**[Cite as *Valentine v. PPG Industries, Inc.*, 158 Ohio App.3d 615, 2004-Ohio-4521.]**

IN THE COURT OF APPEALS OF OHIO
FOURTH APPELLATE DISTRICT
PICKAWAY COUNTY

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| VALENTINE, | : | |
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| Appellant, | : | Case No. 03CA17 |
| | : | |
| v. | : | |
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| PPG INDUSTRIES, INC., | : | DECISION AND JUDGMENT ENTRY |
| | : | |
| Appellee, et al. | : | **Released 8/20/04** |
| | : | |

APPEARANCES:

Terrence W. Larrimer and Gary Dumm, for appellant.

Stephen J. Habash and Kevin L. Shoemaker, for appellee.

HARSHA, Judge.

**{¶ 1}** Linda Valentine appeals the trial court's summary judgment in PPG Industries Ohio, Inc.'s favor and various discovery orders. She contests the court's determination that she failed to establish a right to participate in the workers' compensation system for the death of her husband, David Valentine ("Valentine"). Specifically, she asserts that the court improperly found her experts' opinions regarding proximate cause to be unreliable and, thus, inadmissible. Without expert testimony, the court concluded that no genuine issue of material fact remained concerning whether Valentine's workplace exposure

to various toxic substances proximately caused his brain cancer (glioblastoma multiforme) and subsequent death.

**{¶ 2}** She presents four arguments to support her contention that the court incorrectly determined that no genuine issue of fact exists regarding proximate cause: (1) the court failed to consider her three experts' affidavits, (2) the court wrongly concluded that her medical experts' opinions were unreliable and inadmissible, (3) the court improperly determined that her experts' testimony did not show general causation between the decedent's brain tumor and his exposure to carcinogens in the workplace, and (4) the court imposed an incorrect standard of causation under the workers' compensation statute to determine whether a claimant can establish an occupational disease.

**{¶ 3}** Initially, we conclude that the trial court properly applied traditional tort standards of proximate cause in analyzing the connection between workplace exposure and the occurrence of an occupational disease. Accordingly, we reject the appellant's contention that in order to establish causation, a worker need only establish that a workplace exposure increased the risk of contracting a disease above that of the general population. Furthermore, after carefully reviewing the legal landscape concerning the reliability of expert testimony and the scientific literature that formed the basis of her experts' opinions, we conclude that the trial court did not abuse its

discretion by rejecting this testimony. Without their opinions, appellant possessed no evidence to establish that Valentine's workplace exposure to toxic substances caused his brain tumor. Thus, appellant's failure to show proximate cause as an element of her workers' compensation claim is fatal to her claim, and the trial court appropriately entered summary judgment in PPG's favor. Because her remaining arguments are moot, we affirm the trial court's decision.

BACKGROUND

**{¶ 4}** Valentine worked at the PPG Circleville facility from 1969 until 1997, initially as a lab technician and later as an environmental and wastewater-treatment specialist. During his employment Valentine was exposed to various chemicals. In 1997, Valentine was diagnosed with "glioblastoma multiforme," a rare form of brain cancer that accounts for only two to three percent of all new cancers diagnosed in the United States. The only medically proven cause of glioblastoma multiforme is ionizing radiation. Valentine received treatment for his brain tumor at the Ohio State University Hospital, where he saw Dr. Herbert Newton, Arthur James Cancer Research Center Director, and Dr. Michael Miner, Department of Neurosurgery Chairman. Despite their treatment, Valentine unfortunately died in 1999 at the age of 51.

**{¶ 5}** In November 1999, Mrs. Valentine filed a claim for death benefits with the Bureau of Workers' Compensation. She claimed that Valentine's exposure to a toxic brew of chemicals throughout his career at PPG caused him to contract the glioblastoma multiforme that led to his death. Ultimately, the bureau denied the claim.

**{¶ 6}** In August 2000, Mrs. Valentine filed an administrative appeal in the common pleas court. After lengthy and contentious discovery, PPG filed a summary judgment motion, arguing that no genuine issues of material fact existed concerning whether Valentine's work environment proximately caused his brain tumor. Appellee insisted (1) that the opinions of appellant's experts (Drs. Miner and Newton and industrial hygienist Norman Brusk) were inadmissible because they were not scientifically reliable and (2) that without these opinions, appellant had no evidence regarding proximate cause. Appellee contended that the experts' testimony was unreliable because no scientific discipline has established that a specific chemical agent or combination of chemical agents can cause a brain tumor in humans. The trial court agreed and concluded that without their testimony, no genuine issue of material fact remained regarding proximate cause, so the court granted PPG summary judgment.

**{¶ 7}** Mrs. Valentine timely appealed the trial court's judgment and raises the following assignments of error:

First Assignment of Error. It was an abuse of discretion for the trial court to grant appellee's motion for summary judgment without examining and analyzing the affidavit evidence of appellant's three experts.

Second Assignment of Error. The trial court erred in holding that the opinions of appellant's medical experts were unreliable and therefore inadmissible pursuant to Rule 702.

Third Assignment of Error. The trial court erred in ruling as a matter of law that appellant's experts failed to establish general causation between David Valentine's exposure to carcinogens in his workplace and the development of his brain tumor.

Fourth Assignment of Error. The trial court erred in its assessment of the proof necessary to establish an occupational disease as defined by the workers' compensation statute.

Fifth Assignment of Error. The trial court erred in limiting appellant's discovery on the following matters:

A. Failure to order suspension of the appellee's "retention of records policy" so as to prevent destruction of relevant documents.

B. Limitation on production of exposure records, air sample tests, and ventilation records to only those records taken in the laboratories rather than the production areas.

C. Failure to order appellee to release patent information for materials manufactured during David Valentine's employment.

D. Denial of appellant's request for production of e-mail communications and any other written communications from the appellee's research and development department in Pennsylvania to appellee's industrial hygienists.

E. Denial of appellant's request for review of OSHA and EPA records pertaining to the operation of appellee's Circleville plant.

F. Failure of the court to order the completion of depositions of appellee's industrial hygienists, Lewis Jordan and Nick Cleary, to answer questions on the presence of a risk of cancer from chemicals in the appellee's workplace.

G. Failure of the court to order the release of the ENSR computerized data and imposition of a ten-day time limit for the appellant to commit to full payment of unspecified expenses.

H. Failure of the court to require the appellee to provide affidavits that identify the appropriate individuals with the corporation that carried out discovery searches and to document what efforts have been taken to find the documents ordered released.

I

**{¶ 8}** Appellant directs her first four assignments of error to the trial court's decision granting appellee summary judgment. First, she asserts that the trial court erred by failing to consider her three experts' affidavits when ruling on appellee's summary judgment motion. Second, appellant contends that the trial court erred by concluding that Dr. Miner's and Dr. Newton's testimony regarding the proximate cause of Valentine's brain tumor was not reliable and, therefore, inadmissible. Appellant argues that the court misinterpreted Evid.R. 702 and the evidence she submitted. Third, appellant argues that the trial court erred by concluding that her experts failed to establish general causation linking the exposure to carcinogens in the workplace to the development of brain cancer. Fourth, appellant contends that the trial court erred by determining that appellant was required to establish causation between specifically identified chemicals and brain tumors to a degree of medical or scientific certainty.

**{¶ 9}** As a prelude to addressing appellant's contention that the trial court applied an improper legal standard of causation, we set forth some general principles governing summary judgment proceedings and the workers' compensation statutes.

A. Summary Judgment Standard

**{¶ 10}** We review a trial court's decision to grant summary judgment on a de novo basis. See, e.g., *Grafton v. Ohio Edison Co.* (1996), 77 Ohio St.3d 102, 105, 671 N.E.2d 241. Accordingly, we conduct an independent review of the record and afford no deference to the trial court's determination. See, e.g., *Brown v. Scioto Cty. Bd. of Commrs.* (1993), 87 Ohio App.3d 704, 711, 622 N.E.2d 1153. Under Civ.R. 56(C), summary judgment is appropriate when (1) no genuine issue as to any material fact remains to be litigated, (2) the moving party is entitled to judgment as a matter of law, and (3) it appears from the evidence, when viewed most strongly in favor of the nonmoving party, that reasonable minds can come to a conclusion only in favor of the moving party. See, e.g., *Grafton*, supra. The burden of showing that no genuine issue exists as to any material fact falls upon the moving party. See, e.g., *Mitseff v. Wheeler* (1988), 38 Ohio St.3d 112, 115, 526 N.E.2d 798. If the moving party satisfies this burden, "the nonmoving party then has a reciprocal burden outlined in Civ.R. 56(E) to set forth specific facts showing that there is a genuine issue for

trial, and if the nonmovant does not so respond, summary judgment, if appropriate, shall be entered against the nonmoving party." *Kulch v. Structural Fibers, Inc.* (1997), 78 Ohio St.3d 134, 145, 677 N.E.2d 308, citing *Dresher v. Burt* (1996), 75 Ohio St.3d 280, 295, 662 N.E.2d 264.

B. Workers' Compensation

**{¶ 11}** Every employee who is injured or contracts an occupational disease in the course of employment is entitled to receive compensation under R.C. 4123.54. Courts must liberally construe the workers' compensation laws in favor of employees. R.C. 4123.95; *Bailey v. Republic Engineered Steels, Inc.* (2001), 91 Ohio St.3d 38, 40, 741 N.E.2d 121. In *Bailey*, the court explained that liberal construction of the workers' compensation laws require courts to adopt "the most comprehensive meaning of the statutory terms." Id. The court stated: "A liberal construction has been defined as giving 'generously all that the statute authorizes,' and 'adopting the most comprehensive meaning of the statutory terms in order to accomplish the aims of the Act and to advance its purpose, with all reasonable doubts resolved in favor of the applicability of the statute to the particular case. Interpretation and construction should not result in a decision so technical or narrow as to defeat the compensatory objective of the Act.' Fulton, Ohio Workers'

Compensation Law (2 Ed.1998) 9, Section 1.7." *Bailey*, 91 Ohio St.3d at 40.

**{¶ 12}** Although a court must liberally construe the workers' compensation laws in favor of the injured employee, a court may not "'read into the statute something which cannot reasonably be implied from the language of the statute.'" *Phillips v. Borg-Warner Corp.* (1972), 32 Ohio St.2d 266, 268, 291 N.E.2d 736, quoting *Szekely v. Young* (1963), 174 Ohio St. 213, 188 N.E.2d 424, paragraph two of the syllabus.

**{¶ 13}** R.C. 4123.01(F) defines an "occupational disease" as one that satisfies the following three elements: "(1) the disease is contracted in the course of employment; (2) the disease is peculiar to the claimant's employment by its causes and the characteristics of its manifestation or the conditions of the employment result in a hazard which distinguishes the employment in character from employment generally; and (3) the employment creates a risk of contracting the disease in a greater degree and in a different manner than in the public generally." See, also, *State ex rel. Ohio Bell Tel. Co. v. Krise* (1975), 42 Ohio St.2d 247, 327 N.E.2d 756, syllabus.

