This is the 1st Affidavit
of Dr. Samuel S. Lee in this case
and was made on January 26, 2016

No. C965349
Vancouver Registry

In the Supreme Court of British Columbia

Between:

Anita Endean, as representative plaintiff

Plaintiff

and:

**The Canadian Red Cross Society
Her Majesty the Queen in Right of the Province of
British Columbia, and The Attorney General of Canada**

Defendants

and:

**Prince George Regional Hospital, Dr. William Galliford, Dr.
Robert Hart Dykes, Dr. Peter Houghton, Dr. John Doe,
Her Majesty the Queen in Right of Canada, and
Her Majesty the Queen in Right of the Province of British Columbia**

Third Parties

Proceeding under the Class Proceedings Act, R.S.B.C. 1996, C. 50

Court File No. 98-CV-141369-CP00

ONTARIO
SUPERIOR COURT OF JUSTICE

B E T W E E N :

DIANNA LOUISE PARSONS, MICHAEL HERBERT CRUICKSHANKS, DAVID TULL, MARTIN HENRY GRIFFEN, ANNA KARDISH, ELSIE KOTYK, Executrix of the Estate of Harry Kotyk, deceased and ELSIE KOTYK, personally

Plaintiffs

and

THE CANADIAN RED CROSS SOCIETY, HER MAJESTY THE QUEEN IN RIGHT OF ONTARIO and THE ATTORNEY GENERAL OF CANADA

Defendants

and

HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF ALBERTA
HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF SASKATCHEWAN,
HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF MANITOBA,
HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF NEW BRUNSWICK
HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF PRINCE EDWARD ISLAND,
HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF NOVA SCOTIA
HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF NEWFOUNDLAND,
THE GOVERNMENT OF THE NORTHWEST TERRITORIES,
THE GOVERNMENT OF NUNAVUT and THE GOVERNMENT OF THE YUKON TERRITORY

Intervenors

Proceeding under the Class Proceedings Act, 1992

Court File No. 98-CV-146405

B E T W E E N:

JAMES KREPPNER, BARRY ISAAC, NORMAN LANDRY, as Executor of the Estate of the late SERGE LANDRY, PETER FELSING, DONALD MILLIGAN, ALLAN GRUHLKE, JIM LOVE and PAULINE FOURNIER as Executrix of the Estate of the late PIERRE FOURNIER

Plaintiffs

and

THE CANADIAN RED CROSS SOCIETY, THE ATTORNEY GENERAL OF CANADA and HER MAJESTY THE QUEEN IN RIGHT OF ONTARIO

Defendants

and

HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF ALBERTA, HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF SASKATCHEWAN, HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF MANITOBA, HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF NEW BRUNSWICK, HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF PRINCE EDWARD ISLAND HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF NOVA SCOTIA
HER MAJESTY THE QUEEN IN THE RIGHT OF THE PROVINCE OF NEWFOUNDLAND,
THE GOVERNMENT OF THE NORTHWEST TERRITORIES,
THE GOVERNMENT OF NUNAVUT AND THE GOVERNMENT OF THE YUKON TERRITORY

Intervenors

Proceeding under the Class Proceedings Act, 1992

CANADA

PROVINCE OF QUÉBEC

DISTRICT OF MONTRÉAL

NO : 500-06-000016-960

SUPERIOR COURT

Class action

DOMINIQUE HONHON

Plaintiff

-vs-

THE ATTORNEY GENERAL OF CANADA
THE ATTORNEY GENERAL OF QUÉBEC
THE CANADIAN RED CROSS SOCIETY

Defendants

-and-

MICHEL SAVONITTO, in the capacity of the Joint Committee member for the province of Québec

PETITIONER

-and-

FONDS D'AIDE AUX RECOURS COLLECTIFS

-and-

LE CURATEUR PUBLIC DU QUÉBEC

Mis-en-cause

CANADA

PROVINCE OF QUÉBEC

DISTRICT OF MONTRÉAL

NO : 500-06-000068-987

SUPERIOR COURT

Class action

DAVID PAGE

Plaintiff

-vs-

THE ATTORNEY GENERAL OF CANADA
THE ATTORNEY GENERAL OF QUÉBEC
THE CANADIAN RED CROSS SOCIETY

Defendants

-and-

FONDS D'AIDE AUX RECOURS COLLECTIFS

-and-

LE CURATEUR PUBLIC DU QUÉBEC

Mis-en-cause

AFFIDAVIT OF SAMUEL S. LEE
(affirmed January 26, 2016)

I, DR. SAMUEL S. LEE, of the City of Calgary in the Province of Alberta, physician and professor of medicine, **AFFIRM AND SAY**:

1. The Attorney General of Canada has engaged my services in this matter. For reasons to be discussed below, I have been generally aware of the FPT Governments 1986-1990 Hepatitis C Settlement Agreement ("**Agreement**") from the time it was created, and understand that the Agreement compensates persons who received blood or blood products contaminated with hepatitis C virus ("**HCV**") during the 4 ½ year period preceding the introduction of direct viral testing for HCV in the Canadian blood system.

2. I understand from materials provided by counsel for the Attorney General of Canada that the courts have declared a part of the funds comprising the trust capital within the FPT Governments 1986-1990 Hepatitis C Settlement Trust Fund ("**Trust**") to be unallocated assets within the Trust that will not have to be called on when honouring all current and forthcoming financial commitments made under the Agreement. In connection with these judicial declarations I have been asked to provide medical opinion evidence to assist the courts in reaching their decision on an appropriate allocation of the actuarially and judicially declared unallocated assets within the Trust as at December 31, 2013. I understand that the Attorney General of Canada, as the representative of the federal government in this matter, has a financial interest in the funds forming the capital of the Trust.

a) Duty to the Court and Expert Qualifications

3. Although I am offering my expert evidence pursuant to my engagement by Canada, I understand and affirm that the evidence to be provided by me herein must be fair, objective and non-partisan, and that my duty to tender evidence in accordance with these principles prevails over any obligations I may owe to the Attorney General of Canada ("**Canada**") under the terms of my engagement. I also understand that the evidence I am offering must relate only to areas within the scope of my professional expertise. In the event that the courts find they require additional assistance from me in determining this matter, I am ready, willing and able to offer such assistance.

4. My qualifications to offer evidence requiring expertise in the field of viral hepatitis and specifically HCV are extensive and reflect many decades of work in the field. I am a licensed physician specializing in gastroenterology and hepatology. I have been Board-certified by the American Board of Internal Medicine in internal medicine since 1981 and in gastroenterology since 1983. I have been a Fellow of the Royal College of Physicians of Canada in Internal Medicine since 1982, and a member of the Alberta College of Physicians and Surgeons since 1988. In my clinical practice I have been a member of the active staff in Internal Medicine at Foothills Hospital, Calgary, Alberta, from 1988 to the present. I also have been a courtesy member of the consulting staff in the Department of Medicine at Peter Lougheed Hospital and Rockyview Hospital in Calgary from 1988 to the present. In the course of my clinical practice over the past 27 years, I have seen at least 3,000 patients infected with hepatitis C.

5. In my academic career, my present appointment as a full professor in the Department of Medicine within the Faculty of Medicine at University of Calgary was made in 2000. I previously had served as an assistant professor specializing in gastroenterology in the Faculty's Department of Medicine from 1988 to 1993, and as an associate professor in its Department of Medicine from 1993 to 2000. Prior to accepting my academic appointment at University of Calgary in 1988, I had held a research fellowship in hepatology and portal hypertension at the University of Paris in France from 1984 to 1987, and had taken advanced training in internal medicine and gastroenterology at University of Toronto from 1978 to 1984. I received my M.D. degree from Memorial University of Newfoundland in 1978. Attached hereto as **Exhibit "A"** to this my affidavit is a copy of my *curriculum vitae* updated to January 1, 2016.

6. In addition to my clinical practice and my teaching responsibilities, I have conducted medical research, published and lectured extensively in the field of viral hepatitis and specifically HCV infection and its treatment. For many years my responsibilities have included the review and oversight of work performed by other professionals in my field, both scholars pursuing advanced degrees in my area of expertise and other viral-hepatitis specialists seeking publication of their research in peer-reviewed professional journals.

b) <u>Canadian Association for Study of Liver and Medical Modeling</u>

7. In the course of my professional activities I also have contributed to a number of specialist organizations in my field, including the Canadian

Association for Study of the Liver ("**CASL**") and the International Association for Study of the Liver ("IASL"). I am currently serving as President of IASL. I previously served as president of CASL from 1998 to 2000.

8. During my tenure as CASL president, I was approached by legal counsel acting for the various stakeholders involved in the dispute over personal injuries suffered by persons that received HCV-contaminated blood or blood products in the Canadian blood system between January 1, 1986 and June 30, 1990. The lawyers were seeking neutral medical expertise from CASL to guide them in their efforts to create a compensatory benefits framework that reflects the health experience and outcomes of HCV-infected persons.

9. As CASL president I arranged for the formation of an expert committee of hepatologists, disease/outcome modellers and other experts as part of a CASL *ad hoc* committee known as the Medical Model Working Group ("**MMWG**") in order to provide expert assistance to the courts on this matter. Although I was not part of the working committee chaired by Dr. Murray Krahn, I read the report the MMWG prepared for this matter in 1999 and delivered it to legal counsel under my cover letter as president of CASL.

10. The MMWG study is entitled "Estimating the Prognosis of Canadians Infected with the Hepatitis C Virus through the Blood Supply, 1986-1990" and has gone through several revisions over the past fifteen years. I recently have reviewed the contents of the original MMWG study and some of its revisions in preparation for offering my own evidence in this matter.

11. For persons infected with HCV through contaminated blood and blood products during the class period, the MMWG Report presents a model for estimating the average rate of transition from one stage of HCV infection to the next over the course of the natural history of the disease. Historically, the HCV disease stages and their rates of transition have been important considerations for treatment of patients with HCV infections. The drug therapies available prior to the regulatory approval of direct-acting anti-viral agent ("**DAA**") therapy in Canada in 2011 were characterized by different cure rates at different stages of HCV infection. Prior to the advent of DAA therapy, the stage of the patient's disease was a very important factor in making treatment decisions. Of more immediate concern, the disease-stage progression model also was adopted and adapted to serve as the framework for compensating persons infected through exposure to contaminated blood from the Canadian blood system under the Settlement.

