

# AIDS AND “THE OTHER ORIGINAL SIN.”



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**ABOVE PAINTING BY MICHELANGELO: HIS VERSION OF “ORIGINAL SIN.”**

**THE TIMELINE AND STORY PRESENTED IN THE CHRONICLES THAT FOLLOW DOCUMENT HOW “ORIGINAL ANTIGENIC SIN” HAS COME ABOUT DURING THE ERA OF “HIV/AIDS.”**

For instance, toward the end of a Congressional document prepared by the Government Office of Technology Assessment, the term “**original antigenic sin**” was advanced to describe why by **1995**, 30 “HIV” vaccines had failed:

*A) When a vaccinated individual is exposed to a non-cross-reactive strain of HIV that induces the production of antibodies specific for the vaccine strain that are unable to neutralize the newly encountered strain (in other words when a vaccine doesn't work).*

*B) The fixing of an immune response in a “non-adaptive pattern.”*

*C) When vaccinated individuals may be no worse off than unvaccinated individuals because non-vaccinated individuals also have a lag in generation of antibody to HIV because their immune response has not been “primed” by vaccination.*

The conclusions of The Office of Technology Assessment Book were presented to the 1995 Congress of the United States by the AIDS Research Advisory Committee (ARAC) of the National Institute of Allergy and Infectious Diseases (NIAID). The document recommended that Phase III clinical trials with enveloped vaccines should not proceed in the United States because of scientific, political, and ethical issues, and because of the significant level of scientific uncertainty about the wisdom of immediate trials (1995 Congress of the United States: Office of Technology assessment. Adverse Reactions to HIV Vaccines: Medical, Ethical, and Legal Issues. Roger C. Herdman, Director).

The following story explains how and why “HIV/AIDS” has come to change the Jennerian paradigm regarding the principle of vaccination, and how and why there have to date been more than 100 failed “HIV” vaccines as of 2012, and why there are currently 210 in the “HIV/AIDS” vaccine “pipeline.”

# SURVEY OF COMPETING THEORIES OF AIDS: A BRIEF HISTORY

Andrew Maniotis, PhD. Updated, 2/2012

Below is a list of 30+ hypotheses, and a time-line of important events and publications that have been advanced about “HIV/AIDS” that have evolved during the past 3 decades. Following the hypotheses, I identify the source, institutions, and dates from which these hypotheses emerged in chronological sequence. Only a brief description as to the nature of the hypothesis is provided in the list below to demonstrate how the field of “HIV/AIDS” has evolved, and, a more in depth description and discussion entitled, “**A brief history of AIDS**” follows this survey section.

As you will hopefully discern, the field of “HIV/AIDS” and has departed from the “scientific and medical consensus” that had advanced the notion that Acquired Immune Deficiency Syndrome was caused by “HIV, ” toward the recent 2009 documented claim made by Luc Montagnier, the Nobelist who is credited for the initial discovery of “HIV” and who now points to many co-factors, different causes than “HIV,” and other hopeful news regarding “AIDS,” such as his belief that:

*“you can get rid of it in a few weeks.”*

Some AIDS researchers and front-line AIDS doctors, following the lead of this Pasteur-Institute Nobelist credited for the discovery of “HIV” now recognize that AIDS is most likely caused by multi-factorial reasons that induce the set of at least 58 previously known syndromes that are collectively called Acquired Immune Deficiency Syndrome. You may be surprised to learn, in addition, that lymph node fibrosis, not the ill-termed and ill-conceived “viral load” readings (that never measured anything viral), or T-cell subset counts predict progression to fatal immune suppression, as researched and published by leading research institutions such as The Cleveland Clinic, or as seen in journals such as AIDS Clinical Care.

Here is the survey list in chronological order:

**1.** Cytomegalovirus causes AIDS because Dr. Michael Gottlieb found CMV in **100%** of his first 5 patients who all were gay drug-using men who had no contact with one another (UCLA, Dr. Michael Gottlieb, 1981, reported by the CDC’s MMWR, and in Nature, December **1981**).

