IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF PENNSYLVANIA

JAMIE GANNON and REBECCA GANNON	:	CIVIL ACTION
	:	
vs.	:	
	:	
UNITED STATES	:	NO. 03-6626

MEMORANDUM

ROBERT F. KELLY, Sr. J. **JULY 17, 2007**

Plaintiffs, Jamie and Rebecca Gannon, have filed suit against Defendant, the United States of America, under the Federal Tort Claims Act, 28 U.S.C. §§ 2671-2680. Jamie Gannon was born on July 22, 1973. Between 1973 and 1976 in Upper Darby, Pennsylvania, he was administered multiple doses of Orimune, an oral poliomyelitis vaccine ("OPV") manufactured by Lederle Laboratories. In November 2000, Jamie Gannon was diagnosed with a medulloblastoma, which is a type of cancerous brain tumor. Plaintiffs allege that this tumor was caused by a monkey virus, known as Simian Virus 40 ("SV40") and that the Orimune he received was contaminated with this allegedly cancer-causing SV40. Their claim against the United States rests on the argument that the United States government negligently licensed Lederle to produce Orimune and to release it to the public. Plaintiffs claim that the United States did not confirm the absence of SV40 at each stage of manufacture allegedly in violation of the federal regulations concerning the licensing, testing, and manufacture of live oral polio vaccine.

On January 23, 2007, this Court commenced a bench trial in this case. The trial began with a Daubert examination of Dr. Adi Gazdar, Plaintiffs' causation expert. For the convenience of the parties, the witness, and this Court, Dr. Gazdar also presented his full

testimony as to causation. Defendant then presented the testimonies of its causation experts: Dr. Robert Garcea, Dr. Harald zur Hausen, and Dr. Neal Halsey. These witnesses presented a rebuttal to Dr. Gazdar with respect to Daubert and also presented their full testimonies. The witnesses were presented in this way so that they would not have to be recalled later in the trial.

In a bench trial, this Court's "role as gatekeeper pursuant to Daubert is arguably less essential" because a judge rather than a jury is the fact finder. Clark v. Richman, 339 F. Supp. 2d 631, 648 (M.D. Pa. 2004). However, the Third Circuit has given no indication as to how and if Daubert hearings differ for bench trials. Id. "[I]n the absence of prohibition or direction from the Third Circuit, reliability and relevancy challenges to an experts' opinions may be considered during a bench trial." Id. That is what was done here. Because this Court sits as the trier of fact in this case, it was appropriate for testimony as to Daubert as well as to the merits of the case to occur at the same time and out of the standard order of a trial. The bench trial format provided the flexibility to proceed in this manner.

At the conclusion of the testimonies of Dr. Gazdar and Defendant's three experts, this Court denied Defendant's Daubert motion. Defendant also made a Motion pursuant to Federal Rule of Civil Procedure 52(c) for Judgment on Partial Findings as to causation; whether SV40 causes human medulloblastomas and whether it caused Mr. Gannon's medulloblastoma. Plaintiffs filed a Motion to Strike Defendant's Rule 52(c) Motion. Plaintiffs argue that a Rule 52(c) Motion is improper at this time because Plaintiffs, the party bearing the burden of proof, have not presented all of their evidence.

Federal Rule of Civil Procedure 52(c) governs judgments on partial findings in bench trials. Rule 52(c) states:

> If during a trial without a jury a party has been fully heard on an issue and the court finds against the party on that issue, the court may enter judgment as a matter of law against that party with respect to a claim or defense that cannot under the controlling law be maintained or defeated without a favorable finding on that issue, or the court may decline to render any judgment until the close of all evidence. Such a judgment shall be supported by findings of fact and conclusions of law as required by subdivision (a) of this rule.

This Rule "authorizes the court to enter judgment at any time that it can appropriately make a dispositive finding of fact on the evidence." Fed. R. Civ. P. 52(c) advisory committee's notes.

Rule 52(c) replaced part of Rule 41(b) that "formerly authorized a dismissal at the close of the plaintiff's case if the plaintiff has failed to carry an essential burden of proof." Id. This now defunct part of Rule 41(b) was referred to as involuntary dismissal. With the enactment of Rule 52(c), judgment on partial findings became the procedural successor to involuntary dismissal. Id.; Rule 41(b) advisory committee's notes. Accordingly, this Court will look to the legal standard previously articulated under Rule 41(b). Fechter v. Ct. Gen. Life Ins. Co., 800 F. Supp. 182, 196 (E.D. Pa. 1992); see also 9A Charles Alan Wright & Arthur R. Miller, Federal Practice & Procedure § 2573.1 (2d ed. 1995) ("The case law developed under Rule 41(b) . . . is applicable under Rule 52(c)."). Thus, that legal standard is:

> the court is not as limited in its evaluation of the nonmovant's case as it would be on a motion for a directed verdict. The trial judge is not to draw any special inferences in the nonmovant's favor nor concern itself with whether the nonmovant has made out a prima facie case. Instead the court's task is to weigh the evidence, resolve any conflicts in it, and decide for itself where the preponderance lies.

Giant Eagle, Inc. v. Fed. Ins. Co., 884 F. Supp. 979, 982 (W.D. Pa. 1995); Fechter, 800 F. Supp. at 196. "Rule 52(c) expressly authorizes the district judge to resolve disputed issues of fact." Ritchie v. United States, 451 F.3d 1019, 1023 (9th Cir. 2006).

While a judge's legal approach to ruling on a Rule 52(c) motion is the same as that under Rule 41(b), Rule 52(c) has a broader procedural application than Rule 41(b). 9 James Wm. Moore, et al., Moore's Federal Practice § 52.50 (3d ed. 2007). First, a judgment on partial findings pursuant to Rule 52(c) can be entered against either the plaintiff or the defendant, rather than just the plaintiff. Id. Second, it allows a judgment to be entered at any time after the affected party has been fully heard with respect to the issue. Id. The court need not wait until the conclusion of that party's case. Id. "The failure of a party to establish an essential issue justifies the immediate termination of the case or claim. Judgment on partial findings conserves time and resources by making it unnecessary for the court to hear evidence on additional facts when the result would not be different even if these additional facts were established." Id.

Plaintiffs' argue that a decision under Rule 52(c) "is based solely after the completion of the non-movant's evidence." (Pl.'s Mot. To Strike Def.'s Rule 52(c) Mot., 5). Plaintiffs are incorrect. Their argument ignores the procedural differences between Rule 52(c) and its predecessor Rule 41(b). While the legal standard for making a judgment under both rules is the same, the clear language of Rule 52(c) shows that a motion for judgment on partial findings need not be made after the close of plaintiff's evidence. In re Anthem Communities/ RBG, LLC, 267 B. R. 867, 876 (Bankr. D. Col. 2001) ("[Rule 52(c)] does not contain language requiring a motion at the close of plaintiff's evidence."). Rather, the motion can be made when "a party has been fully heard on an issue." Fed. R. Civ. P. 52(c).

The advisory committee notes for Rule 52(c) lend further support to the understanding that it is procedurally different than Rule 41(b). Those notes state that a judgment on partial findings can be entered "at any time that the court can appropriately make a dispositive

finding of fact on the evidence." Fed. R. Civ. P. 52(c) advisory committee notes. Furthermore, a judgment should only be "made after the court has heard all the evidence bearing on the crucial issue of fact." Id. The advisory committee notes for Rule 41(b) also clearly state that involuntary dismissal is replaced by "the new provisions of Rule 52(c), which authorize entry of judgment against the defendant as well as the plaintiff, and earlier than the close of the case of the party against whom judgment is rendered." Fed. R. Civ. P. 41(b) advisory committee notes. Thus, Plaintiffs' argument that this motion is improper because they have not completed their case-in-chief is meritless in light of the Federal Rules.

To determine if Defendant's Rule 52(c) Motion on causation is proper, this Court must decide if Plaintiffs have been fully heard on the causation issue. Plaintiffs argue that they have not because they have other expert and fact witnesses besides Dr. Gazdar. According to Plaintiffs, these witnesses would address the issue of how OPV was contaminated with SV40 and how this vaccine contamination issue shows flaws in the epidemiological data discussed by Defendant's causation experts.

Epidemiology is the field of study in public health and medicine that researches what risk factors cause a disease in human populations. Epidemiologists seek "to determine whether individuals exposed to an agent have a greater risk of developing the disease in question." Siharath v. Sandoz Pharm. Corp., 131 F. Supp. 2d 1347, 1356 (N.D. Ga. 2001). Here, the epidemiological data addressed whether SV40 is a risk factor that causes human cancers.

Plaintiffs are arguing that the epidemiological evidence referenced and discussed by Defendant's experts incorrectly assumed that only the inactivated polio vaccine ("IPV"), administered between 1955 and 1962, and OPV used for clinical trials prior to its licensure were

contaminated with SV40. Plaintiffs contend that their vaccine contamination evidence challenges this assumption because it shows that OPV used after 1963 (after it was licensed) was also contaminated with SV40.

Plaintiffs made the same argument at the close of Defendant's experts' testimonies. When this Court asked Plaintiffs if Dr. Gazdar was their last witness on causation, Plaintiffs' counsel, Stanley P. Kops, Esq., responded with this answer:

> Not necessarily, Your Honor. Mr. Mitsch [Defendant's counsel] has told this Court that it is. We don't have anyone else who's going to testify about testing Mr. Gannon's tumor. On the other hand, a lot of the discussion has been about whether or not SV40 was in the vaccine post-1961, '62, '63. That changes the entire epidemiological history of this occurrence. Mr. Mitsch says no, it doesn't. The experts don't say that. Every one of the experts assumed, as part of their opinion, that nobody was infected post-1962. And none of them think they were infected by SV40 other than – and OPV, other than perhaps 10,000 people. That has to play a role in a determination of whether or not someone is or isn't suffering from SV40 today, post-1962.

(Jan. 29 Tr. 130: 3-15). Mr. Kops' response at trial as well as now focuses overwhelmingly on the issue of vaccine contamination rather than the issue of causation.

The additional witnesses the Plaintiffs deem necessary would also overwhelmingly testify only as to the contamination issue. These potential witnesses, to the best of this Court's knowledge, are: Dr. Michael Sulzinski, Dr. Janet Butel, Dr. Ronald Lundquist, Dr. Paul Parkman, Dr. Ruth Kirschstein, Dr. Koralnik, and Dr. Andrew Conrad. Dr. Sulzinski and Dr. Butel are Plaintiffs' experts on whether OPV was contaminated by SV40. The expert reports of these two doctors solely discuss vaccine contamination. See Sulzinski's Report (Pl.'s Sur Reply to Def.'s Reply Br. Summ. J. (Doc. No. 116), Ex. A); Butel's Reports (Def.'s Mot. Summ. J. (Doc. No. 79), Exs. 13, 14, 15; Def's Reply Summ. J. (Doc. No. 103), Ex. 26). Drs. Sulzinski and Butel cannot

testify to opinions that are not in their expert report.[1] See Fed. R. Civ. P. 26(a)(2)(B) ("The report shall contain a complete statement of all opinions to be expressed and the basis and reasons therefor . . . ."). Furthermore, in Dr. Sulzinski's and Dr. Butel's depositions they both stated that they were not rendering an opinion with respect to causation.[2] (Def's 52(c) Mot., Exs. 1, 2). As

---

[1] Plaintiffs also argue that Dr. Sulzinski and Dr. Butel could have testified about PCR testing to show that Dr. Gazdar's PCR testing of Gannon's tumor was appropriately conducted. Neither Dr. Sulzinski's nor Dr. Butel's reports addressed Dr. Gazdar's PCR testing.

