Suit No. 33976

IN THE SUPREME COURT OF CANADA
(ON APPEAL FROM THE COURT OF APPEAL FOR MANITOBA)

BETWEEN:

HER MAJESTY THE QUEEN,

APPELLANT,

- and -

CLATO LUAL MABIOR

RESPONDENT.

RECORD OF THE APPELLANT
MANITOBA PROSECUTION SERVICE
Volume V Tabs 1 to 6

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RECORD OF THE APPELLANT: CLATO LUAL MABIOR

VOLUME 1:

VOLUME 2:

EVIDENCE

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VOLUME 5:

EXHIBITS

000117

File No. CR 07-01-27848

THE COURT OF QUEEN'S BENCH
WINNIPEG CENTRE

BETWEEN:

HER MAJESTY THE QUEEN,

Applicant,

- and -

CLATO LUAL MABIOR,

Respondent.

DR. SMITH'S MEDICAL REPORTS AND CURRICULUM VITAE

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THE COURT OF QUEEN'S BENCH
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DR. SMITH'S MEDICAL REPORTS AND CURRICULUM VITAE

000117

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MEDICAL EDUCATION

The London Hospital Medical School, University of London, 1962-1967

PROFESSIONAL DEGREES AND QUALIFICATIONS

M.B. B.S. London, 1967
L.R.C.P, M.R.C.S, 1967
Primary F.R.C.S. (Eng.), 1969
L.M.C.C., 1975
E.C.F.M.G., 1974
Fellow of the College of Family Physicians of Canada, 1995
A.C.L.S., 1998

PROFESSIONAL LICENSES

Medical Register, General Medical Council, U.K., 1968-1974; 2004-5
The College of Physicians and Surgeons of Manitoba, 1972-2004, 2005-6

HONORARY APPOINTMENTS

Honorary Research Fellowship at the Ian Charleson Clinic, 2005
Prosectorship, The Royal College of Surgeons (Eng.), 1969

HOSPITAL APPOINTMENTS

Misericordia Hospital
Active staff - 1980-1999
Medical Executive - 1994-1999
Secretary 1994-1995
President elect 1995-1996
President 1997-1999
Member of the Misericordia Foundation Board
Representative to the Winnipeg-Brandon Inter Hospitals Medical Services Committee

MEMBERSHIP OF PROFESSIONAL ASSOCIATIONS

College of Family Physicians of Canada
Member of Manitoba Chapter Executive 1990-1997
Secretary 1991-1992
Treasurer 1992-1994
Chair AIDS Education Committee, 1992-1988
President-Elect 1994-1995
President, Manitoba 1995-1996
Past President 1996-1997
Editor of the Newsletter
Member of the Awards Committee
Chair of constitution and by-laws committee
Chair of nominating committee
Member of the National Board, 1995-1997

Canadian Medical Association
Member of the Venereal Disease Committee, 1983-1985

Bay Area Physicians for Human Rights 1977-1984
American Association of Physicians for Human Rights now called Gay and Lesbian Medical Association 1983-2002
Toronto HIV Primary Care Physicians Group (founding member), 1988-1995.

UNIVERSITY APPOINTMENTS
Lecturer for the Department of Family Medicine at The University of Manitoba, 1993- 1994
Clinical Research Fellow, Department of Infectious Diseases Toronto General Hospital 1988-1989
Clinical Instructor for the Department of Family Medicine at The University of Manitoba, 1982-1987.

RESEARCH
"*Saccharomyces boulardii* treatment for persistent toxin A (-), toxin B (+) *Clostridium difficile* colitis in a patient in a patient with advanced HIV infection." P. Van Caeseele, J. R. M. Smith, S. Choudhri, M. J. Alfa, and G. K. M. Harding. Publication pending.

"CD8+ Cytotoxic T-Cells Are Target of HIV Infection *in vivo*." A. Balakrishnan, J. R. M. Smith, E. S. Rector, B. M. Sahai. Poster Session, 7th Conference on Retroviruses and Opportunistic Infections, San Francisco, U.S.A., February 2, 2000.

"Severe Cortisol Deficiency Related to High Dose Megestrol Acetate Therapy." S. H. Choudhri, E. Salamon, J. R. M. Smith." Poster Session, 7th Annual Canadian Conference on HIV/AIDS Research, Quebec City, April 30-May 3, 1998.

"Nature, Time, Course, and Dose Dependence of Zidovudine Related Side Effects: Results of the Multicentre Canadian Azidothymidine Trial. K. Gelman, J.S. Montaner, M. Fanning, J.R.M. Smith, J. Falutz, C.Tsoukas, J. Gills, G. Wells, M. O'Shaughnessy, M. Wainberg, et al. AIDS Vol. 3, No. 9 (September 1989). pp. 555- 561.

