IN THE SUPREME COURT OF APPEALS OF WEST VIRGINIA

September 2013 Term

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SUPREME COURT OF APPEALS
OF WEST VIRGINIA

No. 12-1135

DEBORAH KAY HARRIS, ADMINISTRATRIX OF THE ESTATE OF RONALD K. HARRIS, DECEASED,
Plaintiff Below, Petitioner

V.

CSX TRANSPORTATION, INC.,
Defendant Below, Respondent

Appeal from the Circuit Court of Marshall County
Honorable David W. Hummel, Judge
Civil Action No. 08-C-171M(H)

REVERSED AND REMANDED

Submitted: October 15, 2013
Filed: November 13, 2013

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JUSTICE DAVIS delivered the Opinion of the Court.

JUSTICE LOUGHRY dissents and reserves the right to file a dissenting opinion.

SYLLABUS BY THE COURT

1. When a circuit court excludes expert testimony as unreliable under the *Daubert v. Merrell Dow Pharmaceuticals, Inc*., 509 U.S. 579, 113 S. Ct. 2786, 125 L. Ed. 2d 469 (1993), and *Wilt v. Buracker*, 191 W. Va. 39, 443 S.E.2d 196 (1993), gatekeeper analysis, we will review the circuit court's method of conducting the analysis *de novo*.

2. When a trial court is called upon to determine the admissibility of scientific expert testimony, in deciding the "reliability" prong of admissibility the focus of the trial court's inquiry is limited to determining whether the expert employed a methodology that is recognized in the scientific community for rendering an opinion on the subject under consideration. If the methodology is recognized in the scientific community, the court should then determine whether the expert correctly applied the methodology to render his or her opinion. If these two factors are satisfied, and the testimony has been found to be relevant, and the expert is qualified, the expert may testify at trial.

Davis, Justice:

Deborah Kay Harris, administratrix of the Estate of Ronald K. Harris ("Petitioner"), appeals an order of the Circuit Court of Marshall County granting summary judgment in favor of CSX Transportation, Inc. ("CSX"). The circuit court granted summary judgment after ruling that Petitioner was precluded from calling her three expert witnesses at trial. The dispositive issue presented by the Petitioner in this appeal is whether the circuit court committed error in finding the scientific testimony of Petitioner's three expert witnesses was not reliable.[1] After a careful review of the briefs, the record submitted on appeal and listening to the arguments of the parties, we reverse and remand this case.

I.

FACTUAL AND PROCEDURAL HISTORY

This action was originally filed by Ronald K. Harris under the Federal Employers' Liability Act[2] and the Locomotive Inspection Act[3] against his employer, CSX.[4] The complaint alleged that Mr. Harris' exposure to diesel exhaust fumes while employed by

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[1]The Petitioner set out three issues as assignments of error. However, we only need to address the issue of the reliability of the testimony of Petitioner's experts to resolve this case.

[2]*See* 45 U.S.C. §51 *et seq*. (1939).

[3]*See* 49 U.S.C. § 0701 *et seq*. (1994).

[4]The record submitted on appeal did not include the complaint.

CSX caused him to develop a type of cancer called multiple myeloma. While the case was pending, Mr. Harris died as a result of the cancer. Petitioner, Mr. Harris' wife and administratrix of his estate, was substituted as the plaintiff. Petitioner amended the complaint to allege that Mr. Harris' death resulted from his exposure to diesel exhaust fumes.[5]

When the parties concluded expert witness discovery, CSX filed a motion to exclude the testimony of Petitioner's three expert witnesses because their methodology was not reliable. At the request of CSX, the trial court held an evidentiary hearing regarding the admissibility of Petitioner's expert witnesses' testimony. The evidentiary hearing lasted two days. During the hearing, Petitioner called her three experts, Dr. Peter Infante, Ph.D.; Dr. Lawrence Goldstein, Ph.D.; and Dr. Brian Durie, M.D. CSX called two expert witnesses: Dr. Peter Shields, M.D. and Dr. Laura Green, Ph.D. These evidentiary hearings in West Virginia are commonly referred to as "*Daubert*/*Wilt*" hearings.

At the conclusion of the two-day evidentiary hearing, the circuit court entered three orders excluding Petitioner's experts' testimony. The circuit court entered findings of fact which, in essence, determined that Petitioner failed to prove to the court that diesel exhaust exposure causes multiple myeloma. As a result of not having an expert, Petitioner

[5]The amended complaint was not made part of the record on appeal.

agreed with CSX to jointly move for summary judgment in CSX's favor so that Petitioner could appeal the adverse expert witness rulings. The circuit court entered an order granting summary judgment. This appeal followed.

II.

STANDARD OF REVIEW

In this proceeding, the circuit court granted summary judgment in favor of CSX after excluding the testimony of Petitioner's expert witnesses. We stated in Syllabus point 1 of *Painter v. Peavy*, 192 W. Va. 189, 451 S.E.2d 755 (1994), that "[a] circuit court's entry of summary judgment is reviewed *de novo*." The parties agree. Without expert testimony by the Petitioner, summary judgment is appropriate. Consequently, the dispositive ruling in this case is not the summary judgment order. It is the orders precluding Petitioner's three experts from testifying. If those orders fail, summary judgment is not appropriate.

As a general matter, we have long held that "[t]he admissibility of testimony by an expert witness is a matter within the sound discretion of the trial court, and the trial court's decision will not be reversed unless it is clearly wrong." Syl. pt. 6, *Helmick v. Potomac Edison Co.*, 185 W. Va. 269, 406 S.E.2d 700 (1991). However, we have indicated, and so hold, that "when a circuit court excludes expert testimony as unreliable under the [*Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 113 S. Ct. 2786, 125

L. Ed. 2d 469 (1993); and *Wilt v. Buracker*, 191 W. Va. 39, 443 S.E.2d 196 (1993),] gatekeeper analysis, we will review the circuit court's method of conducting the analysis *de novo*." *San Francisco v. Wendy's Int'l, Inc*., 221 W. Va. 734, 740, 656 S.E.2d 485, 491 (2007) (citations omitted).

With these standards in mind, we turn to the issues presented by this appeal.

III.

DISCUSSION

In order to adequately address the dispositive issue in this case and give guidance to trial judges in future cases similar to the instant matter, we have outlined our discussion as follows: (1) general principles of Rule 702; (2) the nature of multiple myeloma; (3) epidemiological methodology; (4) toxicological methodology; (5) weight of the evidence methodology; (6) Bradford Hill methodology; (7) qualification, methodology and opinion of the expert witnesses; and (8) the circuit court's orders excluding the testimony of Petitioner's experts.

A. General Principles of Rule 702

Rule 702 of the West Virginia Rules of Evidence provides in full 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." "Rule 702 reflects an attempt to liberalize the rules governing the admissibility of expert testimony." *Weisgram v. Marley Co.*, 169 F. 3d 514, 523 (8th Cir. 1999). What this means is that "[t]he rule 'is one of admissibility rather than exclusion.'" *In re Flood Litig. Coal River Watershed*, 222 W. Va. 574, 581, 668 S.E.2d 203, 210 (2008) (quoting *Arcoren v. United States*, 929 F.2d 1235, 1239 (8th Cir. 1991). "Disputes as to the strength of an expert's credentials, mere differences in the methodology, or lack of textual authority for the opinion go to weight and not to the admissibility of their [sic] testimony." *Gentry v. Mangum*, 195 W. Va. 512, 527, 466 S.E.2d 171, 186 (1995) (citation omitted).

The decisions of this Court have "explained that circuit courts must conduct a two-part inquiry under Rule 702 and ask: (1) is the witness [qualified as] an expert; and, if so, (2) is the expert's testimony relevant and reliable?" *San Francisco v. Wendy's Int'l, Inc.*, 221 W. Va. at 741, 656 S.E.2d at 492 (citations omitted). *See also* Robin Jean Davis, *Admitting Expert Testimony in Federal Courts and Its Impact on West Virginia Jurisprudence*, 104 W. Va. L. Rev. 485, 513 (2002) ("Trial courts are required to assess scientific expert testimony for relevancy and reliability."). In Syllabus point 5 of *Gentry* we set out the steps that a trial court should take to determine if an expert is qualified to render an opinion under Rule 702:

> In determining who is an expert, a circuit court should conduct a two step inquiry. First, a circuit court must determine whether the proposed expert (a) meets the minimal educational or experiential qualifications (b) in a field that is relevant to the subject under investigation (c) which will assist the trier of fact. Second, a circuit court must determine that the expert's area of expertise covers the particular opinion as to which the expert seeks to testify.

195 W. Va. 512, 466 S.E.2d 171.

The general standard for determining whether an expert's scientific opinion is relevant and reliable was set out in Syllabus point 2 of *Wilt v. Buracker*, 191 W. Va. 39, 443 S.E.2d 196:

> In analyzing the admissibility of expert testimony under Rule 702 of the West Virginia Rules of Evidence, the trial court's initial inquiry must consider whether the testimony is based on an assertion or inference derived from the scientific methodology. Moreover, the testimony must be relevant to a fact at issue. Further assessment should then be made in regard to the expert testimony's reliability by considering its underlying scientific methodology and reasoning. This includes an assessment of (a) whether the scientific theory and its conclusion can be and have been tested; (b) whether the scientific theory has been subjected to peer review and publication; (c) whether the scientific theory's actual or potential rate of error is known; and (d) whether the scientific theory is generally accepted within the scientific community.

As is illustrated later in this opinion, the trial court's decision to exclude Petitioner's three experts resulted from its determination that the scientific opinions of all

three of Petitioner's experts were not reliable. The circuit court's ruling shows a misunderstanding of the meaning of "reliable" under West Virginia jurisprudence. We previously have noted the contours of the meaning of "reliable'" as follows:

> The assessment of whether scientifically-based expert testimony is "reliable," as that term is used in [*Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 113 S. Ct. 2786, 125 L. Ed. 2d 469 (1993), and *Wilt v. Buracker*, 191 W. Va. 39, 443 S.E.2d 196 (1993)], does not mean an assessment of whether the testimony is persuasive, convincing, or well-founded. Rather, assessing "reliability" is a shorthand term of art for assessing whether the testimony is to a reasonable degree based on the use of knowledge and procedures that have been arrived at using the methods of science—rather than being based on irrational and intuitive feelings, guesses, or speculation. If the former is the case, then the jury may (or *may* not, in its sole discretion) "rely upon" the testimony.

In re Flood Litig., 222 W. Va. at 582 n.5, 668 S.E.2d at 211 n.5.

In *Gentry*, Justice Cleckley made the following relevant observation:

> Under *Daubert/Wilt*, the circuit court conducts an inquiry into the validity of the underlying science, looking at the soundness of the principles or theories and the reliability of the process or method as applied in the case. *The problem is not to decide whether the proffered evidence is right, but whether the science is valid enough to be reliable*.

Gentry, 195 W. Va. at 523, 466 S.E.2d at 182 (emphasis in original). It is noteworthy that Justice Cleckley felt it was important to italicize the quoted second sentence: "*The problem is not to decide whether the proffered evidence is right, but whether the science is valid enough to be reliable*." *Id.* As will be shown later in this opinion, the circuit court

misapplied this Court's opinion in *Gentry*. That is, the circuit court decided the opinions from Petitioner's three experts were wrong. Under *Gentry*, right or wrong is not an issue of the admissibility of scientific evidence. The circuit court made right or wrong a central test for the admission of scientific evidence. In doing so, the circuit court removed from the jury its exclusive role of deciding which expert opinion to believe.

Rule 702 and the decisions of this Court clearly state that it is of no moment that the opinions of the parties' experts reach different conclusions on all dispositive issues. This is to be expected. Our legal system is adversarial, not cordial. As a result of the adversarial essence of our legal system, we rely upon the jury to make the ultimate determination as to which expert is right and which expert is wrong. To place the decision in the hands of trial judges denies litigants their constitutional right to a jury trial.

The decision in *State ex rel. Wiseman v. Henning*, 212 W. Va. 128, 569 S.E.2d 204 (2002), illustrates this Court's hostility to stripping litigants of the right to have a jury decide if an expert is right or wrong. The plaintiff in *Wiseman* was injured in an automobile accident and later developed multiple myeloma. The plaintiff filed a negligence action against the truck driver and truck owner, alleging that his multiple myeloma resulted from

a rib cage injury he suffered in the traffic collision with the truck driver.[6] The circuit court granted defendants' motion in limine to exclude testimony of the plaintiff's expert witness on causation. The plaintiff filed a petition for a writ of prohibition with this Court seeking to prevent enforcement of the trial court's order. This Court granted the writ after concluding that plaintiff's expert's proffered opinion was sufficiently reliable to be admissible. The opinion in *Wiseman* addressed the issue as follows:

> Examining the record in the instant case, we believe that the circuit court exceeded its authority in its decision to exclude the testimony of Dr. Hussein. The record reflects that Dr. Hussein was a member of several specialized cancer research societies, and had substantial interaction with other cancer specialists. He was a specialist in cancers such as that suffered by Mr. Wiseman, and was director of the Myeloma Program at the Cleveland Clinic. Dr. Hussein's proffered opinion that multiple myeloma can result from a trauma was based upon: his extensive treatment of Mr. Wiseman; his treatment of five other patients at the Cleveland Clinic who had trauma-induced myelomas; his study of the physiological process of tissue injury causing chronic inflammation and overstimulation of cells, which triggers the growth of cancerous cells; his interaction with other specialists who also believe that trauma can trigger the occurrence of myeloma; and the handful of published studies by other cancer centers that have identified local tissue injury, including a bone fracture, as a risk factor for causing multiple myeloma.
>
> We recognize that Dr. Hussein's opinion is novel and unorthodox, and may not have yet received, as the circuit court found, "general acceptance in the scientific community." However, the *Rules of Evidence* do not require that a scientific opinion be "generally accepted," because such a requirement is

[6]A loss of consortium claim was also brought by the plaintiff's wife.