[2] Dr. Butel testified in her deposition as such:

Q[by Mr. Mitsch]. In your reports, you do not render an opinion as to whether or not SV40 causes human cancer, correct?
A [by Dr. Butel]. That is correct.
Q. And to follow up to the questions earlier today, I take it that you're not going to be offering an opinion on that in this case?
A. That is correct.
Q. And in your reports, you don't render an opinion as to whether or not SV40 caused Mr. Gannon's medulloblastoma, correct?
A. That is correct.
Q. And you're not going to be offering an opinion on that in this case?
A. Correct.

(Def.'s 52(c) Mot., Ex.1).

Dr.Sulzinski also testified as such:

Q[by Mr. Mitsch]. Have you been asked to gave [sic] an opinion in this case as to whether or not Mr. Gannon's tumor has SV40 in it?
A [by Dr. Sulzinski]. No, that's not part of my report.
Q. And that's not something that, at least as far as you are aware, that you are going to be asked about at trial?
A. As far as I am aware, that is correct.
Q. And have you been asked to give an opinion as to whether or not SV40 can cause a Mentula blastoma [sic]?
A. That is not part of my report.
Q. And as far as you know that is not something you are going to be asked about at trial?
A. As far as I know.
Q. And, generally, as to the relationship between SV40 and cancer, is that something that you have been asked to comment about in this case?
A. Well, it is certainly something that I, you know, I read the reports, and I have my own opinion on it. I don't know that I will be asked to comment about that.
MR. MITSCH: Mr. Kops, just to save some time, is he going to be asked, are you going to be proffering him as an expert on –
MR. KOPS: I don't know if I am going to ask him that.
MR. MITSCH: Are you telling me that you are going to be going into areas that have not been discussed in his report.
MR. KOPS: I didn't say that.
MR. MITSCH: Is his expert testimony going to be limited to what he has written in his reports, and the subject matter he has discussed in those reports?
MR. KOPS: And the subject matters discussed in his reports, that is correct.

ascertained from reading Plaintiffs' Motion to Strike and its Reply to Defendant's Trial Brief, the purpose of Drs. Lundquist, Parkman & Kirshcstein is solely to testify as to their knowledge about whether SV40 was present in OPV. In addition to calling their own witnesses, Plaintiffs contend that they did not have a chance to question two of Defendant's experts, Dr. Koralnik and Dr. Conrad. The Defendant chose not to call these two experts as to the issue of causation, but Plaintiffs assert these experts have tested Mr. Gannon's tumor tissue for SV40. Defendant simply chose not to call these witnesses and Plaintiffs did not raise them as potential causation witnesses when asked by the Court if they had any other causation witnesses.

Plaintiffs' key objection to this present Motion is that the polio vaccine contamination issue effects the epidemiological data which in turn effects the causation issue.[3] This objection obscures the issue at hand: whether SV40 causes human cancer and whether it caused Mr. Gannon's medulloblastoma. The testimonies of Dr. Gazdar and Defendant's three

---

> MR. MITSCH: Would it be fair to say, doctor, that you have not discussed the issue of whether or not SV40 can cause cancer in your reports?
> THE WITNESS: I have not discussed that in the report.

(Id., Ex. 2).

[3] Mr. Kops cites to the Insitute of Medicine's ("IOM") Immunization Safety Review: SV40 Contamination of Polio Vaccine and Cancer, (United States Exhibit ("U.S. Ex.") 200), as support for his concern about flawed epidemiological data. The IOM stated:

> Claims have been made that some oral polio vaccines might have been contaminated after 1963 (Kops, 2000). The committee urges that FDA or other agencies address these claims to try and resolve the uncertainty regarding the possibility of exposure to SV40 after 1963. Appropriate assumptions about exposure are essential for conducting valid epidemiological analyses of the risks that might be associated with contaminated OPV.

(Id. at 71). The interesting aspect about this argument is that the IOM made this statement because of claims made in a 2000 article written by Mr. Kops himself. See Kops, Stanley P., Oral polio vaccine and human cancer: a reassessment of SV40 as a contaminant based upon legal documents, Anticancer Res. 20(6C):4745-9. While Mr. Kops implies that he has unbiased support because of the IOM's report, the IOM's support for its concern is Mr. Kops' article. So in the end, Mr. Kops "support" for his argument is none other than himself.

Moreover, the IOM stated that "[t]hough the suggestion has been made (Kops, 2000) that some OPV administered after 1963 might also have contained SV40, there is not evidence to support this. In three studies that sought to detect SV40 in OPV, SV40 was not found (Rizzo et al. 1999; Sangar et al, 1999; Sierra-Hoingmann and Krause, 2000)." (U.S. Ex. 200, at 67).

expert witnesses addressed these issues of general and specific causation. The vaccine contamination issue involves whether OPV that Mr. Gannon received was contaminated with SV40. These are distinct and separate issues. Plaintiffs are merely trying to graft the issue of vaccine contamination onto the issue of causation to prevent their case from ending.

Moreover, this vaccine contamination issue does not bolster Plaintiffs' case for causation. The witnesses that Plaintiffs want to put forth would introduce no additional or affirmative evidence on causation, but rather would just present a critique of the epidemiological data. Plaintiffs have the burden to show causation and cannot satisfy that burden by merely playing the role of contrarian to Defendant's defense.

Plaintiffs also assert that the issue of general causation is no longer before this Court. According to Plaintiffs, "[g]eneral causation is not an issue; it has already been determined to be present. The issue now before the Court is whether Mr. Gannon's tumor contained SV40." (Pl.'s Reply to Def.'s Trial Br., 7). Plaintiffs argue that general causation is no longer an issue because Dr. Gazdar survived Defendant's Daubert challenge. This is a misunderstanding of Daubert. The United States Supreme Court in Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579 (1993), held that for expert testimony to be admissible pursuant to Federal Rule of Evidence 702, the testimony needs to be based only on reasoning or methodology that is scientifically valid and fits with the facts in issue. Id. at 592-593; Heller v. Shaw, 167 F.3d 146, 152 (3d Cir. 1999). In a Daubert hearing, a federal district court performs a "gatekeeper" function with respect to the admissibility of expert testimony, examining whether the testimony is relevant, reliable, and helpful to the trier of fact. Schneider ex rel. Estate of Schneider v. Fried, 320 F.3d 396, 404 (3d Cir. 2000); Shetterly v. Sony Electronics, Inc., No. 02-862, 2005 WL 2219473, *8 n.17 (W.D. Pa. Sept. 13, 2005).

Thus, the Daubert hearing's purpose is only to determine the admissibility of expert evidence; it is not to determine "whether such evidence is sufficient with respect to a matter upon which the plaintiff has the burden of proof." Id. The denial of the Daubert motion only meant that this Court decided that it was proper to consider Dr. Gazdar's testimony in determining the ultimate issues of general and specific causation.

In conclusion, Plaintiffs have been fully heard on the issue of causation because Dr. Gazdar was their only causation witness. Plaintiffs' other potential witnesses would have testified as to SV40 contamination of OPV and not causation. Vaccine contamination's sole and remote connection to the causation issue is with respect to the epidemiological data. Nevertheless, the result of this Rule 52(c) Motion would be no different if these additional facts were established. Therefore, Defendant's Rule 52(c) Motion is proper and Plaintiffs' Motion to Strike is denied.

FINDINGS OF FACT

1. In order to prevail Plaintiffs must prove that SV40 causes cancer in humans and that SV40 caused Mr. Gannon's medulloblastoma.

2. To prove these necessary elements of their case Plaintiffs called as their lone expert, on causation, Dr. Adi Gazdar. Dr. Gazdar did his undergraduate study in London, England and obtained his medical degree at the University of London. He did his internship in England and a residency and a second internship in Milwaukee, Wisconsin. He also did a residency in pathology in Boston, Massachusetts. He has been a pathologist since 1968. At that time, he took employment with the National Cancer Institute in Bethesda, Maryland where he studied forms of cancer. He was an investigator and ultimately a section head. He studied viral etiology of cancer,

which he went on to explain as, "whether virus is associated with the appearance and origin of that cancer." He remained at the National Cancer Institute until 1991 when he went to the University of Texas Southwestern Medical Center in Dallas, Texas. He went there as a full professor and deputy head of the Hammond Cancer Center. In the late 1990's, he started to work with SV40. To date Dr. Gazdar has published approximately 600 papers in peer reviewed medical literature. Jan. 23, 2007 Test. of Dr. Adi Gazdar ("Jan. 23 Gazdar Tr.") at 5:1-10:8.

3. Dr. Gazdar testified in part confirming the concluding opinions of his written report:

> Based on my review of relevant and reliable literature, it is my opinion to a reasonable degree of scientific and medical certainty that SV40 plays a causal role in the subset of human tumors in which it has been frequently found, including brain tumors and [medulloblastomas].
>
> Based on the above, it is my opinion to a reasonable degree of scientific and medical certainty that SV40 played a causal role in the development of Jamie Gannon's medulloblastoma.

Dr. Gazdar Causation Report 5/10/06. Concluding opinions p. 23, 24. Jan. 23 Gazdar Tr. at 158:6-22; 158:23-159:1.

4. He based his opinion on his view of the biological evidence, conceding that "the current epidemiology evidence does not support the conclusion that SV40 causes human cancer, let alone medulloblastoma." Jan. 24 Gazdar Tr. at 60:2-12.