"Nature, Time, Course and Dose Dependency of Zidovudine Related Side Effects: Results from the Multicentre Azidothymidine Trial." J. Ruedy, J.S.G. Montaner, K. Gelman, M. Fanning, J.R.M. Smith, J.Falutz, C. Tsoukas, J. Gills, G. Wells, M. O'Shaughnessy, and M.Wainberg. 5th International Conference on AIDS, Montreal June 4-9, 1989.

"Hematological Effects on Zidovudine: Results of the Multicentre Canadian Azidothymidine Trial." J. Ruedy, J.S.G. Montaner, K. Gelman, M. Fanning, J.R.M. Smith, J. Falutz, C. Tsoukas, J. Gills, G. Wells, M. O'Shaughnessy, and M. Wainberg. AIDS in Southeast Asia and the Pacific Region, June 28-30, 1989, Hong Kong.

LECTURES ON AIDS (SELECTED)

"The Antinomies of AIDS 1979-2007," The faces of HIV/AIDS in Manitoba - Past, Present and Future, Winnipeg, November 2007

"Report on the 2nd IAS Conference on HIV Pathogenesis and Treatment," September, 2003
"Report on the 14th International AIDS Conference," August 2002
"Talk on Human Papillomavirus," Nine Circles Community Health Centre
"Update on the 8th Conference on Retroviruses and Opportunistic Infections"
"The Pharmacist's Role in HIV," Manitoba Pharmaceutical Association, 2000
"Update on the 7th Conference on Retroviruses and Opportunistic Infections," Manitoba HIV Interest Group, 2000
Medical Presenter at the Canadian Summit on Aboriginals and HIV, March 2000
" HIV in Your Practice: What Family Physicians Should Know," Annual Scientific Assembly, Manitoba College of Family Physicians, 2000.
Facilitator, Workshop for Physicians with HIV Patients, Annual Scientific Assembly, Manitoba College of Family Physicians, 1998.
"New Antiretrovirals and Treatment of Opportunistic Infections," Report from the 5th Conference on Retroviruses and Opportunistic Infections, HIV Journal Club, St. Boniface Hospital, 1998.
"HIV Sero-Conversion Syndrome: Detecting HIV in Your Practice," A workshop for Core Area Physicians, Manitoba College of Family Physicians, 1997.
"Caring for the HIV/AIDS Patient in the Community," Manitoba Pharmaceutical Association, 1997.
Report on the 4th Conference on Retroviruses and Opportunistic Infections, HIV Clinical Rounds, HSC 1997

"Introduction to AIDS," The National Working Group on HIV and Rehabilitation, Victoria, 1997
Report on the XI International AIDS Conference, AIDS Manitoba 1996
"AIDS Testing - A Family Physician's Strategy" (1990), "HIV care to CD4 200" (1992), "HIV Testing and Work-Up of the Newly Diagnosed" (1995-1996). Outreach education to Family Physicians of Manitoba (CCFP).
Talk to emergency medical officers, Health Sciences Centre, St. Boniface and Misericordia Hospitals.
Participating Speaker Grand Rounds, Health Sciences Centre, University of Manitoba.
Talks on AIDs to Manitoba Chapter CFPC Annual Scientific Assembly, Department of Family Practice, St. Boniface Hospital, The Staff of the Misericordia Hospital, Manitoba Association of Laboratory Technologists, Manitoba Health Organizations Annual Meeting, Manitoba Chapter of Canadian Hospital Infection Control Association, Manitoba Association of School Trustees.

OTHER LECTURES

"Are You Looking After Gay and Lesbian Patients?" College of Family Physicians of Canada, National Conference, Winnipeg, October 11, 2007 and 14th Annual Rural and Remote Medicine Conference, Winnipeg, April 21, 2006, Winnipeg (both co-presented with Shelly Smith)
"Parasitic Bowel Disease in homosexually active men," second year medical students, University of Manitoba.
Forum on Controversial Topics in Medicine for second and third year medical students University of Manitoba.
Presentation to U of M Family Medicine Residents on Gay/Lesbian Health, January 2001.

COMMUNITY WORK
CCFP Representative, Canadian Working Group on HIV Rehabilitation, 1999-
Member, Manitoba AIDS Implementation Advisory Committee, 1999-
Member, Minister's Advisory Committee on Infectious Diseases, 1997-
Member, Community Strategy Committee of the Urban Planning Partnership (a provincial government appointed Committee to address health care reform) 1995- 97
Member of the committee on bloodborne pathogens, College of Physicians and Surgeons of Manitoba, 1995-present
Member AIDS Manitoba - Medical and Nursing Committee
Member Manitoba Health and Wellness HIV Drug Review Committee, 1992.
Panelist in the Consensus Conference of HIV Therapies, Vancouver, 1992.
Member of the Manitoba Health and Wellness HIV Physician Network Committee, 1991-1992.
Member of The Manitoba Education Committee on AIDS, 1989-1992.
Delegate to The College of Family Physicians of Canada HIV Think Tank Conference on education for family physicians and family practice residents, June 1991.
Chair of Aids Awareness Days Committee 1986-1987.