12. The original 1999 MMWG Report has been revised and updated from time to time over the past 15 years. The initial disease-stage transition model relied primarily on medical literature reporting on studies conducted in Canada and around the world rather than on the then very sparse claims data generated by research involving persons eligible to be compensated under the Settlement. The Fourth Revision of the MMWG Report, delivered in April 2011, builds on refinements developed in earlier revisions that were made to the model as greater volumes of claims data became available in the course of administering the Settlement. What is notable in the Fourth Revision is that, while the MMWG

continues to work from a blended pool of data, it also discusses the results obtained from a review of data drawn from Settlement claims alone.

13. That Fourth Revision, which was prepared in support of actuarial forecasting of Trust sufficiency as at December 31, 2010, has now been followed by the Fifth Revision delivered in September 2014 in support of the actuarial forecasts made as at December 31, 2013. The Fifth Revision also places significant reliance on actual claims data compiled by the Settlement administrator during the period preceding December 31, 2013, but its mode of presentation focuses on the percentage of patients progressing to cirrhosis rather than the average time required for patients to progress through the disease stages from infection to cirrhosis. I have reviewed excerpts from the Fourth Revision, and the Fifth Revision of the MMWG Report and will discuss these topics further in the body of my evidence.

c) <u>My Continuing Role with the CADTH Agency</u>

14. Since 2013 I also have contributed to clinical assessments of antiviral drugs under the auspices of the Canadian Assessment of Drugs, Technology and Health ("**CADTH**"). This independent, not-for-profit organization was established in 1989 by the federal, provincial and territorial governments to offer a coordinated approach for the evidence-based evaluation of medical devices and drugs that might be adopted by the Canadian health-care system. My own continuing role has been to help CADTH committees develop guideline

recommendations for use of new generations of antiviral drugs in the treatment of HCV infections.

15. I draw attention to my contributions to CADTH because they connect my own clinical and research experience to a coordinated national effort that provides updated, evidence-based guidance on the use of drugs to clinically manage and treat chronic HCV infections. CADTH clinical and economic evaluations of drug therapies are used to maintain the currency of the drug formularies for all provincial and territorial authorities other than Quebec. Although CADTH recommendations are subject to Health Canada's prior regulatory approval of a given drug for use in Canada, the pace required to bring timely, thorough clinical and economic evaluations of new therapies to decision-makers means that a CADTH application for common drug review often is set in motion while the regulatory review still is proceeding.

16. Contributors like me to CADTH clinical evaluations are well positioned to speak to the potential impact of emerging drug therapies that have a high likelihood of obtaining Canadian regulatory approval in the coming year or so. This rapid change in available drug therapies has been particularly evident in the field of DAA therapies for eliminating HCV infections since the first Canadian regulatory approvals were granted in 2011. My CADTH consultations have related to the introduction of DAA therapies to the Canadian health care system.

d) <u>Opinions Sought by Counsel</u>

17. I have been asked by counsel for Canada to provide expert advice regarding three aspects of the clinical management and treatment of HCV infections: (a) the impact of new drug therapies in eliminating HCV from all infected persons, including their effectiveness in eradicating genotypes that have been resistant to previous drug regimens; (b) the duration of the natural history of HCV disease from the date of infection to cirrhosis, end-stage liver disease and death; and (c) the HCV disease stages when infected persons are fully symptomatic and under the care of a physician.

e) The continuing revolution in drug therapies for managing HCV infections

18. One of my longstanding research interests has been the development of more efficacious antiviral therapies to eliminate HCV infections with a minimal treatment burden imposed on the patient. That interest has been reflected in my publications in the field over many years. In my opinion, within a very short time, new drug therapies will be available to eradicate HCV from almost 99% of all infected patients with minimal side effects arising during the course of treatment. In the future, a patient's viral genotype, co-morbidities and HCV disease stage probably will not have a major impact upon decisions taken to commence a course of treatment to eliminate the infection. At the same time I believe it to be an open question whether the retail price of next-generation medications will continue to raise ever higher financial barriers to universal access to treatment for HCV-infected persons. Manufacturers' competition for market share of newly

approved drugs and the eventual release of generic versions of DAA medications may help make the elimination of HCV infections more affordable.

19. From 2000 to approximately 2011, the standard antiviral therapy offered to patients infected with HCV was pegylated interferon plus ribavirin ("PR"). The efficacy of PR treatment often was disappointing, especially among patients infected with the most common HCV genotype in Canada, genotype 1. PR treatment also imposed a heavy health burden on many patients. Such patients frequently experienced significant side effects over a 24- to 48-week course of medication with the result that many of them abandoned their course of treatment prior to completion. In addition, PR drug therapy is expensive. HCV-infected patients, their private insurers or the public health-care system had to assume a significant economic burden for each course of individual PR treatment that was followed.

20. Developments in this area have been extremely positive since 2011 when Health Canada granted regulatory approval to the first of a new class of drugs described collectively as DAA agents. The past five years have witnessed tremendous advances in HCV management and therapy. As recently as 2010, clinicians relying on the older PR regimen were able to achieve cure rates of only some 50% for patients infected with the dominant genotype 1 subtype of HCV. Given that some two-thirds of HCV infections in Canada are genotype 1 infections, the PR regimen failed to meet the hopes of many patients who were prepared to endure the rigours of a course of treatment. The PR regimen typically required forty-eight weeks of subcutaneous injections of pegylated

interferon in addition to oral medications and induced many troublesome side effects such as flu-like symptoms, fever, fatigue, itching and even mental or emotional changes including depression. Many HCV-infected patients found the treatment burden to be too great and abandoned their therapy before its completion.

21. With the introduction of DAA agents in 2011, the treatment burden of HCV-infected persons declined markedly and health outcomes improved greatly for those receiving treatment. Even so, in some instances the success of the first generation of these agents was qualified. Not all HCV genotypes responded equally well to DAA agent therapy, and for some genotype infections optimal responses required the addition of ribavirin to ensure a sustained viral response ("**SVR**") that marks the elimination of the virus from the patient. In patients achieving SVR, hepatitis C virus is consistently undetectable, confirming that such individuals have been cured of their HCV infection.

22. In 2013 and 2014, Health Canada granted regulatory approvals to new all-oral DAAs that make possible treatment regimens having a much shorter duration without reliance on the use of pegylated interferon and its harsh side effects. Current DAA treatment consists of one to six pills per day, usually over a period of eight to twelve weeks with no discernible side effects and a cure rate exceeding 90%.

23. Today, in the vast majority of treatment situations, clinicians prescribe Harvoni, an oral medication requiring no combination with ribavirin or other PR

medications. Another oral medication, Holkira PAK, is sometimes less expensive than Harvoni and also quite effective in eradicating HCV from patients but it may have to be supplemented with ribavirin when treating certain HCV genotypes. If some side effects result from the Holkira PAK regimen that might be avoided through the use of Harvoni, they nevertheless are minimal.

24. The Holkira PAK treatment process is more elaborate than that for Harvoni with its single daily pill and has found much less favour with clinicians. If for some reason, perhaps the difference in retail price points of the two medications, a patient were to request specifically that he or she be treated with Holkira PAK rather than Harvoni, most clinicians would accede to his or her request. No appreciable disadvantage in treatment effectiveness would follow from prescribing Holkira PAK. There is no medical reason to suggest that any patient would undergo a hardship in following either Holkira PAK or Harvoni treatment regimens.

25. On January 19, 2016, Health Canada granted regulatory approval for another all-oral DAA combination drug, Zepatier, for treatment of patients with HCV genotypes 1 and 4. I expect to see regulatory approval granted later in 2016 for yet another generation of DAA medications that will offer even greater advantages for patient care, including those few patients who have had the misfortune to be infected with one of the less prevalent HCV genotypes that have proven to be more treatment resistant to earlier regimens. With the arrival of this next generation of DAA medications, very few cases will be seen where the virus cannot be eradicated.

26. Under the Agreement the parties created a benefits structure that reflects therapeutic management of HCV infections in 1999. That was a different therapeutic era for HCV-infected persons. Clinical diagnosis and management of the disease in 2016, or in 2014, or even in 2011 when DAA therapies first became available, have benefited from a series of substantial advances achieved in the treatment of viral hepatitis since 1999. The era of PR regimens with ALT counts to determine who should receive treatment is gone. Under today's treatment regimens, cure rates are very high and associated pain and discomfort correspondingly diminished.

27. Despite great medical progress over the past fifteen years, infection with HCV remains a serious problem for Canadian health-care providers. Current epidemiological estimates of infected Canadians from all sources of contamination range from 260,000 to 300,000 persons, a cohort that includes the approximately 4,000 living persons who are known to have been infected by HCV-contaminated blood between 1986 and 1990.

28. Although the virus can be eliminated very effectively from the great majority of persons who present for treatment, the prevalence of this infectious disease in the Canadian population as a whole has not decreased substantially. No vaccine yet has been developed to check infections and reinfections of the population exposed to this extremely heterogeneous virus cluster. Nor has Canada followed the example of other countries that have developed national HCV strategies to reduce substantially, if not eliminate altogether, this debilitating infectious disease from their populations.

29. Risk factors for a poor prognosis remain a concern despite the advent of DAA therapies. The liver is a major human organ and can suffer insult from agents other than viral hepatitis. Alcohol consumption, auto-immune conditions, obesity, gender and age all can influence the extent and progression of harm suffered by a liver infected with HCV. DAA therapies cannot eliminate these risk factors but they have reduced substantially the treatment burden formerly faced by patients taking a PR regimen. The result is that today a course of treatment can be initiated at almost any stage in the natural history of an HCV infection without significant additional risk to the patient. Even those patients suffering from quite advanced liver disease can benefit from the elimination of the burden imposed by HCV on their compromised health state.

30. To date the problem of patient affordability is one area where the new DAA therapies have failed to show any improvement over their PR predecessors. The former PR course of treatment ran for forty-eight weeks at a list price of some $20,000 per patient. That therapy no longer is used to treat genotype 1 HCV infections but the greater efficacy of the new DAA therapies has come with even greater list prices for treatment than PR therapies. A course of therapy to eliminate a patient's genotype 1 HCV infection now costs $44,000 for 8 weeks, and $67,000 for a twelve-week course of the highly effective Harvoni regimen. A twelve-week course of treatment with the slightly more cumbersome Holkira PAK regimen has a list price of $56,000. The treatment of genotype 2 and 3 HCV infections places as great an economic burden on health-care providers as does genotype 1 treatment. A twelve-week course of treatment relying on sofosbuvir

and ribavirin has a list price of $60,000 and a twenty-four-week regimen of these drugs is listed at $119,000. Even if emergent DAA-based regimens prove capable of eradicating all HCV infections regardless of genotype, the expense attached to the treatment of these infections remains a very real concern for health care.

