In the United States Court of Federal Claims

No. 01-190VC
(Filed: January 7, 2010)[1]

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THOMAS D. HENNESSEY,

Petitioner,

v.

SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES,

Respondent.
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Vaccine Act; off-table claim; significant aggravation; Hepatitis B vaccine; type 1 diabetes

Sylvia Chin-Caplan, Boston, MA, for petitioner.

Darryl R. Wishard, United States Department of Justice, Civil Division, Torts Branch, Washington, DC, with whom were *Gabrielle Fielding*, Assistant Director, *Mark Rogers*, Deputy Director, *Timothy P Garren*, Director, and *Tony West*, Assistant Attorney General for respondent.

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OPINION

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BRUGGINK, *Judge*.

Petitioner, Thomas Hennessey, seeks review of a decision entered by the special master denying compensation under the National Vaccine Injury Compensation Program ("Vaccine Act"), 42 U.S.C. § 300aa-1 to -34 (2006).

[1] In accord with the Rules of the Court of Federal Claims, App. B, Rule 18(b), this opinion was initially filed under seal on December 14, 2009. The parties were afforded fourteen days in which to propose redactions. Neither party proposed any redactions.

Petitioner alleges that the Hepatitis B vaccine caused or significantly aggravated his Type 1 Diabetes ("T1D"). The special master, after considering the parties' submissions and hearing testimony from five medical experts, concluded that Mr. Hennessey failed to establish that any vaccine he received either caused or significantly aggravated his T1D. Accordingly, she denied Mr. Hennessey's petition for compensation.

Mr. Hennessey concedes there is no conclusive scientific proof supporting his allegations but contends he submitted sufficient evidence to establish causation and that the special master improperly concluded his theory of causation was unreliable. Specifically, he alleges the special master wrongly elevated his evidentiary burden, placed excessive reliance on certain evidence, and was otherwise arbitrary, capricious, and abused her discretion. The matter has been briefed and this court heard oral argument on November 9, 2009. For the reasons set forth below, petitioner's motion for review is denied.

BACKGROUND[2]

Mr. Hennessey was born on May 25, 1987. His childhood medical history was relatively unremarkable and he was generally healthy and active. He received the usual childhood vaccinations and had the usual childhood illnesses. His medical history indicates a somewhat reduced rate of growth in the two years preceding the vaccinations at issue.[3] Several months before the vaccinations at issue in this case, Mr. Hennessey visited an optometrist and was prescribed glasses. Two months prior to his vaccinations, he contracted an upper respiratory infection and an ear infection.

On September 15, 1998, at age eleven, Mr. Hennessey received his first Hepatitis B vaccination. Although there was no immediate observable reaction, his mother noted that his stamina decreased throughout the autumn months, becoming more noticeable after receiving his second vaccination about two months later on November 17, 1998. Within days of this second vaccination,

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[2] The facts are drawn from the parties' filings and the special master's decision and, unless otherwise noted, are undisputed.

[3] Although both parties agree that Mr. Hennessey's rate of growth slowed prior to his vaccinations, they disagree whether this was merely a normal "growth spurt" variation or the early onset of diabetic symptoms prior to the vaccinations.

Mr. Hennessey began to display classic symptoms of diabetes, including excessive thirst and urination.

Mr. Hennessey visited his family physician on November 30, 1998, where lab tests revealed extremely high blood and urinary glucose levels. His blood glucose level was 571 milligrams per decaliter, a level considered critical, and his urinary glucose was over 1000 milligrams per decaliter, a measure that was "off the chart." His medical records also reveal weight loss.[4]

He was admitted that day to the children's hospital, where his admission history noted the increased thirst and urination had begun about a week and a half earlier, thus placing the onset of symptoms shortly after the second vaccination. The hospital measured his hemoglobin A_{1c} at 12.1 percent.[5] Four days later, after determining the insulin level needed to control his blood glucose, the hospital discharged Mr. Hennessey with a diagnosis of T1D under good control. About two weeks after his diagnosis, Mr. Hennessey was seen by his pediatric endocrinologist, who found his blood glucose levels were under good control. His hemoglobin A_{1c} had declined to 11.7 percent.

Mr. Hennessey received his third Hepatitis B vaccination on January 19, 1999. Over the next year, his condition remained under good control. His hemoglobin A_{1c} declined to six percent by March and remained relatively steady at seven to eight percent for the rest of the year. In December 1999, he was screened for celiac disease.[6] Subsequent lab tests confirmed the diagnosis and he was placed on a gluten-free diet.

Over the following year, Mr. Hennessey's condition remained under control. Beginning in 2001, however, he experienced some trouble controlling his blood glucose levels. These problems persisted into 2003, and in July 2004 he experienced an episode of severe hypoglycemia which required emergency medical assistance. He experienced similar episodes in August 2005 and July

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[4] The parties dispute whether Mr. Hennessey had lost four pounds or ten pounds since the beginning of the school year.

[5] Hemoglobin A_{1c}, discussed in greater detail below, is a measurement linked to blood glucose levels. A normal level is five to six percent, and a level of 6.5 percent is considered diagnostic of diabetes.

[6] Celiac disease is the inability to digest gluten (found in wheat, rye, and barley). Dorland's Illustrated Medical Dictionary 530 (30th ed. 2003).

2006. Despite his condition, Mr. Hennessey remained active, playing high school sports and college football, and sustained several sports-related injuries.

PROCEDURAL HISTORY[7]

This case was filed on April 2, 2001, by Mrs. Hennessey, the mother of the then-minor petitioner. In addition to its import to the named parties, this case also serves as a "test case" for an omnibus proceeding, a device whereby a special master seeks to answer a question that is common to multiple cases involving the same vaccine and injury and implicating the same medical expertise. The test case allows the special master to hear evidence, make findings, and issue an opinion in a specific case, often regarding a general theory of causation. By prior agreement of the parties, the evidence advanced in the test case is then applied to the other cases in the omnibus proceeding. The parties in the other cases are not bound by the result of the test case but may rely on the expert opinions and the evidence underlying that decision.

At the time this case was filed, the petitioner was not prepared to offer evidence on his theory of vaccine causation, so the case was stayed in early 2003. It was subsequently transferred to the "mercury toxicity" group of Hepatitis B cases. In 2004, action in this case was stayed pending the outcome of the Omnibus Autism Proceeding, which relied upon a theory of causation similar to the mercury toxicity group. By late 2006, the petitioner indicated he was ready to proceed and could produce evidence of causation. The parties selected this case as a test case for approximately 15 others. The special master held a two-day hearing in January 2008, at which she heard evidence from one expert witness for the petitioner and four expert witnesses for the respondent.