Member of the Manitoba Provincial HIV Advisory Committee, Manitoba Medical Association Liaison, 1985-1986
Organizer of "Manitoba AIDS Forum", 1983-1984.

b

GENERAL PRACTICE EXPERIENCE

November 2007-present	Physician (part-time), Four Rivers Medical Clinic
November 1996-April 2001	Physician, Northern Medical Unit, University of Manitoba
December 1997- April 2008, (LOA 2004-05)	Village Clinic (Nine Circles Community Health Centre)
1992 –2001	Physician, Convalescent Home of Winnipeg
December 1989-October 1991	Physician - The Village Clinic
September 1979-December 1997	Fort Rouge Medical Clinic
January 1979 – March 1979	Locum Charleswood Medical Clinic
November 1978 – December 1978	Locum, Gimli, Manitoba
October 1978-November 1978	Locum, Neepawa, Manitoba
September 1973-September 1976	Principal, Shoal Lake, Manitoba
May 1972-September 1973	Assistant, Birtle, Manitoba
March 1972-April 1972	Locum, Neepawa, Manitoba
May 1971-March 1972	Various Locums in General Practice, Surgery and Orthopedics in London, U.K.

TRAINING

November 1988-Oct. 1989	Clinical Research Fellow to Dr. Mary Fanning, Toronto General Hospital.
October 1976-June 1978	Resident, Internal Medicine Program Health Sciences Centre, Winnipeg
December 1970-April 1971	Senior House Officer (General Surgery and Urology), St. Bartholemew's Hospital , Rochester U.K.
April 1970-November 1970	House Officer (Traumatology), The Birmingham Accident Hospital
October 1968-March 1970	Casualty Officer, London Hospital
April 1968-September 1968	Pediatric House Officer, London Hospital
October 1967-March 1968	Receiving Room Officer, London Hospital

PRECEPTORSHIPS ETC. (LAST 5 YEARS)

Sept-Dec., 2004	Observership, Ian Charleson Clinic, Royal Free Hospital, London., U.K.
April 28-May 2, 2004	Resistance Forum and Clinical Care Options for HIV Symposium, Miami, Florida
Nov 20-21, 2003	Wayne State U/Detroit Medical Center Genotyping Course, Detroit
April 13-15, 2002	Whistler HIV Update
Sept 10-12, 2001	Preceptorship, Spectrum Clinic, Vancouver
Sept 7-9, 2001	Western HIV/AIDS Update, Malahat, B.C.
August 23-25, 2001	ACOG Postgraduate Course, Colposcopy in the Management of Lower Genital Tract Diseases, Toronto
August 21-22, 2001	Preceptorship w. Dr. D Fletcher, Immunodeficiency Clinic, Toronto General Hosptal
April 16-21, 2001	Preceptorship in Anal Colposcopy w. Joel Palefsky, Mt Zion Hospital, San Francisco
March, 2001	Preceptorship, HIV Clinic, UCSF, San Francisco (Volberding et al)

CONFERENCES ATTENDED (LAST 5 YEARS)

Oct 7-9, 2005	HIVMA Meeting, San Francisco
Oct 8-9, 2004	British HIV Association Conference, London
Jan 31-Feb 1, 2004	Canadian National Forum on HIV and Rehabilitation, Toronto
Oct 3, 2003	HIV Management and Treatment Strategies Within the Canadian Correctional System, Toronto
July 13-16, 2003	2nd IAS Conference on HIV Pathogenesis and Treatment, Paris
Oct 24-26,2002	Gay and Lesbian Medical Association, Toronto
July 2002	International AIDS Conference, Barcelona, Spain

(Revised May 2006)

Report on HIV/AIDS

HIV causes AIDS

AIDS is an acronym for the Acquired Immunodeficiency Syndrome. It was first reported in the U.S. Centers for Disease Control and Prevention's Morbidity & Mortality Weekly Report on June 25 1981[1]

The Human Immunodeficiency Virus (HIV) was first isolated by Luc Montagner in Paris in May 1983. The same virus was also identified by Ernest Gallo and Jay Levy in the USA. A massive body of body of evidence subsequently confirmed beyond any doubt that this retrovirus (HIV) is the cause of AIDS. The attached document *The Evidence that HIV causes AIDS* published by the U S National Institutes of Health and updated in February 2003 documents this evidence in detail.[2]

Early causation theories and the beliefs of a small number of HIV denialists concerning the causes for AIDS (immune system overload, poverty, malnutrition, drugs or syphilis) have been convincingly disproved. HIV infection is now accepted as the precondition for development of AIDS not only in the scientific community but by all the national and international organizations addressing the AIDS pandemic. However conditions similar to AIDS can sometimes occur in patients who are immunosupressed and do not have HIV (e.g. immune system malignancies or medically induced immunosupression in organ transplant patients). Furthermore a tiny number of people with unexplained low CD4 counts are classified as having Idiopathic Lymphocytopenia (IL).

