with vinyl chloride."

Genardi also smelled VCM in the warehouse when he transported the bags of PVC resin by forklift. About three times a week a bag of PVC powder broke, spilling its contents into the warehouse. He said he "would actually smell, not strong, very faintly you would smell vinyl chloride and the resin . . . . It's a smell you never forget."

Michael Rapavi, Lewis' foreman, testified at depositions that prior to 1975, he often smelled VCM in the reactor rooms, in the vicinity of the slurry tanks, in the warehouse, and toward the front area of the plant if the wind was blowing in that direction and if VCM was being vented out from one of the buildings. Lewis' son, Nicholas Lewis, Jr., who began working at the facility in 1976, testified that once while he was walking outside plant 1, he asked his father what that odor was, and his father responded that he "believed" it was VCM. Lewis, Jr., also smelled VCM by the unloading area near the railcars and outside the storage area.

C. Exposure Limits, Toxicity and Monitoring of VCM.

Although VCM was known to be highly flammable and had anesthetic effects when inhaled in high concentration, it was initially considered to be very low in toxicity. In 1947, the American Conference of Government Industrial Hygienists (ACGIH) recommended a maximum allowable concentration (MAC) of exposure to VCM of 500 ppm. MACs, subsequently renamed "threshold limit values" (TLV), are the maximum average concentrations of contaminants to which workers may be safely exposed in an eight-hour day. Also in 1947, an experiment, the Seeler study, revealed no signs of toxicity to animals fed a diet supplemented with PVC.

The "Chemical Safety Data Sheet SD-56" (SD-56), published in 1954 by the Manufacturing Chemists Association (MCA), of which Pantasote and defendants were members, reflected that assessment, and provided that "[a]side from the risk of fire and explosion, vinyl chloride presents no other very serious problem in general handling. The presently accepted upper limit of safety as a health hazard is 500 ppm."

However, in 1959, the industry learned that there were indications of VCM toxicity in ongoing laboratory animal experiments conducted by Dow Chemical scientists, T.R. Torkelson and V.K. Rowe. The Torkelson study exposed rats and other small

animals to VCM levels ranging from 50 ppm to 500 ppm, levels that had been considered safe for human exposure, for seven hours a day over the course of seven months.

In a letter dated May 12, 1959, Rowe, commented on the ongoing Torkelson study, and informed the Director of Goodrich's Department of Industrial Hygiene and Toxicology that the 500 ppm threshold established by the ACGIH was based on a flawed animal study. Rowe wrote that "[w]e feel quite confident . . . that 500 ppm is going to produce rather appreciable injury when inhaled 7 hours a day, five days a week for an extended period." In another comment on the study in an interoffice correspondence dated November 24, 1959, a Union Carbide employee wrote that

> [a]n off-the-record phone call from V.K. Rowe gives me incomplete data on their current repeated inhalation study. Six months at 500, 200 and 100 [ppm] has not found a no-effect level. Even 100 [ppm] produced organ weight changes and gross pathology, with micropathology expected. [VCM] is more toxic than has been believed.

An experiment conducted and published in 1960 by E. Mastromatteo and others also found some congestion in the livers of laboratory animals exposed to high levels of VCM. And, the Torkelson study, published in 1961, concluded that VCM exposure had a "slight capacity" to cause liver damage in laboratory animals. The authors recommended that the TLV be lowered to 50

ppm, and Dow began applying this exposure value in some of its plants.

However, in a subsequent study published by D. Lester and others, the authors found only increased liver weights in rats exposed to VCM, changes they did not consider significantly pathologic. Thus, they concluded that a TLV of 500 ppm for VCM "seems to offer an adequate margin of safety for human exposure."

Faced with these differing results, in 1962, the ACGIH sided with Lester, and found that, although the available data concerning the toxicity of VCM was "conflicting, the preponderance indicates a compound of relatively low toxicity with which a threshold limit of 500 ppm is consistent."

In 1963, Goodrich began testing employees at its Louisville plant for liver function. In a letter dated June 7, 1965, Rex Wilson, a physician employed by Goodrich, stated that it had been his experience that VCM is a "hepatoxin when exposure is prolonged and high in amount."

In the mid-1960's, reports surfaced linking VCM exposure to acroosteolysis in humans. In Wilson's 1967 published report of thirty-one cases of acroosteolysis, twenty-seven of the workers were autoclave workers, that is, they cleaned polymer from PVC reactors. Goodrich reported observations of acroosteolysis in

some of its PVC workers at an October 6, 1966 MCA meeting. Some industry members predicted that the TLV level for VCM would be reduced to 50 ppm.

As a result, Pantasote installed a hydraulic washing system in plant 2, which obviated the need for autoclave workers to enter the reactor vessel and clean the walls. It also installed additional exhaust fans to increase ventilation, and restricted access to the reactor areas and tank farms to only essential personnel in plants 1 and 2.

In August 1968, scientists employed by Dow Chemical Company presented a report at the Gordon Research Conference on Industrial Hygiene that suggested "long-term weighted exposures at or above 300 ppm could result in adverse functional changes." In 1970, the newly created Occupational Safety & Health Administration (OSHA) followed the established exposure limits and set federal standards for VCM of 500 ppm, the maximum average concentration of VCM to which a worker could be exposed in an eight-hour workday.

In that same year, P. L. Viola, an Italian researcher, presented his findings at a conference, reporting that rats developed cancerous tumors of the skin, lungs and bones after being exposed to 30,000 ppm of VCM four hours a day, five days a week, for twelve months. The results were subsequently

published in 1971, with Viola stating that "[n]o implications to human pathology can be extrapolated from the experimental model reported in this paper."

The Viola study was extensively discussed by members of the VCM industry. On November 16, 1971, a Pantasote representative attended an MCA meeting during which the results of the Viola study were described. The association's members agreed to sponsor independent research regarding the carcinogenicity of VCM. The MCA also found that "[t]he seriousness of Dr. Viola's findings, if properly substantiated, can have potentially damaging results for the entire vinyl chloride industry."

In 1972, the MCA revised SD-56, warning that "[c]hronic overexposure [to vinyl chloride] may produce liver injury." It stated that "[r]ecent research studies from Italy indicate that repeated, long-term high level exposures of rats to [VCM] vapor can result in the development of malignant tumors. However, many years of industrial experience . . . have not demonstrated any carcinogenicity to humans." And, it stated that VCM "does not present a serious industrial health hazard provided workers are adequately supervised and observe the proper means of handling it." OSHA "has set a 500 ppm Ceiling Value on permitted employee exposures. Based upon animal and human observations, this level provides considerable margin of safety

for industrial exposures." However, in 1970 ACGIH recommended that the TLV level for VCM exposure be reduced from 500 to 200 ppm.

In November 1972, several members of MCA, including defendants, executed a "Secrecy Agreement," securing release of data, and pledging not to reveal information outside the organization about a European animal study on the effects of VCM, which was being conducted by Cesare Maltoni at the University of Bologna. The MCA learned that Maltoni had found positive carcinogenic effect in rats at doses of 250 ppm of VCM, and one case of ASL.

At a meeting in February 1973, MCA members expressed the need to defocus their concern about carcinogenicity and VCM, and to refute the European research. In July of 1973, an MCA task force met with OSHA, but withheld information about the European studies and called into question the results of the Viola study.

On January 23, 1974, Goodrich issued a press release disclosing that three of its VCM-exposed workers at its Louisville plant had died of ASL; two died in 1973, and the third died in 1971. Four other deaths from ASL were reported at Goodrich's plant in Shawinigan, Quebec, with the earliest death occurring in 1968. As a result, in April 1974, OSHA issued a temporary emergency VCM exposure limit of 50 ppm, and effective

April 1975, set a permissible exposure limit of 1 ppm as an eight-hour time-weighted average, and 5 ppm as a ceiling value averaged over a period exceeding fifteen minutes. 29 C.F.R. § 1910.1017 (2010). Areas exceeding that standard were designated as "regulated areas."

In conformance with those standards, Pantasote installed a monitoring system in plants 1 and 2, areas that had exceeded the OSHA standard. All other areas, including calendaring, compounding, and thermoforming, did not exceed the OSHA action level.

No sampling data for the period prior to 1974, other than as set forth by Pantasote in correspondence to the USEPA, was available for the facility. For example, in a memo to the USEPA, dated June 14, 1974, Pantasote reported that atmospheric levels of VCM at the plant perimeter, on the side where tank cars were unloaded and the reactor was located, ranged from .9 to 5.5 ppm. Pantasote used devices to collect PVC emissions, including a monomer recovery system, dust collectors, and vent bags on the storage silos. The systems collected approximately 8% of the total monomer used. It estimated that without the systems, 724 pounds of PVC per hour would be released into the atmosphere, and that the systems reduced emissions to about .36 pounds per hour.

On October 17, 1974, a representative from the National Institute for Occupational Safety and Health (NIOSH) visited the Pantasote facility and reported that the company had an

> active health, safety, and sampling program and [is] interested in cooperating with the NIOSH survey program. [It is] concerned that the vinyl chloride levels may be somewhat higher in their processing plant in that it is located between the two . . . [PVC] resin production plants. Information on vinyl chloride is transmitted to their employees through union-management meetings . . . and direct meetings [with] the resin plant employees. Sampling is conducted in the resin . . . and processing plants . . . . . The sampling program is under constant change to comply with OSHA regulations. The . . . samples are analyzed in [its] laboratories at this site.

In interoffice correspondence dated March 23, 1976, Pantasote reported that there had been high exposure levels of VCM loose at all times since the plant opened in 1958, and that the company was doubtful that they could lower the exposure levels to comply with the federal standards.

By the mid-1970's, Pantasote conducted medical tests on its plant employees, including Lewis. Not only were signs posted in the plant warning that VCM was a "Cancer Suspect Agent," but also warnings were imprinted on the bags of PVC manufactured at the plant. Pantasote also provided some of its workers with respirators, although Lewis, Jr., testified that his father did not use one.

Industry-wide, occupational exposure to VCM decreased substantially after 1975. In 1981, the Pantasote plant reduced the content of VCM in the PVC resin, thereby further lowering VCM exposure.

D. Expert Evidence (Exposure and Warning).

James Jones, a certified industrial hygienist, was first contacted by plaintiff sometime after Lewis' death, and after demolition of Pantasote plants 1 and 2. Jones received a B.S. in chemical engineering and completed some graduate work. As an undergraduate, he worked at Goodrich's Louisville plant. Later, at the NIOSH, he directed the exposure assessment portion of its VCM study, and gathered data on employee exposures to VCM by visiting six or seven PVC production facilities. In 1974, he assisted the NIOSH director in preparing to testify before OSHA concerning establishing new exposure limits for VCM.

Jones opined that from 1961 to 1974, Lewis had been exposed to a significant quantity of VCM. He explained that no quantitative exposure sampling data existed prior to 1974, and that because Pantasote's manufacturing facility no longer existed he was not able to monitor the current exposure levels. Thus, he relied significantly upon VCM odor threshold levels to estimate Lewis' exposure. He used a 2,000 ppm odor threshold, as set forth in some scientific literature, although the USEPA

had established a higher threshold of 3,000 ppm, and VCM manufacturers had shown thresholds ranging upwards between 1,200 to 3,800 ppm. He explained that whenever a worker smelled VCM, that worker would have then already been exposed to VCM levels above 2,000 ppm, which he said constituted a "substantial exposure."