31. The economic barrier to universal treatment of the estimated 260,000 to 300,000 HCV-infected persons in Canada is reflected in provincial formularies that all require at least the non-bridging fibrosis of disease stage F2 on the Metavir scale (roughly the upper half of Settlement Level 3) before they will consider funding a treatment regimen. This means that the chance of a patient at some less advanced stage of fibrosis receiving treatment is almost nil unless that patient happens to have a very generous private drug plan. It may be objected that the only modest benefit of treatment of early-stage HCV infection may be psychological. Treatment offers no useful medical or public health benefits in such patients other than alleviating anxiety from the stress of knowing that they are infected.

32. No such financial constraints will inhibit access to treatment for approximately 1.2% to 1.5% of the national cohort of HCV-infected persons. The approximately 4,000 living beneficiaries of the Settlement can be treated effectively without having to face the financial burden of treatment confronting the non-transfused general HCV population of Canada. A beneficiary of the Settlement with an RNA+ result confirming the presence of live HCV on his/her PCR test but no evidence of fibrosis can be treated with Harvoni or the

sofosbuvir+ribavirin combination oral medication to eliminate his/her asymptomatic HCV infection without side effects. With the availability of all-oral DAAs to clear the virus, most clinicians would be prepared to accede to the wishes of such a patient so long as he/she understood the financial burden of the antiviral therapy. Absent a private insurance plan to cover the cost of treatment for this Metavir stage 0 (Settlement Level 2) patient, the $44,000 - $119,000 retail price for his/her medication will be the responsibility of the Fund under the terms of the Settlement.

33. Not all 4,000 living members of the Settlement cohort will require DAA therapy in order to clear their HCV infection. Some living members of the settlement claimants already have been treated successfully with PR therapy or perhaps with first-generation DAA therapies. Absent re-infection from some other source these persons are highly unlikely to require further treatment to eliminate live virus from their bodies.

34. Another group of the Settlement beneficiaries will have suffered an acute HCV infection and then cleared the virus before experiencing the onset of a chronic disease process. Most of these persons will have cleared the virus within a year of their infection but the process of viral clearance by the immune system can occur in some patients after a longer time period. These are the Settlement beneficiaries who have been identified as Level 1 for compensation purposes.

35. According to paragraph 147 and related Tables 146a and 146b showing cohort size in the Morneau Shepell Actuarial Report Assessing Financial

Sufficiency of the Trust as at December 31, 2013 (dated April 8, 2015), some 700 (542 + 148) so-called RNA- claimants have been identified for compensation under the FPT Settlement within the group of 4,000 living claimants. All these RNA- claimants have been exposed to HCV without experiencing a chronic infection. Although for the rest of their lives these claimants probably will register their previous exposure to HCV by way of an antibody reaction on a blood-serum test, they will not present with any detectable viral particles on the standard diagnostic procedure known as a polymerase chain reaction test ("PCR Test"). They are not HCV-infected persons and their treatment with DAA medications would be pointless.

36. The phenomenon of spontaneous viral clearance ("**SVC**") is well recognized among clinicians even if clearance rates apparently do not occur uniformly across the population of all HCV-infected persons. Where the phenomenon occurs, it usually takes place within one year of exposure to HCV. Rates of viral clearance seem to be affected by risk factors such as alcohol consumption, obesity, age, gender and co-morbidities.

37. The SVC phenomenon received recognition in the 1999 medical model developed by CASL for purposes of compensating claimants under the FPT Settlement. SVC was assumed by CASL, at section 3.5.1, pages 7-8 of its report, to occur in some 15% of the cases of HCV infection caused by contaminated blood and blood products. Dr. Murray Krahn, the MMWG team leader, followed up with a letter dated June 10, 1999, and filed in these proceedings, in which he estimated an SVC rate of 20-25% in the Settlement cohort. According to the

1999 actuarial report prepared by Eckler Partners at its section 3.2 (page 8), class counsel instructed Eckler to use a 20% SVC rate in its analysis of the HCV-exposed persons eligible to be compensated under the Settlement.

38. Studies from the American blood system in the 1980s have suggested a somewhat higher spontaneous viral clearance rate, perhaps as much as 30%, while a European study of accidental infection of pregnant women through administration of an HCV-contaminated medication has documented clearance rates in the range of 50% for what was a non-transfused group of young adult females. From my own clinical experience with thousands of HCV-infected patients I would expect a clearance rate of at least 25% among the persons eligible to claim compensation under the FPT Settlement.

39. Spontaneous clearance of an acute HCV infection illustrates a broader problem for the clinical management of this disease. The symptoms associated with an acute HCV infection are not particularly debilitating. Most victims would not seek medical attention for treatment of such symptoms. The transition from an acute to a chronic phase of the disease also is not marked by symptoms that would prompt an infected person to seek medical attention. Chronically infected HCV sufferers can remain asymptomatic for many years. Without diagnostic testing to identify exposure to HCV, these asymptomatic infected persons are unlikely to be aware that they are carrying the virus.

40. Unlike the United States of America which has enacted a national HCV-identification and HCV-management strategy that is supported by the Center for

Disease Control, arguably the best viral hepatitis epidemiology/information centre in the world, the Canadian health-care system does not pursue a national strategy for anti-HCV antibody testing of the general population. Asymptomatically HCV-infected persons may receive diagnostic testing for previous HCV exposure if on an intake examination they present with risk factors and complaints that are not inconsistent with possible HCV infection. They also would be HCV-antibody or HCV-PCR tested where a procedure could create a risk of viral transmission to other persons such as in cases of blood or organ donations.

41. Persons who test positive for spontaneous viral clearance or for asymptomatic chronic infection often will have no immediate explanation for when or how they were exposed to HCV. Nonetheless, as part of their initial history-taking, all medical specialists would seek to determine the mode of acquisition of HCV infection. In taking each patient's history, medical personnel will canvass known risk factors such as intravenous drug use ("**IVDU**"), immigration from an HCV-endemic region of the world or blood-product transfusion. From my years of clinical experience, I believe that the great majority of patients will not deny their exposure to a known risk factor during the taking of their medical history once the particular risk factor has been brought to their attention during the course of the interview. Essentially all patients who have had a transfusion or taken blood product in the past, and who can remember having done so, will be identified on an initial history-taking by a medical specialist.

42. Most of the patients I see in my clinical work belong to the non-transfused general HCV population making up almost 99% of the HCV-infected persons in Canada rather than to the roughly 1% belonging to the transfused group eligible for compensation under the Settlement. A very large proportion of the patients from the general HCV population have some history of IVDU or are immigrants or refugees from high- or medium-endemic countries such as Egypt (12% HCV prevalence) or Pakistan (3-5% HCV prevalence). Many of my IVDU patients were asymptomatic when diagnosed and were shocked to learn that they had been carrying a hepatitis virus for decades after a brief experiment with injection-drug taking.

43. In my clinics I have found that my HCV-infected patients belonging to the transfused group are much more likely to be aware of their HCV infection than are members of the non-transfused general HCV population. The IVDU and immigrant groups of patients frequently are unfamiliar with HCV epidemiology and transmission risks. On the other hand, my clinical experience has taught me that a considerable percentage of patients who previously have had a transfusion cannot recall its occurrence with certainty when asked about blood transfusions during their intake examination. Patients having a history of treatment for motor-vehicle accidents, bleeding, shock or trauma, may have been subject to conditions involving altered sensorium, even coma. Although they usually will recall hospitalizations for serious injuries, their personal recollection of past treatment details may be fragmentary. If patient medical records for previous

hospitalizations are available for review, such records will be more reliable than the patient's personal recollection.

44. At my clinics HCV-positive patients who report having received blood transfusions prior to 1993 are highly likely to be reminded that compensation may be available under this Settlement or another one. Having practised my specialty for many years, I am well aware of the concerns that led to the Commission of Inquiry on the Blood System in Canada and later to the Agreement whose administration now is being supervised by the courts. Moreover, my clinical staff is experienced, reliable and equally aware of the Settlement under which persons infected with HCV through contaminated blood taken between 1986 and 1990 can obtain financial compensation.

45. In the time I spend with each patient at my clinic, my first priority certainly would be to discuss with them the diagnosis, prognosis and management plan for their disease rather than to raise their potential eligibility for benefits available under the Settlement. Where the patient's history raises the possibility of HCV-contaminated blood having been taken, my usual practice would be to ask whether the patient is aware of the Settlement and to suggest that the patient obtain more information from my clinic staff members if he or she wishes to pursue a claim. I am confident that any of my patients making such an inquiry of my staff will receive the information required to initiate a claim under the Settlement.

46. I cannot offer precise data concerning the number of patients with whom I raised the possibility of Settlement benefits or the number who already were aware of the Settlement when I raised it with them or whether fewer patients today are familiar with the Settlement than in previous years. My general impression is that a majority of my patients were not aware of the Settlement at the time when I informed them of their possible eligibility to claim benefits.

f) HCV natural history from infection to cirrhosis requires decades

47. Infection with HCV is an extremely slow disease process. The disease also can remain undetected for many years. Patients who have been referred to me as being RNA+ on their PCR test often are asymptomatic when they present. Patients who exhibit mildly cirrhotic livers on fibroscan testing often report no symptoms despite the discernible scarring of their liver that the disease process already has caused. The rate of fibrosis progression toward cirrhosis and late-stage liver disease is influenced by risk factors such as gender, age, alcohol consumption, obesity and co-infection with hepatitis B virus or human immunodeficiency virus. Patients who are symptomatic usually have carried the virus for many years and are at or approaching late-stage liver disease.

48. HCV infects liver cells causing them to become inflamed, scarred and eventually to die. The spreading patterns of scarring on an infected liver enable clinicians to assess the stage in the natural history of an HCV infection to which the disease has progressed as it advances toward cirrhosis and liver failure. The usual method of breaking out liver fibrosis into stages of development is the so-

called Metavir scale consisting of five levels or grades running from F0 through F4.

49. The Metavir scale begins from F0, where the liver exhibits no fibrosis at all. At stage F1 the liver shows some scarring in its portal areas. By stage F2 the fibrosis has begun to extend beyond the portal areas. Stage F3 is identified by the condition described as bridging fibrosis. At this stage the scarring extends from one or more of the portal triads through which blood enters each liver lobule all the way to the central vein from which blood passes out of that liver lobule, and/or to another portal triad. At stage F4 the fibrosis has grown so extensive that it involves all the portal triads and veins within the liver lobule compromising the flow of blood through the liver. This stage is also known as cirrhosis.

