

HEPATITIS C

Sonya Norris
Science and Technology Division

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HEPATITIS C

BACKGROUND

In recent years, hepatitis C has become a familiar disease to most Canadians as a result of the much-publicized scandal about its transmission via blood transfusion or blood products. Although the disease itself is not new, the virus responsible for it has only recently been identified, and much work remains to gain a full understanding of how it affects the progression of the disease. This paper will review some of the major aspects of hepatitis C, including ongoing research aimed at developing an effective method of treatment, immunization and cure, and will summarize the issues surrounding compensation for Canadians who contracted hepatitis C through the blood supply.

TYPES OF HEPATITIS⁽¹⁾⁽²⁾

A wide spectrum of diseases are referred to as “hepatitis.” Each has a different cause, method of infection, symptomology, progression, treatment, and degree of lethality. All hepatitis, however, is characterized by hepatic inflammation, i.e., swelling of the liver tissue, which often results in permanent organ damage. Non-viral agents, such as alcohol, chemical poisons and drugs (both illicit and medicinal) can cause hepatitis. Infectious agents – viruses – can also produce the disease, and there is a growing “alphabet” of viral forms, from hepatitis A to hepatitis G. Hepatitis C and its cousins A and B are the most common types. Vaccines are available for both hepatitis A and hepatitis B.

(1) C. Everett Koop Institute, “Hepatitis C – An Epidemic for Anyone,” <http://www.epidemic.org>.

(2) S. D. Shafran and J. M. Cooly, “ABCDEFGH ...,” *The Canadian Journal of Infectious Diseases*, Vol. 7, No. 3, May/June 1996, pp. 181-2.

One method of characterizing the viral forms of hepatitis is to define whether the virus is transmitted through food (or water) or through blood (or other bodily fluids). Forms of hepatitis transmitted through contaminated food or water do not cause chronic (long-lasting) disease and tend to produce serious complications in only a small percentage of cases. Hepatitis A, for example, is spread principally through water or food that has become contaminated with infected fecal matter; as a result, it is more prevalent in less-developed countries. Hepatitis E is another food-borne viral hepatitis that is clinically very similar to hepatitis A, although the patient may be ill for a slightly longer time. Hepatitis F is a recent addition to the list of food-borne forms of hepatitis.

In blood-borne forms of viral hepatitis, transmission of the disease occurs primarily through contact with infected blood, or, less frequently, other bodily fluids. One such form is hepatitis B, which is highly infectious (more so than HIV) and is easily transmitted through sexual contact and breastfeeding, or even by casual family contact, as well as through blood-to-blood routes. Only 5-10% of those infected with the hepatitis B virus (HBV) become chronically infected; most of those infected successfully fight off the virus and are said to have suffered an acute infection. D and G are other blood-borne hepatitis viruses. The former appears only as a co-infection with HBV and serves to exacerbate symptoms. Hepatitis G is a recent addition to this list about which very little is known at this time.

Hepatitis C, the main subject of this document, is also transmitted primarily through blood, for example through intravenous drug use by means of shared needles. Before 1990, transmission took place largely during blood transfusions and the use of blood products. The hepatitis C virus is not as highly infectious as HBV and is not easily transmitted through intimate contact or bodily fluids. More than 80% of hepatitis C virus infections become chronic, however, and most are believed to lead to liver disease.

CLINICAL DESCRIPTION OF HEPATITIS C

A. Some Details About the Virus

The virus that causes hepatitis C (HCV) was identified in May 1987 by Chiron Corporation.⁽³⁾ Until that time, patients who tested negative for both hepatitis A and hepatitis B but who still showed symptoms of hepatitis were designated as having non-A,

(3) See Chiron Internet site at <http://www.chiron.com/public/about/pr/101100cbt.jsp>.

non-B hepatitis. In 1990, a test became available that specifically tested for the antibody produced against hepatitis C, but only since 1993 has it been possible to test directly for the virus itself. This is an important distinction, as a person is typically infected with the virus for four to six weeks before antibodies can be detected.

All organisms, with the exception of RNA (ribonucleic acid) viruses, store their permanent genetic information as DNA (deoxyribonucleic acid). Although some virus types use DNA like all other organisms, a large proportion of viruses – including HCV – store their genetic information in the form of RNA. This is a much less stable molecule than DNA and is susceptible to mutation at a much greater rate. Because RNA serves as an intermediary in the replication of genetic material in organisms that have DNA, it is not detected as a foreign entity by those organisms. These factors provide the key to the success of the virus in producing chronic infection that eludes the immune system.