> at odds with the liberal thrust of the . . . Rules and their general approach of relaxing the traditional barriers to opinion testimony. The record suggests a substantial degree of reliability underlying the formation of Dr. Hussein's opinion. Accordingly, we find that the circuit court erred in excluding his testimony. . . . The proffered opinion is valid enough to be reliable; *whether the proffered evidence is right is a question for the finder of fact*.

Wiseman, 212 W. Va. at 133-34, 569 S.E.2d at 209-10 (internal quotations and citations omitted; emphasis added).[7] *See also Ruiz-Troche v. Pepsi Cola of Puerto Rico Bottling Co.*, 161 F.3d 77, 85 (1st Cir. 1998) ("*Daubert* does not require that a party who proffers expert testimony carry the burden of proving to the judge that the expert's assessment of the situation is correct.").

B. The Nature of Multiple Myeloma

In the case *sub judice*, Mr. Harris was diagnosed with having multiple myeloma. He died from this disease. This disease has been described as follows:

> "[M]ultiple myeloma is a cancer of the plasma cell, a cell which arises in bone marrow and is an important part of the immune system as it provides antibodies which help fight infection and other diseases. If a plasma cell becomes malignant, it is called a myeloma cell. An individual with myeloma has an abnormal build-up of myeloma cells in the bone marrow with displacement of normal marrow and which results in tumors that involve and destroy surrounding bone.

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[7]This opinion will conclude the discussion of the legal principles of admissibility of expert testimony in the context of the circuit court's ruling in Section III G, *infra*.

World City Found., Inc. v. Sacchetti, No. 114829/03, 2008 WL 344131, at*4 (N.Y. Sup. Ct. Jan. 28, 2008). Specifically, "'[m]ultiple myeloma' refers to the presence of numerous myelomas in various bones of the body." *Hawaii Med. Serv. Ass'n v. Adams*, 209 P.3d 1260, 1263 n.4 (Haw. Ct. App. 2009). During the underlying proceedings herein, multiple Myeloma was described by the expert for CSX, Dr. Shields, as follows:

> So multiple myeloma is a cancer of one of the types of blood cells. It's actually in plasma cells, which is a type of B cell, and the plasma cells are responsible for making antibodies to fight infection. And what happens is that if you get a myeloma, all the plasma cells or one type of plasma cell, actually started growing uncontrollably and pushing everything out . . .
>
> . . . .
>
> . . . So plasma cell is one of the – is one of the blood cells that's part of the immune system that makes those antibodies fight, you know, the common cold, pneumonia, that sort of thing.
>
> And so what happens is as those cells grow, those plasma cells, and become plasmacytomas; where they like live . . . in the bones. And so that's the myeloma. So the myeloma part is the bones, and multiple is you get multiple bone lesions. And as it's – as it's living in the bones, it starts crowding up the bone marrow, and you start having other bood count effects. You have immune system problems and that sort of thing.

See also Toney v. State, 961 N.E.2d 57, 60 (Ind. Ct. App. 2012) ("[M]ultiple myeloma [is] a cancer of the plasma cells in bone marrow."); *Williams v. Superior Uniform Grp., Inc.*, 847 So. 2d 244, 246 (La. Ct. App. 2003) ("Multiple myeloma is a type of cancer that affects the bone marrow, the body's blood-forming system.").

C. Epidemiological Methodology

One of Petitioner's experts, Dr. Infante, is an epidemiologist. Epidemiology "refers to the science that studies the distribution of diseases within populations[.]" *Chesson v. Montgomery Mut. Ins. Co.*, No. 97, Sept Term, 2012, 2013 WL 5311126, at *17 (Md. Sept. 24, 2013) (internal quotations and citation omitted). Moreover,

> [e]pidemiology is a methodology. The practice of epidemiology involves sampling and matching so as to minimize systematic bias and statistical analysis designed to estimate the effect of random errors on results. Epidemiology is not a theory of how a substance causes cancer, or birth defects, or autoimmune disease. These theories come from other disciplines.

4 David L. Faigman et al., Modern Scientific Evidence: The Law and Science of Expert Testimony § 35-1.1, at 132 n.18 (2002). "[E]pidemiological studies examine existing populations to attempt to determine if there is an association between a disease or condition and a factor suspected of causing that disease or condition." *Merrell Dow Pharms. v. Havner*, 953 S.W.2d 706, 715 (Tex. 1997). The issue of an epidemiological "association" has been more fully described as follows:

> [T]he field of epidemiology is not intended to utilize the results of a group study to demonstrate causation for any individual plaintiff. Instead, the studies are performed or undertaken to first determine if a statistically significant association exists between an exposure and an outcome. If such an association is revealed and the studies are determined to be free of confounding, bias, or other error, then an association can be established. At this point, epidemiologists and others interpreting the epidemiologic data can make an inference vis-à-vis the existence of a causal relationship or the lack

thereof.

Frank C. Woodside, III and Allison G. Davis, *The Bradford Hill Criteria: The Forgotten Predicate*, 35 T. Jefferson L. Rev. 103, 108 (2013).

It should be clearly understood that the term "association" is a term of art in epidemiology. It has been defined as "[t]he degree of statistical dependence between two or more events or variables." *In re TMI Litig.*, 193 F.3d 613, 710 n.159 (3d Cir. 1999) (internal quotations and citation omitted). Moreover, an association is not the same as causation. An epidemiological association identified in a study may or may not be causal. "Although epidemiological studies cannot prove causation, they do provide a basis for an epidemiologist to infer that a chemical agent can cause a disease." Syl. pt. 7, *King v. Burlington Northern Santa Fe Ry. Co.*, 762 N.W.2d 24, 28 (Neb. 2009). "Assessing whether an association is causal requires an understanding of the strengths and weaknesses of the study's design and implementation, as well as a judgment about how the study findings fit with other scientific knowledge." Michael D. Green et al., *Reference Guide on Epidemiology, in* Reference Manual on Scientific Evidence 549, 553 (3d ed. 2011). Moreover, the methodological soundness of an epidemiological study and its use in resolving causation require answering three questions. First, does the study reveal an association between a chemical agent and disease? Second, did any errors in the study cause an inaccurate result? Third, is the relationship between the chemical agent and the disease causal? *See id*. at 554.

In determining whether an association exists between a suspected chemical agent and a disease, epidemiologist primarily rely upon three types of studies: (1) experimental studies, (2) cohort studies, and (3) case-control studies.[8] *See King*, 762 N.W.2d at 35. Finally, the strength of an association between exposure to a chemical agent and disease can be stated as a relative risk, an odds ratio, or an attributable risk. "Each of these measurements of association examines the degree to which the risk of disease increases when individuals are exposed to an agent." Green et al., *supra*, at 566. To better understand this epidemiological association, we will review these three main types of studies – experimental studies, cohort studies, and case control studies as well as studies that examine the association of relative risk, odds ratio, and attributable risk.

1. <u>Experimental studies</u>. An experimental study has been defined as "a study in which a population is selected for a planned trial of a regimen whose effects are measured by comparing the outcome of the regime in the experimental group with the outcome of another regimen in a control group." 4 Faigman et al., *supra*, at 184. This type of study goes by several names including, randomized trial, clinical trial, and true experiment. Green et al. *supra*, at 555.

In order to answer the question of whether a chemical agent is related to a

[8]There are also additional types of specialized studies.

certain disease, an epidemiologist may conduct an experimental study in which selected participants are randomly assigned to one, of two groups: a group exposed to the chemical agent and a group that was not exposed. After a predetermined observation period, the participants in both groups are evaluated for the development of the disease. An experimental study is often used to evaluate new drugs or medical treatments. Green et al., *supra*, at 555. *See also In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166 (N.D. Cal. 2007) (wherein a clinical study that revealed Celebrex increased cardiovascular risk was relied upon by the court to conclude that the plaintiff's experts' testimony on causation was admissible); *McDarby v. Merck & Co.*, 949 A.2d 223 (N.J. Super. Ct. App. Div. 2008) (explaining how clinical trials of Vioxx revealed an association with heart disease).

Of course, if a chemical agent's effects are harmful, a researcher cannot knowingly expose participants to the chemical. In situations where the chemical agent is harmful, a researcher will typically "observe" selected participants who have already been exposed to the chemical, *e.g.*, comparing those already exposed to an industrial chemical agent with another group of participants who have not been exposed. In this situation, the researcher compares the rate of disease or death of the exposed group with that of an unexposed group. Green et al., *supra*, at 555-56.

2. <u>Cohort studies</u>. A cohort study has been defined as an "analytical method of epidemiologic study in which subsets of a defined population can be identified who . . . have been . . . exposed . . . to a factor . . . hypothesized to influence the probability of occurrence of a given disease[.]" Faigman et al., *supra*, at 183. A cohort study is also called a prospective study and followup study. Green et al., *supra*, at 557.

A cohort study involves the use of a study population without regard to the disease status of the participants. A researcher may define a study population in the present and follow it into the future, or design a study population retrospectively at a point in the past and follow it over historical time toward the present. In either situation, the researcher will classify the study population into groups based on whether the group members were exposed to the chemical agent of interest. The task of a researcher in a retrospective population study is to determine the number of people in the exposed group who developed the disease of interest, from all available reliable sources, and compare that number of people with the number of people of the group who were not exposed. With respect to a prospective study, the exposed and unexposed populations are followed for a predetermined length of time, and the number of persons in each group who develop the disease of interest are compared. Green et al., *supra*, at 557. *See also* Faigman et al., *supra*, at 162-65.

A cohort study has the advantage of allowing the temporal relationship between

exposure and disease to be established more quickly than in other study design. As a result of following a study population that is not initially affected by the disease, the researcher is able to determine the exact onset time of the disease and its relation to exposure to the chemical. For a researcher, "[t]his temporal relationship is critical to the question of causation, because exposure must precede disease onset if exposure caused the disease." Green et al, *supra*, at 558.

3. <u>Case control studies</u>. A case control study involves selecting a group of individuals who have a disease of interest (cases), and choosing a similar group of persons who do not have the disease of interest (controls). When the groups are selected, the researcher will then compare them in terms of past exposures. In doing so, the researcher is seeking to determine whether a certain exposure that is associated with the disease resulted in a higher proportion of past exposure among the "cases" than among the "controls". "[C]ase-control studies are . . . particularly useful in the study of rare diseases, because if a cohort study were conducted, an extremely large group would have to be studied in order to observe the development of a sufficient number of cases for analysis." Green et al., *supra*, at 559. *See also* 4 Faigman et al., *supra*, at 166-69.

4. <u>Relative risk</u>. The strength of an association between exposure to a chemical agent and disease can be stated as a relative risk. This concept of "relative risk"

is defined as the ratio of the incidence rate of a targeted disease in an exposed population to the incidence rate in an unexposed population. Additionally, the "incidence rate of a targeted disease" is defined as the total number of cases of the disease that manifests itself during a predetermined time period divided by the number of individuals in the population being studied. In sum, the incidence rate illustrates the risk that an individual in a population group will develop the targeted disease within a predetermined time period. Green et al., *supra*, at 566-67.

For example, assume that a group composed of 100 individuals is exposed to a chemical agent, and a group composed of 200 individuals is not exposed to the chemical. After a researcher studies both groups for one year, it is learned that 40 of the individuals exposed to the chemical have the targeted disease, and 20 of the individuals who were not exposed to the chemical are also found to have the disease. The relative risk of contracting the disease would be determined as follows:

> [1] The incidence rate of disease in the exposed individuals is 40 cases per year per 100 persons (40/100), or 0.4.
>
> [2] The incidence rate of disease in the unexposed individuals is 20 cases per year per 200 persons (20/200), or 0.1.
>
> [3] The relative risk is calculated as the incidence rate in the exposed group (0.4) divided by the incidence rate in the unexposed group (0.1), or 4.0.

Green et al., *supra*, at 567. As a general matter, the relative risk is interpreted as follows:

> [1] If the relative risk equals 1.0, the risk in exposed individuals is the same as the risk in unexposed individuals. There is no association between exposure to the agent and disease.
>
> [2] If the relative risk is greater than 1.0, the risk in exposed individuals is greater than the risk in unexposed individuals. There is a positive association between exposure to the agent and the disease, which could be causal.
>
> [3] If the relative risk is less than 1.0, the risk in exposed individuals is less than the risk in unexposed individuals. There is a negative association, which could reflect a protective or curative effect of the agent on risk of disease. . . .

Green et al., *supra*, at 566-67. *See also* Daniel J. Brown, *Clear as Mud – The Role of Epidemiological Data in Assessing Admissibility under Delaware Rule of Evidence 702*, 13 Del. L. Rev. 71, 79 (2012) ("The size of the relative risk indicates the strength of that association. For example, a relative risk of 3.5 means the risk of disease in those exposed to the substance is three and half times higher than the risk of disease in those who were not exposed.").

