REFEREE'S DECISION
HEPATITIS C CLASS ACTION
JANUARY 1, 1986 – JULY 1, 1990

Claimant:	Claimant #1000277
File No.:	416611 – 8
Province of Infection:	Alberta
Province of Residence:	Alberta
Date:	April 28, 2006

DECISION

1. On September 4, 2001, the Administrator denied the claim of the Claimant for compensation as a Primarily-Infected Person pursuant to the Transfused HCV Plan on the basis that negative tracebacks had resulted from testing the blood donations from which the Claimant had received transfusions within the Class Period and no sufficient evidence was adduced to establish that the Claimant was first infected with HCV by any of the said transfusions.

2. The Claimant requested an in person hearing by a Referee to review the decision of the Administrator.

3. Hearings began in Edmonton, Alberta on May 13 and 14, 2002 and by agreement of the parties were adjourned to August 7, 2002, then again to February 19 and 20th 2003 and then to January 20 and March 17, 2005. Many telephone conferences occurred in between the above dates. The Claimant personally attended at the first morning of the hearings but thereafter was represented by his father.

4. The Claimant's father retained legal counsel on October 10, 2002 to assist him through to the conclusion of the appeal.

5. On November 25, 2005 the parties agreed to finalize the list of exhibits for this appeal and to submit all written arguments by March 21, 2006.

6. 101 exhibits were entered and included typewritten notes of the proceedings prepared by the Referee during the first two in person hearings.

The Issue

7. The parties agree that the issue on this appeal is whether the Appellant has satisfied the burden of proof on a balance of civil probabilities that the infection probably was due to one of the in Class transfusions as required by Article 3.04 of the Settlement Agreement.

8. Article 3.04 of the Settlement Agreement provides as follows:

> (1) Notwithstanding any other provision of this Agreement, if the results of a Traceback Procedure demonstrate that one of the donors or units of Blood received by a HCV-Infected Person or Opted-Out HCV Infected Person before 1 January 1986 is or was HCV antibody positive or that none of the donors or units of Blood received by a Primarily-Infected Person or Opted-Out Primarily Infected Person during the Class Period is or was HCV antibody positive, subject to the provisions of Section 3.04(2), the Administrator must reject the Claim of such HCV Infected Person and all Claims pertaining to such HCV Infected Person or Opted-Out HCV Infected Person including Claims of Secondarily-Infected Persons, HCV Personal Representatives, Dependants and Family Members.

(2) A claimant may prove that the relevant Primarily-Infected Person or Opted-Out Primarily Infected Person was infected, for the first time, with HCV by a Blood transfusion received in Canada during the Class Period or that the relevant Secondarily-Infected Person or Secondarily-Infected Person who opted out of the Class Action in which he or she would otherwise be a Class Member was infected for the first time with HCV by his or her Spouse who is a Primarily Infected Person or Opted-Out Primarily Infected Person or Parent who is an HCV Infected Person or Opted-Out HCV person, notwithstanding the results of the Traceback Procedure. For greater certainty, the costs of obtaining evidence to refute the results of a Traceback Procedure must be paid by the claimant unless otherwise ordered by a Referee, Arbitrator or Court.

9. On January 15, 2002 the Claimant asserted that blood transfusions remained the single most likely source of infection and those transfusions within the Class Period were, on the balance of probabilities, the most likely source of infection, on the following grounds:

(a) due to his age, lifestyle and medical treatment, there was no likelihood of high risk activity to cause the infection;

(b) the blood products in Canada during the Class Period were less safe than those introduced in June 1990 with improved screening and virus detection procedures and lower risk of contamination;

(c) The blood transfusions after 1990 consisted of "irradiated Blood" which further reduced the risk of infection after June 1990 because irradiation of blood can inactivate or reduce blood borne viruses;

(d) The test results of the traceback are not reliable.

Agreed Facts

10. Neither party disputed the following facts:

(a) The Claimant was born on February 1, 1985.

(b) On March 19, 2002 the Claimant's mother tested negative for Hepatitis C.

(c) The infection in this instance could not have occurred through high risk activities such as use of illicit drugs, tattoos, piercing, perinatal infection or other socially risky behaviour .

The Evidence

11. One of the documents filed in the initial application was a Tran 2 completed by Dr. McGonigle on August 22, 2000 indicating (at page 5, Section F - Disease Verification paragraph 1) that risk factors included blood transfusions outside the period January 1, 1986 to July 1, 1990.

12. The Claimant's father testified that

(a) the Claimant in the spring of 1990 was diagnosed with neuroblastoma, a cancer with cure rates of no known success in Canada;

(b) the Claimant underwent surgery on May 2, 1990 to remove a mass on the renal gland over one of his kidneys and had a blood transfusion on that day;

(c) further transfusions were given on May 31 and June 12, 1990;

(d) the Claimant was given two injections of a blood product known as MZVIG on June 22, 1990;

(e) in October 1990 he required red blood cells and again in December of 1990 and January of 1991 required MZVIG due to chicken pox reported at his school;

(f) the Claimant required further transfusions outside the Class Period on February 5, 1991, March 1, 1991 and April 1991;

(g) the Claimant's father researched alternative treatments and located an experimental program in Florida that might offer some hope within a six-month window of time when the Claimant was and would remain in remission;

(h) based on his research, the Claimant's father estimated the chances of success were in the range of 50 percent;

(i) the Claimant was entered into the treatment program at what the father described as "University of Florida Shands University Hospital";

(j) bone marrow was drawn from the Claimant via treatment with protobody irradiation two times a day for three days for the purpose of destroying all the cancer cells. He was then given oblitive chemotherapy to destroy the remaining cells and to stop all blood reproduction;

(k) on May 10, 1991 the Claimant had his own harvested bone marrow reinjected into his body;

(l) as a result of the above-described process, the Claimant required further blood transfusions to provide platelets and to promote blood coagulation;

(m) the Claimant received about 30 transfusions in Florida;

(n) because of pooling, the Claimant's father was not sure if the 30 transfusions involved multiple donors;

(o) the Claimant's father did not know if there had been any Hepatitis C testing at the Florida hospital that time;

(p) the Claimant became jaundiced and test results at that time were positive for Hepatitis A;

(q) the Claimant's father was not sure how old the Florida blood products were, what the nature of the surrogate testing was in Florida or when it would have been introduced;

(r) the Claimant's father understood the blood product provided in Florida consisted of red blood cells, platelets and plasma but was unable to verify the nature of the product or other details, such as whether the products could be a year old, when tested or when the units were bled;

(s) the Claimant's father had not sought to have the Claimant joined in any United States based Hepatitis C class action cases;

(t) the Claimant returned from Florida to Alberta on August 9, 1991;

(u) the irradiation affected the kidneys and the liver such that the kidneys stopped producing the hormone required to produce blood;

(v) in August 1991, the Claimant received 2 more units which lifted his hemoglobin level to 15;

(w) the Claimant was unable to produce hemoglobin and his level then dropped to 7;

(x) another transfusion raised the hemoglobin again but as the Claimant still could not produce his own hemoglobin, it dropped again to 7;

(y) the Claimant had further blood transfusions in December 1991, February and March 1992;

(z) in April, he began receiving artificial blood products and injections;

(aa) in April 1992, Dr. McGonigle, an Alberta paediatrician became the coordinator of the Claimant's cancer treatment regime;

(bb) Dr. McGonigle did not know the details of the 30 transfusions in Florida;

(cc) the Claimant's father became concerned by June 1992 about blood transfusions and in particular, whether the Claimant could have contracted Hepatitis C and requested the treating professionals to provide artificial blood products;

(dd) the Claimant's father understood the treating doctors did not think testing for Hepatitis C would then produce meaningful results because the Claimant at that time remained immunocompromised;

(ee) due to the concern of the Claimant's family about Hepatitis C, a test was performed on June 10, 1992 for Hepatitis A, B, and C that revealed, *inter alia*, that no Hepatitis C virus antibodies were detected;

(ff) in October 1992, the Claimant was exposed to chickenpox and again received MVZIG, which according to the Claimant's father, was the last date he received blood products;

(gg) five months later, the Claimant's kidneys began producing hormones and he began producing his own blood;

(hh) by 1998 the Claimant was progressing well and while he had kidney damage, he nevertheless had 50% of his kidney function;

(ii) the Claimant's kidney function appeared to diminish, causing the Claimant's father to be concerned about Hepatitis C and request testing for it;

(jj) the test was performed on July 5, 1999 and produced a positive finding that the Claimant was infected with Hepatitis C;

(kk) no records from the Florida hospital or details of the Florida transfusions were ever requested by the Claimant's father. The Claimant's father testified that he saw them as too difficult to obtain.