The hepatitis C virus must attach to and infect liver cells in order to carry out its life cycle. The virus injects its RNA into a liver cell, which, perceiving it as its own “transient” RNA (“messenger” RNA or mRNA), proceeds to replicate it. In doing so, the infected cell shuts down most of its normal functions in order to conserve energy. Hundreds to thousands of copies of the viral RNA are produced in each infected cell. The host cells continue to cooperate by manufacturing the components needed for the viral RNA to assemble into virus particles until the host cell bursts, releasing the particles, which then infect new host liver cells.

The immune system of the infected individual is quick to recognize the invading virus and attempts to eliminate it. The high rate of mutation of the virus as it replicates, however, ensures that an “evolved” variant will be always able to elude the immune response and go on to replicate without interruption until the immune system makes its next attempt. In this way these viruses can evolve faster than the immune system can mount an effective response. This game of genetic hide-and-seek is also the reason why scientists have so far failed to discover an effective vaccine against hepatitis C.

B. Transmissibility of the Virus

It is believed that HCV is transmitted only by blood. It is unlike other blood-borne viruses, however, in that any source of blood or blood product appears capable of carrying it – even indirectly, such as through a razor or toothbrush.

In the 1970s and 1980s, many people became infected with HCV through blood transfusions. Hemophiliacs were particularly susceptible to contracting the disease, as the blood products they needed were derived from the blood of thousands of donors. Since the virus has been identified and tests for it have become available, the number of transmissions through the Canadian blood system has fallen to a negligible level. The most significant risk behaviour for contracting hepatitis C is illicit drug use; this accounts for as many as 40% of all cases. Indeed, the majority of injection drug users are HCV positive since the virus is transmitted not only through sharing needles but also through sharing other drug paraphernalia.

Other significant risk factors for infection are needlestick injuries, tattooing, body piercing, acupuncture, ear piercing, contact with contaminated medical equipment and sexual activity with multiple partners. Transmission through casual day-to-day contact (such as the sharing of razors) as well as from mother to child at birth is also possible, but the extent of transmissibility by these routes remains unknown.

C. Symptoms and Diagnosis of Hepatitis C⁽⁴⁾⁽⁵⁾

In most cases, the patient does not exhibit symptoms when newly infected with the virus (in the acute phase). Any symptoms are sometimes dismissed as the flu or a general malaise and are not followed up appropriately so that the infection can be identified. In a smaller percentage of cases (3-5%), the infected person experiences an acute reaction two to three weeks after infection, with severe abdominal pain, nausea, vomiting and extreme fatigue. Jaundice, loss of appetite, weight loss and lethargy usually follow, but the severity of these symptoms usually decreases over time.

The most common symptoms of chronic infection, which may not appear for several years, are mild fever, muscle and joint aches, nausea, vomiting, loss of appetite, vague abdominal pain and sometimes diarrhea. Another complaint not uncommon among hepatitis C sufferers is itchiness of the skin; however, because this symptom is itself poorly understood, its association with HCV status is not clear. Other, less frequently reported symptoms include dark urine, light-coloured stools, and weight loss. As with the milder acute reactions, many individuals dismiss these symptoms as flu-like or may not even recognize them as being sufficiently serious to require medical attention.

(4) Paul R. Gully and Martin L. Tepper, "Hepatitis C," *Canadian Medical Association Journal*, Vol. 156, No. 10, 1997, p. 1427.

(5) World Health Organization, "Hepatitis C," Fact sheet, rev. October 2000, <http://www.who.int/mediacentre/factsheets/fs164/en/index.html>.

In a small proportion of hepatitis C patients, progression of the disease, usually over several decades, produces symptoms associated with poor liver functioning. In addition to the symptoms already listed, patients may also experience swelling of the arms and feet, readiness to bruise, intermittent confusion, disorientation or inability to carry out complex mental tasks.

Many infected individuals are diagnosed with the disease when they seek medical attention for chronic fatigue. Others are not diagnosed until severe liver problems prompt them to see a doctor. Some individuals are identified as having hepatitis C when routine blood tests show abnormally high liver enzymes⁽⁶⁾ levels or when they are screened before donating blood.