Type 1 Diabetes

Diabetes is not technically a disease. Rather, it is a condition caused by some underlying disease and characterized by elevated blood glucose. In the case of T1D, the condition is a result of a decrease in the body's production of insulin, a growth hormone that transports glucose from the blood into the muscles to be used as fuel.

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[7] The special master related the procedural history of this case in great detail in her decision. Because neither party contests the accuracy of that recounting, we will only briefly summarize it here.

Insulin is normally produced naturally by specialized clusters of cells known as β islet cells, which are produced by the pancreas. These β islet cells secrete insulin into the bloodstream in response to a rise in blood glucose, for example, after eating. When for some reason the β islet cells are destroyed, the result is progressive insulin insufficiency and a corresponding rise in blood glucose. Although blood glucose levels continue to rise, without insulin the muscles are unable to use the glucose as fuel, and the body effectively starves.

Experts generally agree that T1D is an autoimmune condition—one in which the immune system malfunctions and begins attacking the body's own tissue. Specifically, in the case of T1D, immune system cells designed to attack invading pathogens instead target and destroy the insulin-producing β islet cells. When the immune system antibodies cease to identify and neutralize foreign substances and instead are directed at the body's own tissue, they are known as autoantibodies. Like other autoimmune diseases, T1D requires a genetic predisposition; however, not all who are genetically susceptible develop the condition. An additional environmental inducement is necessary to trigger the production of autoantibodies.

Autoantibodies typically appear early in life, often many years before the onset of clinical symptoms of diabetes. For reasons that are unknown, the rate of destruction of β islet cells varies widely: some patients develop T1D as infants, others develop T1D only decades after the detection of autoantibodies, and some never progress to clinical symptoms. After the first appearance of autoantibodies, there is a latency period during which the destruction of the β islet cells takes place gradually. During this period, there are enough remaining β islet cells to produce sufficient insulin to control blood sugar levels. When 50–70 percent of the β islet cells have been destroyed and blood glucose exceeds 200 milligrams per decaliter, clinical symptoms of T1D begin to appear. If not detected and controlled, this process eventually leads to diabetic ketoacidosis, a potentially fatal condition.

Blood glucose is typically measured in two ways. It can be precisely determined at any given time, sometimes referred to as "spot readings," expressed in milligrams per decaliter. This measurement fluctuates throughout the day, most significantly in response to eating. Blood glucose can also be measured indirectly by calculating the percentage of hemoglobin A_{1c} which is formed when blood glucose binds to new red blood cells as they are produced. As the β islet cells are destroyed, blood glucose levels rise, thus more glucose is available to bind to the hemoglobin. Accordingly, as blood glucose rises, so does the percentage of hemoglobin A_{1c} in the blood.

Because hemoglobin A_{1c} has a known finite life span, the percentage of hemoglobin A_{1c} at any given time can be used to determine the average level of blood glucose for a period prior to the test. Unlike spot readings of current blood glucose levels, a hemoglobin A_{1c} test result changes very slowly and represents an average of blood glucose levels over the previous three to four months. Thus, this one test serves the same function as multiple spot readings of the constantly fluctuating glucose levels.

THE SPECIAL MASTER'S DECISION[8]

The special master heard the testimony of four expert witnesses, received the written report of a fifth expert, and reviewed over 200 medical and scientific journal articles, ultimately concluding "that petitioner has failed to establish by preponderant evidence that any vaccine he received either caused or significantly aggravated his condition." *Hennessey* at *2. Although Mr. Hennessey's expert acknowledged that his theory of causation conflicted with the many studies that do not show an increased incidence ofT1D following Hepatitis B vaccination or any other vaccines, the special master nonetheless considered several theories advanced by petitioner to explain how vaccination could initiate or accelerate autoimmune responses.

The special master also considered evidence put forward by the government's experts, who she concluded "persuasively testified that there is no evidence that vaccines play any role in initiating the autoimmune process or in supplying the sometimes-postulated 'second hit' that pushes an individual into insulin dependence." *Id.* at *26. The special master cited over 30 epidemiological studies investigating possible causes for T1D, many of which examined the possible role of vaccines generally and the Hepatitis B vaccine in particular. *Id.* at *29–35. The special master concluded that "[a]ll of the well-conducted epidemiologic studies have failed to find any relationship between vaccines and the onset of T1D."[9] *Id.* at *31 (footnote omitted).

The special master determined that Mr. Hennessey failed to establish a plausible medical theory of causation because his primary theory—molecular

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[8] *Hennessey v. Sec'y of HHS*, No. 01-190V, 2009 WL 1709053 (May 29, 2009) [hereinafter "*Hennessey"*].

[9] In her review of the evidence, the special master addressed two exhibits suggesting a link between the Hepatitis B vaccination and T1D, *id.* at *29–30, but gave them no weight. *See generally infra* notes 26, 34.

mimicry[10]—was not a reliable one. *See id.* at *52 n.156, *53. She also found that Mr. Hennessey "failed to establish any logical connection between his Hepatitis B vaccinations and his T1D" and that his deteriorated condition was caused by "the natural progression of insulin dependence, rather than his vaccines." *Id.* at *52. She further determined that Mr. Hennessey was already in an advanced overt stage of diabetes at the time of his vaccination based on the testimony of the experts, the hemoglobin A_{1c} tests, and other circumstantial evidence such as his blurred vision,[11] weight loss, and reduced growth rate. *See id.* at *45–51. The special master further determined that Mr. Hennessey failed to demonstrate an appropriate time frame for the onset of symptoms after vaccination.

Despite concluding that Mr. Hennessey had not established a *prima facie* case, the special master considered the government's alternate explanation for his condition. She found "logical and compelling"—though not preponderant—evidence that Mr. Hennessey's T1D was caused by an enterovirus infection several months prior to his diagnosis. *Id.* at *58. She also found by preponderant evidence that the deterioration of his condition after vaccination was due to the "natural progression of insulin dependence, not the vaccines." *Id.*

Finally, the special master found that because Mr. Hennessey's T1D claim failed, his claim for celiac disease, which was based on the T1D causation claim, necessarily failed as well. The special master concluded that "[p]etitioner has not demonstrated by a preponderance of the evidence that his condition was significantly aggravated by the Hepatitis B vaccinations" and, thus, denied the petition for compensation. *Id.* at 59.

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[10] The theory of molecular mimicry posits that autoimmune conditions may result when an invading pathogen contains a molecular sequence that resembles a sequence found in the body. The immune system, primed to attack the invader, inadvertently targets the body's own tissue, continuing the attack on the "innocent" tissue even after destroying the invading pathogen.