**2.** Black slaves caused cancer because art work evidence proved to a scientifically established and epidemiologically justified racist construct, that these slaves that were depicted in works of art caught and contagiously transmitted a *cancer virus* called “HTLV-I” and then HTLV-II in the 16<sup>th</sup> Century from monkeys during the slave trade, which caused **0.6 %** of approximately **330,000** serologically tested Japanese near Nagasaki to develop a leukemia 40 years or so after the atomic bombs were dropped on them. This hypothesis was advanced for the origins of “HIV’s” suspected “cancer virus” relative or precursor(s), “HTLV-I and II” by Robert Gallo in 1979-81, to explain why a few (out of hundreds of thousands of) “HTLV-1” seropositive individuals acquired cancer in Southern Japan near Nagasaki. This is also why, a year later, when the announcement was made to the American public by Dr. Gallo and Margaret Heckler in 1984, the phrase, “the probable cause of AIDS has been found...to be “HTLV-III,” **a known “cancer virus”** was used. As mentioned, “HTLV-III,” was later renamed “HIV” by oncogene Nobelist Varmus’s committee (please see National Institute of Health: Gallo R.C., Sliski A., Wong-Staal F. Origin of T-cell leukaemia-lymphoma virus. Lancet (ii): 962-963, **1983**).

**3.** The virus “LAV” (the Pasteur group’s so-called non-productive “isolate,” named “lymphadenopathy ASSOCIATED virus), was associated with AIDS because Luc Montagnier’s “Patient 1” had many diseases and was said to have pre-AIDS. Montagnier’s so-called “LAV-BRU” or “LAV-LAI” “isolate’s” **association** with **pre-AIDS** was based on the fact that “Patient One” had been treated for syphilis, gonorrhea, cytomegalovirus, Epstein-Barr virus, and herpes viruses I and II. Perhaps not the ideal patient to isolate anything from or launch the AIDS era? Yet, even though “Patient One’s” lymphocytes were believed to harbor “LAV,” (later named

“HIV” by Varmus’s committee), Montagnier later admitted they couldn’t purify “HIV” despite “a Roman effort,” a required technical step in proving the association of any virus with disease (Pasteur Institute: Barre-Sinoussi, F, J.C. Chermann J.C., Rey F., Nugeyre MT., Chamaret, S., Gruest J., Dauguet C., Axler-Blin C., Vezinet-Brn F., Rouzioux, C., Rozenbaum R., & Montagnier L. Isolation of a T-lymphotropic retrovirus from a patient at risk for Acquired Immune Deficiency Syndrome. *Science* 230: 868-871, **1983**).

**4.** Gallo’s press conference with HHS director Margaret Heckler in 1984 in front of the nation proved that “HIV” was not a cancer virus as he tried to previously establish (to lay claim to discovering the first Human retrovirus), but was described instead as a cancer virus **variant**. Gallo’s claim that “HIV” causes AIDS, was based on the finding that **36%** of 72 of his AIDS patients tested “HIV-positive,” even though many were not sick. This claim was the basis of the first “HIV-test kits, and the American-French patent for that kit. (It also was 64% less of an association that Gottlieb reported for CMV’s association with his original AIDS patients). Four papers published in *Science* in 1984 are the papers said to establish the causal association between “HIV” and “AIDS,” although many have since disputed the data in these papers, as they were subject to intense controversy and still are (National Institutes of Health: Popovic M, Sarngadharan MG, Read E, et al. Detection, Isolation, and Continuous Production of Cytopathic Retroviruses (HTLV-III) from Patients with AIDS and Pre-AIDS. *Science* **1984**; 224:497-500.; Gallo RC, Salahuddin SZ, Popovic M, et al. Frequent Detection and Isolation of Cytopathic Retroviruses (HTLV-III) from Patients with AIDS and at Risk for AIDS. *Science* **1984**; 224:500-502; Schupbach J, Popovic M, Gilden RV, et al. Serological analysis of a Subgroup of Human T-Lymphotropic Retroviruses (HTLV-III) Associated with AIDS. *Science* **1984**; 224:503-505; Sarngadharan MG, Popovic M, Bruch L, et al. Antibodies Reactive to Human T-Lymphotropic Retroviruses (HTLV-III) in the Serum of Patients with AIDS. *Science* **1984**; 224:506-508).

**5.** Robin Weiss says “HIV” attacks brain cells through the T4 receptor ( Paul Jay Maddon, Angus G. Dalgleish, J. Steven McDougal, Paul R. Clapham, Robin A. Weiss, and Richard Axel (*Cell*, Vol 47, 333-348, 7 November **1986**).

**6.** Smallpox vaccine campaigns in Africa caused AIDS as they woke up latent “HIV” harbored by blacks who were vaccinated. (The World Health Organization, published in *Letters to The Lancet*, and *NEJM*, London Times Edition 1 MON 11 MAY **1987**). A Competing hypothesis advanced for the origin of “HIV” was that the virus emerged from barbaric African sexual practices to stimulate a man or a woman and induce them to intense sexual activity, monkey blood [for a man] or she-monkey blood [for a woman] was directly inoculated in the pubic area and also the thighs and back. These magic practices would therefore have unequivocally constituted an efficient experimental transmission model and could be responsible, according to this blatantly racist thinking, for the emergence of AIDS in man, as was published in *The Lancet*. (Noireau F. HIV transmission from monkey to man-*Lancet* (i):1498-1499, **1987**).