Elevated liver enzymes will prompt the physician to test for the antibody to the HCV and other conditions associated with liver disorders. A newly infected individual will test negative, as it can take three to four months for the immune system to produce any antibody to the virus. A patient found to have the HCV antibody is often said to be “anti-HCV positive.” Such a person will generally have acquired a chronic infection, although a very small percentage of people successfully eliminate the virus from their system. Since 1993, it has been possible to test for the virus itself. The testing technique, which analyzes for the virus RNA, has now been adopted by Canadian Blood Services for standard screening of blood products. If drug treatment is going to be initiated, or if there is reason to suspect severe liver damage (which can be confirmed only by tissue analysis) a biopsy of the liver is required.

D. Progression of the Disease

Although the progression of hepatitis C is relatively slow, the consequences can be quite debilitating or even fatal. As yet, because of the relatively short time since the virus has been identified, the percentage of sufferers who progress to liver disease and death is not known. The chronic inflammation of the liver associated with hepatitis C leads to scarring (“cirrhosis”) as the liver attempts to protect itself from the inflammation. Approximately 20% of those chronically infected will develop cirrhosis after 20 years of infection. Of these, 1-5% will develop cancer of the liver (hepatocellular carcinoma) each year.⁽⁷⁾ Hepatitis C also exacerbates

(6) Liver enzymes are enzymes specific to the liver and normally found in the blood. Any assault on the liver can result in higher levels of these enzymes, which are always present in low amounts due to the normal death of liver cells and release of the enzymes into the blood.

(7) World Health Organization (2000).

coexisting liver conditions. Once the disease has progressed to cirrhosis or cancer, a liver transplant is the only option for survival. Liver disease caused by HCV infection is a leading cause for liver transplantation in Canada, currently accounting for almost one-quarter of liver transplants.⁽⁸⁾

A large number of additional complications have also been linked to this disease, although the relationships are at present not entirely understood. It is believed that as many as 20% of hepatitis C patients also suffer from disorders of the thyroid, intestine, eyes, joints, blood, spleen, kidneys or skin.⁽⁹⁾ Many of these complications are suspected of being associated with interferon therapy, described below, which is used by a significant number of patients.

CURRENT TREATMENTS AND MANAGEMENT

Once diagnosed, the individual must consider whether treatment or management of the disease, by means of drugs, alternative treatment, or lifestyle modification, is appropriate. In cases where the patient is not suffering any physical symptoms and the liver enzymes levels are clinically stable, doctors frequently do not advocate aggressive treatment.

A. Conventional Drug Therapy

Since hepatitis C was identified, the only approved drug therapy has been an interferon given the trade name Intron-A. Interferon is a family of glycoproteins derived from the human cells normally involved in fighting viral infections by preventing virus multiplication in cells. Interferon therapy is aimed primarily at patients with HCV infection and in whom persistent elevation of the liver enzymes indicates chronic hepatitis. Interferon is not considered a cure. Only 25% of infected patients may be candidates for this therapy and, of those treated, only 10-25% will show prolonged reduction of liver enzyme and virus levels.

Advances in drug therapy have always included interferon, in varying dosages and perhaps in conjunction with a second drug. Interferon therapy originally consisted of injections three times weekly over a 12-month period or longer. This therapy would be discontinued if no improvement in liver enzymes was seen after a reasonable time (two or

(8) Information from the Canadian Institute for Health Information at <http://www.cihi.ca>.

(9) "Hepatitis C – An Epidemic for Anyone," <http://www.epidemic.org>.

three months). Of those individuals who showed improvement while on therapy, a large proportion would relapse upon discontinuation of the drug. Subsequently, higher doses of interferon were prescribed by increasing either the amount of the drug or the frequency of administration; this approach appeared to have more success in viral eradication during therapy and a higher post-treatment success. Unfortunately, the incidence of relapse remained high, and larger doses did not appear to improve sustained response rates substantially. Interferon itself is a harsh drug to take, with many debilitating side effects, and several physicians have questioned its use, given the very low long-term remission rates. Another form of interferon, called PEGASYS (peginterferon alfa-2a), lasts longer in the body and is slightly better tolerated.