C. Proximate Cause

**{¶ 14}** Appellant contends that the liberal nature of workers' compensation laws requires us to lower the standard for admitting expert testimony or to lower the standard for proving

proximate cause. Regrettably, we cannot agree. While workers' compensation statutes should be liberally construed in favor of the injured worker, this does not mean we are free to ignore the legal principles that control the admissibility of expert testimony and the pronouncements of the Supreme Court of Ohio on proximate cause.

**{¶ 15}** In order to demonstrate that the employee contracted the disease while in the course of employment, the employee must prove that the occupational disease proximately resulted from employment. See *State ex rel. Ohio Bell Tel. Co. v. Krise*, 42 Ohio St.2d at 254, 327 N.E.2d 756 ("the basic subject matter [of the first element of an occupational-disease claim] is causation"); see, also, *Hutchinson v. Ohio Ferro Alloys Corp.* (1994), 70 Ohio St.3d 50, 52, 636 N.E.2d 316 ("Proof of the three definitional criteria of 'occupational disease'--causal connection, hazard and risk--creates a compensable claim"); *Cook v. Mayfield* (1989), 45 Ohio St.3d 200, 204, 543 N.E.2d 787; *Fox v. Indus. Comm.* (1955), 162 Ohio St. 569, 576, paragraph one of the syllabus; *Sheeler v. Ohio Bur. of Workers' Comp.* (1994), 99 Ohio App.3d 443, 451, 651 N.E.2d 7 (noting that in *Fox* the court "held that, in order to establish a right to workers' compensation, a claimant must show by a preponderance of the evidence that a direct or proximate causal relationship existed between his employment and the disability sustained").

**{¶ 16}** "The proximate cause of an event is that which in a natural and continuous sequence, unbroken by any new, independent cause, produces that event and without which that event would not have occurred." *Aiken v.* Indus. Comm. (1944), 143 Ohio St. 113, 117, 53 N.E.2d 1018. "The definition of and principles governing * * * the determination of 'proximate cause' in the field of torts are applicable" in workers' compensation cases. *Murphy v. Carrollton Mfg. Co.* (1991), 61 Ohio St.3d 585, 587, 575 N.E.2d 828, citing *Aiken*, 143 Ohio St. 113, 53 N.E.2d 1018, syllabus; and *Oswald v. Connor* (1985), 16 Ohio St.3d 38, 42; see, also, *Click v. S. Ohio Correctional Facility*, 152 Ohio App.3d 560, 2003-Ohio-2208, 789 N.E.2d 643, at ¶8. Thus, we are forced to reject appellant's contention that the trial court improperly applied standard principles of tort law to the context of a worker's compensation claim.

**{¶ 17}** To prove the proximate cause of a medical condition, here, a brain tumor, expert medical testimony ordinarily is necessary. See, e.g., *Darnell v. Eastman* (1970), 23 Ohio St.2d 13, 261 N.E.2d 114, syllabus. Furthermore, to prove that a toxic substance caused the plaintiff's medical condition, the plaintiff must establish both (1) that the toxic substance is capable of causing the condition (general causation) and (2) that the toxic substance in fact caused the plaintiff's medical

condition (specific causation).[1] See, generally, *Downs v. Perstop Components, Inc.* (1999), 126 F.Supp.2d 1090, 1095; *Hall v. Baxter Healthcare Corp.* (D.Ore. 1996), 947 F.Supp. 1387. Expert testimony ordinarily will be required to prove both general and specific causation.

**{¶ 18}** Appellant cites *Olinger v. Pretty Products, Inc.* (Nov. 7, 1997), Coshocton App. No. 96-CA-29, as support for her argument that the burden of proof regarding causation in tort cases is not applicable to an occupational-disease claim in the workers' compensation context. In *Olinger*, the court stated: "Throughout its brief, appellant consistently misstates the law in Ohio concerning the of a [sic] cause of action for recovery of workers' compensation benefits for an occupational disease. Appellant cites cases concerning proof of causation in a tort action. The instant action is not based on tort. An occupational disease is compensable under R.C. 4123.68(BB) where the following criteria exist: (1) the disease is contracted in the course of employment, (2) the disease is peculiar to the claimant's employment by its causes and the characteristics of its manifestation, or the conditions of employment result in a

[1] The plaintiff must show that he was exposed to the toxic substance and that the level of exposure was sufficient to induce the complained-of medical condition (commonly called a "dose-response relationship"). See, generally, Wiley, Expert Witness Update: New Developments in Personal Injury Litigation (2000), Section 1.04, at 18-19, and Section 1.05[C], at 28 ("the dose makes the poison"). Here, PPG asserts that appellant lacks exposure evidence. However, we assume for the sake of argument that appellant has established exposure and a dose-response relationship.

hazard which distinguishes the employment in character from employment generally, and (3) the employment creates a risk of contracting the disease in a greater degree and in a different manner than the public generally."

**{¶ 19}** Based on Ohio's unique statutory scheme, the *Olinger* court went further and rejected the requirement that a claimant must establish both general and specific causation in order to participate in the fund. Unfortunately, *Olinger* is neither persuasive nor controlling in light of the Ohio Supreme Court's pronouncement that the definition and principles governing proximate cause in tort actions are equally applicable in workers' compensation cases. *Murphy,* 61 Ohio St.3d at 587; *Oswald v. Connor*, supra; *Aiken v. Indus. Comm.*, supra. In fact, our research failed to reveal a single Ohio case that cites *Olinger,* with approval or otherwise.

**{¶ 20}** Appellant construes the statutory requirement that a claimant must establish a risk of contracting a disease that is greater that that of the public generally as a liberalized causation standard. We disagree. The requirement dealing with comparative risk is simply a necessary predicate to participation in the worker's compensation system; it is a definitional component of the term "occupational disease" that is intended to ensure that the injury to be compensated is truly workplace-related. The Supreme Court's ruling in *Murphy* negates

the contention that it is intended to abrogate the standard principles of proximate cause that require proof of both general and specific causation within a reasonable degree of expert certainty. While we concede that appellant's argument is appealing, we cannot adopt it in light of *Murphy*.[2] Appellant's fourth assignment of error is meritless.

II. Admissibility of Expert Testimony

**{¶ 21}** Apologetically, we now turn to an extended discussion of expert testimony, which must comply with Evid.R. 702 to be admissible during summary judgment proceedings. "Pursuant to Civ.R. 56(C), a court may not consider any evidence when ruling on a motion for summary judgment unless it conforms with Civ.R. 56." *Douglass v. Salem Community Hosp.*, 153 Ohio App.3d 350, 2003-Ohio-4006, 794 N.E.2d 107, at ¶21. According to Civ.R. 56(E), "[s]upporting and opposing affidavits shall be made on personal knowledge, shall set forth such facts as would be admissible in evidence, and shall show affirmatively that the affiant is competent to testify to the matters stated in the affidavit." Thus, affidavits containing opinions must meet the requirements in the Rules of Evidence governing the

[2] We have rejected the *Olinger* court's position on causation based upon the Supreme Court's pronouncement in *Murphy v. Carrollton Mfg. Co.*, supra. However, *Olinger's* conclusion that strict application of general and specific causation is not warranted in worker's compensation cases arguably seems reasonable in light of the remedial purpose of the statutes. We encourage certification of the conflict on this issue to the Supreme Court for final resolution.

admissibility of opinions. See *Tomlinson v. Cincinnati* (1983), 4 Ohio St.3d 66, 446 N.E.2d 454, paragraph one of the syllabus; see, also, *Douglass*.

**{¶ 22}** The trial court has broad discretion in determining the admissibility of expert testimony, and we may reverse only if the trial court abused its discretion. See *Kumho Tire Co. v. Carmichael* (1999), 526 U.S. 137, 152-153, 119 S.Ct. 1167, 143 L.Ed.2d 238; see, also, *Miller v. Bike Athletic Co.* (1998), 80 Ohio St.3d 607, 616, 687 N.E.2d 735. "Abuse of discretion" implies that a court acted in "an unreasonable, arbitrary, or unconscionable manner." See, e.g., *State ex rel. Sartini v. Yost*, 96 Ohio St.3d 37, 2002-Ohio-3317, 770 N.E.2d 584, at ¶21; *State v. Herring* (2002), 94 Ohio St.3d 246, 255, 762 N.E.2d 940; *Blakemore v. Blakemore* (1983), 5 Ohio St.3d 217, 219, 450 N.E.2d 1140. An abuse of discretion amounts to more than an error of judgment and instead equates to "perversity of will, passion, prejudice, partiality, or moral delinquency." *Pons v. Ohio State Med. Bd.* (1993), 66 Ohio St.3d 619, 621, 614 N.E.2d 748. Furthermore, when applying the abuse-of-discretion standard, an appellate court may not substitute its judgment for that of the trial court. See, e.g., *Berk v. Matthews* (1990), 53 Ohio St.3d 161, 169, 559 N.E.2d 1301.

**{¶ 23}** In general, courts should admit expert testimony whenever it is relevant and satisfies Evid.R. 702. *State v.*

*Nemeth* (1998), 82 Ohio St.3d 202, 207, 694 N.E.2d 1332; see, also, *State v. Williams* (1983), 4 Ohio St.3d 53,58. Thus, the trial judge must perform a "gatekeeping" role to ensure that expert testimony is sufficiently (a) relevant and (b) reliable to justify its submission to the trier of fact. See *Kumho Tire*, 526 U.S. at 152; *Daubert v. Merrell Dow Pharmaceuticals, Inc.* (1993), 509 U.S. 579, 589, 113 S.Ct. 2786, 125 L.Ed.2d 469; *Nemeth*, 82 Ohio St.3d at 211; *Douglass*, 153 Ohio App.3d 350, 2003-Ohio-4006, 794 N.E.2d 107, at ¶32.

**{¶ 24}** In performing its gatekeeping function, the trial court's starting point should be Evid.R. 702, which provides that a witness may testify as an expert if all of the following apply: "(A) The witness' testimony either relates to matters beyond the knowledge or experience possessed by lay persons or dispels a misconception common among lay persons; (B) The witness is qualified as an expert by specialized knowledge, skill, experience, training, or education regarding the subject matter of the testimony; (C) The witness' testimony is based on reliable, scientific, technical, or other specialized information. To the extent that the testimony reports the result of a procedure, test, or experiment, the testimony is reliable only if all of the following apply: (1) The theory upon which the procedure, test, or experiment is based is objectively verifiable or is validly derived from widely accepted knowledge,

facts, or principles; (2) The design of the procedure, test, or experiment reliably implements the theory; (3) The particular procedure, test, or experiment was conducted in a way that will yield an accurate result."