**7.** “HIV” is a harmless passenger virus: “HIV” exists but does no harm, and doesn’t cause AIDS. (University of California, Berkeley: Peter H. Duesberg, “Retroviruses as Carcinogens and Pathogens: Expectations and Reality *Cancer Research* 47, 1199-1220, March 1, **1987**).

**8.** AIDS is actually syphilis, “the great imitator,” which presents with identical T—cell disturbances, profound immune suppression, and dementia (Washington’s National Library of Medicine. Coulter HL. AIDS and Syphilis-The Hidden Link, CA, pps. 20, 21, **1987**).

**9.** AIDS is caused by Redox imbalance and proteins and behaviors that oxidize cells and tissues (Royal Perth Hospital, Perth Australia, Papadopulos-Eleopulos E. *Medical Hypotheses* (**1988**) 25: 151-162).

**10.** Malnutrition, recreational and prescription drugs, and foreign proteins cause AIDS (University of California, Berkeley: Peter H. Duesberg, *Proc. Natl. Acad. Sci. USA* Vol.86, pp. 755-764, February **1989**).

**11.** Anti-HLA alloantigens cause AIDS (Michigan State University: Robert Root-Bernstein. Do we know the causes of AIDS. *Perspectives in Biology and Medicine*: Summer, 33:480-500, **1990**).

- 12.** Mycoplasma incognitus causes AIDS: **22/34** patients with AIDS demonstrated high titres of M. incognitus in their tissues while **0** non-AIDS patients have the microbe (Armed Forces Institute of Pathology: Lo SC, Hayes MM, Wang RY, Pierce PF, Kotani H, Shih JW. Newly discovered mycoplasma isolated from patients infected with HIV. *Lancet*. Dec 7;338 (8780):1415-8, **1991**).
- 13.** AIDS is caused by multiple –antigen-mediated-autoimmunity (MAMA) through idiotypic/anti-idiotypic immune reactions (Michigan State University: Robert Root-Bernstein, ‘Rethinking AIDS; The tragic cost of premature consensus’ The Free Press/Macmillan USA, 527 pages, **1993**. ISBN 0-02-926905-9).
- 14.** AIDS is caused by “HIV” hiding in lymph nodes and destroying them. Pantaleo G, Graziosi C, Demarest JF, Butini L, et al. HIV infection is active and progressive in lymphoid tissue during the clinically latent stage of disease. *Nature*; 362(6418):355-8, **1993b**).
- 15.** AIDS is caused by exposure to too many viruses, which is why no vaccine is possible: AIDS is a chemical chain reaction because of all these virus infections, and the manner by which they destroy or confuse the immune system (Kary Mullis, multiple institutions, inventor of PCR, Nobel Prize Recipient. A hypothetical disease of the immune system that may bear some relation to the Acquired Immune Deficiency Syndrome. *Genetica* 95: 195-**1995**).
- 16.** “HIV” is caused by oxidizing T-cell cultures with a plant lectin, phytohemagglutinin, and IL2. (Royal Perth Hospital, Perth Australia, Eleni Papadopulos-Eleopulos, Valendar F.Turner, John M. Papadimitriou, David Causer, Bruce Hedland-Thomas, Barry Page. a critical analysis of the HIV-T4-Cell-AIDS hypothesis. *Genetica* 95: 5-24, **1995**).
- 17.** Influenza vaccines cause “HIV.” (Simonsen L, Buffington J, Shapiro CN, et al. Multiple false reactions in viral antibody screening assays after influenza vaccination. *Am J Epidemiol* 141:1089-1096,**1995**).
- 18.** “HIV” isn’t sexually transmissible between 175 serodiscordant sexual partners (where one tests “HIV-positive and the other “HIV-negative) who don’t use condoms, if folks receive expert counseling, therefore lack of expert counseling causes AIDS (Padian, et al. Heterosexual Transmission of HIV in Northern California: Results from a Ten-Year Study.” *American Journal of Epidemiology*. August, **1997**).
- 19.** “HIV”’s primary target of destruction is the gut, and specifically the Peyer’s patches, which causes AIDS. (12th International conference on Retroviruses and Opportunistic infections Jon Cohen, Science Writer, *Science*, 4 March,: Vol 307, p. 1395, **2005**)
- 20.** Cigarette smoking causes “HIV” but cigarettes don’t cause AIDS. (Dr Andrew Furber, of the South East Sheffield Primary Care Trust, London, Reuters, Thursday, Sep 21, **2006**).
- 21.** The Thy-1/Thy-2 lymphocyte imbalance caused by many stressors to the body causes AIDS and can be reversed. (German Federal HIV Study, Parliamentary HIV/AIDS Commission biostatistics unit of Wuppertal University, *Raum & Zeit*, **2006**).
- 22.** Cross presentation of caspase-cleaved apoptotic self-antigens is induced by “HIV-infection” to cause AIDS. (University of Italy: Rawson et al., Cross presentation of caspase-cleaved apoptotic self antigens in HIV infection. *Nature Medicine* Vol. 13, No 12, December **2007**).
- 23.** “HIV” requires exactly 237 cellular proteins to cause AIDS. Harvard Medical School, Howard Hughes Medical Institute Stephen J. Elledge, January 10, **2008**, in *Science Express*).
- 24.** There is no AIDS because the world’s AIDS epidemic is over, except in Africa (WHO epidemiologist, Dr. Kevin de Cock, May 19, 2008, Geneva AIDS conference).

