In the United States Court of Federal Claims

No. 01-357 V

(Filed Under Seal: May 3, 2004)

(Reissued: May 18, 2004)[1]

EMMA HART, as Representative	*	
of the Estate of MANASSEH MICLEA,	*	National Childhood Vaccine Injury Act;
Deceased,	*	Measles, mumps and rubella (MMR)
Petitioner,	*	vaccination; Causation of fatal condition known
	*	as hemophagocytic lymphohistio-cystosis
v.	*	(HLH); *Knudsen*; Improper use of "bare" or
	*	"naked" statistics; Unreliable random
SECRETARY OF THE DEPARTMENT	*	observations; Findings arbitrary and capricious
OF HEALTH AND HUMAN SERVICES,	*	and contrary to law; Remand for further findings
	*	consistent with opinion.
Respondent.	*	

OPINION

Richard Gage, Gage and Moxley, P.C., Cheyenne, Wyoming, for petitioner.

Traci Manning, Torts Branch, Civil Division, U.S. Department of Justice, Washington, D.C., for respondent, with whom was *Assistant Attorney General Peter D. Keisler*.

ALLEGRA, Judge:

"It is vain to do with more what can be done with fewer."

[1] An unredacted version of this opinion was issued under seal on May 3, 2004. The court afforded the parties an opportunity to propose redactions in the opinion prior to its publication, but no such redactions were proposed. Accordingly, the opinion is reissued solely with the correction of a few typographical errors.

So said the mediaeval philosopher William of Occam, known for his popular razor, which can lead medical doctors to choose parsimoniously from a set of otherwise equivalent diagnoses the simplest one.[2] But, is this simplest diagnosis always the right one – under the law?

In this vaccine case, before the court on petitioner's motion for review, the diagnosis accepted by the Special Master hinged on probabilistic statistics that appeared to show that it was more likely that the tragic death of a young boy was not the result of the measles, mumps and rubella (MMR) vaccine, but an unrelated virus. Because the Special Master's reliance on such statistics was not only incompatible with the decisional law, but particularly inapropos given the shortcomings of the statistics themselves, the court believes that a razor of a different sort must be plied here – one that transects from the factual findings here any taint attributable to the statistics in question. This matter accordingly is remanded to allow the Special Master, in the first instance, to perform this task in rendering new findings.

I. FACTUAL AND PROCEDURAL BACKGROUND

On January 1, 1998, Manasseh Miclea was born to Emma Hart and, by all accounts, was a normal, healthy boy for the first fifteen months of his life. On April 10, 1999, Manasseh received several vaccinations, including a measles, mumps, rubella (MMR) immunization. During the next several days, Manasseh's mother called his pediatrician once or twice because he was not feeling well and had begun to develop a rash. On April 22, 1999, Manasseh was brought to his pediatrician's office. Fever and rash are common reactions to the measles vaccine and usually appear one or two weeks post-vaccination and, apparently for that reason, Manasseh's treating pediatrician recorded on April 22, 1999, that his symptoms likely constituted either a "viral exanthem," *i.e.*, a rash due to a virus, or an "MMR reaction." During the ensuing two weeks, his parents made several calls to the pediatrician, and he was taken into the office on May 2 and May 5, 1999, with symptoms including crying, fever, vomiting, red throat, rash, and swollen gums. More symptoms developed, prompting additional physician visits on May 6, 7, 8 and 9, 2000. Finally, Manasseh was hospitalized on May 9, 1999, and would remain so, except for a brief release on May 20, until his untimely death on June 20, 1999. It was determined, and it is not disputed, that Manasseh's death was the result of a condition known as hemophagocytic lymphohistiocystosis (HLH).

Certain basic facts regarding HLH are undisputed. HLH is a rare, and generally fatal, disease primarily affecting infants. The disease is not fully understood, but appears to result from the defective, overreactive operation of the victim's immune system. Cases of HLH appear to be related to some stimulus such as an active infection. Such infections produce "cytokines," a type of hormone which is strongly suspected as the trigger for the disease.

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[2] *See* Principia Cybernetica, Occam's Razor, at http://pespmc1.vub.ac.be/OCCAMRAZ.html.

On June 14, 2001, petitioner filed a claim under the National Childhood Vaccine Injury Act of 1986, 42 U.S.C. §§ 300aa-1 to 300aa-34 (the Vaccine Act), contending that Manasseh's death was caused by his vaccinations of April 10, 1999. The Secretary of Health and Human Services (respondent) contested that claim. An evidentiary hearing was held on November 8, 2002; following posthearing briefs, a second hearing to clarify various expert opinions was held on September 2, 2003. Special Master Hastings issued two opinions denying relief: the first on October 1, 2003, and the second, responding to petitioner's motion for reconsideration, on November 24, 2003. In these opinions, he concluded that petitioner had failed adequately to establish that Manasseh's MMR shot triggered the HLH and found instead that it was likely that the HLH was triggered by an infection caused by the Epstein-Barr virus (EBV). In reaching this conclusion, the Special Master noted that because petitioner's claim did not involve a so-called "table injury,"[3] petitioner was required to show that the MMR vaccine "actually caused," or "caused-in-fact," Manasseh's death. 42 U.S.C. § 300aa-13(a)(1), § 300aa-11(c)(1)(C)(ii). As the Special Master further explained, petitioner did not need to prove that the vaccine was the only cause, or even the predominant cause, of Manasseh's death, but only that the MMR shot was at least a "substantial factor," and a "but for" cause. *See Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999).

In analyzing this case, Special Master Hastings began by noting that there was evidence that a vaccine like the MMR could trigger HLH and that the nature and timing of the symptoms experienced by Manasseh were consistent with a reaction to the measles or rubella vaccinations. The Special Master, indeed, indicated that were there not evidence that Manasseh had EBV, he likely would have awarded compensation, stating "based upon the HLH literature and the expert testimony contained in the record of this case, if there were no indication that Manasseh was infected by the Epstein-Barr virus, then I likely ***would*** conclude that Manasseh's HLH was probably triggered by one of his vaccinations received on April 10, 1999." *Hart v. Sec'y of Health & Human Servs.*, 2003 WL 23218077, at *3 (Fed. Cl. Spec. Mstr. Nov. 24, 2003) (emphasis in original). He noted that in two other vaccine cases, special masters had found that vaccinations triggered cases of HLH, and that one of these cases specifically involved the MMR vaccine. *See Ackley v. Sec'y of Health & Human Servs.*, 2002 WL 985435 (Fed. Cl. Spec. Mstr. Apr. 29, 2002) (MMR vaccination); *Gall v. Sec'y of Health & Human Servs.*, 1999 WL 1179611 (Fed. Cl. Spec. Mstr. Oct. 31, 1999) (DPT and/or OPV vaccinations). The Special Master held, however, that those cases were *sui generis* because, unlike the case *sub judice*, there was no proof that the children in those cases were infected with Epstein-Barr.