5. This is inconsistent with statements made in correspondence with plaintiffs' counsel in other SV40 litigation, Dr. Gazdar opined that the *Gannon* case was not an "optimal" one to pursue and that it will be "difficult . . . to show causation":

> For more than 6 months I have tried to impress on Don [MacLachlan, Plaintiffs' counsel] the importance of getting in live patients for extensive testing of blood urine etc and for immortalization of B

> lymphoblastoid cells. If we can conclusively demonstrate the virus or evidence of virus exposure in a live patient your job (and mine) would be greatly simplified. While Don always agrees in principle, I have yet to see the patient. I have no idea when the deadline for lab testing for the [redacted] case is or was. Yet there was a lot we could do to strengthen our findings, which have not even been discussed. . . .
>
> I am concerned about our cases. [redacted], I regard [redacted] as a toss up at best. [redacted] Hicks both have lots of virus and we have an opportunity to do things not possible with the other two cases. **Both patients are alive. Our testing and reporting are light years more advanced than a couple of years ago. However they are medulloblastoma and meningioma cases. Both are going to be difficult cases to show causation**. Medulloblastoma has a very different origin than the run of the mill brain tumors (gliomas), and to compare them to gliomas is like comparing apples and oranges. Most series (including my own) indicate very low frequencies of SV40 positivity in medulloblastomas. Meningiomas are even more difficult because they are not brain tumors at all. There is one report on meningioma from mainland China, and there is no animal model. It is not even a malignant tumor, and I know of no benign tumors induced by SV40. I had forgotten about the case report Janet Butel described a couple of years ago (see enclosure). If there is a weakness with [Lederle's counsel], he has failed to hire an expert with a good general knowledge of cancer medicine/biology. We cannot presume the opposite side will always fail to exploit our inherent deficiencies in these areas. **Obviously to win these cases we have to walk the extra mile. I feel that you lawyers should consult one of your medical experts before selecting cases to litigate**. **I think we have failed to select a single optimal case in these first four cases, and if we lose them all, it may be the end of SV40 litigation without having fought the "right" case.**

U.S. Ex. 36, February 23, 2006 E-mail from Dr. Gazdar (emphasis added); accord Jan. 24 Gazdar Tr. at 5:24-20:4

6. To determine whether a virus causes human cancer, scientists routinely examine two essential types of evidence: (1) epidemiological evidence, which statistically demonstrates that exposure to the virus increases the risk that a tumor will develop; and (b) biological evidence, which demonstrates that the virus transforms otherwise healthy human cells into malignant cells.

Jan. 25, 2007 Test. of Dr. Robert Garcea ("Garcea Tr.") at 12:13-15:16; Jan. 26, 2007 Test. of Dr. Harald zur Hausen ("zur Hausen Tr.") at 21:22-22:22, 157:14-19, 158:9-16; Jan. 29, 2007 Test. of Dr. Neal Halsey ("Halsey Tr.") at 9:24-10:17; Halsey Tr. at 10:18-10:23.

7. Epidemiology, the study of diseases in populations determines whether an agent is causally associated with an increased risk of disease. Halsey Tr. at 10:18-10:23; see also zur Hausen Tr. at 28:1-19. Epidemiology is the core discipline used to determine whether an infectious disease agent causes cancer in humans. Halsey Tr. at 10:24-11:6; accord Jan. 23 Gazdar Tr. at 145:1-8 ("It's obvious that epidemiology is one of the major methods by which we determine whether an agent is cancer causing or not.").

8. No virus has ever been determined to cause human cancer without both supporting epidemiological and biological evidence. See Halsey Tr. at 11:15-12:20; see also Jan. 24 Gazdar Tr. at 66:8-19 (explaining that six viruses are known to cause human cancer and that epidemiological evidence exists for all of them).

9. The National Academy of Sciences, which advises the federal government on scientific and technical matters, established the Institute of Medicine ("IOM") to examine policy matters pertaining to public health. United States Exhibit ("U.S. Ex.") 200, "Immunization Safety Review: SV40 Contamination of Polio Vaccine and Cancer" ("IOM Report") at iv. Consistent with this mandate, the IOM periodically conducts immunization safety reviews. Id. at ix; Jan. 23 Gazdar Tr. at 143:25-144:12. In 2002, the IOM conducted an immunization safety review concerning human cancer and possible SV40 contamination of polio vaccines. Jan. 23 Gazdar Tr. at 143:25-144:12; U.S. Ex. 200 at 25-26. As it had in the past, the IOM analyzed both the biological and the epidemiological evidence. See U.S. Ex. 200, IOM Report, at 1-4.

10. This analytical and scientific framework - - examining both epidemiological and biological evidence - - has been used routinely by the IOM to determine whether an agent causes human disease. See id. at 1-4; see generally Halsey Tr. at 61:14-65:7. In each case, the IOM analyzed whether the pathogen or substance under study could be considered the cause of a disease or condition by evaluating both the biological and the epidemiological evidence. See generally Halsey Tr. at 61:14-65:7.

11. Several well-known and frequently used methodologies exist for evaluating whether pathogens cause human disease. These methodologies all rely on both biological and epidemiological lines of evidence. The most famous of these was developed by Sir Austin Bradford Hill. Halsey Tr. at 18:17-19:20, 64:19-65:3, see also Jan. 24 Gazdar Tr. at 69:16-70:6 (testifying that the Bradford Hill criteria are "well-recognized" and widely used in the science community to assess general causation").

12. Other preeminent scientists have relied on and adapted the Bradford Hill criteria to determine whether a virus can be deemed to cause human cancer. One such methodology was developed by Dr. Harald sur Hausen, who testified as an expert for the United States at this trial. E.g., Jan. 24 Gazdar Tr. at 69:16-70:6; zur Hausen Tr. at 17:5-18:14. As with the Bradford Hill criteria and the IOM's analytical framework, the causation framework developed by Dr. zur Hausen requires an evaluation of both biological and epidemiological evidence in evaluating causation.

13. The International Agency for Research on Cancer ("IARC"), a part of the World Health Organization, examines whether agents cause human cancer. Jan. 24 Gazdar Tr. at 23:18-4. IARC examines both the epidemiological evidence and the biological evidence in

making this determination. See id. at 27:17-28:25, 66:8-67:2; Jan. 23 Gazdar Tr. at 57:9-58:14.

14. In other words, epidemiological and biological evidence are key components to all well-recognized scientific frameworks that examine causation of human diseases. If either epidemiological or biological evidence fails to support a causal connection or is otherwise inconclusive, one cannot conclude with any degree of certainty that a pathogen such as a virus is the cause of a disease such as human cancer. Halsey Tr. at 11:15-12:1; see also Garcea Tr. at 15:17-23.

15. The United States called three expert witnesses to testify on causation: Robert Garcea, M.D., Harald zur Hausen, M.D., D. Sc., and Neal Halsey, M.D. Dr. Garcea is a Professor of Pediatrics, Cell and Developmental Biology, and Microbiology at the University of Colorado School of Medicine. Garcea Tr. at 3:10-14; U.S. Ex. 3, Garcea Curriculum Vitae. Dr. Garcea has extensive experience studying SV40 and the other human polyomaviruses, JC and BK viruses. Garcea Tr. at 6:22-8:5. His laboratory conducted, in Plaintiffs' expert's own words, the "landmark Bergsagel study," which was the first one to use Polymerase Chain reaction ("PCR") testing to detect SV40-like sequences in pediatric tumors. Jan. 24 Gazdar Tr. at 37:7-37:18; Garcea Tr. at 27:18-28:3. Dr. Garcea also participated in a sero-epidemiology study conducted by Dr. Denise Galloway that sought to determine whether SV40 transmission was occurring within the human population. Currently, Dr. Garcea's area of interest is in development a low-cost vaccine for cervical cancers caused by the human papillomavirus

16. After reviewing the available biological and epidemiological evidence, Dr. Garcea concluded, to a reasonable degree of medical certainty, that SV40 has not been shown to be a cause of human cancer, including medulloblastoma. Garcea Tr. at 91:9-22; accord U.S. Ex.

4, Garcea Expert Report. Dr. Garcea reached this conclusion based on two essential points. First, although the biological evidence demonstrates that SV40 can cause cancer in laboratory animals and can transform human cells when they are exposed *in vitro* - - i.e., in cell culture experiments conducted in the laboratory - - insufficient biological evidence exists to demonstrate that SV40 acts the same way *in vivo* - - i.e., in live human beings. Garcea Tr. at 136:23-138:9. Indeed, Dr. Garcea emphasized that older reports claiming to find SV40 DNA in various human tumor samples have recently been called into doubt by more recent studies that conclude that positive findings may be the result of contamination from DNA plasmids routinely used by microbiological labs conducting cancer research. Id. at 36:9-37:20. Second, the more recent epidemiological studies, which rely on new methods to detect SV40 antibodies in human blood, strongly suggest that no causal connection exists between SV40 and human cancer. Garcea Tr. at 22:17–25:7.

17. Dr. zur Hausen, a Professor Emeritus at the German Cancer Research Center, also testified on behalf of the United States. Dr. zur Hausen helped determine that the Epstein-Barr virus causes cancer in humans and discovered that HPV 16 and 18 cause cervical cancer in women. zur Hausen Tr. at 5:21-6:24; 7:21-9:21; Jan. 23 Gazdar Tr. at 57:18-58:4. His discovery concerning HPV led to the development of the first vaccine to prevent cancer. zur Hausen Tr. at 7:21-9:21. He was described by Plaintiffs' expert as a "learned man" and a "very distinguished scientist." Jan. 24 Gazdar Tr. at 19:9-18. Dr. zur Hausen is a member of numerous cancer advisory panels, was a member of IARC, is a member of multiple journal editorial boards, and is editor-in-chief of the International Journal of Cancer. Id. at 12:25-13:21; U.S. Ex. 11, zur Hausen Tr. at 9:21-12:24.

18. Dr. zur Hausen concluded, to a reasonable degree of medical certainty, that the available evidence does not support the conclusion that SV40 causes human cancer, including medulloblastoma. Id. at 42:23-43:8; accord U.S. Ex. 17, zur Hausen Expert Report. He testified that it is inappropriate to conclude that SV40 causes human cancer based just on: (1) reports that SV40 causes cancer in laboratory animals, and (2) the ability of SV40 to transform human cells *in vitro.* zur Hausen Tr. at 14:14-25. Dr. zur Hausen also reiterated Dr. Garcea's concerns that laboratory contamination may be the cause of the positive SV40 results in PCR testing on tumors, including Mr. Gannon's medulloblastoma.

19. Dr. Halsey, the only epidemiologist to testify in this case, is a Professor at the Johns Hopkins University Bloomberg School of Public Health and Director of the Bloomberg School's Institute of Vaccine Safety. Halsey Tr. at 3:16-4:1, U.S. Ex. 253, Halsey Curriculum Vitae. Dr. Halsey has worked extensively on vaccine safety for the past quarter century. Halsey Tr. at 5:25-8:7. He recently participated in a sero-epidemiological study seeking to determine the prevalence of SV40 in persons with tumors as compared to persons without cancer. See generally Halsey Tr. at 36:20-40:21; U.S. Ex. 249.

20. Dr. Halsey testified, to a reasonable degree of medical certainty, that it is not scientifically valid to conclude that SV40 causes cancer, including brain cancers, in humans. Halsey Tr. at 46:4-16; accord U.S. Ex. 2, Halsey Expert Report. Specifically, he testified that: (1) the available epidemiological evidence does not support the claim that SV40 causes cancer in humans; (2) the epidemiological evidence reviewed by the IOM, while flawed in certain respects because of classification issues preventing researchers from determining which participants - - if any - - were truly exposed to SV40, suggests that there is no connection between SV40 and human

cancer; (3) the Bradford Hill criteria have not been met with regard to SV40 and human cancer; and (4) recent sero-epidemiological studies demonstrate that SV40 plays no role in causing human cancer. Halsey Tr. at 17:22-18:16, 31:9-32:11, 33:2-35:23.