5. <u>Odds ratio</u>. The odds ratio, like the relative risk, is used to illustrate in quantitative terms the association between exposure to a chemical agent and a disease. This tool is considered an easy way to estimate the risk in a case-control study when a rare disease is under investigation.[9] The odds ratio permits an approximation of the risk when a rare

[9]It has been noted that

(continued...)

disease is the focus of the study. The odds ratio, in a case-control study, is the ratio of the odds that a case group (one with the disease) was exposed to a chemical agent to the odds that a control group (one without the disease) was exposed to the same chemical. However, in a cohort study, the odds ratio is expressed as the ratio of the odds of developing a disease when exposed to a chemical to the odds of developing the disease when not exposed to the chemical. Green et al., *supra*, at 568.

For example, a researcher conducts a case-control study that has 100 individuals with a disease who act as the "case" group, and 100 individuals who do not have the disease act who were the "control" group. It is found that 40 of the 100 case group individuals were exposed to a chemical agent, and 60 were not. In the control group, 20 individuals were exposed to the chemical, and 80 were not. The calculation of the odds ratio would be as follows:

(40/60)

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[9](...continued)

> [a] relative risk cannot be calculated for a case-control study, because a case-control study begins by examining a group of persons who already have the disease. That aspect of the study design prevents a researcher from determining the rate at which individuals develop the disease. Without a rate or incidence of disease, a researcher cannot calculate a relative risk.

Green et al., *Reference Guide on Epidemiology*, *in* Reference Manual on Scientific Evidence 549, 568 n.58 (3d ed. 2011).

$$OR = \frac{\quad}{(20/80)} = 2.67$$

Green et al., *supra*, at 569.

> [B]ecause an odds ratio approximates the relative risk, the same general rules of interpretation apply, i.e., an odds ratio of 1.0 indicates that there is no association between exposure and disease, whereas an odds ratio above 1.0 indicates a positive association and an odds ratio below 1.0 indicates a negative association.

Brown, *supra,* 13 Del. L. Rev. at 79.

6. <u>Attributable risk</u>. Another epidemiological measurement of risk is called attributable risk. This measurement tool represents the amount of disease that individuals are exposed to that may be attributed to such exposure. Attributable risk also can be formulated as the proportion of the disease among exposed individuals that is linked to the exposure. "[T]he attributable risk reflects the maximum proportion of the disease that can be attributed to exposure to an agent and consequently the maximum proportion of disease that could be potentially prevented by blocking the effect of the exposure or by eliminating the exposure." Green et al., *supra*, at 570. Stated differently, if the epidemiological association of the disease and chemical agent is causal, "the attributable risk is the proportion of disease in an exposed population that might be caused by the agent and that might be prevented by eliminating exposure to that agent." *Id.*

The following example has been given to illustrate the determination of attributable risk:

> For example, if the incidence rate in the unexposed group is ten and the incidence rate in the exposed is fifty then the attributable risk is 80 percent (i.e., 50-10 = 40; 40/50 = 80%). This would mean that 80 percent of the disease in the exposed group is attributable to the exposure to the suspect substance. This, however, is not the same as stating that 80 percent of the disease is caused by the exposure.

Brown, *supra*, 13 Del. L. Rev. at 80.

D. Toxicological Methodology

Another of the Petitioner's experts, Dr. Goldstein, is a toxicologist. The record also shows that one of the experts called by CSX, Dr. Green, is likewise a toxicologist. "[T]he science of toxicology can help understand whether the dose of a substance achieved following a particular exposure has any relationship to toxicity or disease." David L. Eaton, *Scientific Judgment and Toxic Torts–Primer in Toxicology for Judges and Lawyers*, 12 J.L. & Pol'y 5, 12 (2003). Toxicology is a science that focuses on understanding and identifying the harmful effects of chemical agents.[10] Toxicological studies alone do not purport to provide direct evidence a disease was caused by a chemical exposure. This discipline can, however, be instrumental in offering scientific data regarding the increased risk of

[10]"The discipline of toxicology is based primarily upon the sciences of chemistry and biology." 4 Faigman et al., Modern Scientific Evidence: The Law and Science of Expert Testimony § 35-1-1, at 104 (2002).

contracting a disease based upon dosage. Bernard D. Goldstein and Mary Sue Henifin, *Reference Guide on Toxicology*, *in* Reference Manual on Scientific Evidence 633, 635-37 (3d ed. 2011). Courts have held that toxicologists can provide expert testimony on whether a chemical agent caused a disease. *See Bonner v. ISP Techs., Inc*., 259 F.3d 924, 928-31 (8th Cir. 2001); *Loudermill v. Dow Chem. Co.*, 863 F.2d 566, 569–70 (8th Cir. 1988).[11]

"[D]ata from properly designed and evaluated studies in experimental animals have been and continue to be reliable sources of information for the identification of potential human health hazards and the estimation of risks in exposed populations." Ronald L. Melnick and John R. Bucher, *Determining Disease Causality From Experimental Toxicology Studies*, 15 J.L. & Pol'y 113, 133 (2007). *See also* 4 Faigman et al., *supra*, at 109

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[11]One commentator summarized the science of toxicology as follows:

> There are three basic tenets of toxicology: (1) all chemicals have the potential to be harmful given the right dosage; (2) many chemical agents have a signature pattern of toxic effects that are used to establish causation; and (3) responses in laboratory animals are useful in determining the potential effects on humans. Toxicology generally seeks to identify chemicals that pose a threat to human populations and the risks associated with a chemical exposure at a given dose. Unlike epidemiology, which seeks primarily to establish causation, toxicology seeks primarily to estimate the given risks associated with potential exposure.

Carl H. Johnson, *When Science Is Too Daunting: Multiple Chemical Sensitivity, Federal Courts, and the Struggling Spirit of Daubert,* 1 Vill. Envtl. L.J. 273, 291-92 (2000). *See also* 4 Faigman et al., *supra* note 10, at 107.

("There is an overwhelming biological similarity between humans and other animals, particularly mammals."). The general testing procedure used by toxicologists involves exposing laboratory animals[12] or cells/tissues[13] to a chemical agent, monitoring changes, and comparing those changes with those for an unexposed control group. Of course, there is an ongoing debate as to the extent to which animal testing will validly reflect human responses to a chemical agent. This debate, however, is circular because it is unethical and potentially criminal to experiment on humans by exposing them to hazardous doses of a chemical agent. Thus, animal toxicological studies provide the best readily accessible scientific data concerning the risk of disease from a chemical exposure. Goldstein and Henifin, *supra*, at 639.[14]

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[12]This is called in vivo research.

[13]This is called in vitro research.

[14]The justification and reliability of animal studies for the potential effects of chemicals on humans has been stated as follows:

> Why are animal models used to evaluate human risk? The most obvious explanation is that it is unethical to test for adverse health effects, such as cancer, in humans through intentional exposures. Just as animal models are used in preclinical trials of new pharmaceutical agents before testing in humans, experimental studies performed on animals have been used to assess potential health risks of toxic and carcinogenic agents in our workplace and general environment. The predictive value of animal studies is based on species similarities in the biological processes of disease induction. Another major advantage of animal studies is the elimination of the need to wait for a high incidence of human cancers, which

(continued...)

A central component of a toxicological study will involve dose-response relationships. 4 Faigman, *supra*, at 107-08. That is, experiments with animals are conducted to determine the dose-response relationship of a chemical agent by measuring how the response varies with different doses. Information obtained from this technique "is useful in understanding the mechanisms of toxicity and extrapolating data from animals to humans." Goldstein and Henifin, *supra*, at 641. In making a causation opinion about a chemical and a disease, a toxicologist will consider the extent of a person's dose exposure. Goldstein and Henifin, *supra*, at 638.[15]

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[14](...continued)
may take as much as 30 years from time of first exposure to clinical manifestation of disease, before implementing public health protective strategies.

Melnick and Bucher, *Determining Disease Causality From Experimental Toxicology Studies*, 15 J.L. & Pol'y 113, at 115-16 (2007).

[15]The following is an explanation and illustration of dose:

> Dose is a function of both concentration and duration. Haber's rule is a century-old simplified expression of dose effects in which the effect of a concentration and duration of exposure is a constant (e.g., exposure to an agent at 10 parts per million for 1 hour has the same impact as exposure to 1 part per million for 10 hours). Exposure levels, which are concentrations, are often confused with dose. This can be particularly problematic when attempting to understand the implications of exposure to a level that exceeds a regulatory standard that is set for a different time frame. For example, assume a drinking water contaminant is a known cause of cancer. To avoid a 1 in 100,000 lifetime risk caused by this contaminant in drinking water, and assuming that the average person will drink

(continued...)

The approach taken by toxicologists for assessing exposure to a harmful chemical agent has been summarized as follows:

> Exposure assessment methodologies include mathematical models predicting exposure resulting from an emission source, which might be a long distance upwind; chemical or physical measurements of media such as air, food, and water; and biological monitoring within humans, including measurements of blood and urine specimens. An exposure assessment should also look for competing exposures. In this continuum of exposure metrics, the closer to the human body, the greater the overlap with toxicology.

Goldstein and Henifin, *supra*, at 657.

A toxicologist's opinion on causation should be based upon three preliminary assessments:

> First, the expert should analyze whether the disease can be related to chemical exposure by a biologically plausible theory. Second, the expert should examine whether the plaintiff was exposed to the chemical in a manner that can lead to absorption into the body. Third, the expert should offer an opinion about whether the dose to which the plaintiff was

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> [15](...continued)
> approximately 2000 mL of water daily for a lifetime, the regulatory authority sets the allowable contaminant standard in drinking water at 10 µg/L. Drinking one glass of water containing 20 µg/L of this contaminant, although exceeding the standard, does not come close to achieving a "reasonably medically probable" cause of an individual case of cancer.

Goldstein and Henifin, *Reference Guide on Toxicology, in Reference Manual on Scientific Evidence* 633, 638 n.12 (3d ed. 2011).

exposed is sufficient to cause the disease.

Goldstein and Henifin, *supra*, at 661. *See also* Eaton, *supra*, 12 J.L. & Pol'y at 38-40; Robert C. James, *Role of Toxicology in Toxic Tort Litigation: Establishing Causation*, 61 Def. Couns. J. 28, 29 (1994). Courts also have recognized a "three-step methodology for toxicologists endorsed by the World Health Organization[.]" *Young v. Burton*, 567 F. Supp. 2d 121, 129 (D.D.C. 2008). The risk assessment methodology has been described as follows:

> First, an evaluation is made of the chemicals to which the individual might have been exposed, and of the concentrations of these chemicals in air breathed by the individual. The second step involves an evaluation, based on the published scientific literature, of the exposures necessary to produce the adverse effects associated with the chemicals to which individuals may be exposed. These two evaluations are then combined in the final step of the risk assessment to provide an estimate of the likelihood that any of the harmful properties of any or all of the chemicals might have been expressed in the exposed individual.

Bombardiere v. Schlumberger Tech. Corp., 934 F. Supp. 2d 843, 848-49 (N.D. W. Va. 2013). *See also Evans v. Toyota Motor Corp.*, No. V-03-09, 2005 WL 3454456, at*4 (S.D. Tex. Aug. 9, 2005); *Roche v. Lincoln Prop. Co.*, 278 F. Supp. 2d 744, 754 (E.D. Va. 2003); *Mancuso v. Consolidated Edison Co. of New York, Inc.*, 967 F. Supp. 1437, 1445 (S.D.N.Y. 1997); *Cavallo v. Star Enter.*, 892 F. Supp. 756, 764 (E.D. Va. 1995), *aff'd, in part, and rev'd, in part*, 100 F.3d 1150 (4th Cir. 1996); Craig T. Smith, *Peering into the Microscope: The Rise of Judicial Gatekeeping after Daubert and its Effect on Federal Toxic Tort Litigation*, 13 B.U. J. Sci. & Tech. L. 218, 227 (2007); Neal C. Stout and Peter A. Valberg,

Bayes' Law, Sequential Uncertainties, and Evidence of Causation in Toxic Tort Cases, 38 U. Mich. J.L. Reform 781, 900 (2005).

E. Weight of the Evidence Methodology

One of the Petitioner's experts, toxicologist Dr. Goldstein, indicated during his testimony that he relied upon the weight of the evidence methodology in rendering his opinion.[16] "[T]he term 'weight of evidence' is used to characterize a process or method in which all scientific evidence that is relevant to the status of a causal hypothesis is taken into account." Sheldon Krimsky, *The Weight of Scientific Evidence in Policy and Law*, 95 Am. J. Pub. Health S129 (2005). Under this approach, an "expert considers all available studies and determines the weight to be afforded to each on the basis of the strengths and weaknesses of the individual studies." Thomas O. McGarity and Sidney A. Shapiro, *Regulatory Science in Rulemaking and Tort: Unifying the Weight of the Evidence Approach*, 3 Wake Forest J.L. & Pol'y 65, 78 (2013).

The phrase "weight of the evidence" is often accorded different meanings by scientists. As explained by one court:

> The weight of evidence method [WOE] is used in

[16]It was previously indicated that CSX's expert, Dr. Green, is a toxicologist. However, her testimony was far too acrimonious and rambling to clearly understand what precise methodology she used.

