DECISION

BACKGROUND

1. On October 17, 2003, the Administrator denied the Personal Representative's claim on behalf of the deceased's estate for compensation under the Transfused HCV Plan. The claim was denied on the basis that the Personal Representative had not provided sufficient evidence that the deceased's death was caused by HCV.

2. On November 26, 2003, the Personal Representative requested that the Administrator's denial of the claim on behalf of the deceased's estate be reviewed by a referee.

3. On May 13, 2004, a hearing began before me. The hearing was adjourned to allow the Personal Representative to obtain further evidence in support of her claim.

4. On August 31, 2005, the hearing resumed before me. The hearing was adjourned to allow the Personal Representative to obtain more evidence and to allow both parties to summon witnesses. I issued a summons to obtain all medical records relating to the deceased from the Health Sciences Centre in Winnipeg. The records were obtained in January 2006.

5. On April 9, 2007, the hearing continued and was concluded.

FACTS:

6. In May 1986, the deceased underwent coronary bypass surgery. At that time, he underwent multiple blood transfusions. Two months later, further transfusions were received when he had an abscess drained.

7. In August 1986, the deceased was admitted to hospital suffering from acute jaundice and hepatitis which was noted in the discharge summary as post-transfusional non-A non-B hepatitis.

8. On February 18, 2003, Dr. James Parrott, the deceased's cardiologist, completed the Treating Physician Form. He answered "no" to the following questions:

It is my opinion that the HCV Infected Person's infection with HCV materially contributed to his or her disease level 6 medical condition.

It is my opinion that the HCV Infected Person's infection with HCV materially contributed to his or her disease level 5 condition.

In answer to questions 11 and 12 where Dr. Parrott was asked if the HCV infection materially contributed to the deceased's death, he answered "yes". Having answered "yes", he was asked how the HCV infection materially contributed to the death. He stated: "May have been contributory to the development of gastric cancer." On May 6, 2003, when the Fund sent a fax asking Dr. Parrott to clarify his response, he stated: "if we don't know for sure, there may still be evidence that I do not know of, or may be forthcoming in the future that may confirm this. Therefore HCV may indeed be contributory to this man's cancer and subsequent death."

9. The Administrator requested that Dr. Gary Garber review the deceased's file and provide the Administrator with an opinion on whether the HCV infection materially contributed to the deceased's death.

Dr. Gary Garber is head of the Division of Infectious Diseases at the Ottawa Hospital. In his report dated September 10, 2003, Dr. Garber's opinion was that there was no evidence that the deceased's HCV infection had any correlation with his stomach adenocarcinoma. He stated:

> Furthermore, it usually takes 10 or more years of a hepatitis C infection before liver damage becomes manifest and in most cases at least 20 years is required. The normal hepatocites seen on a liver biopsy is not in keeping with cirrhosis which can be a possible prelude to developing hepatic carcinoma.

Dr. Garber concluded that there is "absolutely no evidence to suggest that hepatitis C virus played a role in the death of this unfortunate individual."

10. On August 10, 2004, Dr. Rosemary Danielli, a family physician, forwarded a medical opinion with respect to the deceased. She reviewed some of the deceased's medical records and concluded:

> The attending physician to [the Claimant]diagnosed his post transfusion hepatitis during a hospital admission from August 12 to August 20, 1986. He was very sick with hepatitis at that time. The hepatitis caused significant morbidity and suffering in the form of weakness, anorexia, 40 pound weight loss, progressive jaundice, nausea, chills and rigors. Unfortunately, [the Claimant]died just two years later, on March 20, 1988. His unfortunate death was caused by complications arising from gastric cancer, hepatitis C (non-A, non-B hepatitis) and pneumonia.

EVIDENCE:

11. On the first hearing day, the Personal Representative testified about the deceased's health subsequent to his heart bypass surgery on May 29, 1986. The Personal Representative nursed him after the surgery and his discharge from hospital. The deceased was extremely weak and not recovering well. He was readmitted to hospital on August 12, 1986 and discharged on August 20, 1986. His discharge diagnosis stated that he had post-transfusional hepatitis non-A, non-B and pernicious anaemia. The Personal Representative testified that the deceased was badly jaundiced during this period of time.

The deceased was readmitted to the Health Sciences Centre in Winnipeg, Manitoba on January 22, 1988 and discharged on February 2, 1988. He had a high fever, was jaundiced, had abdominal pain, diarrhea and was lethargic. He died on March 20, 1988.

On January 25, 1988, a lab report noted adenocarcinoma of the stomach and distension of the liver. The discharge summary dated February 2, 1988 and signed by Dr. E.G. Brownell states:

> Prior examinations over the last couple of months including abdominal ultrasound and CT scan of the liver had both suggested the possibility of metastatic carcinoma in his liver. In spite of this a biopsy done about two months previously under echo control had shown normal hepatocites. Nevertheless the assumption is that he has metastatic disease of his liver and that the echo control biopsy just missed the appropriate area.

12. Ms. Carol Miller, the Appeal Coordinator for the Fund, whose role it is to review claims and prepare them for the Committee of Evaluators, testified at the hearing. As the deceased passed away before January 1, 1999, a traceback of the seventeen units of transfused blood was conducted. Ms. Miller testified that the traceback was inconclusive. Thirteen units were traced to donors who were not infected with Hepatitis C. However, it was impossible to trace the donors for 4 units of blood.