21. Dr. Gazdar's opinion fails to satisfy the well-recognized and broadly accepted criteria for evaluating causation that have been developed by scientists such as Sir Bradford Hill. Halsey Tr. at 18:17-19:20, 64:19-65:3, see also Jan. 24 Gazdar Tr. at 69:16-70:6 (testifying that the Bradford Hill criteria are "well-recognized" and "widely used in the science community to assess general causation"). Dr. Gazdar's opinion also fails to satisfy the criteria developed by Dr. zur Hausen.

22. The Bradford Hill criteria consist of nine factors that address causality: (1) Strength of Association, (2) Consistency, (3) Specificity, (4) Temporality, (5) Biologic Gradient, (6) Plausibility, (7) Coherence, (8) Experimental Evidence, and (9) Analogy. See generally Halsey Tr. at 19:21-29:18.

23. Dr. Halsey testified that most expert bodies use the Bradford Hill criteria and he puts the most weight on the first five. Id. at 19:15-20.

24. The first criterion, Strength of Association, means the comparison of patients with cancer who have been exposed to the virus as to those who were unexposed. Id. at 19:23-20:4. Dr. Halsey is of the opinion that SV40 and human cancer do not satisfy the Strength of Association criterion. Id. at 20:5-9.

25. The second criterion, Consistency, has not been satisfied according to Dr. Halsey because most evidence from recent studies show no association and have been inconsistent. Id. at 22:7-23:2.

26. Specificity, in laymen's terms generally means that an agent usually causes one type of human cancer. When an agent is associated with a broad array of different types of diseases it weakens the evidence because it is non-specific. Most agents that cause cancer cause a single form of cancer. Id. at 23:9-17. SV40 does not meet the Specificity criteria because it is found in at least half a dozen different tumors. "[P]eople even found it in breast cancer which we know it is not associated with, . . ." Id. at 24:1-18.

27. The fourth criterion, Temporality, means that the exposure must have occurred prior to the onset of the tumor. You have to be exposed to something before you can conclude that it caused the disease in question. Most experts in the field of causal assessment consider this an essential criteria. Id. at 25:1-10.

28. The fifth criterion, Biologic Gradient, means the higher the level of exposure the greater the risk. This is not used as much with regard to infections in cancer. Id. at 25:23-26:10.

29. The sixth criterion, Plausibility, means does it make sense that this virus could cause this type of tumor based upon everything we know about virus and its potential to cause a specific disease. Dr. Halsey concluded that it is plausible that it can cause human cancer. Id. at 26:12-27:3.

30. The seventh criterion, Coherence, means you have to look at everything that has been generated, all of the criteria that have been listed before, does it all fit together with regard to your knowledge about the disease and about the infection. In other words is it a nice neat package that suddenly explains everything. Id. at 27:5-12. Dr. Halsey is of the opinion that this criterion linking SV40 and human cancer has not been met. Id. at 27:13-14.

31. The eighth criterion, is Experimental Evidence, this is where scientists set up a prospective trial and deliberately give some people a drug and others a placebo. In the case of an infectious agent, scientists would have to deliberately expose someone to an infectious agent and not expose others. This has been done with animals and SV40 and those experiments demonstrate that SV40 can cause some tumors, not the medulloblastoma, but it can cause other tumors. In humans there are no experimental data that the infection causes a specific tumor. No one can deliberately select people to be injected with SV40 in order to compare them with a control group and then follow them for many years. Such a study would not be permitted. Id. at 27:17-28:12.

32. The ninth and last criterion, Analogy, is a weak criteria which, according to Dr. Halsey, is almost never used and even ignored by some. Id. at 28:19-25. According to Dr. Halsey, this criterion has not been met for the association with SV40. In this case, the other two members of the polyomavirus that infect humans, the BK and JC viruses, do not cause cancer. Id. at 29:4-9. Therefore, if the analogy is used properly in its narrow meaning you would have to say that by analogy the other polyomaviruses do not cause cancer. Id. at 29:7-18.

33. In Dr. Halsey's opinion the first two criteria are the most important, Strength of Association and Consistency. The first four are the ones we weigh the most. Id. at 29:19-30:2. Satisfying one or even two criteria is not sufficient evidence to establish a causal relationship. Id. at 30:3-11.

34. Even though medulloblastoma is a rare tumor epidemilogical analysis could be done with respect to medulloblastoma and human cancer and the Bradford Hill criteria could be applied to the purported link between SV40 and human cancer. Id. at 30:17-31:8.

35. Dr. Gazdar's testimony regarding causation is also inconsistent with the

criteria developed by Dr. zur Hausen, which have been specifically tailored to viruses and human tumors. zur Hausen Tr. at 17:5-18:14. Under these criteria, a virus is considered to be the cause of tumors when: (1) the regular presence of the virus is established in the tumor cells; (2) the virus is found to "immortalize" cells in vitro; (3) the infected cells return to a normal state when the cancer-causing agent in the virus is blocked; and (4) epidemiological evidence demonstrates that the viral infection represents the prime risk factor for development of the tumor at issue. Id. These criteria have not been met with respect to whether SV40 causes human cancer, including medulloblastoma.

36. The first factor, finding the regular presence of the virus in tumor cells, has not been met with respect to SV40 and human cancer. zur Hausen Tr. at 25:7-9. To satisfy this factor, at lease one viral copy must be found in every tumor cell. Id. at 23:19-24:1. For example, with respect to certain strains of HPV that cause cervical cancer, a viral copy is found in every tumor cell. Id. at 25:1-6; Jan. 24 Gazdar Tr. at 149:20-23. In contrast, viral copies are not found in all of the cells of tumors purportedly caused by SV40. In the tests on Mr. Gannon's tumor, for example, Dr. Gazdar testified that "perhaps" he found less than one viral copy per ten tumor cells. Jan. 24 Gazdar Tr. at 149:8-12. Dr. Garcea recalculated this figure and determined that Dr. Gazdar actually found far fewer than one copy per ten tumor cells; instead, one SV40 viral copy was detected in every 3,000 to 15,000 tumor cells. Garcea Tr. at 34:12-35:4, 75:2-76:7.

37. The second factor, cell immortalization, is satisfied when the viral genome is placed into cells that then continue to divide indefinitely. zur Hausen Tr. at 25:10-26:3. While this factor has been satisfied in some animal models, SV40 immortalizes human tissue cells slowly and inefficiently. See id. at 25:25-26:3. Satisfying this factor alone, moreover, fails to

demonstrate that SV40 causes cancer, as "it needs to be considered in the context of the other [criteria], as well." Id. at 26:4-8.

38. The third factor, whether the infected cells return to a normal state when the cancer-causing agent in the virus is blocked, has not been met with respect to SV40 and human cancer. Id. at 27:23-25. This factor demonstrates that the oncogene is actually causing the cancer, and is "the strongest point in favor of a causal role of [the] agent in the respective type of tumor." Id. at 26:19-27:15. For example, in HPV 16 and 18, this factor was satisfied "convincingly." Id. at 27:16-22.

39. The fourth and final factor, epidemiological evidence that the viral infection represents the prime risk factor for development of the tumor at issue, has not been met with respect to SV40 and human cancer, including medulloblastoma.

40. The 2002 IOM Report on SV40 and cancer concluded "that the evidence is inadequate to accept or reject a causal relationship." U.S. Ex. 200, IOM Report, at 6; accord Jan. 23 Gazdar Tr. at 149:5-11; Halsey Tr. at 13:21-25. According to the IOM Report, "[i]f the evidence is not reasonably convincing either in support of or against causality, the category 'inadequate to accept or reject a causal relationship' is used. Evidence that is sparse, conflicting, of weak quality, or merely suggestive either toward or away from the causality falls into this category." U.S. Ex. 200, IOM Report at 23; see also Halsey Tr. at 60:11-61:13.

41. In reaching its conclusion, the IOM found that the available epidemiologic studies had a number of substantial limitations. See generally Halsey Tr. at 14:21-15:19. Most of the epidemiologic studies cited in the IOM Report were "ecologic" studies, which relied upon participants' birth year to determine if he or she received SV40-contaminated vaccine. Id. at 31:9-

32:11; Garcea Tr. at 116:17-117:19, 180:17-21; Jan. 23 Gazdar Tr. at 145:18-148:2; U.S. Ex. 200, IOM Report at 5-6, 33-59. This methodology does not accurately reflect who was actually exposed to SV40. Halsey Tr. at 31:9-32:11, 123:4-124:11; Jan. 23 Gazdar Tr. at 147:10-15. Other epidemiologic studies cited in the IOM Report relied upon blood tests for SV40 exposure; however, these serology tests were not specific for SV40, and the tests could "cross-react" with the JC and BK viruses, i.e., purportedly test positive for one virus when the test actually detected a different virus. Halsey Tr. at 31:9-32:11, 123:4-124:11. "[A]lmost every human on the planet is infected with" the JC and BK viruses, yet "they cause no known disease in normal people." Garcea Tr. at 25-20-27:7. Therefore, these tests likely resulted in false positives for SV40. Halsey Tr. at 31:9-32:11, 123:4-124:11.

42. While the IOM Report concluded that, based on the biological evidence, it is plausible that SV40 could contribute to some human cancers, the IOM specifically emphasized that any conclusion that SV40 causes human cancer must also be supported by epidemiological evidence:

> When other evidence of causality is available, biological data add supportive evidence, but they cannot prove causality on their own.
>
> This committee is often faced with a set of circumstances in which the epidemiologic evidence is judged inadequate to accept or reject a causal association between vaccine exposure and an adverse event of concern. It is then left with the task of examining proposed or conceivable biologic mechanisms that might be operating *if* an epidemiologically sounds association could be shown between a vaccine exposure and and adverse event.

U.S. Ex. 200, IOM report at 24 (emphasis in original); Halsey Tr. at 65:8-24; U.S. Ex. 200, IOM Report at 6-11, 59-69; accord id. at 59 ("Although biological data do not provide an independent basis for evaluating causality, they can help validate epidemiologically based conclusions for or

against causal associations. Such data can also guide further investigation when epidemiologic evidence is inconclusive."). Moreover, the IOM Report recognized that some of the biological evidence was questionable:

> The detection of SV40 in tumors does not, by itself, demonstrate a causal relationship. SV40 could be a passenger virus, infecting the cells but causing no pathology. Findings from studies examining SV40 in mesothelioma demonstrate a great deal of variability which precludes the ability at present to draw conclusions regarding the frequency with which SV40 can be detected in specific neoplasms and/or normal tissues in humans. Some studies have detected SV40 in normal tissue from healthy subjects (Martini et al., 1996, Woloschak et al, 1995). Its detection in multiple types of tumors (i.e. its lack of specificity for a single type of cancer) also leads to doubts about a causal link (Strickler, 2001b).