HIV is a member of the Retrovirus family. On entering the cell it is infecting, its own enzyme Reverse Transcriptase enables it to replicate its RNA genome in a DNA version of itself thus permitting it to enter the cells genome. Viruses copy their genetic material into the genetic material of the cell they have infected and remain there for the rest of the cell's life. Some of the cells HIV infect (immune system memory cells) are extremely long lived. This results in a lifelong chronic infection. Since the development of powerful antiretroviral agents HIV can be very well controlled but not, as yet, eradicated. Researchers have been looking for strategies to eradicate HIV and the possibility of a complete cure has not been ruled out.

HIV can infect many different cells in the body. HIV's most harmful effects result from its predilection for infecting a type of white blood cell called the CD4 lymphocyte (CD4 cell) and neurons (nerve cells). Infection of neurons is one of the mechanisms by which HIV can cause damage to the brain, spinal cord, or peripheral nervous system. CD4 lymphocytes are the co-coordinators of the immune system's response to infections. In untreated HIV infection the number of CD4 Lymphocytes progressively decline. A small number of individuals are unusually resistant to HIV for a variety of reasons and maintain high CD4 counts. They are known as "long term slow progressors". Others are able to control the amount of HIV in their blood to very low levels and are known as "elite suppressors". These people have a better prognosis when compared to other persons with HIV not receiving treatment.

NATURAL HISTORY OF HIV

The course of untreated HIV infection is well described.[3] The amount of HIV in the blood peaks at around 6 weeks and after it is brought under control by the body's immune system and falls by around 12 weeks after initial infection. This initial phase is the period of greatest infectivity. Unfortunately a large proportion of persons infected with HIV may have minor and transient symptoms of the initial infection and be unaware they have been infected and are now themselves highly infectious to others. After a variable period of time , symptoms of HIV (such as fever, night sweats, fatigue, diarrhea, or weight loss, thrush, multi-dermatomal shingles) or an AIDS-defining illness develop.[4] This period can be approximately predicted by the quantity of HIV in the blood – the Viral Load (VL) – and the extent of immune system damage – the CD4 cell count (CD4). The interval from HIV infection if untreated to the development of symptomatic HIV disease or AIDS averages about 8 years. During this late stage the viral load again increases but ill health and possibly other factors usually results in less transmission of HIV than in the initial phase of infection. If no treatment is given for either the AIDS defining illness or HIV infection the median survival time to death is 1.3 years.[5]

AIDS Classification

Untreated HIV infection almost invariably causes progressive immune system damage evidenced by, but not solely due to, a fall in the number of CD4 cells. Eventually the immune system is so damaged it is unable to suppress certain life-threatening infections and cancers or stop HIV from damaging the brain and causing Dementia. The development of one of these 15 Opportunistic Infections, 3 Tumours , an HIV Wasting Syndrome or HIV associated Dementia in an individual with documented HIV constitutes the current definition of AIDS.[6] In the USA for reasons of medical financial coverage the definition of AIDS includes a CD4 level <200 cells/mm3 . This is not the case in Europe or Canada.

Before the advent of highly effective antiretroviral therapy (HAART), which is able to suppress HIV to viral loads 'below the level of detection' (200, 40 or 50 copies per ml. of blood depending on the sensitivity of the test) a diagnosis of AIDS was considered akin to an inevitable death sentence. This is no longer the case.

The course of treated HIV disease

The molecular structure and biology of HIV have been defined by scientists with an unprecedented rapidity. Potential target points in HIV's life cycle have been used to design drugs to prevent HIV from reproducing itself or even entering the cell to infect it. There is now a large and highly effective armamentarium of potent antiretroviral agents. There are now at least 20 highly effective medications from 5 classes of drugs available to patients in Manitoba. The absence of cross resistant mutation patterns between different classes of drugs and even within the same classes means that patients now

usually have options of 3rd or 4th line regimens if necessary. A number of these agents cause minimal if any side-effects, or side effects that are manageable. There are several antiretroviral regimens now available that require only once daily dosing and small numbers of pills. This has entirely altered the outlook for persons infected with HIV. The arduous symptomatic side-effects of some of the early medications are rarely seen with newer medications. Demanding food and fluid restrictions, medication spacing requirements, and constant need for refrigeration of medication of earlier drugs are almost entirely a thing of the past. This has made it much easier for patients to adhere precisely to the prescribed regime of medications, not miss doses, suppress their virus for decades with the same regime and still have backup regimens available should their virus become resistant or unacceptable side-effects develop.

Guidelines for the best time to start HIV medications have evolved over time. It is currently recommended that patients start treatment when their CD4 has fallen to 350 or when they develop symptoms that can be attributed to HIV.

The advent of HIV genotyping assays has enabled physicians to select a combination of HIV medications to which the strains of virus in an individual's blood are susceptible. Transmission of HIV strains resistant to any of the HIV medications is a relatively rare occurrence in Manitoba and would be extremely unlikely from someone who had not themselves evidenced resistance to HIV medications and whose virus remained suppressed with their first antiretroviral regimen of medications. Baseline genotyping of a new patient's HIV is now a standard of care to enable selection of the optimum first regime. In my experience it is now extremely rare for HIV not to be fully suppressed within 4 weeks of starting HIV treatment.