13. Canadian Blood Services ("CBS") provided a letter to the Appeals Co-ordinator dated February 20, 2002 (Exhibit 3) which explained its Litigation Notification Program to conduct tracebacks requested by the Fund Administrator and directed requests to the appropriate CBS regional blood centre which then conducted the traceback investigation and that it received a traceback request from the Fund Administrator on November 6, 2000.

14. In this instance a traceback investigation had been commenced in 1999 which confirmed that eight units of packed red blood cells and MVZIG 32 x 2 were transfused to the Claimant between December 12, 1990 and March 31, 1992 at Cross Cancer Institute, and 6 units of packed red blood cells and five of MVZIG were transfused between May 2, 1990 and October 29, 1992 at the University of Alberta Hospital.

15. It was determined from the traceback investigation that the three donors associated with the units transfused during the Class Period subsequently tested negative for anti-HCV.

16. As regards the eleven units of red blood cells and five of MVZIG in the post Class Period between October 18, 1990 and October 29, 1992, ten donors associated with 10 units of red blood cells transfused during the post Class Period subsequently tested negative for anti-HCV and the eleventh donor, associated with unit number A-388525-9, received and

signed for a registered letter from CBS but did not respond to a request for testing, and received a subsequent request which was refused on January 29, 2001.

17. CBS used the HCV 3.0 screening test approved in June 1996 for use in Canada by Health Canada.

18. All in class donors and post class Canadian transfusions were screened with EIA 1.

19. Fund Counsel produced a letter from Saskatchewan Health which confirmed that the Provincial Laboratory is the public health and reference laboratory for the Province; is accredited by the College of Physicians and Surgeons of Saskatchewan and used the Abbott AXSYM system HCV3 assay, Version 1.00.1 which is a third generation ELISIA(sic) method. (Exhibit 8)

20. Fund Counsel provided the Claimant's father on April 15, 2002 with notice that it was relying on the letter from Saskatchewan Health, a copy of website information from the manufacturer of MVZIG-58, a product identified in the transfusion information that was excluded from the definition of "blood" in the Class Action Settlement Agreement, as well as a decision in British Columbia appeal #1300750.

Expert Evidence

21. Fund Counsel submitted Hepatitis C Class Action decision #1300750 which referenced a medical opinion from Dr. Frank Anderson that stated it was rare for an infected person to clear a Hepatitis C antibody and it would not seem possible to do so in less than 20 years.

22. Fund Counsel submitted a report of Dr. Stephen Kleinman dated April 15, 2002 (Exhibit 11), together with his curriculum vitae which stated that Dr. Kleinman is an expert in the field of transfusion-transmitted infection as evidenced by six years in chairing the American Association of Blood Banks Transfusion Transmitted Disease Committee, principal investigator in numerous large-scale US government sponsored research studies on transfusion-transmitted infections and author of numerous peer reviewed scientific articles and book chapters on transfusion-transmitted infections and diseases including Hepatitis C.

23. Dr. Kleinman attended in person to present his opinion and to submit to cross examination by the Claimant's father.

24. Dr. Kleinman reported that the ELISA 3 test is the current "gold standard" test in detecting Hepatitis C antibody. He said there are multiple manufacturers of Hepatitis C antibody ELISA 3 tests in Europe, but only two in North America, and that the test used by CBS had the best sensitivity of all tests evaluated. He said the CBS is using the test that best detects the Hepatitis C antibody and, based on the current level of knowledge, specimens that test negative by this ELISA 3 can be interpreted as lacking Hepatitis C antibody.

25. In his report he offered a number of definitions including the following definition of seroversion:

> Seroreversion: the Loss of Hepatitis C antibody after it was previously detected. For seroreversion to occur, a specimen obtained from a patient at a later time must test Hepatitis C antibody negative, whereas a specimen obtained at an earlier time tests Hepatitis C antibody positive. In such case, seroreversion has occurred. The exact time of occurrence of seroreversion often cannot be determined due to a significant interval between the time that the positive and negative specimens were collected.

26. As to the subject of seroreversion, he stated that it was not known until recently that persons previously infected with Hepatitis C could lose the Hepatitis C antibody over the passage of time, but it is now known that seroreversion can occur, albeit infrequently, and this phenomenon is the least frequent outcome associated with Hepatitis C infection. The rate of seroreversion appears related to the length of time elapsed from the onset of infection and the sensitivity of the test used to detect the Hepatitis C antibody. In some people with resolved Hepatitis C infection, the level of Hepatitis C antibody declines over time such that the antibody becomes undetectable even by the most sensitive assays.

27. Dr. Kleinman reviewed the known data on seroreversion and concluded, from the studies he examined, that approximately 5% of persons infected with Hepatitis C will no longer have detectable Hepatitis C antibody at approximately 20 years after the infection. The studies provided no information as to whether the Hepatitis C antibody would have been present at shorter intervals than the 17-20 years reported in the studies.

28. He opined that the most likely time course in persons who serorevert or lose the Hepatitis C antibody is as follows:

    (a) infection with Hepatitis C;

    (b) development of a positive Hepatitis C PCR test approximately 2 weeks post infection;

    (c) development of a positive Hepatitis C antibody test at 2-3 months post infection;

    (d) loss of virus in peripheral blood within 6 months of Hepatitis C diagnosis or within 8-9 months of infection. Such resolved infection occurs in about 15-20% of persons infected with Hepatitis C. The majority of such persons will show the continued presence of Hepatitis C antibody for decades;

    (e) A small number of persons who lose the virus will show decreasing quantitative levels of antibody for prolonged periods of time, resulting in the documented lack of detectable antibody at approximately 20 years after the infection.

29. He concluded it would be rare for a donor who now tests Hepatitis C antibody negative to have transmitted Hepatitis C at the time of a previous donation since it would require a donor who subsequently loses Hepatitis C antibody (itself a rare event) to have made a donation during the nine months in which that donor was potentially infectious.

30. He noted that another use of Hepatitis C PCR testing is in persons whose immune systems are suppressed. Such persons may be infected with the Hepatitis C virus and not be capable of mounting an immune response. He reported that the study data indicated the phenomena of a positive Hepatitis C PCR test and a negative Hepatitis C antibody test in HIV infected persons was rare.

31. He reported that MVZIG is an abbreviation for Viricella Zoster Immune Globulin, manufactured by the Massachusetts Public Health Biological Laboratories which is infused into severely immunosuppressed patients to provide immunity to the Viricella Zoster virus which is the agent of chicken pox, since chicken pox can be lethal to an immunosuppressed patient.