[11] The special master noted that high blood glucose can cause blurred vision due to swelling of the eyes' lenses and that Mr. Hennessey was prescribed with glasses several months before his diagnosis. Additionally, Mr. Hennessey's post-diagnosis followup visit noted his vision "improved greatly since getting better control of his diabetes." *Id.* at *50 (citing Ptr. Ex. 5, p. 12).

ARGUMENT

This court has jurisdiction to review the special master's decision. *See* 42 U.S.C. § 300aa-12(e)(1)–(2). On review, we may sustain the decision, set aside the decision and issue our own findings of fact and conclusions of law, or remand to the special master. *Id.* § 300aa-12(e)(2). We may set aside only those findings of fact and conclusions of law that are "arbitrary, capricious, an abuse of discretion, or otherwise not in accordance with law." *Id.* § 300aa-12(e)(2)(B). This standard of review applies differently to different aspects of the special master's decision: findings of fact are reviewed under the deferential "arbitrary and capricious" standard, legal conclusions under the "not in accordance with law" standard, and discretionary rulings for an "abuse of discretion." *Munn v. Sec'y of HHS*, 970 F.2d 863, 870 n.10 (Fed. Cir. 1992).

In his memorandum in support of his motion for review, Mr. Hennessey alleges four numbered objections: (1) the special master impermissibly elevated his evidentiary burden, (2) the special master's conclusion that his T1D predated the vaccinations was arbitrary, capricious, and an abuse of discretion, (3) the special master arbitrarily and capriciously concluded that he failed to prove an appropriate temporal relationship, and (4) the special master's reliance on epidemiology was arbitrary and an abuse of her discretion. *See* Ptr.'s Mem. in Supp. of Mot. for Rev. [hereinafter "Ptr.'s Mem."] at *i*.

I. General Overview of the Vaccine Act.

In enacting the Vaccine Act, Congress recognized that "[w]hile most of the Nation's children enjoy greater benefit from immunization programs, a small but significant number have been gravely injured." H.R. Rep. No. 99-908, at 4 (1986). Because the traditional tort system proved ineffective for these plaintiffs, "Congress created a federal no-fault compensation scheme under which awards were to be 'made to vaccine- injured persons quickly, easily , and with certainty and generosity.'" *Andreu v. Sec'y of HHS*, 569 F.3d 1367, 1374 (Fed. Cir. 2009) (quoting H.R. Rep. No. 99-908, at 3 (1986)).

The Vaccine Act provides two routes for a petitioner to obtain compensation. To establish a claim under the easier of the two methods, known as a "table injury," the claimant merely must show he received a vaccination listed on the Vaccine Injury Table and suffered one of the listed injuries within the prescribed period. *Pafford v. Sec'y of HHS*, 451 F.3d 1352, 1355 (Fed. Cir. 2006) (citing *Capizzano v. Sec'y of HHS*, 440 F.3d 1317, 1319

(Fed. Cir. 2006)). Upon this showing, the vaccine is presumed to have caused the injury. *Id.*

The other route, called an "off-table" case, does not carry with it the presumption of causation. Instead, the petitioner must prove that the vaccination caused the harm. To prevail in an off-table claim, the claimant must demonstrate by a preponderance of the evidence that: (1) while within the United States, he received a vaccine listed on the Vaccine Injury Table, (2) the vaccine caused or significantly aggravated an illness, disease, disability or condition, and (3) the effects of that injury lasted more than six months or resulted in surgery or death. 42 U.S.C. § 300aa-11(c). Litigation rarely concerns the first or third requirement. In most cases, including this one, the issue is one of causation.[12] In *Althen v. Sec'y of HHS*, the Federal Circuit set out a three-prong test for establishing causation in an off-table claim:

> Concisely stated, [petitioner's] burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury.

418 F.3d 1274, 1278 (2005). The first prong focuses on whether the vaccine in question *can* cause the injury alleged. *Pafford*, 451 F.3d at 1356. The second prong applies the medical theory and considers whether the vaccine *did* cause the petitioner's injury. *Id.* The third prong focuses on whether symptoms occurred within a medically appropriate time frame that is neither too late nor too soon after the allegedly causal vaccination. *See De Bazan v. Sec'y of HHS*, 539 F.3d 1347, 1352 (2008). These three prongs "must cumulatively show that the vaccination was a 'but-for' cause of the harm, rather than just an insubstantial contributor in, or one among several possible causes of, the harm." *Pafford*, 451 F.3d at 1355.

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[12] Here, Mr. Hennessey presents a claim for significant aggravation, as opposed to a claim for initial causation. *See Hennessey* at *40; *see also* 42 U.S.C. 300aa-33(4) (defining "significant aggravation"). The Federal Circuit has called significant aggravation the "most slippery and difficult to apply" concept in the Vaccine Act. *Whitecotton v. Sec'y of HHS*, 81 F.3d 1099, 1105 (Fed. Cir. 1996).

II. The special master appropriately considered and weighed the evidence in evaluating Mr. Hennessey's medical theory.

Although not framed in terms of the *Althen* test, Mr. Hennessey's numbered objections challenge the special master's findings on each of the three *Althen* prongs. His first and fourth numbered objections relate primarily to *Althen's* first prong and assert that the special master improperly evaluated the evidence relating to his medical theory of causation. Specifically, Mr. Hennessey claims that the special master erroneously elevated his evidentiary burden and that her reliance on epidemiology was incorrect. We disagree and conclude that the special master examined the evidence under an appropriate legal standard.

The Federal Circuit has consistently acknowledged the inherent uncertainty of vaccine causation and has reiterated the relaxed standard of proof in such cases. *See Knudson v. Sec'y of HHS*, 35 F.3d 543, 549 (Fed. Cir. 1994) ("[T]o require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program."); *Bunting v. Sec'y of HHS*, 931 F.2d 867, 873 (Fed. Cir. 1991) ("The standard of proof required by the Act is simple preponderance of evidence; not scientific certainty."). Consequently, a petitioner's theory of causation must be supported by a preponderance of the evidence. *Althen*, 418 F.3d at 1278. This standard of proof reflects the uncertain nature of "a field bereft of complete and direct proof of how vaccines affect the human body." *Id.* at 1280.

Although the "preponderant evidence" standard does not demand scientific certainty, neither is it satisfied by mere speculation. Rather, a petitioner's theory "must be supported by a sound and reliable medical or scientific explanation." *Knudsen*, 35 F.3d at 548 (citing *Jay v. Sec'y of HHS*, 998 F.2d 979, 984 (Fed. Cir. 1993)). A theory must be "persuasive" and should be "supported by 'reputable medical or scientific explanation.'" *Althen*, 418 F.3d at 1278 (quoting *Grant v. Sec'y of HHS*, 956 F.2d 1144, 1148 (Fed. Cir. 1992)). A medical theory satisfies this standard when reliable evidence makes its truth "more probable than not." *Id.* at 1279 (citing *Hellerbrand v. Sec'y of HHS*, 999 F.2d 1565, 1572–73 (Fed. Cir. 1993)).