**25.** AIDS originated 125 years ago when the virus jumped from chimpanzees to blacks. (University of Arizona, Nature, October, **2008**).

**26.** A new strain of “HIV” jumped from a gorilla to a black woman living in Cameroon as reported in **2008**, proving unequivocally through molecular phylogenetic analyses of “HIV” and “SIV” sequences that although the woman is not ill in any way, this new strain of virus rapidly mutated and adapted to her body, and now threatens to ignite a new deadly global AIDS pandemic (University of Rouen, France, Jean-Christophe Plantier, reported in Nature, **2008**).

**27.** Viral loads or T-cell killing don’t cause AIDS: fibrosis of the lymph nodes causes AIDS. Evaluation of the pathogenesis of decreasing CD4<sup>+</sup> T cell counts in human immunodeficiency virus type 1–infected patients receiving successfully suppressive antiretroviral therapy. Nies-Kraske E. et al. J Infect Dis. Jun 1; 199:1648, **2009**).

**28.** A defect in innate immunity causes AIDS that is mediated by the cellular protein tetherin, because tetherin inhibits the release of HIV-1, EBOLA, and Kaposi’s associated herpesvirus 8, and probably all viruses from infected cells (University of California, San Diego, and the Veterans Affairs San Diego Healthcare System. 362:6 February 11, 2010. John Guatelli, M.D. 362:6 February 11, **2010**).

**29.** Not enough smallpox vaccinations caused AIDS because of the CCR5 receptor binds to both smallpox viruses, and “HIV,” and Petri-dish experiments prove that a competition of smallpox virus with “HIV” for the CCR5 receptor inhibits “HIV” (George Mason University, Manassas, Virginia, worked with a team of researchers from George Washington University and UCLA ScienceDaily (May 18, **2010**))

**30.** “HIV” doesn’t cause AIDS because if you have a good immune system, you can get rid of it in a few weeks, according to the Nobelist Montagnier, who first described “HIV.” (Interview and documentary, House of Numbers: **2010**).

**Brief Youtube clip:** <http://www.youtube.com/watch?v=WQoNW7lOnT4l>,

**Entire award winning documentary:** [http://www.facebook.com/l.php?u=http%3A%2F%2Fhiv-dissidents.tabaru.com%2Fvideo%2F%2F26508\\_Dom\\_chisel.html&h=f6d5eYwjJV-GDsjmQI3gQp8u\\_Vg](http://www.facebook.com/l.php?u=http%3A%2F%2Fhiv-dissidents.tabaru.com%2Fvideo%2F%2F26508_Dom_chisel.html&h=f6d5eYwjJV-GDsjmQI3gQp8u_Vg)

### **THE ORTHODOX VERSION, AND THE BEST THIRTY + HYPOTHESES:**

On June 5th, 1981, the first article about AIDS appeared in the medical literature, “Pneumocystis Pneumonia—Los Angeles,” in Morbidity and Mortality Weekly Report (vol. 30, pp. 250-52), by Dr. Michael Gottlieb and colleagues of University of California at Los Angeles [1].

Gottlieb and colleagues detailed the five case studies, noting the commonalities among the cases, such as sexual preference and quick development of a rare form of pneumonia. All five patients were previously healthy individuals who had laboratory-confirmed cytomegalovirus (CMV) infection within five months of PCP diagnosis and candida mucosal infection (<http://www.infectiousdiseaseneews.com/200606/discovery.asp>).