**{¶ 25}** Here the issue is whether appellant's experts' testimony "is based on *reliable* scientific, technical, or other specialized information." (Emphasis added.) Evid.R. 702(C). In general terms, the reliability of an expert's opinion depends upon (1) the validity of the underlying theory, (2) the validity of the technique used to apply that theory, and (3) the proper application of the technique on a particular occasion. In *Daubert* the United States Supreme Court identified a series of specific reliability inquiries that apply in the context of the "hard" or quantitative sciences. These factors include (1) whether a theory or technique can be and has been tested, (2) known error rates, (3) peer review and publication, and (4) general acceptance in the field.

**{¶26}** The court made it clear in *Kumho Tire Co.* that the reliability analysis adopted in *Daubert* for scientific experts also applied to experts with other types of technical or specialized knowledge. But it is critical to realize that the analysis of reliability is flexible and its indicators may vary from discipline to discipline. *Daubert*, 509 U.S. at 593; see, also, *Moore v. Ashland Chem., Inc.* (C.A.5 1997), 126 F.3d 679,

at 686-688. Thus, the court should proceed in a two-step process that first identifies the indicators of reliability that are appropriate for the discipline involved and then applies them. In this instance, we will focus primarily on general acceptance, testing, known error rates, and "fit." For our purposes we assume without deciding that the studies cited by appellant's experts meet the peer-review criterion.

**{¶27}** In order to determine reliability, a court must assess whether the reasoning or methodology underlying the testimony is valid. *Miller*, 80 Ohio St.3d at 611, citing *Daubert*, 509 U.S. at 592-593. Thus, an expert may not base an opinion upon "subjective belief or unsupported speculation." *Daubert*, 509 U.S. at 590; see, also, *State v. Hurst* (Mar. 7, 2000), Franklin App. No. 98AP-1549. Instead, the expert's opinion must be based on methods and procedures that meet the level of intellectual rigor demanded by the relevant discipline. See In re Paoli (C.A.3, 1994), 35 F.3d 717, 742, citing *Daubert*, 509 U.S. at 590. The "[p]roposed testimony must be supported by appropriate validation--i.e., 'good grounds,' based on what is known." *Daubert*, 509 U.S. at 590. And "where such testimony's factual basis, data, principles, methods, or their application are called sufficiently into question, * * * the trial judge must determine whether the testimony has a 'reliable basis in the knowledge and experience of [the relevant] discipline.'" *Kumho*,

526 U.S. 137, quoting *Daubert*, 509 U.S. at 592; see, also, Daniel J. Capra, The *Daubert* Puzzle (1998) 32 Ga.L.Rev. 699, 705 ("In deciding the question of admissibility, trial judges must consider the degree to which the accuracy of scientific information has been established. The less certain the scientific community is about information, the less willing courts should be to receive it"). In other words, "[s]cientific evidence and expert testimony must have a traceable, analytical basis in objective fact before it may be considered on summary judgment." *Bragdon v. Abbott* (1998), 524 U.S. 624, 653, 118 S.Ct. 2196, 141 L.Ed.2d 540; see, also, *Gen. Elec. Co. v. Joiner* (1997), 522 U.S. 136, 144-146, 118 S.Ct. 512, 139 L.Ed.2d 508. However, "[t]he grounds for the expert's opinion merely have to be good[;] they do not have to be perfect." *Paoli*, 35 F.3d at 744.

**{¶28}** A court resolving a reliability question should consider the "principles and methods" the expert used "in reaching his or her conclusions, rather than trying to determine whether the conclusions themselves are correct or credible." *Nemeth*, 82 Ohio St.3d at 210; see, also, *Miller*, 80 Ohio St.3d 607, paragraph one of the syllabus. As the *Daubert* court stated, in assessing reliability, "[t]he focus * * * must [generally] be * * * on principles and methodology, not on the conclusions that they generate." *Daubert*, 509 U.S. at 595.

**{¶29}** A trial court may not, therefore, exclude expert testimony simply because it disagrees with the expert's conclusions. Instead, if the expert followed methods and principles deemed valid by the discipline to reach his opinion, the court should allow the testimony. See *Paoli*, 35 F.3d at 742 ("an expert's testimony is admissible so long as the process or technique the expert used in formulating the opinion is reliable").[3] The traditional adversary process is then capable of weeding out those shaky opinions. See *Daubert*, 509 U.S. at 596.

**{¶30}** In addition to being scientifically or technically reliable, expert testimony also must "fit" the case at hand; that is, the testimony must be "'relevant to the task at hand' in that it logically advances a material aspect of the proposing party's case." *Daubert*, 509 U.S. at 597; see, also, *Hall v. Baxter Healthcare Corp.,* 947 F.Supp. at 1396; *Moore v. Ashland Chem. Inc.* (C.A.5, 1998), 151 F.3d 269, 275 ("Rule 104(a) requires the judge to conduct preliminary fact-finding and to make a 'preliminary assessment of whether the reasoning or methodology underlying the testimony * * * properly can be applied to the facts in issue,'" quoting *Daubert*, 509 U.S. at 592-593). Thus, "admissibility depends in part on 'the

[3] Because of the lack of Ohio cases addressing the precise issue here (i.e., the admissibility of expert testimony that a toxic substance proximately caused the plaintiff's condition), we look to the federal courts for guidance.

proffered connection between the scientific research or test result to be presented and particular disputed factual issues in the case.' *Downing*, 753 F.2d at 1237." *Paoli*, 35 F.3d at 743.

**{¶31}** In *Cavallo v. Star Ent.* (E.D.Va.1995), 892 F.Supp. 756, 761, the court gave this explanation of the requirement of fit: "The distinction between 'scientific validity' and 'fit' is not always clear and the two inquiries may overlap in a particular case. For instance, there may be times where an expert relies on published literature and widely accepted, tested theories in forming her opinion, and her ultimate conclusion is clearly relevant to an issue in the case. Yet, if those published theories and studies purport to prove XYZ, and from them, the expert concludes ABC, it may be that the expert's reasoning process itself is not scientifically valid. Put another way, there may be a lack of 'fit' between the tested theories relied upon and the ultimate conclusion reached." Accordingly, courts have an obligation to ensure that there is a valid link between the sources or studies the expert consulted and the conclusion the expert reached. *Cavallo*, 892 F.Supp. at 762. "[A] determination regarding the scientific validity of a particular theory requires not only an examination of the trustworthiness of the tested principles on which the expert opinion rests, but also an analysis of the reliability of an expert's *application* of the tested princip[les] to the

particular set of facts at issue." (Emphasis sic.) *Cavallo*, 892 F.Supp. at 762-763.

**{¶32}** Thus, an expert's opinion would not fit if sources relied upon by the expert did not actually support the expert's opinion. As the *Paoli* court more specifically explained: "[A]nimal studies may be methodologically acceptable to show that chemical X increased the risk of cancer in animals, but they may not be methodologically acceptable to show that chemical X increases the risk of cancer in humans. *Daubert* explains that ' "[F]it" is not always obvious, and scientific validity for one purpose is not necessarily scientific validity for other, unrelated purposes.' Id. [509 U.S.] at 591. Thus, even if an expert's proposed testimony constitutes scientific knowledge, his or her testimony will be excluded if it is not scientific knowledge for purposes of the case. 'Rule 702's "helpfulness" standard requires a valid scientific connection to the pertinent inquiry as a precondition to admissibility.' Id. For example, in order for animal studies to be admissible to prove causation in humans, there must be good grounds to extrapolate from animals to humans, just as the methodology of the studies must constitute good grounds to reach conclusions about the animals themselves. Thus, the requirement of reliability, or 'good grounds,' extends to each step in an expert's analysis all the way through the step that connects the

work of the expert to the particular case." *Paoli*, 35 F.3d at 743.

**{¶33}** In cases alleging that chemical exposure caused a plaintiff to contract cancer, the question whether the expert's testimony regarding general causation is reliable, or fits, looms large. This is so because in most cases, the only thing that scientists know for certain about the causes of cancer is the limited nature of their knowledge. See Michael D. Green, Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of Agent Orange and Bendectin Litigation (1992), 86 N.W.U.L.Rev. 643, 644, quoting *Environmental Defense Fund v. Environmental Protection Agency* (C.A.D.C.1978), 598 F.2d 62, 89. Ascertaining the reliability and fit of such testimony can be difficult because of the very fact that science has demonstrated so little as to the causes of cancer (with certain rare exceptions, of course, such as cigarette smoking and lung cancer). As Green writes: "Ideally, to demonstrate that a given agent was a necessary link in the causal chain that led to an individual's disease, one would trace each of the steps in the biology of the development of the disease, including the essential role played by the agent. To state this ideal is to recognize its futility, at least given the current state of

scientific affairs. * * * Because the biological mechanisms of most diseases are understood marginally at best, other devices are necessary to attempt causal attributions. In the absence of direct evidence, scientific methods that permit causal inference—the essence of science is to permit generalizations from observed phenomena—are employed." (Footnotes omitted.) Id. at 644-645.

**{¶34}** Nonetheless, in attempting to show that a chemical substance can cause a particular medical condition (general causation), the expert ideally proceeds in "a stepwise fashion." Federal Judicial Center, Reference Manual on Scientific Evidence (2000) 468. "In the first step the physician must establish the characteristics of the medical condition. Second, he or she carefully defines the nature and amount of the environmental exposure. The third step is to demonstrate that the medical and scientific literature provides evidence that in some circumstances the exposure under consideration can cause the outcome under consideration. This step is synonymous with establishment of general causation. As part of this step, the clinician attempts to establish the relationship between dose and response, including whether thresholds exist, ultimately defining the clinical toxicology of the exposure. The fourth step is to apply this general knowledge to the specific

circumstances of the case at hand, incorporating the specifics of exposure, mitigating or exacerbating influences, individual susceptibilities, competing or synergistic causes, and any other relevant data." (Footnotes omitted.) Id. at 468-470.

**{¶35}** Because of the limitations of hard scientific knowledge, especially about the causes of cancer, experts commonly extrapolate from existing data. Extrapolation is a valid expert technique when properly performed and explained. "But nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered." *Gen. Elec. Co. v. Joiner* (1997), 522 U.S. 136, 146, 118 S.Ct. 512, 139 L.Ed.2d 508. *Daubert* and its progeny require a court to examine the rationale and methods behind the extrapolation to determine whether it is scientifically valid or whether the analytical gap is too wide. See *Hall*, 947 F.Supp. at 1400 ("'*Daubert* * * * not only allows, but requires, courts to determine whether an expert's extrapolations from underlying studies or data are proper, or whether the expert has committed scientific or mathematical errors'"), quoting David E. Bernstein, The Admissibility of Scientific Evidence After

*Daubert v. Merrell Dow Pharmaceuticals, Inc.* (1999), 15 Cardozo L.Rev. 2139, 2165-2166.