A. The special master did not wrongly elevate Mr. Hennessey's burden.

Mr. Hennessey claims that the special master imposed an elevated evidentiary burden in his case. He cites *Andreu v. Secretery of Health and*

Human Services, 569 F.3d 1367 (Fed. Cir. 2009), decided after the special master issued her opinion in *Hennessey*, and states that the special master here committed the same error identified in that case.[13] Specifically, he claims that, as in *Andreu*, the special master here "used epidemiology to impose an 'elevated' burden in his case." Ptr.'s Mem. at 2. We disagree and conclude that the special master did not run afoul of *Andreu's* guidance.

In *Andreu*, the Federal Circuit found that a special master had erroneously rejected the petitioners' theory of causation by imposing on the petitioners an elevated evidentiary burden. *Andreu*, 569 F.3d 1367. The Federal Circuit explained that petitioners need not "submit conclusive proof in the medical literature." *Id.* at 1375. Medical articles and epidemiological studies, while permissible, are not required, and should be viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. In addition, the Federal Circuit faulted the special master for discounting the petitioner's expert testimony "under the rubric of a 'credibility' determination," *id.* at 1379, and noted that a special master cannot "cloak the application of an erroneous legal standard in the guise of a credibility determination, and thereby shield it from appellate review." *Id.*

1. The special master did not err in determining that Mr. Hennessey failed to present preponderant evidence of his medical theory.

The special master concluded that Mr. Hennessey failed to present a *prima facie* case. Although Mr. Hennessey concedes "there is no scientific certainty that a hep B vaccine has ever caused *any* individual to suffer *any* autoimmune disease," he contends that he has submitted preponderant circumstantial evidence sufficient to establish causation. Ptr.'s Mem. at 19–20. We disagree and conclude that the special master did not err when she

[13] While *Andreu* is in some respects similar to this case, the two are not entirely analogous. *Andreu* involved only the second prong of the *Althen* test, *see Andreu*, 569 F.3d at 1375 ("There is no dispute that the [petitioners] met the first and third prongs of the *Althen* test."), while this case involves all three prongs. Even more significant, in *Andreu*, the government's lone expert witness "did not dispute the biologic plausibility of [petitioners'] medical theory." *Id.* at 1377. Here, however, the government's experts strenuously contest Mr. Hennessey's proposed medical theory.

determined that Mr. Hennessey failed to prove his medical theory by a preponderance of the evidence.

Both this court and the special master recognize that petitioners are not required to submit conclusive proof of causation. *See Hennessey* at *39 (citing *Capizzano*, 440 F.3d at 1325). The special master may not demand scientific certainty, but neither may she accept mere speculation. Here, it was not the lack of scientific certainty that was petitioner's undoing. Rather it was his failure to present sufficient evidence to constitute a *prima facie* case. A petitioner's theory must be plausible and supported by reliable evidence. The special master simply considered all the evidence and determined that Mr. Hennessey's evidence did not rise to the level of preponderance.

In essence, the theory of Dr. Yehuda Shoenfeld, plaintiff's expert, is that because some vaccines can cause some autoimmune disorders, any vaccine can cause any autoimmune disorder.[14] *See* Transcript of Entitlement Hearing [hereinafter "Tr. __"] 50–51. He testified that if one virus can cause autoimmune disease, so can other viruses, *id.* at 58, and that "every vaccine potentially can cause an autoimmune disease."[15] *Id.* at 81. This theory is not entirely surprising in light of Dr. Shoenfeld's view that "[a]ll autoimmune diseases are the same," *id.* at 10, a view without evidentiary support and strongly contested by the government's experts. Accordingly, one of the special master's well-founded concerns with Dr. Shoenfeld's theory was its vagueness. Even without considering the questionable merits of molecular mimicry (the mechanism on which his testimony focused),[16] Dr. Shoenfeld's theory is so broad as to be meaningless.

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[14] At oral argument, petitioner's counsel conceded this was an accurate summary of the theory propounded by Dr. Shoenfeld.

[15] In response, one of the government's experts, Dr. Whitton, stated he found this hypothesis "quite remarkable." Tr. 374A. He subsequently clarified that he was expressing incredulity, not awe. *Id.* at 375A.

[16] In his testimony, Dr. Shoenfeld described five different mechanisms by which infectious agents can induce autoimmune disease. *See* Tr. 35–37. For further discussion of molecular mimicry, see *infra* note 21 and accompanying text.

Additionally, Dr. Shoenfeld's theory allows for an unlimited period of time in which symptoms can manifest.[17] *See* Tr. at 83–84. According to his testimony, in a genetically predisposed person, a vaccination can induce autoantibodies that lay dormant for as long as several years until a second event triggers overt autoimmunity. Alternatively, his theory posits that if some previous event initiated the production of autoantibodies, a subsequent vaccination can lead to clinical symptoms within days or even hours. Whether symptoms present immediately or not until years later, any time frame fits into his theory, thereby transforming *Althen's* "proximate temporal relationship" into a virtual Procrustean bed. The effect of these two ambiguities, when combined, is to posit that any vaccine can cause any autoimmune condition in any period of time. This essentially renders *Althen's* first and third prongs meaningless.

Not only was petitioner's medical theory vague, it also lacked support. Understandably, it would be difficult for his theory to be more specific given the paucity of any medical or scientific literature supporting a link between the Hepatitis B vaccine and T1D.[18] The special master weighed the evidence submitted by both sides and determined the proposed theory was not supported by a preponderance of the evidence. A review of the exhibits filed in support of Dr. Shoenfeld's testimony confirms the special master's conclusion that they "were largely literature reviews" that "repeat the same case reports, reference the same animal studies, and treat speculation as proven fact." *Hennessey* at *44. Many of these articles make no mention T1D. *See, e.g.*, Ptr. Ex. 27, 29–37. Those that discuss T1D do not even suggest, much less prove, a causal role for the Hepatitis B vaccine. *See, e.g.*, Ptr. Ex. 25–26, 28, 38–44. At best, these articles support Dr. Shoenfeld's assertion that certain viruses can cause specific autoimmune diseases, including T1D, and indicate that Dr. Shoenfeld has long suspected molecular mimicry as a possible mechanism in such a link.