32. This product is a specialized type of immunoglobulin prepared from pooling plasma obtained from many different blood donors and then purifying the immunoglobulin through a manufacturing process.

33. Dr. Kleinman noted that the Claimant's test of June 7, 1992 was negative for the Hepatitis C antibody, but it was unclear to Dr. Kleinman what version of the test was utilized (ELISA 1, 2 or 3), to what extent the patient was immunosuppressed at that time and whether it was enough to prevent the making of antibody responses to foreign viruses. If not enough to prevent the making of antibody responses, the Claimant would not have acquired Hepatitis C prior to July 1, 1990 because persons with normal immune systems infected with Hepatitis C develop detectable Hepatitis C antibody within two years even using older versions of the ELISA test.

34. Dr. Kleinman listed possible sources for the infection other than blood transfusion including:

    (a) infusions of MVZIG;

    (b) infused bone marrow during the bone marrow transplant procedure;

    (c) iatrogenic exposure from invasive medical treatment/procedures;

    (d) perinatal exposure (which however is rare and could be excluded by Hepatitis C testing of his mother- which was done in this case).

35. Of these possibilities, he noted:

    (a) could occur but was rare;

    (b) has been documented, but is extremely uncommon;

    (c) is a possibility.

36. As to the possibility of infection occurring from one of the three in class donations, he said:

(a) Of the three units of blood received within the Class Period, one of those donors continued to donate blood a total of 59 times from the date of the transfusion to the Claimant to 2002 and has consistently tested negative and is ruled out as the source of the Claimant's infection;

(b) The other two made no further donations, but returned for Hepatitis C antibody testing in early 1999 and tested negative by the ELISA 3 test.

37. Ten of the 11 donors whose units were transfused out of the Class Period were also subsequently tested in periods ranging from 2 to 7.5 years and were negative. Four were tested by ELISA 2, four by ELISA 3 and two by an unspecified test, but Dr. Kleinman thought probably by ELISA 3 given the test occurred in 1999. One out of class donor refused to submit to retesting.

38. In his oral evidence, Dr. Kleinman restated his opinion that while seroversion can occur, it was the least frequent outcome associated with Hepatitis C infection. He contended that the best data was a US study that showed 7 percent of those who had the Hepatitis C antibody detected by ELISA 2 in the mid 1970s no longer had the antibody 20 years later. He considered the Irish study and the East German study and concluded that 5 percent of persons infected would no longer have it detectable after 20 years, but the studies do not show if the Hepatitis C antibody would be present or absent earlier than 17 years. He concluded that the possibility of seroreversion in the case of one of the in class donors was remote because such donor would have to have donated blood only during a 9 month interval when he or she was infectious.

39. Dr. Kleinman opined it is unlikely that the infection came from the MVZIG.

40. He examined the probabilities and considered it more likely that the infection came from someone who did not have a negative test result than from a case where someone was infected and then lost the antibody.

41. He had never heard it suggested that irradiated blood could reduce the risk of infection of Hepatitis C. In his opinion the doses would not be sufficient to kill viruses in blood.

42. He concluded that those who are not tested are a higher *a priori* risk.

43. He further stated that the proposition that a donor may have lost the antibody between donation and testing must be applied to all donors in 1992.

44. The Claimant submitted a letter from Dr. Paul Grundy dated April 23, 2002. Dr Grundy had been involved as the Claimant's pediatric oncologist since April 1990. The report noted:

(a) (the Claimant's) liver enzyme, AST, was documented to be mildly elevated on June 19th at 61 units/litre having been shown to be normal at diagnosis. The AST remained mildly elevated between 44 and 94 between June and December 1990. The bilirubin remained within normal range during this time. The Claimant was receiving multi-agent chemotherapy,

although none of the agents he was receiving "is particularly known to result in chemical Hepatitis";

(b) he had known the Claimant since his presentation in 1990 and to the best of his knowledge, the Claimant has not been or likely to have been involved with any high risk activities for contracting Hepatitis C other than the blood products he was known to have received;

(c) It was also impossible for the Claimant to have been infected through the bone marrow since he received autologous bone marrow.

45. Exhibit 27 is a report from Dr. Roggendorf dated May 7, 2002 which reviews papers published in the former German Democratic Republic reporting findings that indicate that after acute Hepatitis C infection and solution of the antibodies, different antigens persist for many years, but some patients lost antibodies already two to three years after infection and show no serological marker of past infection. As such, it is possible that those persons may have transmitted Hepatitis C virus during the acute phase, e.g. blood transfusion.

46. The papers attached to Dr. Roggendorf's report relate to findings from a study of women infected in East Germany who had received contaminated blood from a woman who had fulfilled the legal requirements (normal transaminiase levels and negative Hepatitis B surface antigen) on the day of blood donation and again on reexamination after 40 days, but retrospectively had been identified as an HCV carrier.

47. An email exchange between the Claimant's father and Professor Dr. M. Roggendorf from the Institute of Virology at the University of Essen in Germany confirmed Dr. Roggendorf's opinion that about 20-25% of Hepatitis C infected individuals are able to clear the virus over a period of several weeks to months and may lose their antibodies to Hepatitis C "between to Ten years or later"(*sic*).

48. Dr. Roggendorf was also asked if it was possible that women in his East German study could have gone from HCV positive to clearing the virus and antibodies between the time of infection and testing. He indicated it was possible however "many patient where (*sic*) tested frequently after exposure but were negative."

49. A letter was submitted by the Claimant from Dr. Klaus Gutfreund dated March 24, 2003 which stated that the testing for Hepatitis C that was available in 1992 was not very accurate. In his opinion, considering that the Claimant received blood products in 1990, and had an elevated AST, in the absence of any other liver disease, he in all likelihood contracted Hepatitis C from the blood transfusions received in 1990. His report made no mention of the out of class donations.

50. Dr. Gutfreund is shown as being attached to the Division of Gastroenterology of the University of Alberta and, from his letterhead, that he practiced, researched and taught as well in Hepatology, Nutrition and Therapeutic Endoscopy. He did not testify, nor was he cross examined and his curriculum vitae was not presented.

Submissions of the Claimant at May 2002 hearing dates

51. The Claimant's father provided his *curriculum vitae* that cited his professional training as an engineer and published research involving statistical analysis of hydrology and hydrogeology issues. He appeared to have applied this type of professional expertise to the issue of probabilities of the infection in the instant case, which made for a very high level of discussion and debate despite the fact that he did not have legal training or initially engage legal counsel on this appeal.

52. He cross examined Dr. Kleinman on his report and put his own statistical theory to Dr. Kleinman.

53. He located his own statement from Professor Roggendorf dated May 7, 2002 which engaged the opinion of Dr. Kleinman and required Dr. Kleinman to review and consider that research of which he did not appear to have been previously aware.

54. The Claimant's father asserted that the screening and surrogate testing was at a higher level in the United States at the time of the Claimant's treatment in Florida and was probably safer there than in Alberta. However he produced no evidence to substantiate this contention.

55. He contended that the blood products received by the Claimant from May 1991 to February 1992 were restricted to irradiated blood products that would inactivate or reduce blood borne viruses and thus further reduce the risk of infection from those products by a figure that cannot be quantified.

56. He conceded that the literature does not verify that irradiation reduces or eliminates Hepatitis C, but he contended that irradiation probably had the effect of reducing the risk of further contracting Hepatitis C.