> medical literature either in a rigorous scientific or metaphorical sense. It is used as methodology where WOE points to established interpretative methodologies (*e.g.*, systematic narrative review, meta-analysis, causal criteria, and/or quality criteria for toxicological studies). . . . The metaphorical use of the term is, if nothing else, a colorful way to say the body of evidence we have examined and judged using a method we have not described but could be more or less inferred from a careful between-the-lines reading of our paper.

Reeps ex rel. Reeps v. BMW of N. Am., LLC, No. 100725/08, 2013 WL 2362566, at *3 (N.Y. Sup. Ct., May 10, 2013) (internal quotations and citations omitted)).[17] The weight of the evidence methodology is used by regulatory agencies such as the Environmental

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[17]The different ways in which weight of the evidence may be used by scientists have been summarized as follows:

> WOE has several distinct uses in contemporary scientific practice. First, it most often appears in a metaphorical sense, pointing to a body of scientific evidence without reference to any specific methodology. . . . Second, in some situations, a WOE approach specifically refers to a technique in which "all available evidence" should be examined and interpreted. . . . Third, often a WOE method refers directly to some other synthetic method, such as the systematic narrative review, meta-analysis, or the so-called "causal criteria" associated most often with the public health discipline of epidemiology. Fourth, a WOE method may point to an institutional approach to synthesis. . . . Finally, in relatively rare instances of health-risk assessment, a WOE approach involves a method that assigns numerical weights to individual scientific studies and creates summary numeric assessments using mathematical algorithms.

Douglas L. Weed, *Evidence Synthesis and General Causation: Key Methods and an Assessment of Reliability*, 54 Drake L. Rev. 639, 639 (2006).

Protection Agency[18] and the Occupational Safety and Health Administration. "Regulatory agencies or risk analysis panels use [the weight of evidence method] to assess the total value of the scientific evidence that a substance may be dangerous to human health." Krimsky, *supra*, at S139. *See also King*, 762 N.W.2d at 39-40 ("[G]overnment agencies and some experts use a weight-of-the-evidence methodology. That methodology comprehensively analyzes the data from different scientific fields, primarily animal tests and epidemiological studies, to assess carcinogenic risks."). In commenting upon the weight of the evidence methodology, Justice Stevens noted the following:

> [T]he Court of Appeals expressly decided that a "weight of the

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[18]In the EPA's 2005 "Guidelines for Carcinogen Risk Assessment" it described the type of data that would be considered in its weight of the evidence methodology:

> **1.3.3. Weight of Evidence Narrative**
>
> The cancer guidelines emphasize the importance of weighing all of the evidence in reaching conclusions about the human carcinogenic potential of agents. This is accomplished in a single integrative step after assessing all of the individual lines of evidence. . . . Evidence considered includes tumor findings, or lack thereof, in humans and laboratory animals; an agent's chemical and physical properties; its structure-activity relationships (SARs) as compared with other carcinogenic agents; and studies addressing potential carcinogenic processes and mode(s) of action, either in vivo or in vitro. Data from epidemiologic studies are generally preferred for characterizing human cancer hazard and risk.

U.S. Environmental Protection Agency, Guidelines for Carcinogen Risk Assessment 1-11 (2005), http://www.epa.gov/ttn/atw/cancer_guidelines_final_3-25-05.pdf (last visited on Nov. 8, 2013).

> evidence" methodology was scientifically acceptable. To this extent, the Court of Appeals' opinion is persuasive. It is not intrinsically "unscientific" for experienced professionals to arrive at a conclusion by weighing all available scientific evidence—this is not the sort of "junk science" with which *Daubert* was concerned.

General Elec. Co. v. Joiner, 522 U.S. 136, 153, 118 S. Ct. 512, 522-23, 139 L. Ed. 2d 508 (1997) (Stevens, J., concurring, in part, and dissenting, in part).

The court in *Milward v. Acuity Specialty Products Group, Inc.*, 639 F.3d 11 (1st Cir. 2011), explained the weight of the evidence methodology as follows:

> This "weight of the evidence" approach to making causal determinations involves a mode of logical reasoning often described as "inference to the best explanation," in which the conclusion is not guaranteed by the premises. . . . [I]nference to the best explanation can be thought of as involving six general steps, some of which may be implicit. The scientist must (1) identify an association between an exposure and a disease, (2) consider a range of plausible explanations for the association, (3) rank the rival explanations according to their plausibility, (4) seek additional evidence to separate the more plausible from the less plausible explanations, (5) consider all of the relevant available evidence, and (6) integrate the evidence using professional judgment to come to a conclusion about the best explanation.
>
> . . . .
>
> The fact that the role of judgment in the weight of the evidence approach is more readily apparent than it is in other methodologies does not mean that the approach is any less scientific. No matter what methodology is used, an evaluation of data and scientific evidence to determine whether an inference of causation is appropriate requires judgment and

> interpretation. The use of judgment in the weight of the evidence methodology is similar to that in differential diagnosis, which we have repeatedly found to be a reliable method of medical diagnosis.

Milward, 639 F.3d at 17-18 (internal quotations and citations omitted). *See also* Thomas O. McGarity and Sidney A. Shapiro, *Regulatory Science in Rulemaking and Tort: Unifying the Weight of the Evidence Approach*, 3 Wake Forest J.L. & Pol'y 65, 97 (2013) ("Both common law courts and regulatory agencies should consider expert opinion based on weight of the evidence evaluations of the available scientific information in accordance with valid scientific criteria, such as the Bradford Hill criteria, for evaluating evidence."); Kimberly Gordy, *The 9/11 Cancer Conundrum: The Law, Policy, & Politics of the Zadroga Act*, 37 Seton Hall Legis. J. 33, 83 (2012) ("The *Milward* court . . . provides useful guidance for weighing evidence. . . . It endorsed the 'weight of the evidence' approach, which encompasses the Bradford Hill methodology.").

F. Bradford Hill Methodology

Petitioner's experts, Dr. Durie and Dr. Infante, relied upon the Bradford Hill methodology in rendering their opinions. The record also showed that the expert for CSX, Dr. Shield, relied upon the Bradford Hill Methodology. This methodology involves the use of criteria set out by epidemiologist Sir Austin Bradford Hill in an article he published in 1965. *See* Sir Austin Bradford Hill, *The Environment and Disease: Association or Causation?*, 58 Proc. Royal Soc'y Med. 295 (1965). The Bradford Hill criteria, as they are

called,[19] are "considered relevant for determining whether an epidemiologically-observed correlation between a potential causal agent and a disease can or cannot legitimately be treated as a cause rather than as merely an association." Jennifer L. Mnookin, *Atomism, Holism, and the Judicial Assessment of Evidence*, 60 UCLA L. Rev. 1524, 1524 (2013). Stated differently, the Bradford Hill criteria are factors that are considered when a researcher seeks to determine whether an observed epidemiological association between a disease and a chemical agent is causal. *Nonnon v. City of New York*, 932 N.Y.S.2d 428, 433 (2011). *See also Gannon v. United States*, 571 F. Supp. 2d 615, 624 (E.D. Pa. 2007) ("Other preeminent scientists have relied on and adapted the Bradford Hill criteria to determine whether a virus can be deemed to cause human cancer."). "[C]ourts that have considered the question have held that it is not proper methodology for an epidemiologist to apply the Bradford Hill factors without data from controlled studies showing an association." *In re Fosamax Prods. Liab. Litig.*, 645 F. Supp. 2d 164, 188 (S.D.N.Y. 2009).

The Bradford Hill criteria include: (1) strength of the association, (2) consistency of the association, (3) specificity of the association, (4) temporal relationship of the association, (5) biological gradient or dose-response curve of the association, (6) plausibility of the causation, (7) coherence of the explanation, (8) experimental data, and (9)

[19]They are also known as the Bradford Hill viewpoints. *See Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp. 2d 584, 592 n.9 (D.N.J. 2002) ("These factors, first set forth by Sir Austin Bradford Hill, also have been referred to as 'viewpoints'[.]").

existence of analogous causal relationships. Hill, *supra*, 58 Proc. Royal Soc'y Med. at 295-99. *See also Watson v. Dillon Cos., Inc.*, 797 F. Supp. 2d 1138, 1150 (D. Colo. 2011); *Merrell Dow Pharms., Inc. v. Havner*, 953 S.W.2d 706, 718-19 (Tex. 1997).[20]

The Bradford Hill criteria are "not exhaustive and that no one type of evidence must be present before causality may be inferred." *Milward*, 639 F.3d at 17. *See also In re Asbestos Litig.*, 900 A.2d 120, 134-35 (Del. Super. Ct. 2006) ("None of these criteria stand alone; they are all important when considering the issues of association and risk."). That is, "one or more of the factors may be absent even where a causal relationship exists[.]" *Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp. 2d 584, 592 n.9 (D.N.J. 2002).[21] Comments on each of the Bradford Hill criteria follow.

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[20]The "weight of the evidence" methodology can be used along with the Bradford Hill criteria. *See Milward*, 639 F.3d at 17.

[21]This point also was emphasized by Hill, who cautioned in his article:

> None of my nine viewpoints can bring indisputable evidence for or against the cause and effect hypothesis and none can be required as a sine qua non. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question-is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?

Sir Austin Bradford Hill, *The Environment and Disease: Association or Causation?* , 58 Proc. Royal Soc'y Med. 295, 299 (1965).

1. <u>Strength of association</u>. Showing that a strong association exists between a chemical agent and a disease is more likely indicative of a causal relationship. That is, the stronger the relationship between the chemical agent and the disease, the less likely it is that the relationship is due to chance or an extraneous variable (a confounder). Hill provided the following example of this criterion:

> To take a more modern and more general example upon which I have now reflected for over fifteen years, prospective inquiries into smoking have shown that the death rate from cancer of the lung in cigarette smokers is nine to ten times the rate in non-smokers and the rate in heavy cigarette smokers is twenty to thirty times as great. On the other hand the death rate from coronary thrombosis in smokers is no more than twice, possibly less, the death rate in nonsmokers. Though there is good evidence to support causation it is surely much easier in this case to think of some features of life that may go hand-in-hand with smoking – features that might conceivably be the real underlying cause or, at the least, an important contributor, whether it be lack of exercise, nature of diet or other factors. But to explain the pronounced excess in cancer of the lung in any other environmental terms requires some feature of life so intimately linked with cigarette smoking and with the amount of smoking that such a feature should be easily detectable.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 295-96. *See also King*, 762 N.W.2d at 40 ("[R]egarding an association's strength, the higher the relative risk, the greater the likelihood that a relationship is causal. Yet lower relative risks can reflect causality.").

2. <u>Consistency of the association</u>. The consistency of association criterion seeks to determine whether a similar association may be found in a variety of different

situations. Showing numerous observations of an association, with different people in diverse situations with different measurement tools, will increase the credibility of an association finding. Hill provided the following commentary on this factor:

> This requirement may be of special importance for those rare hazards singled out in the Section's terms of reference. With many alert minds at work in industry today many an environmental association may be thrown up. Some of them on the customary tests of statistical significance will appear to be unlikely to be due to chance. Nevertheless whether chance is the explanation or whether a true hazard has been revealed may sometimes be answered only by a repetition of the circumstances and the observations.
>
> Returning to my more general example, the Advisory Committee to the Surgeon-General of the United States Public Health Service found the association of smoking with cancer of the lung in 29 retrospective and 7 prospective inquiries. The lesson here is that broadly the same answer has been reached in quite a wide variety of situations and techniques. In other words we can justifiably infer that the association is not due to some constant error or fallacy that permeates every inquiry. And we have indeed to be on our guard against that.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 296. *See also* Frank C. Woodside, III and Allison G. Davis, *The Bradford Hill Criteria: The Forgotten Predicate*, 35 T. Jefferson L. Rev. 103, 116 (2013) ("Reduced to an elementary level, consistency demonstrates that the results of a particular study are not an outlier result. Consistency indicates that the results are generally concurrent with the results of other studies–not that they are generally accepted.").

3. <u>Specificity of the association.</u> The specificity factor seeks to show that an

effect, *e.g.*, lung cancer, has only one cause, smoking. Hill discussed this factor as follows:

> If, as here, the association is limited to specific workers and to particular sites and types of disease and there is no association between the work and other modes of dying, then clearly that is a strong argument in favour of causation.
>
> . . . .
>
> Coming to modern times the prospective investigations of smoking and cancer of the lung have been criticized for not showing specificity - in other words the death rate of smokers is higher than the death rate of non-smokers from many causes of death. But here surely one must return to my first characteristic, the strength of the association. If other causes of death are raised 10, 20 or even 50% in smokers whereas cancer of the lung is raised 900-1,000% we have specificity–a specificity in the magnitude of the association.
>
> We must also keep in mind that diseases may have more than one cause. It has always been possible to acquire a cancer of the scrotum without sweeping chimneys or taking to mulespinning in Lancashire. One-to-one relationships are not frequent. Indeed I believe that multicausation is generally more likely than single causation though possibly if we knew all the answers we might get back to a single factor.
>
> In short, if specificity exists we may be able to draw conclusions without hesitation; if it is not apparent, we are not thereby necessarily left sitting irresolutely on the fence.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 297. *See also* Woodside and Davis, *supra*, 35 T. Jefferson L. Rev. at 116 ("The crux of the specificity consideration is that causation is likely if a very specific population at a specific site develops a disease with no other likely explanation. More specifically, well performed studies demonstrating an association between a specific exposure and a clearly defined disease or condition–otherwise known as

the case definition–are of more value in inferring the existence of a causal relationship than studies with poorly defined exposures and/or loosely defined diseases or conditions.").