In contrast, the government advanced a substantial amount of testimony and evidence indicating that there is no causal link between the Hepatitis B vaccine and T1D. This included expert testimony and reports and numerous studies finding no link between vaccinations and T1D. It likewise indicated

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[17] For a more thorough discussion of the appropriate time frame for the onset of symptoms, see *infra* Part IV.

[18] For a more detailed discussion of the scientific and medical literature, see *infra* Part II.A.2.c.

that vaccines play no role in accelerating or aggravating T1D. The special master rightly considered all the evidence and found, in contrast to the petitioner's unsupported speculations, that the weight of the medical and scientific evidence falls clearly against the petitioner's theory of causation.[19] After reviewing the evidence presented and the special master's decision, we cannot conclude that her decision to deny compensation was an error of law.

2. The special master did not ignore Mr. Hennessey's circumstantial evidence.

Although Mr. Hennessey concedes that he has no direct proof of causation, he contends that the special master "ignored substantial circumstantial evidence" and thus impermissibly elevated his evidentiary burden. Ptr.'s Mem. at 19, 25. We disagree. The special master's decision thoroughly discusses the evidence she found helpful or important and makes clear that she carefully considered all the evidence filed in this case. The special master was under no obligation to summarize or discuss every piece of evidence submitted and failure to do so is not an error of law.

a. The special master did not ignore Mr. Hennessey's medical records.

Mr. Hennessey claims that the special master ignored his medical records. The "evidence" that the special master allegedly ignored is simply a restatement of the facts: essentially that Mr. Hennessey received a Hepatitis B vaccination and subsequently developed overt T1D. *See* Ptr.'s Mem. at 25–26. Even a cursory perusal of the special master's decision, however, reveals that she did not ignore the medical records or the facts contained in them. Indeed, she plainly was aware of them as noted at various points throughout her decision. *See, e.g., Hennessey* at *6–8, *51. Mr. Hennessey points to no specific medical records or files that the special master overlooked. At its core, this objection is simply a repetition of Mr. Hennessey's *post hoc ergo propter hoc* argument, assuming that a temporal relationship presupposes a causal relationship. The special master neither ignored the medical records nor do they provide proof of causation.

[19] The special master recognized that a petitioner need not show epidemiological studies or consensus in the scientific community to prevail. *See Hennessey* at *39 (quoting *Capizzano*, 440 F.3d at 1325).

b. The special master did not ignore the expert testimony.

Mr. Hennessey next argues that the special master ignored the testimony of his expert, as well as various "concessions" made by the government's experts. This objection, however, is not supported by a review of the special master's decision, which is replete with references to, quotations from, and discussion of all of the expert witnesses. The special master noted the experts' "extraordinary credentials," *id.* at *8, and detailed their education, research, publications, awards, and current practices. The special master thoughtfully evaluated the testimony from both sides and, where they disagreed, determined which she found more persuasive.

The special master considered the testimony of Mr. Hennessey's expert witness, Dr. Yehuda Shoenfeld, noting he was "a very highly qualified immunologist, with significant expertise on autoimmune conditions in general," but that "he was less experienced in diagnosing and treating T1D than several of respondent's experts." *Id.* at *9. Furthermore, the special master noted that Dr. Shoenfeld "has conducted no research directly related to T1D causation, and has not published anything, other than literature surveys, directly related to T1D." *Id.* She evaluated the expert witnesses and determined "that respondent's witnesses were far more qualified to opine on T1D than Dr. Shoenfeld." *Id.* at *44. The special master clearly did not ignore Dr. Shoenfeld's testimony.

Mr. Hennessey also claims the special master erred by failing to consider "substantial concessions" made by the government's experts. Upon a close examination, however, most of the supposed concessions merely affirm widely known facts about T1D and provide no support for petitioner's theory. For example, the government's experts acknowledged there are a number of possible triggers for T1D, including viruses, diet, stress, and physical exertion. *See* Tr. 255; Resp. Ex. AAA at 5–7. They agreed that a genetic susceptibility was a necessary, but not sufficient, condition for T1D. Tr. 185, 382. Dr. Marion Rewers agreed there were a number of potential mechanisms by which a *virus* could trigger T1D,[20] *see* Resp. Ex. AAA at 6, and that vaccines present a similar challenge to the immune system as the disease against which they

[20] Petitioner's brief obscures the difference between a virus and a vaccine when it simply states that "[Dr. Rewers] agreed with Dr. Shoenfeld with respect to the potential mechanisms of the disease." Ptr.'s Mem. at 26–27.

immunize. Tr. 322A. The government's experts agreed that the rate of β islet cell destruction varies from one individual to another, *id.* at 237, that an adjuvant can stimulate the immune system, *id.* at 442A, and that Mr. Hennessey had subclinical T1D prior to the manifestation of overt clinical symptoms. *Id.* at 433A. None of these "concessions," alone or in combination, strengthen Mr. Hennessey's case.

Several of the statements made by the government's experts, however, demand special attention. In particular, Mr. Hennessey claims that one of the government's experts, Dr. J. Lindsey Whitton, "agreed that molecular mimicry exists in animal models" and that "there is compelling data that it exists in humans as well." Ptr.'s Mem. at 28 (citing Tr. 383A, 384A). Mr. Hennessey's assertion, however, does not fully convey the thrust of Dr. Whitton's testimony. Specifically, Dr. Whitton testified that while molecular mimicry can be shown in "highly manipulated [animal] models,"[21] there is "very limited evidence" for its existence in humans. Tr. 384A. He further stated that "[t]he argument that molecular mimicry causes diseases . . . in human virus infections is extraordinarily weak." *Id.* at 385A. He continued that after "22 years of intensive research on molecular mimicry from many laboratories . . . we have got very little evidence for it." *Id.*

If anything, it is Mr. Hennessey, not the special master, who ignores the testimony of the experts who repeatedly and emphatically disagreed with his medical theory of causation. For example, Dr. Rewers testified that he knew of no mechanism by which the Hepatitis B vaccine could accelerate β islet cell destruction. Two of the government's experts testified that Dr. Shoenfeld was incorrect when he stated there was a causal relationship between T1D and celiac disease. Another, Dr. Noel Maclaren, testified that the alleged causal connection was "an allegation from the blue sky that has no foundation at all." *Id.* at 182. He further testified that although the filed medical literature contained multiple references to molecular mimicry, "it's devoid of any proof that such a thing exists." *Id.* at 216. He stated that he knew of no process whereby the Hepatitis B vaccine could accelerate the destruction of β islet cells. *Id.* at 234–35. Dr. Whitton flatly disagreed with Dr. Shoenfeld's theory that all autoimmune diseases are the same disease. Dr. Whitton also found it

[21] Later in his testimony, Dr. Whitton explained how researchers "stack[] the deck" by genetically manipulating the rats' DNA to contain a protein sequence copied from a virus. *See* Tr. 412A He noted that such experiments do not "prove it's biological reality in the real world." *Id.*

"completely implausible" that the Hepatitis B vaccine provided the final hit pushing Mr. Hennessey into clinical T1D. *Id.* at 436A. The government's experts also testified that Dr. Shoenfeld was incorrect about the classification of the Hepatitis B virus.[22] *Id.* at 241, 354A–55. Dr. Rewers stated that "there is no shred of evidence from human studies that either Hepatitis B virus or any form of vaccine against this virus can trigger or precipitate the cause of added autoimmunity accumulating in diabetes."[23] *Id.* at 252.