Jones concluded, based on Lewis' job functions, the reports of pervasive odor of VCM at the facility, and Jones' experience in other PVC plants, that Lewis had been exposed to in excess of 40 to 50 ppm of VCM annually, or a cumulative minimum exposure, from 1961 to 1974, of 520 to 650 ppm. He opined in his report that Lewis'

> VCM exposure would have been variable, but . . . [Lewis] would have been regularly exposed to levels that were very high (probably in the thousands of ppm range) while unloading VCM tank cars and because of ambient VCM concentrations caused by the release of VCM from reactors and resin storage silos. He did not [wear] a respirator during this time [.] . . . Lewis' [cumulative] VCM exposure would likely be in excess of 500 ppm-years [i.e., over the years he worked at Pantasote], primarily because of exposure before 1974.

Jones further testified that the exposure warnings given to the Pantasote workers prior to 1974, about the risk of developing liver damage from exposure to VCM were inadequate. By 1970, defendants were aware of animal studies linking VCM

exposure to liver cancer, reports that workers in foreign PVC plants developed liver damage, and recommendations in the scientific literature that VCM exposure levels should be lowered to 50 ppm. Jones concluded in his report:

> [I]n light of these multiple indications that VCM was harmful to workers at exposure levels as low as 50 ppm and the finding of cancer in VCM exposed animals, users of VCM should have begun monitoring and controlling exposures to VCM, and suppliers of VCM and the MCA should have lowered their exposure recommendations at least as early as 1961 and given adequate warning of the various health effects found in humans and animals. If this had been done, worker exposure levels at this plant would likely have been lowered much earlier than 1974, and the potential for harm, such as liver disease and cancer, to . . . Lewis, and other workers would also have been significantly reduced.

E. Lewis' Diagnosis.

In August 2000, Lewis, then seventy-five years old, physically active, with no history of hepatitis or alcohol abuse, was diagnosed with "metastatic disease to the liver and adrenals." During an examination conducted on November 2, 2000, Dr. Michael Maroules reported that Lewis had "widely disseminated cancer." A computerized tomography (CT) scan revealed that Lewis had a huge tumor in his liver, measuring in excess of 15 centimeters, multiple tumors in his lungs, and tumors in his adrenal glands. Lewis died on December 31, 2000.

It was undisputed that Lewis had not developed ASL. ASL is a sarcoma, or cancer of the connective tissue, which develops in the endothelial lining or sinusoidal cells, and is causally associated with exposure to VCM. HCC, accounting for approximately 80% of all primary liver cancers, is a carcinoma or cancer of the epithelial tissue, which develops in the hepatocytes or liver cells. The major risk factors for developing HCC are infection with the hepatitis-B and -C viruses, and the abuse of alcohol.

There was, however, some dispute as to whether Lewis had primary HCC, and whether the cancer had metastasized from, not to, his liver. Most malignant liver tumors metastasize to the liver from other areas of the body, but there are six types of cancers that originate in the liver, including HCC and ASL. Some records indicated that Lewis' tumor had metastasized to his liver. For example, a radiologist's report dated October 18, 2000, indicated that Lewis' CT scan findings were "consistent with metastatic disease." In Dr. Maroules' oncology report dated November 2, 2000, he stated that a "CT guided biopsy of the liver revealed malignancy, possibly endocrine." Maroules also noted in his report that the biopsy results were "consistent for endocrine source," but testified at depositions that he could not "definitely say what kind of cancer [Lewis]

had." Moreover, Lewis' cause of death was listed on the death certificate as "[m]etastatic carcinoma to the liver, primary unknown."

However, other records supported a diagnosis of HCC. A November 7, 2000, pathology report revealed that the tests results "favor hepatocellular carcinoma." The hospital discharge summary, dated November 12, 2000, listed a final diagnosis of "[h]epatocellular carcinoma with metastasis." Another pathology report dated November 14, 2000, stated that the results of a fine needle aspiration were "suggestive of hepatocellular carcinoma."

Dr. David Groth, a board certified pathologist, opined that Lewis had primary HCC that metastasized to his lungs, possibly his adrenal glands, and caused his death. In reaching that conclusion, Groth noted that Lewis' liver tumor was about the size of a grapefruit, and was one of the largest, if not the largest, tumor he had ever seen in the thousands of cases of cancer he had studied. According to Groth, Lewis also had several small nodules in his liver, a common finding when the primary liver tumor was greater than 5 centimeters in diameter, and Lewis had elevated levels of alpha-fetoprotein, which is consistent with primary HCC.

Groth examined ten of Lewis' fifty biopsy slides before rendering his report, and another ten before his pre-trial deposition. He explained that the tissue of a malignant tumor will look similar to the tissue of the primary source. The slides revealed a canaliculi pattern, which are only found in the liver, thereby indicating that the primary source of the cancer was the liver. However, Groth admitted that at the time he prepared his report he had not viewed the actual slide revealing the canalicular pattern, but instead had derived that information from the pathology report. In a later certification pre-dating the Rule 104 hearing, Groth stated he subsequently reviewed Lewis' fifty pathology slides, and found there was "nothing . . . on the remaining slides to change" his opinion that Lewis had died from HCC.

Groth explained that the blood test results ruled out several other types of cancer including adenocarcinoma, melanoma, pancreatic, prostrate, and carcinoid tumors, which generally occur in the lungs or the gastrointestinal tract. Groth also ruled out lung cancer as the primary source, because the multiple small nodules found in Lewis' lungs indicated that the cancer had metastasized to the lungs.

F. Causation.

Dr. Peter Infante, an epidemiologist, specialized in evaluating occupational exposures and co-authored more than 100 peer-reviewed articles, some of which concerned the effects of exposure to VCM. He had been responsible for industry-wide epidemiological studies, including studies of VCM at NIOSH, and was a director of the department at OSHA that set permissible limits for workplace toxic exposure.

Infante also had served on the World Health Organization's International Agency for Research on Cancer (IARC) committee on the "Evaluation of the Carcinogenic Risk of Chemicals to Humans" from 1977 to 1979. The IARC evaluates data, determines the carcinogenicity of various agents, examines whether agents cause human cancer, and publishes monographs[5] containing critical reviews of the data on carcinogenicity. The IARC classifies agents into four categories; group one is reserved for substances known to be carcinogenic to humans. OSHA relies on IARC evaluations in determining what information industries must include on their labels and on the material safety data sheets (MSDS).

[5] "Monograph" means "a learned detailed thoroughly documented treatise covering exhaustively a small area of a field of learning." Kanter v. Warner-Lambert Co., 122 Cal. Rptr. 2d 72, 77 n.2 (Ct. App. 2002) (quoting Webster's Third New International Dictionary 1462 (1986)).

Infante opined that not only is occupational exposure to VCM a cause of HCC in humans, but also a significant contributing factor and likely cause of Lewis' HCC. He explained that at least one laboratory animal study in the early 1970's had shown VCM exposure caused ASL in animals, and that VCM exposure was first identified as a human carcinogen in 1974 as a result of Goodrich's case reports that three of its employees had died from ASL. Although admitting that HCC and ASL are different types of liver cancer that have "different cell type origination," Infante explained that epidemiological and animal studies conducted after 1974 demonstrated to a statistically significant degree that exposure to VCM causes ASL and HCC.

In determining general causation, Infante primarily relied on two extensive and several minor epidemiological studies published in peer-reviewed scientific literature. In the first extensive study published by Otto Wong and others in 1991, the authors performed an update of a 1973 cohort study,[6] which

[6] "Cohort studies" in the context of epidemiology are "studies that 'involve the identification of two groups of individuals: 1) individuals exposed to a substance that is considered a possible cause of disease; and 2) individuals who have not been exposed. The study takes place over a specified period; researchers determine the proportion of individuals in each group who develop the disease of interest. Where a particular agent causes the disease, one should expect a higher proportion (continued)

tracked the mortality of 10,173 workers exposed to VCM from thirty-seven North American plants. The cohort study compared the number of exposed-workers who had died of cancer with the expected number of cases in the unexposed general population. They acknowledged that, "[w]hile there is little doubt about the relationship between occupational exposure to vinyl chloride and [ASL]," as of 1991 there was still controversy about the association between vinyl chloride and other cancers. Thus, the update was conducted to "monitor the mortality pattern of the cohort of vinyl chloride workers and attempt to resolve some of the outstanding issues," including the relationship between exposure to VCM and the development of other types of cancers.

The O. Wong study reported that from 1942 to 1982, 37 of the 10,173 workers died of liver cancer (including both ASL and HCC), compared with an expected or background rate, based on United States mortality rates of 5.77. The standardized mortality ratio (SMR), or the ratio of observed deaths from liver cancer to expected deaths according to a specific health outcome in a population, was 641.2. An SMR of 100 indicates

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(continued)
of exposed individuals to develop the disease as compared to those who had no exposure.'" Knight v. Kirby Inland Marine, Inc., 482 F.3d 347, 352 n.2 (5th Cir. 2007) (quoting Knight v. Kirby Inland Marine, Inc., 363 F. Supp. 2d 859, 865 n.11 (N.D. Miss. 2005)).

that the death rate due to the disease is the same in the study group as in the general population. Therefore, an SMR of 641.2 means that the exposed workers' risk of dying from liver cancer was 6.4 times greater than that of the general population.

In order to determine the risk of developing liver cancer, excluding ASL which was a known risk, the authors reviewed the workers' death certificates which indicated that out of thirty-seven deaths from liver cancer, fifteen died of ASL, seven died of biliary tract cancer, and fifteen died of other types of liver cancers, including hepatomas (four), HCC (one), hepatic carcinoma (two), carcinoma of the liver (seven), and metastatic liver cancer of unknown primary (one). To verify the diagnosis, the authors compared available pathology reports to the workers' death certificates. The pathology reports revealed that there were twenty-one cases of ASL, not fifteen as stated in the death certificates. Thus, there were sixteen cases of liver/biliary tract cancer (37 (total) - 21 (ASL)), out of an expected rate of 5.7 (SMR = 281). Significantly, they found that even if the ASL cases were eliminated, the incidence of other forms of liver and biliary cancer was higher than expected. Workers with more than twenty years of exposure to VCM had the highest risk of dying from liver and biliary cancer (SMR = 1284.9), and workers first

exposed to vinyl chloride prior to age twenty-five had a significantly increased risk of developing liver cancer.

Infante admitted that the O. Wong study had not found that exposure to VCM was causally associated with HCC. As a result, Infante followed the methodology set forth in the study, but "went one step further," separating out the cases of liver cancer that were not ASL or biliary cancer (37 (total) - 21 (ASL) - 7 (biliary tract cancer) = 9 (unidentified liver cancer)). He assumed that despite the designation on the death certificates, these nine cases were HCC, because HCC was the most common form of liver cancer. The expected rate for a population of that size was three HCC cases, and thus, an exposed worker's risk of contacting HCC was three times greater than for the unexposed population. Infante concluded that the O. Wong study showed there was a statistically significant risk of developing non-ASL or biliary cancer from VCM exposure. However, he admitted that the K.A. Mundt study, a 1999 industry-sponsored update to the O. Wong study, did not support his conclusion that VCM exposure was associated with a significantly elevated risk of HCC.

In the second extensive study conducted by Elizabeth Ward and others published in 2001, the authors updated a 1991 cohort study by L. Simonato involving 12,700 workers exposed to VCM in

nineteen European factories. The study was designed, in part, to evaluate whether VCM induced both HCC and ASL.