The following are summarized excerpts of the patient’s symptoms written in the original MMWR case reports, in which it was noted that 100% of the patients exhibited cytomegalovirus (CMV) infections:

*\* Patient 1: A 33-year-old man developed PCP and oral mucosal candidiasis in March 1981, after two months of fever that was **associated with elevated liver enzymes**, leucopenia and **CMV** viruria. The patient’s condition declined despite treatment*

with trimethoprim-sulfamethoxazole (TMP-SMX), pentamidine and acyclovir. He died on May 3.

\* Patient 2: A 30-year-old man developed PCP in April 1981 after five months of fever **associated with elevated liver enzymes, CMV viruria and seroconversion to CMV**. In addition, he had leucopenia and mucosal candidiasis. PCP responded to treatment with intravenous TMP-SMX, however he continued to have fevers.

\* Patient 3: A 30-year-old man developed esophageal and oral candidiasis around January 1981. He was treated with amphotericin B and responded well. In February, he was hospitalized for PCP that responded to treatment with oral **TMP-SMX**. Esophageal candidiasis reoccurred and he was retreated with **amphotericin B**. Biopsy showed he was positive for **CMV**.

\* Patient 4: A 29-year-old man developed PCP in February 1981. **Three years prior he had Hodgkin's disease; however, was successfully treated with radiation therapy**. PCP did not improve after administration of intravenous TMP-SMX and corticosteroids. He died in March. Postmortem examination showed **PCP and CMV** in lung tissue.

\* Patient 5: A 36-year-old man visited his physician in April 1981 after a four-month history of fever, dyspnea and cough. He had been clinically diagnosed with **CMV** in September 1980. He was diagnosed with PCP, oral candidiasis and **CMV** retinitis. He was administered two short courses of TMP-SMX and was treated for candidiasis with topical nystatin.

## **CYTOMEGALOVIRUS (A HERPESVIRUS) FOUND IN 100% OF**

### **“AIDS” PATIENTS.**

Although the 5 patients didn't know or ever have contact with one another, Gottlieb et al. concluded: *...all of the above observations suggest the possibility of a cellular-immune dysfunction related to a common exposure that predisposes individuals to opportunistic infections, such as pneumocystosis and candidiasis. **Although the role of CMV infection in the pathogenesis of pneumocystosis remains unknown, the possibility of P. carinii infection must be carefully considered in a differential diagnosis for previously healthy homosexual males with dyspnea and pneumonia.***

In December of that same year, another study published by Dr. Gottlieb and his collaborators concluded that **CMV was important** in the pathogenesis of the syndrome they had described:

*Four previously healthy homosexual men contracted Pneumocystis carinii pneumonia, extensive mucosal candidiasis, and multiple viral infections. In three of the patients these infections followed prolonged fevers of unknown origin. **In all four cytomegalovirus was recovered from secretions.** Kaposi's sarcoma developed in **one** patient eight months after he presented with esophageal candidiasis. **All patients were anergic [couldn't respond to foreign antigens] and lymphopenic [had low numbers of T-cells]; they had no lymphocyte proliferative responses to soluble antigens, and their responses to phytohemagglutinin were markedly reduced.** Monoclonal-antibody analysis of peripheral-blood T-cell subpopulations revealed virtual elimination of the Leu-3 / helper/inducer subset, an increased percentage of the Leu-2 + suppressor/cytotoxic subset, and an increased percentage of cells bearing the thymocyte-associated antigen T10. **The inversion of the T helper to suppressor/cytotoxic ratio suggested that cytomegalovirus infection was an important factor in the pathogenesis of the immunodeficient state.** A high level of exposure of male homosexuals to cytomegalovirus-infected secretions **may** account for the occurrence of this immune deficiency.<sup>1</sup>*

Michael Gottlieb recounted his 1981 *New England Journal of Medicine* discoveries in a July 26, 1993 issue of *Current Contents*:

*Because cytomegalovirus (CMV) was cultured from multiple sites, I proposed that it might be **causal. This proved to be an***

**error. CMV had been reactivated because of immune deficiency.** However, I also suggested that a previously unrecognized **toxin, microbe, or virus** might be the culprit.