In sum, the supposed concessions by the government's experts provide no support for Mr. Hennessey's theory. In any event, there is every indication that the special master exhaustively considered the testimony by all the experts. Thus, we cannot conclude that she erred by ignoring the testimony presented.

c. The special master did not ignore the medical and scientific literature.

Mr. Hennessey next claims that the special master failed to consider the medical and scientific literature. He points specifically to 37 of the articles filed in his own and the government's exhibits. We cannot agree that the special master ignored that literature. The special master wrote a thorough opinion citing and summarizing dozens of studies relied upon by both sides. She was under no obligation to discuss each of the more than 200 medical and scientific journal articles filed as exhibits and her failure to do so does not mean she ignored those exhibits.

Mr. Hennessey points first to 21 articles he filed in support of Dr. Shoenfeld's testimony and states that the special master ignored them. In several instances this claim is clearly incorrect, as the special master explicitly discussed the articles in question.[24] Furthermore, the studies Mr. Hennessey

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[22] Dr. Shoenfeld had testified that the Hepatitis B virus was an RNA virus. Dr. Whitton disagreed, stating it is a DNA virus and, thus, unable to stimulate the immune system in the way suggested by Dr. Shoenfeld. *See* Tr. 355–58.

[23] In response to a question by the special master, Dr. Rewers clarified that there was no evidence from either human or animal studies. Tr. 252–53.

[24] *See Hennessey* at *52 n.155 (discussing Ptr. Ex. 25, 26, 28), *44 n.140 (discussing Ptr. Ex. 32, 38), *35 (discussing Ptr. Ex. 37), *34 (discussing Ptr. Ex. 38), *29 (discussing Ptr. Ex. 40), *31 (discussing Ptr. Ex. 41), *28

points to provide no direct support for his theory. Some discuss neither the Hepatitis B vaccine nor T1D. *See, e.g.*, Ptr. Ex. 27, 29–37. Others discuss one but not the other.[25] *See, e.g.*, Ptr. Ex. 25–26, 28, 38–44. None assert any causal connection between the two. While many of the articles mention molecular mimicry, none provide any evidence for its existence or suggest it may be at play in these circumstances. In sum, Mr. Hennessey's literature provides little support for his theory and, in any event, the special master did not ignore it.

Next, petitioner points to 16 scientific or medical journal articles filed by the government, alleging they provide support for his theory but were ignored by the special master.[26] In some instances, again, his claim is simply incorrect, as several of the articles were specifically discussed by the special master.[27] Others merely state uncontroversial facts about T1D—facts about which the special master was clearly aware. *See*, *e.g.*, Res. Ex. D, G, J, K, O, Q, NN, CCC Tab 39 (all stating that various environmental factors such as chemicals, viruses, infections, diet, physical exertion, and stress may trigger T1D), Res. Ex. Z (stating that not all who develop autoantibodies will go on to develop T1D and that the preclinical phase can vary in length).

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n.84 (discussing Ptr. Ex. 42), *23 (discussing Ptr. Ex. 43), *24 n.78 (discussing Ptr. Ex. 44).

[25] Several of the articles authored by Dr. Shoenfeld contain tables listing various autoimmune diseases reported after vaccination. *See*, *e.g.*, Ptr. Ex. 36 Table 1; Ptr. Ex. 38 Table 24.1. Notably, none of these tables suggest an association between the Hepatitis B vaccine and T1D.

[26] One article not discussed in petitioner's motion for review is a 1997 study by Drs. David and John Classen purporting to find a causal link between the Hepatitis B vaccine and an increased risk of T1D. *See* Res. Ex. CCC Tab 86. The special master discounted this study based on testimony that its authors have been "extensively criticized in the medical literature." *See Hennessey* at *29–30; *see also Baker v. Sec'y of HHS*, No. 99-653V, 2003 U.S. Claims LEXIS 290 (Fed. Cl. Spec. Mstr. Sep. 26, 2003). We need not determine the study's probity, as Mr. Hennessey neither relies on it nor claims the special master erred by discounting its importance.

[27] *See Hennessey* at *13 (discussing Res. Ex. TT), *18 (discussing Res. Ex. M), * 22 (discussing Ptr. Ex. 42, also filed as Res. Ex. KK), *24 (discussing Ptr. Ex. 41, also filed as Res. Ex. MM), *30 (discussing Res. Ex. CCC, Tab 28).

The remaining articles supposedly ignored by the special master—Res. Ex. CCC Tabs 54 and 94—likewise contain little support for Mr. Hennessey's proposed medical theory.[28] The former, co-authored by Dr. Whitton, discusses a number of possible mechanisms by which viruses may precipitate autoimmune conditions. It specifically notes, however, that it is "difficult to provide direct evidence for the involvement of viruses in human autoimmune diseases." Res. Ex. CCC Tab 54 at 91. Furthermore, while the article does discuss molecular mimicry and bystander activation, Dr. Whitton's testimony discussed previously demonstrates the error of placing too much weight on this exhibit.[29] The other article provides an extensive list of diseases allegedly caused by the Hepatitis B vaccine. *See* Res. Ex. CCC Tab 94 at 3878 Table 2. Notably absent from the list is T1D. This article provides support for Mr. Hennessey's theory only if one accepts that "all autoimmune conditions are the same" as Dr. Shoenfeld alleges. We cannot agree that the special master ignored the scientific and medical literature or otherwise erred in her evaluation of it.

B. The special master's reliance on epidemiology was not in error.

In his fourth numbered objection, Mr. Hennessey claims the special master wrongly relied on epidemiology in rejecting his medical theory. He is correct that a petitioner is not required to show epidemiological support for his position, and the special master acknowledged that "epidemiology is not dispositive." *Hennessey* at *53 (citing *Grant v. Sec'y of HHS*, 956 F.2d 1144, 1149 (Fed. Cir. 1992)). This, however, does not mean that the special master cannot consider epidemiology. In fact, she is required to consider the entire record, including the epidemiology. *See* 42 U.S.C. § 300aa-13(b)(1) (the special master "shall consider the entire record" including "relevant medical and scientific evidence"). Here, the special master did not make epidemiology dispositive, but properly considered it as part of the whole record.