The authors identified seventy-one cases of liver cancer, comprising of thirty-seven cases of ASL, ten HCC cases, seven cases of other known histology, and seventeen cases of unspecified types of liver cancer. They found that

> [t]he results of the updated study are generally consistent with the original study with respect to liver cancer and [ASL]. A strong relation is observed between cumulative [vinyl chloride (VC)] exposure and occurrence of liver cancer. An even sharper exposure-response is observed for [ASL] . . . . A marked exposure-response trend with both duration of employment and cumulative VC exposure was present for the ten known cases of [HCC], suggesting that VC exposure may be associated with this tumor as well. An association of [HCC] with VC exposure is biologically plausible, given that [HCC has] been induced by VC in rodents . . . . In addition, cases of [HCC] together with [ASL] have been reported among workers highly exposed to VC.

However, Infante conceded that the authors of the Ward study had not determined a "causal" connection between exposure to VCM and HCC. He also admitted that the study showed there was no elevated risk of non-ASL cancer for workers, like Lewis, who were hired after 1964 and not employed as autoclave workers. Nonetheless, Infante testified that the demonstration of significant trends in the dose response, that is, an increase in

dosage yields an increase in side effects for VCM exposure and HCC, evidenced a causal relationship.

As stated, Infante also relied upon several minor epidemiological studies, including a 1998 Taiwanese study by Chung-Li Du and Jung-Der Wang. This case-controlled study compared the hospital records of 2,224 VCM exposed workers to the hospital records of a group of unexposed workers employed by manufacturers of optical equipment and motorcycles. The authors examined the records of the two groups to determine whether there was an increased risk of hospital admission among workers exposed to VCM. Twelve of the exposed workers developed primary liver cancer, including HCC, ASL, and cholangiocarcinoma (originating in the bile duct). They concluded:

> Vinyl chloride monomer has been shown to be a multipotential carcinogen in animals. In humans, a causal relation has been found between occupational exposure to VCM and [ASL] of the liver. It was not until 1983 that Evans[7] reported two cases of [HCC] among VCM workers. Later on, several epidemiological studies (in the United States and Europe) also corroborated such an association in humans. According to recent experimental studies performed by Froment[8] different molecular mechanisms of

[7] Evans DMD, et. al., Angiosarcoma and Hepatocellular Carcinoma in Vinyl Chloride Workers, 7 Histopathology 377 (1983).

[8] Froment O., et. al., Mutagenesis of Ras Proto-oncogens in Rat Liver Tumors Induced by Vinyl Chloride, 54 Cancer Res 5340 (1994).

> exposure to VCM may lead to different cell types of liver tumor, including [ASL] and [HCC]. No data on viral hepatitis markers, however, are available from these studies. Thus, we are among the first to report such a high incidence of primary liver cancer or [HCC] among VCM workers, and the possible synergistic influence of viral hepatitis deserves more attention.

Infante also relied on a 1981 German case-control cohort study of 7,021 workers by H. Weber and others. The study compared the SMRs of workers exposed to VCM with unexposed workers. Among the exposed workers, there were twelve cases of liver cancer, of which four cases were confirmed as ASL. Infante admitted that the study did not conclude there was a causal relationship between HCC and VCM. However, there were eight cases of non-ASL liver cancer (12 (total) - 4 (ASL)), compared with an expected rate of .8, yielding a relative risk of ten. Thus, Infante found there was a statistically significant risk of developing non-ASL liver cancer from exposure to VCM.

Similarly, in a Taiwanese study published in 2003 by Ruey-Hong Wong and others, the authors noted that recent studies indicated that VCM exposure is associated with HCC. The authors conducted a case-control study from a previously established cohort of 4,096 workers from six PVC manufacturing plants. It was estimated that in the 1960's the workers had been exposed to

cumulative doses of about 500 ppm of VCM. They found there was a significant excess of mortality from liver cancer (twenty-five cases) among the workers, although none of the deaths appeared to have been from ASL. They obtained medical records for eighteen of the twenty-five cases, and determined that a diagnosis of HCC was histologically confirmed in only five cases, although five more cases also were regarded as positive for HCC based on high levels of alpha-fetoprotein, and the remaining eight cases were diagnosed as HCC based on clinical manifestations and imaging studies

Infante acknowledged that the authors of the R. Wong study had not concluded that exposure to VCM causes HCC. And he admitted that "[h]epatitis leads to cirrhosis of the liver, which then leads to [h]epatocellular carcinoma." He explained that exposure to VCM by hepatitis-infected workers, results in a "synergistic interaction," which substantially increases the worker's risk of developing HCC. Significantly, he said the authors of the study had concluded that VCM exposure was an "independent risk factor" for developing HCC.

In another 2003 study by Paolo Boffetta and others, the authors compared the results of eight cohort studies, including the previously discussed Ward, Mundt, Weber, and R. Wong studies, and the Pirastu study discussed infra. The authors

found a "weak but statistically significant" increased risk for developing non-ASL liver cancer from VCM exposure. Although they wrote that "[t]o date, the only cancer that has clearly been associated with vinyl chloride exposure is [ASL]," they concluded that "[a]part from the known risk of ASL, workers exposed to vinyl chloride may experience an increased risk of [HCC]." However, they cautioned that "while the meta-analysis supports a small excess of liver cancers apart from [ASL] and other studies and evidence exists that support[] the hypothesis, clearer evidence is still needed."

The Italian 2003 cohort study by Roberta Pirastu and others reported that mortality rates for HCC and liver cirrhosis was higher than expected for VCM exposed workers whose only job title was bagger, and showed a similar pattern for cumulative exposure as for VCM and ASL. "The study results confirm the causal relationship between VCM exposure and liver [ASL] and add supplementary evidence in favor of a causal explanation of the excess risk for [HCC] and liver cirrhosis as well as lung cancer among only baggers."

Infante also relied on another Italian case control study by Giuseppe Mastrangelo and others published in 2004. The authors initially wrote that "although a large body of evidence from experimental and epidemiologic studies [had] demonstrated

the relationship between exposure to [VCM] and [ASL], . . . there [was] little evidence of a causal association between VCM and [HCC] and liver cirrhosis." However, following their study, they concluded that "VCM exposure is an independent risk factor for the development of HCC and [liver cirrhosis]." They also determined that VCM exposure "interact[s] synergistically with alcohol consumption and additively with viral hepatitis infection."

Infante also reviewed a 1984 article by Carlo H. Tamburro in which the author sought to address the effects of chronic low-grade exposure to VCM. The author explained that VCM "reactants have been shown to covalently bind to both hepatocytes and sinusoidal cells." The author reported that there were at least two cases of HCC in VCM-exposed workers, both of whom had engaged in chronic alcohol consumption. However, the author did not find any relationship "between [ASL] occurrence and [vinyl chloride] exposure occurring after 1966 when exposure levels generally were below 200 ppm." But Infante testified the article referred to studies conducted in the 1970's, and given the very long latency period of VCM induced cancer, workers exposed after 1966 would not yet have developed liver cancer.

Based on his review of the epidemiological studies, Infante opined that exposure to VCM causes HCC, and that although alcohol consumption and hepatitis are also risk factors, exposure to VCM is an independent factor for developing HCC. He admitted, however, that some epidemiological studies had not shown any association between VCM and HCC.

In reaching his conclusion on general causation, Infante also relied on several laboratory animal studies. In the 1981 study by M.J. Radike and others on the effects of ethanol on VCM carcinogenesis, experimental rats were exposed to VCM and ethanol. The rats were divided into four groups: 1) those exposed to 600 ppm VCM; 2) those exposed to 600 ppm VCM and 5% ethanol; 3) those exposed to 5% ethanol; and 4) those not exposed to either VCM or ethanol. The study showed that the rats exposed to VCM developed ASL and HCC, and those exposed to VCM and ethanol had the highest rates of both forms of cancer.

Infante also relied on V.J. Feron's 1978 study on the oral toxicity of VCM in which rats were exposed to varying doses of VCM by incorporating PVC powder into their diet. The death rate from liver cancer was higher in all of the VCM-exposed groups, the rate increased with increasing doses, and the rats that developed hepatocellular tumors had the highest levels of alpha-fetoprotein. Importantly, the tumor response in the liver

appeared to shift from a predominance of ASL at the highest dose level, to a mixture of ASL and HCC at the intermediate levels, to the exclusive development of HCC at the lowest level.

Lastly, Infante relied on a 1981 "short-term burst exposure" study by Robert M. Hehir. Mice were exposed to a single one-hour dose of VCM ranging from 50 ppm to 50,000 ppm (group one), and to ten one-hour exposures of 500 ppm of VCM (group two). Hehir discovered dose related effects, or increased incidences of carcinomas in the mice exposed to ten one-hour high levels of VCM (5,000 to 50,000 ppm). The mice exposed to the same cumulative VCM dose (e.g., 5,000 ppm), but at lower doses of 50 ppm over time (50 ppm x 100 hours = 5,000 ppm cumulative dose), did not have an increased incidence of tumors.

In determining specific causation, Infante accepted as factual Dr. Groth's opinion that Lewis died of primary HCC. He reviewed Lewis' history of occupational exposure to VCM, including internal memos regarding levels of VCM at the Pantasote facility, co-worker's deposition testimony regarding the odor of VCM at the facility indicating an exposure of 3,000 ppm of VCM, and Lewis' job responsibilities. Infante also relied on his own experiences in studying the effects of VCM, and on Jones' report regarding Lewis' cumulative exposure. He

found that cumulatively, Lewis had significant exposure to VCM, probably in excess of 500 ppm.

Infante opined that Lewis had an "extremely high risk" of developing liver cancer based on his significant occupational exposure to VCM over twenty-seven years, his age at first exposure, and the length of his exposure. He explained that Lewis fit several of the VCM exposure and latency categories presented in the O. Wong study, and therefore, had a significantly elevated risk of death from liver cancer.

Infante also found that Lewis did not have either of the two major risk factors for developing liver cancer, namely, cirrhosis of the liver or hepatitis. Infante did not address obesity as a possible cause of Lewis developing HCC because according to Infante, there are no studies demonstrating that obesity is a factor in developing the disease. Nonetheless, he admitted that his opinion was limited by the fact that HCC is the most common form of liver cancer, and the number of people developing the disease is on the rise.

Dr. Howard Kipen, a physician who specialized in occupational and environmental medicine for more than twenty years, opined that occupational exposure to VCM is a cause of HCC in humans, and was the cause of Lewis' HCC. In determining general causation, Kipen cited three reported cases of HCC in

exposed workers, and relied on many of the same epidemiological studies as Infante, including the R. Wong study. Kipen also noted that the Ward study had found that the risk for developing HCC "increased with increasing estimated cumulative dose," that is, the study "demonstrated . . . a dose response relationship. That's pretty strong." Additionally, Kipen testified that the Mundt study gave "some credence" to the fact that there was an increased risk of developing HCC from exposure to VCM, although he admitted the study did not find that VCM exposure was causally related to HCC, and it had a "limited ability" to discriminate between HCC and ASL.

According to Kipen, the Mastrangelo study provided further support for the premise that an increase in the quantity of VCM exposure, particularly when combined with high alcohol consumption, resulted in a very significant increase in HCC cases. Importantly, the authors adjusted for alcohol consumption and hepatitis, and found that "independent of that adjustment, vinyl chloride exposure increased the risk for [HCC]." Finally, Kipen relied on a case report published in 1976 by J.M. Gokel and others in which a worker in a PVC facility was diagnosed with HCC. However, Kipen admitted he had not reviewed scientific textbooks in forming his opinion, which did not list VCM exposure as a factor in developing HCC. Kipen

also acknowledged that none of the studies had specifically concluded that a cumulative exposure of 500 ppm of VCM resulted in an increased relative risk of contracting HCC, although the studies showed a dose response trend. He further conceded that some published epidemiological studies found no association between HCC and VCM exposure.