50. Under Dr. Krahn's direction, the CASL *ad hoc* committee known as MMWG created a medical model reflecting the probable rates at which members of the Settlement cohort would progress from one disease stage to the next. The MMWG followed the fibrosis-involvement staging of the Metavir scale through the natural history of HCV infection. In adapting the MMWG disease-progression model for use as a gradient of compensable injury, the parties to the Settlement slightly recalibrated the Metavir model to incorporate six levels of disease progression at which qualified claimants would receive cumulative lump-sum compensation. MMWG/Metavir grade F0 is equivalent to Settlement Plan compensation Level 1, where the claimant is RNA- on an HCV-antibody test, and also Level 2, where the claimant is RNA+ on a PCR test. Metavir stages F1 and F2, which are characterized by non-bridging fibrosis, are roughly equivalent to

Settlement Plan compensation Level 3. Metavir stage F3, which is characterized by progression to bridging fibrosis, is equivalent to Settlement Plan compensation Level 4. Metavir stage F4, which is characterized by progression to cirrhosis, is equivalent to Settlement Plan compensation Level 5. In addition to the MMWG/Metavir stages, the Settlement Plan includes compensation Level 6, which addresses claims arising from hepatocellular cancer, decompensated cirrhosis, and liver transplant.

51. The transition from one fibrosis stage to the next on the Metavir scale adopted by MMWG can be an extremely slow process. Subject to risk factors such as alcohol use and co-morbidities, studies have shown that the progress from Metavir grade F1 to grade F2 may take as long as seven to fifteen years.

52. As stated at page 8 of its Fourth Revision in April 2011, MMWG has refined its model on several occasions in a continuing effort to assist in the process of assuring the sufficiency of the Trust. MMWG reviews the history of these refinements in the opening section of its Fourth Revision which is entitled "Background". MMWG reports that as increasing amounts of actual data from the Settlement administration have been made available for use in the medical model, greater weight has been given to Settlement claims data within a blended pool of information derived largely from results reported in world medical literature.

53. With the Fourth Revision, MMWG has grown quite confident that the administration of the Settlement itself has yielded a useful body of claims data for

estimating stage-transition rates. At subsection 5.5 of its Fourth Revision, MMWG writes: "Given the fact that we know the approximate time at which HCV infection was acquired and have estimated the stage distribution of the claim, it is possible to use data from the [post-transfusion claimant cohort] to estimate transition rates between fibrosis stages. We used adjusted stage distribution data from the non-hemophilic patients without HIV infection and who received first blood transfusion between 1986 and 1990 to derive these rates."

54. Thus, MMWG reports at page 70 of its Fourth Revision that the disease-stage transition rates derived exclusively from the post-transfusion claimant cohort present a somewhat longer natural history of HCV infection than MMWG had posited in earlier, literature-based revisions of its disease-progression model: "Compared to literature-derived rates, [the post-transfusion claimant cohort]-derived rates are much lower for F0 → F1 and F3 → F4, but higher for F1 → F2 ([as encapsulated in] Table 4.2.5 [at page 112]). In addition, the estimated number of years (**60 years**=1/0.029+1/0.118+1/0.137+1/0.103) required to progress from infection (F0) to cirrhosis are somewhat longer than our previous estimates **41.5 years** in 2004 and **55.5 years** in 2007) and the **30 years** (4/0.133) reported by Poynard *et al.*".

55. MMWG's reference to 60 years for progression from infection to cirrhosis is based on the actual class data in 2010. Nonetheless, the stage-transition rates that MMWG finally develop for adoption in their Fourth Revision combine world medical-literature-derived rates and the actual class transitions to derive an average time from infection to cirrhosis of 39.5 years.

56. Based on my experience in the field, counsel have asked me to comment on the reasonableness of the MMWG estimates for the average time required for the natural history of the disease to run its course from infection to cirrhosis. They have asked whether the average time for HCV disease to progress from infection to compensated and decompensated cirrhosis in my patients has changed over the last thirty years.

57. In my opinion, the average time to progress from infection to cirrhosis has not changed in my practice over the past thirty years. As for the MMWG estimates of a mean of approximately 40 years to progress from infection to cirrhosis, I believe these estimates are reasonable. For several reasons, the Transfused cohort shows apparently slower progression rates than those pooled from the medical literature. I speculate that such reasons include the following factors: 1) a subset of the initial transfused cohort comprising the sickest and most critically-ill who might have had faster progression to cirrhosis, had died before making a claim in 1999 or later. 2) A significant number of the 4000 claimants have had their HCV cured by antiviral therapy in the past 2 decades, and thus have had no further progression of their liver disease.

58. Broadly, I agree with most of the MMWG Krahn report dated April 2011. However, one section (page 89) with which I disagree reflects the tremendous increment in antiviral cure rates that occurred from 2011-2014, when the introduction of new DAA treatments dramatically augmented the SVR cure rates from approximately 50% to more than 90%. Thus the specific statements with which I disagree estimate that 35% of the non-hemophilic patients alive in 2010

will develop cirrhosis and 20% will die of liver disease. Further, the report estimates that corresponding rates for hemophiliacs are much higher, at 52% and 36%, respectively. Because we can now cure the vast majority, more than 90% of HCV-infected patients, regardless of age, degree of liver fibrosis, and HIV-coinfected status, I believe it is very unlikely that such relatively large percentages of patients alive in 2010 will progress to cirrhosis or liver death.

g) Awareness of disease by Level 5 and Level 6 patients

59. Counsel also have asked me to identify the disease stage when all or almost all HCV-infected persons would experience disease symptoms that would make them aware of their need for medical attention. In my experience, from two-thirds to three-quarters of patients at the cirrhotic stage of HCV infection likely have sought medical attention and been diagnosed.

60. Cirrhosis is a spectrum of disease. It ranges from mild, asymptomatic cases with full physical and mental function to a syndrome called end-stage liver failure ("ESLF") with severe symptoms and poor mental and physical function. In my opinion, 95% of patients suffering from advanced cirrhosis likely have been diagnosed. These patients have reached ESLF or are nearing that stage and usually have less than a year to live without a liver transplant.

61. In considering the disease-stage distribution within my own practice, I estimate that about 50% of my patients are at Metavir stages F0 to F1; that another 20% have advanced to stage F2; that some 15% have reached stage F3; and that the remaining 15% are at cirrhosis and beyond. Most of my patients

do not belong to the transfused-blood cohort covered by the Settlement but to the non-transfused general HCV population. Keeping that distinction in mind, I estimate the distribution pattern in terms of the Settlement compensation levels to be roughly 50% at levels 1, 2 and early-stage 3, another 20% at level 3, another 15% at level 4, and the final 15% at levels 5 and 6 which include cirrhosis, hepatocellular cancer, decompensated cirrhosis, and liver transplant.

62. Counsel have asked me to distinguish between diagnoses of compensated cirrhosis, where liver function continues despite extensive scarring, and decompensated cirrhosis where liver function is gravely impaired. Under the Settlement, claimants with compensated cirrhosis are rated at Level 5 while those suffering from decompensated cirrhosis are rated at Level 6. In my opinion 70-85% of persons having Level 5 HCV-derived disease will have presented as patients and been diagnosed, 90-95% of persons having Level 6 HCV-derived disease will have presented as patients and been diagnosed, and 99% of persons at ESLF will have presented as patients and been diagnosed.

AFFIRMED before me at the City of Calgary in the Province of Alberta on this 26th day of January, 2016.

Commissioner for Taking Affidavits and Notary Public in the Province of Alberta

Amarinder Virk
Notary Public
Barrister & Solicitor
Signature Attested. No Legal Advice Given.

SAMUEL S. LEE

This is Exhibit "A" referred to in the
affidavit of Samuel S. Lee
sworn before me at Calgary, AB
this 26th day of January, 2016

A Commissioner for taking Affidavits and
Notary Public in the Province of Alberta

Amarinder Virk
Notary Public
Barrister & Solicitor
Signature Attested. No Legal Advice Given.

CURRICULUM VITAE

Current to 1/1/2016

I. BIOGRAPHICAL DATA:

Name: Samuel S. Lee
Address: 3330 Hospital Dr NW
Calgary, AB, T2N 4N1
Canada
Telephone: (403) 220-8457
Fax: (403) 270-0995
Email: samlee@ucalgary.ca

Present Rank: Professor
Department: Medicine
Faculty: Medicine
Institution: University of Calgary

II. ACADEMIC RECORD

Final Degree: M.D.
Date Completed: 1978
Institution/City/Country Memorial University of Newfoundland, St. John's, NL, Canada

I. **Undergraduate:** Memorial University, 1972-74, premedical studies

ii. **Graduate and postdoctoral:**

Internship and Residency:

Internship (RI) Medicine, Sunnybrook Hospital, University of Toronto, 1978 - 79
RII medicine, Sunnybrook Hospital, 1979 - 80.
RIII medicine, Wellesley Hospital, Toronto 1980 - 81.
RIV gastroenterology, Toronto Western Hospital 1981 - 82.
RV gastroenterology, Toronto General Hospital 1982 - 83.

Research Fellowship:
Research fellow, Hepatic Hemodynamics Lab, Unité de Recherches de Physiopathologie Hépatique (INSERM U 481), Hospital Beaujon, Clichy, France, January 1984 - August 1987 (supported by Canadian Liver Foundation and Medical Research Council).