**{¶36}** Several types of improper extrapolation commonly appear, "including extrapolating (1) from a structure analysis for similar compounds, (2) that a substance that causes one type of harm also causes a different type of harm, (3) upon the basis of methodology that is transposed from one area of inquiry to a completely different one, (4) from epidemiological studies with different exposures, and (5) when data regarding the plaintiff's exposure is unknown." (Footnotes omitted.) Judge Harvey Brown, Eight Gates for Expert Witnesses (1991), 36 Hous.L.Rev. 743 at 811, citing Capra, 32 Ga.L.Rev. 699,supra.

## III. The Experts' Opinions[4]

**{¶37}** In this case there is no direct scientific evidence that any particular chemical or group of chemicals to which Valentine was exposed caused his glioblastoma multiforme. The plaintiff's experts agree that the only scientifically proven cause of brain tumors is ionizing radiation, a factor that is not applicable in this case. Thus, in reaching their opinions, appellant's experts have extrapolated from other scientific data.

### A. Epidemiological Studies

[4] See Appendix One for a summary of the evidence that the parties submitted.

**{¶38}** Dr. Newton, Dr. Miner, and Mr. Brusk have extrapolated from epidemiological studies, which Green characterizes as follows: "The most desirable evidence is epidemiologic, because it can best be generalized to support inferences about the effect of an agent in causing disease in humans. Epidemiology studies the causes of disease in humans as inferred from observation of humans. Epidemiologic studies are conducted on groups of individuals, i.e., a sample, to isolate and determine the effect of a given agent or factor on the incidence of disease in the population as a whole. These studies entail a comparison of the incidence of disease in a population exposed to the agent being examined with the incidence of disease in an otherwise similar, but unexposed, population, i.e., the control. When epidemiologists study agents that are thought of as toxins—cigarettes, asbestos, drugs, and chemicals—the results of their study may be helpful to the legal system in assessing causation. Because epidemiology is conducted on human populations, it has greater generalizability to those outside the population studied than other toxicological methods, such as animal studies." Green, supra, 86 N.W.U.L.Rev. at 646.

**{¶39}** While epidemiological studies can be "powerful evidence of causation," their absence "is not fatal to a plaintiff's case." *Rider v. Sandoz Pharmaceuticals Corp.* (C.A.11, 2002), 295 F.3d 1194, 1198. "Epidemiological studies

are merely a tool, not a panacea, for finding toxic causation," Green, supra, 86 N.W.U.L.Rev. at 699, and "[a]t best, epidemiology assesses the likelihood that the agent caused a specific individual disease," id. at 647.

**{¶40}** However, extrapolating from epidemiological studies may not always be proper. See, generally, *Joiner*, 522 U.S. 136.[5] "The epidemiological studies must correspond closely enough with the plaintiff's exposure and injury, and they must find a statistically significant connection between the particular substance and the injury alleged by the plaintiff." Capra, 32 Ga.L.Rev. 719; see, also, *Christophersen v. Allied-Signal Corp.* (C.A.5, 1991), 939 F.2d 1106, 1115-1116 (stating that experts may not rely on studies that do not address the types of diseases at issue); *Allen v. Pennsylvania Eng. Corp.* (C.A.5, 1996), 102 F.3d 194, 197 (studies suggesting that chemical exposure causes lymphatic and hematopoietic cancer are not probative to cause of brain cancer); *Schmaltz v. Norfolk & W. Ry. Co.* (N.D.Ill.1995), 878 F.Supp. 1119, 1122.

**{¶41}** The existence of an epidemiological study alone does not make expert testimony fit a plaintiff's case. "[A] claimant must do more than simply rely on epidemiological studies that show a substantially elevated risk. A claimant must show that

[5] See Appendix Three for a summary of the Joiner court's analysis of improper extrapolation from four studies.

he or she is similar to those in the studies. This would include proof that the injured person was exposed to the same substance, that the exposure or dose levels were comparable to or greater than those in the studies, that the exposure occurred before the onset of injury, and that the timing of the onset of injury was consistent with that experienced by those in the study." *Merrell Dow Pharmaceuticals, Inc. v. Havner*, (1997), 953 S.W.2d at 720; see, also, Capra, 32 Ga.L.Rev. at 726 ("a valid study finding a connection between a substance and an injury will fail the 'fit' requirement if the plaintiff's exposure to the substance is materially different from the exposures considered in the study"); *Cavallo*, 892 F.Supp. at 766 ("While Rule 702 does not necessarily mandate that the expert find a study linking the exact chemical at the exact exposure levels with the exact illnesses at issue, it does require that the expert demonstrate a scientifically valid basis for projecting the findings of a study identifying a different chemical-illness relationship to the proffered causal theory").

**{¶42}** In reaching their causation opinions here, appellant's experts relied on epidemiological studies "suggestive" of a link between various chemical exposures and glioblastoma multiforme. However, none of the epidemiological studies concerned the same industry in which Valentine worked, and, more important, none of the studies could identify a chemical or group of chemicals that

caused the brain tumor excess. Thus, extrapolation produced unreliable results because none of the epidemiological studies the appellant's experts relied upon could conclusively identify the cause of the brain tumor excess. See *Bragdon v. Abbott* (1998), 524 U.S. 624, 653, 118 S.Ct. 2196 ("The study on which petitioner relied was inconclusive, however, determining only that '[f]urther work is required to determine whether such a risk exists,'" quoting Johnson & Robinson, Human Immunodeficiency Virus-l (HIV-1) in the Vapors of Surgical Power Instruments (1991), 33 J. of Med. Virology 47); see, also, *Allen v. Pennsylvania Eng. Corp.* (C.A.5, 1996), 102 F.3d 194, 197 (epidemiological studies that "suggest" a link between chemical exposure and brain cancer are not statistically significant so as to lend credence to expert causation opinion). Because appellant's experts relied on studies that do not support the experts' conclusions, those opinions are not scientifically reliable. These studies fall far short of proving their hypothesis. They are a starting point for further research, not scientific proof of causation. To the extent that the experts based their opinions upon and extrapolated from the epidemiological studies, the trial court did not abuse its discretion in finding them inadmissible.

B. Animal Studies

**{¶43}** Appellant's experts also stated that they relied upon animal studies. "While it is quite true that animal data can be important indicia of human health effects, direct transference from one to the other may or may not be possible, depending, in part, on the type of effect and the dosage used to achieve that effect." Wiley, supra fn.1, Section 1.04[C], at 21. "[T]he more closely those specific organ system effects are known to mimic effects in people and the closer the dosage creating the effect in the animal is to the human dosage, the better the argument for transferability." Id., Section 1.04[D], at 21-22.

**{¶44}** In *Joiner*, the court considered the reliability of an expert's opinion when the expert relied on animal studies to reach a causation opinion. The court noted: "The studies involved infant mice that had developed cancer after being exposed to PCB's. The infant mice in the studies had had massive doses of PCB's injected directly into their peritoneums or stomachs. Joiner was an adult human being whose alleged exposure to PCB's was far less than the exposure in the animal studies. The PCB's were injected into the mice in a highly concentrated form. The fluid with which Joiner had come into contact generally had a much smaller PCB concentration of between 0-to-500 parts per million. The cancer that these mice developed was alveologenic adenomas; Joiner had developed small-cell carcinomas. No study demonstrated that adult mice

developed cancer after being exposed to PCB's. One of the experts admitted that no study had demonstrated that PCB's lead to cancer in any other species." *Joiner*, 522 U.S. at 144.

**{¶45}** The *Joiner* court concluded that "[t]he studies were so dissimilar to the facts presented in this litigation that it was not an abuse of discretion for the District Court to have rejected the experts' reliance on them." Id.

**{¶46}** Here, appellant's experts have not sufficiently explained what enables them to extrapolate from the animal studies to humans. Moreover, the chemical exposures in the animal studies did not cause brain tumors to develop across species. See *Allen*, 102 F.3d at 197 (finding expert's reliance on animal studies in forming causation opinion unreliable when chemical exposure did not consistently result in brain tumors across species). Without further explanation of the logic and reasoning behind extrapolating from the animal studies to humans, the trial court was well within its discretion to exclude appellant's experts' testimony.

C. Experience

**{¶47}** Dr. Miner stated that he reached his opinion based upon his many years of treating patients with brain tumors and upon his experience in the Houston, Texas area, where several petroleum workers contracted brain tumors. Case reports in and of themselves are not invalid. However, they best describe

associations, not causation. The mere coincidence of exposure and the appearance of a disease is never sufficient to prove causation in an individual instance. Susan R. Poulter, Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation? (1992), 7 High Tech.L.J. 189 at 216. Even clustering of a disease can occur by random chance or other causes. Id. In other words, "[t]hey reflect only reported data, not scientific methodology." *Rider*, 295 F.3d at 1199; see, also, *Haggerty v. Upjohn Co.* (S.D.Fla 1996), 950 F.Supp. 1160, 1165 ("while case reports may provide anecdotal support, they are no substitute for a scientifically designed and conducted inquiry"); *Casey v. Ohio Med. Prods.* (N.D.Cal. 1995), 877 F.Supp. 1380, 1385 ("case reports are not reliable scientific evidence of causation, because they simply described reported phenomena without comparison to the rate at which the phenomena occur in the general population or in a defined control group; do not isolate and exclude potentially alternative causes; and do not investigate or explain the mechanism of causation"). Importantly, there is no means of determining the rate of error involved in anecdotal reports. In short, these opinions cannot be tested. Moreover, experience alone does not make an otherwise unfounded conclusion scientifically reliable. See, generally, David L. Faigman et al., How Good Is Good Enough?

Expert Evidence Under *Daubert* and Kumho (2000), 50 Case W.Res.L.Rev. 645, 657.

**{¶48}** Therefore, the trial court did not abuse its discretion in finding that Dr. Miner's reliance on his experience, including his observation of workers in the petroleum industry, does not sufficiently demonstrate the reliability of his opinion.

D. Governmental Regulatory Policy

**{¶49}** In reaching his causation opinion, Brusk also relied upon various regulatory agencies' classification of some of the chemicals as carcinogenic. That a regulatory agency chooses to classify a chemical as carcinogenic does not give credence to an expert's opinion that the chemical caused a plaintiff's specific type of cancer. Two problems inhere in inferring causation from such a classification. First, the regulatory body's classification of the chemical does not determine admissibility of expert testimony on the subject. Second, to classify a chemical as carcinogenic is to say that it causes cancer. What type of cancer is an unresolved question. It is generally well understood that carcinogens cause specific types of cancer. Thus, to state that a chemical is carcinogenic answers only half of the question. See *Allen*, 102 F.3d at 196 ("the fact that [ethylene oxide] has been classified as a carcinogen by agencies responsible for public health regulations is not probative of

the question whether [the plaintiff's] brain cancer was caused by [this] exposure").