[3] Table injuries are those listed in the "Vaccine Injury Table" of 42 U.S.C. § 300aa-14(a) (as administratively altered). If an injury listed on that table occurs within a prescribed time, there is a presumption that the injury is compensable under the Vaccine Act. In other words, petitioners seeking redress for table injuries do not carry the burden of establishing a *prima facie* case of causation.

In concluding that, at the time of the onset of his fatal HLH, Manasseh was infected with EBV, the Special Master relied on statistics indicating that EBV "is by far the most common cause of HLH." He first relied on these statistics independently, observing:

> First, I note that the medical literature introduced by both parties, along with the testimony of the experts, indicates that one particular virus, the Epstein-Barr virus, . . . has been identified as by far the most common trigger for HLH. Dr. Byers acknowledged that, as Dr. Berger indicated, Epstein-Barr virus has been the most commonly identified trigger. Dr. Byers seemed to estimate that, based upon her review of the literature, in about 50% of HLH cases Epstein-Barr virus is identified as the trigger. One article submitted by petitioner's reviewed 219 cases and found the Epstein-Barr virus to be the trigger in 121 cases (55%), with another virus identified as the trigger in 28 cases, non-viral triggers identified in 14 cases, and no trigger identified in 56 cases. Dr. Berger also looked at other literature regarding HLH triggers, and found that Epstein-Barr virus was implicated as the trigger in 18% to 95% of the cases involved in each article.

Hart, 2003 WL 23218077, at *4. The Special Master secondarily drew on the same statistics in crediting the testimony of respondent's expert, Dr. Berger, over that of petitioner's expert, Dr. Byers. Dr. Berger, in his own right, relied on similar statistics, noting in his original report that "[d]epending on the criteria used to implicate EBV and the inclusion or exclusion of patients with pre-existing known immune deficiencies, it has been reported to be involved in 18% to 95% of cases."[4] Along the same lines, in his supplemental report, he further observed –

> In recent multi-case series, EBV is generally the most common cause of this syndrome identified. It was found in 5/8 cases reported by Hoang et al and 9/10 cases in children and young adults reported by Ohshima et al. The relative statistical probability of EBV as the cause was also discussed in my previous report. Indeed, in the reference supplied by the Petitioners as Exhibit 11, EBV accounts for 121 of 149 (81%) virus-associated cases of infection related [HLH].

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[4] Further emphasizing the importance of these statistics to his conclusion, Dr. Berger wrote in this report that "[g]iven that the cause of HLH is unknown, but that it is frequently associated with EBV, which this child did have; and that it is very rarely associated with any of the viruses against which he was immunized, I do not believe there is any reason to conclude that this child was injured by the vaccines, nor that they caused or contributed to his fatal illness."

Dr. Berger considered these statistics in evaluating the other record evidence, which included various test results that, in his view, indicated that, at the time he became infected with HLH, Manasseh was suffering from an active EBV infection.[5]

Regarding the latter tests, the Special Master found that "two different tests indicated that Manasseh was infected by the Epstein-Barr virus" and that "Dr. Berger bases his conclusion chiefly on those two tests." *Hart*, 2003 WL 23218077, at *4. The testimony of Dr. Berger is consistent with these observations; the written test results, it appears, are not – they only indicate that Manasseh had been exposed to the EB virus at some point and do not say that he had the sort of active infection necessary to trigger HLH. In the first of these tests, a bone marrow exam performed on May 10, 1999, the lab report indicates – "occasional EBER-positive cells scattered throughout the biopsy core, suggesting that this patient has been exposed to EBV. However, this analysis is difficult to interpret in bone marrow biopsy core material, and the specificity of the result in this setting is not well-established yet."[6] In the second of these tests, a polymerase chain reaction (PCR) test performed on June 8, 1999, Manesseh's blood tested positive for EBV – the record, however, overwhelmingly indicates that this test provided no definitive indication that Manasseh was currently suffering from an EBV infection;

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[5] In this regard, the Special Master found:

> Dr. Berger . . . relies chiefly on the facts that (1) the Epstein-Barr virus is by far the most common trigger for HLH, and (2) two different tests on Manasseh during his HLH ordeal indicated that the child was infected with the Epstein-Barr virus. Dr. Berger finds it far more likely that the Epstein-Barr virus was the HLH trigger than the possibility that one of the attenuated viruses in the MMR vaccine was the trigger.

Hart, 2003 WL 23218077, at *3. Notably, at various points, Dr. Berger appeared to take views less consistent with the Special Master's finding. For example, in his original report, he noted that "[o]ne recent review concludes that 'despite repeated studies, no virus could be consistently associated with HLH, while the spectrum of reported infections simply mirrors that of common pathogens.'" And, in his supplemental report he stated that notwithstanding that "EBV is the statistically most likely cause" of HLH, "I do not believe we can state with any degree of certainty exactly what caused Miclea's fatal illness."

[6] By comparison, as to the bone marrow test, the Special Master determined that "the bone-marrow test performed on May 10, 1999, did indicate a current, active infection – i.e., it indicated that the Epstein-Barr virus was actively replicating in Manasseh's body at that time." *Hart*, 2003 WL 23218077, at *7. In support of this finding, the Special Master does not cite the test results themselves, but Dr. Berger's interpretation thereof.

relying on genetic traces of the virus, the PCR test, like the earlier bone marrow test, rather indicated only that he had been exposed to the virus at some point.[7]

Notably, two other tests, one performed on May 10, 1999, the other on June 8, 1999, both indicated that Manasseh's immune system was not manufacturing antibodies to the Epstein-Barr virus. Dr. Berger discounted these test results by indicating that several studies showed that some individuals suffering from HLH with EBV as the trigger did not show an antibody response to EBV. While Dr. Berger drew parallels between Manasseh and the individuals in these studies, he did not indicate whether Manasseh's serology and immunology results, as well as his overall symptoms, were consistent with those encountered in the individuals in the cited studies who did not have an antibody response to EBV.[8] In the end, while the negative antibody tests gave the Special Master "pause," he, nevertheless, credited Dr. Berger's testimony that Manasseh suffered from an active EBV infection based upon the bone marrow and PCR test results.