13. Dr. Danielli testified on the final day of the hearing by teleconference. Dr. Danielli had reviewed the deceased's physician's progress notes and Dr. Gary Garber's report. She was asked how she concluded that Hepatitis C materially contributed to the death of the deceased. She responded that the deceased had a complicated medical history. However, he first presented in 1986 with acute jaundice resulting from post-transfusional Hepatitis C. Dr. Danielli testified that the fatigue and weight loss resulting from Hepatitis C were co-morbid factors which materially contributed to the deceased's death. She pointed out that the deceased's weight loss predated his gastric cancer diagnosis, as did the distension of his liver. If the weight loss and the fatigue were attributable to gastric cancer, the deceased would not have been jaundiced. Instead, he was ill with Hepatitis C, unable to eat and growing progressively weaker. Consequently, Dr. Danielli concluded that the deceased's HCV infection materially contributed to his death.

14. Dr. Garber testified on the final day of the hearing via teleconference. He testified about his expertise treating patients infected with HCV and his research publications relating to Hepatitis C.

Dr. Garber acknowledged that the deceased had jaundice after post-transfusional infection with HCV. However, he attributed the deceased's weight loss and bleeding in 1988 to a large gastric tumour. The biopsy of the liver revealed normal hepatocites which confirmed the deceased did not have advanced liver disease. The liver function tests from January 30, 1988 revealed no active inflammation of the liver. Dr. Garber testified that he would not expect to see significant liver damage 2 years after contracting Hepatitis C.

Dr. Garber reviewed the deceased's file and confirmed that there was no evidence that HCV infection played a role in his death. In his opinion, all of the deceased's symptoms in January 1988 could be completely explained by the presence of gastric cancer. Although Dr. Garber conceded that the deceased was likely infected with Hepatitis C, he did not believe that Hepatitis C caused the death of the deceased. Dr. Garber testified that there is no medical link between HCV infection and gastric cancer.

Dr. Garber hypothesized that the fever, chills, and weakness experienced by the deceased could have been an overlap of symptoms. He conceded that his testimony was not the same as being at the bedside seeing the patient but he did not believe it was plausible given the short time from the date of the transfusion with no evidence of liver disease or failure that Hepatitis C had a role to play in the death of the deceased.

SUBMISSIONS:

15. Subsection 3.05(1)(a) of the Plan requires the HCV Personal Representative to provide proof that the death of the HCV Infected Person was caused by his infection with HCV.

16. Fund counsel submitted that the claimant had the burden of proving that the

death of the deceased was caused by HCV infection. Although the traceback was inconclusive with respect to 4 units of blood, in such circumstances, the presumption is in favour of the claimant that the deceased was infected with HCV as a result of the blood transfusion.

17. Fund counsel relied on the decision in Claim No. 2101 to support his submissions. Claim No. 2101 can be distinguished from the case before me in 2 major ways: 1) there was no compelling evidence in Claim No. 2101 that the deceased was infected with HCV; and 2) the opinion of the deceased's doctor was based on the complex clinical relationship between HCV and HBV infection.

18. The Personal Representative submitted that her father could not eat after being infected with HCV and was very weak. She claimed that the HCV infection hastened the deceased's death. The Personal Representative hypothesized that the deceased's HCV infection caused liver cancer which spread to the stomach causing gastric cancer. She countered Dr. Garber's thesis that it would take 10 years for the deceased to succumb to Hepatitis C. In her opinion, this neglected to take into account how vulnerable the deceased was at the time of the transfusions and the large number of transfused units of blood he received while ill. She noted the difficulties she faced obtaining relevant evidence to support her claim as both the deceased's physicians, Dr. Matwichuk and Dr. Brownell, are now deceased and Dr. Parrott had moved away.

ANALYSIS

19. The Personal Representative seeks compensation on behalf of the deceased's estate under the Transfused HCV Plan. In order for this claim to be successful, the deceased must meet the definition of "Primarily-Infected Person". The Transfused HCV Plan defines "Primarily-Infected Person", in part, as meaning "a person who received a Blood transfusion in Canada during the Class Period ...". Class Period is defined as meaning "the period from and including 1 January 1986 to and including 1 July 1990". I find that the deceased did receive more than one Blood transfusion in Canada during the

Class Period. I also find that the deceased was infected with HCV as a result of his blood transfusions.

20. Fund counsel and the Personal Representative's submissions must be considered in the context of the requirements for compensation set out in the Transfused HCV Plan. Subsection 3.05(1) of the Transfused HCV Plan requires that a Personal Representative claiming on behalf of an HCV Infected Person who has died must deliver to the Administrator an application form together with proof that the death of the HCV Infected Person was caused by his or her infection with HCV. The burden is on the Personal Representative to establish that the death of the deceased was caused by HCV infection. This proof need not be beyond a reasonable doubt but must satisfy the civil standard of proof on the balance of probabilities.

21. While I found Dr. Garber to be an impressive and knowledgeable expert witness, I was not persuaded by his conclusion that it was impossible that the deceased's HCV infection materially contributed to his death. I preferred the evidence of Dr. Danielli that the deceased's HCV infection materially contributed to his death together with his gastric cancer and pneumonia. I did not find it implausible that the deceased's HCV infection could have materially contributed to the deceased's death. I found Dr. Danielli's evidence compelling that the deceased's weight loss, jaundice and weakness predated his diagnosis with gastric cancer. I find that the deceased's HCV infection materially contributed to his death.

22. Consequently, I find that the Personal Representative has established on the balance of probabilities that the death of the deceased was caused by HCV infection. She has satisfied the requirements of subsection 3.05(1) of the Settlement Agreement by providing sufficient proof that the deceased's HCV infection materially contributed to his death.

CONCLUSION

23. I accept the claim of the Personal Representative and overturn the decision of the Administrator.

JUDITH KILLORAN
Referee

July 6, 2007
DATE