57. The Claimant contended that the only possible source of infection was from the blood products and contended it was most likely from one of the in class transfusions because:

    (a) the screening and surrogate testing was at a higher level in the US at the time of the Florida treatment and probably safer than in Canada;

    (b) between May 1991 to February of 1992 the Claimant received only irradiated blood products which would further reduce the risk of infection from those transfusions but the reduced risk could not be quantified;

    (c) The risk of infection from any single transfusion between 1985-1990 was estimated at 3-5% but after 1990 it had gone down to a risk of 1/111,000 or according to Dr. Frank Anderson, 1/200,000, and in 2001, the risk had reduced to 1/1,000,000.

Submissions of Fund Counsel at May 2002 hearing dates

58. Fund Counsel submitted that, whereas the three transfusions within the Class Period had been referred to the Traceback Procedure provided by the Plan and were negative, the thirty transfusions from the United States in fact may have involved more donors due to pooling. Then there were 11 additional transfusions after the Class Period. Fund Counsel

argued that, based on the probabilities of the infection occurring from one of the three transfusions in the Class Period as compared to those outside the Class Period, the likelihood had to be that the infection came from one of the donations outside the Class Period.

59. Fund Counsel noted that the screening procedures related to the introduction of a health questionnaire to identify and eliminate from blood donation those applicants that are high risk donors or who have received other blood transfusions and that the first blood test known as "ELISA" was introduced in June 1990.

60. Fund Counsel disputed that irradiated blood could reduce the risk of infection of Hepatitis C.

Testimony of Dr. McGonigle

61. Dr. McGonigle gave the opinion that the raised AST levels of the Claimant in 1990 could have been caused by some other illness at that time, but he thought that the only reasonable explanation in retrospect was that the Claimant had acquired Hepatitis C due to the donations in May and June 1990. He thought it most likely due to the transfusions in Canada even though he was aware of the 30 donations from Florida in May to August of 1991.

62. When asked about the possibility that all the in class donors could have tested negative for the Hepatitis C virus his response was – "What I see and what I know … is that (the Claimant) looked like he had Hepatitis starting in around May, June, July, sometime in June 1990 and he had ongoing Hepatitis until diagnosed in 1998.

63. The reason for his position was that the AST levels were elevated over a prolonged period of time, although he admitted that they dropped to 50 in January of 1991 and 33 in February 28, 1991. He said multiple different illnesses causing elevated liver enzymes does not account for the AST/ALT results.

64. He testified that he was a general paediatrician who had practiced for 16 years. He had a generalist's understanding of Hepatitis C and its treatment. (By my calculation, he would have been in general paediatric practice for about 3 years when he began coordinating the Claimant's treatment regime in 1992). He had no other patients with neuroblastoma that developed Hepatitis C and renal problems. He had about six other patients with Hepatitis C.

65. Dr. McGonigle did not know who managed the Claimant's care before February 1992 and did not have the charts of his prior paediatricians.

66. He said the Claimant was retested in 1999 because the tests were better, because there was a clinical indication that he may have ongoing Hepatitis, and because all the other usual causes of acute rise in liver enzymes such Epstein-Barr virus, Hepatitis B and Hepatitis A, were tested for and the tests were negative.

67. Dr. McGonigle said a high AST reading could be due to illnesses other than liver disease, damaged liver cells, Hepatitis C or other diseases or issues. Whereas AST was the test commonly run at that time, ALT came on line after that time and was known to be more specific for liver cell damage.

68. In the period between 1990 through to 1998 the Claimant's high AST levels were not causing particular alarm to Dr. McGonigle because (a) it could have been caused by things other than Hepatitis C and, (b) it was not very high. It would have been more concerning if the AST was rising and continuing to rise or was very high during that period of time. It was fairly stable prior to 1999 but became significantly higher when he was retested for Hepatitis C in 1999. The AST level was less than double the normal range through most of that period, if normal is somewhat less than 50. It was 60 or 70 or 50 up and down a bit. An AST in the range of 100 plus would be more concerning.

69. The Claimant had ALT of 84 in August 1991, then 74 later in August, then AST of 82 and ALT of 101 in August. The last score would be a concern if it persisted but if it came down it would indicate an insult that would get better on its own.

70. Dr. McGonigle did not know a lot about Hepatitis C in 1991 as the testing was new. No one sought to test the Claimant for Hepatitis C prior to February 1992, because given his condition, there was not much alarm that he might have Hepatitis C.

71. In April 1992, the Claimant's AST was 58; On June 8, 1992 his AST was 141. In June 1992 Hepatitis A, B, and C were all negative. Dr. McGonigle could not remember if the high AST score on June 8 was what prompted the Hepatitis C test.

72. Dr. McGonigle seemed to agree that, in 1991, the AST and ALT levels might have been explained by background factors other than Hepatitis C.

73. When things continued that way in 1992 and 1993 then further on, in retrospect, he said it was clear to him that the onset of Hepatitis C was causing the increased enzymes, even though it did not hit the level of concern for him until June 1992. The reading of 141 was a significant elevation from where he had been prior to that time and was a strong consideration of possible viral Hepatitis.

74. Dr. McGonigle could not forecast when after contracting Hepatitis C virus one would expect his patient to produce an AST reading in the range of 141 because there were too many variables. He said the readings vary considerably from person to person and depend entirely on other conditions going on when immunosuppressive medications are being administered.

75. He agreed that the immunosuppressive drugs may have caused the AST levels to take longer to spike in the Claimant's case. He believed the Claimant was on immunosuppression drugs in 1990 and was immunosuppressed in 1992 which could well cause the AST levels to take longer to spike.

76. He could not account for the raised AST levels of 67 in April 1990 or in June 1990 of 61. He had little doubt that the enzyme elevations through most of that time period related to Hepatitis C, but agreed that he would have to exclude the reading of April 1990.

77. He did not know what caused the spike in April 1990, but said kids get such spikes from illnesses such as mononucleosis, E-B virus, CMV, adnovius, toxoplasmosis and other things. A single spike can occur due to a viral infection and it may be up for a period of time and then come down to normal.

78. With acute Hepatitis due to viral causes like Hepatitis A, it can peak within days or months. Hepatitis C can cause overwhelming infection or a low grumbling inflammation.

79. He did not believe Hepatitis C developed in 1991 because it did not explain what was seen in the AST/ALT results.

80. He was not an expert in Hepatitis C so as to comment or explain why the donors tested negative for the Hepatitis C virus and had no explanation for that. He was aware that the in class donations were all screened before the transfusions, but that did not alter his opinion.

81. He could not say why he did not order another Hepatitis C test between 1992 and 1998. He did not see a reason to test again because the Claimant was up and down but very stable clinically. He could not say whether he was concerned again in July of 1992 when the AST was 262. He then stated that he "guessed" he had an ongoing concern, but Hepatitis C was a relatively new disease and he and the others on the team did not know what was going on there, and while the levels were bouncing up and down, they never got significantly high.

82. He admitted that the only basis for his conclusion that the Hepatitis C was contracted in May or June 1990 was the AST/ALT levels. He admitted that there are risk factors other than blood transfusions, such as medical procedures, scope work or contaminated equipment that could cause Hepatitis C.

83. He was not aware in 1990 or 1992 that there was a window between exposure and when the test will pick up the infection.

84. During my questioning, he told me he did not remember reading the written reports of Dr. Kleinman or Dr. Diaz-Mitoma. He remembered reading a report of a physician who did not think that the Claimant developed the Hepatitis C from blood transfusions but did not have any specific knowledge of that.

85. He told me he recalled filing out a Tran 2, and signing his name to it, but did not remember checking off that the Claimant had as risk factors for the Hepatitis C virus blood transfusions outside the period January 1, 1986 to July 1, 1990.