Visiting lecturer, Department of Pharmacology, Technion Faculty of Medicine, Haifa, Israel, September 1987 - December 1987.

iii. **Licensure and boards:**

LMCC 1980
ABIM (Internal Medicine) 1981
FRCPC (Internal Medicine) 1982
ABIM (Gastroenterology) 1983
Alberta College of Physicians & Surgeons, 1988-present: licence # 9548

III AWARDS AND DISTINCTIONS:

Newfoundland Government Centenary Scholarships, 1971,1972 and 1973
Memorial University Mathematics Competition, Second Prize, 1973
Faculty of Medicine Lange Book Prize, 1977
Faculty of Medicine Lange Book Prize, 1978
Canadian Liver Foundation Fellowship, 1984 - 1985
Medical Research Council Fellowship, 1986 - 1987
Royal College of Physicians and Surgeons of Canada H.K. Detweiler Travel Award, 1986
Faculty of Medicine "Gold Star" teaching awards, 1994,1998, 1999, 2000, 2003, 2004
CMSA Letter of Excellence for teaching, 2002, 2005, 2006
Encana Alberta Stars of Distinction award (Science category), 2002
Queen Elizabeth II Golden Jubilee Medal, 2002
Best Clinical Teacher Award, voted by fellows and trainees in GI division, 2003
Mizne Lectureship in Gastroenterology, Jewish General Hospital, McGill University, 2003
Graduate Students Society Golden Apple Teaching Award, 2003, 2006
Canadian Liver Foundation Tribute Gala 'Roast', Oct 26, 2007
St Louis University International Visiting Speaker, SLU Division of Gastroenterology, St Louis, USA, Dec 2011

IV ACADEMIC APPOINTMENTS:

- Active staff, Internal Medicine, Foothills Hospital, Calgary, Alberta, 1988 - present.
- Courtesy Consulting staff, Department of Medicine, Peter Lougheed Hospital and Rockyview Hospital, Calgary 1988 - present.
- Alberta Heritage Foundation for Medical Research, Clinical Investigator, 1988 - 1994, AHFMR Scholar, 1995 - 2000, Senior Scholar 2000 - 2006.
- Assistant Professor, Department of Medicine (Gastroenterology), University of Calgary, Calgary, Alberta, 1988 - 1993.
- Associate Professor, Department of Medicine, University of Calgary, 1993 - 2000.
- Professor, Department of Medicine, University of Calgary, 2000 - present

V EDUCATIONAL ACTIVITIES:

Coordinator, GI course (432), 2nd year curriculum (2000-03)
Supervisor for MSc studies: J.M. Pak, 1994; D.N. Jaue, 1996; J. Wong, 1997; T. Glenn, 2008; N. Alhassan (leave of absence to finish surgical residency; expected 2017)
Supervisor for Ph.D. studies: Z. Ma, 1996; D. Song, 2003; S.A. Gaskari 2009.

External examiner: M. Belanger, PhD, Univ Montreal 2006; K. Moncrief, MSc, Univ Alberta 2006; Venessa Petullo, PhD, Univ New South Wales, Australia, 2013

VI ADMINISTRATIVE RESPONSIBILITIES:

Chairman, University of Calgary Gastroenterology Research Group, 1997-99
Member, GI fellowship program committee, 1993-2005
Director of Research, GI Division, 2004-2007
Dept of Medicine representative, UCMG Management Committee, 2006-08
GI divisional representative, Alternate Reimbursement Plan (ARP) Committee, 2006-11
Head, Division of Gastroenterology, 11/2007 – 3/2009
Member, Faculty of Medicine Academic Review Committee (promotions cte) 2013- present

VII PROFESSIONAL ACTIVITIES:

Canadian Association for Study of Liver, President, 1998-2000
Canadian Liver Foundation, member, Summer Studentship Committees
Canadian Association of Gastroenterology
Canadian Society for Clinical Investigation
American Association for Study of Liver Disease; member, Ethics committee 2003-06, and abstract review committee (Experimental Portal Hypertension) 2005-08
European Association for Study of Liver
Health Canada Hepatitis C Initiative, Program Advisory Group (steering committee)
World Congress of Gastroenterology, Montreal-2005, member scientific program organizing committee, and chairman, hepatology organizing committee
International Ascites Club; member, Scientific Executive Committee, 2004-07, Chairman and Secretary-Treasurer, 2007-2010
International Association for Study of Liver, Councillor, 2012 – 2016; President, 1/2016-1/2018

ii. Reviewer for:

Granting and scientific agencies:
Medical Research Council of Canada (member, Experimental Medicine committee, 1997-00)
Heart & Stroke Foundation
Canadian Institutes of Health Research
Canadian Liver Foundation
Ontario PSI Foundation
Bayer Blood Services Research Foundation
Manitoba Health Science Centre Foundation
Saskatchewan Health Research Foundation
Israel Science Foundation
Wellcome Trust
US Food and Drug Agency (invited reviewer for lamivudine NDA meeting 1998)
US Veterans Affairs Department

Journals:

Am J Cardiol, Am J Gastroenterol, Am J Pathol, Am J Physiol, Brit J Pharmacol, Brit Med J, Can J Cardiol, Can J Gastroenterol (editorial board 2004-2009), Can J Physiol Pharmacol, Chest, Clin Invest Med, Clin Sci, Digestion, Digest Dis Sci, Digest Liver Dis, Eur J Clin Invest, Eur J Gastro Hepatol, Gastroenterology, Gut, Hepatology, Hepatol Int (editorial board 2007-present) Hepatogastroenterology, J Clin Invest, J Gastroenterol Hepatol, J Hepatol, J Ped Gastro Nutr, J Physiol (Lond), J Viral Hep, Kidney Int, Kor J Hepatol (editorial board 2010-present), Kor J Int Med (editorial board 2008-present), Life Sci, Liver Transpl, Microcirculation, Ped Res, World J Gastroenterol (editorial board 2004-present)
-Editor-in-chief of *Liver International*, 01/2008 – 01/2013

Consultant for (past 5 years):
Abbott, Boehringer-Ingelheim, Bristol Myers Squibb, CV Therapeutics, Genentech, Gilead, Healix, Human Genome Sciences, Idenix, Janssen, Microgenix, Merck, Novartis, Innervision, Pharmasset, Roche, Sequana, Transition Therapeutics, Vertex

VII RESEARCH SUPPORT:

AHFMR Establishment grant: "Pathophysiology of Portal Hypertension", 1988-90, $72,500/yr. "Top-up" award 1997, $12,000.

CLF Supplementary establishment grant: "Mesenteric Veins in Cirrhosis - Human Study", 1990-92, $10,000/yr
Bridging operating grant: "Hyperkinetic circulation in cirrhosis", 1993-94, $60,000/yr.
Operating grant: "Mechanisms of negative inotropic effects of IFN", 2005-07, $60,000/yr
Operating grant: "Myosin heavy chain isoforms in cirrhotic cardiomyopathy", 2011-13, $60,000/yr

MRC/ Operating grant: "Spanchnic capacitance in cirrhosis", 1990-93, $56,000/yr.
CIHR Operating grant: "Cardiac membranes in cirrhosis", 1994-97, $52,000/yr.
Operating grant: "Membrane mechanisms of cirrhotic cardiomyopathy, 1997-2002, $63,000/yr.
Operating grant: "Cirrhotic cardiomyopathy: causes and consequences" 2002-07, $112,000/yr.
Operating grant (co-PI with R.J. Hilsden): "Treatment of active injection drug users infected with HCV with pegylated interferon, 2003-07, $134,000/yr.

Pharmaceutical industry (Amgen Canada, Cytel Corp., Agouron, Gilead, GlaxoSmithkline, Schering-Plough, Serono, Hoffman-LaRoche, Idenix, Leo, Bristol Myers Squibb, Wyeth Ayerst, Genentech, Johnson&Johnson, Intermune), Phase II and III clinical studies of antiviral agents in chronic viral hepatitis, ascites, hepatocellular carcinoma, autoimmune hepatitis and primary biliary cirrhosis 1992-present, (variable funding per study).

IX INVITED NATIONAL AND INTERNATIONAL ADDRESSES PAST 20 YEARS:

1. Japanese Association of Hepatology annual congress, Fukuoka, Japan, June 1995: "Mechanisms of cirrhotic cardiomyopathy"

2. Sapporo Medical College, Sapporo, Japan - March 1996: "Review of cirrhotic cardiomyopathy"

3. Kagawa Medical School, Kagawa, Japan - March 1996: "Cirrhotic cardiomyopathy"

4. Kurume Medical University, Kurume, Japan - March 1996: "Cirrhotic cardiomyopathy mechanisms"

5. 3rd International Korean Scientists Triennial Conference, Seoul, Korea - June 1996: "Pathogenic mechanisms of cirrhotic cardiomyopathy"

6. Chinese University of Hong Kong, Hong Kong - July 1996: 1) "Mechanisms of cirrhotic cardiomyopathy" 2) "Viral hepatitis: the Canadian Perspective'"

7. National Yang-Min University, Taipei, Taiwan - July 1996: "Cirrhotic cardiomyopathy"

8. Canadian Digestive Disease Week annual congress, Banff - March 1997: "Cardiovascular effects of bile salts" and "Hepatitis C treatment"

8. Japanese Portal Hypertension Society Annual Meeting, Fukuoka, Japan - September 1997: "Update on cirrhotic cardiomyopathy" (Keynote address)

9. First Canadian Association for Study of Liver (CASL) Single Topic Conference: "Hepatocellular carcinoma", co-organizer and cochair of congress, Niagara-on-the-Lake, ON, June 1998.

10. CASL Special Topic Conference: Natural History of chronic HCV, an expert panel consensus. Co-organizer, cochair and panelist. Toronto, ON, Sept 1998.

11. Association Française de l'étude du Foie (AFEF) Annual Congress, Portal Hypertension Symposium, Paris, France - October 1998: "Mechanisms of cirrhotic cardiomyopathy" (Keynote address)

12. Laboratory Centre for Disease Control 2nd Public Health symposium on Hepatitis C, Ottawa - Oct 1998: "Treatment of HCV patients"

13. Sunnybrook Gut Club, Toronto - October 1998: "Update hepatitis C"

14. MRC - Health Canada special conference, Defining an agenda for hepatitis C in Canada, Ottawa - Jan 1999: "Viral hepatitis network centres of excellence - where do we go from here"

15. Hepatitis B: Treatment Strategies for the New Millenium, conference chairman and principal organizer, Quebec City, QC, Jan 1999.

16. Hospital Medica Sur, 2nd Annual Liver Day (with teleconferencing to 12 other Mexican

hospitals), Mexico City - February 1999: "Treatment of hepatitis C with IFN and ribavirin"

17. New Brunswick Association of Gastroenterology Annual Meeting, Moncton - April, 1999: "Update viral hepatitis" and "Hepatic complications of IBD"

18. Canadian Association for Study of Liver Viral Hepatitis Consensus Conference, Montreal - March 1999: "Epidemiology of hepatitis B in Canada"

19. Vancouver Gut Club, Vancouver - June 1999: "Treatment of hepatitis B"

20. Gastro99 Pan-American Gastroenterology biannual meeting, Vancouver - Sept, 1999: "Cirrhotic cardiomyopathy"

21. Second annual CASL Single Topic Congress, "Hepatic Fibrosis", chairman and organizer of congress, Kananaskis, AB, Oct 1999.