**{¶50}** Moreover, "substances are regulated because of what they might do at given levels, not because of what they will do." Wiley, supra fn.1, Section 1.07, at 33. The fact of regulation does not imply scientific certainty. It may suggest a decision to err on the side of safety as a matter of regulatory policy rather than the existence of scientific fact or knowledge. See id., Section 1.07, at 34. "A regulatory agency such as the FDA may choose to err on the side of caution. Courts, however, are required by the *Daubert* trilogy to engage in objective review of evidence to determine whether it has sufficient scientific basis to be considered reliable." *Rider*, 295 F.3d at 1201. The mere fact that substances to which Valentine was exposed may be listed as carcinogenic does not provide reliable evidence that they are capable of causing brain cancer, generally or specifically, in Valentine's case. We have no reason to conclude that the trial court erred on this basis.

E. Differential Diagnosis

**{¶51}** Appellant's medical experts have also asserted that they reached their conclusions of specific causation after conducting a differential diagnosis of Valentine.

**{¶52}** Differential diagnosis[6] can be a valid expert technique. See *Hardyman v. Norfolk & W. Ry. Co.* (C.A.6, 2001), 243 F.3d 255, 260 (stating that differential diagnosis is one appropriate method for determining causation in an individual); *Westberry v. Gislaved Gummi AB* (C.A.4, 1999), 178 F.3d 257, 262 (stating that differential diagnosis is a standard scientific technique); *Cutlip v. Norfolk S. Corp.,* Lucas App. No. L-02-1051, 2003-Ohio-1862. "Differential diagnosis" is defined as "'[t]he method by which a physician determines what disease process caused a patient's symptoms. The physician considers all relevant potential causes of the symptoms and then eliminates alternative causes based on a physical examination, clinical tests, and a thorough case history.'" *Hardyman*, 243 F.3d at 260, quoting Federal Judicial Center, Reference Manual on Scientific Evidence (1994) 214. "A reliable differential

[6] Some commentators have noted that the legal system's use of "differential diagnosis" to mean the process of eliminating causes is improper. Instead, medical professionals use the term to describe the process of identifying the disease the patient is suffering from. See Reference Manual on Scientific Evidence at 443-444 (footnotes omitted): "[C]ourts have come to use certain medical terms, such as differential diagnosis and differential etiology in ways that differ from their common usage in the medical profession. For example, although environmental and occupational health physicians may use the term 'differential diagnosis' to include the process of determining whether an environmental or occupational exposure caused the patient's disease, most physicians use the term to describe the process of determining which of several diseases is causing a patient's symptoms. Expert witnesses and courts, however, frequently use the term 'differential diagnosis' to describe the process by which causes of the patient's condition are identified, particularly causes external to the patient. Additionally, courts sometimes characterize causal reasoning as 'differential etiology,' a term not used in medical practice, but one that more closely suggests the determination of a cause." The reference manual suggests that a more appropriate term would be "external causation."

diagnosis typically, though not invariably, is performed after 'physical examination, the taking of medical histories, and the review of clinical tests, including laboratory tests,' and generally is accomplished by determining the possible causes for the patient's symptoms and then eliminating each of these potential causes until reaching one that cannot be ruled out or determining which of those that cannot be excluded is the most likely." *Westberry*, 178 F.3d at 262.

**{¶53}** However, differential diagnosis alone does not always establish proximate cause, particularly when general causation evidence is lacking. "The process of differential diagnosis is undoubtedly important to the question of 'specific causation.'" *Cavallo*, 892 F.Supp. at 771. But a valid differential diagnosis presupposes that general causation has been established, i.e., that agent X is capable of causing brain tumors in humans generally. See *Cavallo*, 892 F.Supp. at 771 ("a fundamental assumption underlying [differential diagnosis] is that the final, suspected 'cause' remaining * * * must actually be capable of causing the injury"); see, also, *Hollander v. Sandoz Pharmaceuticals Corp.* (C.A.10, 2002), 289 F.3d 1193, 1210 ("In many of the decisions in which a differential diagnosis has been deemed reliable, the party relying on the diagnosis has offered independently reliable evidence that the allegedly dangerous drug or substance had harmful effects"); *Hall*, 947 F.Supp. at

1413, citing *Cavallo*. Thus, "the expert must 'rule in' the suspected cause as well as 'rule out' other possible causes." *Cavallo*, 892 F.Supp. at 771. Here, we have concluded there is no other evidence to "rule in" Valentine's exposure as a general causation agent. Moreover, "If other possible causes of an injury cannot be ruled out, or at least the probability of their combination to causation minimized, then the 'more likely than not' threshold for proving causation may not be met.'" *Cavallo*, 892 F.Supp. at 771. In other words, the technique is valid where the causes of the disease are known. Differential diagnosis presupposes the causes are known and then proceeds to eliminate them based upon the premise that the absence of other risk factors increases the likelihood that the patient's disease was caused by exposure to the toxic substance. Where the other causes are unknown, there is nothing to eliminate and thus no increase in likelihood of causation by the toxic agent. See Expert Witnessing: Explaining and Understanding Science, Carl Meyer, CRC Press (1999), Chapter 12, Medical and Scientific Evidence of Causation.

**{¶54}** For example, in *Cavallo*, the plaintiff's expert used differential diagnosis to opine that the plaintiff's exposure to jet fuel caused her respiratory problems. However, the plaintiff presented no reliable evidence that jet fuel fumes could, in fact, cause such respiratory problems. See, also,

*Raynor v. Merrell Pharmaceuticals, Inc.* (C.A.D.C.1997), 104 F.3d 1371 (rejecting differential diagnosis when general causation had not been established).

**{¶55}** Compare *Cavallo* with *Westberry*, where the court found the expert's differential diagnosis reliable to prove causation when general causation already had been established. The court noted that "it was undisputed that inhalation of high levels of talc irritates mucous membranes." *Westberry*, 178 F.3d at 264; see, also, *Mattis v. Carlson Elec. Products* (C.A.8, 2002), 295 F.3d 856 (accepting differential diagnosis for causation when evidence also existed regarding general causation).

**{¶56}** Here, the cause of brain tumors is largely unknown (except for ionizing radiation). To state that nothing else caused the brain tumor is contrary to the medical and scientific fact that the cause of brain tumors is unknown. At this point, medical science does not enable physicians and other scientists to pinpoint a cause of brain cancer (except for ionizing radiation). Both Dr. Miner and Dr. Newton readily acknowledged the current status of medical and scientific knowledge in their depositions. Thus, under the circumstances of this case the trial court did not abuse its discretion in concluding that differential diagnosis is not a reliable technique for identifying causation.

## IV. Conclusion

**{¶57}** Consequently, we reluctantly conclude that the trial court did not abuse its discretion by finding appellant's experts' testimony to be unreliable and inadmissible. There is no relevant direct scientific or technical evidence that identifies the agents of general causation for brain tumors in humans. In forming their opinions, the experts have improperly interpreted epidemiological studies, animal studies, and cancer regulatory policy. Causation opinions based upon the differential diagnosis are not reliable in this case because that technique requires both ruling in the suspected agent and ruling out the other causes of the disease; the present state of scientific knowledge on the cause of brain cancer precludes reliability in this context. To the extent appellant relies on Brusk's testimony to establish causation, his testimony is inadmissible for the same reasons as Dr. Miner's and Dr. Newton's testimony. Without her experts' testimony, appellant possesses no evidence of a cause-and-effect relationship between Valentine's brain cancer and his workplace. The mere fact that no other evidence is available does not warrant the admission of clearly unreliable evidence. Thus, we then are compelled to agree with the trial court's decision entering summary judgment in PPG's favor.

**{¶58}** Based upon the foregoing reasons, we overrule appellant's first through fourth assignments of error.

IV

**{¶59}** In her fifth assignment of error, appellant argues that the trial court abused its discretion when ruling on a wide variety of discovery matters, which are summarized in Appendix Two.

**{¶60}** In this case, any error that the trial court may have committed in ruling on discovery matters is harmless. Civ.R. 61 provides: "No error in either the admission or the exclusion of evidence and no error or defect in any ruling or order or in anything done or omitted by the court or by any of the parties is ground for granting a new trial or for setting aside a verdict or for vacating, modifying or otherwise disturbing a judgment or order, unless refusal to take such action appears to the court inconsistent with substantial justice. The court at every stage of the proceeding must disregard any error or defect in the proceeding which does not affect the substantial rights of the parties." See, also, *Siuda v. Howard*, Hamilton App. Nos. C-000656 and C-000687, 2002-Ohio-2292, at ¶21, citing *Meyers v. Hot Bagels Factory, Inc.* (1999), 131 Ohio App.3d 82, 100-101, 721 N.E.2d 1068 ("harmless error is an error that does not affect the substantial rights of the parties").

**{¶61}** "'In determining whether a substantial right of a party has been affected, the reviewing court must decide whether the trier of fact would have reached the same decision, had the

error not occurred.'" *Prakash v. Copley Twp.*, Summit App. No. 21057, 2003-Ohio-642, quoting *Moore v. Univ. of Akron* (Aug. 1, 2001), Summit App. No. 20320. "'Generally, in order to find that substantial justice has been done to an appellant so as to prevent reversal of a judgment for errors occurring at the trial, the reviewing court must not only weigh the prejudicial effect of those errors but also determine that, if those errors had not occurred, the jury or other trier of the facts would probably have made the same decision.' *Hallworth v. Republic Steel Corp.* (1950), 153 Ohio St. 349, 91 N.E.2d 690, paragraph three of the syllabus." *Cappara v. Schibley* (1999), 85 Ohio St.3d 403, 408, 709 N.E.2d 117.

**{¶62}** In this case, had the trial court ruled in appellant's favor regarding the various discovery requests, none of the requested material would have helped appellant establish the reliability of her experts' testimony. Therefore, no prejudice resulted from the court's discovery rulings.

**{¶63}** Accordingly, we overrule appellant's fifth assignment of error and affirm the trial court's judgment.

Judgment affirmed.

ABELE, J., concurs.

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ABELE, Judge, concurring.

**{¶64}** I reluctantly concur in both the judgment and opinion. I believe that the principal opinion accurately sets forth the current state of the law concerning the admissibility of expert witness opinions. I am troubled, however, with the application of the law to the facts of this workers' compensation case.

**{¶65}** Appellant asserts that the trial court improperly applied general tort law principles to this workers' compensation case. The principal opinion rejects this argument, and I agree with this conclusion. Although courts must liberally construe workers' compensation laws in favor of employees (see R.C. 4123.95), proximate cause determinations are made in the same manner in workers' compensation cases as in other general tort actions. See *Murphy v. Carrollton Mfg. Co.* (1991), 61 Ohio St.3d 585, 575 N.E.2d 828.

**{¶66}** The results in this particular case are unfortunate. This case involved Valentine's prolonged exposure to chemical toxins, not a traditional workplace injury that involved machinery or other types of easily recognizable injuries. I fear that the instant case may very well be a situation in which medical science and the law are not currently equipped to adequately resolve this controversy.[7] In light of the fact that

[7] It is indeed interesting to note that Valentine's coworker (Harold McConnaughy) also had "a similar exposure pattern to neurocarcinogens and also died of a glioblastoma multiforme" (i.e. a rare form of brain cancer that accounts for two to three percent of all new cancers diagnosed in the

this matter is in the context of a workers' compensation case, I do not believe that a relaxed causation standard is unworkable or far fetched. This, however, is a matter that is beyond our reach and must be addressed by the Ohio Supreme Court. As an intermediate appellate court, we are obligated to follow Ohio Supreme Court decisions.