In so concluding, Dr. Berger and, in turn, the Special Master also relied on the reports filed by the physicians who treated Manasseh while he was hospitalized. While noting that Manasseh's

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[7] On this point, the Special Master found:

> Petitioner has argued that the PCR blood test performed on June 8, 1999, showed only that Manasseh had been exposed to the Epstein-Barr virus ***at some time*** in the past, not necessarily an infection recent enough to trigger the HLH. That is true, but, of course, the PCR test does not indicate whether the infection was in the distant past or not. The June 8 test could also mean that Manasseh was ***currently*** suffering from Epstein-Barr infection or had been exposed in the very recent past.

Hart, 2003 WL 23218077, at *7 (emphasis in original).

[8] Among other things, petitioner posits that Manasseh's situation differed from the individuals who did not have an antibody response to EBV in that he did have antibody responses to other viruses, among them the measles. Further, Dr. Byers indicated that if Manasseh had infectious mononucleosis but could not mount an antibody response, he would have been more acutely ill, with symptoms such as cancer of the lymph nodes, liver failure or an encephalopathy. Dr. Stanton, Manasseh's personal physician, agreed with the latter theory, but proposed a different theory why Manasseh may not have had EBV antibodies, testifying that "[t]he MMR virus brings on the HLH which the first part of which is the neutropenia, and then subsequent to that time, the [EB] virus, the dormant virus, is activated, and that's the reason why by that time in the hospital the patient doesn't develop antibodies to it, is because it's present but it hasn't provoked an antibody response because it hasn't been able to do so at that time, that is, after all of this has occurred."

personal physician, Dr. Stanton, testified that the HLH was triggered by a MMR reaction,[9] the Special Master gave greater weight to the reports filed by the three hospital physicians (none of whom testified at trial): Dr. Fryberger, Manasseh's attending physician, who, on Manasseh's death certificate, indicated that Manasseh had suffered an "epstein-barr viral infection;" Dr. Lindquist, an infection disease specialist, who wrote that the bone marrow test was "positive for EBV" and that the virus was the "most likely" trigger for the HLH; and Dr. Kitzis, a resident, who likewise concluded that the HLH was "likely triggered" by the Epstein-Barr virus.

However, these physicians appeared to base their conclusions primarily on the test results cited above, which, as noted, do not themselves indicate the presence of an active infection. Indeed, in an earlier report, Dr. Fryberger indicated that she could not rule out the possibility that Manesseh's HLH was caused by the mumps virus he received as part of his MMR vaccination.[10] And, in a revealing letter subsequently provided to the Special Master, Dr. Lindquist, the aforementioned infectious disease specialist, clarified his earlier report, indicating that "[w]hen I was caring for Manasseh Miclea, the exact trigger of his HLH was not my greatest concern" and that "[n]othing about my treatment would have changed because one virus or another triggered the HLH." Dr. Lindquist further explained –

> I cannot say with certainty what triggered [Manasseh's] HLH. Clearly, there is evidence that supports both an Epstein-Barr virus infection and recent vaccinations with a Measles Mumps Rubella (MMR) vaccine; in fact we isolated mumps from one of his specimens. In my opinion it remains unclear as to the true causative agent of his HLH.
>
> It would be unfair for anyone at this time to rely on my notes as a definitive diagnosis of the trigger for Manasseh's HLH. The trigger of his HLH would not have impacted his treatment and I approached my care of him and my conclusions as such.[[11]]

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[9] Consistent with Dr. Stanton's impressions, Manasseh's hospital admission records for May 9, 1999, indicate that his symptoms were "suggestive of measles," but that "other viral possibilities include EBV."

[10] In this report, dated May 26, 1999, Dr. Fryberger explained –

> Given that he had a prior bone marrow biopsy which stained positive for EBV, and this is a known trigger for hemophagocytic syndrome, the most likely virus in my differential is Epstein-Barr virus. It is also possible that he has CMV or HHV 6, 7 or 8. I doubt his prior urine culture, which is growing mumps virus, is contributing to his current process, however, cannot rule this out.

[11] Consistent with the views expressed by Dr. Lindquist in his letter, Dr. Berger testified, regarding the need to determine whether Manasseh had EBV, that "in treating the patient for [HLH] it's not terribly important to know that."

The Special Master, however, did not treat this letter as an explanation of Dr. Lindquist's original report, but rather as "back[ing] away" from his view that EBV was the most likely trigger for Manasseh's HLH. The Special Master thus concluded that Dr. Lindquist's opinion, as encountered in the hospital record, "still is of *some* probative value, because it indicates Dr. Lindquist's thinking *at that time*." *Hart*, 2003 WL 23218077, at *10 n.15.

Largely absent from the Special Master's factual recitation is any definitive indication that, over the two-month time line involved here, Manasseh's symptoms were more consistent with an EBV infection rather than a MMR reaction or the HLH disease itself. In this regard, the Special Master found:

> Dr. Berger's theory in this case is based primarily on the two laboratory tests discussed above, rather than on any clinical symptoms, as proof that Manasseh experienced Epstein-Barr infection. And I am persuaded by Dr. Berger's logic that these laboratory tests are convincing evidence that Manasseh did experience active Epstein-Barr infection in May of 1999. That is true even though we cannot say with ***certainty*** whether any of Manasseh's many symptoms in May constituted clinical symptoms of Epstein-Barr infection rather than a reaction to HLH symptoms. Some of those symptoms may in fact have been symptoms of an Epstein-Barr infection, or maybe none were. Even in the latter event, the ***laboratory test results*** are enough to persuade me that Manasseh likely did experience Epstein-Barr infection.

Hart, 2003 WL 23218077, at *9 (emphasis in original). Notably, while, at the two evidentiary hearings conducted below, Dr. Berger testified that the symptoms experienced by Manasseh in May were characteristic of EBV, he also admitted that many of the same symptoms (*e.g.*, fever, pharyngitis (sore throat), enlargement of the liver and spleen) were also characteristic of an MMR reaction or of the HLH disease itself. [12] By comparison, Dr. Stanton, who saw Manasseh repeatedly in April and May of 1999, testified that Manasseh's symptoms were initially consistent with a viral infection caused by MMR, rather than EBV, and then later consistent with the onset of HLH. He noted that, at critical junctures, Manasseh lacked several of the classic symptoms of EBV, including swollen lymph nodes and tonsilitis.