22. American Association for Study of Liver Disease (AASLD) Annual Meeting, International Liver Transplant Society Pre-meeting Symposium, Dallas, Texas - November 1999: "Cirrhotic cardiomyopathy and liver transplantation", and co-moderator, workshop: "Are hyperdynamic conditions necessary for successful transplantation"

23. Fourth Annual Update Liver Disease and IBD, Cancun, Mexico - Feb 2000: panelist, interesting cases, and workshop moderator, "Management of borderline patients"

24. Pegylated Interferon (Pegasys) Investigators' meeting, Mar 2000, Athens, Greece: "Histological results of the Pegasys 95 study"

25. Management of hepatitis C at the turn of the new millenium, Acapulco, Mexico, Mar 2000, workshop moderator, "Patterns of response to treatment"

26. International Congress of Infectious Disease, 9th annual meeing, satellite symposium on "New approaches to the difficult HCV patient", Buenos Aires, Argentina, Apr 2000: "Pegylated interferon treatment of cirrhotic HCV patients".

27. Interscience Congress of Antimicrobial Agents and Chemotherapy (ICAAC) 40th annual meeting, symposium on "New directions in treating hepatitis C", Toronto, Sept 2000: "Quality of life in patients with chronic hepatitis C"

28. 5th Annual Update in Liver Disease and Inflammatory Bowel Disease, Tucson, AZ, Feb 2001: workshop leader, "Portal hypertension"

29. Canadian Digestive Disease Week, symposium organizer and chairman, "Management of hepatitis B: new strategies for an old disease", Banff, Alberta, Feb 2001

30. Mexican Association of Hepatology Annual meeting, Hepatitis symposium, "Role of liver biopsy in hepatitis C" and "Pegylated IFN therapy for chronic hepatitis C", Guadalajara, Mexico,

Oct 2001.

31. Correctional Service of Canada annual healthcare congress, "Management of hepatitis C", Ottawa, Feb 2002

32. Canadian Society of Hematology, hemophilia symposium, "Bleeding disorders in liver disease patients", Banff, Alberta, Mar 2002

33. 6th Annual Update in Liver and Inflammatory Bowel Disease, "Interesting Cases" organizer and chairman, Nassau, Bahamas, Mar 2002

34. Lebanese Society of Gastroenterology 1st Annual Congress, "La regulation centrale de la circulation sanguine au cours de l'hypertension portale", Beirut, Lebanon, Apr 2002

35. American Association for Study of Liver Disease, Symposium on Clinical Research in Portal Hypertension, luncheon workshop leader, "Heart in portal hypertension", San Francisco, May 2002

36. Canadian Association of Pathologists, 53rd Annual meeting, "Update in hepatitis C" Calgary, May 2002

37. European Association for Study of Liver monothematic conference on Vascular Mechanisms in Liver Disease, "Cirrhotic cardiomyopathy: definition and basic mechanisms", London, UK, July 2002

38. University of London tri-hospital (St. Mary's, St. Thomas, Chelsea-Westminister) Infectious Disease rounds, "Chronic hepatitis C: a Canadian perspective", London, UK, July 2002

39. Hepatitis C forum: Foundations for Cure, "Early predictability of pegylated IFN alfa-2a therapy in chronic hepatitis C", Rhodes, Greece, Sept 2002

40. Annual Western Canadian Update in Medicine for Psychiatrists course, "ABC's of viral hepatitis", Banff, Alberta, Sept 2002

41. GI Grand Rounds, University Alabama-Birmingham, "Cirrhotic cardiomyopathy: clinical and basic aspects" Birmingham, AL, USA, Nov 2002

42. Western Canadian Peginterferon alfa-2b Update, "Review of peginterferon HCV studies", Edmonton, AB, Dec 2002

43. 7th annual Update Liver and Inflammatory Bowel Disease, "Interesting cases" organizer and co-chairman, San Juan, Puerto Rico, Jan 2003

44. African Association for Study of Liver Disease 6th annual congress, "Liver biopsy in hepatitis C", "Peginterferon: early predictability and individualized treatment of hepatitis C patients", "Risks/Benefits of treating cirrhotic HCV patients" Cairo, Egypt, Mar 2003.

45. Canadian Viral Hepatitis Consensus Conference, "Treatment of HCV relapsers and nonresponders to previous antiviral therapy, and role of maintenance treatment", Ottawa, Nov 2003.

46. McGill University – Jewish General Hospital Mizne Lectureship Award, "The heart in cirrhosis", Montreal, Nov 2003.

47. Queen's University Division of Gastroenterology Rounds, "Cirrhotic cardiomyopathy: what it means to the clinician and researcher", Kingston, ON, Mar 2004

48. Australian Hepatitis C Symposium: Make the difference a cure, "Treating patients with HCV cirrhosis", and "Treatment of HCV genotypes 2 and 3", Sydney, Australia, Mar 2004

49. International Ascites Club EASL pre-meeting symposium on Cirrhotic Cardiomyopathy: "Evidence for cirrhotic cardiomyopathy: animal models", Berlin, Germany, Apr 2004

50. LatinoAmerican HCV symposium, "Treating the cirrhotic patient", "HCV: interesting cases" and breakfast workshop, La Romana, Dominican Republic, Jul 2004

51. Chosun University Hospital International Symposium, "Cardiovascular complications of cirrhosis", Kwangju, Korea, Sept 2004

52. Kyunghee University Hospital, Dept of Medicine Grand Rounds, "Cirrhotic cardiomyopathy", Seoul, Korea, Sept 2004

53. Mexican Society of Internal Medicine, annual congress, "Treatment of chronic HCV" and "Interesting hepatitis cases", Veracruz, Mexico, Nov 2004

54. Asian Pacific Association for Study of Liver (APASL) 14th annual meeting, "Cirrhotic cardiomyopathy", and "Animal models of portal hypertension", "Animal models of cardiovascular dysfunction in liver disease" New Delhi, India, Dec 2004

55. Canadian Association for Study of Liver (CASL) First Winter Meeting, "What is so good about being an academic hepatologist?"; symposium organizer and cochairman, "Treating chronic Hepatitis C: the new reality", Banff, AB, Mar 2005

56. AGA Clinical Symposium: "Cirrhosis: more than a liver disease", 56th Annual DDW meeting, "the heart in cirrhosis", Chicago, USA, May 2005

57. Ecos Internacionales, 17th annual meeting of Mexican Society of Gastroenterology, symposium on Treating Difficult HCV: "Treatment of HCV cirrhosis", and "HCV interesting cases", Puebla, Mexico, Aug 2005

58. Isfahan University Faculty of Medicine GI seminar series, "Management of hepatitis C", Isfahan, Iran, Sept 2005

59. Iranian Society of Physiology and Pharmacology, 17th biennial congress, "Mechanisms of cirrhotic cardiomyopathy" (one of 8 keynote addresses) Kerman, Iran, Oct, 2005

60. Tehran University of Medical Sciences, Faculty of Pharmacology Grand Rounds, "Cardiovascular complications of cirrhosis: new insights", Tehran, Iran, Oct 2005

61. Tehran University, Shariati Hospital GI Rounds, "Cirrhotic cardiomyopathy", and "Treatment of chronic hepatitis C", Tehran, Oct 2005

62. Memorial University of Newfoundland - St. Clare's Hospital Medical Grand Rounds, "Hepatitis C: 2005 update", St. John's, NL, Oct 2005

63. AASLD 56th Annual Meeting, "Presentation of AASLD Distinguished Achievement Award to Dr. Jenny Heathcote" (introduction of awardee), San Francisco, CA, Nov 2005

64. Peruvian Society of Gastroenterology, 25th annual congress, "Cardiovascular complications of liver disease", "cirrhotic cardiomyopathy", "treating the HCV patient with normal transaminases", "treatment of HCV cirrhosis", Lima, Peru, Nov 2005

65. Blood Ties Four Directions congress "HIV and HCV: Bringing Expertise to the North", "Hepatitis C: from Shakespeare to the 21st century" (keynote address), Whitehorse, Yukon, Feb, 2006

66. Whitehorse General Hospital, Grand Rounds, "Update Hepatitis C 2006", Whitehorse, YK, Feb, 2006

67. Chronic Liver Disease Foundation Speakers Bureau symposium, "Management of hepatocellular carcinoma", Las Vegas, NV, Feb 2006

68. CASL 2nd annual Winter Meeting, "Treating hepatitis C: the Canadian EAP experience", Toronto, ON, Apr 2006

69. Canadian EAP/RAP Investigators meeting, debate: "borderline-compensated patients with HCV cirrhosis should be treated" (con), Scottsdale, AZ, Apr 2006

70. University of Alberta, Dept of Physiology, "Cardiovascular abnormalities in portal hypertension: new paradigms", Edmonton, AB, Apr 2006

71. New treatment concepts of chronic hepatitis C, "Chronic HCV: early or late treatment?", "Tailoring treatment duration in chronic hepatitis C", Dubai, UAE, Jun 2006

72. 15th Annual Post-DDW Review Course, "Review of liver abstracts: nonviral", Lake Louise, AB, Jun 2006

73. International Association for Study of Liver (IASL) / African Association for Study of Liver

Disease (AfASLD) 2006 Congress, "Cirrhotic cardiomyopathy", "Pharmacokinetics of peginterferons" "Helicobacter in cirrhosis", Cairo, Egypt, Sept 2006

74. AASLD 57th annual meeting, breakfast workshop moderator "Experimental portal hypertension", Boston, MA, Oct 2006

75. 4th Canadian Consensus Meeting for Viral Hepatitis, "Natural history of chronic hepatitis B", Toronto, Jan 2007

76. University of British Columbia Dept of Medicine Grand Rounds, "New paradigms in HCV management", Vancouver, BC, Feb 2007

77. Asian-Pacific Association for Study of Liver (APASL) 17th annual meeting, "Optimizing Hepatitis C treatment duration", Kyoto, Japan, Mar 2007

78. Virginia Commonwealth University Division of Gastroenterology Grand rounds, "Cirrhotic cardiomyopathy", Richmond, VA, USA, Apr 2007

79. 2nd Symposium on Coagulopathy in Liver Disease, "Endothelial function in cirrhosis", Charlottesville, VA, Apr 2007

80. Korean Association for Study of Liver (KASL) annual meeting, "New paradigms in portal hypertension", "How to publish in English-language journals" (both keynote lectures) Busan, Korea, May 2007

81. PROPHESYS Investigators' Meeting, "Optimizing treatment outcomes in chronic hepatitis C", Oslo, Norway, June 2007

82. Barcelona Liver Unit 7th International Symposium on Treatment in Liver Disease, "Cardiac dysfunction in cirrhosis", Barcelona, Spain, Sept 2007