Since the advent of HAART persons who are able to take their HIV medications as instructed (and not miss doses) rarely if ever develop mutant strains that are resistant to those medications. I am unable to remember a single instance of a patient in my practice doing so.

AIDS may be lethal or endanger life

In the past HIV was unquestionably a lethal condition. The situation is now much more nuanced. Advances in HIV treatment have dramatically improved life expectancy. For example one of the best attempts to address more recent mortality from HIV is based on the Danish HIV Cohort Study (which comprises all persons treated with HIV in Danish HIV clinics since January 1995).[7] It was estimated, using data from 1995 till 2005, that a 25 year old infected with HIV but not Hepatitis C treated in the21st century could expect an additional 35 years of life – living to the age of 60. While this is clearly a reduced life expectancy, a person infected today would probably have an even better prognosis for several reasons. This study includes all the patients infected before the availability of HAART in 1996 as well as those with predictors of lower survival such as advanced disease (AIDS diagnosis), low CD4 count, high viral load, poor adherence and poor response to treatment. Current standard of care for a person diagnosed with HIV in a timely fashion would dictate treatment before progression to a low CD4count or AIDS. Furthermore 3 of the regimens used by this study population (Abacavir containing triple nucleoside, efavirenz with ritonavir boosted lopinavir , and protease inhibitors without ritonavir

boosting) are known to be less effective combinations and are no longer used. Finally it appears that some at least of these patients had planned treatment interruptions which are now known to significantly impair prognosis. Thus changes in the standard of care during and since the period studied could be expected to provide an even better life expectancy than that reported. It is now believed that with the advances thus far achieved in HIV care many if not most persons infected with HIV who receive and are compliant with optimal care will die of a non-AIDS cause.

Furthermore there is now no HIV specific reason why appropriately managed HIV infected persons of either sex should not have their own children without recourse to artificial insemination or sperm washing. The risk of mother to child transmission of HIV is extremely low in women who have maintained virologic control for some time.

At present there is no doubt that HIV patients co-infected with Hepatitis C, or conditions making it difficult to comply with treatment who are unable to overcome them (some psychiatric conditions, addictions or fears of HIV status disclosure at home) do have significantly shortened life expectancy. There is also a small increase in non AIDS defining illnesses and cancers in persons with HIV that might shorten life. However some of these (e.g., ischemic heart disease or diabetes) may actually be detected earlier (because the persons with HIV are regularly monitored) permitting introduction of preventive strategies that others might not receive till later in the course of these illnesses - thus actually prolonging life.

HIV can be transmitted through unprotected Intercourse

Sexual transmission of HIV has been widely studied; male to female transmission has been well documented.[8] It is a major mode of transmission globally and is universally accepted as such in the scientific community and by national and international organizations. A large study in Uganda of couples where one partner was infected with HIV and the other was not (sero-discordant couples) showed a male to female transmission rate in that setting ranging from 1 per 2000 to 1 per 384 coital acts.[9] The authors concluded that this rate would be insufficient to sustain the epidemic and that in the early stage of infection there is a period of increased transmission which is brief but efficient. This was supported by a study showing a massive increase in HIV RNA in genital secretions and an 8-9 fold increase in the probability of HIV transmission from male to female at day 20 after infection compared with the 54 day.[10] Another study in Uganda found not a single instance of HIV transmission between sero-discordant couples where the HIV positive partner's viral load was less than 1500 copies per ml.[11] A longitudinal study of 393 sero-discordant heterosexual couples showed that in 14 years not a single case of transmission occurred over a 14 year period when the infected partner was treated with antiretrovirals compared with an 8.6% transmission rate in couples where no antiretrovirals were being taken.[12]

In addition to the viral load of the infected partner, other factors have been reported to affect the male to female transmission rate of HIV from men with unsuppressed plasma HIV viral loads, including oral

contraceptives, gonococcal cervicitis, candida vaginits, genital ulcers, bacterial vaginosis, herpes genitalis, Vitamin A definciency, CD4 count < 200. oral contraceptives.[13] [14]

A recent statement by the **Swiss Federal Commission for HIV/AIDS**[15] "authored by four of Switzerland's foremost HIV experts" is the first ever consensus statement to say that 'an HIV –infected person on antiretroviral therapy with a completely suppressed viremia ("effective ART") is not sexually infectious, i.e. cannot transmit HIV through sexual contact'

"It goes on to say that this statement is valid as long as:

- The person adheres to antiretroviral therapy, the effects of which must be monitored regularly by the treating physician, and
- The viral load has been suppressed (<40 copies/ml) for at least six months, and
- There are no other sexually transmitted diseases."