In determining whether the observed association between exposure to VCM and development of HCC in the epidemiological studies were causal, Kipen considered whether the following factors identified by Sir Austin Bradford Hill (the Hill factors),[9] had been satisfied: (1) temporal relationship; (2) strength of association; (3) dose response relationship; (4) replication; (5) biological plausibility; (6) consideration of alternative explanations; (7) specificity; and (8) consistency with other relevant knowledge. He concluded that they were.

Kipen found that in each of the epidemiological studies there was a temporal relationship between VCM exposure and HCC, that is, the timing of the exposure to VCM and the onset of HCC was consistent with the lengthy latency period for development of the disease. Next, the strength of association, whether the association is statistically significant, was established in the

[9] A.B. Hill, The Environment and Disease: Association or Causation, 58 Proceedings of the Royal Society of Medicine 295, 299 (1965).

Ward and Mastrangelo studies where the authors found that the relative risk of developing HCC was "quite strong." Dose response was established in the Ward and Mastrangelo studies, which demonstrated that an increase in exposure to VCM yielded an increase in risk of developing HCC. Concerning replication, Kipen believed it was satisfied not only by the Ward and Mastrangelo studies, but also by the Taiwanese studies.

He also determined that there is a biologically plausible mechanism by which VCM could cause HCC. Kipen explained that it is widely recognized that exposure to VCM causes damage to the liver, including non-malignant liver fibrosis and liver cancer. When VCM is metabolized in the liver, either in the hepatocyte cells or in the sinusoidal or endothelial cells, the metabolites can directly damage the cell's deoxyribonucleic acid (DNA),[10] or create an "oxidized environment" that results in cell damage. This damage forms "the first step in the process of malignant transformation of a cell." If the sinusoidal cells become malignant, it results in ASL, and if the hepatocellular cells become malignant, it results in HCC.

[10] "DNA" is a "molecule of genetic materials shaped like a double-helix or spiral ladder." State v. Kemp, 195 N.J. 136, 143 n.9 (2008) (quoting State v. Harvey, 151 N.J. 117, 156 (1997), cert. denied, 528 U.S. 1085, 120 S. Ct. 811, 145 L. Ed. 2d 683 (2000)).

In considering alternative explanations, Kipen explained that scientists generally do not "exclude alternatives," but rather, consider whether the association could be more accurately accounted for by other factors. According to Kipen, the other risk factors, liver cirrhosis and hepatitis, interacted with VCM exposure to significantly increase the risk of developing HCC.

Further, the "specificity" factor involved a consideration of whether VCM is associated with HCC, and the studies showed that there was an association between VCM exposure and liver damage. Finally, as to the last factor, the consistency of the relationship, Kipen found it was established because the results of the multiple scientific studies did not otherwise contradict established "laws of biology or physics or medicine."

In determining specific causation, Kipen initially reviewed Lewis' medical records to establish the diagnosis of HCC. In making that determination, he applied the criteria developed by the British Society of Gastroenterology and used by the authors of the Mastrangelo study, which are an alpha-fetoprotein level above 400 micrograms per liter and the existence of a liver mass. Kipen found that Lewis met both criteria in that his alpha-fetoprotein level was 495 mcg, and he had a tumor in his liver measuring 15 centimeters. Moreover, although Kipen did

not review the pathology slides, he reviewed the pathology report, which provided that the tests results "favor [HCC]." According to Kipen, the pathology report "trump[ed]" all of the other medical records, and thereby confirmed the diagnosis of primary HCC.

Kipen noted that Lewis did not have any of the other risk factors of developing HCC, including infection with the hepatitis B or hepatitis C virus, or alcohol abuse. Kipen conceded that it had been suggested, but not widely accepted, that obesity is another risk factor in developing HCC, and Lewis' weight fluctuated between obese and overweight. Kipen also admitted that non-alcoholic fatty liver disease (NAFL) can develop as a result of obesity, and that it is associated with HCC. However, he opined that Lewis did not have NAFL.

In determining Lewis' cumulative exposure to VCM, Kipen reviewed Lewis' work history, and the experts' reports. According to Kipen, the fact that Lewis had worked for twenty-seven years at the PVC plant, thirteen of which were before 1974, placed him in the high risk group for liver cancer. Lewis' work duties included unloading the rail tank cars and working in the reactor and warehouse buildings, which presented opportunities "for quite high exposure." He had not calculated Lewis' specific numerical cumulative dose exposure.

Nonetheless, Kipen said he generally makes determinations as to specific causation in the absence of quantitative data. It is against this factual backdrop that we consider the issues raised on the appeal and cross-appeals.

III.

On appeal, plaintiff argues that the trial court erred in precluding Drs. Infante and Kipen from testifying as to general causation because the court: 1) improperly excluded from evidence the IARC's 1987 Monograph (supplement 7),[11] the Lancet article,[12] the IARC's 2007 Monograph volume 97,[13] and Harrison's Principles of Internal Medicine (16th ed. 2005) (Harrison's); and 2) improperly applied the standard for admissibility of scientific evidence by conducting its own analysis of the epidemiological studies as reported in the scientific literature. Plaintiff also contends that the court erred in

[11] IARC Monographs on the Evaluation of Carcinogenic Risks to Humans; Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42 (supplement 7) (1987).

[12] An article by Yann Grosse, et. al., Carcinogenicity of 1,3-Butadiene, Ethylene Oxide, Vinyl Chloride, Vinyl Fluoride, and Vinyl Bromide, 8 Lancet 679-80 (2007), reporting on the IARC's reassessment that there was "sufficient evidence" that exposure to VCM caused HCC, which was to be published in IARC Monograph 97.

[13] 97 IARC Monographs on the Evaluation Of Carcinogenic Risks To Humans: 1,3-Butadiene, Ethylene Oxide and Vinyl Halides (Vinyl Floride, Vinyl Chloride and Vinyl Bromide) 311 (2008).

precluding Kipen and Infante from testifying as to specific causation. We agree.

A. Exclusion of Evidence.

In forming his opinion on causation, Infante set forth in his report, and testified during the N.J.R.E. 104 hearing, that he had relied on the IARC's 1987 Monograph (supplement 7), which had been published at the time of the hearing, in which VCM was classified as a group 1 carcinogenic. In its supplement the IARC stated that the evidence for carcinogenicity to humans was "sufficient," a "large number of epidemiological studies and case reports [had] substantiated the causal association" between vinyl chloride and ASL of the liver, and "several studies [had] also confirm[ed] that exposure to vinyl chloride causes other forms of cancer, i.e., hepatocellular carcinoma." It referenced several of the epidemiological studies relied on by Infante, including those by Weber, Hehir, and Maltoni.

Defendants objected, arguing that supplement 7 had only reported "what some other studies [had found]." The court sustained the objection, finding supplement 7 was "conclusionary," failing to "set out the rationale, methodology, or the means by which" the IARC reached its conclusion.

Generally, "[p]roofs offered at a N.J.R.E. 104(a) hearing need not comply with the other rules of evidence, except that

N.J.R.E. 403 may be invoked and valid claims of privilege will be recognized." Biunno, Weissbard & Zegas, Current N.J. Rules of Evidence, comment 4 on N.J.R.E. 104 (2011). Courts are granted broad discretion in determining the relevance of evidence and whether its probative value is substantially outweighed by its prejudicial nature. Verdicchio v. Ricca, 179 N.J. 1, 34 (2004).

The IARC, through its monographs, seeks, with the help of international working groups of experts, to identify causes of human cancer. In evaluating agents, the IARC reviews exposure data, epidemiological studies, cancer bioassays in experimental animals, and other data. See Allen v. Pa. Eng'g Corp., 102 F.3d 194, 198 (5th Cir. 1996) (stating that "[r]egulatory and advisory bodies such as IARC . . . utilize a 'weight of the evidence' method to assess the carcinogenicity of various substances in human beings and suggest or make prophylactic rules governing human exposure"). Thus, the IARC's classifications are the end products of detailed investigations.

The purpose of supplement 7 was to summarize and update the data on carcinogenicity in humans and in certain animals for agents that had previously been evaluated. The IARC Monographs are used by national and international agencies, including OSHA, and are probative evidence of causation. See Calumet Indus.,

Inc. v. Brock, 807 F.2d 225, 226 (D.C. Cir. 1986) (characterizing the IARC's Monographs as a definitive source in determining whether certain chemicals are carcinogenic); Taylor v. Airco, Inc., 494 F. Supp. 2d 21, 24 (D. Mass. 2007) (noting the IARC found sufficient evidence of carcinogenecity in humans to classify VCM as group 1 carcinogen), aff'd sub nom. Taylor v. Am. Chemistry Council, 576 F.3d 16 (1st Cir. 2009).

Supplement 7 was relevant to a determination of general causation and was the type of scientific data relied on by experts in the field of study. It should have been admitted by the trial court as an opinion contained in a learned treatise upon which Infante relied. N.J.R.E. 803(c)(18); see Jacober v. St. Peter's Med. Ctr., 128 N.J. 475, 493-97 (1992). Supplement 7 was not "conclusory" because the extensive methodology by which the IARC evaluated and categorized agents is set forth in its Preamble.[14] And, although the 1987 Monograph summarized prior findings, the IARC supported its conclusion by referencing epidemiological and laboratory animal studies, many of which had been testified to by Infante. Further, the IARC Monographs, including its supplements, are used by national and

[14] The Preamble can be located on the IARC's website. See World Health Organization, International Agency for Research on Cancer, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Preamble (2006), available at http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf.

international agencies, including OSHA, in determining what information industries must include on their labels and on the MSDS.

Infante testified that in forming his opinion on general causation he had relied upon the IARC's 2007 reassessment of the carcinogenicity of VCM, which was to be published as volume 97 of the IARC Monographs. Defendants objected, arguing that Infante had not cited to that Monograph in his report. Infante explained that although Monograph volume 97 had not yet been published, a summary of the IARC's reassessment had been published in the Lancet, a leading peer-reviewed medical journal. The Lancet article, published in August 2007, set forth that in June 2007, twenty-five scientists from eight countries met at the IARC to reassess the carcinogenicity of VCM and other chemicals. The article summarized the IARC's findings, in pertinent part, stating that the results of various studies (Ward, Mundt, Piratstu, and Mastrangelo) provide "sufficient evidence" in humans that VCM causes ASL and HCC, "leading to the overall classification of vinyl chloride as 'carcinogenic to humans' (Group 1)." Nonetheless, the court sustained defendants' objection, and excluded references to the IARC's reassessment and the Lancet article.

In denying plaintiff's first motion for reconsideration, the trial court said it excluded the IARC's 2007 reassessment because it "had not been prepared and authenticated in an official monograph." The court excluded the Lancet article because, although the article referred to various studies, the published assessment did not reference methodology or scientific data, and did not "establish the proposition, other than in conclusory terms, that the risk for cancer increases substantially with cumulative exposure, a cumulative exposure which is not defined in the assessment to cause [HCC]."