83. Iranian Gastroenterology Hepatology Society, 7th annual congress, "Optimizing antiviral treatment in hepatitis C", and "Treatment of hepatitis C in endstage renal failure", Tehran, Iran, Nov 2007

84. APASL 18th annual meeting: "Portal hypertension: new concepts", Seoul, Korea, Mar 2008

85. Japanese Gastroenterology Society annual congress, "Real world experience in treating chronic hepatitis C", Fukuoka, Japan, May 2008

86. Hong Kong International Liver Congress 2008: "Optimizing hepatitis C treatment", Hong Kong, Jun 2008

87. AASLD Single Topic Conference (Circulatory and renal complications of cirrhosis): "Cardiac dysfunction in cirrhosis", Atlanta, USA, Sept 2008

88. ALEH (Latin American Assoc Study of Liver) annual congress, "new concepts in portal hypertension", "Cirrhotic cardiomyopathy", Isla Margarita, Venezuela, Sept 2008

89. Japanese Portal Hypertension Society, 7th Annual Single Topic Congress, "Cardiovascular complications of portal hypertension: new paradigms in pathogenesis", Fukuoka, Japan, Nov 2008

90. 2nd Ditan International Symposium on Infectious Diseases, "Optimizing hepatitis C treatment duration", "Cardiovascular complications of endstage liver failure", Beijing, China, Nov 2008

91. Paris Hepatitis Congress 2009, "Treatment of hepatitis B with nucleos(t)ide analogues", Paris, France, Jan 2009

92. Intl Ascites Club (IAC) – Norwegian Society of Gastroenterology joint CME symposium: 'Complications of chronic liver failure' (co-organizer), "Circulatory changes in cirrhosis: new paradigms", Lillehamer, Norway, Feb 2009

93. APASL 19th annual meeting, "Cardiac complications of endstage liver disease", "How to write a clinical research paper", Hong Kong, Feb 2009

94. Colombian-Venezuelan Liver Society annual meeting, "Cirrhotic cardiomyopathy", "How to write and publish a paper", Bogota, Colombia, Mar 2009

95. EASL annual congress, pre-meeting IAC symposium, "Bacterial infections and ascites" (symposium organizer and chairman), Copenhagen, Denmark, April 2009

96. Asian-Pacific Digestive Week, "Cirrhotic cardiomyopathy: bench to bedside", Taipei Sept 2009

97. AASLD 60th annual meeting Postgraduate course on "Complications of endstage liver failure": "cirrhotic cardiomyopathy: clinical significance and outcomes", Boston, Oct, 2009

98. Iranian Society of Physiology and Pharmacology, 19th biennial congress, "Cardiovascular complications of liver failure", Tehran, Iran, Nov 2009

99. Iranian Society of Gastroenterology annual congress, "Management of Hepatitis C", Tehran, Nov 2009

100. Indian Society of Gastroenterology Congress: "The heart in cirrhosis: from ignorance to consequence", Kolkata, India, Dec 2009

101. Ontario Association of Gastroenterology winter meeting: "Update in hepatitis C: 2010", Collingwood, Ontario, Jan 2010

102. AASLD/EASL Single Topic Conference on Acute on Chronic Liver Failure, "Management of cardiovascular complications in CLF", Atlanta, Mar 2010

103. APASL 20th annual meeting, IAC Symposium, 'Renal dysfunction in liver failure' (symposium co-chair), "Treatment of hepatitis C: global data", Beijing, Mar 2010

104. Hospital Medica Sur / Instituto de Nutricion 2nd annual international congress, "Hepatopulmonary syndrome", "Cardiovascular complications post-liver transplantation", Mexico City, July 2010

105. Falk Symposium 174: Gut and Liver, "Cardiac dysfunction in cirrhosis", Beijing, Aug, 2010

106. First Shanghai International Symposium on Liver Diseases, "How to write a paper", Hangzhou, China, Sept 2010

107. Intl Association of Surgery, Gastroenterology, Oncology (IASGO) course on Liver Transplantation, "Cirrhotic cardiomyopathy: what it means to the transplant unit", Essen, Germany, Sept 2010

108. Beijing Youan Hospital, and Sino-Japanese Friendship Hospital, visiting lectures: "How to write a paper for English-language journals". Beijing, Nov 2010

109. 2nd International Congress of the Liver Cirrhosis Clinical Research Center, "Cardiovascular changes in cirrhosis: new paradigms" Seoul, Korea, Jan 2011

110. Yonsei University Severance Hospital GI division rounds, "How to write a paper", Severance Hospital, Seoul, Jan, 2011

111. 4th Paris Hepatitis Congress 2011, workshop moderator, "Using quantitative HBsAg and HBV-DNA in management of HBV", Paris, Jan 2011

112. EASL annual congress, pre-meeting symposium on 'the Heart in Liver Disease', 'Cirrhotic cardiomyopathy: pathogenic mechanisms', Berlin, Apr 2011

113. 4th International Coagulopathy in Liver Disease Meeting, "Endothelial dysfunction, coagulopathy and liver disease", London, UK, Sept 2011

114. AASLD 61st annual meeting, lunch workshop: "Cardiovascular assessment of the transplant patient" San Francisco, Nov 2011.

115. Beijing Youan Hospital, Special Symposium on 'How to do clinical research and write up the results' (course organizer and main lecturer). Beijing, Dec 2011

116. 5th Paris Hepatitis Congress 2012, workshop speaker, "Managing complications of DAA triple-therapy in HCV."; session chairman, 'Treating special populations of HCV patients', Paris, Jan 2012

117. Canadian Digestive Disease Week annual congress, "Management of HCC" workshop

leader, Montreal, Mar 2012

118. 22nd APASL annual congress, "Heart in cirrhosis: clinical consequences and pathogenic mechanisms", Taipei, Taiwan, Feb 2012

119. EASL Monothematic congress: Vascular Liver Disease. "Hepatic venous outflow obstruction due to cardiac and pericardial disease", Tallinn, Estonia, Jun 2012

120. International Ascites Club Special Consensus Development meeting, 'New definitions of AKI and HRS', "Cardiac dysfunction in cirrhosis: basic mechanisms and pathophysiology", Padova, Italy, Dec 2012

121. 6th Paris Hepatitis Congress 2013, "Optimizing therapy in HCV genotype 1 patients: management of side effects", Paris, Jan 2013

122. King Saud University Liver Unit congress, 'Building International Bridges', "The joys and pains of doing research: 3 decades of experience", "How to write a paper" (keynote lecture), "Cardiovascular complications of cirrhosis: why it matters", Riyadh, Saudia Arabia, Feb 2013

123. Canadian Digestive Disease Week (CDDW) annual congress, Portal Hypertension symposium, debate: Early TIPS vs rescue TIPS for treatment of variceal bleeding (pro debator); workshop leader, "Management of hepatorenal syndrome", Victoria, BC, Mar 2013

124. 23rd APASL annual congress, "HCV and renal disease", "Managing side effects of HCV antiviral therapy", "Dual therapy in the new DAA age: is there still a place?"; "Treating HCV-HIV coinfected patients"; workshop moderator, "Difficult HCV". Singapore, Jun 2013

125. Middle Eastern KOL HCV symposium: "HCV treatment with new DAAs", "HCV genotype 1: current management and real life data", Dubai, UAE, Oct 2013

126. 5th Coagulopathy of Liver Disease congress: chairman, session on Basic mechanisms of coagulopathy in cirrhosis. Padua, Italy, Sept 2013

127. LCCDC Biennial International congress: "New paradigms in cardiovascular disturbances of cirrhosis", Seoul, Korea, Oct 2013

128. Peking Union Medical College Hospital: "How to write and submit a paper for international journals", PUMC Hospital, Beijing, Oct 2013

129. Capital Medical University – Youan Hospital: 'How to do clinical research and write the papers' symposium organizer and lecturer. Beijing, Oct 2013

130. 63rd annual AASLD congress, Postgraduate Course: "Prophylaxis of variceal bleeding: screening and management", Washington DC, Nov 2013

131. 7th Paris Hepatitis Congress: chairman session on 'Hard-to-treat HCV', Paris, Jan 2014

132. CDDW annual congress, 'Management of HCV' Symposium: "Which patients should be treated now?", Toronto, Mar 2014

133. India speaking tour: "How to treat genotype 1 and 3 HCV in the DAA age", lectures delivered in Chandigahr, Kolkata, Hyderabad, Mumbai, New Delhi, Mar 2014

134. Digestive Disease Week, AASLD symposium on new advances in liver disease. "Coagulopathy and bleeding in cirrhosis: should we be worried?", Chicago, May 2014

135. Chinese Society of Hepatology – IASL co-sponsored symposium: Hepatology review for clinicians: "How to organize and conduct clinical research", "How to present at scientific meetings", Beijing, Sept 2014

136. United European Gastroenterology Week, "Antiviral treatment of the HCV cirrhotic patient", Vienna, Oct 2014

137. 5th Kolkata Liver Society congress: New Advances in Portal Hypertension, "New paradigms in cardiovascular abnormalities of cirrhosis: insights from animal models", "Extrahepatic complications of portal hypertension", Kolkata, Dec 2014

X PUBLICATIONS:

i Peer-reviewed Publications:

1. **Lee SS**, Girod C, Valla D, Geoffroy P, Lebrec D: Effects of pentobarbital sodium on the splanchnic hemodynamics of normal and portal hypertensive rats. *Am J Physiol* 1985; 249: G528 - 532.

2. Valla D, **Lee SS**, Moreau R, Hadengue A, Sayegh R, Lebrec D: Effets de la glypressine sur les circulations splanchnique et systemique des malades atteints de cirrhose. *Gastroenterol Clin Biol* 1985; 9: 877 - 880.

3. **Lee SS**, Braillon A, Girod C, Geoffroy P, Lebrec D: Haemodynamic rebound phenomena after abrupt cessation of popranolol therapy in portal hypertensive rats. *J Hepatol* 1986; 3: 38 - 41.

4. **Lee SS**, Girod C, Braillon A, Hadengue A, Lebrec D: Hemodynamic characterization of the chronic bile duct - ligated rat: effect of pentobarbital sodium. *Am J Physiol* 1986; 251: G176 - 180.

5. Braillon A, Capron-Chivrac D, Valla D, **Lee SS** Capron JP, Lebrec D: Domperidone-induced increase in lower esophageal sphincter pressure does not affect azygous blood flow in patients with cirrhosis. *Scand J Gastroenterol* 1986; 21: 1080 - 1082.