4. <u>Temporal relationship of the association</u>. This factor seeks to assure that the exposure to a chemical agent preceded the disease by a reasonable amount of time, *i.e.*, a cause must precede an effect in time. Hill commented briefly on this factor as follows:

> My fourth characteristic is the temporal relationship of the association–which is the cart and which the horse? This is a question which might be particularly relevant with diseases of slow development. Does a particular diet lead to disease or do the early stages of the disease lead to those peculiar dietetic habits? Does a particular occupation or occupational environment promote infection by the tubercle bacillus or are the men and women who select that kind of work more liable to contract tuberculosis whatever the environment–or, indeed, have they already contracted it? This temporal problem may not arise often but it certainly needs to be remembered, particularly with selective factors at work in industry.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 298. *See also* Woodside and Davis, *supra*, 35 T. Jefferson L. Rev. at 119 ("Not only must the exposure precede the development of the alleged symptoms, but the period of time between the alleged exposure and the onset of symptoms for which compensation is sought must be consistent with the known latency period for the exposure in question. The latency period is the period of time between exposure to an agent and manifestation of disease symptoms.").

5. <u>Biological gradient or dose-response curve of the association.</u> The

biological gradient factor seeks to show or determine whether increased exposure to a chemical agent increases the incidence of the disease. Hill addressed this factor as follows:

> [I]f the association is one which can reveal a biological gradient, or dose-response curve, then we should look most carefully for such evidence. For instance, the fact that the death rate from cancer of the lung rises linearly with the number of cigarettes smoked daily, adds a very great deal to the simpler evidence that cigarette smokers have a higher death rate than non-smokers. That comparison would be weakened, though not necessarily destroyed, if it depended upon, say, a heavier death rate in light smokers and a lower rate in heavier smokers. We should then need to envisage some much more complex relationship to satisfy the cause-and-effect hypothesis. The clear dose-response curve admits of a simple explanation and obviously puts the case in a clearer light.
>
> The same would clearly be true of an alleged dust hazard in industry. The dustier the environment the greater the incidence of disease we would expect to see. Often the difficulty is to secure some satisfactory quantitative measure of the environment which will permit us to explore this dose-response. But we should invariably seek it.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 298. *See also King*, 762 N.W.2d at 40 ("A dose-response relationship is primarily a hallmark of toxicology. If higher exposures to the agent increase the incidence of disease, the evidence strongly suggests a causal relationship.").

6. Plausibility of the causation. Showing that an association is causal is easier when biological or other facts support such a conclusion. However, such evidence is not essential. Hill tersely commented on this factor as follows:

> It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 298. *See also King*, 762 N.W.2d at 41-42 ("When experts know how a disease develops, an association should show biological consistency with that knowledge. . . . An expert's inability to explain a disease's pathology or progression goes to the weight of the evidence, not to its admissibility.").

7. <u>Coherence of the explanation</u>. The viability of an association is enhanced when it does not conflict with what is known about the study variables, and when competing plausible theories or hypotheses do not exist. In other words, an association should be coherent with relevant other knowledge. Hill commented on this factor as follows:

> [T]he cause-and effect interpretation of our data should not seriously conflict with the generally known facts of the natural history and biology of the disease–in the expression of the Advisory Committee to the Surgeon-General it should have coherence.
>
> Thus in the discussion of lung cancer the Committee finds its association with cigarette smoking coherent with the temporal rise that has taken place in the two variables over the last generation and with the sex difference in mortality–features that might well apply in an occupational problem. The known urban/rural ratio of lung cancer mortality does not detract from coherence, nor the restriction of the effect to the lung.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 298. *See also* Woodside and Davis, *supra*, 35 T. Jefferson L. Rev. at 123 ("The difference between coherence and plausibility would seem,

in part, to be one of semantics. While plausibility is worded positively (an association should be in line with substantive knowledge), coherence is presented negatively (an association should not seriously conflict with substantive knowledge). Consideration of coherence would reject an observed result as non-causal if it contradicted a predominant theory; while plausibility leaves the researcher more room regarding which particular piece of substantive knowledge to evaluate the results against.").

8. <u>Experimental data</u>. An association can be enhanced by any related research that is based on experiments. Hill said the following about this factor:

> Occasionally it is possible to appeal to experimental, or semi-experimental, evidence. For example, because of an observed association some preventive action is taken. Does it in fact prevent? The dust in the workshop is reduced, lubricating oils are changed, persons stop smoking cigarettes. Is the frequency of the associated events affected? Here the strongest support for the causation hypothesis may be revealed.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 298-99. *See also* Woodside and Davis, *supra*, 35 T. Jefferson L. Rev. at 124 ("From a scientific standpoint, it is unfortunate that this type of evidence is generally not available. When an agent's effects are suspected to be harmful, researchers cannot knowingly expose people to the agent. It is difficult to design these types of studies due to the ethical implications of experimentation on humans.").

9. Existence of analogous causal relationships. This factor seeks to

determine whether an accepted phenomenon in one area can be applied to another area. Hill tersely commented on this issue as follows:

> In some circumstances it would be fair to judge by analogy. With the effects of thalidomide and rubella before us we would surely be ready to accept slighter but similar evidence with another drug or another viral disease in pregnancy.

Hill, *supra*, 58 Proc. Royal Soc'y Med. at 299. *See also* Woodside and Davis, *supra*, 35 T. Jefferson L. Rev. at 125 ("Recent case law has cast caution upon the extent to which evidence of analogy may be considered in developing opinions on causation. Courts have warned that a reliable methodology must still be utilized in drawing analogies.").

G. Qualification, Methodology and Opinion of the Expert Witnesses

As previously mentioned, the three expert witnesses who testified for the Petitioner at the evidentiary hearing were Dr. Infante, Dr. Goldstein and Dr. Durie. CSX called Dr. Shields and Dr. Green as expert witnesses. In this section we will summarize each expert's qualifications, methodology and opinion.

1. Dr. Infante's qualifications, methodology and opinion. Dr. Infante was called as an expert witness by Petitioner. Dr. Infante received a Ph.D. in public health from

the Department of Epidemiology at the University of Michigan in 1973.[22] Dr. Infante has published approximately 118 peer reviewed articles in scientific journals, the majority of which involve epidemiology causation. Dr. Infante's area of expertise is occupational environmental epidemiology.

In 1973, Dr. Infante worked as a research associate at the University of Michigan and as an epidemiologic consultant for the World Health Organization in Washington, D.C. Dr. Infante was employed as an epidemiologist for the Ohio Department of Health from 1974-1975. During the period 1975-1978, Dr. Infante worked as an epidemiologist for the National Institute for Occupational Safety and Health ("NIOSH"), Center for Disease Control, in Cincinnati, Ohio. While working for NIOSH, Dr. Infante performed epidemiological studies of workers who were exposed to chemical substances that included benezene, pesticides and vinyl chloride. From 1978-2002, Dr. Infante worked for the Occupational Safety and Health Administration ("OSHA"), United States Department of Labor, in Washington, D.C. While with OSHA, Dr. Infante was employed as the Director of the Office of Carcinogen Identification and Classification ("OCIC") for five years and as the Director of the Office of Standards Review ("OSR") for nineteen years. Dr. Infante's work at OCIC included identifying workplace substances that had the ability to cause cancer

[22]Dr. Infante also received a D.D.S. degree from the College of Dentistry at the Ohio State University in 1966.

and classifying them. Dr. Infante's work at OSR involved evaluating workplace exposure to harmful substances and developing occupational exposure limits for substances that were causing cancer in the workplace. This research work involved developing standards for toxic workplace substances that included asbestos, arsenic, benzene, cadmium, ethylene oxide and formaldehyde. From 2002-2011, Dr. Infante was an adjunct professor and lecturer at the School of Public Health and Health Service, George Mason University.

During his career Dr. Infante has been a consultant or advisor for the World Health Organization, National Institute of Environmental Health Sciences, Department of Health and Human Services, National Safety Council, National Academy of Sciences, National Cancer Institute, Federal Asbestos Taskforce, and the American Public Health Association. Dr. Infante is a Fellow in the American College of Epidemiology.

Dr. Infante was retained in this litigation to render an epidemiological opinion as to whether there was an association between diesel exhaust and multiple myeloma, and whether diesel exhaust caused Mr. Harris' multiple myeloma. Dr. Infante relied upon the epidemiological methodology in conjunction with the Bradford Hill criteria.

Dr. Infante reviewed epidemiology literature involving railroad worker diesel exhaust exposure and multiple myeloma; animal cancer studies related to diesel exhaust

exposures; the effects of diesel exhaust on DNA and human lymphocytes; components of diesel exhaust that demonstrate an elevated risk of multiple myeloma; and data involving exposure to two components of diesel exhaust: pristane and benzene.

Dr. Infante testified to reviewing a study by Dr. Tomoko Sonoda et al., *Meta-Analysis of Multiple Myeloma and Benzene Exposure*, 11 J. Epidemiol. 249 (2001),[23] which demonstrated a significant association between engine exhaust and multiple myeloma. Dr. Infante testified that the International Agency for Research on Cancer issued Technical Publication Number 42 in 2009, and that the publication stated that diesel exhaust exposures have been linked to multiple myeloma and leukemia. Dr. Infante further testified that in the third edition of a treatise by David Schottenfeld and Joseph F. Fraumeni, Jr., *Cancer Epidemiology and Prevention*, it was reported that studies show an association between diesel exhaust and elevated risk of multiple myeloma.

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[23]Dr. Infante defined "meta-analysis" as follows;

> A [meta] analysis is an analysis where you pull the data from a number of studies, and you combine the data, and then you evaluate the studies that you then select to determine whether or not there's an elevated risk of – of the associations that you're interested in evaluating.

"Meta-analyses do not involve conducting any new experiments, but are nevertheless highly regarded in the scientific community for their ability to synthesize a large amount of data and illustrate a general consensus in a particular field." *State v. Lawson*, 291 P.3d 673, 700 n.12 (Or. 2012).

The report Dr. Infante prepared for the Petitioner summarized the bases for his opinion as follows:

> Cohort and case-control studies have demonstrated that workers exposed to diesel exhaust (DE) have a significantly elevated risk of death from [multiple myeloma] MM. Epidemiological studies have also demonstrated chromosomal damage to B-lymphocytes of workers exposed to diesel exhaust. Another cancer of the B-cell line, chronic lymphatic leukemia, also demonstrated a significant association with exposure to diesel exhaust. Furthermore, benzene, a component of DE, also has been significantly associated with an elevated risk of developing MM, and pristane, an additional component of DE, has demonstrated the induction of plasmacytomas in experimental animals. These latter tumors are similar to human MM.
>
> The association between diesel exhaust exposure and MM has been derived in the face of several factors that limit the ability to detect such an association through epidemiological study. The difficulties in identifying an association with MM in epidemiological study are a reflection of several factors. . . . In spite of the . . . limitations, several cohort studies of workers exposed to diesel exhaust now demonstrate elevated risks of death from MM . . . .
>
> Case-control studies which allow for the recruitment of much larger cases of MM can be identified in cohort studies also have been conducted. A large number of these studies demonstrate a significant association between exposure to diesel exhaust and MM.
>
> Diesel exhaust also has been demonstrated to cause DNA damage to the lymphocytes of exposed workers. In addition, experimental studies demonstrate that diesel exhaust and components of diesel exhaust, *e.g.*, polycyclic aromatic hydrocarbons, are mutagenic in experimental test systems, and cause cancer in experimental animals. Diesel exhaust itself as well as additional components of diesel exhaust are known to

> cause cancer in experimental animals, including lymphoma, and additional components of DE also demonstrate the induction of cancer in experimental animals, including lymphomas. This information provides biological plausibility to the epidemiological observations related to diesel exhaust and risk of developing MM.

Ultimately, Dr. Infante opined that there is a significant association between diesel exhaust and the risk of multiple myeloma and, that "Mr. Harris' occupational exposure to [diesel exhaust] between 1978 and 2007 were [sic] significant contributing factors and the most likely cause of his development of [multiple myeloma]."[24]

2. Dr. Goldstein's qualifications, methodology and opinion. Dr. Goldstein was called as an expert witness by Petitioner. Dr. Goldstein received a Ph.D. in biology in 1962 from the State University of New York, at Buffalo. Dr. Goldstein has published roughly 60 peer reviewed articles in scientific journals. Dr. Goldstein's area of expertise is animal toxicology, specifically with respect to polycyclic aromatic hydrocarbons.[25]

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[24]During his testimony, Dr. Infante acknowledged that he reviewed literature that did not support his opinion.

[25]"Polycyclic aromatic hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. PAHs are usually found as a mixture containing two or more of these compounds, such as soot." Agency for Toxic Substances and Disease Registry, http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=25 (last visited Nov. 8, 2013).