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[12] While Dr. Berger strenuously maintained that Manasseh's HLH was most likely triggered by an EBV infection, under cross-examination, he admitted that: (i) Manasseh had an immunological response to the MMR vaccine; (ii) his immunological response was indicated by his fever; and (iii) the fever was an indication that Manasseh's immune system had responded with increased levels of cytokines because of the MMR vaccination. Dr. Berger also agreed with the testimony of other witnesses that cytokines are viewed as the likely trigger of HLH.

As noted, on December 3, 2003, petitioner in this case filed a motion for review of Special Master Hastings' November 24, 2003, decision denying her compensation under the Program. Oral argument on petitioner's motion was held, via videoconference, on April 14, 2004.

II. DISCUSSION

A. Background

We begin with common ground.

The National Childhood Vaccine Injury Act of 1986, 42 U.S.C. §§ 300aa-1 to 300aa-34, provides two methods for establishing eligibility for compensation. *See Munn v. Sec'y of Health & Human Servs.*, 970 F.2d 863, 865 (Fed. Cir. 1992). First, a petitioner may demonstrate that the injury is one listed in the Vaccine Table, occurred within the time provided within the table, and meets the further requirements of the statute. *Id*. For such "table injuries," causation is presumed. *Id*. Secondly, if the facts of the case do not comport with the requirements of the Vaccine Table, revealing the presence of a so-called "non-table injury," the petitioner must prove, by a preponderance of the evidence, that the injury was caused by the vaccine. *Id*. Thus, for table injuries, once petitioner demonstrates that symptoms manifested within the prescribed time from the inoculation, causation is presumed. But, in proving a non-table injury, "a proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury." *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992).

In *Shyface, supra,* the Federal Circuit clarified these proof standards. It determined that to establish a *prima facie* entitlement to compensation for a non-table, or causation-in-fact injury, a petitioner is required "to prove, by a preponderance of the evidence, that the vaccine was not only a but-for cause of the injury but also a substantial factor in bringing about the injury." 165 F.3d at 1352. As such, a petitioner "must show 'a medical theory causally connecting the vaccination and the injury'" to establish that the vaccine was a "substantial factor." *Id*. at 1353 (quoting *Grant*, 956 F.2d at 1148). To establish this, "[t]here must be a 'logical sequence of cause and effect showing that the vaccination was the reason for the injury.'" *Id*. (quoting *Grant*, 956 F.2d at 1148). Nonetheless, while the theory of causation must be supported by a "reputable medical or scientific explanation," *Grant*, 956 F.2d at 1148, a petitioner need not prove her theory of causation is "medically or scientifically certain." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994).

In the case *sub judice*, the Special Master decided that plaintiff had not met this burden – that the evidence instead indicated that Manasseh's HLH derived from a preexisting EBV. He, therefore, denied compensation. When deciding a motion for review of a special master's decision, the court may:

> (A) uphold the findings of fact and conclusions of law of the special master and sustain the special master's decision,

(B) set aside any findings of fact or conclusion of law of the special master found to be arbitrary, capricious, an abuse of discretion, or otherwise not in accordance with law and issue its own findings of fact and conclusions of law, or
(C) remand the petition to the special master for further action in accordance with the court's direction.

42 U.S.C. § 300aa-12(e)(2) (2000). Burnishing and combining these standards, the Federal Circuit has stated that this court "may set aside the decision of a special master only if the special master's fact findings are arbitrary and capricious, its legal conclusions are not in accordance with law, or its discretionary rulings are an abuse of discretion." *Turner v. Sec'y of Health & Human Servs.*, 268 F.3d 1334, 1337 (Fed. Cir. 2001) (citing 42 U.S.C. § 300aa-12(e)(2)(B) (2000); *Munn.*, 970 F.2d at 870 n.10); *see also Wagner v. Sec'y of Health & Human Servs.*, 25 F.3d 1031, 1033 (Fed. Cir. 1994); *Guillory v. Sec'y of Health & Human Servs.*, 59 Fed. Cl. 121, 123 (2003).

Petitioner assaults the Special Master's decision on a number of fronts, but her primary thrust is that the Special Master erred in relying on statistical evidence to conclude that EBV, rather than the MMR vaccine, was the most likely cause of Manasseh's HLH. According to petitioner, this reliance on statistics flies in the face of *Knudsen, supra*, in which the Federal Circuit held that "bare" statistical evidence is irrelevant in determining causation under the Vaccine Act. In petitioner's view, the Special Master failed properly to follow *Knudsen* not only by directly relying on statistics allegedly showing that EBV was the most common trigger for HLH, but also in using those statistics to find that respondent's expert was more credible than petitioner's. The result, petitioner contends, is that the Special Master's findings are arbitrary and capricious, as well as contrary to law.

B. ***Knudsen* and "bare" statistics**

Petitioner's focus require this court, *ab initio*, to consider the Federal Circuit's decision in *Knudsen*. There, a special master found that petitioners had made out a *prima facie* case and were entitled to a presumption that the DPT caused Debra Ann Knudsen to suffer an encephalopathy. 35 F.3d at 546. However, the special master also found that "at the time Debra suffered the encephalopathy, she was also suffering from a systemic viral infection, and that the viral infection in fact caused the encephalopathy and the DTP vaccine did not." *Id.* The special master based this decision partially on a finding that "encephalopathies caused by DTP vaccine occur less frequently than encephalopathies caused by viral infection." *Id*. This court upheld the findings; the Federal Circuit, however, reversed.

Initially, the Federal Circuit drew a parallel between the petitioner's burden in a non-table case and the respondent's burden in a table case, observing that "the standards that apply to a petitioner's proof of actual causation in fact in off-table cases should be the same as those that apply to the government's proof of alternative actual causation in fact." 35 F.3d at 549. The court then held that

evidence showing that viral infections more often cause encephalopathies than do vaccines was not proof in an individual case that the virus, and not the vaccine, was the cause of encephalopathy –

> We also reject the government's argument, which again was relied on in the special master's decision, that evidence that there are more occurrences of encephalopathies caused by viral infections than there are encephalopathies caused by DTP vaccines is relevant. Viral infections themselves occur infinitely more often than do DTP vaccinations. Moreover, as the witnesses testified, in the 1950s doctors rarely explained an encephalopathy as being caused by a DTP vaccination. The bare statistical fact that there are more reported cases of viral encephalopathies than there are reported cases of DTP encephalopathies is not evidence that in a particular case an encephalopathy following a DTP vaccination was in fact caused by a viral infection present in the child and not caused by the DTP vaccine.