86. He did not explore any of the transfusions from Florida or look at the records in terms of dates, times or numbers of transfusions.

87. He had not consulted with any other medical experts who specialize in the area of Hepatitis C in formulating his opinion. His opinion was based on knowing the Claimant's family and him and that, other than medical procedures and blood transfusions, there were no other risk factors for that Claimant getting Hepatitis C at that time.

88. He agreed that the origination of the Hepatitis C in this case was not well settled, but he maintained it was likely that the infection occurred around 1990 and was related to (in class) transfusions.

89. He then said **"I don't think it is provable one way or the other, and I think there will be lots of opinions and differences of opinions of medical professionals about that.**

90. He believed other Edmonton physicians thought the infection occurred in May or June of 1990, but he admitted he had not consulted with or spoken to any of them directly about this. He was asked if he had reviewed or considered the opposing opinions to test against his own conclusion and although he claimed that he had done so, he could not explain to what extent he considered and weighed the opposing views of those experts. He said " I am sure that I reviewed them if they came across my desk and I do remember seeing an opposing opinion.

Testimony of Dr. Grundy

91. Dr. Grundy gave the opinion that, in retrospect, it appeared more likely to him that Hepatitis C was accounting for the whole pattern. When asked why he would assign more weight to one than the other he said "in looking at all these different factors, it makes more sense that the Hepatitis C did start in 1990 and that the explanations for the negative tracebacks are as talked about." He said he was assigning more importance to the evidence of hepatic dysfunction in 1990 and 1991 than the other experts were.

92. He conceded that, in formulating his opinion, he was putting more weight on the fact of the patient apparently showing symptoms or laboratory test indications of Hepatitis for which they don't have other good explanation.

93. He said he could accept the explanation that some Hepatitis C patients serorevert as possible and his explanation was that one of the two in the Class Period who had not made other donations had seroreverted.

94. Dr. Grundy is a specialist in paediatric hemotology and oncology. He conceded he was not an expert in Hepatitis C and if one were desired, it would be an infectious disease specialist.

95. He said Hepatitis C affects about 10-15 out of 500-600 of his patients.

96. He testified that he was but one of a team of doctors who followed the Claimant during his illness and was not the first oncologist to see the Claimant, whose condition was diagnosed as a malignancy or cancer of the developing sympathetic nervous system which is seen predominantly in young children.

97. He also noted that Dr. McGonigle was not on the team treating the Claimant until later.

98. Dr. Grundy supervised the chemotherapy treatment which involved repeated cycles of the same chemotherapy. One of the medications, Dycarbazine (DTIC) would be one reason for increase in AST levels. He agreed that DTIC could cause hepatitic dysfunction that could then cause mild elevations in AST/ALT levels. However he said he has not

knowingly seen DTIC affect liver enzyme. He admitted there were occasions when one cannot be certain of the cause of raised AST/ALT levels.

99. He monitors the kidney and liver function of patients every week or two through the whole course of their chemotherapy. He agreed that AST is not a specific marker for what may cause liver dysfunction, but could be due to passing influenza, liver inflammation or Hepatitis C.

100. In clarifying this point for me, Dr. Grundy stated

> *" There are occasions when—with specific reference to a case like this, when the AST is elevated and you cannot conclusively know whether that's elevated for one reason or another. –For instance...we're using combination chemotherapy of several of these drugs together. If the AST was elevated because of one of those drugs, it would not be possible to know from—in our experience, whether it was because one of the drugs or several of the drugs or none of the drugs---or...some other reason. It's only by looking partly from research studies and partly by other patterns over time that sometimes allow you to conclusively pinpoint cause.*

101. Dr. Grundy said the levels of AST in the case at hand were not of a high enough magnitude to alter his treatment or to cause him to make further inquiry. In the day to day priorities, when these children are affected in critical ways by the diseases themselves, using chemotherapy which can be associated with critical toxicities and side-effects, he prioritizes what he looks into and what is of concern. It becomes a concern more in retrospect when one looks at the AST levels and realizes they continued to remain elevated.

102. He agreed that the Claimant was on chemotherapy for around a year between 1990 and 1991 when the AST levels were elevated, but the increased levels were not of a concern sufficient to modify the treatment plan or to send the Claimant somewhere else because

> *" at the time there were not other sort(s) of causes of liver dysfunction that we were worried about in the context of the other priorities that we were already dealing with. Certainly, at that point in time, as you know, for instance, Hepatitis C was not a big concern on people's priority list.*

103. He agreed the AST levels were not particularly disconcerting because it was expected that there were other causes for the increase in levels of a non-threatening nature due to the medical treatment. It at least suggested to him that there was an ongoing process that was not evident at the time, but looking in retrospect at everything, it seemed to him that it was clearly an ongoing process, which as much as anything makes it abnormal, all the way from 1990 throughout.

104. He agreed that:

> *"yes, there are other possible causes, and at the time, on a day to day basis some of those other causes were probably thought to be, but....what's abnormal here is the fact that it continued to be abnormal and the key is that it continued beyond the treatment period.*

105. He agreed it was possible that some of the (chemotherapy) drugs could have caused transient elevations in the AST although he does not commonly see it with these drugs, but even if it had, he said it should have disappeared yet the AST continued to be elevated.

106. The chemotherapy treatment ended in the early to mid-1991 and at the completion of the recommended protocol, the Claimant was in complete remission and has been since that time.

107. He estimated then the chance of remaining in remission was less than 20%. It was not standard practice at that time to undertake high dose chemotherapy with stem cell rescue. Dr. Grundy agreed that both radiation and chemotherapy is used in the process undertaken in Florida and both would transiently affect AST levels for about a month.

108. He said there is no need for immunosuppressant drugs during that procedure but after the dosage of chemotherapy and radiation, he opined that the Claimant would be immune suppressed for at least a year. For that reason, patients like the Claimant may be more susceptible to infection and thus treated more aggressively. When asked if the increased AST levels in the case of an immune suppressed pediatric patient could indicate a viral or other infection, he said:

> *" Yes, I mean, yes, it could, you know, so that, yes, I mean, a lot of these things are multifactorial and again you prioritize...*

109. He said after the Claimant returned from Florida it was not long before it was obvious that he had impairment of his kidney function and also a variety of problems. He then did have periodic investigations into why the AST was elevated without cause being found. He said patients post chemotherapy have abnormal liver enzymes but:

> *"that doesn't mean we can always find what that cause is..." [but] once you're out of that period where you might accept that this was transient elevation associated with something, they should be normal.*
>
> *...his ASTs as he went along there were not normal, even though- that doesn't mean we could explain the cause for it.*

110. He said he had not done a study on abnormal AST levels in his patients, but would guess that in most patients their ASTS would virtually always be in the normal range unless there is a problem. If, when the Claimant's chemotherapy had finished in March of 1991, and he never saw another abnormal value, he would probably think the occasional elevations during chemotherapy must have been secondary to the chemotherapy. But they continued elevated, so looking back, Dr Grundy concluded there is a pattern here.

111. He said from the date chemotherapy started from May 1990 to March 1991, they look at what is important to address from a diagnosis or management perspective bearing in mind that there is a 90% chance of the patient dying from the underlying disease and a 10-15% chance of dying from the treatment. He conceded that, with those priorities, they did not spend a lot of time struggling to think about what could be causing the AST levels to elevate during the chemotherapy.

112. When asked if the radiation could affect the liver such that it affects the AST and ALT levels in terms of the Florida treatment, he stated, while he was not a radiation specialist, he thought it could be, but did not think it was very common.