From 1972 to 1989, Dr. Goldstein worked at the University of California, at San Francisco, in various capacities, including associate professor in the Department of Radiology Oncology. From 1989 to 2002, Dr. Goldstein was employed as a researcher by Electric Power Research Institute ("EPRI"), Palo Alto, California. While at EPRI, Dr. Goldstein conducted and supervised research involving the toxicological hazards caused by polycyclic aromatic hydrocarbons that are found in coal tars.[26] The World Health Organization employed Dr. Goldstein in 2002 to evaluate and compare radiation hazards associated with cell phone use with that of carcinogenic hazards associated with coal tars. In 2004, the federal Environmental Protection Agency hired Dr. Goldstein to be part of a group that was charged with the responsibility of revising the approach used to evaluate the polycyclic aromatic hydrocarbon hazard caused by complex mixtures such as coals.

Dr. Goldstein was retained in this litigation to render an opinion as to whether diesel exhaust caused multiple myeloma. Dr. Goldstein testified that he relied upon the weight of the evidence methodology to render his opinion.

Dr. Goldstein reviewed literature from governmental and international agencies that addressed the issue of whether diesel exhaust caused cancer in general. This literature

[26]Dr. Goldstein testified that polycyclic aromatic hydrocarbons are found in diesel exhaust as well as coal tar. He also testified that "[c]hemically they are the same, but their distribution and concentration within the two sources would vary."

included publications from the Environmental Protection Agency,[27] International Agency for Research on Cancer,[28] National Institute of Occupational Science and Health, National

[27]The EPA's published report concluded the following:

> **II.A.1. Weight-of-Evidence Characterization**
>
> Using U.S. EPA's revised draft 1999 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1999), diesel exhaust (DE) is likely to be carcinogenic to humans by inhalation from environmental exposures. The basis for this conclusion includes the following lines of evidence:
>
> [1] strong but less than sufficient evidence for a causal association between DE exposure and increased lung cancer risk among workers in varied occupations where exposure to DE occurs;
>
> [2] extensive supporting data including the demonstrated mutagenic and/or chromosomal effects of DE and its organic constituents, and knowledge of the known mutagenic and/or carcinogenic activity of a number of individual organic compounds that adhere to the particles and are present in the DE gases;
>
> [3] evidence of carcinogenicity of DPM and the associated organic compounds in rats and mice by other routes of exposure (dermal, intratracheal, and subcutaneous and intraperitoneal injection); and
>
> [4] suggestive evidence for the bioavailability of DE organic compounds from DE in humans and animals.

Diesel Engine Exhaust (CASRN N.A.) Integrated Risk Info. Syst., U.S. Envtl. Prot. Agency, http://epa.gov/IRIS/subst/0642.htm (last visited Nov. 8, 2013).

[28]Dr. Goldstein reported data from a 1988 study by IARC, which found diesel exhaust "probably carcinogenic to humans (Group 2A)." It appears that after Dr. Goldstein's testimony and report in 2011, IARC released a new study on June 12, 2012, which "classified (continued...)

Toxicology Program of the National Institutes of Environmental Health Science, and the American Conference of Certified Industrial Hygienists. Based upon his review and analysis of the literature on the subject, Dr. Goldstein opined that diesel exhaust can cause cancer in general.

In determining whether diesel exhaust caused multiple myeloma, Dr. Goldstein focused his research on the polycyclic aromatic hydrocarbon chemicals that are found in diesel exhaust. After reviewing literature involving animal studies and the effects of polycyclic aromatic hydrocarbons, Dr. Goldstein found that polycyclic aromatic hydrocarbon, ingested through the lungs and carried through the bloodstream, can travel into bone marrow and impact blood forming organs that are responsible for the development of multiple myeloma. In other words, Dr. Goldstein opined that polycyclic aromatic hydrocarbons caused multiple myeloma.[29]

Dr. Goldstein's report set out an analysis of the degree to which Mr. Harris was

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[28](...continued)
diesel engine exhaust as **carcinogenic to humans (Group 1)**, based on sufficient evidence that exposure is associated with an increased risk for lung cancer." International Agency for Research on Cancer, World Health Org., *IARC*:*Diesel Engine Exhaust Carcinogenic*, http://www.iarc.fr/en/media-centre/pr/2012/pdfs/pr213_E.pdf (last visited on Nov. 8, 2013).

[29]Dr. Goldstein made clear that no study that he reviewed stated definitively that polycyclic aromatic hydrocarbons caused multiple myeloma. His opinion was based upon the weight of the evidence.

exposed to diesel exhaust:[30]

> In evaluating the risk posed to Mr. Harris by diesel exhaust in his work environment it is important to get some idea of the dose. Unfortunately no contemporaneous measures of relevant contaminants were made in the time before his diagnosis. . . . What I will therefore attempt to do is put the dose ratio into perspective by using available data (including anecdotal evidence) as well as guidelines for diesel exhaust proposed by the American Conference of Governmental Hygienists for a Threshold Value limit for diesel exhaust of 0.15mg/m^3 (Time Weighted Average). It is intended to provide a perspective of the conditions when a train was dragging a full load uphill in an unvented tunnel.
>
> For this calculation I assume that the engines on Mr. Harris' run were 3000 hp and met the exhaust standards of 0.6 g particulate matter and 1.0 g total hydrocarbons (PAH) per bhp-hr for diesel engines manufactured between 1973 and 2001 (63 CFR 18997-19084, 16 Apr. 1998). Thus each locomotive in the consist taking 10-20 minutes to pass through one of the longer tunnels on the Allegheny and New River routes would have produced 300-600 g of particulate matter. Using Stretcher's Neck for this example, the tunnel is 1588 feet long and the bore is 21' x 25 feet (estimated from photos of the tunnel), the tunnel has a volume of 30878 cubic yards. For a roughly 10-20 minute

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[30]Courts have

> recognized that in toxic tort cases it is generally difficult or impossible to quantify a plaintiff's exposure to a toxin[.] [Therefore], [i]t is not always necessary for a plaintiff to quantify exposure levels precisely or use the dose-response relationship, provided that whatever methods an expert uses to establish causation are generally accepted in the scientific community.

Nonnon v. City of New York, 932 N.Y.S.2d 428, 436-37 (N.Y. App. Div. 2011).

> exposure the trainmen would have been in an environment of 9.7-19.5mg/yd^3 of particulate matter, though the concentration in the engine cab would likely be less. To put this in perspective, the ACGLH proposed (subsequently withdrawn) a threshold limit of value 0.5-0.15mg/m^3 (one m^3 and one yd^3 are essentially the same) time weighted average of particulate matter for an 8 hour workday in its recommendation. Using the 0.15mg/m^3 value, Mr. Harris found himself surrounded by an environment that exceeded the proposed 8 hour average concentration limit by 65-to-130-fold. . . .
>
> . . . .
>
> It is my opinion that Mr. Harris through his employment by CSX was exposed to high levels of diesel exhaust, an agent determined by scientific and medical experts to be a probable or likely human carcinogen. Absent other factors, it can be reasonably concluded that this exposure was a major factor in his multiple myeloma. The weight of scientific and medical evidence from humans, animal studies, studies with tissues and cells using diesel exhaust, closely related pyrogenic materials and chemicals known to be in diesel exhaust supports this conclusion as does an understanding of the conditions under which Mr. Harris worked for 29 years.

3. Dr. Durie's qualifications, methodology and opinion. Dr. Durie was called as an expert witness by Petitioner. Dr. Durie received a medical degree in 1966 from the University of Edinburgh Medical School, Edinburgh, Scotland. Dr. Durie has published approximately 400 peer reviewed articles in scientific journals, the majority of which involve multiple myeloma. He has been recognized as one of the top ten multiple myeloma researchers in the world. Dr. Durie is board certified in internal medicine, hematology and oncology.

From 1972-1992, Dr. Durie was on the faculty at the University of Arizona College of Medicine. Dr. Durie was on the faculty at Charing Cross and Westminster Medical School, University of London, from 1989-1992. From 1993 to the present, Dr. Durie has been the Director of Hematologic Research and Myeloma Programs at Cedars-Sinai Comprehensive Cancer Center at the University of California, Los Angeles ("UCLA"). During his career, Dr. Durie has spent roughly thirty years doing laboratory research involving multiple myeloma. Additionally, for many years, Dr. Durie prepared summaries of every article that was published on multiple myeloma and presented the material at the Annual Review of Medicine.

Dr. Durie was retained in this litigation to render an opinion as to whether diesel exhaust caused multiple myeloma. Dr. Durie testified that he relied upon the Bradford Hill methodology to render his opinion.

Dr. Durie reviewed reports by the Environmental Protection Agency, International Agency for Research on Cancer, and National Toxicology Program of the National Institutes of Environmental Health Science, which concluded that diesel exhaust contained chemicals that were carcinogenic in humans, such as benzene and polycyclic aromatic hydrocarbons. He consulted a report linking diesel exhaust with multiple myeloma and epidemiologic literature concerning diesel exhaust and multiple myeloma. He reviewed

animal studies involving exposure to benzene, polycyclic aromatic hydrocarbons and pristane. Dr. Durie reviewed the literature showing that benzene caused the loss of certain chromosomes, and that Mr. Harris suffered the same chromosomal damage. Dr. Durie testified that during his career, he has treated thousands of patients with multiple myeloma and that when he asked "them what their job is, it is amazing how frequently they'll say they're an engineer or that they're working with chemicals. And so the occurrence of occupations where there is a risk of exposure is remarkably frequent."

Dr. Durie's written report summarized his findings indicating the causal relationship of diesel exhaust and multiple myeloma as follows:

> [1] Martyn T. Smith and the group al-Berkley California have detailed the chromosomal changes linked to human benzene exposure. These chromosome changes include specific findings in the bone marrow myeloma cells from Ronald Harris. . . . Ronald Harris's myeloma thus manifests a chromosomal pattern characteristic of benzene exposure.
>
> . . . .
>
> [2] The linage between diesel exhaust carcinogen exposure and the development of multiple myeloma in the case of Ronald Harris is thus both plausible and highly probable.
>
> [3] Of note the more likely than not association between multiple myeloma and diesel exhaust exposure is supported by the known presence of multiple other toxic compounds in the exhaust [such as the] pristane chemical studied extensively by Michael Potter since the 1960's and known to [have] induced plasmacytomata in mice (analogous to human multiple myeloma). Recent studies at UCLA have shown that pristane

> levels can be measured in vivo in humans and linked to immune regulatory dysfunction with increased B-cell activation. Myeloma is derived from abnormal B-lymphocytes. In addition, diesel exhaust contained many of the same polycyclic aromatic hydrocarbons found in coal tar and pitch blends both of which are known human carcinogens. Heavy metals such as nickel are also present with known carcinogenic potential. All this reinforces the plausible and probable causative relationship between diesel exhaust and the development of multiple myeloma.
>
> . . . .
>
> [4] I strongly support the fact that in the case of Ronald Harris the workplace exposures at CSX Transportation Inc. were more probably than not a causative factor in the development of multiple myeloma.

4. **Dr. Shields' qualifications, methodology and opinion.** Dr. Shields was called as an expert witness by CSX. Dr. Shields received a medical degree in 1983 from Mount Sinai School of Medicine, New York. Dr. Shields has published approximately 154 peer reviewed articles in scientific journals. Dr. Shields' area of expertise includes hematology and oncology. Dr. Shields is board certified in internal medicine and oncology.

From 1984-1989, Dr. Shields worked as a civilian physician at three medical facilities in Washington, D.C. Dr. Shields served as a commissioned officer in the United States Public Health Service Commissioned Corps from 1990-1999[31] and ultimately attained

[31]Commissioned Corps officers serve in a variety of positions throughout the (continued...)

the rank of captain. From 2000 to the 2011, Dr. Shields was on the faculty at Georgetown University Medical Center. From 2006-2008, Dr. Shields was the senior medical director at Capital Breast Care Center in Washington, D.C. During the hearing in this case, Dr. Shields testified that he was now employed with the Ohio State University Comprehensive Cancer Center.

Dr. Shields was retained by CSX to render an opinion as to whether diesel exhaust caused multiple myeloma. Dr. Shields testified that he relied upon the Bradford Hill methodology to render his opinion.

Dr. Shields testified that he went on the internet to a website run by the National Institute of Health and researched articles dealing with diesel exhaust and multiple myeloma. Dr. Shields indicated that he reviewed twenty or more papers that involved railroad workers and cancers. Dr. Shields testified that he "inferred" from this data that myeloma was not found in the studies because it was not mentioned. Specifically, Dr. Shields stated that "if myeloma was going to arise from the way they're describing Mr. Harris' exposure, these studies would show it." Dr. Shields acknowledged that he was aware

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[31](...continued)
United States Department of Health and Human Services and certain other federal agencies. *See* United States Pub. Health Serv. Commissioned Corps, U.S. Dep't. of Health & Human Servs., http://www.usphs.gov/aboutus/mission.aspx (last visited on Nov. 8, 2013).

of a study that showed a significant association between myeloma and railroad workers. Dr. Shields discounted the study because, in his opinion, the study did not implicate diesel exhaust as a cause for any of the cancers. Several other studies linking myeloma and diesel exhaust were found not to be significant by Dr. Shields. Studies that showed an association between benzene and myeloma were also rejected by Dr. Shields as not significant to establish causation. Ultimately, Dr. Shields opined that, from his review of the literature, "there's no evidence or there's insufficient evidence that railroad workers are at increased risk of myeloma." At the conclusion of Dr. Shields direct examination, counsel for CSX asked the following question:

> Q. Do you have an opinion as to whether the hypothesis in this case that exposure to diesel exhaust causes multiple myeloma has been proven?
>
> A. Yes. It's my opinion that it – that it's not been proven.