*The release of my MMWR article set off an explosion of interest. People began reporting cases of AIDS from New York, San Francisco, and many other cities. Publication of the NEJM report on December 10, 1981, changed my life, it was one of the most heavily quoted publications in the medical literature during the first several years of the epidemic.*

*Since I described those early cases, I have been continuously involved with clinical research on AIDS and with the care of people in various stages of HIV infection. I authored 50 papers, mostly on clinical aspects of HIV disease, including several other first reports of phenomena associated with AIDS.<sup>2-3</sup> I became active in AIDS causes and was willing to speak candidly with reporters at a time when there were few “experts” on the disease. Despite my record of productivity in clinical research and community service, in 1987 I was **denied tenure** at UCLA.*

Gottlieb’s early observations were rapidly confirmed by the observations of other clinicians, who also reported patients that presented with anergy and shortage of lymphocytes (anergic is an inability to produce an immune response to immune stimulants or irritants). For example, in December of 1981:

***All patients were anergic and lymphopenic; they had no lymphocyte proliferative responses to soluble antigens, and their responses to phytohemagglutinin were markedly reduced. Phytohemagglutinin is a plant lectin “phyto” that causes T-cells to become “activated” and which was used to stimulate all cultures said to propagate “HIV” several years later.***

Other clinicians reported in the NEMJ a similar puzzling cohort **whose humoral immunity was “intact,”** but **believed** that cellular immunity was compromised:

***Eleven cases of community-acquired Pneumocystis carinii pneumonia occurred between 1979 and 1981 and prompted clinical and immunologic evaluation of the patients. Young men who were drug abusers (seven patients), homosexuals (six), or both (two) presented with pneumonia. Immunologic testing revealed that absolute lymphocyte counts, T-cell counts, and lymphocyte proliferation were depressed, and that humoral immunity was intact. Of the 11 patients, one was found to have Kaposi’s sarcoma, and another had angioimmunoblastic lymphadenopathy. Eight patients died. In the remaining three, no diagnosis of an immunosuppressive disease was established, despite persistence of immune defects. These cases of pneumocystosis suggest the importance of cell-mediated immune function in the defense against P. carinii. The occurrence of this infection among drug abusers and homosexuals indicates that these groups may be at high risk for this infection.***<sup>2</sup>

In July of that same year, a Task Force on Kaposi’s Sarcoma and Opportunistic Infections was established at the CDC under the direction of Dr. James Curran [1]. However, much of the scientific knowledge that was being accumulated on the spread of AIDS in America came from the surveillance and blood testing of large groups of gay and bisexual men who volunteered as human test subjects in the original Hepatitis B vaccine trials which took place in six American cities during the years 1978-1981.

Volunteers in the program were required to be homosexual or bisexual men under the age of 40, healthy and promiscuous. In New York, after screening the blood of almost ten thousand men, a final group of 1,083 were selected to participate in the first Hepatitis B Vaccine Study. The experiment took place at the New York City Blood Center in Manhattan, during November 1978.

*“In November 1978, the first gay man was inoculated at the New York City Blood Center. By October 1979, all the men in (the) study were inoculated, and within a decade, most of the men in the experiment would be doomed to die of AIDS.”*

According to two physicians, Dr. Strecker and Dr. Alan Cantwell:

*“the appearance of AIDS in New York City in 1978 coincided exactly with the Hepatitis B vaccine trials conducted there. The first appearance of AIDS in San Francisco and Los Angeles in 1980 closely followed a similar vaccine program undertaken in those cities.”*

**ART WORK EVIDENCE PROVES THAT BLACK SLAVES CAUGHT A CANCER VIRUS FROM MONKEYS IN THE 16<sup>TH</sup> CENTURY DURING THE SLAVE TRADE WHICH CAUSED JAPANESE LIVING NEAR NAGASAKI TO CATCH CANCER CENTURIES LATER, AFTER THE ATOMIC BOMB WAS DROPPED ON THEM.**

In 1975, Robert Gallo published a paper saying he had isolated a new human virus – human leukaemia virus 23. <sup>3</sup>

*Gallo was jubilant, it was the justification for years of dedication. ‘We got permanently growing cell lines eventually, and it was a great eureka. We succeeded ten times in ten different cell lines, and **we thought** we had made the discovery, the genuine article, **that retroviruses exist in humans**. A year or more of analysis went by. We thought it was a triumph.’”*

This period of research turned from being Gallo’s greatest triumph to date into his greatest disaster. When other scientists looked at this virus they discovered it was a mixture of three animal viruses: from a gibbon, a baboon and a woolly monkey.

*As Gallo said: ‘I was depressed, dumbfounded, angry. It was the low point of my whole career. It was **almost** the last nail in the coffin of the field of retrovirology. The programme died, and all the good that came out of it, **like interleukin-2, which would be so important in fighting cancers**, didn’t seem to matter, to me or to the world. I became more cynical, tougher, less happy. I mean, what could it be **butsabotage**? One contamination can occur, but three? In fifteen years I had had one contamination from a mouse. But three?’*