Mr. Hennessey disputes the usefulness of epidemiology both in general terms and as applied to him specifically. He first argues that epidemiology, as a general matter, is never applicable to an individual case. This, he alleges, is due to both the rarity of vaccine injuries and the presence of "confounders" for which the study may not account. In support of this contention, Mr. Hennessey

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[28] We deal with Res. Ex. CC, as does Mr. Hennessey, primarily in regard to his fourth numbered objection. *See infra* II.B.

[29] *See supra* note 21 and accompanying text.

points to an article co-authored by Dr. Whitton, one of the government's experts, and offered into evidence by the government. In this article, the authors propose a hypothesis known as the "fertile field," whereby an individual's susceptibility to autoimmune conditions waxes and wanes over time and in response to other factors. *See* Res. Ex. CC. Mr. Hennessey argues that this article demonstrates the complexity of the various confounders for which epidemiology cannot possibly account.

Mr. Hennessey, however, argues beyond what the record supports. For example, Mr. Hennessey states that "respondent's expert Dr. Whitton conceded it would take a very large study to detect rare adverse events." Ptr.'s Mem. 47 (citing Tr. 380A). He fails, however, to mention Dr. Whitton's caveats and explanation, which lead to the opposite conclusion, namely, that the epidemiology in this case is reliable and verifiable. *See* Tr. 378–80A. Specifically, Dr. Whitton noted that many epidemiologic studies select subjects of known genetic risk, thus allowing these studies to detect even rare associations while still using a manageable number of participants. Furthermore, some of these studies are quite large. In his written report, Dr. Barry Bercu mentions a study involving every child born in Denmark from 1990–2000 and finding no increased risk of diabetes after vaccination.

Likewise, Dr. Rewers admitted the challenges of epidemiology, but testified that epidemiological studies are nonetheless well-suited for studying rare diseases. Using the example of the two most significant long-term studies of T1D, DAISY[30] and TEDDY,[31] Dr. Rewers explained these studies screen large numbers of infants, select those who have an enhanced risk of developing T1D, and then follow the health of these individuals over many years. In the latter study, researchers screened 350,000 newborns in multiple countries and selected 8,000 of those at high genetic risk to follow for up to 15 years. As a result of these methods, such studies are able to rely on narrowed groups and still detect rare occurrences. These studies and others, however, have consistently and repeatedly found no link between vaccines and T1D.[32]

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[30] Diabetes AutoImmunity Study in the Young. *See* Tr. 248; Res. Ex. CCC Tab 6.

[31] The Environmental Determinants of Diabetes in the Young. *See* Tr. 248; Res. Ex. C.

[32] One of Mr. Hennessey's own exhibits, a book chapter co-authored by his expert, Dr. Shoenfeld, confirms that most of the well-conducted studies do

In addition to his complaints about epidemiology in general, Mr. Hennessey also claims these specific epidemiological studies are inapplicable to his particular case. In essence, his argument focuses on the age of the participants in these studies. He suggests that because susceptibility to autoimmune disease may vary with an individual's age, the only useful study here would be one examining eleven-year-olds. This argument seems designed to capitalize on an abstract of a scientific article filed inadvertently by the government.[33] This abstract, which apparently never resulted in a published paper, reports an elevated risk of T1D in children receiving the Hepatitis B vaccine at age twelve when compared to those who received the vaccine as infants.[34] Mr. Hennessey's argument on this point falls short. As Dr. Rewers explained, the epidemiological studies submitted by the government included a range of ages, including children up to the age of eleven—the same age as Mr. Hennessey at the time he was vaccinated. Tr. 291A, 304A. He testified further that the largest studies contain "enough variability to tell if taking or not taking a given vaccine or the timing of the vaccination may make a difference." *Id.* at 330.

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not support a causal link between vaccinations and T1D. *See* Ptr. Ex.38 at 313. Dr. Shoenfeld explained his change of opinion was due to recent research on the protective effects of vitamin D, which undermined the reliability of these studies. This research, however, was not recent and had in fact been available at the time Dr. Shoenfeld authored the textbook chapter. *See* Res. Ex. CCC Tabs 30, 31, 33; *Hennessey* at *44. Additionally, Dr. Rewers testified that the DAISY study measured and accounted for vitamin D intake. Tr. 266.

[33] The abstract (P. Pozzilli, *et al*., *Hepatitis B Vaccine Associated with an Increased Risk of Type 1 Diabetes in Italy*, Abstracts from the American Diabetes Association 60th Scientific Session, 272-OR, A67) appears on the same page as several other abstracts relied on by the government at Res. Ex. CCC, Tab 9.

[34] Because Mr. Hennessey does not directly rely on the contents of this abstract or object to the special master's treatment of it, we need not discuss it in great detail. The special master discounted it on the basis of Dr. Whitton's testimony that an unpublished "meeting abstract" is accorded very little weight in the scientific community. *See Hennessey* at *32 n.112. Additionally, Dr. Rewers noted that two of the abstract's authors, the Drs. Classen, had been widely discredited in the scientific community for having significant conflicts of interest in these studies. Tr. 301–02A.

At the end of the day, regardless of the relative strengths and weaknesses of epidemiology in general and as applied specifically to this case, we cannot say that the special master's consideration of this evidence was erroneous. The special master did not rely solely on the epidemiological evidence, but properly considered it along with the other evidence.

III. The special master's decision that Mr. Hennessey's condition was not logically connected to his vaccination was not arbitrary and capricious.

Mr. Hennessey's second numbered objection relates primarily to the special master's finding on *Althen's* second prong: a logical connection between the vaccination and the petitioner's injury. The special master concluded that Mr. Hennessey's T1D predated his vaccinations and thus could not have been caused or aggravated by them,[35] a conclusion Mr. Hennessey contends was arbitrary and capricious. Specifically, he contests the special master's reliance on the hemoglobin A_{1c} test and other circumstantial evidence. We need not decide the underlying substantive question of when Mr. Hennessey developed clinical symptoms. Rather, we need only review the special master's decision to determine if she articulated a reasonable basis for her decision. *See Turner v. Sec'y of HHS*, 268 F.3d 1334, 1338–39 (Fed. Cir. 2001).