**{¶67}** I recognize that *Daubert* and *Kumho Tire* stand for the proposition that "junk science" has no place in our courtrooms. I, however, question whether the expert opinions elicited in this case should be placed in that category.

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APPENDIX ONE

**{¶68}** The evidence relevant to the trial court's summary judgment decision follows.

**{¶69}** Appellant retained three expert witnesses: Valentine's treating physicians at OSU hospital, Dr. Newton and Dr. Miner, and industrial hygienist Norman Brusk. Drs. Newton and Miner opined that Valentine's work environment, specifically his exposure to various toxic substances, proximately caused his brain tumor.

**{¶70}** Appellee retained two expert witnesses: Dr. Darrell A. Bigner and Charles R. Buncher, a professor of Biostatistics

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United States each year). See Dr. Newton's August 29, 2000 letter to appellant's counsel, Appendix I, ¶ 88.

and Epidemiology in the Department of Environmental Health University of Cincinnati College of Medicine who testified favorably for PPG.

A. APPELLANT'S EXPERTS

1. Dr. Miner

**{¶71}** Dr. Miner conceded that medical science has not identified a specific etiology for glioblastoma multiforme, with the exception of ionizing radiation, and that medical science has not established a definite link between a specific chemical and glioblastoma. Nevertheless, Dr. Miner opined that Valentine was at a greater risk of developing a malignant brain tumor than the general population, that he had an increased risk of developing malignant brain tumor secondary to the exposure to the chemicals at PPG, and that his tumor was related to his employment and exposure to chemicals. In an affidavit, Dr. Miner asserted: "It is my professional opinion, based on a reasonable medical probability, that Mr. Valentine's brain tumor was directly and proximately caused by his exposure to chemical toxins in his workplace, especially in his employment for ten years as a laboratory technician at PPG, and that his death was directly caused by that exposure." He further explained: "It is my professional opinion that it is not possible to identify a specific chemical that by itself would have resulted in Mr. Valentine's brain tumor. Indeed, it is more likely that it was

the long exposure over years to a combination of chemicals that was the cause of his malignant brain tumor. Since he was exposed for a long period of time to many known carcinogens, it is not possible to identify a single causative agent. There are no laboratory experiments that can duplicate his total exposure to these chemicals. However, other industries have reported the results of people exposed to similar carcinogens and in those reports, the incidence of brain tumors is higher than expected."

**{¶72}** Dr. Miner readily admitted, however, that medical science has not yet proven a known etiology for glioblastoma multiforme. He qualified this statement by explaining: "I believe it's very difficult to prove what would or wouldn't cause brain tumors. One thing that seems to have been well proven is ionizing radiation. I believe there's other things that certainly contribute to formation of tumors, but it's difficult to prove in people that they are the cause of them."

**{¶73}** Dr. Miner stated that he did not agree with PPG's expert, Dr. Bigner, that "the only known cause of primary brain tumors * * * [is] ionizing therapeutic radiation." Dr. Miner believed Dr. Bigner's opinion to be "an overly dogmatic statement; and taken in the context of everything else he says, I would have trouble agreeing with." He explained his disagreement with Dr. Bigner: "Well, after reading [Dr. Bigner's opinion], you conclude that nothing else contributes to

causing---I would come to the conclusion that he thinks that nothing else contributes to the cause of brain tumors besides essentially bad luck, your parents' genes and ionizing radiation, and I don't think that's true."

**{¶74}** He cannot state within a reasonable degree of medical probability which specific chemical caused Valentine's glioblastoma. He cannot state which chemical or group of chemicals caused Valentine's glioblastoma.

**{¶75}** He thinks that benzene, acrylonitriles, ethylene oxide, and toluene can contribute to glioblastoma: "[E]xposure to them increases the chances of people developing brain tumors or glioblastoma multiforme." He believes that exposure to toluene increases the risk of glioblastoma. "[L]ong-term exposure to benzene I believe within a reasonable degree of medical probability enhances the likelihood that you'll develop a glioblastoma."

**{¶76}** Appellant's counsel questioned him: "[W]hen you consider the totality of Mr. Valentine's exposure to the range of chemicals identified in Mr. Brusk's report, this would be exposure both through air contact and dermal contact and under situations where strict safety standards were not being applied and that this exposure to the range of chemicals taking place over approximately a 30-year period, do you have an opinion based on a reasonable medical probability as to whether Mr.

Valentine was exposed to a risk of developing cancer, including brain cancer, that was greater than the public as a whole because of his employment at PPG?"

**{¶77}** Dr. Miner answered: "I think assuming the things that you said are true from what I glean from the Brusk report, that he was at an increased risk of developing a malignant brain tumor than the general population." Dr. Miner opined that Valentine's tumor was related to his employment and his total exposure to various chemicals, not that any one specific chemical or group of chemicals caused the brain tumor.

**{¶78}** To support his opinion, Dr. Miner relied upon (a) epidemiological studies, (b) animal studies, (c) his prior experience treating patients with brain tumors, and (d) differential diagnosis.

a. Epidemiological Studies

**{¶79}** Dr. Miner's review of an epidemiological study from the petrochemical industry led him to believe that benzene increases the likelihood of developing a brain tumor. Dr. Miner explained: "[The whole purpose of the epidemiological study is to] show these people were working and very highly exposed to these chemicals and they had a very high instance of glioblastoma. That's the usual way the epidemiologists make associations. Epidemiologists almost never prove anything. They just make the association so significant that you can't

believe otherwise." In explaining his reliance on epidemiological studies, Dr. Miner stated that "it was very clear that there was a relationship between exposures to some chemicals and the onset of brain tumors." He explained: (1) "A number of epidemiological studies document an increased risk of cancer, including brain cancer * * * , in the occupation of a lab technician/chemist whose employment involved routine handling and exposure to a number of different solvents similar to what Mr. Valentine had"; and (2) "Epidemiological studies from the petroleum industry have documented an increased risk of the development of brain tumors * * * from exposure to solvents, especially benzene."

b. Animal Studies

**{¶80}** Dr. Miner stated that some animal studies have shown certain chemicals, like benzene, capable of producing brain tumors.

c. Prior Experience

**{¶81}** Dr. Miner also stated that he based his opinion upon his past experience treating people diagnosed with brain tumors. When Dr. Miner worked in the Houston, Texas area, he treated at least 50 patients diagnosed with glioblastomas. He asserted: "[I]t's been my experience that people that work around some of the chemicals that I believe that David Valentine was exposed to have an unusually high instance of brain tumors." He stated

that he was "often struck at the frequency of people that worked in the petrochemical industry having glioblastomas."

d. Differential Diagnosis

**{¶82}** Dr. Miner stated that the following facts formed the basis for his opinion: (1) Valentine's personal history is negative for any family history of similar problems, (2) he did not have any exposure to chemical toxins outside of his employment, (3) industrial hygienist Brusk's report, (4) a review of the medical literature, including articles on primary medical and genetic research, animal studies and epidemiological studies, (5) Valentine's exposure to chemicals that are known to cause or suspected to cause changes in human cells that result in cancer, and (6) animal studies identifying acrylonitrile and ethylene oxide as chemicals capable of producing brain tumors in rats.

2. Dr. Newton

**{¶83}** Dr. Newton agrees that the only known cause of glioblastoma multiforme is ionizing radiation. Dr. Newton has treated over 1,000 cases of glioblastoma multiforme, and he has yet to notice any pattern or common links between patients.

**{¶84}** Dr. Newton opined that Valentine contracted the glioblastoma multiforme as a result of the risks he was exposed to at PPG. Dr. Newton believes that there is a suspicious proximate relationship between PPG and Valentine's brain tumor.

Dr. Newton also stated that Valentine's employment as a lab technician and environmental specialist presented a risk of developing brain cancer greater than the risk presented to the general population.

**{¶85}** Dr. Newton stated that he agreed with Dr. Bigner that ionizing radiation is the only proven cause of primary brain tumors, but emphasized that Dr. Bigner discussed proven causes. Dr. Newton stated: "[T]here are many things that could be related that have not been proven yet. They may be part of a multiple causation of cancer, which is typical in most cases. If you really have the cancer, it's often not just one thing, it's numerous things that are involved in the cancer causation. It's not typically a single proximate cause disease."

**{¶86}** Dr. Newton stated that he could not identify the "exact single proximate cause of [Valentine's] glioblastoma, but [he] suspect[ed] there are numerous proximate causes like there typically are in most of these cases." Dr. Newton also explained that he is "suspicious that [Valentine's] exposures over 28 years at PPG were part of that list of potential proximate causes." Dr. Newton stated that he could not name a specific chemical that caused Valentine's brain tumor, but thinks "it might have been generalized exposure to probably more than one of those chemicals over time that could have acted as a proximate cause or one of the proximate causes."

**{¶87}** Dr. Newton stated that he was, within reasonable medical probability, "suspicious that [Valentine's employment] was possibly one proximate cause that contributed to his brain tumor development." Dr. Newton explained that his use of the word "suspicious" meant "[m]ore likely than not." Dr. Newton opined that Valentine's death was more likely than not contracted as a result of risks he was exposed to in his employment. Dr. Newton gave the following reasons underlying his opinion: "Because of such intense constant exposure over a long number of years, which is the way cancer is often thought to develop. With environmental carcinogens, it's often long-term constant exposure to different agents that we feel lends to the initiation and promotion of a tumor."

**{¶88}** In Dr. Newton's August 29, 2000 letter that he sent to appellant's counsel, he more specifically explained his opinion as follows: "The etiology of PBT remains unclear, but most investigators agree it is a combination of familial genetic loading and endogenous metabolic processes and, less importantly, environmental exposures. However, some recent literature would suggest that exposure to environmental agents accounts for 60-80% of sporadic cancers, including PBT. It is important at this point to note that Mr. Valentine did not have a particularly strong family history of cancer, although there was some on his mother's side. Environmental carcinogens are

suspected of acting as promoters, more so than as initiators, in the multistep process of neurocarcinogens and brain tumor development. In humans, cancer development is a long-term process that often evolves over decades. Several types of chemicals have been implicated as neurocarcinogens in animals and humans, including triazenes, hydrazines, vinyl chloride, pesticides, and others. Vinyl chloride, in particular, has been implicated in numerous studies as a potential neurocarcinogen in the work-place. Epidemiological studies are fairly consistent that industrial workers given long-term exposure to the above chemicals have an increased incidence of, and elevated odds ratios for, developing a brain tumor. Mr. Valentine was exposed to many of the above chemicals, as well as many others, over a 30 year career at PPG Industries. Although I agree with Dr. Bigner that the only *proven* exogenous cause of a brain tumor in humans is irradiation, I do not agree with his assessment concerning long-term exposure to neurocarcinogens in the work-place. I feel that long-term exposure to neurocarcinogens can add to the lifetime risk of developing a brain tumor and that they add to the intrinsic risk a person has from familial genetic loading for cancer. Considering that Mr. Valentine did not have a strong family history for cancer and that he was exposed to several types of neurocarcinogens over a 30 year period, I feel it is quite likely that his occupation

contributed to the development of his brain tumor. This is supported by the fact that his co-worker, Harold McConnaughy, also had a similar exposure pattern to neurocarcinogens and also died of a glioblastoma multiforme. I conclude that Mr. Valentine's employment at PPG Industries constituted a risk for developing cancer that was greater than the public as a whole and that the exposure to neurocarcinogens in the workplace contributed to his contraction of the brain tumor."