After this so-called NM23 mistake, in 1978-9 Gallo’s group believed they isolated a retrovirus they thought might cause leukemia, and called it HTLV-I. The results were published in 1980 and early 1981. The molecular signature of HTLV-1 and the way it was obtained, was critical for what was to follow in the pursuit of “the AIDS virus.” Detailed and accurate descriptions of the initial characterization methods and hypotheses can be found in the new book, “*Dissecting a Discovery*,” by Nikolas Kontaratos, who is [a non-firearm-carrying security guard and filter salesman](http://dissectingadiscovery.com/) Gallo hired to ghost write his recollections

(<http://exlibhollywood.blogspot.com/2009/05/doctors-without-boundaries.html> ), and perhaps is a period of discovery best recounted on line in a well-written account by Jad Adams. <sup>4</sup>

*Gallo is credited with isolating and describing **the first** human retrovirus. Japanese and American researchers confirmed by analyzing **the RNA** of both isolates that the Japanese and American viruses were related strains of the same virus. **They could never be exactly the same** because of the **mutations** which occur **as the virus replicates**, but the RNA sequence was **close enough** in the two isolates.*

*Once the virus had been described, other laboratories looked for it. It was found in **black patients** born in the US, Caribbean countries or South America; **Caribbean-born black people in England, Africans and Japanese [living 175 miles from Nagasaki]**. What could tie these disparate regions together mused Gallo.*

*The answer he came up with was **the slave trade**. Miyoshi in Japan found Japanese macaques had antibodies to HTLV-I and he **suggested** the monkeys had the disease first and infected people. Researchers at Gottingen, Germany, and in Gallo’s lab found*



that **many species** of African monkeys had **antibodies** which reacted with HTLV-I. **African green monkey and chimpanzee viruses [antibodies]** were most closely related to the virus Gallo had found **in leukaemic cells**.

Gallo suggested: HTLV-I originated in **Africa** where it **infected** many species of Old World primates including human beings. It reached the Americas along with **the slave trade**.

**Curiously**, it may well have arrived in Japan the same way. In the sixteenth century Portuguese traders traveled to Japan and stayed specifically in the islands where HTLV-I is now **endemic**. Along with them they brought both **African slaves and monkeys**, as contemporary Japanese **works of art** show, **and either one or the other may have carried the virus**.

The discovery of HTLV-I infection on Hokkaido, one of the northern islands of Japan, immediately challenged this view of events **but Gallo and his colleagues** have remained attached to **the monkey-virus theory. [The monkey slave-virus theory]**.

So, why is it thought that this virus causes the leukaemia? 'First because of the coincidence between virus and leukaemia – **find one and you will find the other**,' Gallo says.

The incidence of adult T-cell leukaemia in Japan is estimated to be only **0.06** per cent based on **339** cases of T-cell leukaemia among **600,000** subjects who are antibody-positive for HTLV-I. Why is this?

Because of **the latency period**, responds Gallo. **It will cause** leukaemia, but it may take as long as **forty years**.

Despite the art work evidence that "The leukemia virus" originated during "the slave trade," and the observation by Dr. Joseph Mercola presented in the Introduction that scid marks cause car accidents, these hypotheses and notions of Gallo regarding the origin of "HTLV-1, and later, "HIV," should be considered in light of what Africans and African scholars have written. For instance, Rosalind Harrison-Chirimuuta and Richard Chirimuuta <sup>5</sup> suggested that:

*Racism and "AIDS from Africa"*

*The first black people diagnosed as suffering from AIDS in any number were Haitians living in the United States. The possibility that they may have caught the infection from Americans in the United States or in Haiti was not given serious consideration and Haiti was immediately accused of being the source of the epidemic.(11) Soon Haitians with AIDS were being reported from all over the Western world (12,13,14,15) and the Centers for Disease Control (CDC) in Atlanta, Georgia, included Haitians as a group at risk for AIDS along with homosexuals, intravenous drug users and haemophiliacs. It was only in 1985 that CDC, faced with overwhelming evidence that Haitians per se were no more at risk for AIDS than anyone else,(16) removed them from the high risk classification, but not before Haitians en masse were **dismissed from their jobs, evicted from their homes, and even housed in separate prisons**. Abandoning Haiti, the researchers then turned their attention **to Africa**.*

*One of the reasons given by scientists for this turn to Africa was the high incidence of Kaposi's sarcoma (KS) in Africa, although it was clear from the beginning that **the benign clinical course of African KS** was **very different** from the aggressive, **disseminated form** of KS in AIDS patients.(17) A number of AIDS-like cases were reported **retrospectively**, the most cited being a Danish surgeon who worked in Zaire and died in 1977.(18) This patient was given prominence in the best-selling book by Randy Shilt's "And The Band Played On" where, under "Dramatis Personae" she is listed as "Danish surgeon in Zaire, first westerner to have died of AIDS," and is described in the following manner:*

*The battle between humans and disease was nowhere more bitterly fought than here in the **fetid** equatorial climate, where heat and humidity fuel **the generation of new life forms**... Here, on the frontiers of the world's harshest realities, Grethe Rask*

tended the sick(19).