Id. at 550. Examining the other evidence presented by the government in support of its assertion that petitioner's encephalopathy was caused by a viral infection, the court observed that "[t]he special master did not specifically find whether the above evidence preponderated in favor of alternative causation due to the special master's heavy reliance on grounds we have rejected," among them "the fact that viral encephalopathies are reported more often than DTP encephalopathies." *Id*. at 550-51. Refusing to speculate on this count, particularly in light of the "generosity of the Vaccine Act," the court instead remanded the matter back to the special master for further findings consistent with its opinion. *Id*. at 551.

Although the Federal Circuit cited nothing in support of its assertion that "bare" statistics showing that viral infection more often caused encephalopathies than the DTP vaccine were irrelevant, its holding is well-grounded in the decisional law. To be sure, in recognition of the Kantian dilemma, every form of observation carries with it some probability of corresponding to the truth. *See Victor v. Nebraska*, 511 U.S. 1, 13 (1994).[13] Moreover, while there certainly is an active and continuing

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[13] In *Victor*, the Supreme Court observed –

> In a judicial proceeding in which there is a dispute about the facts of some earlier event, the factfinder cannot acquire unassailably accurate knowledge of what happened. Instead, all the factfinder can acquire is a belief of what probably happened.

511 U.S. at 14 (quoting *In re Winship*, 397 U.S. 358, 370 (1970) (Harlan, J., concurring)); *see also Riordan v. Kempiners*, 831 F.2d 690, 698 (7th Cir. 1987) ("All evidence is probabilistic – statistical evidence is merely explicitly so"); T. Starkie, Law of Evidence 478 (2d ed. 1833) ("Even the most direct evidence can produce nothing more than such a high degree of probability as amounts to moral certainty. From the highest degree it may decline, by an infinite number of gradations, until it produces in the mind nothing more than a mere preponderance of assent in favour of the particular fact.").

debate on the proper use of probabilistic statistics,[14] it is beyond peradventure that courts commonly use certain forms of statistics in reaching some factual findings – for example, in determining disparate treatment in Title VII discrimination cases, *see Griggs v. Duke Power Co.*, 401 U.S. 424, 429-33 (1971), or in applying DNA evidence in criminal cases, *see, e.g.*, *United States v. Wright*, 215 F.3d 1020, 1028 (9th Cir. 2000). *See also Branion v. Gramly*, 855 F.2d 1256, 1263-64 (7th Cir. 1988), *cert. denied*, 490 U.S. 1008 (1989) (citing other appropriate uses of probability statistics). The problem that the Federal Circuit identified in *Knudsen* then, is not that facts cannot be understood in terms of probability or even statistics more generally, but that probability in the form of certain types of statistics is not an appropriate substitute for hard evidence.

In particular, courts are hesitant to determine the causation of a past event using statistics. This hesitancy was first exhibited in state cases, among them, the casebook favorite *Smith v. Rapid Transit, Inc.*, 317 Mass. 469, 58 N.E.2d 754 (1945). There, the plaintiff alleged that she collided with a parked car when she was forced off the road by an oncoming bus. To establish that the bus was owned by the defendant, plaintiff showed that the defendant was the only bus company that had a charter to operate buses on the road in question. The trial court granted a directed verdict for the defendant. On appeal, the Supreme Judicial Court of Massachusetts upheld the verdict finding that ownership of the bus in question was a matter of conjecture. 58 N.E.2d at 755. It reasoned –

> it is 'not enough that mathematically the chances somewhat favor a proposition to be proved' . . . The most that can be said of the evidence in the instant case is that perhaps the mathematical chances somewhat favor the proposition that a bus of the defendant caused the accident. This was not enough.

Id. (quoting *Sargent v. Mass. Accident Co.*, 307 Mass. 246, 250, 29 N.E.2d 825, 827 (1940)). A similar rationale was enunciated in *People v. Collins*, 68 Cal. 2d 319, 438 P.2d 33 (1968), in which a

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[14] For a highly abbreviated sampling of competing articles on this subject, *see, e.g.*, Ronald J. Allen, "On the Significance of Batting Averages and Strikeout Totals: A Clarification of the 'Naked Statistical Evidence' Debate, the Meaning of 'Evidence,' and the Requirement of Proof Beyond a Reasonable Doubt," 65 Tul. L. Rev. 1093 (1991) (hereinafter "Allen"); Richard W. Wright, "Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying Concepts," 73 Iowa L. Rev. 1001 (1988) (hereinafter "Wright"); Daniel Shaviro, "Statistical-Probability Evidence and the Appearance of Justice," 103 Harv. L. Rev. 530 (1989); Michael O. Finkelstein & William B. Fairley, "The Continuing Debate over Mathematics in the Law of Evidence: A Comment on 'Trial By Mathematics,'" 84 Harv. L. Rev. 1801 (1971); Lawrence H. Tribe, "A Further Critique of Mathematical Proof," 84 Harv. L. Rev. 1810 (1971); Lawrence H. Tribe, "Trial By Mathematics: Precision and Ritual in the Legal Process," 84 Harv. L. Rev. 1329 (1971) (hereinafter "Tribe"); Michael O. Finkelstein & William B. Fairley, "A Bayesian Approach to Identification Evidence," 83 Harv. L. Rev. 489 (1970). For a symposium dedicated to this subject *see* Probability and Inference in the Law of Evidence, 66 B.U. L. Rev. 377-952 (1986).

prosecutor used probability statistics to infer that, because of their distinctive characteristics, a husband and wife who were accused of robbery were, indeed, the perpetrators. The Supreme Court of California held that the use of such probability statistics resulted in prejudicial error because the statistical evidence lacked an adequate foundation and the methodology employed by the prosecutor "could only lead to wild conjecture without demonstrated relevancy to the issues presented." 68 Cal. 2d at 329, 438 P. 2d at 38-39. In so concluding, the court admonished that "[m]athematics, a veritable sorcerer in our computerized society, while assisting the trier of fact in the search for the truth, must not cast a spell over him." 68 Cal. 2d at 320, 438 P.2d at 35; *see also People v. Risley*, 214 N.Y. 75, 86, 108 N.E. 200, 203 (1915).