This statement by this authoritative body was made "after review of the medical literature and extensive discussion". The article cites three studies showing that effective antiretroviral therapy reduces HIV in sexual secretions to below the level of detection.[16] [17] [18] acknowledges that "medical and biologic data available today do not permit proof that HIV-infection during effective antiretroviral therapy is impossible, because the non-occurrence of an improbable event cannot be proven." The article states that the situation is analogous to 1986 when the statement "HIV cannot be transmitted by kissing" was publicized. This statement has not been proven, but after 20 years' experience its accuracy appears highly plausible." The article asserts that their statement about the relationship between treatment and sexual HIV transmission is much more informed than what was available in 1986 regarding transmission of HIV through kissing. The authors also make the point that there has not been a single case of transmission of HIV in the situation described above reported to date . They state that if there is any risk of HIV transmission in this situation it is less than I : 100,000 a level of risk considered acceptable in other situations such as flying in a plane. The Swiss Commission is careful to say that they only recommend that couples in a stable and durable relationship and meet the 3 criteria have unprotected sex and then only if that is the choice of the uninfected partner.[19]

This has been a controversial statement. Not because there is any convincing scientific refutation of the Swiss Commission's facts and risk assessment but because corroborating evidence and further study in an attempt to obtain further reassurance are felt necessary by some. The conditionality of the statement might not be understood; certainly alarm was caused by the fear that individuals not in a steady relationship with a partner (a partner who would also be committed to ensuring the HIV medications are taken without fail) might conclude it would be safe for them also to have unprotected sex with multiple partners without using a condom. Because sexually transmitted infections are known to markedly increase the amount of HIV in sexual secretions in HIV untreated individuals thus increasing the risk of transmission of HIV and because sexually transmitted infections present in the HIV-uninfected partner increase the susceptibility to HIV infection, it is highly advisable that persons even with an undetectable viral load who are having sex with more than one partner unfailingly and correctly use a condom.[20] [21] [22]

However Important studies quoted by the Swiss HIV Commission indicate that treating an STI results in a reduction in semen HIV RNA levels[23], and that the increase in seminal HIV caused by an STI is much less marked in someone with a suppressed viral load compared with someone with uncontrolled HIV viremia.[24] Another study done in Australia showed that seminal plasma HIV levels in patients on antiretrovirals who had asymptomatic STIs remained below the limits of detection of the test whereas there was a minimal increase in some of the men not on antiretrovirals.[25] In response to concerns about HIV DNA present in cells in the semen the authors note that this HIV lack markers for viral replicative capacity and that this indicates there is no transmission risk. Obviously if a patient stops his/her antiretrovirals long enough HIV in the blood will no longer be controlled and infectious HIV would appear in the genital secretions. There has been one case of transmission in this circumstance reported.

The Swiss Federal Commission for HIV/AIDS says that "when they evaluate the reprehensible character of an exposure to HIV, courts will have to take into account the fact that HIV-positive people not suffering from any other STI and following an effective antiretroviral treatment do not transmit HIV sexually."[26]

Even with a condom, HIV can be transmitted through sexual Intercourse.

HIV is unable to pass through good quality condoms.[27] "The proper use of the male or the female condom has been shown to reduce the risk of HIV transmission during vaginal intercourse. There is evidence of transmission due to condom failure, however, so receptive penile-vaginal intercourse with a condom is considered to be 'low risk', not 'no risk'. Condoms are not 100 percent reliable. It is difficult to define a condom failure rate because the information is dependent on the history of study participants. This issue is also a matter of controversy and researchers may bring their own biases. A Cochrane review of condom effectiveness concludes that consistent use of condoms results in an 80% reduction in HIV incidence. The studies used in this review did not report on the "correctness" of use.[28] Quality control of condom manufacture is now rigorous but is unable to prevent some defective condoms coming to market. There is enough evidence of transmission due to condom breakage or improper use to classify this activity as low (rather than negligible) risk"[29]

The correct use of the male condom requires that it is not used after the expiry date on the package and that the condom has not been subject to excessive heat or trauma (eg kept in the wallet or back pocket and sat on). Correct usage requires that the packet be opened carefully so as not to puncture the condom. The condom must be applied correctly to the penis with air expressed from the tip of the condom and the condom fully rolled down the shaft of the penis. It is essential to ensure that there is adequate vaginal lubrication with a water soluble lubricant unless there is sufficient physiologic lubricant. Care should be taken when withdrawing the penis and some educators recommend the base of the condom should be held during withdrawal to ensure the condom does not slip off the penis

spilling ejaculate into the vaginal cavity. If a condom comes off it should not be reused. Good health educators/ post test counselors will recommend partner referral for consideration of post exposure prophylaxis if there is condom failure. However in the absence of sexually transmitted infections in a person who has had an undetectable viral load for 6 months some experts would question the benefit of this.

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[1] CDC. Pneumocystis pneumonia – Los Angeles. MMWR 1981; 30: 250-2

[2] National Institute of Allergy and Infectious Diseases, "The Evidence That HIV Causes AIDS," http://www.niaid.nih.gov/factsheets/evidhiv.htm (updated Feb 27 2003).