U.S. Ex. 200, IOM report at 9; accord Halsey Tr. at 69:9-70:21.

43. Dr. Gazdar opined that the purported link between SV40 and human cancer was strengthened by the similarity of tumors caused in rodents and humans. Jan. 23 Gazdar Tr. at 121:4-122:23. Both Drs. Garcea and zur Hausen convincingly refuted this proposition. Garcea Tr. at 54:8-56:9; zur Hausen Tr. at 39:3-40:1. Dr. zur Hausen pointed out that investigators' choice of tumors is biased because research was focused on human tumors that had been previously induced in rodents. zur Hausen Tr. at 39:3-40:1. Dr. Garcea also noted that SV40 does not even cause medulloblastoma in rodents. Garcea Tr. at 56:1-6.

44. Because humans and rodents are inherently different, the results of rodent studies cannot be extrapolated to humans. Garcea Tr. at 54:8-56:9; zur Hausen Tr. at 39:3-40:1. Dr. Garcea likened Dr. Gazdar's reasoning to "comparing apples and oranges." Garcea Tr. at 54:8-55:9. Indeed, Dr. Gazdar conceded that the "mechanism by which the virus causes cancer in hamsters and humans is very different." Jan. 24 Gazdar Tr. at 81:17-22.

45. Importantly, rodent studies are conducted in a manner much more likely to result in a tumor. Garcea Tr. at 54:8-56:9. SV40 is injected directly into newborn rodents, which are extremely susceptible to the effects of SV40 at this stage in their development. Id. In contrast, oral polio vaccine is ingested orally. Moreover, newborn rodents are not age-equivalent to newborn humans. See id. Because of rodents' gestational development, they are more similar to humans born prematurely. See id.

46. In a recent study, investigators failed to detect SV40 in any of the medulloblastoma tumors that they tested. This study relied on a full range of state of the art tests, including real-time PCR, PCR immunohistochemistry and sensitive serological testing. Garcea Tr. at 39:21-42:1; J.Y. Kim, et al., Medulloblastomas and Primitive Neuroectodermal Tumors Rarely Contain Polyomavirus DNA Sequences, 4 Neuro-oncology 165 (2002) (The only portions of this article admitted into evidence are those that were testified to by the witnesses at trial).

47. A 2005 study used PCR tests to determine the presence of SV40 in 225 brain tumor samples, including 21 medulloblastoma tumors. Rollison, et al., Investigation of Human Brain Tumors for the Presence of Polyomavirus Genome Sequences by Two Independent Laboratories, 113 Int.'l J. Cancer 769 (2205); U.S. Ex. 222 at 769; Halsey Tr. at 41:14-24, 42:12-14. Each of the 225 tissues was sampled by two independent laboratories using PCR testing, masked positive controls, and masked negative controls, thereby reducing the possibility of laboratory contamination and false positives. U.S. Ex. 222 at 770; Halsey Tr. at 42:15-43:8. Of all 225 samples tested, only four tumors tested positive for SV40; however, no sample tested positive for SV40 in both laboratories. U.S. Ex 222 at 770; Halsey Tr. at 43:9-21. Although one medulloblastoma tested positive for SV40 in each laboratory, no medulloblastoma tested positive

for SV40 in both laboratories. U.S. Ex. 222 at 770; Halsey Tr. at 43:9-21.

48. Dr. Gazdar acknowledged in this Rollison study, U.S. Ex. 222, the authors concluded that "[o]ur findings suggest that JCV, BKV and SV40 are not present in most brain tumors." U.S. Ex. 222 at 773; Jan. 24 Gazdar Tr. at 85:24-86:11. Dr. Gazdar also testified that the authors of an earlier Rollison study, U.S. Ex. 254, concluded that "[t]here was not evidence of an association between the presence of antibodies to polyomaviruses up to 22 years before the diagnosis of cancer and the subsequent development of brain tumors." U.S. Ex. 254 at 462; Jan. 24 Gazdar Tr. at 89:1-90:4. Dr. Gazdar felt that there was a cloud over these studies because one of the ten authors he contended had a conflict of interest. Jan. 24 Gazdar Tr. at 86:2-88:8.

49. The Eric A. Engels study looked at possible association between SV40 and human brain tumors in northern India. The residents of northern India are at risk for SV40 infection because they live in close proximity to SV40-infected rhesus monkeys. U.S. Ex. 230 at 351; Jan. 24 Gazdar Tr. at 90:5-25. The authors studied 33 ependymomas, 14 choroid plexus tumors, and 18 control brain tumors. U.S. Ex. 230. Real-time PCR was used to detect and quantify SV40. Id. SV40 was detected in one ependymoma specimen; however, fewer than one SV40 viral copy was found per 350 tumor cells. Id. In addition, this finding could not be reproduced, suggesting that the positive result may have been due to laboratory contamination. Id. The authors concluded that "[o]ur results do not support a role for SV40 in human brain tumors in northern India." Id. at 350; Jan. Tr. at 91:2-9.

50. Dr. Gazdar, himself, participated in a study in which medulloblastoma tumors were tested for SV40. Jan. 24 Gazdar Tr. at 78:12-79:17. There, Dr. Gazdar reported that just one of sixteen medulloblastoma tumors tested positive for SV40. Id.; Shivapurkar, N., et al.,

Presence of Simian Virus 40 DNA Sequences in Human Lymphomas, Lancet 359:851 (2002) (The only portions of this article admitted into evidence are those that were testified to by the witnesses at trial).

51. Dr. Gazdar failed to consider the most recently published sero-epidemiological studies. Sero-epidemiological studies use blood testing to determine whether "the respective human population has been exposed to a certain agent." zur Hausen Tr. at 22:24-23:4. If SV40 causes human tumors, a sero-epidemiological study should find SV40 antibodies in a greater proportion of individuals with tumors than in a control population of individuals without tumors. Garcea Tr. at 52:2-54:7.

52. In the 2003 Rollison study, the authors examined blood samples from 44 people who suffered brain tumors for possible SV40 infections. U.S. Ex. 254 at 461. To do so, the authors used a new, sensitive serology test that distinguishes between SV40, the BK virus, and the JC virus. Id. at 460-461. In addition, the authors used two control subjects for each tumor whose samples were masked to prevent investigator bias. Id. at 461; Halsey Tr. at 33:25-34:12, 35:24-36:7. The investigators found that no statistically significant differences exist between cases and controls for the presence of SV40 antibodies. U.S. Ex. 254 at 462; Halsey Tr. at 35:13-35:19 ("The findings were that the percentage of people who had brain tumors who had antibody to SV40 were basically identical to the controls."). In conclusion, the authors wrote that "no association with brain tumors was observed" as a result of SV40 infection. U.S. Ex. 254 at 462; Halsey Tr. at 35:20-35:23.

53. In the 2003 Carter Study, which was co-authored by Dr. Garcea, the authors examined whether SV40 could be found in 122 osteosarcoma and 90 prostate cancers. U.S. Ex.

246 at 1522; Garcea Tr. at 23:10-19. To do so, the authors again used a serology test that differentiated between SV40 antibodies and the antibodies produced for the related JC and BK polyomaviruses. U.S. Ex. 246 at 1523-28. The authors used 487 control subjects. Id. at 1523. The study found that positive results for "SV40 [were] lower among the osteosarcoma patients than among the control subjects." Id. at 1526; Garcea Tr. at 23:21-24:9. Similar findings were made with respect to the prostate tumors. U.S. Ex. 246 at 1526; Garcea Tr. at 23:21-24:9. Accordingly, the authors concluded that no association existed between SV40 and these tumors, as their "data could not confirm the presence of SV40-specific antibodies in the general population or in individuals with prostate cancer or osteosarcoma." U.S. Ex. 246 at 1528; Garcea Tr. at 24:10-25:19; zur Hausen Tr. at 29:11-30:13, 34:3-18.

54. In the 2005 Rollison study investigating Non-Hodgkin Lymphoma ("NHL"), the authors - - including Dr. Halsey - - tested serum samples from 170 individuals who contracted NHL. U.S. Ex. 249 at 1449; Halsey Tr. at 36:20-37:14. This study addressed the "cross-reactivity" and "ecologic" flaws, by testing people born after 1963 with the sensitive serology test that distinguished SV40 from the JC and BK viruses. Halsey Tr. at 37:8-14, 103:1-104:21. The authors used two control subjects for each patient. The samples were masked to prevent investigator bias. U.S. Ex. 249 at 1449; Halsey Tr. at 37:18-38:2, 40:9-21. A number of prominent scientists, including one of Plaintiffs' experts, Dr. Janet Butel, reviewed and approved the study design before it was conducted. Id. at 39:6-23. Based on the testing, only 1.8% of the patients whose tumor samples had tested positive for SV40 had SV40 antibodies, while 1.6% of the controls tested positive for SV40 antibodies. U.S. Ex. 249 at 1448; Halsey Tr. at 38:5-9. The authors wrote "[i]f SV40 infection is truly associated with NHL, then SV40 antibody levels

should be higher among NHL cases than controls in a case-control study, regardless of route of SV40 infection." U.S. Ex. 249 at 1451; accord Garcea Tr. at 52:2-54:7. Therefore, the authors concluded that "[o]ur results indicate that past SV40 infection is not associated with the development of NHL." U.S. Ex. 249 at 1452; see also Halsey Tr. at 38:5-15.

55. Numerous investigators in the past five years have failed to detect SV40 DNA sequences in human tumors. Garcea Tr. at 25:6-19. These published studies conflict with earlier studies reporting higher frequencies of SV40 DNA sequences detected in a range of human tumors. Id. Both Drs. Garcea and zur Hausen testified that the relative lack of negative reports in earlier years had more to do with the nature of scientific publications - - many negative reports were not previously published because scientists typically only publish when the agent of interest is detected. Id.; zur Hausen Tr. at 10:18-11:25. Indeed, Dr. zur Hausen testified that his laboratory had conducted a series of experiments in the 1990s looking for the presence of SV40 in human tumors, but the virus went undetected. zur Hausen Tr. at 10:18-11:25. Those negative results were never published. Id.

56. Both Drs. Garcea and Gazdar divide the biological reports into "pre-PCR era" and "PCR era" studies. Garcea Tr. at 25:20-28:3; Jan. 23 Gazdar Tr. at 138:19-140:1. Reports from the pre-PCR era have questionable scientific value. Garcea Tr. at 26:6-27:9; Jan. 23 Gazdar Tr. at 138:19-140:1. These studies were typically suspect because the tests used to detect SV40 were cross-reactive with the BK and JC human polyomaviruses. In other words, a positive test result could not necessarily distinguish among JC, BK, or SV40. Consequently, no "cause-effect" association can be made with any of these other studies. Garcea Tr. at 26:6-27:9; Jan. 23 Gazdar Tr. at 138:19-140:1; 140:20-141:4; U.S. Ex. 4, Garcea Expert Report at 7.