Smith and *Collins* have spawned numerous hypotheticals, generated both by jurists and scholars. Could, for example, evidence that 95 percent of the drivers on a certain stretch of highway speed be enough to convict all who use the highway (that is every driver arrested) of speeding?[15] Is the fact that 95 percent of a particular type of wheel is manufactured by a given company enough to hold that company liable in 100 percent of the cases in which that type of wheel causes injury?[16] Or what if a plaintiff is hit by a blue bus, and it is known that 51 percent of the blue buses on the road are owned by bus company A – is that fact adequate to support a judgment against A?[17] Along the same lines is the hypothetical posed by Dr. Stanton, Manasseh's personal physician (undoubtedly unaware of either *Smith* or *Collins*), who, in rejecting Dr. Berger's reliance on statistics allegedly indicating that EBV is the most common trigger for HLH, explained:

> So, you know, it's, to my mind, it's kind of like you have two guys, in the store, one has a gun and is holding up the clerk, but he has a history of not doing that very often, whereas somebody else that's elsewhere in the store looking for bubblegum is a known bank robber; does that mean he did it?

Consistent with the Federal Circuit's decision in *Knudsen*, the law answers each of these hypotheticals in the negative – the statistics cited are not an adequate basis for decision. But, why? Why are some statistics so patently and intuitively unreliable, while others seemingly are more persuasive? And do some of the statistics that, at first blush, appear cogent ultimately suffer from the same deficiencies that underlie those instantly deemed capricious?

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[15] *United States v. Hannigan*, 27 F.3d 890, 901 (3d Cir. 1994) (Becker, J., concurring).

[16] *Baker v. Bridgestone/Firestone Co.*, 966 F. Supp. 874, 876 (W.D. Mo. 1996).

[17] *Howard v. Wal-Mart Stores, Inc.*, 160 F.3d 358, 359-60 (7th Cir. 1998); *see also United States v. Veysey*, 334 F.3d 600, 605 (7th Cir. 2003). For further variations on the "blue bus" leitmotif, *see* Charles Nesson, "The Evidence or the Event? On Judicial Proof and the Acceptability of Verdicts," 98 Harv. L. Rev. 1357, 1378-79 (1985).

Perhaps questions such as these prompted the Seventh Circuit to note – "The lesson of *Collins* is not that statistical methods are suspect but that people must be sure of what they are looking for, and how they can prove it, before they start fooling with algebra." *Branion*, 855 F.2d at 1264. Indeed, the courts have described what is meant by "naked" or "bare" statistics in several ways. First, they have made clear that mathematical evidence, standing unsupported in the setting of a lawsuit, rarely establishes the proposition to which it is directed. *See Hannigan*, 27 F.3d at 899 n.5; *Prashker v. Beech Aircraft Corp.*, 258 F.2d 602, 609 (3d Cir. 1958). Rather, such evidence is useful only when properly combined with more conventional evidence, so that probabilistic statistics constitute but a single link in a longer chain of proof. *See United States v. Davis*, 200 F.3d 1053, 1054-55 (7th Cir. 2000); *United States v. Shonubi*, 103 F.3d 1085, 1091-92 (2d Cir. 1997); *see also* Tribe, 84 Harv. L. Rev. at 1350. Where such statistical evidence is used without adequate factual foundation, courts have not hesitated to make short shrift of it based, *inter alia*, upon the "desirability of forcing [a party] to identify specific and particularized evidence to support their case." *Baker*, 966 F. Supp. at 876; *see also Veysey*, 334 F.3d at 605; *Howard*, 160 F.3d at 360.[18] Such corroborating, non-statistical evidence is demanded not simply to establish a proper chain of causation, but to ensure that general probability evidence based upon the relative frequency of various events in one population is truly transferable to prove the occurrence of a prior event relevant to the case before the court. *See Veysey*, 334 F.3d at 604; *Hannigan*, 27 F.3d at 897 (Becker, J., concurring); *Baker*, 966 F. Supp. at 876; *see also* Tribe, 84 Harv. L. Rev. at 1346. When these various principles are violated – when one lacks, according to a prominent commentator, "particularistic evidence" and "case-specific probability," Wright, 73 Iowa L. Rev. at 1050-51 – one has what proverbially has been referred to as "naked" or "bare" statistics.[19]

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[18] For examples of this principle in practice, *see Shonubi*, 103 F.3d at 1091-92 (evidence of the quantities of heroin swallowed in balloons by other drug smugglers, not adequate to prove the amount defendant smuggled); *Lynch v. Merrell-Nat'l Labs.*, 830 F.2d 1190, 1197 (1st Cir. 1987) (that chances "somewhat favor" the defendant drug manufacturer being the cause of injury is "not preponderant evidence"); *United States v. Rangel-Gonzales*, 617 F.2d 529, 532 (9th Cir. 1980) (that very few aliens, when advised of right to consult with Consulate, do so, has no bearing on what a particular individual would have done); *United States v. Massey*, 594 F.2d 676, 680-81 (8th Cir. 1979) (testimony stating probability of match to be one chance in 4,500 unfairly confusing where no foundation for statement provided); *Guenther v. Armstrong Rubber Co.*, 406 F.2d 1315, 1318 (3d Cir. 1969) (although 75% to 80% of the tires marketed by Sears were made by defendant manufacturer, plaintiff could not recover even if it showed it was injured by tire bought at Sears); *Spencer v. Baxter Int'l, Inc.*, 163 F. Supp. 2d 74, 80 (D. Mass. 2001) (fact that AIDS patient could show statistically that it was more likely that she was infected from transfusion inadequate to prove liability). For additional cases, *see* Wright, 73 Iowa L. Rev. at 1050 nn. 271 & 272.

[19] By way of further explaining this point, Professor Wright indicated:

A judgment on what actually happened on a particular occasion is a judgment on which

Illuminated by the principles, what may have been obscured before becomes nose-on-the-face plain: the "statistics" employed here are the same sort of "bare" or "naked" statistics that the court in *Knudsen* and other courts have rejected as irrelevant in establishing causation in a particular case. Various reasons buttress this conclusion. Principal among these is that the percentages cited below do not represent probabilities at all, but merely random observations. The case studies brandished by Dr. Berger and others here were not designed to estimate, based upon random sampling, the actual frequency of EBV-infected patients in a known population of HLH-affected individuals. Instead, they involved *ad hoc* groups of individuals who were the victims of HLH, with little revealed about how the members of those groups were selected.[20] As further evidence of this, these studies do not set forth as findings any of the statistics relied upon below – instead, those statistics, expressed in the form of various percentages, were derived by Dr. Berger and others by simply dividing the number of HLH patients for whom EBV was identified as the trigger by the total number of patients for whom any trigger was identified. The percentages so calculated, in fact, considerably overstated the incidence of EBV triggering HLH because they failed to account for the fact that no trigger was identified for a significant portion (in some cases, nearly half) of the patients studied. In sum, while these studies certainly indicate that EBV can cause HLH, they provide no reasonable assurance that the observations extrapolated by Dr. Berger and others therefrom could be projected validly to the particular facts of the case *sub judice*. *See United States v. Yee*, 134 F.R.D. 161, 211 (N.D. Ohio 1991) ("The consistent basis for rulings that either exclude probability estimates or express reservations about such evidence is that the estimate of frequencies on which the computation is made is speculative.").[21]

> causal generalization and its underlying causal law was fully instantiated on the particular occasion. Particularistic evidence connects a possibly applicable causal generalization to the particular occasion by instantiating the abstract elements in the causal generalization, thereby converting the abstract generalization into an instantiated generalization. Without such particularistic evidence, there is no basis for applying the causal generalization to the particular occasion.