After the N.J.R.E. 104 hearing, in mid-February 2009, the IARC released its reassessment. In preparing that evaluation, an IARC working group reviewed case reports of HCC in VCM-exposed workers, epidemiological studies, including the Mundt, Piratstu, Mastrangelo, Weber, O. Wong, Boffetta, Ward, and Du studies, and laboratory animal studies, including the Feron and Maltoni experiments. In its study, the IARC stated that based on the working group's analysis of the Ward and Piratstu studies, "[t]ogether with the observation that vinyl chloride increases the risk for liver cirrhosis, which is a known risk factor for [HCC], these findings provide convincing evidence that vinyl chloride causes [HCC] as well as [ASL]." The IARC indicated that "[t]here was suggestive evidence that the risk

for [HCC] from vinyl chloride is substantially higher among workers who are infected with hepatitis virus or report high levels of alcoholic beverage consumption." Accordingly, the IARC concluded that "[t]here is sufficient evidence in humans for the carcinogenicity of vinyl chloride. Vinyl chloride causes [ASL] of the liver and [HCC]." It characterized VCM as a Group 1 carcinogen to humans.

In denying plaintiff's second motion for reconsideration, the trial court found: 1) it was unclear whether plaintiff could have discovered the 2007 Monograph prior to the February 2009, decision; 2) the Monograph was almost identical to the Lancet article, which had also been "rejected"; and 3) in any event, plaintiff failed to adduce sufficient evidence on specific causation.

Parties are obligated under Rule 4:17-4(a) and (e) to furnish expert reports requested through interrogatories on a continuing basis. Gaido v. Weiser, 227 N.J. Super. 175, 192 (App. Div. 1988), aff'd, 115 N.J. 310 (1989). "The purpose of [an expert's] report is to forewarn the propounding party of the expected contents of the expert's testimony in order to enable preparation to counter such opinions with other opinion material." Maurio v. Mereck Constr. Co., 162 N.J. Super. 566, 569 (App. Div. 1978). An expert's report must include a

complete statement of the expert's opinion and the facts considered in making that opinion. R. 4:17-4(e).

Determining whether to preclude an expert from testifying to opinions not contained in his or her report or in other discovery materials is within the discretion of the trial court. Mauro v. Owens-Corning Fiberglass Corp., 225 N.J. Super. 196, 206 (App. Div. 1988), aff'd sub nom. Mauro v. Raymark Indus., Inc., 116 N.J. 126 (1989). Factors that would support a trial court's decision to suspend the imposition of sanctions are "the absence of a design to mislead," "the absence of the element of surprise," and the "absence of prejudice." Wymbs v. Twp. of Wayne, 163 N.J. 523, 544 (2000). Furthermore, when the challenged testimony is "pivotal" to the party offering the testimony, "a court should seek to avoid exclusion where possible." Ibid. An appellate court, in reviewing a trial court's ruling concerning the admissibility of evidence, is limited to examining the decision for abuse of discretion. Hisenaj v. Kuehner, 194 N.J. 6, 16 (2008).

Because the Lancet article and the IARC Monograph can be considered the substance of Infante's facts and opinions, plaintiff should have provided an updated report. See R. 4:17-4(e). The report should have referred to the IARC's reassessment, which had, at the time of the N.J.R.E. 104

hearing, not yet been published, but had been summarized in the Lancet article. However, because we conclude that defendants were not misled, surprised or prejudiced by Infante's testimony about the IARC's reassessment, we determine that the court erred in excluding that evidence.

Defendants had been forewarned that Infante would testify about the IARC's evaluation of the carcinogenicity of VCM, because he had referenced the IARC's 1987 Monograph in his report. The reassessment was substantially similar to the 1987 Monograph, in that VCM was categorized as a group 1 carcinogenic, and the IARC cited evidence that VCM exposure was associated with ASL and HCC. The reassessment, as summarized in the Lancet article, referred to many of the same studies referenced by Infante. Thus, defendants were not deprived of the opportunity to test the accuracy of the opinions in supplement 7. Additionally, there was no design to mislead because Infante referred to the Lancet article, which was published after he testified at depositions, not as a new source of information, but in response to defendants' objection to his testimony about the yet unpublished Monograph.

The fact that the IARC's reassessment had been summarized in the Lancet, a scientific peer-reviewed journal, should have allayed any concerns the court had as to the reliability of

Infante's testimony about the as-yet unpublished Monograph. See Hisenaj, supra, 194 N.J. at 22 ("Publication itself, although not necessarily dispositive of general acceptance in the scientific community, does provide additional evidence of acceptance."). Moreover, the evidence was the type of data reasonably relied on by experts, and it supported the scientific reliability of Infante's and Kipen's opinions. Thus, the evidence should have been admitted as an opinion contained in a peer-reviewed learned treatise upon which Infante relied. N.J.R.E. 803(c)(18).

Finally, to the extent that the trial court excluded the Lancet article on the basis that it was "conclusory," we disagree. As previously stated, in summarizing the IARC's reassessment, the Lancet article referred to epidemiological studies, many of which had been extensively discussed by Infante, and the IARC's methodology in arriving at its conclusions was fully explained in its Preamble.

Turning to the exclusion of Harrison's, Kipen testified that Harrison's was "the best standard textbook of internal medicine." Harrison's states that "HCC may occur with long-term . . . exposure to . . . vinyl chloride." Defendants objected to Kipen referencing Harrison's because Kipen had failed to refer to the textbook in his report. Plaintiff countered that Kipen

referred to Harrison's during depositions in another VCM case against the same defendants, involving the same attorneys.[15] Kipen had been deposed in the Burlington County case prior to his deposition in this case, and the parties agreed that Kipen's testimony in that case would constitute his testimony on general causation in this case. Nevertheless, the court sustained the objection, because Harrison's was not referenced "in the report."

In denying plaintiff's first motion for reconsideration, the court found it "noteworthy" that Kipen had failed to include Harrison's in his report, even though he referenced other textbooks. Additionally, the court stated that "Harrison's standing alone . . . substantively and otherwise, does not, in this court's opinion, represent a consensus" of a relationship between VCM and HCC.

We conclude that the trial court erred in excluding Harrison's from evidence during the hearing. Although Kipen should have included a reference to Harrison's in his report, R. 4:17-4(e), defendants were not misled, surprised or prejudiced by his testimony during the N.J.R.E. 104 hearing. Kipen testified about the text during depositions in the Burlington

[15] Holmes v. Airco, Superior Court of New Jersey, Law Division, Burlington County, Docket No. L-1307-04.

County case, testimony the parties agreed could be used in this case. We also determine that the court erred in barring Kipen from referencing Harrison's based on the court's conclusion that Harrison's "standing alone and by itself . . . does not represent a consensus" of a causal relationship between VCM and HCC. Harrison's was not offered to stand by itself but rather as one of several authorities relied upon by Kipen in formulating his opinion on causation. See Tyndall v. Zaboski, 306 N.J. Super. 423, 427-29 (App. Div. 1997) (providing that a statement from a learned treatise established as reliable by expert testimony is admissible for substantive purposes if relied upon by the expert in direct examination), certif. denied, 153 N.J. 404 (1998).

In sum, we conclude that the trial court erroneously prohibited plaintiff's experts from referencing the IARC's 1987 Monograph, the IARC's 2007 Monograph, which has now been officially published in volume 97, the Lancet article, and Harrison's.

B. Admissibility of Infante's and Kipen's Testimony.

Plaintiff argues that the court applied the wrong standard for the admissibility of scientific evidence by conducting "its own analysis of the articles, independently decided whether they have scientific value, and reached scientific conclusions

diametrically opposed to those appearing in the published literature." We agree.

At the conclusion of the hearing, the trial court found that the epidemiological studies relied on by Infante did not support his opinion. The court concluded that Infante had "extrapolat[ed] from those studies to come to a conclusion that the authors do not reach," namely, that exposure to VCM causes HCC. Additionally, the court found that Infante's general causation opinion was "flawed by his so-called short burst theory," as set forth in the Hehir study, wherein laboratory animals that were exposed to short "bursts" of VCM developed tumors. The court determined that conclusions in that study were not "meaningful" to this case because the animals developed lung tumors, not HCC, and because there was no indication that Lewis had been exposed to similar bursts of VCM. Thus, the court ruled that Infante's opinions were not supported by epidemiological or animal studies and lacked sufficient reliability. The court concluded that Infante's opinions were "his own . . . [for] which there is no supporting authentication or justification, no methodology of reliability, [and] no reliability in terms of extrapolation from all of those studies." The court determined Infante's conclusions were "reached from dissimilar information [-] there is a

misclassification in terms of his epidemiological analysis of his sources and in terms of his extrapolation [which] render[s] his general theory of causation inadmissible."

Similarly, the court found that Kipen's reliance on the Mastrangelo study rendered his opinion on general causation unreliable. In so doing, the court noted that although the study found a "synergistic effect" between cirrhosis, hepatitis, and VCM, "[i]t did not, and it could not find that there was an independent risk factor" between VCM exposure and HCC.

Where there is a claim of a defective product under a theory of strict liability, a plaintiff must prove: 1) the product was defective; 2) "the defect existed when the product left the defendant's control"; and 3) "the defect caused injury to a reasonably foreseeable user." Coffman v. Keene Corp., 133 N.J. 581, 593 (1993). In a failure to warn action, the defect "is the absence of a warning to unsuspecting users that the product can potentially cause injury." Id. at 593-94.

General and specific causation are fundamental elements of the claim. Id. at 594. In a toxic-tort products liability action, a plaintiff must prove both product defect and medical causation. James v. Bessemer Processing Co., 155 N.J. 279, 299 (1998). "[A] plaintiff in an occupational-exposure, toxic-tort case may demonstrate medical causation by establishing: (1)

factual proof of the plaintiff's frequent, regular and proximate exposure to a defendant's products; and (2) medical and/or scientific proof of a nexus between the exposure and the plaintiff's condition." Id. at 304. The exposure must be a "substantial factor in causing or exacerbating" the complained of disease. Id. at 299.

N.J.R.E. 702 provides that "[i]f scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education may testify thereto in the form of an opinion or otherwise." To comply with N.J.R.E. 702, three requirements must be established:

> (1) the intended testimony must concern a subject matter that is beyond the ken of the average juror; (2) the field testified to must be at a state of the art such that an expert's testimony could be sufficiently reliable; and (3) the witness must have sufficient expertise to offer the intended testimony.
>
> [State v. Kelly, 97 N.J. 178, 208 (1984).]

The proponent of the expert's opinion bears the burden of proving that the testimony satisfies those threshold requirements. Hisenaj, supra, 194 N.J. at 15. The admissibility of expert testimony is a matter within the sound discretion of the trial court, and on appeal, "an appellate

court is limited to examining the decision for abuse of discretion." Id. at 12. The second Kelly requirement, that is, whether Infante's and Kipen's causation testimony was sufficiently reliable is at issue here.

Generally, a proponent of scientific evidence must demonstrate that the opinions are "generally accepted, within the relevant scientific community" ("the Frye[16] standard"). State v. Chun, 194 N.J. 54, 91, cert. denied, ___ U.S. ___, 129 S. Ct. 158, 172 L. Ed. 2d 41 (2008); Harvey, supra, 151 N.J. at 169-70. Proving general acceptance "entails the strict application of the scientific method, which requires an extraordinarily high level of proof based on prolonged, controlled, consistent, and validated experience." Rubanick v. Witco Chem. Corp., 125 N.J. 421, 436 (1991). Scientific literature can support general acceptance of a test if "the existing literature reveals a consensus of acceptance regarding a technology." Harvey, supra, 151 N.J. at 174.