Rebetron was approved by the Therapeutic Products Directorate at Health Canada in March 1999. This is a form of therapy in which interferon is combined with a drug called ribavirin, which, like interferon, acts by modulating the immune response. This therapy combination does increase the percentage of patients showing a sustained decrease of virus levels in the blood over interferon alone. Significant adverse effects from ribavirin can limit the use of combination therapy for many individuals.⁽¹⁰⁾

B. Alternative Drug Therapy

Many herbal medicines are claimed to be helpful in treating liver diseases in general, and some are said to be effective specifically for hepatitis C. Some studies suggest that the most widely acclaimed of these, silymarin (derived from the milk thistle plant), may help liver cells regenerate and stabilize liver cell membranes. Proponents claim that silymarin boosts the ability of the liver to filter blood and prevents damage to it from toxins, including solvents, alcohol, drugs, most pesticides and herbicides, and bacterial compounds such as those associated with food poisoning, and may help treat or prevent cirrhosis, hepatitis and other liver diseases.⁽¹¹⁾ However, other studies argue that the data are too limited to establish the benefits of silymarin.⁽¹²⁾

(10) Mitchell Schiffman, "Hepatitis C: Dilemmas in Treatment," Presented at the Digestive Disease Week 1999 Annual Meeting, 17 May 1999.

(11) M. I. Thabrew, "Phytogenic Agents in the Therapy of Liver Disease," *Phytotherapy Research*, Vol. 10, No. 6, September 1996, pp. 461-7.

(12) B. P. Jacobs et al., "Milk thistle for the treatment of liver disease: a systematic review and meta-analysis," *American Journal of Medicine*, Vol. 113, No. 6, October 2002, pp. 506-15.

C. Lifestyle Management

Many individuals suffering from hepatitis C feel that they can minimize fatigue by having healthy sleeping habits, allowing for short naps, eating wisely, and maintaining a constant modest level of physical exercise. In terms of diet, patients are encouraged to ensure they have the adequate protein intake essential for repairing liver cells, consume complex carbohydrates liberally, and restrict fat, while still having an adequate intake of essential fatty acids. Caloric intake should not be limited unless there is a need for the patient to lose weight. HCV-positive individuals are advised against being overweight as this is associated with some other liver abnormalities such as fatty deposits, which can lead to inflammation of the liver. Because any additional stresses on the liver will exacerbate the effects of the virus, alcohol should be avoided. It is also recommended that patients get immunized against hepatitis A and B, and avoid unnecessarily consuming substances that require liver metabolism.⁽¹³⁾ In this regard, many medications should be kept to a minimum. Common medications such as aspirin, and ibuprofen can be toxic if used habitually by someone with compromised liver function.⁽¹⁴⁾

CURRENT RESEARCH INTO THE DISEASE

There is currently no cure for hepatitis C. Below is a brief description of the current state of research in the areas of treatment and prevention.

A. Treatment

Treatment of the disease can encompass several areas, including strategies to alleviate or eliminate symptoms, slow down or reverse tissue damage (with or without symptoms), or cure the disease through elimination of the virus. Most current research is focusing on the last two categories.

(13) Public Health Agency of Canada, "Hepatitis C: Nutrition Care Canadian Guidelines for Health Care Providers," <http://www.phac-aspc.gc.ca/hepc/pubs/nc-hcp-sn-is/index.html>.

(14) T. R. Riley 3rd and J. P. Smith, "Ibuprofen-Induced Hepatotoxicity in Patients with Chronic Hepatitis C: A Case Series," *American Journal of Gastroenterology*, Vol. 93, No. 9, September 1998, pp. 1563-5.

A significant amount of research continues on existing drug therapies. Interferon therapy has proved to be helpful in a limited proportion of HCV infections (20-30% response rate), but the proportion increases when interferon is combined with ribavirin (40-60% response rate). The harshness of these drugs detracts from their appeal as treatments, however. Researchers have been interested in identifying which hepatitis C sufferers would be most likely to benefit from them, in order to minimize the application of ineffective therapies. Other research consists of analyzing and overcoming possible reasons for non-response to interferon.⁽¹⁵⁾ In addition, there is some evidence that antiviral therapy, even in non-responders, may slow progression of the disease.