113. He noted that the total dose of radiation was fairly low but he had body irradiation to the whole body which could cause some adverse effects. He would not have received a very high dose to the liver, so Dr. Grundy thought it unlikely to see elevations of liver enzymes from that dose of radiation.

114. When asked if the AST levels were taken before or after the Claimant's chemotherapy treatments, his answer was "I'm going to guess that they would have been both." When asked if the AST levels tended to be low before chemotherapy and high after, Dr. Grundy said he could find examples where it went up and down one way and other examples where it didn't.

115. When asked if the Claimant was immune suppressed 10 or 11 months after the last Florida treatment, Dr. Grundy said

> "he certainly could have been. I don't – we don't have real good—
> yes, he certainly could have been."

116. Dr. Grundy did not know who decided to test for the Hepatitis C virus in June of 1992 and did not recall being consulted on it. He did not recall if there was a discussion at that time as to whether testing then could produce a negative result due to immunosuppression, or that the negative result might not mean anything.

117. He said that even if the tests don't show what you think might be the problems, if the patient continues to exhibit those problems you retest at a later date because the test might be inadequate. When asked why there was no further test for Hepatitis C in January of 1993 as opposed to 1998 he said it was not a top priority and did not know why no second Hepatitis C test was requested when the AST level was 262 in July 1992.

118. Dr. Grundy did not totally discount the negative antibody test of June 1992. He agreed that the negative test in June 1992 could have been because the Claimant was not infected with Hepatitis C or it could mean he was unable to mount an antibody response to it.

119. Dr. Grundy advised Dr. McGonigle on October 1, 1998 that he was going to initiate the trace back in respect of the in class Canadian donations and that the Claimant's mother would initiate a traceback of the Florida donations. He explained that this note was not patient specific but was an initiative with the Cross Cancer Institute and the Red Cross to traceback all the transfusions between 1986 to 1990. He did not know if anything happened with respect to the traceback of donations in Florida.

120. He did not think he had the results of the Claimant's traceback on his file.

121. He agreed that short segments or AST levels or a specific AST level may not be a factor to be used to establish the source of Hepatitis C in this instance. He said

> *"there are inexplicable sources of variation in individual results that we have to be careful of. We are always working with patterns and whether the patterns of the lab results are consistent with what seems to be happening with the patient."*

122. He did not agree that the AST level in April 1990 was indicative of any pre-existing liver pathology because none ever came to light except Hepatitis C. He did not think one could assign any meaning whatsoever to the height of the spikes or the occurrence. He found a number of patients with totally normal liver enzymes yet were positive for Hepatitis C. Clearly the disease and the measures of the disease like the enzymes follow an extremely variable pattern.

123. He disagreed with the opinion of Dr. Diaz-Mitoma, a virologist, who concluded that the Claimant most likely acquired the Hepatitis C infection after transfusions given to him outside the Class Period. He said that other of his patients who had Hepatitis C had it without causing any symptoms or laboratory findings that made it obvious they had Hepatitis C. He said

> *"We have all these patients who we missed the fact completely that they had been infected originally, and their liver enzymes vary all over the place."*

124. Dr. Grundy from his experience did not assign any significance to the fact that the increased level follows a period of time after the transfusions in terms of trying to pinpoint its onset. He thought that meant that either the virus or the infection became more active or the liver responded differently at that point.

125. When asked about the statement in his written opinion that "we believe that the screening in place for the blood transfusions received in the United States were extremely sensitive and effective, he indicated that he had "accepted other things that I've now reviewed from some of the other letters from the other physicians that you have supplied me with."

126. He then stated that he thought the other experts were providing their opinion on the balance of probability of which unit was infected with Hepatitis C rather than "what is the balance of probability on when the patient was infected with Hepatitis C" which would not focus on the efficacy of the screening procedures and the number of units, but would take into account the patient's factors. He has chosen to reformulate the question which caused him to look more stringently at the pattern of the AST which suggested to him, in retrospect, that there had been something wrong with the Claimant's liver for a long time and a continual pattern of raised ASTs that started in April of 1990. The pattern is there without explanation and, if it had been the chemotherapy, it should have resolved but it didn't.

127. He then said that "the liver enzymes can go to normal even when you have Hepatitis C infection."

128. He was asked to comment on the distinction between the AST levels which went up and down before Florida and were consistently up after Claimant went to Florida and he acknowledged there was that variability, but contended that if you were to look at any kind of trend, over time the infection does persist and get worse.

129. When asked to explain how, under his theory, the in class donors who tested negative would have caused the Claimant's infection, he responded by asking if that had not already been covered by other more expert people than he.

130. When asked about the comment in his report that the data could not prove directly the timing of the Hepatitis C infection, but the data are consistent with the Hepatitis C infection occurring in the spring of 1990, he conceded that he was not saying that the Hepatitis C came from the donated blood from the in class donors.

131. When asked what he understood to be the period for seroreversion to take place, he said he did not think there is enough known about this to put limits on that. He conceded he was not an expert in, nor had he read extensively on, that area.

132. He restated that he did not assign a lot of significance to the fact that the AST levels were higher in 1992 than in 1991 but rather to the fact that the AST was abnormal throughout the period. He agreed that one could assign some of the chemotherapy to cause some of the values and that something else could have caused other of the values and then finally Hepatitis C could have caused other values.

133. He told me that he had not seen data or records from the Florida treatment, but then indicated that "I'm not saying I've never seen them but I have not reviewed any records from Florida recently that I can comment to you on now." He said his only source of information as to the number of transfusions was from documentation he had received from the Referee on behalf of both parties.

134. He did not know if there was any traceback on any of those units or not. He agreed that it was equally possible that the donors in the Florida transfusions could have seroreverted.

135. When I asked him if the Hepatitis C could have been contracted from a Florida donor, he did not disagree, but stated that the whole essence of his perspective was that the Claimant seemed to have liver dysfunction to elevated AST levels prior to his attendance in Florida. He also pointed out that Heptatitis C has been picked up in their patients only well afterward because it is not clinically obvious when they do get infected.

Evidence of Dr. Diaz-Mitoma

136. Dr. Diaz-Mitoma, the Chief of the Regional Virology Laboratory of the Children's Hospital of Eastern Ontario in Ottawa gave a written opinion dated January 13, 2004 (exhibit 87).

137. He gave the opinion that, after reviewing the clinical records and doctor's reports regarding the Claimant's condition, including the facts of all the blood donations, that on the balance of probabilities the Claimant was not infected for the first time with Hepatitis C by a blood transfusion within the Class Period, but it is more likely that he was infected either by the transfusions given in Florida in 1991 or by the blood transfusion on February 1, 1002 in which a traceback procedure was not performed.

138. He concluded that the cumulative residual risk from the more than 30 units of blood outside the Class Period is 1 in 200.

139. He concluded that the chronological listing of his liver enzymes do not support the assertion that he was infected with Hepatitis C during the Class Period because his liver enzymes were abnormal before any blood transfusion was given to him.

140. No cross examination was undertaken of Dr. Diaz-Mitoma.

Written submission of the Claimant on March 2006

141. The Claimant, at the outset of this hearing, had contended that the Traceback system was fallible, however after the Reasons for Judgment released by Pitfield, J. in case number 53, he indicated that argument is no longer to be pursed on this Appeal.

142. The Claimant contends that there is no evidence, medical or otherwise, that the Claimant could have contracted HCV other than by transfusion.

143. The Claimant contends that the in class donors may have incorrectly tested negative for the HCV antibody due to three possibilities:

    (a) there were errors in the testing procedures;

    (b) seroreversion occurred; and

    (c) immunosuppression occurred.