The special master articulated several reasons for her finding that Mr. Hennessey failed to satisfy *Althen's* second prong, including Mr. Hennessey's hemoglobin A_{1c} test, the lack of reliable evidence that the wild Hepatis B virus causes or aggravates T1D, the lack of evidence linking the vaccine's adjuvant or preservative to T1D, and the absence of any effect when Mr. Hennessey received his third Hepatitis B vaccination. *Hennessey* at *53. The special master also noted that other circumstantial evidence, such as Mr. Hennessey's blurred vision, weight loss, and slowed growth rate, bolstered her finding that his T1D predated his vaccination. *Id.* at *50–51. Additionally, she considered testimony that, had Mr. Hennessey been examined in August prior to his vaccination, he would have been diagnosed with clinical T1D at that time.

[35] Mr. Hennessey concedes he had T1D before his vaccination, but argues his condition was subclinical and that clinical symptoms would never have manifested but for his vaccination. Ptr.'s Mem 36.

A significant amount of testimony and the special master's opinion were spent explaining the hemoglobin A_{1c} test and discussing its usefulness in this case.[36] Mr. Hennessey, who strenuously opposes the special master's reliance on this test, offered very little evidence to rebut its usefulness and reliability. His expert, Dr. Shoenfeld, referred only briefly to the measurement, noting that "there are false positives with these tests."[37] Tr. 90. In contrast, the government's experts provided repeated and consistent testimony that the hemoglobin A_{1c} test is highly reliable as an indicator of blood glucose over a lengthy period of time. In this case, the results showed a prolonged period of elevated blood glucose prior to Mr. Hennessey's vaccinations, indicating an advanced stage of T1D that was worsening even prior to the vaccinations. The special master's findings of fact plainly were not arbitrary and capricious.

The special master also relied on other circumstantial evidence in her determination that Mr. Hennessey failed to establish a logical connection between his vaccinations and T1D. This evidence included testimony about his reduced growth rate in the years prior to his vaccinations, suggesting it may reflect a gradual decrease in insulin production. Furthermore, Dr. Rewers testified that blurred vision is a common occurrence among children with elevated blood sugar and that this condition develops very gradually over extended periods of time. Additionally, the special master considered evidence submitted regarding Mr. Hennessey's weight loss prior to his hospitalization.[38] The special master considered this testimony and circumstantial evidence among other factors and her conclusion was rational. We cannot say that the special master's decision was arbitrary and capricious.

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[36] *See Hennessey* at *14, *47–50; Tr. 166–70A, 200–05, 276–81A, 346–48, 435A–36C, 456B–59.

[37] Mr. Hennessey has neither claimed nor submitted evidence that either of his hemoglobin A_{1c} tests were in error.

[38] Mr. Hennessey disagrees with the special master's finding that he had sustained an 11% weight loss since the beginning of the school year, contending instead that he lost only four pounds (i.e. 5%). We need not resolve this dispute, but note that Mr. Hennessey's mother states in her affidavit that Mr. Hennessey "had lost 10% of his weight by the time he was hospitalized." Ptr. Ex. 8, ¶ 4.

IV. The special master's decision that Mr. Hennessey failed to prove an appropriate temporal relationship was not arbitrary and capricious.

In his third numbered objection, Mr. Hennessey disputes the special master's finding on Althen's third prong: the appropriate temporal relationship between the vaccination and the onset of disease. After reviewing the evidence and testimony submitted by both parties, we cannot say that the special master acted arbitrarily or capriciously in reaching this conclusion.

In support of this objection, Mr. Hennessey argues that his expert, Dr. Shoenfeld, "specifically addressed this issue at trial." Ptr.'s Mem. at 44. While Dr. Shoenfeld certainly did address the issue of timing, his testimony was less than specific. *See* Tr. 51–55; *Hennessey* at *54 (noting that Dr. Shoenfeld's testimony on this point was "wandering and unfocused"). He testified that traditionally, scientists would expect to see the onset of an autoimmune disease three to six weeks after the environmental trigger.[39] He further testified, however, that recently his opinion has changed completely. According to his current opinion, an initial environmental trigger could induce the production of autoantibodies.[40] At some point potentially years later, a second triggering event can induce or accelerate the destruction of β islet cells, and clinical symptoms of T1D may manifest between two and eight weeks later. For a third triggering event, he testified that symptoms could occur within hours.

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[39] Dr. Shoenfeld cited no support for this view, which differs from that of the government's experts, and was unclear if he referred to autoimmune diseases generally or to T1D specifically. Even assuming this "traditional" view to be correct, Mr. Hennessey's symptoms did not fit within the predicted timeframe. The onset of classic symptoms of diabetes such as polydypsia and polyuria occurred more than eight weeks after the first vaccination but only two days after the second—both too late after the first vaccination and too soon after the second vaccination to satisfy what Dr. Shoenfeld testified was the classically accepted timing for onset of symptoms.

[40] The presence of autoantibodies is a precursor to developing T1D, but not all who develop autoantibodies go on to develop T1D. Such an individual does not yet "have" T1D, either subclinical or overt. Dr. Shoenfeld's testimony was not clear whether, under his theory, this initial trigger also initiated the subclinical disease.

As previously noted, Dr. Shoenfeld's proposed timing suffers from the same overly broad scope as his proposed medical theory. In effect, Dr. Shoenfeld's testimony renders *Althen's* third prong a nullity. Under his theory, nearly any conceivable timing could qualify as an appropriate temporal relationship. For example, if symptoms occur immediately after vaccination, it would be considered the "second hit" exacerbating a process started an indefinite time earlier by some unknown initial trigger. Likewise, this theory accounts for symptoms which do not occur until years after vaccination, exacerbated by some other environmental trigger. Additionally, in the latter example, it would be virtually impossible to determine that the vaccine, and not some other environmental condition, was the original triggering event.

In sum, the petitioner has attempted to satisfy *Althen's* third prong by defining it away, positing that any time frame is appropriate. Furthermore, the government's experts persuasively testified that the timing of events in this case precluded assigning a causal or aggravating role to the vaccination. Both Dr. Maclaren and Dr. Rewers stated that clinical symptoms occur only after a lengthy preclinical period, typically measured in years, during which the β islet cells are gradually destroyed. Dr. Rewers further testified that he knew of no mechanism by which the Hepatitis B vaccine could accelerate β islet cell destruction. Thus, we cannot conclude that the special master's decision on this point was arbitrary and capricious.

V. Conclusion

Petitioner has failed to demonstrate that the special master's decision was arbitrary, capricious, an abuse of discretion, or otherwise not in accordance with the law. Accordingly, we sustain the decision of the Special Master, and deny petitioner's motion for review. The Clerk is directed to enter judgment accordingly.

s/Eric G. Bruggink
Eric G. Bruggink
Judge