However, our Supreme Court relaxed the standard for admissibility of novel scientific evidence relating to causation in toxic-tort litigation. Rubanick, supra, 125 N.J. at 449. Under the relaxed standard, applicable here, "a scientific theory of causation that has not yet reached general acceptance

[16] Frye v. United States, 293 F. 1013, 1014 (D.C. Cir. 1923).

may be found to be sufficiently reliable if it is based on a sound, adequately-founded scientific methodology involving data and information of the type reasonably relied on by experts in the scientific field." Ibid. "[I]t is not essential that there be general agreement with the opinions drawn from the methodology used. There must merely be some expert consensus that the methodology and the underlying data are generally followed by experts in the field." Id. at 450. Thus, "Rubanick changed the focus of the inquiry from the scientific community's acceptance of the substance of the opinion to its acceptance of the methodology and reasoning underlying it." Clark v. Safety-Kleen Corp., 179 N.J. 318, 337 (2004).

In determining whether causation testimony is sufficiently reliable, the Court has cautioned that "[g]reat difficulties can arise when judges, assuming the role of scientist, attempt to assess the validity of a complex scientific methodology." Rubanick, supra, 125 N.J. at 451. For example, in Rubanick, the trial court "independently reviewed" each of the studies on which the expert relied, and decided that they "do not say what plaintiff's expert concludes." Ibid. The Court found that "[i]n engaging in such an analysis, the [trial] court substituted its own assessment of the studies for that of an acknowledged expert." Ibid. The court should not "directly and

independently determine as a matter of law that a controversial and complex scientific methodology is sound." Ibid. Simply stated:

> [T]he inquiry is not the reliability of the expert's ultimate opinion nor is it whether the expert thought his or her own reliance on the underlying data was reasonable, nor whether the court thinks that the expert's reliance was reasonable. The proper inquiry is whether comparable "experts in the field [would] actually rely" on that information.
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> [Id. at 452 (internal citations omitted).]

We conclude that the trial court failed to heed the Court's instruction in Rubanick.

The court improperly conducted its own independent review of the epidemiological studies, instead of determining whether the studies were generally relied on by experts in their fields and focusing on the methodology used by Infante and Kipen in reaching their conclusions. See Landrigan v. Celotex Corp., 127 N.J. 404, 417 (1992) (indicating that a trial court should review epidemiological studies to determine if they are of a kind on which such experts ordinarily rely). Notably, the trial court found that the Mastrangelo study "cannot stand for, nor can it be extrapolated to the conclusion that . . . vinyl chloride can serve as an independent risk factor for the development of [HCC]." That finding directly contradicts the conclusion of the scientists in the study, who found that "VCM

exposure appears to be an independent risk factor for HCC and [liver cirrhosis] interacting synergistically with alcohol consumption and additively with viral hepatitis infection."

Defendants' argument that admissions by Infante and Kipen that workers in the Mastrangelo study were exposed to much greater concentrations of VCM, and as a result, suffered from greater incidences of hepatitis and liver cirrhosis, go to the weight, not the admissibility of their opinions. See Hisenaj, supra, 194 N.J. at 21 (acknowledging that identical data in scientific testing is not a prerequisite to admission of expert testimony).

Further, the methodology used by Infante, namely extrapolating from existing data contained in epidemiological studies by separating those cases of HCC from the cohort and determining the SMR, is an accepted methodology. See Gen. Elec. Co. v. Joiner, 522 U.S. 136, 146, 118 S. Ct. 512, 519, 139 L. Ed. 2d 508, 519 (1997) (noting that "[t]rained experts commonly extrapolate from existing data"); Hisenaj, supra, 194 N.J. at 17, 25 (finding expert's testimony extrapolated from seventeen engineering studies was properly admitted). And, the data utilized by Infante, that is, the findings in the several cohort studies regarding workers exposed to VCM who suffered from liver cancer including HCC, is the type of data reasonably relied on

by experts. Challenges to the fact that Infante, in extrapolating from the results of the O. Wong study, "assumed" that some non-ASL cases were HCC, similarly goes to the weight of his opinion, rather than the admissibility of it.

Defendants correctly point out that experts cannot rely on data extrapolated from articles to support conclusions not drawn by the authors. However, that is not what Infante and Kipen did. Although both Infante and Kipen admitted that the epidemiological studies do not conclusively establish a causal relationship between HCC and VCM exposure, the studies point to an association, a biological plausibility, and a dose response between VCM and HCC. Thus, the data supported their opinions.

Moreover, in evaluating the epidemiological studies, Kipen correctly applied the Bradford Hill factors to determine whether the association between VCM and HCC, as reported in the studies, was causal. Magistrini v. One Hour Martinizing Dry Cleaning, 180 F. Supp. 2d 584, 592-93 (D.N.J. 2002), aff'd, 68 Fed. App'x. 356 (3d Cir. 2003). Because statistical associations do not necessarily imply causation, scientists widely use those factors to assess general causation from epidemiological studies. Ibid.; see also In re Viagra Prods. Liab. Litig., 658 F. Supp. 2d 950, 958 (D. Minn. 2009). Kipen found that each of the

factors had been established, and thus, the epidemiological studies supported a finding of causation.

This is a difficult case, and the trial court made great efforts to assess the complicated scientific material by conducting a lengthy N.J.R.E. 104 hearing, considering extensive oral argument, and reviewing complex epidemiological studies. Typical of many toxic tort cases, the theory that VCM exposure causes HCC is contested. However, there are several epidemiological studies which show an association between VCM exposure and HCC, and experts generally rely on these studies in assessing causation. There are other publications, notably the IARC Monographs, which were excluded from evidence that have also assessed these studies and determined that they support a finding of a causal relationship. Thus, given the exclusion of the highly relevant IARC Monographs and the court's failure to focus on the experts' methodology, as opposed to their conclusions, we reverse the trial court's ruling that Infante's and Kipen's general causation testimony is inadmissible.

IV.

In determining that Infante's opinion on specific causation lacked the requisite scientific reliability, the court found that in addition to improperly relying on the epidemiological studies, Infante had failed to review Lewis' medical records,

account for the death certificate, conduct a differential diagnosis, and consider Lewis' obesity as a factor in his development of HCC.

In a toxic-tort products liability action, a plaintiff must prove both product-defect and medical causation. James, supra, 155 N.J. at 299. Differential diagnosis is an acceptable manner to establish causation in toxic tort cases. Creanga v. Jardal, 185 N.J. 345, 356 (2005). "[A] differential diagnosis is a medical construct for determining 'which one of two or more diseases or conditions a patient is suffering from, by systematically comparing and contrasting their symptoms.'" Id. at 355 (quoting Dorland's Illustrated Medical Dictionary 377 (23d ed. 1957)). Courts use the term "in a more general sense to describe the process by which causes of the patient's condition are identified." Id. at 356.

In performing a differential diagnosis, the expert first rules in all plausible causes for the patient's condition by compiling a comprehensive list of hypotheses that might explain the clinical findings under consideration, and then rules out by process of elimination all causes that did not produce the patient's condition. Ibid. "A reliably performed differential diagnosis includes considering plausible alternative causes." Magistrini, supra, 180 F. Supp. 2d at 609. However,

> [w]hile an expert is not required to rule out all alternative possible causes of a plaintiff's disease, "where a defendant points to a plausible alternative cause and the doctor offers no reasonable explanation" for why he still concludes that the chemical was a substantial factor in bringing about the plaintiff's disease, "that doctor's methodology is unreliable."
>
> [Ibid. (quoting In Re Paoli R.R. Yard PCB Litig., 35 F.3d 717, 759 n.27 (3d Cir. 1994), cert. denied, 513 U.S. 1190, 115 S. Ct. 1253, 131 L. Ed. 2d 134 (1995)).]

Infante performed a differential diagnosis in that he accepted Groth's findings that Lewis had developed HCC, and then ruled in and then out, the two major risk factors for developing HCC--cirrhosis of the liver and hepatitis. During the hearing, Infante testified that the only possible risk factor that Lewis "may have had" was that he "may have been obese." However, Infante also testified that no studies existed to prove that obesity was a factor in developing the disease. Nevertheless, the trial court sustained defendants' objection to Infante's testimony concerning obesity because of his failure to include it in his report.

Infante did not consider obesity as an "accepted" risk factor in Lewis developing HCC. Accordingly, he did not rule the factor in or out in performing his diagnosis. We discern no prejudice to defendants by Infante explaining why he chose not to consider obesity as a risk factor.

Defendants also counter that the court properly excluded Kipen's testimony on specific causation because he failed to review the majority of Lewis' medical history and all of the pathology slides. We disagree. Kipen reviewed the pathology reports, which he stated "trump[ed]" all of the other medical records and confirmed the diagnosis of primary HCC. He also performed a differential diagnosis, ruling in and then out, cirrhosis of the liver and hepatitis as factors for Lewis developing HCC. Thus, Kipen should be allowed to render an opinion on specific causation. Additionally, like with Infante, Kipen should be permitted to testify whether Lewis' obesity is an accepted risk factor for HCC.

Accordingly, we reverse the trial court's decision precluding Infante and Kipen from testifying on specific causation.

V.

We now turn to defendants' cross-appeals. Goodrich, PPG, and Shell argue in Point III of their brief that the trial court erred in denying their motion to exclude Dr. David Groth from testifying. Defendants assert that Groth was not qualified to testify about the diagnosis of HCC or its pathology because he had not reviewed a single case of HCC in the last forty years,

or diagnosed liver cancer, or conducted any independent research on HCC or VCM exposure. We reject these contentions.

N.J.R.E. 702 requires that an expert be qualified "by knowledge, skill, experience, training, or education." The decision to allow an individual to testify as an expert rests within the sound discretion of the court. Hisenaj, supra, 194 N.J. at 12. On appeal, we will only reverse a trial court's decision to admit expert testimony upon a finding of abuse of that discretion. Ibid.

Groth is a board certified pathologist who received his medical degree from Yale University, and whose principal interest is in cancer. In addition to the general knowledge implied by his license, see Sanzari v. Rosenfeld, 34 N.J. 128, 137 (1961), Groth also acquired knowledge from more than forty years of occupational experience. Although his major interest was in the area of asbestos-related pulmonary cancer, and he had not reviewed a single case of primary liver cancer, he had extensive experience in diagnosing and researching occupational cancer, as well as in the pathology of VCM. For example, during his internship and residency, he conducted 200 to 300 autopsies, and witnessed approximately 3,000 autopsies, of which 30% involved some form of cancer. While employed at the OSHA and the NIOSH, he conducted animal experiments, which required him

to review slides of tumor cells, including HCC. As a member of a committee formed to test the toxic effect of industrial chemicals on the liver, he reviewed HCC and ASL tissue slides.

Additionally, while working as a consultant for the NIOSH, Groth reviewed medical records and histopathology slides on 700 cases of cancer. He also authored two peer-reviewed articles on HCC, and had significant experience with the pathology of VCM, in that he participated in toxicological studies while at the NIOSH, attended conferences, and reviewed literature.

We conclude that based on his training and experience with HCC and VCM, the trial court correctly determined Groth was qualified to testify as an expert in this case. What is more, the fact that Groth had not seen a slide of human HCC tissue since his residency and had not previously reviewed a single HCC case, goes to the weight to be accorded his opinion, not to his competency. See Rubanick v. Witco Chem. Corp., 242 N.J. Super. 36, 48 (App. Div. 1990), modified and remanded, 125 N.J. 421 (1991).