6. Braillon A, **Lee SS**, Girod C, Valla D, Peignoux-Martinot M, Lebrec D: The role of portasystemic shunts on the hyperkinetic circulation of the portal hypertensive rat. *J Lab Clin Med* 1986; 108: 543-548.

7. Valla D, Gaudin C, Geoffroy P, Braillon A, **Lee SS**, Lebrec D: Reversal of adrenaline-induced increase in azygous blood flow in patients with cirrhosis receiving popranolol. *J Hepatol* 1986; 4: 86 - 92.

8. Moreau R, **Lee SS**, Hadengue A, Braillon A, Lebrec D: Hemodynamic effects of clonidine-induced decrease in sympathetic tone in patients with cirrhosis. *Hepatology* 1987; 7: 149- 154.

9. **Lee SS**, Hadengue A, Girod C, Braillon A, Lebrec D: Reduction of intrahepatic vascular space in the pathogenesis of portal hypertension. In vitro and in vivo studes in the rat. *Gastroenterology* 1987; 93: 157 - 161.

10. Hadengue A, **Lee SS**, Moreau R, Braillon A, Lebrec D: Beneficial hemodynamic effects of ketanserin in patients with cirrhosis: evidence for serotonergic mechanisms in portal hypertension. *Hepatology* 1987; 7: 644 - 647.

11. Koshy A, Hadengue A, **Lee SS**, Jiron MI, Lebrec D: Possible deleterious hemodynamic effects of nifedipine on portal hypertension in patients with cirrhosis. *Clin Pharm Ther* 1987; 42: 295 - 298.

12. Hadengue A, **Lee SS**, Moreau R, Lebrec D: Content of oxygen and bile salts in the azygous venous blood: clues to the azygous derivation in patients with portal hypertension. *J Hepatol* 1987; 7: 98 - 101.

13. **Lee SS**, Hadengue A, Moreau R, Girod C, Jiron MI, Lebrec D: Naloxone does not alter haemodynamics in cirrhosis. Studies in humans and rats. *J Hepatol* 1987; 5: 149 - 153.

14. Cerini R, **Lee SS**, Hadengue A, Koshy A, Girod C, Lebrec D: Circulatory effects of somatostatin analogue in two conscious rat models of portal hypertension. *Gastroenterology* 1988; 94: 703 - 708.

15. Valla D, Girod C, **Lee SS**, Braillon A, Lebrec D: Lack of vasopressin action during bleeding. A study in conscious portal hypertensive rats. *Hepatology* 1988; 8: 10 - 15.

16. Hadengue A, Moreau R, **Lee SS**, Gaudin C, Rueff B, Lebrec D: Liver hypermetabolism during alcohol withdrawal: role of sympathetic overactivity. *Gastroenterology* 1988; 94: 1047 - 1052.

17. Jiron MI, **Lee SS**, Cerini R, Pugliese D, Hadengue A, Lebrec D: Effects of nitroglycerin on forearm hemodynamics in patients with cirrhosis. *Clin Sci* 1988; 74: 433 - 436.

18. Hadengue A, **Lee SS**, Koshy A, Girod C, Lebrec D: Regional blood flows by the microsphere method: validity in portal hypertensive rats and influence of a portal vein catheter. *Proc Soc Exp Biol Med* 1988; 187: 461 - 468.

19. Pugliese D, **Lee SS**, Koshy A, Cerini R, Ozier Y, Lebrec D: Hemodynamic effects of intravenous hypertonic glucose in patients with cirrhosis. *Hepatology* 1988; 8: 643 - 646.

20. **Lee SS**, Hadengue A, Moreau R, Sayegh R, Hillon P, Lebrec D: Postprandial hemodynamic responses in patient with cirrhosis. *Hepatology* 1988; 8: 647 - 651.

21. Braillon A, **Lee SS**, Valla D, Geoffroy P, Lebrec D: Comparative hemodynamic effects of betaxolol and popranolol in patients with cirrhosis. *Scand J Gastroenterol* 1988; 23:691-695.

22. Moreau R, **Lee SS**, Soupison T, Roche-Sicot J, Sicot C: Abnormal tissue oxygenatin in patients with cirrhosis and liver failure. *J Hepatol* 1988; 7: 98 - 105.

23. **Lee SS**, Moreau R, Hadengue A, Cerini R, Koshy A, Lebrec D: Glucagon selectively increases splanchnic blood flow in patients with well-compensated cirrhosis. *Hepatology* 1988; 8: 1501 - 1505.

24. Braillon A, Koshy A, **Lee SS**, Girod C, Lebrec D: Effet du developpement des shunts porta-systemiques dans le maintien de l'hypertension portale chez le rat. *Gastroenterol Clin Biol* 1988; 12: 641 - 645.

25. **Lee SS**: Alcohol infusion to measure hepatic blood flow: vanquishing the bete noire? *Hepatology* 1989; 10: 1021 - 1022 (commentary).

26. Hadengue A, Moreau R, Cerini R, Koshy A, **Lee SS**, Lebrec D: Combination of ketanserin and verapamil or popranolol in patients with alcoholic cirrhosis: search for an additive effect. *Hepatology* 1989; 9: 83 - 87.

27. Moreau R, **Lee SS**, Hadengue A, Ozier Y, Sicot C, Lebrec D: Relation between oxygen transport and consumption during vasoactive drug administration in patients with cirrhosis. *Hepatology* 1989; 9: 427 - 432.

28. Koshy A, Girod C, **Lee SS**, Hadengue A, Cerini R, Lebrec D: Discrepancy between systemic and splanchnic hemodynamic changes after incremental doses of popranolol in awake portal-hypertensive rats. *Hepatology* 1989; 9: 269 - 273.

29. Cerini R, Koshy A, Hadengue A, **Lee SS**, Garnier P, Lebrec D: Effects of glucagon on systemic and splanchnic circulation in conscious rats with biliary cirrhosis. *J Hepatol* 1989; 9: 69 - 74.

30. Cerini R, Braillon A, Hadengue A, Koshy A, **Lee SS**, Lebrec D: Somatostatin analogue improves survival in conscious cirrhotic rats subjected to gastrointestinal bleeding. *Clin Sci* 1989; 77: 7 - 9.

31. **Lee SS**: Cardiac abnormalities in liver cirrhosis. *West J Med* 1989; 151: 530 - 535.

32. **Lee SS**, Koshy A, Hadengue A, Lebrec D: Heterogeneous hepatic venous pressures in patients with liver cancer. *J Clin Gastroenterol* 1990; 12: 53 - 56.

33. MacColl C, MacCannell KL, Baylis B, **Lee SS**: Treatment of acute colonic pseudo-obstruction (Ogilvie's syndrome) with cisapride. *Gastroenterology* 1990; 98: 773 - 776.

34. Kong, K, Kelly JK, **Lee SS**: Pseudotumor appearance in chronic hepatitis. *J Clin Gastroenterol* 1990; 12: 437 - 440.

35. **Lee SS**, Marty J, Mantz J, Samain E, Braillon A, Lebrec D: Desensitization of myocardial beta-adrenergic receptors in cirrhotic rats. *Hepatology* 1990; 12: 481 - 485.

36. **Lee SS**, Hadengue A, Braillon A, Lebrec D: A pitfall in azygous vein cannulation in cirrhotic patients: mistaken cannulation of the mammary vein. *Angiology* 1990; 41: 942 - 945.

37. Johansen KH, Girod C, **Lee SS**, Lebrec D: Mesenteric venous stenosis reduces hyperammonemia in the portocaval shunted rat. *Eur Surg Res* 1990; 22: 170 - 174.

38. **Lee SS**, Hadengue A, Girod C, Braillon A, Lebrec D: Divergent circulatory effects of betaxolol in conscious and anesthetized normal and portal hypertensive rats. *J Hepatol* 1991; 12: 157 - 161.

39. Bomzon A, Jacob G, **Lee SS**, Meddings JB: In vitro vascular responsiveness to norepinephrine in experimental portal hypertension. *Clin Invest Med* 1991; 14: 63 - 71.

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41. Fraser KA, **Lee SS**: Autonomic regulation of splanchnic circulation. *Can J Gastroenterol* 1991; 5: 147 - 153.

42. Jacob G, Bishara B, **Lee SS**, Hilzenart N, Bomzon A: Cardiovascular responses to serotonin in experimental liver disease. *Hepatology* 1991; 14: 1235 - 1247.

43. **Lee SS**, Johansen K, Lebrec D: Circulatory changes induced by portal venous diversion and mesenteric hypertension in rats. *Hepatology* 1992; 15: 117 - 121.

44. **Lee SS**, Chilton EL, Pak JM: Adenosine receptor blockade reduces splanchnic hyperemia in cirrhotic rats. *Hepatology* 1992; 15: 1107 - 1111.

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Gastroenterol 1993; 7: 9 - 10 (editorial).

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56. Pak JM, Adeagbo ASO, Triggle CR, Shaffer EA, **Lee SS**: Mechanism of bile salt vasoactivity: dependence on calcium channels in vascular smooth muscle. *Br J Pharmacol* 1994; 112: 1209 - 1215.

57. Beck PL, **Lee SS**: Vitamin K1 improves survival in bile duct-ligated cirrhotic rats. *J Hepatol* 1995; 23: 235. (letter)

58. Sadowski D, **Lee SS**, Wanless IR, Kelly JK, Heathcote J: Progressive type of focal nodular hyperplasia characterized by multiple tumors and recurrence. *Hepatology* 1995; 21: 970 - 975.

59. Ma Z, Meddings JB, **Lee SS**. Cardiac plasma membrane physical properties and β-adrenergic receptor function are unaltered in portal-hypertensive rats. *Hepatology* 1995;22: 188 - 193.

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effects and mesenteric arterial receptor characteristics. *Hepatology* 1996; 23: 1174 - 1180.

63. Kanwar S, Kubes P, Tepperman BL, **Lee SS**. Nitric oxide synthase activity in portal-hypertensive and cirrhotic rats. *J Hepatol* 1996; 25: 85 - 89.

64. Jalink D, Urbanski SJ, **Lee SS**. Bilioenteric anastomosis reverses hyperkinetic circulation in bile duct-ligated cirrhotic rats. *J Hepatol* 1996; 25: 924 - 931.

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66. Wong J, Zhang Y, **Lee SS**. Effects of portocaval shunting on hyperdynamic circulation in bile duct-ligated cirrhotic rats. *J Hepatol* 1997; 26: 369 - 375.

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transfusion recipients, 1986-1990. *Can J Publ Health* 2003; 94: 130-134.

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ii **Non-Peer Reviewed Book Chapters, Reviews, Letters, Editorials**

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