57. Likewise, a recent review article by James DiCaprio and Danielle Poulin concluded that the evidence is inadequate that SV40 plays a role in human cancer and there is no strong evidence in support of its role in human tumors. zur Hausen Tr. 35:13-15; 38:16-39:2; Danielle L. Poulin, et al., Is There a Role for SV40 in Human Cancer?, 24 J. Human Oncology 4356 (2006) (The only portions of this article admitted into evidence are those that were testified to by the witnesses at trial).

58. Another article entitled "Mesothelioma Mortality in Europe: Impact of Asbestos Consumption and Simian Virus 40" concluded that "using currently-existing data on SV40 prevalence, no association between SV40 prevalence and asbestos-corrected male pleural cancer [mesothelioma]." Jan. 24 Gazdar Tr. at 96:9-97:17; Katharina Leithner, et al., Mesothelioma Mortality in Europe: Impact of Asbestos Consumption and Simian Virus 40, 1 Orphanet J. of Rare Diseases 44 (2006) (The only portions of this article admitted into evidence are those that were testified to by the witnesses at trial).

59. Another study, published by Dr. Fernando Lopez-Rios and others, concluded that the "inability to detect SV40 T-antigen transcripts or proteins and our essentially negative SV40 T-antigen DNA PCR results with the primer pair not prone to plasmid contamination suggest that SV40 is at most rarely present in human mesotheliomas." Jan. 24 Gazdar Tr. at 114:1-9; Fernando Lopez-Rios, et al., Evidence Against a Role for SV40 Infection in Human Mesotheliomas and High Risk of False-Positive PCR Results Owing to Presence of SV40 Sequences in Common Laboratory Plasmids, 364 Lancet 1157 (2004) (The only portions of this article admitted into evidence are those that were testified to by the witnesses at trial).

60. The Lopez-Rios study also discovered that ordinary laboratory plasmid

contamination could account for many of the positive results in the early SV40 studies. See Garcea Tr. at 36:6-37:7. These authors first tested their mesothelioma samples using two sets of primers targeting a specific region of the SV40 genome and obtained positive results for 56%-62% of the sample tissues. Jan. 24 Gazdar Tr. at 110:18-111:14. The sequences amplified by these two sets of primers, however, are within the section of the SV40 genome that is included in many common laboratory-engineered plasmids - - that is, portions of DNA that are created for use in research. These plasmids are present in research laboratories throughout the world. Id. at 111:24-112:13; Garcea Tr. at 36:6-37:7. The investigators conducted additional tests looking for sections of SV40 DNA that are outside of this particular research region and found that the mesothelioma samples were essentially negative for SV40; dropping from 62% to 6%. Jan. 24 Gazdar Tr. at 112:14-113:1. After confirming the results using immunohistochemistry tests, they concluded that the original primers were at "high-risk" for providing false-positive data. Id. at 113:2-25. The authors indicated that at least some of the previously published reports with high rates of SV40 positivity were because of plasmid contamination. Id. at 113:23-114:18.

61. These findings were confirmed by the Aoe study, which Dr. Gazdar co-authored. Jan. 24 Gazdar Tr. at 92:7-14; Keisuke Aoe, et al., Infrequent Existence of Simian Virus 40 Large T Antigen DNA in Malignant Mesothelioma in Japan, 97 Cancer Science 292 (2006) (The only portions of this article admitted into evidence are those that were testified to by the witnesses at trial). "In conclusion, [this study] found a low rate of SV40 infection in Japanese malignant mesothelioma specimens, suggesting that SV40 may not be involved in the pathogenesis of malignant mesothelioma in Japan." Jan. 24. Gazdar Tr. at 91:22-92:14.

62. Plaintiffs' expert, Dr. Gazdar, rendered an opinion that, "Based on my review

of relevant and reliable literature it is my opinion to a reasonable degree of scientific and medical certainty that SV40 plays a causal role in the substantive human tumors in which it had been frequently found including brain tumors. And I wrote (indiscernible). I meant to say medulloblastomas." Jan. 23 Gazdar Tr. at 158:7-12. Dr. Gazdar went on to state that "based on the above it is my opinion to a reasonable degree of scientific and medical certainty that SV40 played a causal role in the development of Jamie Gannon's medulloblastoma. Id. at 158:24-159:1.

63. The only tests that Dr. Gazdar conducted to determine whether SV40 was the specific cause of Mr. Gannon's medulloblastoma were PCR tests to detect the presence of SV40 DNA in Mr. Gannon's tumor tissue. *See, e.g.,* Garcea Tr. at 85:25-86:5. Dr. Gazdar was unable to point to any methodology by which the mere detection of SV40 DNA in a tumor is enough to prove specific causation. Moreover, the design of Dr. Gazdar's PCR test also creates substantial doubts as to whether what he detected in Mr. Gannon's tumor was actually SV40.

64. Both Dr. zur Hausen and Dr. Garcea testified that, to a reasonable degree of medical certainty, SV40 did not specifically cause Mr. Gannon's medulloblastoma. zur Hausen Tr. at 43:5-8, Garcea Tr. at 91:9-14.

65. Drs. Garcea and zur Hausen testified that because of the substandard design of Dr. Gazdar's PCR tests, the evidence failed to establish that SV40 was even detected in Mr. Gannon's tumor tissue. zur Hausen Tr. at 43:14-44:1, Garcea Tr. at 84:18-87:6. Dr. Garcea additionally testified that, even if SV40 was convincingly detected in the tumor, Dr. Gazdar failed to rule out laboratory contamination as the source of the virus. See generally, Garcea Tr. at 67:21-68:16; 71:11-72:6; 88:24-90:3.

66. After examining Dr. Gazdar's testing methodology, Dr. zur Hausen testified

that he "wouldn't permit a PhD student in [his] group to base a statement on this type of demonstration." sur Hausen Tr. at 43:9-44:1; see also id. ("Working in this field for quite some time, I am aware of so many artifacts which may occur in this type of technique that, without sufficient additional controls, I would not at all be convinced that this test provides you with a real result."). Dr. Garcea similarly concluded that SV40 studies conducted over a decade ago were more precise than the tests conducted by Dr. Gazdar on Mr. Gannon's tumor. Garcea Tr. at 35:16-36:5 ("Dr. Gazdar's analysis was basically less than the experiments that we had done in 1992.").

67. The SV40 genome consists of approximately 5,200 DNA base pairs. Garcea Tr. at 32:1-5; Jan. 24 Gazdar Tr. at 116:20-22. Because researchers cannot practically detect all 5,200 base pairs when conducting PCR tests, they must examine discrete portions of the genome to determine whether "SV40-like sequences" are present. Garcea Tr. at 31:16-25. Typically, researchers examine approximately 170 SV40 base pairs when conducting PCR testing. Id. at 32:6-16.

68. Moreover, as a standard practice, researchers use several different sets of primer pairs that target disparate regions of the SV40 genome to determine whether authentic SV40-like sequences are present. zur Hausen Tr. at 42:2-4; Garcea Tr. at 86:13-24.

69. Dr. Gazdar's PCR testing on Mr. Gannon's medulloblastoma used just one primer pair that targeted between 80 and 90 base pairs - - approximately 1.5% of the SV40 genome sequence. Jan. 24 Gazdar Tr. at 117:1-5; Garcea Tr. at 32:6-16, 85:6; zur Hausen Tr. at 43:9-44:1.

70. Most researchers would not conclude that SV40 had been identified on the

basis of the amplification of so few base pairs. zur Hausen Tr. at 43:14-44:1; id. at 123:16-22 ("[Y]ou test for 1.5 percent of the - - of the total DNA. And, so, you may end up with - - if you work for a long time with PCRs on tumors, and you take only very small pieces of the DNA, you would be surprised how many artifacts occur, most particularly with tumor specimens.").

71. The Lopez-Rios study, mentioned above, discovered that earlier researchers using PCR tests to detect SV40 often used primer pairs that could also yield a "positive" result if certain widely used laboratory plasmids were present in a sample. Garcea Tr. at 36:6-37:7. That study concluded that researchers "should be very wary of SV40 positivity in that plasmid contamination must be very stringently ruled out." Id.

72. Standard laboratory practice since the publication of the Lopez-Rios article requires researchers to rule out plasmid contamination when conducting PCR testing for SV40. Id.; see also zur Hausen Tr. at 31:12-25. To do so, researchers "need to design their primers in different ways outside of these regions that are common to many laboratory plasmids and also that, for example, that they should check and see whether their amplification reactions contain these plasmids by amplifying up genes that are common in these plasmids." Garcea Tr. at 37:8-18; see also zur Hausen Tr. at 31:21-35. If researchers continue to use "high risk" primers, it is essential that they also add additional primer pairs targeting portions of the SV40 genome that fall outside the "high-risk" region. zur Hausen Tr. at 32:1-8.

73. Dr. Gazdar used just one primer pair in testing Mr. Gannon's medulloblastoma for SV40. This primer pair fell within the Lopez-Rios "high-risk" area that could result in a false-positive test result. Garcea Tr. at 84:18-85:10. Despite recognizing that he only used a "high-risk" primer pair to test Mr. Gannon's tumor, Dr. Gazdar failed to rule out laboratory plasmid

contamination. Jan. 24 Gazdar Tr. at 116:23-25; Garcea Tr. at 37:8-20.

74. Moreover, Dr. Gazdar admitted that when he has used low risk primers in his own research, he has obtained far fewer positive SV40 results than when using Lopez-Rios "high-risk" primers. Jan. 24 Gazdar Tr. at 201:4-16.

75. Without ruling out laboratory contamination, Dr. Gazdar's conclusion that he detected SV40 DNA in Mr. Gannon's tumor - - based on PCR testing involving just one "high-risk"primer pair - - fails to meet the scientific standards in this field and is inherently unreliable. Garcea Tr. at 40:11-21; 85:11-24.

76. In addition to examining just 1.5% of the SV40 genome with a single "high-risk" primer pair, the portion of the SV40 genome that Dr. Gazdar targeted was also very similar, or "homologous," to certain human chromosomes. sur Hausen Tr. at 44:5-20.

77. Eighteen of the twenty-four base pairs used in Dr. Gazdar's "F" primer are identical to the human Chromosome 20 DNA sequence. Id. at 46:2-47:8. Eighteen of the twenty-four base pairs in the same F primer are identical to the human Chromosome 3 DNA sequence. Id. at 47:9-21. Eighteen of the twenty-three base pairs used in Dr. Gazdar's "R" primer are also identical to the human Chromosome 2 DNA sequence. Id. at 47:22-48:2. Dr. Gazdar's PCR test probe was also highly homologous to the human chromosome 18 DNA sequence; "for a long stretch, [the sequences are] virtually identical." Id. at 48:3-18.