144. In respect of the argument for seroreversion, the Claimant noted that a period of 9-10 years had elapsed between the last donations of 2 of the in-class donors' donations and their subsequent testing, and based on expert evidence produced by Dr. Roggendorf, seroreversion could occur in as short an interval as three years. Despite Dr. Kleinman's opposing view, it is argued that I should prefer the opinion of Dr. Roggendorf.

145. It is then submitted that I cannot assume the health status of the said 2 in class donors currently would be the same as at the time of their last donation because of the potential fore immunosuppression due to underlying medical treatment or conditions. It was noted that the Claimant sought the particular donor's records from the CBS to support this position, but I ruled against such production and the pursuit was abandoned by the Claimant after Justice Pitfield made his ruling in case 53.

146. Finally, the Claimant contends that the negative traceback is irrelevant to the appeal and the sole issue is whether he has adduced sufficient evidence to find that he was infected for the first time by one of the in class donations.

ALT/AST Readings

147. The Claimant contends that his experts' evidence established that the pattern of the above scores supported their conclusions that the infection must have resulted from one of the in-class donations. There was a dispute as to the meaning of the one recorded AST score prior to the Class Period transfusions that recorded a higher than normal score, however, the Claimant contended that his experts' evidence established that, subsequent to the transfusions, the overall scores remained consistently higher than or in the upper range of normal until the 1999 Hepatitis C positive test.

148. The Claimant contends that his experts established that individual spikes and lows in scores should not be given any significance because it could be a transient process occurring in the patient at the time of testing, such as a reaction to chemotherapy or a fleeting cold. They testified that the Claimant's ALT score immediately prior to his positive Hepatitis C test was one of the lowest. They opined the high AST score prior to the first transfusion was anomalous.

Written submission of Fund Counsel March 2006

149. The Fund contends that

(a) The Claimant has not produced very persuasive contradictory objective evidence but has produced a theory only which is based upon too many highly improbable events;

(b) The CBS Traceback Procedure established that all three in class donors tested negative for the antibody and one had donated a further 59 times including 2 in 1990 and has not been identified in any other lookback or traceback program. The remaining two donors, while they made no subsequent donations, tested negative after submitting to retesting during the traceback process. Neither have either of those two been implicated in any lookback or traceback program;

(c) It is relevant and permitted by the prevailing case authority to examine the risk analysis to show the probabilities of the infection occurring due to the in-class donations as opposed to the out of class donations;

(d) Dr. Kleinman opined that the per unit risk for EIA-1 1.0 screened blood in Canada was 0.06% whereas the studies showed that the risk in the US was 0.07%.He further opined that the risk of infection by the in-class donations was 6.2% and the risk of infection from the out of class Canadian transfusions was 22.6% and the out of class US risk was 71.2%. He considered the risk factor would further decrease given that the in class

donors were both screened with EIA-1 and tested negative for the antibody at later dates;

(e) He further opined that, on the balance of probabilities, the infection most likely occurred from one of the US transfusions and least likely due to one of the in class donations;

(f) A further opinion from Dr. Diaz-Mitoma, Chief of Virology of an Ontario Children's Hospital, was that the likelihood of an in-class donation causing the infection was "nil".

Review of Case Authority

150. The decision of Pitfield, J. in Case 53 provided analysis of the proof permitted by Article 3.04(2).

He stated at paragraph 14 as follows:

> *While the primary basis for the determination of eligibility is the traceback process, a Claimant may adduce evidence on appeal in support of the claim that he or she was infected for the first time in the class period notwithstanding a negative traceback result. In my opinion, Article 3.04(2) does not permit a Claimant to conduct his or her own traceback procedure. The Article contemplates that there might be evidence which would establish that the source of the infection, more likely than not or on the balance of probabilities was a transfusion received in the period. It is not an answer to a Claimant's attempt to provide such evidence to say that some small percentage of the population may be infected by HCV from unknown sources. Were such an assertion an answer, a Claimant could never refute the traceback result because the Claimant could never prove that he or she was not one of that small percentage of population who might have been so infected.*
>
> *The evidence the Claimant would be required to adduce on appeal would include, at the least, complete family and personal medical history and detailed evidence of all aspects of the Claimant's lifestyle including evidence of the absence of opportunity to be infected by needles or injections, however and for whatever purpose received.*

And at paragraph 16:

> *The reliability of the assertion which is subjective in nature would have to be tested by reference to all known objective evidence. One of the pieces of objective evidence is the negative traceback result following upon the application of, and adherence to, the approved traceback protocol. Contradictory objective evidence would have to be very persuasive if the traceback result is to be refuted.*

Analysis

151. One cannot help but have the greatest sympathy and admiration for the courage and hard work displayed by the Claimant and family during the course of this challenging history. In addition, it would be difficult to find a parent who has expended more time and effort in studying and familiarizing himself with all the known literature on every aspect of his child's medical condition and the legal rights and remedies available to him, than this Claimant's father.

152. In my following analysis I do not intend that my evaluation of the evidence be perceived as any criticism of the conduct of the Claimant's family or health professionals who all provided me with their full cooperation and made their best efforts throughout this proceeding to respond in a timely way to permit this hearing to proceed to conclusion.

Relevant medical records

153. Counsel for the Claimant asserted that all relevant medical records were produced in this hearing, however, no medical charts were produced of the paediatricians who treated the Claimant between April 1990 and February 1992 prior to the involvement of Dr. McGonigle. Dr. McGonigle bases his opinion on the AST/ALT scores and his patient's condition from April 1990 and February 1992, an important time period which preceded his involvement.

154. Further, the Claimant has not produced the available hospital records about 30 transfusions of blood to the Claimant by the Florida treating institution. His representative indicated he thought they would be too difficult to obtain.

155. On the other hand, Dr. Grundy's testimony suggested he had or may have seen such records. Further, he indicated that there had been discussion between him and the Claimants' family about initiating tracebacks of the donations in Florida, but it evidently never proceeded.

156. This suggests to me that the Claimant's family and the expert medical witnesses considered the possibility, after the Claimant tested positive for Hepatitis C, that the infection may have occurred in Florida and appreciated that tracebacks would be relevant to the question of the cause of the infection.

157. Moreover, the Claimant's representative did not reveal the fact of the Florida donations until the first day of the hearing of the appeal.

158. However on the date when that revelation occurred, Fund Counsel raised with the Claimant's representative and me whether it would not be more probable that the Hepatitis C infection occurred in one of the 30 Florida transfusions for which there was no evidence of any request for tracebacks, or from one of the further 11 in Canada that occurred outside the Class Period than from 3 in class transfusions all of which were shown to be negative. This did not appear to me to surprise the Claimant's representative, as he had a very cogent argument ready at that time on the weakness of the probability theory raised by Fund Counsel.

159. The anomaly in the evidence as to whether Dr. Grundy did or did not review the Florida hospital records leaves me in doubt as to whether the records were obtained at some point and reviewed by Dr. Grundy or, in any event, whether there was any serious impediment to producing those records for this hearing.

160. If Dr. Grundy had considered and ruled out the involvement of the 30 Florida transfusions for examinable reasons, that would have been relevant and persuasive evidence to support the Claimant's contention. Moreover, Dr. Grundy's awareness of the Florida records suggests they may not have been impossible or even difficult to obtain. Further, I note from his testimony that there was discussion about requesting tracebacks from the Florida donors yet no evidence was presented to me about the results of any such request or traceback.

161. There was evidence that the Claimant had not sought to be joined to any Hepatitis C class actions in the United States, but no evidence as to whether there were or were not any such actions that might be afoot in the Florida area or its surrounding jurisdictions at any material time.