Defendants argue next that Groth's testimony lacked sufficient reliability, contending that Groth failed to review all the pathology slides and medical records.

An expert's opinion must be supported by facts or data either in the record or of a type usually relied on by experts

in the field, which need not be admissible. N.J.R.E. 703. An expert's bare conclusion unsupported by factual evidence is inadmissible as a net opinion. Creanga, supra, 185 N.J. at 360. However, "[t]he failure of an expert to give weight to a factor thought important by an adverse party does not reduce his testimony to an inadmissible net opinion if he otherwise offers sufficient reasons which logically support his opinion." Rosenberg v. Tavorath, 352 N.J. Super. 385, 402 (App. Div. 2002).

We conclude that Groth's opinion was amply supported by appropriate facts and data. Groth reviewed all of the clinical data sent to him by counsel, reviewed histopathology slides of Lewis' liver tumor, conducted a differential diagnosis, and determined that even excluding the slides, the clinical data alone was sufficient to support the HCC diagnosis. Significant factors to support the diagnosis that Lewis had developed HCC included the large size of the liver tumor, the presence of smaller nodules in the liver, and elevated alpha-fetoprotein levels. Groth ruled out lung cancer as a primary site based on the presence of small bilateral nodules in Lewis' lungs, and a "clear" 1998 chest x-ray. Biochemical tests ruled out other cancers.

Moreover, Groth reviewed the hospital pathology report in which the carcinoembryonic antigen and feritin levels revealed canalicular staining patterns, which indicated that the tumor cells originated in the liver. Other medical records also supported Groth's opinion. One report set forth that the tumor had a trabecular pattern of growth, indicating HCC. A pathology report set forth that the results of a fine needle aspiration were "suggestive of hepatocellular carcinoma," and another report revealed that the tests results "favor hepatocellular carcinoma." Additionally, the hospital discharge summary listed the final diagnosis as "[h]epatocellular carcinoma with metastasis."

Groth did not review all of Lewis' slides, request copies of depositions, or rule out all other possible forms of cancer. For example, Groth admitted that he initially only reviewed ten out of fifty available slides, and had never reviewed the actual slide showing the canalicular staining patterns. However, he maintained that many of the slides simply showed smears of blood, and that he had all of the clinical data he needed to render a diagnosis. Because Groth offered other sufficient reasons in support of his opinion, the fact that he did not review all of the material deemed significant by defendants goes to the weight, not the admissibility, of his testimony. See

Rosenberg, supra, 352 N.J. Super. at 402 (instructing that omission of a factor by an expert, who otherwise supports his opinion, that the opposing party deems relevant is more properly accounted for on cross-examination at trial). Accordingly, the trial court did not err in denying the motion to exclude Groth's testimony.

VI.

Goodrich, PPG, and Shell argue in Point IV of their brief that the trial court erroneously denied their motion seeking to exclude James Jones from testifying as to the state-of-the-art knowledge in the industry regarding the toxicity of VCM and the adequacy of the warnings. Defendants contend that Jones' opinions on the state-of-the-art knowledge in the industry and adequacy of the warnings should have been excluded because they were not based on a proper methodology. Defendants assert that Jones was not qualified to render an opinion about the "timing or type" of the warnings that should have been provided because he was not a warnings expert or toxicologist. They also argue that Jones failed to analyze what the industry knew prior to January 1974. We disagree.

Jones, a certified industrial hygienist, received a Bachelor of Science Degree in chemical engineering and completed some graduate work. As an undergraduate, he worked at

Goodrich's Louisville plant, and later, at the NIOSH, where he directed the exposure assessment portion of their VCM study. He visited six or seven PVC production facilities to gather information on exposures to VCM for the NIOSH. In 1974, he assisted in preparing the OSHA exposure limits for VCM. He co-authored peer-reviewed articles, three of which addressed studies on VCM, and taught courses at the University of Cincinnati on industrial hygiene. As an industrial hygienist, he identified harmful workplace exposures to chemicals and other agents, determined safe exposure levels with epidemiologists and physicians, and assisted employers in reducing risks of exposures. Jones also testified to the adequacy of warnings in several other VCM cases, including rendering an opinion on the state-of-the-art knowledge in the industry.

Jones opined that the warnings given to workers prior to 1974 about the risk of developing liver damage from exposure to VCM were inadequate. According to Jones, defendants were aware of laboratory animal studies linking VCM exposure to liver cancer, of reports that workers in foreign PVC plants developed liver damage, and of recommendations in the scientific literature that VCM exposure levels should be lowered to 50 ppm. Jones concluded that "in light of these multiple indications that VCM was harmful to workers at exposure levels as low as 50

ppm and the finding of cancer in VCM exposed animals, users of VCM should have begun monitoring and controlling exposures to VCM, and suppliers of VCM and the MCA should have lowered their exposure recommendations at least as early as 1961 and given adequate warning of the various health effects found in humans and animals." Jones did not render an opinion on the design of the warning, but rather testified to what information should have been included in the warning.

We conclude that the court did not abuse its discretion in finding Jones qualified by his knowledge, skill, experience, training, and education to testify as to what information should have been provided to workers to protect them from workplace hazards. Jones was amply qualified to testify to the dangers of VCM exposure, to the toxicity of VCM as contained in toxicology reports, and how employers could protect workers from such exposure. See Beadling v. William Bowman Assocs., 355 N.J. Super. 70, 80 (App. Div. 2002) (discussing certified industrial hygienist's report and opinion in products liability case in which he reviewed the defendant's warning label for compliance with federal and industry labeling requirements); Kapsis v. Port Auth. of N.Y. & N.J., 313 N.J. Super. 395, 401-02 (App. Div.) (summarizing industrial hygienist's testimony that the plaintiff should have been warned about dangers of asbestos in the

workplace), certif. denied, 157 N.J. 544 (1998). Although Jones was not qualified to testify to the specific format or wording of the warnings, he did not.

Defendants further contend that Jones' opinions on the state-of-the-art knowledge in the industry and adequacy of the warnings should have been excluded because they were not based on a proper methodology. They assert that Jones failed to analyze what the industry had known prior to January 1974.

In a toxic tort failure-to-warn case, the plaintiff must establish that the defendant had a duty to warn. James, supra, 155 N.J. at 297-98. "To establish such a duty, the plaintiff must satisfy 'a very low threshold of proof in order to impute to a manufacturer sufficient knowledge to trigger the duty to provide a warning of the harmful effects of its product.'" Ibid. (quoting Coffman, supra, 133 N.J. at 599). In strict liability cases, "knowledge of the harmful effects of a product will be imputed to a manufacturer on a showing that 'knowledge of the defect existed within the relevant industry.'" Id. at 298 (quoting Coffman, supra, 133 N.J. at 599). "The plaintiff need not prove that the defendant manufacturer was cognizant of a defect, but rather that knowledge of the defect existed within the relevant industry." Coffman, supra, 133 N.J. at 599. If proceeding under a theory of negligence, "the plaintiff must

demonstrate that the specific defendant knew or should have known of the potential hazards of the product." James, supra, 155 N.J. at 298.

"Once proof of such knowledge in the industry has been established, triggering the duty to warn, the plaintiff must show that an adequate warning was not provided." Ibid. The manufacturer's conduct generally "should be measured by knowledge at the time the manufacturer distributed the product." Feldman v. Lederle Labs., 97 N.J. 429, 452 (1984). "The duty to warn exists not only to protect and alert product users but to encourage manufacturers and industries, which benefit from placing products into the stream of commerce, to remain apprised of the hazards posed by a product." Coffman, supra, 133 N.J. at 599. The manufacturer "should keep abreast of scientific advances," and is held to the standard of an expert. Feldman, supra, 97 N.J. at 452-53.

Contrary to defendants' assertion, Jones extensively discussed in his report and testimony the numerous studies conducted prior to 1974 that showed VCM toxicity in laboratory animals and to workers in the VCM industry. He was also aware of the Lester study that concluded that VCM was not harmful. Jones testified that the NIOSH recommended providing workers with information regarding the results of animal toxicity

studies. He also cited recommendations by Torkelson and the ACGIH that the industry lower threshold ceiling exposure limits for VCM. Thus, Jones considered the state of the knowledge of the industry prior to 1974.

Lastly, defendants contend that Pantasote was a typical manufacturer in the industry, and it did not consider VCM to be toxic to humans prior to 1974. Thus, defendants assert that the state of the knowledge in the industry did not require a stronger warning. In support of this argument, defendants cite testimony by Jacob Jaglom, Pantasote's former president, who stated that Pantasote had "all" the same knowledge about the potential health hazards of VCM as the rest of the industry, and Pantasote did not interpret the then state-of-the-art knowledge to require revised warnings prior to 1974. However, Jaglom also testified that before the 1970's he did not know that VCM exposure caused liver damage in animals or workers, or that some scientists had recommended lowering the TLV for VCM to 50 ppm, or that other companies had been monitoring their workers for liver damage. Thus, there was evidence that Pantasote did not have the same knowledge as defendants regarding the toxicity of VCM, and therefore, the company did not possess representative knowledge of the industry. Accordingly, the court did not err in denying the motion to exclude Jones from testifying as to the

state-of-the-art knowledge in the industry regarding the toxicity of VCM and the adequacy of the warnings.

VII.

Goodrich, PPG, and Shell argue that the trial court erred in denying their motion for summary judgment on the inadequate warnings claim. Defendants contend plaintiff not only failed to present any evidence that they had a duty to warn, but also failed to present evidence of a "feasible alternative warning." Defendants also assert that plaintiff failed to present expert evidence showing that defendants should have known prior to 1974 that vinyl chloride was a potential human carcinogen. Hexion joins in the argument.

A court will grant summary judgment "if the pleadings, depositions, answers to interrogatories and admissions on file, together with the affidavits, if any, show that there is no genuine issue as to any material fact challenged and that the moving party is entitled to a judgment or order as a matter of law." R. 4:46-2(c); Brill v. Guardian Life Ins. Co. of Am., 142 N.J. 520, 540 (1995). "An issue of fact is genuine only if, considering the burden of persuasion at trial, the evidence submitted by the parties on the motion, together with all legitimate inferences therefrom favoring the non-moving party, would require submission of the issue to the trier of fact." R.

4:46-2(c). On appeal, we apply the same standard. Atl. Mut. Ins. Co. v. Hillside Bottling Co., 387 N.J. Super. 224, 230 (App. Div.), certif. denied, 189 N.J. 104 (2006).

We have considered defendants' arguments in light of the record and applicable law. We affirm substantially for the reasons expressed by the trial court in its oral decision of October 25, 2007. Nevertheless, we add the following comments.

Defendants first contend that plaintiff failed to present evidence that they, as opposed to Pantasote, had a duty to warn Lewis of the dangers of exposure to VCM.

The manufacturer of a product has a duty to warn about any risk relating to the product that it knows or ought to know "on the basis of reasonably obtainable or available knowledge." Feldman, supra, 97 N.J. at 434. "In the employment context, a manufacturer's duty to warn of the dangers posed by its products extends to both the employer and the employees of the recipient entity." James, supra, 155 N.J. at 298.

Plaintiff presented evidence that Goodrich, PPG, and Shell sold VCM to Pantasote. Jones opined that the "suppliers of VCM and the MCA," which included all defendants--even Hexion, "should have lowered their exposure recommendations at least as early as 1961 and given adequate warning of the various health effects found in humans and animals." Thus, plaintiff presented

sufficient evidence for purposes of summary judgment that defendants had a duty to warn.