5. Dr. Green's qualifications, methodology and opinion. Dr. Green was called as an expert witness by CSX. Dr. Green received a Ph.D. in food science and technology from the Department of Nutrition and Food Science at Massachusetts Institute of Technology in 1981. Dr. Green has published approximately 139 peer reviewed articles in scientific journals. She is also the author of "In Search of Safety: Chemicals and Cancer Risk" (Harvard University Press 1988). Dr. Green's area of expertise is toxicology. Dr. Green is a board certified toxicologist.

Dr. Green was a research director of Scientific Conflict Mapping Project at Harvard University from 1983-1985. From 1985-1989, Dr. Green was employed at Meta Systems, Inc., as vice president of Environmental Health and Toxicology. From 1989 to the present, Dr. Green has been president of Cambridge Environmental, Inc.

Dr. Green was retained by CSX to render an opinion as to whether diesel exhaust caused multiple myeloma. Dr. Green did not indicate any specific methodology that she used to render her opinion. However, her testimony suggests she followed the Bradford Hill methodology.

Dr. Green testified that she does not know of any literature linking any type of cancer through the inhalation of pristane. Dr. Green also testified that neither the Environmental Protection Agency's Health Assessment Document for Diesel Engine Exhaust nor the National Toxicology Program support the assertion that diesel exhaust causes myeloma cancer. Dr. Green found a study of Swedish workers by Dr. Paolo Bofetta was irrelevant, even though the study showed that over 800 workers exposed to diesel exhaust contracted multiple myeloma. Dr. Green found the study was not significant because over 800 other men who were studied contracted multiple myeloma, but there was no evidence that they also were exposed to diesel exhaust. Dr. Green opined that "diesel engine exhaust might cause lung cancer, but there is no credible evidence that it causes multiple myeloma."

H. The Circuit Court's Orders Excluding the Testimony of Petitioner's Experts

We have no hesitancy in finding that the opinions of Petitioner's three experts regarding the causal link between diesel exhaust and multiple myeloma satisfy certain requirements of Rule 702. Their opinions would "assist the trier of fact to understand the evidence or to determine a fact in issue." W. Va. R. Evid. 702. All three experts are witnesses "qualified as an expert by knowledge, skill, experience, training, or education." *Id*. Additionally, the testimony of the experts was relevant to issues in the case. W. Va. R. Evid. 402. Thus, the question before us is whether the trial court abused its discretion in concluding that the reliability prong of Rule 702 was not met. That issue, properly framed, is whether Petitioner's three experts used reliable methodologies in rendering opinions on the causation issue linking diesel exhaust with multiple myeloma. As we will explain, below, the trial court's analysis exceeded this narrow issue. Instead, the court in rendering its ruling, addressed the jury question: Did Petitioner's three experts prove causation? Because the trial court exceeded the scope of its narrow review of the reliability prong of Rule 702, we find it necessary to examine cases that have demonstrated the narrow focus used to make the reliability determination.

To begin, the court in *King v. Burlington Northern Santa Fe Railway Co.*, 762 N.W.2d 24 (Neb. 2009), provided an excellent analysis of the limited gatekeeper role of trial courts. In *King*, the wife of a deceased former railroad employee brought an action seeking

damages against the railroad under the Federal Employers' Liability Act.[32] The plaintiff alleged that her husband contracted multiple myeloma due to his exposure to diesel exhaust fumes while working for the railroad as a brakeman. The defendant moved the court to exclude the plaintiff's expert. The opinion in *King* summed up the arguments and the trial court's ruling as follows:

> Differing epidemiological studies supported the experts' deposition testimony. [Plaintiff's] expert, Dr. Arthur Frank, blamed [decedent's] multiple myeloma on his exposure to diesel exhaust. Of course, [defendant's] expert, Dr. Peter G. Shields, disagreed. He believed that the causes were unknown and that the majority of epidemiological studies failed to show that diesel exhaust can cause multiple myeloma. The district court sustained [defendant's] motion to exclude Frank's testimony, concluding that it failed to pass muster under our *Daubert/Schafersman* framework. It reasoned that his methodology was unreliable because the studies he relied on failed to conclusively state that exposure to diesel fuel exhaust causes multiple myeloma.

King, 762 N.W.2d at 31.

After the trial court excluded the plaintiff's expert witness in *King*, it granted summary judgment to the defendant. The plaintiff appealed to a Nebraska appellate court. The appellate court affirmed. The plaintiff then appealed to Nebraska's Supreme Court. The high court in *King* reversed the ruling of the trial court after concluding that it applied an

[32]The plaintiff's husband was the original plaintiff, but he died during the pendency of the litigation.

improper standard for reviewing the admissibility of expert testimony. The opinion in *King* outlined the following limited gatekeeper role of trial courts:

> Here, the parties do not dispute Frank's qualification to give expert medical testimony or to interpret epidemiological studies. We see the broad issue as whether under our *Daubert/Schafersman* framework, Frank based his opinion on a reliable, or scientifically valid, methodology. . . .
>
> In determining the admissibility of an expert's opinion, the court must focus on the validity of the underlying principles and methodology—not the conclusions that they generate. And reasonable differences in scientific evaluation should not exclude an expert witness' opinion. The trial court's role as the evidentiary gatekeeper is not intended to replace the adversary system but to ensure that an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field. In sum, while the trial court acts as the evidentiary gatekeeper, it is not a goalkeeper.
>
> . . . .
>
> . . . Absent evidence that an expert's testimony grows out of the expert's own prelitigation research or that an expert's research has been subjected to peer review, experts must show that they reached their opinions by following an accepted scientific method or procedure as it is practiced by others in their field.
>
> Epidemiological statistical techniques for testing a causation theory have been subject to peer review and are generally accepted in the scientific community. The studies Frank relied upon were subject to peer review, and the researchers did not develop the statistical techniques used in the studies for this litigation. . . . Accordingly, the district court needed to consider only two issues regarding Frank's opinion on . . . causation. Were the results of the epidemiological studies

> Frank relied on sufficient to support his opinion regarding . . . causation? And did he review the scientific literature or data in a reliable manner? In other words, did too great an analytical gap exist between the data and Frank's opinion?
>
> . . . .
>
> We believe the district court erred in concluding that Frank's causation opinion was unreliable because Frank could not point to a study that concludes exposure to diesel exhaust causes multiple myeloma. As explained, individual epidemiological studies need not draw definitive conclusions on causation before experts can conclude that an agent can cause a disease. If the expert's methodology appears otherwise consistent with the standards set out above, the court should admit the expert's opinion. But here, the court did not inquire into Frank's methodology.

King, 762 N.W.2d at 42-49 (internal quotations and citations omitted).

Another case, though it did not involve multiple myeloma, which illustrates a trial court's limited gatekeeper role is *Milward v. Acuity Specialty Products Group, Inc*., 639 F.3d 11 (1st Cir. 2011). In *Milward*, the plaintiffs, husband and wife, filed an action against manufacturers of products used in refrigerators. The plaintiff husband worked as a refrigeration technician. The plaintiffs alleged that the husband contracted acute promyelocytic leukemia ("APL") as a result of exposure to benzene that was contained in the defendants' products. The trial court held a four day hearing to determine whether plaintiff's expert on causation would be allowed to testify that benzene caused APL. The trial court, "in a detailed opinion, ruled that 'Dr. Smith's proffered testimony that exposure to benzene

can cause APL lacks sufficient demonstrated scientific reliability to warrant its admission under Rule 702.'" *Milward*, 639 F.3d at 13. The trial court thereafter dismissed the action. The First Circuit Court of Appeals reversed after concluding that the trial court exceeded its discretion in finding that the opinion of plaintiffs' expert was wrong. The First Circuit outlined the limited role of the trial court in deciding the admissibility of expert testimony:

> [T]rial courts are [not] empowered to determine which of several competing scientific theories has the best provenance. *Daubert* does not require that a party who proffers expert testimony carry the burden of proving to the judge that the expert's assessment of the situation is correct. The proponent of the evidence must show only that the expert's conclusion has been arrived at in a scientifically sound and methodologically reliable fashion. The object of *Daubert* is to make certain that an expert, whether basing testimony on professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field.
>
> So long as an expert's scientific testimony rests upon good grounds, based on what is known, it should be tested by the adversarial process, rather than excluded for fear that jurors will not be able to handle the scientific complexities. Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence.
>
> . . . .
>
> . . . [T]he alleged flaws identified by the court go to the weight of Dr. Smith's opinion, not its admissibility. There is an important difference between what is unreliable support and what a trier of fact may conclude is insufficient support for an expert's conclusion.
>
> The court's analysis repeatedly challenged the factual

> underpinnings of Dr. Smith's opinion, and took sides on questions that are currently the focus of extensive scientific research and debate—and on which reasonable scientists can clearly disagree. In this, the court overstepped the authorized bounds of its role as gatekeeper. The soundness of the factual underpinnings of the expert's analysis and the correctness of the expert's conclusions based on that analysis are factual matters to be determined by the trier of fact. When the factual underpinning of an expert's opinion is weak, it is a matter affecting the weight and credibility of the testimony—a question to be resolved by the jury.
>
> . . . .
>
> . . . The sum of Dr. Smith's testimony was not merely that it is possible, or even biologically plausible, that benzene causes APL. Rather, the sum of his testimony was that a weighing of the Hill factors, including biological plausibility, supported the inference that the association between benzene exposure and APL is genuine and causal.
>
> The record clearly demonstrates that Dr. Smith's opinion was based on an analysis in which he employed the same level of intellectual rigor that he employs in his academic work. In excluding Dr. Smith's testimony, the district court did not properly apply *Daubert* and exceeded the scope of its discretion. We reverse the district court's judgment for the defendants and its exclusion of Dr. Smith's testimony, and we remand for proceedings consistent with this opinion.

Milward, 639 F.3d at 15-26 (internal quotations and citations omitted)..

In *Wagoner v. Exxon Mobil Corp.*, 813 F. Supp. 2d 771 (E.D. La. 2011), the plaintiff, widow and legal representative of decedent, filed a products liability action against manufacturers of benzene-containing products alleging that, as a result of the decedent's

exposure to benzene, the decedent contracted and died of multiple myeloma. The defendants filed motions to exclude plaintiff's two causation experts. The basis of the motions, and the district court's rejection of the same, were addressed by the court as follows:

> Defendants have raised five arguments with regard to the reliability of the testimony of Dr. Butler and Dr. Saux . . . : 1) their opinion rests on studies that do not show statistically significant findings; 2) their opinion relies on studies that do not examine benzene specifically; 3) their opinion rests on studies that are not published in peer-reviewed journals and are otherwise flawed; 4) their opinion reflects an incomplete review of the relevant literature; and 5) their opinion fails to articulate a biologically plausible mechanism for benzene to cause [multiple myeloma] and thus does not meet the Bradford Hill criteria.
>
> . . . .
>
> None of the arguments raised by Defendants in support of their motions to exclude Dr. Butler and Dr. Saux are persuasive. The two individuals are qualified to render an opinion . . ., and at least two studies support the notion that there is a statistically significant association between benzene and [multiple myeloma]. The fact that those studies may be flawed, that there are studies that cut against the two doctors' opinion, and that the doctors could not articulate a biologically plausible mechanism for benzene to cause [multiple myeloma] all go to the weight of their opinion, and not the question of admissibility. . . . Accordingly, the motions to exclude and for summary judgment must be denied.

Wagoner, 813 F. Supp. 2d at 800-05.

Finally, in *Moreland v. Eagle Picher Technologies*, 362 S.W.3d 491 (Mo. Ct. App. 2012), a Missouri appellate court addressed the admissibility of an expert

opinion under its rules of evidence in the context of a workers' compensation claim. The employee in *Moreland* alleged that he developed multiple myeloma as a result of years of inhalation of chemicals from plastics that his employer produced. The chemicals in the plastics included benzene, trichloroethylene, cadmium, nickel, and platinum. The employee produced an expert witness at the administrative level who testified that his exposure to benzene caused him to develop multiple myeloma. The employer called an expert witness who opined that benzene had never been proven to cause multiple myeloma. The ALJ found in favor of the employee and awarded him workers' compensation benefits. An appellate court affirmed the award. In doing so, the appellate court in *Moreland* set out the following relevant discussion regarding the employee's causation expert:

> Dr. Bernard Goldstein ("Dr. Goldstein"), a professor of medicine at the University of Pittsburg [sic] Graduate School of Public Health and School of Medicine, and also a physician, toxicologist, and hematologist, testified on behalf of Moreland. Dr. Goldstein testified he had studied benzene toxicity and published close to one hundred papers or reviews upon the subject since the 1960s. Dr. Goldstein also specifically published and instructed members of the federal judiciary on issues concerning toxicology and, in particular, the issue of causation and whether chemical agents should be deemed to have caused or contributed to the development of multiple myeloma.
>
> Dr. Goldstein testified that benzene was reasonably probable to be a cause of multiple myeloma based upon epidemiological data, bioassays (experiments on laboratory animals), and mechanistic data. Dr. Goldstein testified that these sources of information are recognized by the International Agency for Research on Cancer and could be applied to substantiate that benzene caused multiple myeloma. . . .

. . . .