Research into a cure for the disease has focused mainly on genetically engineered therapeutic vaccines and drugs intended to “overwhelm” the virus before it is able to mutate sufficiently to elude treatment. Recently, a technique called ribonucleic acid interference (RNAi) has been explored as a possible approach for treatment of HCV infection.⁽¹⁶⁾ RNAi can selectively inhibit the expression of genes and can cause the degradation of any RNA. Unlike the vast majority of living things that have DNA as their genetic material as described earlier, HCV stores its genetic information as RNA and is therefore vulnerable to RNAi.⁽¹⁷⁾

B. Prevention

Research into the prevention of HCV infection centres on developing a vaccine. This research has proved to be very frustrating because of the mutative nature of the virus. It is hoped that biotechnology will make a genetically engineered vaccine a reality in the near future. Any successes in the race to produce a vaccine against HIV would speed the search for a vaccine against HCV, as these two viruses share a number of physical properties.⁽¹⁸⁾

(15) W. Wayt Gibbs, “In Focus: R_x for B and C,” *Scientific American*, Vol. 280, No. 3, March 1999, pp. 17-8.

(16) Mario Stevenson, “Therapeutic Potential of RNA Interference,” *New England Journal of Medicine*, Vol. 351, 2004, pp. 1772-7.

(17) A schematic explanation of this technique is available at http://www.ambion.com/techlib/append/RNAi_mechanism.html.

(18) David B. Weiner and Ronald C. Kennedy, “Genetic Vaccines,” *Scientific American*, Vol. 281, No. 1, July 1999, pp. 50-7.

HOW IMPORTANT IS THE HEPATITIS C ISSUE FOR CANADA?

A. How Many People Suffer?

According to Health Canada, about 250,000 people in Canada (approximately 0.8% of the population) may be infected with HCV. The incidence (i.e., rate of new cases) has slowed significantly in recent years and now stands at 10-20 per 100,000 people per year.⁽¹⁹⁾ The prevalence of HCV infection in Canada is thought by some to be higher than 0.8%, however, since the Centers for Disease Control and Prevention in Atlanta has reported the American prevalence to be about 1.8%.⁽²⁰⁾ Many infected individuals are undiagnosed because of the high proportion of asymptomatic, or slightly symptomatic, infected persons who do not seek medical attention. Hepatitis C is thus referred to as the “silent epidemic.” From the estimate of 250,000 cases, it has been projected that approximately 50,000 cases (20%) will progress to cirrhosis, which, based on a rate of 1% to 5% per year, could result in between 500 and 2,500 cases of hepatocellular carcinoma a year.

B. Compensation for Tainted Blood Victims

1. The Debate Over Surrogate Testing⁽²¹⁾

In the early 1970s, donated blood began to be screened for hepatitis B, the only form of chronic viral hepatitis known at the time. It was expected that this would eliminate all post-transfusion hepatitis; however, hepatitis continued to be associated with transfusion. Many scientists felt that occurrences of post-transfusion hepatitis would be significantly reduced as a result of screening donated blood for an elevated level of the liver enzyme alanine amino transferase (ALT) and for the antibody to the hepatitis B core antigen (anti-HBc). Such screening, which does not test specifically for the infectious agent, is referred to as surrogate testing. Other scientists and authorities believed that the benefits of such testing were questionable, and that it would be ethically and morally difficult, if not impossible, to design the research

(19) Health Canada, Hep C Fact Sheet, Blood-borne pathogens section, 2003.

(20) US Centers for Disease Control and Prevention, Hepatitis C fact sheet, <http://www.cdc.gov/ncidod/diseases/hepatitis/c/fact.htm>.

(21) This section is adapted from Mr. Justice Horace Krever, *Commission of Inquiry on the Blood System in Canada – Final Report*, Vol. 2, Chapters 23 and 24, Minister of Public Works and Government Services Canada, 1997.

studies needed to confirm its usefulness. Nevertheless, the United States ordered surrogate testing of all donated blood by 1986, although many centres within the country had done this independently as early as 1982.

Canada followed this issue as it developed in the United States and on several occasions considered surrogate testing of all donated blood. Conflicting opinions among different authorities (such the Canadian Red Cross Society, the Bureau of Biologics and the Canadian Blood Agency), however, in addition to budgetary considerations, delayed a decision on implementing such testing. When a study of surrogate testing was begun in Canada in 1989 (such a study having been considered unacceptable in the United States), the Red Cross was required by regulators at Health Canada not to implement surrogate testing while the study was in progress. Eventually, with the introduction of a specific test for the hepatitis C virus in early 1990 and the subsequent availability of Chiron's first-generation anti-HCV test kit, the debate about whether to implement surrogate testing in Canada became obsolete.