78. Compounding this homology, medulloblastoma tumors ordinarily contain "chromosome rearrangements." Id. at 119:17-120:7. Because of the possible chromosome rearrangements and Dr. Gazdar's use of such a high number of PCR cycles, "you will end up with - - in all likelihood, with some [PCR] bands, in these cases, which, in the end, turn out to be

artifacts." Id. at 119:17-120:7.

79. Under the conditions used by Dr. Gazdar, his PCR test probe would, in all likelihood, detect human chromosomes. Id. at 46:2-47:8; 47:9-21; and 48:3-18. By only targeting a region of the SV40 genome that is homologous to human chromosomes, Dr. Gazdar failed to rule out the possibility that he was merely detecting human DNA - - as opposed to authentic SV40. Id. at 32:9-22.

80. SV40 is also closely homologous to two other human polyomaviruses, the JC and BK viruses. Id. at 32:23-33:16; Garcea Tr. at 26:6-27:7. The presence of either virus in a sample can produce false positive results for SV40 when conducting PCR testing. Garcea Tr. at 35:5-15; id. at 26:6-27:7 ("[T]hese viruses look identical to SV40 and their proteins are almost identical to those of SV40 and their nucleic acid, their DNA sequence is very similar to that of SV40".).

81. Even though standard practice requires testing for the JC and BK viruses in brain tumors when searching for SV40, Dr. Gazdar failed to conduct any such tests on Mr. Gannon's tumor sample. Id. at 35:5-15; zur Hausen Tr. at 33:11-16; Jan. 24 Gazdar Tr. at 155:1-7. Regardless of the specificity of his PCR primer pair, by using only one primer pair to target such a small section of SV40 DNA, Dr. Gazdar cannot rule out the possibility that his PCR tests detected JC or BK viral DNA instead of SV40. Garcea Tr. at 35:5-15.

82. When conducting his real PCR testing on Mr. Gannon's tumor, Dr. Gazdar only detected SV40 when he used more than 40 PCR cycles. Jan. 24 Gazdar Tr. at 148:21-149:5; Garcea Tr. at 79:19-80:8.

83. Dr. Garcea explained that "the way PCR works is that DNA is synthesized in

cycles, in rounds, and so these primers are put down on the DNA, allowed to amplify the DNA between them and then a new cycle is started by heating and cooling, denaturing the products of the DNA, re-hybridizing on the probes and starting new cycles, generating basically daughters of daughters of daughters of daughters of daughters of the original product." Garcea Tr. at 73:11-20. The risk of obtaining an "artifact" - - a by-product of the PCR testing - - is much more likely when conducting PCR testing above 40 cycles. zur Hausen Tr. at 119:17-120:6. As a result, positive results for SV40 generated after 40 PCR cycles are typically considered negative or inclonclusive. Garcea Tr. at 79:19-80:8.

84. Dr. Garcea testified that the number of cycles that Dr. Gazdar used in this case was "very, very high," and above the number that Dr. Garcea's laboratory would consider as yielding a true positive result. Id. at 74:8-14.

85. In addition, even if Dr. Gazdar's methodology was otherwise sound, he only detected between one viral copy per 3,000 to 15,000 cells in Mr. Gannon's tumor. Garcea Tr. at 74:1-17. This low viral detection rate undercuts Plaintiffs' argument that SV40 is causing the tumor. Garcea Tr. at 34:12-35:4; id. at 75:22-76:7 ("[H]ow can you have a tumor [caused by SV40] where 99.99 per cent of the cells don't have the virus in it?"). Finally, although it was not mentioned in his expert report, Dr. Gazdar also failed to detect SV40 using conventional PCR tests on the one section of Mr. Gannon's tumor that had previously tested positive in his real-time PCR testing. Id. at 84:8-17. The fact that Dr. Gazdar's results were not reproducible raises additional doubts as to the validity of his positive SV40 results on Mr. Gannon's tumor.

86. Mr. Gannon's tumor blocks were sectioned - - *i.e.*, cut into pieces suitable for laboratory testing - - in one of the core facilities at the University of Texas Southwestern Medical

Center. Garcea Tr. at 67:21-25; Jan. 24 Gazdar Tr. at 125:15-21. Dr. Gazdar is just one of many researchers who send samples to the core facility for sectioning. Garcea Tr. at 68:1-16; see generally Jan. 24 Gazdar Tr. at 129:9-15.

87. Dr. Gazdar failed personally to supervise the sectioning process at the core facility, and he did not know whether any SV40-related samples had been recently handled in the core facility before Mr. Gannon's tumor was sectioned. Id. at 138:15-23, 134:9-15.

88. Negative tissue block controls were not used during the sectioning of Mr. Gannon's tumor at the core facility. Garcea Tr. at 68:1-6; Jan. 24 Gazdar Tr. at 130:13-24. Dr. Garcea testified that using negative control tissue blocks is important because "including a control sample of tissue at the time of preparation of the sample being tested . . . is a necessary control to determine whether there might be fortuitous contamination at that point in the preparation of the DNA for testing." Garcea Tr. at 68:7-16. Even Dr. Gazdar admitted that negative controls were necessary for a researcher to confirm that a positive test result was not due to contamination. Jan. 24 Gazdar Tr. at 129:25-130:12.

89. Although Dr. Gazdar testified that it was standard practice to change blades before and during sectioning, he was unable to provide any contemporaneous records to show that the blades were actually changed before or during the sectioning of Mr. Gannon's tissue. Id. at 126:1-18. This failure to provide records is important, as Dr. Gazdar tested the core facility in 2006 and discovered SV40 on a cutting blade even after the blade had been decontaminated by the lab personnel. Id. at 137:2-138:8.

90. Dr. Gazdar also testified that the core facility did not routinely monitor for viral contamination; thus, researchers would be unaware if SV40 contaminated the core facility.

Id. at 133:1-24.

91. Accordingly, Dr. Gazdar could not exclude the possibility that there was SV40 contamination in the core facility prior to the sectioning of the Gannon tissue. Id. at 134:9-18. This raises additional doubts as to the validity of his positive SV40 results.

92. DNA from the sections cut in the core facility was extracted in Dr. Gazdar's laboratory. Dr. Gazdar conceded, however, that he grew SV40 in his own laboratory in 2003, and admitted that he subsequently found SV40 contamination and took steps to decontaminate his laboratory. Jan. 24 Gazdar Tr. at 142:6-21; accord Garcea Tr. at 193:2-22.

93. However, growing SV40 in a laboratory setting is "a very messy procedure." Garcea Tr. at 88:24-90:3. Once SV40 contamination has occurred, "it's very, very difficult to remove that contamination," and that it may take years to do so. Id. Scientists have found it necessary to shut down a laboratory for an entire year after such contamination. Id. at 193:23-194:6. Dr. Garcea testified that he did not "think any laboratory who's ever grown SV40 should do PCR in the laboratory," as the "DNA and virus can probably exist on your bench tops for years." Id. at 88:24-90:3.

94. No evidence was present at trial to suggest that Dr. Gazdar had shut down his laboratory for any extended period of time to ensure that SV40 contamination had been eliminated.

95. Despite having grown SV40 in his laboratory only two years previously, Dr. Gazdar failed to use negative tissue controls when extracting DNA from Mr. Gannon's tissue samples. Jan. 24 Gazdar Tr. at 139:6-9; Garcea Tr. at 71:18-21. This is critical because DNA extraction involves the removal of DNA from material in a series of steps during which

contamination can occur. An investigator can only rule out potential contamination by using negative controls during the extraction procedure. Since Dr. Gazdar did not use negative control during the DNA extraction he cannot rule out the possibility that contamination from his laboratory produced the positive SV40 results obtained on Mr. Gannon's tissue. Garcea Tr. at 71:22-72:6.

96. Dr. Gazdar admitted that using negative tissue controls during the DNA extraction process would have been "a useful . . . precaution" and "not a bad idea" Jan. 24 Gazdar Tr. at 139:10-20.

97. Plaintiffs did not conduct additional available tests on Mr. Gannon's tumor samples that, if positive, could have "strengthened" Plaintiffs' claim that SV40 played a role in the formation of Mr. Gannon's tumor (assuming proof of general causation). Jan. 24 Gazdar Tr. at 158:8-17:1; id. at 197:17-198:6.

98. For example, standard scientific practice requires serology testing to examine whether an SV40 antibody response exists in individuals suspected of having tumors caused by SV40. Garcea Tr. at 40:11-21 ("I think it's almost obligatory to include a study of antibodies"); accord id. at 87:7-88:3.

99. Plaintiffs failed to conduct serology tests to determine whether SV40 antibodies were present in Mr. Gannon's bodily fluids. Jan. 24 Gazdar Tr. at 15:25-16:17. If Mr. Gannon's medulloblastoma was caused by an SV40 infection, he likely would have developed detectable SV40 antibodies. Garcea Tr. at 22:11-23:6; 40:22-41:16; zur Hausen Tr. at 34:19-35:1.

100. Plaintiffs also failed to conduct immunohistochemistry tests on Mr. Gannon's tumor. These tests would have shown whether the SV40 allegedly detected in his tumor was

expressing the large T-antigen protein. If, contrary to the evidence, SV40 causes human cancer, the large T-antigen is the portion of SV40 thought to play a role in tumor formation. Id. at 30:2-16; 86:13-87:6. Dr. Garcea testified that standard scientific practice requires immunohistochemistry tests to determine whether a virus potentially plays a role in the formation of a tumor. Id. at 40:11-21. The only immunohistochemistry tests run on Mr. Gannon's tumor samples were negative for large T-antigen expression. Id. at 87:7-11. Without the expression of the large T-antigen protein, no evidence exists demonstrating that SV40 played a pathogenic role in this tumor. Garcea Tr. at 86:25-87:4.

101. Plaintiffs could have also conducted "very easy" PCR tests on Mr. Gannon's white blood cells to determine whether SV40 was circulating in his bloodstream. Id. at 87:12-88:14.

102. In his February 23, 2006 e-mail, Dr. Gazdar expressed his belief that these types of tests are important to making a case for specific causation. Dr. Gazdar wrote that he has "tried to impress on Don [MacLachlan, Plaintiffs' counsel] the importance of getting in live patients for extensive blood urine etc. and for immortalization of B lymphoblastoid cells. If we can conclusively determine the virus or evidence of virus exposure in a live patient your job (and mine) would be greatly simplified. . . . [T]here is a lot we could do to strengthen our findings, which have not even been discussed." U.S. Ex. 36.

103. Dr. Gazdar explained that if these additional tests were conducted and positive for SV40, it "would mean finding evidence of the virus or virus sequences or live virus in the patient's tissues, human blood, etcetera, which would indicate that the virus had established itself in the patient and had persisted." Jan. 24 Gazdar Tr. at 198:12-19.