Next, Goodrich, PPG, and Shell assert that plaintiff failed to present evidence of a "feasible alternative warning that would have prevented Lewis' alleged occupational exposure." We determine under the facts that plaintiff was not required to present evidence of a feasible adequate warning.

Once a plaintiff establishes a duty to warn, he or she must then establish that an adequate warning was not provided. Ibid. To reinforce the duty to provide necessary warnings, New Jersey applies a rebuttable "heeding presumption" to ease the plaintiff's burden in establishing that the lack of a warning was a proximate cause of the injury. Id. at 297. The presumption is that the plaintiff would have followed an adequate warning had one been provided. Coffman, supra, 133 N.J. at 591. In the employment context, the presumption is not only that the employee would have heeded the warning, but also that the employer would have heeded the warning and communicated it to the employees, thereby enabling the employees to take precautions. Theer v. Philip Carey Co., 113 N.J. 610, 621 (1993).

From 1962 to 1974, Goodrich, PPG, and Shell provided no warnings to either Pantasote or Lewis, even though knowledge

existed of the toxic effects of VCM in the industry. In that regard, Jones opined that defendants should have "given adequate warning of the various health effects found in humans and animals." He testified that the warning should have included information that VCM exposure causes liver damage and should have suggested that employers lower exposure standards and monitor VCM levels. Therefore, for summary judgment purposes, the record contained sufficient evidence that those defendants had failed to provide an adequate warning to withstand the motion, and the presumption is that plaintiff would have followed an adequate warning had one been provided. Coffman, supra, 133 N.J. at 591.

Finally, Goodrich, PPG, and Shell argue the court erred in finding that a genuine issue of fact existed as to whether they "acted like reasonable experts in the field" in investigating and warning of the potential health hazards of VCM. They claim that prior to 1974, there was no evidence that exposure to VCM was a "potential human carcinogen." Not so.

"The question in strict liability design-defect and warning cases is whether, assuming that the manufacturer knew of the defect in the product, [it] acted in a reasonably prudent manner in marketing the product or in providing the warnings given." Feldman, supra, 97 N.J. at 451. "Generally, the state of the

art in design defect cases and available knowledge in defect warning situations are relevant factors in measuring reasonableness of conduct." Ibid.

The record contained ample evidence showing that prior to 1974 knowledge of the toxicity of VCM existed within the industry. As early as 1959, there were indications of VCM toxicity in animals (e.g., the Rowe study), and as a result, at least one member of the industry noted that VCM "is more toxic than [was previously] believed." The Mastromatteo, Torkelson and Viola animal experiments conducted in 1960, 1968, and 1970 also showed liver damage to animals. As noted by the trial court, correspondence existed among industry members discussing the animal studies and the toxicity of VCM to humans. Indeed, in 1963, Goodrich began testing its workers for liver damage, indicating it had some knowledge of the danger of VCM exposure.

Because a "duty to warn is triggered by early warning flags of danger from a product, so that people are not needlessly exposed to the possible dangers of a product during the time that extensive testing is being done," Magistrini v. One Hour Martinizing Dry Cleaning, 109 F. Supp. 2d 306, 313-14 (D.N.J. 2000), aff'd 68 Fed. App'x. 356 (3d Cir. 2003), we agree with the trial court that a material question of fact existed as to

whether defendants had knowledge prior to 1974 of the link between VCM exposure and cancer.

VIII.

Hexion argues that the court erred in denying defendants' motion for summary judgment on plaintiff's civil conspiracy claim because once plaintiff's fraud claim was dismissed, plaintiff failed to assert another underlying intentional tort in support of the conspiracy claim. We agree.

In denying defendants' motion, the trial court accepted the proposition that a conspiracy charge founded solely on negligence will not suffice. Although the court found no New Jersey cases addressing the issue, it was persuaded by the rationale of several out-of-state cases that held "a strict liability claim can serve as an underlying tort and co-conspirators can be liable in the products liability arena for the intentional acts of a defendant manufacturer." Adopting the rationale underlying the out-of-state decisions, the court found that plaintiff had produced sufficient facts "to establish a conspiracy claim with respect to the secrecy pact in 1972 through 1974." But the court made "no ruling" as to "any claim of a conspiracy among the defendants with regard to an effort to withhold information relative to the adverse effects of VCM at any point prior to 1972 and that issue is deferred."

Under New Jersey law, "a civil conspiracy is a combination of two or more persons acting in concert to commit an unlawful act, or to commit a lawful act by unlawful means, the principal element of which is an agreement between the parties to inflict a wrong against or injury upon another, and an overt act that results in damage." Banco Popular N. Am. v. Gandi, 184 N.J. 161, 177 (2005) (internal quotation omitted); see also Morgan v. Union County Bd. of Chosen Freeholders, 268 N.J. Super. 337, 364 (App. Div. 1993), certif. denied, 135 N.J. 468 (1994). A party is liable if he or she understands the general objectives of the conspiracy, accepts them, and makes an implicit or explicit agreement to further those objectives. Banco Popular, supra, 184 N.J. at 177. "Most importantly, the gist of the claim is not the unlawful agreement, but the underlying wrong which, absent the conspiracy, would give a right of action." Id. at 177-78 (internal quotation omitted).

We could not find any New Jersey State court opinions addressing the issue of whether the tort underlying a civil conspiracy claim must be intentional. Nonetheless, we note that the reported cases sustaining civil conspiracy claims are based on underlying intentional torts. See ibid. (holding that an allegation of a conspirator agreeing to perform a fraudulent transfer of assets in violation of the Uniform Fraudulent

Transfer Act, N.J.S.A. 25:2-20 to -34, was sufficient to state a conspiracy claim); Bd. of Educ. of Asbury Park v. Hoek, 38 N.J. 213, 238-39 (1962) (upholding a verdict based on a civil conspiracy where there was evidence showing that the conspirators attempted to deceive the plaintiff to purposely avoid a public bidding statute); Middlesex Concrete Prods. & Excavating Corp. v. Carteret Indus. Ass'n, 37 N.J. 507, 516-17 (1962) (holding that a complaint alleging a civil conspiracy based on the conspirators malicious interference with plaintiff's contractual rights and business or economic relations with a municipality was sufficient to state a cause of action); State, Dep't of Treasury, Div. of Inv. ex rel. McCormac v. Qwest Commc'ns Int'l Inc., 387 N.J. Super. 469, 485-86 (App. Div. 2006) (reversing trial court's order dismissing complaint on civil conspiracy claim where plaintiff pled common law fraud as an underlying wrong); Morgan, supra, 268 N.J. Super. at 367 (reversing a trial court's dismissal of a civil conspiracy claim to deny a plaintiff employment because of his political affiliations because the record contained evidence of the defendants' intentional harassment to force the plaintiff into an involuntary resignation).

Although, no New Jersey State court has specifically addressed this issue, a majority of other courts that have

addressed it held that the underlying tort in a civil conspiracy claim must be intentional. See, e.g., Brown v. Philip Morris Inc., 228 F. Supp. 2d 506, 517 n.10 (D.N.J. 2002) (noting that absent the plaintiff's fraud claim or other intentional tort, her conspiracy claim fails for lack of an underlying tort); United States v. Mitlof, 165 F. Supp. 2d 558, 564 (S.D.N.Y. 2001) (disapproving principle that one can conspire to act unintentionally as logical impossibility), aff'd sub nom. United States v. Sheehan, 89 Fed. App'x. 307 (2d Cir. 2004); Sackman v. Liggett Group, Inc., 965 F. Supp. 391, 395-96 (E.D.N.Y. 1997) (acknowledging that civil conspiracy requires an underlying intentional tort but declining to grant summary judgment because of the possibility that strict product liability may be a sufficient basis for civil a conspiracy claim); Sonnenreich v. Philip Morris Inc., 929 F. Supp. 416, 419-20 (S.D. Fla. 1996) (noting conspiracy to commit negligence is illogical, and conspiracy must be based on intentional tort); Goldstein v. Philip Morris, Inc., 854 A.2d 585, 590 (Pa. Super. Ct. 2004) (affirming dismissal of civil conspiracy claim because complaint only alleged strict liability and negligence); Rosen v. Brown & Williamson Tobacco Corp., 782 N.Y.S.2d 795, 795 (N.Y. App. Div. 2004) (granting summary judgment because civil conspiracy requires showing of intentional conduct).

However, as cited by the trial court, other out-of-state cases provide support for the proposition that a plaintiff may assert a conspiracy claim solely upon a products liability cause of action. See Sackman, supra, 965 F. Supp. at 396; Wright v. Brooke Group Ltd., 652 N.W.2d 159, 174 (Iowa 2002) (permitting claim of civil conspiracy on wrongful conduct that did not constitute an intentional tort); see also In re Methyl Tertiary Butyl Ether Prods. Liab. Litig., 175 F. Supp. 2d 593, 633 (S.D.N.Y. 2001) (permitting a civil conspiracy claim in a strict liability design defect action based on allegations that the defendants had marketed the alleged defective product intentionally).

Here, the court found that plaintiff's strict liability claim could provide the underlying tort for her civil conspiracy claim. A strict liability claim does not, however, involve an element of intent. Myrlak v. Port Auth. of N.Y. & N.J., 157 N.J. 84, 97 (1999) (stating that fault is not a necessary element in a strict liability action); see also O'Brien v. Muskin Corp., 94 N.J. 169, 180 (1983) ("Under strict liability, a manufacturer that produces defective products is liable even if those products are carefully produced.)."

Although our Supreme Court has not specifically addressed whether the tort underlying a civil conspiracy claim can be

anything other than an intentional tort, we determine the rationale adopted by the majority of courts that have addressed the issue is more persuasive, and that the Court would follow the majority and require the underlying tort in a civil conspiracy claim to be intentional. Accordingly, we conclude that the trial court erred in holding plaintiff could assert her conspiracy claim based upon a products liability cause of action. Therefore, we reverse that part of the November 8, 2007 order that denied defendants' motions for summary judgment seeking to dismiss plaintiff's civil conspiracy claims. In so doing, we need not address Hexion's alternative argument that it did not conspire with remaining defendants.

IX.

In sum, we reverse those parts of the December 15, 2008 order excluding Infante and Kipen from testifying; affirm those parts of the December 15, 2008 order denying defendants' motions seeking to exclude Groth and Jones from testifying; reverse that part of the February 5, 2009 order granting defendants' motions for summary judgment dismissing the complaint; affirm that part of the November 8, 2007 order denying defendants' motions for summary judgment on the adequacy of the warning claim; and reverse that part of the November 8, 2007 order denying

defendants' motions for summary judgment on the civil conspiracy claim.

Affirmed in part; and reversed in part.

APPENDIX

ACRONYM - DEFINITION

ACGIH - American Conference of Government Industrial Hygienists

ASL - Angiosarcoma

CT - Computerized Tomography

HCC - Hepatocellular Carcinoma

IARC - International Agency for Research on Cancer

MAC - Maximum Allowable Concentration

MCA - Manufacturing Chemists Association

MSDS - Material Safety Data Sheet

NAFL - Non-Alcoholic Fatty Liver Disease

NIOSH - National Institute for Occupational Safety and Health

OSHA - Occupational Safety and Health Administration

ppm - Parts Per Million

PVC - Polyvinyl Chloride

SD-56 - Chemical Safety Data Sheet SD-56

SMR - Standardized Mortality Ratio

TLV - Threshold Limit Values

USEPA - United States Environmental Protection Agency

VCM - Vinyl Chloride Monomer