Thus, between the 1986 implementation of surrogate testing in the United States and the introduction of the anti-HCV kit in 1990, blood in Canada was not screened at all for "non-A, non-B post-transfusion hepatitis." It is on this ground that those who contracted the disease through blood products in Canada between those dates demanded compensation from the federal and provincial governments.

2. Federal Compensation Packages

a. Compensation for Those Infected Between 1986 and 1990

On 27 March 1998, the federal government announced a compensation package of \$1.1 billion for those who had contracted hepatitis C through the Canadian blood supply between 1986 and 1990. These were estimated to number as many as 10,000 individuals,⁽²²⁾ although the figure could well be less.⁽²³⁾ This compensation arrangement had been forged with the provincial and territorial governments and was made up of \$300 million in provincial/territorial funds and \$800 million in federal funds. This figure was reduced slightly on 16 December 1998, when it was decided that about \$58 million of it would go to compensate secondarily infected HIV victims.

(22) Hepatitis C Class Action – Class Counsel Statement, Canada News-Wire, 15 June 1999.

(23) Health Canada, *Report on the Meeting of the Expert Panel on Hepatitis C Epidemiology*, 24 July 1998.

On 18 December 1998, details of the package were revealed after much negotiation between a federal–provincial/territorial legal negotiating team and the counsel for the class action suits. In May 1999, a settlement valued at \$1.118 billion plus interest was reached; this included compensation for those individuals secondarily infected with HIV. The proposed settlement calls for an initial payment of \$10,000 to every person who became infected with HCV through the blood system between 1 January 1986 and 1 July 1990. Individuals would also be eligible for additional compensation, depending upon the severity of their disease. Claimants might also be eligible to receive compensation for loss of income, loss of services in the home, costs of care, costs of HCV drug therapy, costs of uninsured treatment and medication and out-of-pocket expenses. They would be able to apply for more compensation as their disease progressed, up to a maximum payment of \$240,000. In addition, a death benefit category would compensate the patient’s estate, should death be directly attributable to hepatitis C contracted through the blood supply between the relevant dates.⁽²⁴⁾ The award would not be taxable and would not affect social assistance benefits. In return, those accepting the offer would have to sign a waiver giving up their right to sue the Red Cross and the federal, provincial or territorial governments.⁽²⁵⁾ The offer was filed with the courts of Ontario, British Columbia and Quebec and accepted by all three in 1999. Upon acceptance of this package, the terms would be binding and cover all class action and non-class action suits that had been filed, including lawsuits in all other provinces. Generally, all claims must be submitted prior to 30 June 2010.

b. Compensation for Those Infected Outside of the 1986-1990 Window

At the time that this federal compensation package was announced, many people felt that all victims who acquired hepatitis C through tainted blood should be compensated, not just those infected between 1986 and 1990. When, in September 2004, it was revealed that the compensation fund still contained over \$1 billion, there were renewed calls to open it up to all those infected with hepatitis C through tainted blood. In November 2004, the Minister of Health announced that the government would enter into discussions regarding the options for compensating people who were infected outside of the 1986-1990 timeframe. These discussions began in February 2005 and included the overseers and administrator of the fund, lawyers for

(24) Health Canada, “Proposed 1986-1990 Hepatitis C Settlement Agreement – Summary Overview,” http://www.hc-sc.gc.ca/ahc-asc/media/nr-cp/1999/1999_84bk1_e.html.

(25) Health Canada, “Proposed 1986-1990 Hepatitis C Settlement Agreement: Chronology of Key Developments,” http://www.hc-sc.gc.ca/ahc-asc/media/nr-cp/1999/1999_84bk2_e.html.

ongoing class action lawsuits, as well as federal, provincial and territorial governments. In November 2005, the federal government entered into a memorandum of understanding with the lawyers representing those infected before 1 January 1986 and after 1 July 1990, committing the federal government to compensate these individuals. In July 2006, the prime minister announced that an agreement had been reached on the elements of a settlement for those who had been infected with hepatitis C outside of the 1986-1990 window through the blood supply. The package would be separate from the previous one established for those infected between 1986 and 1990, but would be similar in size, that is, approximately \$1 billion; specifics of the compensation would also be similar. The proposed settlement was sent to the courts of British Columbia, Alberta, Ontario and Quebec for approval, which was announced on 8 June 2007. The Fund Administrator and plaintiffs' counsel now must create the administrative structure of the settlement before compensation payments can be made.