**{¶89}** In reaching his opinion, Dr. Newton relied upon scientific literature, animal studies, and epidemiological studies.

a. Scientific Literature

**{¶90}** Dr. Newton recognized that some of the chemicals that industrial hygienist Brusk identified in his report are carcinogens or suspected carcinogens (as classified by cancer regulatory agencies), but states that "[n]othing's been proven," meaning that no medical proof exists that any one chemical or any combination of the chemicals causes glioblastoma multiforme.

b. Animal Studies

**{¶91}** Dr. Newton stated that the following chemicals are neurocarcinogens in animals and are looked at as potential or possible causes in humans: hydrazines, vinyl chloride, acrylonitrile, and polycyclic hydrocarbons.

**{¶92}** He stated that the animal studies showing a neurocarcinogenic effect would make one "suspicious for having that same effect in people." He further explained, however, that "we don't know the dosing, we don't know how long they need to be exposed, we don't know the details of any of that stuff because those studies can't be done [in humans]." Thus, Dr. Newton opined that "it's hard to say" whether the animal studies show that a specific chemical proximately causes brain tumors in humans.

c. Epidemiological Studies

**{¶93}** Dr. Newton recognized that "some epidemiological studies * * * suggest increased relative risk compared to the general population for people exposed to [solvents, hydrocarbons, and associated chemicals]." Dr. Newton referred to an article entitled "Occupational Risk Factors of Women in Shanghai, China." The study stated: "Previous studies have suggested that organic solvents are associated with brain tumors, particularly gliomas. Our results provide supporting evidence for that hypothesis."

3. Industrial Hygienist Brusk

**{¶94}** Brusk reviewed Valentine's employment with PPG to determine what chemicals Valentine would have been exposed to. Brusk stated that when Valentine worked in the Quality Control Laboratories, "he had contact with chemicals that are considered

to be potentially carcinogenic." The International Agency for Research on Cancer rates chemicals as carcinogenic, probably carcinogenic, or possible carcinogenic. The American Conference of Governmental Industrial Hygienists rates chemicals as confirmed human carcinogen, suspected human carcinogen, or animal carcinogen. The chemicals included acrylamide (possible carcinogenic/animal carcinogen), acrylonitrile (probably carcinogenic/suspected human carcinogen), benzene (carcinogenic/confirmed human carcinogen), ethyl acrylate (possibly carcinogenic), ethylene oxide (probably carcinogenic/suspected human carcinogen), propylene imine (possibly carcinogenic/animal carcinogen), propylene oxide (probably carcinogenic/animal carcinogen), styrene (possibly carcinogenic/animal carcinogen), toluene (possibly carcinogenic), dioxane (possibly carcinogenic), and polychlorinated biphenyls ("PCBs") (probably carcinogenic). Valentine also had "extensive exposures to additional solvents including toluene, xylene, MIK, MEK, and butyl alcohol. Additional effects of these chemicals is undetermined at this time. Toluene in particular is of concern because this product, as manufactured in the 60s, was known to have significant Benzene contamination."

**{¶95}** Brusk stated: "It is my professional opinion that Mr. Valentine's particular employment as a laboratory technician and

environmental specialist subjected him to a risk of developing cancer from both respiratory and skin contact with the numerous carcinogens identified above that was substantially different from and greater than the risk of developing cancer faced by workers in general and/or the public as a whole." Brusk based his opinion "not only on the specifically identified chemicals, but also on the cumulative effect of Mr. Valentine's long term exposures to many toxic chemicals."

**{¶96}** Brusk opined that Valentine had "unquantifiable, but significant exposures to cancer causing and potentially cancer causing chemicals. His exposures would be from inhalation of vapors and from absorption of chemicals directly though his unprotected skin. He may also have had exposure from the transfer of chemicals from his hands to food or to his mouth by wiping."

**{¶97}** He reviewed the epidemiological study "A General Mortality Study of Production Workers in the Paint and Coatings Manufacturing Industry." He does not believe that it "reflect[s] the situation of Mr. Valentine during his work career at PPG. The study was considerably too broad to isolate the laboratory personnel. They were lumped in, if included at all, with the general work population. Therefore if there were an increase in brain cancers for the laboratory personnel, it would not show up in the study."

**{¶98}** Brusk refers to other epidemiological studies that linked employment and brain cancer: "Case-Control Study of Intracranial Tumors Among Employees at a Petrochemical Research Facility" and "Cancer Incidence Among Employees at a Petrochemical Research Facility." He recognizes that neither study could identify a specific agent, but the study concluded that the "'patterns suggest that the brain cancer excess resulted from occupational exposures.'" He also referred to "The Hazard of a Chemical Laboratory Environment—A Study of the Mortality in Two Cohorts of Swedish Chemists." The study concluded: "'An increase in mortality due to leukemias * * * and possibly brain tumors was observed. It is probable that employment in a chemical laboratory, and particularly in organic chemistry, is associated to some extent with the increase.'" He also referred to a study entitled "The Cancer Mortality Among Swedish Chemists Graduated During Three Decades." That study concluded: "'[T]his study supports the suggestions that chemical exposure is a cause of both leukemic and hematopoietic tumors and brain tumors and thus indicates the occupational hazards in chemical work.'" Brusk also noted that the study "Brain Cancer in Petrochemical Workers: A Case Series Report" concluded, " 'The information available indicates that the number of brain tumors is excessive in a population of this size, and that the tumors are likely to be occupationally

related.'" Brusk also referred to the study "Occupational Risk Factors for Brain Tumors Among Women in Shanghai, China," which concluded that brain cancer "'[r]isks were significantly elevated in occupations thought to entail exposure to organic solvents.'"

**{¶99}** In his affidavit, Brusk stated: "Based on my assessment of Mr. Valentine's workplace, the above studies, my experience as an industrial hygienist as well as a chemist, and the occurrence of clusters of brain cancers in chemical related fields, it is my professional opinion that Mr. Valentine had a higher risk of developing brain cancer because of his position as a laboratory technician than of employees in the general workforce and of the population as a whole."

B. PPG'S EXPERTS

1. Dr. Bigner

**{¶100}** Dr. Bigner challenged appellant's experts' opinions. Relevant quotations from his affidavit follow: "[T]here is no reliable or reproducible scientific evidence for any opinion or conclusion that chemicals or environmental agents other than ionizing (therapeutic) radiation can cause primary brain tumors in humans. There are no peer reviewed medical or scientific reports, studies, or articles that demonstrate chemicals or environmental agents cause primary brain tumors in humans, except ionizing (therapeutic) radiation. There are no

scientific reports or data in the medical or scientific literature that establishes that acrylamide, acrylonitrile, benzene, ethyl acrylate, ethylene oxide, propylene imine, propylene oxide, styrene, toluene-diisocyanate, dioxane, [PCBs], polycyclic aromatic hydrocarbons, toluene, xylene, MIK, MEK and butyl alcohol can cause or contribute to the development of primary brain tumors in humans. There is no study or report in the scientific literature that any of these chemicals can cause mutations or other DNA damage in human brain cells that is not repaired, especially glial cells and astrocytic cells which are the precursor cells of David Valentine's brain tumor. Acrylamide, acrylonitrile, ethylene oxide, propylene imine, polycyclic aromatic hydrocarbons, and hydrazines given at maximum tolerated doses through most of the lifetime of rodents have been demonstrated to induce brain tumors. Nevertheless, there is no reliable or reproducible epidemiological evidence that shows that chemicals capable of causing brain tumors in animals at maximum tolerated doses over a lifetime can cause brain tumors in humans. The biological plausibility of those chemicals causing brain tumors in humans is lacking. First of all, there is no documented exposure of the decedent, David Valentine, to those chemicals and if there was any exposure, it was far less than the maximum tolerated lifetime doses given to animals. At low doses, these chemicals in humans would not

likely be converted by enzyme systems to their reactive metabolites which would be necessary to cause DNA damage. The high doses given to animals are necessary to saturate enzyme systems which inactivate these chemicals and make them available to enzyme pathways which could convert them to their reactive metabolites. The human brain has many defense mechanisms against the development of brain tumors. These include the blood-brain barrier and the extremely low levels of expression of genes such as P450 enzymes and glutathione-S transferase enzymes which are necessary to convert chemicals to their reactive metabolites. In the absence of reactive metabolites, DNA damage does not occur. Moreover, even if chemicals can cross the blood-brain barrier and there are reactive metabolites that can cause DNA damage, the brain is equipped with large numbers and high concentrations of DNA repair proteins which repair DNA damage as it occurs."

**{¶101}** Dr. Bigner's thus opined: "[T]here are no reliable or reproducible epidemiological studies that show that more likely than not any chemical or environmental agent has caused or contributed to the development of primary brain tumors in humans." Therapeutic radiation "is the only known and established cause of primary brain tumors in humans." "It is my opinion to a reasonable degree of medical and scientific probability and certainty that David Valentine's condition was

not caused by exposure to chemicals or environmental agents in his work place."

**{¶102}** Dr. Bigner stated: "Plaintiff's contention that Decedent David Valentine was at an increased risk of developing a malignant brain tumor than the general population, that he had an increased risk of developing malignant brain tumor secondary to the exposure to the chemicals at PPG, and that his tumor was related to his employment and exposure to chemicals is not supported by any peer-reviewed medical or scientific literature or data in this specific case. There is no medical or scientific basis to determine what caused David Valentine's glioblastoma, and there is no medical or scientific basis to conclude that Mr. Valentine's glioblastoma was caused by any exposures at PPG."

2. Buncher

**{¶103}** Charles R. Buncher, professor of biostatistics and epidemiology in the Department of Environmental Health University of Cincinnati College of Medicine, explained the significance of epidemiological studies in his affidavit: "In order to establish that a chemical or environmental agent more likely than not * * * causes a glioblastoma multiforme, the epidemiological study must first establish that there is a statistically significant association between the agent and the disease."

**{¶104}** Buncher stated: "Based upon my experience and review of the medical and scientific literature there are no scientifically reliable or reproducible epidemiological studies that establish any chemical or environmental agent as a cause of a glioblastoma multiforme. There are no epidemiological studies that demonstrate that there is a statistically significant association between a specific chemical or environmental agent (with the exception of ionizing radiation) and contraction of glioblastoma multiforme in humans."