[3] John G Bartlett & Joel Gallant, *Medical management of HIV Infection* (2003) 1.

[4] Ibid. 2.

[5] Ibid. 1.

[6] Ibid. 4.

[7] Nicolai Lohse, et al, "Survival of persons with and without HIV Infection in Denmark, 1995-2005," *Annals of Internal Medicine*," vol. 146. No. 3 (16 January 2007), 87-95.

[9] Gray RH et al Probability of HIV transmission per coital act in monogamous heterosexual, HIV1 discordant couples in Rakai Uganda. *Lancet.*(2001) vol 357: 1148 - 1153

[10] Pilcher CD et al Brief but efficient: acute HIV infection and the sexual transmission of HIV. *J Infect Dis.* 2004;189:1785-1792

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Review of Clato Mabior's medical and public health records

received and reviewed

1. Copy of Brandon Public Health Nurses' clinical records for period December 12 2003 toMarch30 2006 (there appears to be incompletion of the note on March 10 or 1 or more pages missing for the period between March 10 and March 29 2004 notes)
2. A letter to Dr Kasper from Darlene McDonald faxed Dec 2 2004
3. Documents relating to Clato Mabior's Social Assistance in Brandon application and coverage
4. Unsigned letter (? a copy of a letter sent or delivered) from Darlene McDonald to "Clato". no address on letter. Dated Nov 26 2004
5. Copy of letter to Dr Elise Weiss from Dr Carol Kurbis MOH WRHA (undated)
6. Copy of letter from Dr Elise Weiss MOH Brandon & Assinaboine RHAs
7. Copy of Manitoba Health STD control Contact information record on Clato Mabior relating to 10/22/04 HIV + GC- Chlamydia- Positive
8. Copy of positive Non-nominal Anti-HIV1/HIV2 serology result for code OR197801M15R7A drawn Dec 22 2003 for Dr Scott Blyth, Western medical Clinic, Brandon. Handwritten note '+ GC Clato Mabior'
9. Copy of HIV Notification Form for patient code OR197801M15R7A

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10. Copy of Clato Mabior's medical records from Western Medical Clinic Brandon with documents from Dec 19 2003 to Dec 23 2004
11. Letters from Dr K Kasper to Dr Blyth dated Dec 23 2004 and February 9 2005

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12. Copy of letter from Marcia Dzik to the Winnipeg Police Sex Crimes Unit

13. Copy of letter to Clato Mabior from Pierre Plourde, Medical Officer of Health, WRHA, December 30, 2005.

14. WRHA Health Records from the period June 22, 2005-February 6, 2006.

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15. Copy of the Health Sciences records from the period, December 23, 2004-February 8, 2006.

I reviewed the above listed copies of documents relating to Clato Mabior's health.

Was Clato Mabior's able to transmit infectious HIV? (Updated May 25 2008)

In my opinion Mr Mabior was already well past the initial 3 month phase of his HIV illness by the time was first diagnosed with gonococcal urethritis in Brandon on October 8 2003. This opinion is based on his CD4 count being 224 cells/mm3 on February 6 2004, 310 cells/mm3 on February 25 2004, and 361 cells /mm3 on April 9 2004. This reduction in CD4 count in the context of his low viral load levels indicates he had already had HIV for a period of years. In other words the period when Mr Mabior would have been most able to infect another person was long past before his HIV test was drawn on December 22 2003.

His HIV viral load tests 6,100 copies per ml ordered on February 6 (collected Feb 11 2004), and subsequently 6,300 copies per ml (collected

Feb 25 2004) are significantly low viral loads levels and would be consistent with probably low but possible infectivity. In any event from August 4 2004 till December 28 2005 his HIV viral load was below the level of detection. In my opinion there is a very high probability that Mr Mabior was not infectious ie could not have transmitted HIV throughout this period from 6 months after initiation of antiretroviral treatment in April 2004 ie October 22 2004 till December 28, 2005 – the last date for which we have a viral load). There is no evidence that he had any sexually transmitted infection during this time frame.

Although named as a contact of Chlamydia and epitreated for this by Dr Blyth on October 22 2004 his swab negative for Chlamydia according to the PHN note November 25 2004.If he had had an STI his viral load might have increased above the level of detection but as he was taking a potent antiretroviral combination throughout this period it seems unlikely that his viral load would have achieved even the low levels that he had in Brandon at the time of his gonoccal urethritis. He was again tested for STI contacts on June 21 2005 and February 8 2006. On each of these 2 occasions he tested negative for gonorrhea and Chlamydia lending support to the use of condoms.

Was Mr Mabior aware that his HIV was below the level of detection?

It is a routine and standard part of an HIV visit to advise a patient whether their viral load is detectable or not and what their CD4 count is. I think it is very unlikely indeed that his clinicians would not have discussed his viral load with Mr Mabior; however this is not specifically documented in his chart. Dr Kasper does mention in his letters that Mr Mabior is pleased with his response to treatment, is 100% compliant, and despite some symptomatic side-effects does not wish to change his Sustiva (Efavirenz) because of his good reponse to treatment. This suggest Mr Mabior was aware that his viral load was controlled.