3. Provincial Compensation Packages

When the federal compensation package was initially announced in March 1998, it was with the cooperation of all provinces and territories, the provinces having expressed support for compensating only those individuals infected between 1986 and 1990 through their endorsement of the federal package. Over the next several months, however, some provinces began to speak of expanding the compensation to all those who had acquired HCV infection through the blood system, regardless of when. Most of these provinces felt that such expanded compensation should come from federal funds.

Four provinces have compensation packages for those infected pre-1986 and post-1990. In November 1998, the Ontario government announced that it would compensate these victims; before the end of December that year, it began to distribute a lump-sum payment of \$10,000 per claimant, which subsequently increased to \$25,000.⁽²⁶⁾ The Government of Quebec has also established a compensation program for those infected outside of the 1986-1990 time frame. Following the program's establishment in May 1999, Quebec offered \$10,000 lump-sum payments to those who qualified. In January 2004, a new agreement was reached allowing for an additional \$14,500 to be paid out per claimant.⁽²⁷⁾ On 18 January 2001, the Government of Manitoba announced that it would offer a one-time payment of \$10,000 to

(26) Government of Ontario Hepatitis C Assistance Plan:
http://www.health.gov.on.ca/english/public/program/hepc/hepc_assistplan.html.

(27) Régie de l'assurance maladie Québec, "Financial Assistance for Persons Infected With the Hepatitis C Virus," http://www.ramq.gouv.qc.ca/en/citoyens/contributionetaidefinancieres/hepatite_c.shtml.

those infected outside of the 1986-1990 window.⁽²⁸⁾ In 2003, British Columbia settled a class action lawsuit by providing the proceeds from the sale (\$6.5 million) of a Vancouver Red Cross building for compensation. Average individual compensation was about \$5,000.⁽²⁹⁾

4. Liability of the Canadian Red Cross Society

The Canadian Red Cross, just prior to relinquishing its control of the blood supply in the fall of 1998, filed for bankruptcy protection so that it could restructure its finances in the face of \$8 billion in lawsuits filed primarily by those who had contracted hepatitis C through the blood system outside the 1986-1990 time frame. The charity has expressed a desire to fulfil its moral obligation to these victims but emphasizes its financial constraints in offering compensation. It had hoped to create an acceptable special compensation fund for hepatitis C victims; however, in March 1999, its offer of \$60 million to victims excluded from the federal compensation package was rejected.⁽³⁰⁾ In June 2001, a settlement of \$63 million was ordered by the Court of Ontario. The administrator of this settlement fund is KPMG.⁽³¹⁾

CONCLUSION

The slow progression of Hepatitis C to its potentially fatal end, combined with the large proportion of affected individuals who are unaware of their infection, has led it to be labelled the “silent epidemic.” Now that the disease has been identified and many people are known to have suffered from it for many years, the associated health problems are receiving more attention. Each year, more cases of infection are reported. This does not point to a surge in new infections, but rather to increased diagnoses of chronic infection after symptoms finally emerge. Hepatitis C is a major contributor to liver disease, cirrhosis and liver cancer. Research continues in the areas of treatment and prevention; to date, no cure has been found.

(28) Government of Manitoba, “Manitoba Extends Extraordinary Assistance to Hepatitis C Patients: Chomiak,” News release, <http://gov.mb.ca/chc/press/top/2001/01/2001-01-18-01.html>.

(29) Government of British Columbia, Ministry of Health, “Supporting British Columbians Infected With Hepatitis C,” www.health.gov.bc.ca/library/publications/year/2004/Supporting_HepC.pdf.

(30) Mark Kennedy, “Blood Victims Reject \$60M Offer: Hep-C Victims Say Red Cross Proposal Not Enough,” *Ottawa Citizen*, 30 March 1999, p. A7.

(31) See KPMG website, <http://www.kpmg.ca/en/ms/hepatitisc>.