C. Epidemiological Studies

**{¶ 105}** Following is a basic summary of some of the epidemiological studies upon which appellant's experts relied.

1. Cancer Incidence Among Employees at a Petrochemical Research Facility, JOEM, Vol. 43, Number 2, February 2001.

**{¶ 106}** This study evaluated cancer incidence among employees at an Amoco Research Center. The study notes, "A few of the many studies of petrochemical industry workers have noted an excess of brain cancer, but none has identified a specific agent likely to be responsible for the positive associations. Most studies of chemical and petrochemical industry workers in technical and salaried occupations have reported a deficit of brain cancer and of chemists." The study states: "Brain cancer was increased in the overall study group, and this excess was restricted to white men who worked for the ACC as scientists or technicians; all cases

in the latter group had worked in the 500 complex. These patterns suggest that the brain cancer excess resulted from occupational exposures, but the present study and other investigations of ARC employees have not identified a causal occupational agent, nor do they entirely rule out random variability as an explanation."

2. Occupational Risk Factors for Brain Tumors Amongst Women in Shaghai, China, JOEM, Vol. 37, Number 3, March 1995.

**{¶ 107}** This study states: "Previous studies have suggested that organic solvents are associated with brain tumors, particularly gliomas. Our results provide supporting evidence for that hypothesis. Risks also appeared to increase with probability and level of benzene exposure in these data. No previous studies, however, have provided evidence of an association of benzene with brain tumors. The association we observed may be confounded by exposure to other solvents, particularly because the association with the general class of solvents was about the same magnitude as that observed with benzene. Solvents, including benzene, may be partly responsible for the excesses observed in painters and rubber workers, but both occupations entail complex exposures. Additional detailed, exposure-specific analyses are needed to determine the agents responsible for these excesses."

3. Cancer Mortality among Swedish Chemists Graduated during Three Decades.

**{¶ 108}** This study states that it supports the suggestions that chemical exposure is a cause of both leukemic and hematopoietic tumors and brain tumors and thus indicates the occupational hazards in chemical work.

4. Brain Cancer in Petrochemical Workers.

**{¶ 109}** This study examined 18 brain cancer deaths at one petrochemical plant. "The plant is a diversified petrochemical manufacturing facility with a large number of major product lines which over the years have included ethylene, butadiene, naphtha, ethylene dichloride, diethyl sulfate, glycols, aldehydes, acetates, alcohols, amines, organic acids, and plastics and resins (polyethylene, vinyl chloride—vinyl acetate co-polymers, phenol-formaldehyde resin). Review of the plant's chemical inventory reveals the presence of at least ten recognized or suspected carcinogens in significant quantities as raw materials." However, the study did not find a common link of chemical exposure.

**{¶ 110}** The study also states: "A detailed literature search for compounds inducing brain cancer has identified 26 different chemicals from experimental animal studies, as shown in Table VI. Review of available epidemiological studies provided in Table VII offers several intriguing clues, but little solid documentation for occupationally related brain tumors, except in the case of vinyl chloride. Recent mortality studies of oil refinery workers in Canada and petrochemical workers in Texas suggest the

possibility of an excess of brain cancer risk among these groups, although no specific causative agent has yet been identified." The study also reported: "A comparison of experimental studies, epidemiological data, and the plant chemical inventory shows that vinyl chloride and diethyl sulfate are possible suspect agents for inducing brain cancer in these workers. On the basis of prior knowledge from human and animal studies, and its presence in the work environment under poorly controlled circumstances in the past, vinyl chloride must be the first compound considered. However, to date, examination of the work histories of the 18 cases does not support a significant positive association with vinyl chloride exposure, and other agents must therefore be carefully evaluated. The information available indicates that this number of brain tumors is excessive in a population of this size, and the tumors are likely to be occupationally related. There is no good evidence so far to implicate non-plant, general environmental factors. A very careful and thorough investigation of this situation must be completed, since a previously unsuspected chemical exposure may be responsible for this striking appearance of brain cancers among workers in a single petrochemical plant."

APPENDIX TWO

**{¶ 111}** The trial court denied appellant's motion to expand discovery. Appellant argued that because Valentine's employment took him into the production area as well as the laboratory areas, the air sample testing of workers on the production floor is relevant and appropriate evidence for discovery. Appellant asserted that she is entitled to 27 years' worth of air sampling results of PPG's production work force because Valentine might have passed through production areas when working as a laboratory technician and as an environmental specialist. Appellant did not direct the court to any specific incident where it is alleged that Valentine was exposed to specific chemicals within the production area of PPG. The court stated even if "air sample tests alone from the production area would benefit [appellant], this Court is not persuaded that [appellant] needs to be provided with twenty-seven years worth of air sampling results of PPG's production work force. [Appellant's] request for air sampling data for all production workers is too broad. When conducting air sampling, it appears that PPG used a charcoal sample tube that was placed on an individual worker for approximately an eight hour shift. Consequently, as argued by PPG, air samples were taken of every place that the laboratory technician went, including the production floor. Thus, the air sampling information that has been produced already includes any exposure that a laboratory technician had when the laboratory technician passed through the

production floor. Absent documentation concerning a specific incident or date whereby plaintiff demonstrates that [the deceased] was possibly exposed to chemicals similar to production workers, this Court finds that the request for the expansion of discovery is unreasonable."

**{¶ 112}** The court also denied appellant's request for an order compelling the completion of Louis Jordan's deposition. "The Court agrees with [appellee] that [appellant] is asking Mr. Jordan to testify regarding a risk analysis that is the subject of expert testimony and clearly beyond the scope of Mr. Jordan's qualifications to answer."

**{¶ 113}** The court denied appellant's request to order Nick Cleary to appear for redeposition and specifically order him to answer all questions regarding increased risk of injury from chemical exposure at PPG. The court found that based upon Cleary's affidavit, he is not able to answer appellant's counsel's question whether someone working in a chemical plant like PPG would have a risk of exposure to harmful chemicals greater than the general public. Cleary swears that he is not qualified by education or training to do a comparative analysis between the risk of exposure to harmful chemicals in a paint plant and the risk of exposure to harmful chemicals to the general public.

**{¶ 114}** The court denied appellant's request for transmittal packages, messages, e-mails, and any other forms of communication

sent from the Research and Development Departments in Pennsylvania to the Circleville plant's Safety Department or industrial hygiene specialist, from 1969 through 1996. The court found that it was too broad and unduly burdensome.

**{¶ 115}** The court denied appellant's request for PPG to produce "all product analysis prepared by process engineers to document by-products given off in the manufacturing process as discussed in Nick Cleary's deposition of December 20, 2002 at page 52." The court found appellant's request "overly broad and unduly burdensome."

**{¶ 116}** The court found that it already addressed appellant's request for the production of all ventilation test results and all problem analysis and memorandum accompanying such testing procedures that are described by Ralph Copeland in his deposition. The court previously ordered PPG to produce all existing documents relating to air sample testing performed from 1969 to 1996 at the Circleville facility in the labs where Valentine worked, and to produce documents relating to engineering controls or any problem analysis concerning ventilation problems at the labs where Valentine worked.

**{¶ 117}** The court denied appellant's request for PPG to produce all engineering and architectural designs to document the ventilation function for all of the buildings where Valentine worked from 1969 to 1996.

**{¶ 118}** The court denied appellant's request for all patent information to identify chemical processes and all products manufactured at the Circleville facility from 1969 to 1996. The court found that appellant's request seeking all patent information for a 30-year period is overly broad, unduly burdensome, and ambiguous.

**{¶ 119}** The court denied appellant's request for disclosure of the volume of every raw product purchased by the Circleville facility from 1969 to 1996. The court found appellant's request for the total volume of every raw product used by PPG's facilities over approximately 30 years to be overly broad.

**{¶ 120}** The court denied appellant's request for all OSHA and EPA records for inspection. The court noted that in its June 12, 2002 entry, it ordered PPG to produce all existing accident and/or injury reports from 1969 to 1996 related specifically to chemical exposure at the Circleville facility in the labs where Valentine worked.

**{¶ 121}** The court denied appellant's request to order PPG to produce an affidavit from appropriate PPG employees who have carried out the search to obtain the requested document information. The court found that it had already instructed PPG to supply an affidavit verifying the nonexistence of the items that the court had ordered PPG to produce and that it claims do not exist.

APPENDIX THREE

**{¶ 122}** In *Joiner*, the court upheld the trial court's finding that the four epidemiological studies on which the plaintiff relied were not a sufficient basis for the experts' opinions. The court discussed the four studies:

**{¶ 123}** "The first such study involved workers at an Italian capacitor plant who had been exposed to PCBs. Bertazzi, Riboldi, Pesatori, Radice, & Zocchetti, Cancer Mortality of Capacitor Manufacturing Workers, 11 American Journal of Industrial Medicine 165 (1987). The authors noted that lung cancer deaths among ex-employees at the plant were higher than might have been expected, but concluded that 'there were apparently no grounds for associating lung cancer deaths (although increased above expectations) and exposure in the plant.' Id. at 172. Given that Bertazzi et al. were unwilling to say that PCB exposure had caused cancer among the workers they examined, their study did not support the experts' conclusion that Joiner's exposure to PCB's caused his cancer.

**{¶ 124}** "The second study followed employees who had worked at Monsanto's PCB production plant. J. Zack & D. Musch, Mortality of PCB Workers at the Monsanto Plant in Sauget, Illinois (Dec. 14, 1979) (unpublished report), 3 Record, Doc. No. 11. The authors of this study found that the incidence of lung cancer deaths among these workers was somewhat higher than would ordinarily be

expected. The increase, however, was not statistically significant and the authors of the study did not suggest a link between the increase in lung cancer deaths and the exposure to PCB's.

**{¶ 125}** "The third and fourth studies were likewise of no help. The third involved workers at a Norwegian cable manufacturing company who had been exposed to mineral oil. Ronneberg, Andersen, & Skyberg, Mortality and Incidence of Cancer Among Oil-Exposed Workers in a Norwegian Cable Manufacturing Company, 45 British Journal of Industrial Medicine 595 (1988). A statistically significant increase in lung cancer deaths had been observed in these workers. The study, however, (1) made no mention of PCB's and (2) was expressly limited to the type of mineral oil involved in that study, and thus did not support these experts' opinions. The fourth and final study involved a PCB-exposed group in Japan that had seen a statistically significant increase in lung cancer deaths. Kuratsune, Nakamura, Ikeda, & Hirohata, Analysis of Deaths Seen Among Patients with Yusho--A Preliminary Report, 16 Chemosphere, Nos. 8/9, p. 2085 (1987). The subjects of this study, however, had been exposed to numerous potential carcinogens, including toxic rice oil that they had ingested." Joiner, 522 U.S. at 145-146.