Wright, 73 Iowa L. Rev. at 1051.

[20] *Compare* McCormick on Evidence § 210 (5th ed. 2003) ("When the statistical analyst takes properly collected sample data, computes some statistics such as a proportion, a difference between two means, or a regression coefficient, and calculates a P value or a confidence interval for each such statistic, the courts are willing to rely on the probabilities in assessing the force of the statistical evidence."); *see also* Federal Judicial Center, Reference Manual on Scientific Evidence 115-33 (2d ed. 2000).

[21] Various commentators refer to probabilities which may be projected to entities that are not members of the studied class as being "counterfactualizable." L. Jonathan Cohen, The Dialogue of Reason 165 (1986). As noted by another commentator –

But, there are still more problems with these numbers. For one thing, no effort was made to determine whether there were biasing factors, referred to in statistics as confounders, that impacted the headcounts derived from these studies – for example, whether a particular study was conducted by a clinic focusing on EBV that was derivatively studying HLH or, conversely, whether the fact that measles was not encountered as a more common trigger for HLH derives from the effectiveness of the MMR vaccine.[22] Nor was anything more than a glancing effort made to compare Manasseh's overall medical situation with that of the study participants, some of whom had severely impaired immune systems, others of whom were not infants, but young adults. These and the other defects and weaknesses identified above, moreover, are magnified and compounded by the fact that the Special Master and Dr. Berger essentially aggregated the statistics they extrapolated from these various studies in concluding that EBV was the "most common" trigger of HLH. Indeed, that the percentage range cited in the opinion was so broad – from 18 percent to 95 percent – should have been the first clue that these statistics were neither probabilistic nor projectable. In sum, the raw statistics relied upon below share critical features with those rejected in other cases, in which courts have held that "epidemiological data that is not statistically significant cannot provide a scientific basis for an opinion of causation." *In Re Norplant Contraceptive Prods. Liab. Litig.*, 215 F. Supp. 2d 795, 830 (E.D. Tex. 2002); *see also Casey v. Ohio Med. Prods*., 877 F. Supp. 1380, 1385 (N.D. Cal. 1995) (rejecting statistics derived from "a compilation of case reports" which "simply described reported phenomenon").

Seeking to deflect these problems, the Secretary advances several arguments in support of its position that *Knudsen* is inapposite here. First, he contends that decision only applies where the government relies upon a bare statistical analysis to rebut a finding of causation in a table case. This assertion, however, is rebutted by *Knudsen* itself, in which Chief Judge Archer, writing for the majority,

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> "[i]f a statistic has no counterfactual implications, if it really is just an accidental property, then it tells us nothing about an event that is not in the particular set that generated the statistic. It is merely a summary of someone's counting the members of a set formed in an ad hoc fashion, and thus cannot be extended outside the actual reference class. Only if the characteristic may be projected beyond the reference class may it inform about some nonmember of that class.

Allen, 65 Tul. L. Rev. at 1099. The statistics here were plainly, in a word (albeit, a lengthy one), noncontrafactualizable.

[22] In his testimony, Dr. Stanton, Manasseh's personal physician, similarly observed:

> You can say statistically from the literature that EB virus is more likely cause of it than the measles virus. For one thing, EB virus is extremely nowadays more common than measles, and the only measles virus we see now in this country to speak of is the vaccine.

analogized the government's burden in a table case to the plaintiff's burden in a non-table case. *Knudsen*, 35 F.3d at 549. In this court's view, the Federal Circuit thus made clear that bare statistical information cannot be used to rebut the plaintiff's evidence in a case such as this. And other decisions have so held.[23] The Secretary also asserts that this case is distinguishable from *Knudsen* in that it does not involve a "bare statistical fact" because the Secretary provided other evidence that Manasseh's HLH derived not from the MMR vaccine, but rather from EBV. Again, though, this argument misses the main thrust of *Knudsen* and the *ratio dicendi* from which it is derived. Under the law, the question is not whether a party's case consists solely of statistics, which is rarely true – indeed, in *Knudsen*, the Federal Circuit held that the statistical evidence was "bare," notwithstanding the fact that there was other evidence that purported to support the notion that the child involved had a virus unrelated to the vaccine. Rather, to remedy nakedness – to supply the emperor with clothes, as it were – the additional evidence adduced must both show that the probabilities expressed are extendable to the facts of a given case and link the so validated statistical evidence into an otherwise plausible chain of causation. In this regard, the evidence provided by the Secretary here provides wholly inadequate cover.[24]

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[23] At least four decisions by special masters of this court have applied *Knudsen* in non-table cases, where, as here, respondent offered statistics to counter the petitioner's case. *See Watson v. Sec'y of Health & Human Servs.*, 2001 WL 1682537 (Fed. Cl. Spec. Mstr. Dec. 18, 2001); *Rice v. Sec'y of Health & Human Servs.*, 2001 WL 363929 (Fed. Cl. Spec. Mstr. Mar. 26, 2001); *Herkert v. Sec'y of Health & Human Servs.*, 2000 WL 141263 (Fed. Cl. Spec. Mstr. Jan. 19, 2000); *Housand v. Sec'y of Health & Human Servs.*, 1996 WL 282882 (Fed. Cl. Spec. Mstr. May 13, 1996). Other cases have invoked *Knudsen* in still other procedural contexts. *See, e.g.*, *Jenkins v. Sec'y of Health & Human Servs.*, 1999 WL 476255 (Fed. Cl. Spec. Mstr. June 23, 1999) (applying *Knudsen* to reject the use of statistics by petitioner's expert in a non-table case); *Aalders v. Sec'y of Health & Human Servs.*, 1998 WL 408794 (Fed. Cl. Spec. Mstr. June 23, 1998) (same rule applied to petitioner's case in an alleged table case).