ALT/AST readings

162. The Claimant contended that the in class donations must have caused the infection because the Claimant had varying but elevated AST levels from April 1990 onward over the 1.5 years since the in class donations. The Claimant relies upon the evidence of three physicians, Dr. Gutfreund, Dr. McGonigle and Dr. Grundy, as well as Dr. Roggendorf.

163. They contend that I must disregard the AST reading from April 1990, the negative Hepatitis C test in 1992 which was a possibly false negative, and rely on the AST levels which varied, but were elevated, from April 1990 to April 1991.

164. I must now consider the opinions of Dr. McGonigle and Dr. Grundy in light of the requirement to examine the reliability of a subjective assertion against all known objective evidence including the negative traceback result following upon the application of and adherence to the approved traceback protocol and determine if the contradictory objective evidence was persuasive enough to refute the traceback result.

165. Dr. McGonigle is, without doubt, a caring and concerned medical professional whose treatment program for his patient and patient's family is top of mind and rightly so. He was very firm in his view that the cause of the infection was the in class transfusions. However, I did not find the foundation for his opinion to be nearly as firm. In particular,

    (a) He admitted not much was known about Hepatitis C in 1990 to 1992 and he had no particular expertise or much experience with it in his practice;

    (b) He had not obtained the previous charts of the paediatric physicians who treated the Claimant before 1992 and thus had no personal knowledge of the Claimant's week to week condition in the period between April of 1990 to 1992;

(c) He did not have a clear memory of the Claimant's week to week condition in 1992 when he first assumed responsibility for the Claimant's treatment that would rule in or out any symptoms of viral infections that might explain periodic high AST readings;

(d) He did not have a clear recollection of what his concerns, apart from the primary issue of cancer, were about the Claimant's prevailing condition in the years between 1992 to 1998 when he first began treatment of the Claimant;

(e) For example, despite the high AST reading of 262 in July of 1992, he did not recall any concern sufficient to order a further Hepatitis C test.

166. I am not confident in accepting his conclusion for a number of reasons. First, he concedes it is based entirely on the AST/ALT readings and he maintains that no individual reading is reliable. Second, he has no particular note or memory of the Claimant's condition in 1992. Third, he concedes that in formulating his opinion, he did not consult with any other medical colleagues more knowledgeable in this area. If he read or considered the opposing opinions in this case, he did not have any clear recollection of the basis for rejecting the opposing opinions. I do not find the basis for his opinion, by itself, to meet the requirement of being highly persuasive contradictory objective evidence.

167. Dr. Grundy is clearly an experienced, competent and caring paediatric oncologist and I have no hesitation in accepting his evidence as it relates to his field of expertise. It is clear to me that the priority of Dr. Grundy and his team during 1990 and 1991 were quite properly on the issue the Claimant's survival.

168. However Dr. Grundy made it very clear that he does not have expertise in the area of infectious diseases, radiation or Hepatitis C and that only a very small number of his patients have Hepatitis C. From his own testimony, it appears that some of the increased AST levels in 1990 could be due to transient viral conditions, such as a cold or flu, and some of the other increased levels could have been due to the chemotherapy medications. He further testified that some patients with Hepatitis C have normal AST levels. I do not see that his theory that the Claimant must have had Hepatitis C since 1990 solely because the AST levels were elevated, in the face of all the other evidence before me, meets the test enunciated by Pitfield J.

Conclusions

169. On the basis of the evidence before me, I am entirely satisfied that, based on the Claimant's age, lifestyle and medical treatment, there was no likelihood of high risk activity to cause the Claimant's infection.

170. I am satisfied that the blood products in Canada during the Class Period were less safe than those introduced in June 1990 with improved screening and virus detection procedures and lower risk of contamination.

171. I did not receive very persuasive evidence to establish that

(a) blood transfusions after 1990 consisted of "irradiated blood" which further reduced the risk of infection after June 1990 because irradiation of blood can inactivate or reduce blood borne viruses;

(b) there were errors in the testing procedures of the traceback program in this instance;

(c) the Claimant was sufficiently immunosuppressed at June 7, 1992 such as to render the negative Hepatitis C test result on that date irrelevant;

(d) screening and surrogate testing was at a higher level in the United States at the time of the Claimant's treatment in Florida or that as a result the process of blood transfusions was probably safer there and at that time than in Alberta in May of 1990;

(e) the Claimant was free of transient viruses or effects of chemotherapy medications such as to eliminate those factors as an explanation for some or all of the elevated AST levels in the period between April 1990 and June 1992;

172. I have considered the statistical evidence that one of the donors in the Class Period may have seroreverted and note that none of the evidence presented by either side was conclusive. I noted that Dr. Kleinman gave the opinion that seroreversion while possible, was extremely unlikely in this instance. Dr. Kleinman was subjected to very intense cross examination and provided plausible explanations and answers to the challenges put to his opinion and the basis for them.

173. While the Claimant's father has called the Claimants very competent treating doctors and presented well researched articles relating to seroreversion to support his contention that the Claimant was infected by an inclass donation and provided his own very moving testimony and able arguments, I consider the criterion imposed upon me by the HCV Settlement Agreement is more stringent.

174. The evidence on seroreversion is clearly evolving and not yet well settled in the medical research field. It appears to me that the argument put forward by the Claimant to the effect that seroreversion could have occurred makes it equally likely that such effect could have occurred in case of any of the Florida donations at the time of screening or donation and thus undermines the Claimant's argument that infection most probably occurred in only one of the three in class donations.

175. In the result, I am not satisfied that that seroreversion theory put forth by the Claimant meets the required test of highly persuasive evidence given the fact of 30 donations from Florida without evidence of tracebacks, and evidence of 11 out of Class Canadian donations for which only one donor declined to submit to retesting.

176. Finally, I have considered the theory of immunosuppression having an effect on the negative test of the Claimant in 1992 and providing an explanation for the negative traceback results.

177. The Claimant's father's testimony that the Claimant was without doubt immunosuppressed at the date of the June 7, 1992 negative Hepatitis C test was based on his understanding of the views of the treating physicians. I found their views were not unequivocal. While Dr. McGonigle concluded that the Claimant was immunosuppressed at the time of the 1992 test, it seemed to me that Dr. Grundy had more experience and had been involved in the Claimant's treatment for a longer period. I noted that Dr. Grundy's opinion on that question established no more than a possibility that it was the case. Moreover, Dr. Grundy was not prepared to rule out the possibility that the June 7, 1992 test was valid.

178. As stated earlier, I was not satisfied on the test enunciated by Pitfield, J. that the opinions of Drs. McGonigle, Grundy and Gutfreund on the significance of the AST/ALT levels are sufficiently well grounded to justify the conclusion that one of the inclass donors as opposed to one of the out of class Canadian donors who did not submit to a traceback, or one of the 30 or more U.S. donors must have seroreverted and caused the Claimant's infection. In the result, I must conclude that the theory of immunosuppression on the facts of this appeal does not meet the requirement of highly persuasive evidence.

179. I note that the Administrator under the Settlement Agreement is required to administer the Transfused HCV Plan in accordance with its terms. The Administrator does not have the authority to vary the terms of the Plan. Neither an arbitrator nor a referee may vary the terms of the Plan when asked to review the Administrator's decision.

180. In the result, I must uphold the Administrator's decision.

181. However, I do consider that due to the very sincere and considerable effort by this Claimant to establish his grounds for appeal, he should be entitled to his reasonable costs in seeking evidence to refute the results of the negative traceback.

Dated: April 28, 2006

Shelley L. Miller, Q.C. Referee

EDMDOCS|SHMILLER|113762-1