Would Mr Mabior have had reason to believe he could not transmit HIV (ie was no longer infectious) at the time of the alleged offences?

No he would not. Prior to the publication of the report of the Swiss Federal Commission for HIV/AIDS in January 2008 the standard of care was to ensure that patients were aware that an undetectable viral load did not necessarily mean that they could not infect a sex partner and that they should continue to use condoms. Although his medical records do not specifically record that Mr Mabior was given this precise information it is implied by the fact that he was advised to use condoms on several occasions, was given or requested condoms, and that he consistently reported using condoms, purchasing them on one occasion.

Since Mr Mabior met the Swiss Commision's three criteria for non-infectiousness why should he use a condom?

Had Mr Mabior been **in a monogamous relationship, or entering a monogamous relationship** (with someone who was screened negative for HIV and Sexually Transmitted Infections - STIs), disclosed his HIV status to his partner, both of them discussed it with Mr Mabior's HIV physician, and Mr Mabior received her consent to not wear condoms, they would have been able to safely have unprotected sex providing he continued to take his antiretroviral medications and attend for his medical appointments regularly.

All persons having **multiple partner or casual sex** should be expected to understand the need and their own responsibility for unremitting universal precautions. This is to protect themselves from HIV and other STIs (or if they themselves have undiagnosed HIV or STI to protect their partners). Since this was Mr Mabior's sexual life style even though he met the three Swiss criteria for non–infectiousness he should have consistently worn a condom for two reasons. Firstly to maintain the third Swiss criteria of having no other sexually transmitted infections he **must** consistently use a correctly applied condom to prevent himself from being infected with an STI by his non-monagamous partner. Secondly to avoid exacerbating the

course of his HIV disease Mr Mabior should consistently use a condom to protect himself from the possibility of superinfection with a strain of uncontrolled HIV his partner might already have that could be resistant to the antiretrovirals he is currently on causing treatment failure and perhaps the loss of sensitivity to other antiretroviral agents he could otherwise have used. Since it is believed that approximately 1 in 3 persons with HIV in Manitoba have not taken the test and do not know they have HIV, Mr Mabior would like anyone else be well advised to adhere to condom usage until he has a regular sex partner who has also been HIV and STI tested.

Would it be medically necessary for an HIV infected person meeting, and maintaining, the Swiss criteria to disclose their HIV status prior to casual sex?

There is no scientific justification to require HIV status disclosure if a condom is always used. There is a **mutual responsibility** for casual sex partners to be aware of the innate risks of non-monogamy and to ensure their own safety by adhering to consistent and correct condom use. A person who knows they are HIV infected has **two additional responsibilities** to decline sex when a casual partner wishes to have unprotected sex, and if there is condom failure with possible sexual secretion or blood exposure, to advise their partner at that point to seek immediate evaluation for possible HIV post-exposure prophylaxis. Addendum. The rationale for requiring disclosure and the offer to accompany the casual partner for assessment for any possible (though unlikely) need for HIV post-exposure prophylaxis in this situation is to protect the interests of both persons. The person who knows themselves to be HIV infected needs to know that his/her partner is not already infected from a previous exposure. The person who does not know themselves to be infected needs to know whether her/his partner has a lost virologic control and has a detectable viral load and if that were the case to be evaluated for possible post-exposure prophylaxis. In the event that it turned out that both partners were HIV infected there might

possibly be benefit in knowing the history of previous antiretroviral resistance in case a superinfection occurred.

Had Mr Mabior been taught the correct way to use a condom to ensure maximum safety?

I could not find any specific documentation in the clinical records that Mr Mabior had received detailed instruction on correct condom usage or demonstrated an understanding of that from the Public health Nurses in Brandon or Winnipeg, or from the clinicians in Brandon or Winnipeg. On February 10 2004 the Public Health Nurse in Brandon recorded that information on HIV transmission and ways to reduce risk were discussed with Mr Mabior. The standard HIV test couselling requires this education and it would have been very unusual for it not to have been given.

What might cause Cato Mabior's antiretroviral regimen of Combivir (AZT+3TC) twice daily and Sustiva (Effavirenz) once daily to fail?

1. Primary Resistance. The information available does not support this
2. Failure to take his antiretroviral medications. There are several documentations of a high degree of adherence or compliance with his regimen. The fact that his viral load has never become detectable lends strong support to this. Even had he missed an occasional dose his HIV would remain well controlled. Sustiva levels would remain sufficiently elevated to control his HIV for at least 72 hours after the last dose.
3. Serious and prolonged vomiting or diarrhea. There is no documentation that he had either of these.
4. Drug-drug interactions. Co-administration of certain medications might result in lower blood levels of AZT (Clarithromycin, Rifabutin, rifampin, Nevirapine , Nelfinavir) or Sustiva (Rifampin, Phenytoin, Phenobarbitone). None of these medications were prescribed for Mr Mabior.