[24] For similar results, *see Rice*, 2001 WL 363929, at *10 ("[respondent's expert] admits that much of his opinion is based on the statistical probabilities. Reliance on statistical probabilities has been held inadequate evidence of causation"); *Herkert*, 2000 WL 141263, at *13 ("bare statistical fact that the incidence of reactions following acellular pertussis vaccination is reduced one hundredfold and that TM is a rare but accepted complication of CMV is not evidence that, in a particular case, TM following a DPaT vaccination was in fact caused solely by the CMV and not substantially by the DPat vaccine"); *Jenkins*, 1999 WL 476255, at *17 ("courts have objected to using statistical analysis in individual cases to demonstrate actual causation except in certain instances [not here relevant]"); *Aalders*, 1998 WL 408794, at * 4 (petitioner cannot prevail on the "mere statistical fact" that roseola, of which petitioner apparently exhibited symptoms, rarely results in encephalopathy and seizures); *Housand*, 1996 WL 282882, at *6 (explaining that "respondent may not defeat petitioner's case by arguing that it is statistically unlikely that tetanus toxoid caused petitioner's [Guillain Barre Syndrome]," because, "[w]hile epidemiological studies are relevant," relying on them in this manner would make it "virtually impossible for claimants to prove off-Table cases").

Indeed, while the Secretary argues that apart from the statistics employed here, there is more than ample evidence in the record demonstrating that Manasseh's HLH was more likely triggered by EBV than his MMR vaccination, it appears that much of the evidence received by the Special Master was tainted by those same statistics. For example, the Special Master placed greater reliance on Dr. Berger's testimony, at least in part, because his testimony was consistent with the statistics extrapolated from the studies. Moreover, Dr. Berger not only directly relied upon these same statistics, but also indirectly did so, interpreting the other information he reviewed through the prism of what he believed was the most likely cause of Manasseh's HLH. Thus, for example, while the Special Master indicated that Dr. Berger's testimony was independently supported by the bone marrow and PCR test results here, the fact remains that neither of the lab reports in question actually indicated that Manasseh had an active EBV infection, but rather stated only that he had been exposed to the virus. This is critical because all agree that HLH is not sparked by the mere presence of a virus, but rather requires an active infection that, in turn, produces cytokines. Finally, to the extent that the Special Master and Dr. Berger rely on the reports of the three hospital physicians who treated Manasseh, the record reveals that the reports of two of these doctors, Drs. Fryberger and Lindquist, are more or less equivocal regarding whether Manasseh actually had an EBV infection. Indeed, in the letter that he furnished below, Dr. Lindquist did not, as the Special Master found, change his view as to what caused Manasseh's HLH, but rather indicated, emphatically, that his original position was that it was "unclear" what caused Manasseh's HLH. In light of these conflicting facts, the court is unprepared to take the fact-finding equivalent of a Kierkegaardian leap of faith and conclude that the reliance on statistics below was, as the Secretary asserts, essentially harmless error.

Nor, on the other hand, does the court accept petitioner's argument that the record, as a whole, leads only to the conclusion that Manasseh's HLH was triggered by the MMR vaccination. In the court's view, even excluding the statistics in question, the record here remains somewhat mixed, albeit now weighted more in petitioner's favor. It thus seems advisable to allow the Special Master, who was present when the various witnesses testified, to render additional findings based on that record. Those findings should address whether a detailed time line of the symptoms experienced by Manasseh is more consistent with either of the party's theories of causation. And they also should shed light on another matter that appears from the record, but which was not discussed by the Special Master – that Manasseh had a brother who, years before Manasseh's death, mysteriously died, apparently shortly after receiving a vaccination. In the end, then, it will be for Special Master Hastings, in whom this court has great confidence, to reweigh what remains of the record to ascertain whether the evidence provided by petitioner indicating that MMR could generate cytokines and thereby trigger HLH is still rebutted by what is left of respondent's evidence indicating that it is more likely than not that Manasseh's HLH was triggered by EBV.[25]

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[25] Petitioner argues that once it made out a *prima facie* case that Manasseh's HLH was triggered by the vaccine, it was for respondent to prove alternate causation, that is, that the HLH was triggered by EBV. Although petitioner does not cite any authority for this proposition, the case that

III. CONCLUSION

This court need go no further. For the foregoing reasons, the court determines that the Special Master's findings are arbitrary and capricious and that his construction of *Knudsen*, as it applies to this case, is contrary to law. The petitioner's motion for review, therefore, is **GRANTED**. The Special Master's Entitlement Decision of November 24, 2003, is hereby **VACATED** and this matter is **REMANDED** to the Office of Special Masters for further proceedings consistent with this opinion. Pursuant to Vaccine Rule 28, the period of this remand shall not exceed 90 days.[26]

IT IS SO ORDERED.

s/Francis M. Allegra
Francis M. Allegra
Judge

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comes closest to this claim is *Wagner v. Sec'y of Health & Human Servs.*, 37 Fed. Cl. 134 (1997). In *Wagner*, however, the issue was whether the petitioner was required to disprove what effectively was an idiopathic illness (*i.e.*, one whose cause is unknown). Under the Program, such illnesses cannot be "factors unrelated to the administration of the vaccine" under 42 U.S.C. § 300aa-13(a)(2)(A). This case, however, does not involve an idiopathic illness, but rather whether Manasseh's HLH was caused specifically by EBV. In these circumstances, it would appear that the *Wagner* rationale is inapplicable. *See also Wagner v. Sec'y of Health & Human Servs.*, 1997 WL 617035 (Fed. Cl. Spec. Mstr. Sept. 22, 1997) (explaining, in *dicta*, why this court's decision in *Wagner* was wrong). Rather, it would seem that if petitioner proves, by a preponderance of the evidence, that the MMR vaccine was a substantial factor in Manasseh's contracting HLH, she will also have shown that his fatal illness was not caused by EBV. Nonetheless, the court reserves a definitive ruling on this point until the Special Master has an opportunity to render new findings in this matter.

[26] This opinion shall be unsealed, as issued, after May 17, 2004, unless the parties, pursuant to Vaccine Rule 18(b), identify protected and/or privileged materials subject to redaction prior to said date. Said materials shall be identified with specificity, both in terms of the language to be redacted and the reasons for that redaction.