> Dr. Goldstein testified that multiple myeloma is an identifiable disease and it is reasonably probable that exposure to benzene, either by air or dermal absorption, or both, is a substantial factor to cause the compounding of cells that lead [sic] to multiple myeloma.
>
> . . . .
>
> Here, [the employer] specifically argues only that Dr. Goldstein's opinion is not based on medical certainty, and is not based on any medical or scientific facts that are reasonably relied upon by experts in the field of medical expertise. However, . . . Dr. Goldstein extensively explained many of the studies which show causation between benzene and multiple myeloma. Further, Dr. Goldstein testified that these sources of information are recognized by the International Agency for Research on Cancer and could be applied to substantiate that benzene causes multiple myeloma. Thus, the facts and data on which Dr. Goldstein based his opinions are a type reasonably relied on by experts in the field.
>
> Accordingly, the Commission's finding that Dr. Goldstein's testimony meets the standard required of expert testimony was supported by competent and substantial evidence.

Moreland, 362 S.W.3d at 500-04 (internal citations omitted).[33]

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[33]In the context of an administrative workers' compensation claim, this Court addressed the issue of the reliability of evidence showing a link between benzene exposure and a cancer called chronic myelogenous leukemia. In *Casdorph v. West Virginia Office Insurance Commissioner*, 225 W. Va. 94, 690 S.E.2d 102 (2009), the claimant worked as an auto mechanic for the State Police for nearly twenty-two years. After the claimant was diagnosed with chronic myelogenous leukemia, he filed a claim for workers' compensation benefits. (The claimant died while the case was pending at the administrative level). The claimant alleged that his cancer was caused by his exposure to benzene in the workplace. During a hearing before an ALJ the claimant provided evidence from several experts, (continued...)

The foregoing authorities consistently demonstrate the narrow scope of a trial court's consideration of the admissibility of scientific expert testimony: [a] narrow focus that our cases have acknowledged, but which far too often has been misunderstood. Therefore,

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[33](...continued)
including Dr. Infante, who testified that claimant "had ample opportunity for occupational exposure to benzene and other solvents contaminated with benzene due to his occupation and stated that benzene is the cause of leukemia and CML is a type of leukemia associated with benzene exposure." *Casdorph*, 225 W. Va. at 102, 690 S.E.2d at 110. The ALJ found that the claimant developed chronic myelogenous leukemia from his exposure to benzene and therefore ruled the claim was compensable. The Board of Review reversed the decision of the ALJ. On appeal, this Court reinstated the ALJ's decision. We concluded as follows:

> The medical literature and expert and fact witness testimony in this case sufficiently established that a causal link between the Appellant's benzene exposure and CML existed. Although the Appellees assert that the case studies cited by Appellant showing a causal connection between benzene exposure and CML have not been able to get peer reviewed textbooks to acknowledge and print them as common or accepted consensus medical opinion, we find that these case studies, although small, are valid studies that have been peer reviewed and published. We acknowledge, as Appellees contend, that this Court recognized in *State v. Leep*, 212 W. Va. 57, 569 S.E.2d 133 (2002) that "whether a scientific theory is generally accepted within a scientific community" is a factor that must be weighed in determining whether to allow such testimony as evidence. However, we must also be reminded that the Rules of Civil Procedure and the Rules of Evidence do not strictly apply to workers' compensation claims.

Casdorph, 225 W. Va. at 104-05, 690 S.E.2d at 112-13 (footnote added). It is important to note that the decision in *Casdorph* made clear that it was not evaluating the admissibility of the expert testimony under the standards of the rules of evidence, because those rules did not strictly apply to workers' compensation litigation. *Casdorph* is distinguishable from *Moreland* in that regard because Missouri applies its rules of evidence to workers' compensation litigation.

we believe it is necessary to carefully and clearly articulate our standard for reviewing the reliability prong of the admission of scientific expert testimony. Thus, we make clear, and so hold that, when a trial court is called upon to determine the admissibility of scientific expert testimony, in deciding the "reliability" prong of admissibility the focus of the trial court's inquiry is limited to determining whether the expert employed a methodology that is recognized in the scientific community for rendering an opinion on the subject under consideration. If the methodology is recognized in the scientific community, the court should then determine whether the expert correctly applied the methodology to render his or her opinion. If these two factors are satisfied, and the testimony has been found to be relevant, and the expert is qualified, the expert may testify at trial.

We wish to clarify that the standards outlined above are not new principles under this Court's *Daubert/Wilt* jurisprudence. These principles have always been an implicit part of the *Daubert/Wilt* analysis. Simply put, however, these principles have not been clearly understood or followed by trial courts. For instance, this Court made the following observations in *Wilt*:

> We . . . are of the view that, under Rule 702, there is a category of expert testimony based on scientific methodology that is so longstanding and generally recognized that it may be judicially noticed, and, therefore, a trial court need not ascertain the basis for its reliability.
>
> Thus, we believe that *Daubert* is directed at situations where the scientific or technical basis for the expert testimony

> cannot be judicially noticed and a hearing must be held to determine its reliability.

Wilt, 191 W. Va. at 46, 443 S.E.2d at 203. This limitation recognized in *Wilt* has been lost in practice. Litigants invariably have crowded trial court calendars with purported *Daubert/Wilt* evidentiary hearings whenever an expert is called to testify. This was never the intent of our *Daubert/Wilt* analysis.

In *Gentry v. Mangum*, 195 W. Va. 512, 466 S.E.2d 171 (1995), Justice Cleckley attempted to clarify how *Daubert/Wilt* was to be applied by "giv[ing] circuit courts more guidance from a procedural standpoint in resolving scientific evidence issues." *Gentry*, 195 W. Va. at 521, 466 S.E.2d at 180. *Gentry* pointed out in crystal clear terms that,

> [a]ctually, most scientific validity issues will be resolved under judicial notice pursuant to Rule 201. Indeed, most of the cases in which expert testimony is offered involve only qualified experts disagreeing about the interpretation of data that was obtained through standard methodologies. *Daubert/Wilt* is unlikely to impact upon those cases. Therefore, circuit courts are right to admit or exclude evidence without "reinventing the wheel" every time by requiring parties to put on full proof of the validity or invalidity of scientific principles. Where judicial notice is appropriate, the circuit court should use it.

Gentry, 195 W. Va. at 522, 466 S.E.2d at 181. In Syllabus point 4 of *Gentry*, Justice Cleckley simplified and reformulated our *Daubert/Wilt* standard as follows:

> When scientific evidence is proffered, a circuit court in its "gatekeeper" role under *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 113 S. Ct. 2786, 125 L. Ed. 2d 469 (1993), and *Wilt v. Buracker*, 191 W. Va. 39, 443

> S.E.2d 196 (1993), *cert denied*, 511 U.S. 1129, 114 S. Ct. 2137, 128 L. Ed. 2d 867 (1994), must engage in a two part analysis in regard to the expert testimony. First, the circuit court must determine whether the expert testimony reflects scientific knowledge, whether the findings are derived by scientific method, and whether the work product amounts to good science. Second, the circuit court must ensure that the scientific testimony is relevant to the task at hand.

195 W. Va. 512, 466 S.E.2d 171.

Gentry attempted to show that a full blown evedentiary *Daubert/Wilt* analysis was required only for evaluating a new and/or novel scientific methodology. Recognized methodologies are the subject of judicial notice. Moreover, this Court explained in Syllabus point four of *Mayhorn v. Logan Medical Foundation*, 193 W. Va. 42, 454 S.E.2d 87 (1994), that

> [p]ursuant to West Virginia Rules of Evidence 702 an expert's opinion is admissible if the basic methodology employed by the expert in arriving at his opinion is scientifically or technically valid and properly applied. The jury, and not the trial judge, determines the weight to be given to the expert's opinion.

See 2 Franklin D. Cleckley, Louis J. Palmer, Jr. and Robin Jean Davis, Handbook on Evidence for West Virginia Lawyers § 702.02[2][c] (5th ed. 2012) ("The [*Daubert/Wilt*] regime contemplates that trial judges will perform a gatekeeping function, determining whether the . . . methodology underlying proffered expert testimony is scientifically valid and whether that . . . methodology properly can be applied to the facts in issue."). We note that

we are not alone in limiting an evidentiary hearing to determine the reliability of experiments conducted for litigation and/or novel scientific methodology. *See Nonnon v. City of New York*, 932 N.Y.S.2d 428, 429 (N.Y. App. Div. 2011) ("[W]e [have] determined that plaintiffs' expert evidence did not require that a hearing be held [because] neither the deductions of the expert epidemiologists and toxicologists, nor the methodologies employed by them, in reaching their conclusions are premised on . . . novel science[.]" (internal quotations and citations omitted)). The court in *Nonnon* observed that

> epidemiology and toxicology are hardly novel sciences, but rather, well-established and accepted methodologies. In such a case, the focus moves from the general reliability concerns . . . to the specific reliability of the procedures followed to generate the evidence proffered and whether they establish a foundation for the reception of the evidence at trial.

Nonnon, 932 N.Y.S.2d at 435.

In the instant case, the trial court erred by holding a mini-trial to set out and resolve issues that were purely matters for jury consideration.[34] The three orders excluding Petitioner's three experts set out and resolved an array of disputed factual matters that were exclusively grist for the jury and which had no relevancy to the limited role the trial court had

[34]This Court is fully aware that litigants have abused the limited resources of our trial judges by demanding full-blown evidentiary hearings in most cases where expert testimony is offered. This opinion is intended to make unequivocally clear that the admissibility principles under *Daubert/Wilt* were never intended to allow the abuse that has become routine in our trial courts.

under the facts of this case. For instance, as noted by Petitioner, the orders found:

> 1. If a difference between a case group and control group is not statistically significant then there is no difference at all.
>
> 2. It is acceptable scientific practice to interpret as "not different" a study that shows an elevated risk that is not statistically significant.
>
> 3. There is substantially more benzene in cigarette smoke than diesel exhaust.
>
> 4. Benzene is present only in trivial doses in diesel exhaust.
>
> 5. The hypothesis that diesel exhaust causes multiple myeloma is confounded by the fact that cigarette smoking does not.
>
> 6. Most epidemiologic studies must be positive for purported causal association to be real.
>
> 7. Of forty-seven (47) studies of diesel exposed workers only eight (8) purport to be positive.
>
> 9. The epidemiologic literature investigating a causal association between railroad employment and multiple myeloma is null and not supportive of the subject hypothesis.
>
> 10. There are approximately ten (10) published studies investigating [sic] causal link between benzene and multiple myeloma. None of them are positive.
>
> 11. The epidemiologic literature regarding PAH exposure and multiple myeloma does not support the subject hypothesis.
>
> 12. IARC Technical Publication 42 was not intended to make a causation statement but to express a research agenda.
>
> 13. The general causation hypothesis that exposure to

diesel exhaust causes multiple myeloma has not been proven.

Clearly, the above findings made by the trial court should never have been considered as part of its limited gatekeeper role in this case. All of the above findings involve disputed opinions between the experts. They have nothing to do with the reliability of the methodologies used by the Petitioner's experts. In fact, the trial court could have resolved the question of the relevancy and reliability of Petitioner's experts through arguments by the parties and without their experts' testimony. It is undisputed that the methodologies employed by Petitioner's experts are recognized in the scientific community. Ironically, CSX's experts relied upon the same methodologies. There is also no reasonable dispute that Petitioner's three experts employed the methodologies in a manner consistent with how they are employed in the scientific community. The only issue that was in dispute was whether Petitioner's experts were correct in reaching the conclusions they reached. Challenging the latter issue is a matter for jury determination.[35]

[35]This Court is aware that some courts have excluded expert testimony on the issue of whether multiple myeloma is caused by diesel exhaust. *See Aurand v. Norfolk S. Ry. Co.*, 802 F. Supp. 2d 950 (N.D. Ind. 2011) (excluding plaintiff experts on multiple myeloma); *Morin v. United States*, 534 F. Supp. 2d 1179 (D. Nev. 2005) (same); *Castellow v. Chevron USA*, 97 F. Supp. 2d 780 (S.D. Tex. 2000) (same); *Estate of Mitchell v. Gencorp, Inc*., 968 F. Supp. 592 (D. Kan. 1997) (same); *Sutera v. Perrier Grp. of Am. Inc*., 986 F. Supp. 655 (D. Mass. 1997) *(*same); *Richardson v. Union Pac. R.R. Co.*, 386 S.W.3d 77 (Ark . Ct. App. 2011) (same); *Missouri Pac. R.R. Co. v. Navarro*, 90 S.W.3d 747 (Tex. Ct. App. 2002) (same). The decisions of the courts in those cases are inconsistent with the standards of admissibility of scientific expert testimony that are followed in this jurisdiction.

We understand there will be cases where a party seeks to offer a new and novel methodology to explain causation, or where a party's expert performed a specific experiment for trial to show causation. In either of those situations, the rigorous prong of the *Daubert/Wilt* gatekeeper analysis is implicated. In stark contrast, the experts in the instant case did not offer new or novel methodologies. The epidemiological, toxicological, weight of the evidence and Bradford Hill methodologies they used are recognized and highly respected in the scientific community. And, as is detailed in this opinion, those experts applied the methodologies consistently with the "level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Milward*, 639 F.3d at 15.

IV.

CONCLUSION

We reverse the circuit court's orders excluding the testimony of Petitioner's three experts. Furthermore, we reverse the order granting summary judgment in favor of CSX. Finally, this case is remanded for further proceedings consistent with this opinion.

Reversed and Remanded.