104. Notwithstanding Dr. Gazdar's view that additional tests were needed to win these cases - - as Plaintiffs will have to "walk the extra mile" - - Dr. Gazdar failed to conduct any additional testing. U.S. Ex. 36; Jan. 24 Gazdar Tr. at 15:8-17:22.

CONCLUSIONS OF LAW

1. Jurisdiction for this Federal Tort Claims Act case is based upon 28 U.S.C. § 1346(b). Under the Federal Tort Claims Act, 28 U.S.C. §§ 2671, et seq., liability is determined in accordance with the substantive law of the place where the alleged negligent act or omission occurred. 28 U.S. C. § 1346(b)(1); see also, e.g., Matsko v. United States, 372 F.3d 556, 559 (3d Cir. 2004). In Richards v. United States, 369 U.S. 1, 11 (1962), the Supreme Court held that 28 U.S.C. § 1346(b) "requires application of the whole law of the State where the act or omission occurred." Here, the alleged negligent acts committed by the United States occurred in Maryland where the Food and Drug Administration is located. Maryland follows the lex loci delicti choice of law rule in tort cases. Hauch v. Connor, 453 A.2d 1207 (1983). This rule "requires a tort action to be governed by the substantive law of the state where the wrong occurred." Id. at 1209, "The place of injury is the place where the injury was suffered, not where the wrongful act took place." Johnson v. Oroweat Foods, Inc., 785 F.2d 503, 511 (4th Cir. 1986) (applying Maryland law). Here, because Mr. Gannon was vaccinated in Pennsylvania, the law of this state applies.

2. For Plaintiffs to recover under a negligence theory in Pennsylvania, they must demonstrate that: (1) a duty existed, (2) the United States breached that duty, (3) the United States' breach of the duty was the "cause in fact" of the injury, (4) the breach of the duty was "proximate cause" of the injury, and (5) damages resulting from the injury. Redland Soccer Club v. Dep't of the Army, 55 F.3d 827, 851 (3d Cir. 1995); Miterman v. United States, No. 01-5352,

2003 U.S. Dist. LEXIS 12052, at *17-18 (E.D. Pa. Mar. 6, 2003); R.W. v. Manzek, 346 888 A.2d 740, 746 (Pa. 2005).

3. Plaintiffs have the burden of proof on these issues by a preponderance of the evidence. Rippee v. Grand Valley Mfg. Co., 762 F.2d 25, 27 (3d Cir. 1985); Cope v United States Dep't of Justice, No. 99-CV-0238, 2000 U.S. Dist. LEXIS 10289, at *20-21 (E.D. Pa. July 13, 2000); Young v. Pennsylvania Dep't of Transp., 744 A.2d 1277, 1277-78 (Pa. 2000).

4. Under Pennsylvania law, Plaintiffs have the burden of proving general causation - - whether SV40 causes cancer and, particularly, medulloblastoma in humans. Kemmerer v. State Farm Ins. Co., No. 01-5445, 2004 WL 87017, at *3 (E.D. Pa. Jan. 19, 2004); Soldo v. Sandoz Pharm. Corp., 244 F.Supp. 2d 434, 524-25 (W.D. Pa. 2003); Heller v. Shaw Indus., Inc., No. 95-7657, 1997 WL 535163, at *6 (E.D. Pa. Aug. 18, 1997); Blum v. Merrell Dow Pharms., Inc.,705 A.2d 1314, 1316 (Pa. Super. Ct. 1997); see also Heller, 167 F.3d at 155 (explaining that general causation requires a showing that a particular agent causes a particular illness).

5. Under Pennsylvania law, Plaintiffs also have the burden of proving specific causation - whether SV40 caused Mr. Gannon's medulloblastoma. Kemmerer, 2004 WL 87017, at *3; Soldo, 244 F. Supp. 2d at 524-25; Heller, 1997 WL 535163, at *6; Blum, 705 A.2d at 1316.

6. "In a case involving complex issues of causation not readily apparent to the fact finder, plaintiff must present admissible expert testimony to carry her burden." Kemmerer, 2004 WL 87017, at *3 (citing Soldo, 244 F. Supp. 2d at 525); accord Guy M. Cooper, Inc. v. E. Penn Sch. Dist., 903 A.2d 608, 617 (Pa. Commw. Ct. 2006); see also In re Dobrowsky, 762 F.2d 90, 92 (3d Cir. 1984) ("Under Pennsylvania law . . . when an injury is not the 'obvious, natural or

probable result' of an accident, the plaintiff has the preliminary burden of demonstrating causation by introducing expert medical testimony.").

7. Federal Rule of Civil Procedure 52(c) allows for the entry of judgment on partial findings. That rule provides:

> If during a trial without a jury a party has been fully heard on an issue and the court finds against the party on that issue, the court may enter judgment as a matter of law against that party with respect to a claim or defense that cannot under the controlling law be maintained or defeated without a favorable finding on that issue, or the court may decline to render any judgment until the close of all the evidence. Such a judgment shall be supported by findings of fact and conclusions of law as required by subdivision (a) of this rule.

8. The advisory committee notes discussing Rule 52(c) state, in pertinent part, that:

> Subdivision (c) is added. **It parallels the revised Rule 50(a), but is applicable to non-jury trials. It authorizes the court to enter judgment at any time that it can appropriately make a dispositive finding of fact on the evidence. . . .** Judgment entered under this rule differs from a summary judgment under Rule 56 in the nature of the evaluation made by the court. A judgment on partial findings is made after the court has heard all the evidence bearing on the crucial issue of fact, and the finding is reversible only if the appellate court finds it to be clearly erroneous."

Fed. R. Civ. P. 52(c) advisory committee notes (emphasis added). Thus, since Plaintiffs were fully heard on the causation issue, the Court now "can appropriately make a dispositive finding" on this issue pursuant to Rule 52(c).

9. A trial court can resolve factual disputes under Rule 52(c). Rego v. ARC Water Treatment Co., 181 F.3d 396, 400 (3d Cir. 1999); accord Ritchie v. United States, 451 F.3d 1019, 1022-23 (9th Cir. 2006). An appellate court then "reviews a district court's [Rule 52(c)] findings of fact for clear error, and its conclusions of law de novo." Id. (internal citations omitted); accord

Bursztajn v. United States, 367 F.3d 485, 488-89 (5th Cir. 2004); see also Newark Branch, NAACP v. City of Bayonne, 134 F.3d 113, 119-20 (3d Cir. 1998) (explaining the appropriate standard of review for findings of fact for judgment granted on partial findings).

10. Plaintiffs were fully heard on the causation issues. Plaintiffs' only evidence on these issues came from their expert, Dr. Adi Gazdar.

11. Plaintiffs have failed to demonstrate, by a preponderance of the evidence, that SV40 causes cancer, let alone medulloblastoma, in humans. Dr. Gazdar's opinion that SV40 generally causes medulloblastoma is not supported by the evidence produced in this case.

12. "Well-recognized" criteria for determining causality which rely upon epidemiological and biological evidence, such as the Bradford Hill criteria and Dr. zur Hausen's criteria, have not been met with respect to SV40 and human cancer. Even Dr. Gazdar admits that many of the Bradford Hill criteria have not been met. More significantly, although he recognizes the importance of epidemiological evidence, Dr. Gazdar concedes that supporting epidemiological evidence for this general causation opinion does not exist.

13. Dr. Gazdar acknowledges that the IOM Report concluded that available epidemiologic evidence was "inadequate to accept or reject a causal relationship between" SV40 and human cancer. However, in contrast to the explicit statements in the IOM Report explaining that biological data "cannot prove causality on their own," Dr. Gazdar relies solely on biological evidence.

14. Dr. Gazdar's opinion ignores recent sero-epidemiological evidence which has not detected a higher incidence of SV40 infections in tumor samples than in control samples. These recent studies address the flaws cited in the IOM Report concerning earlier "ecological"

epidemiological studies, and demonstrate that SV40 is not causing human tumors.

15. Dr. Gazdar's opinion is inconsistent with recent biological evidence concluding that SV40 is not associated with human tumors. Most of the recent studies failed to detect SV40 in human tumors when conducting PCR testing. These studies have addressed the plasmid contamination issues discovered by Lopez-Rios. Significantly, Lopez-Rios' findings suggest that many of the older reports detecting SV40 in human tissues were actually detecting ordinary laboratory contamination.

16. Dr. Gazdar's opinion is inconsistent with studies that have failed to detect SV40 in human brain tumors - - including medulloblastoma. The recent articles, which take into account findings made by Lopez-Rios, have found, at best, sporadic evidence of SV40 in human brain tumors.

17. Dr. Gazdar's opinion relies on experiments conducted on rodents. However, even Dr. Gazdar admits that animals form tumors in a different manner than humans and that the results of these rodent studies cannot be extrapolated to humans. Moreover, SV40 does not even cause medulloblastoma tumors in rodents.

18. In contrast, the United States presented evidence from Robert Garcea, M.D., Harald zur Hausen, M.D., D. Sc., and Neal Halsey, M.D. (who was the only epidemiologist to testify in this case). These experts all relied upon the established scientific framework that relies on both epidemiological and biological evidence in determining causality. All three experts testified that the epidemiological evidence does not support the conclusion that SV40 causes human cancer. I find their testimony in this regard credible based upon sound reasoning and experience and I accept it.

19. Drs. Garcea and zur Hausen also testified that the biological evidence does not support the conclusion that SV40 causes human cancer. Both experts pointed out the many flaws and inconsistencies in Dr. Gazdar's opinion. Both experts also discussed newer serological testing that distinguishes between the common JC and BK viruses and SV40. This recent development, as well as the findings made in the Lopez-Rios article, demonstrate that it is highly unlikely that researchers were finding authentic SV40 DNA in the tumor samples studied in the older articles upon which Dr. Gazdar relies. I find their testimony in this regard credible based upon sound reasoning and experience and I accept it.

20. Plaintiffs also have failed to demonstrate by a preponderance of the evidence that SV40 caused Mr. Gannon's medulloblastoma. I find that Dr. Gazdar failed to conduct accurate, complete and scientifically reliable testing on Mr. Gannon's medulloblastoma sample.

Plaintiffs have failed to meet their burden on the dispositive issues of general and specific causation. Judgment is granted in favor of the United States.

UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF PENNSYLVANIA

JAMIE GANNON and REBECCA GANNON	:	CIVIL ACTION
	:	
vs.	:	
	:	
UNITED STATES	:	NO. 03-6626

O R D E R

AND NOW, this 17th day of July, 2007, for the reasons set forth in the foregoing Memorandum, Findings of Fact, and Conclusions of Law, Judgment is hereby entered in favor of the Defendant United States and against the Plaintiffs' Jamie Gannon and Rebecca Gannon.

BY THE COURT:

/s/ Robert F. Kelly
ROBERT F. KELLY
SENIOR JUDGE