Jonathan Mann, former director of the AIDS program for the World Health Organisation (WHO) and medical text books are now citing **the case** as evidence that AIDS originated in central Africa.(20,21) It was claimed that she acquired the infection from her patients, at least one of whom had KS, but there was no firm evidence that she died of AIDS, and other diagnostic possibilities were not considered. In 1988, five years after the case was published, we learned that her serum had been tested and found human immunodeficiency virus (HIV) enzyme linked immunosorbent assay (ELISA) **negative**, (22) but the author of the original paper has not published this information in the scientific literature.

Although such AIDS-like cases are presented as evidence that the human immunodeficiency virus existed in Africa **prior** to the American epidemic, only African cases are considered and the many instances of AIDS-like cases documented in Europe and America are conveniently ignored.(23) Indeed, on the opposite page to the report of the Danish surgeon in the same issue of **The Lancet** was an account of an AIDS-like illness in a young German homosexual,(24) but whilst **non-AIDS** in a Danish surgeon heads the citation index **proving** an African origin, the German case has been completely **ignored**.

The next source of support for the African hypothesis came from the seroepidemiological studies undertaken in Africa or on African serum stored in the West. This research, **more than any other**, has been at the foundation of all the fantastic stories of **millions** dying in Africa. Using an enzyme linked immunosorbent assay seropositive figures of **25%** of patients attending a clinic in rural Zaire in 1984,(25) **50%** of the Turkhanas in Kenya from 1980-1984,(26) and **66%** of children in Uganda in 1972(27) were reported. **As AIDS was rare or unknown in the areas where the serum was collected, one would expect the authors to have had serious doubts about the reliability of the tests**, but sadly scientific scepticism has never been a feature of AIDS research in Africa.

One of the most cited studies was undertaken on serum collected in Zaire **in 1959**.(28) Using **a number of tests** in addition to ELISA, only **one** of **1,213** plasmas was positive, but the identity of the donor, described as “rural Bantu”, was no longer known. As with the sporadic AIDS-like cases, only seroepidemiology in Africa is considered relevant to the question of the origin of HIV. A study using the same tests was undertaken on stored serum taken from “**aboriginal**” Amazonian Indians in Venezuela in **1968/69**, and **9of 224** samples were positive on **all** the tests.(29) The results were challenged by other researchers as **probable false positivity**.(30) but **the single** positive sample from Zaire continues to be cited as evidence that the world AIDS epidemic began in Zaire 35 years ago.

In an interview shown on British television Professor Hunsmann, head of virology and immunology section and professor of medicine at the (West) German Centre of Primate Research at Gottingen, discussed the problems of seroepidemiology:

“We had conducted quite extensive experiments in respect to the epidemiology... **first human retrovirus... HTLV [Human T-Lymphotropic Virus]**... For this reason we had several thousand serum samples frozen and saved in our refrigerated stock. When the news came that there was another, and new human retrovirus discovered, the AIDS virus... we could immediately search among our stock and probe for an earlier presence of this virus in Africa... These tests quickly and clearly gave results, namely, that the first “positive” probes which we could find among our more than **7,000** serum samples are dated only after the beginning of the ‘eighties, from the years 1982-83; and that among samples from before that date — and we had quite a lot of that earlier time in our stock- **nota single one** proved positive. We have concluded from all this that most other researchers had probably fallen victim to **the technical difficulties connected with the conservation and analysis of older serum samples**. And the American authors who originally had produced those high percentage data had to correct them — but certainly, once some wrong information like that has been put into circulation it continues to go on. This has led to quite a lot of friction between some African states and the United States.”(31)

Later in the same interview when asked why AIDS is not considered to have originated in the United States, Professor Hunsmann made the following comment:

“Testing of the kind being done in Africa and to that volume **has never been done** by anyone in America. Nobody has looked at the stocked blood serum in the USA and there certainly is much more there than in Africa. Nor has anyone asked what happened to the general population. Only one single group, the homosexual community in San Francisco, has been analysed and the results showed a high percentage of HIV positivity already **by the mid 1970’s**. But no other samples have been tested to the extent done in Africa. I think this should be clearly said.”(31)

Why, then, if this research is valid (and there have not been any serious criticisms) have other AIDS researchers persisted in arguing that the African AIDS epidemic preceded the epidemic elsewhere in the world? And **if the tests are unreliable**, why are the predictions that millions of Africans will soon die from AIDS still presented **without comment**? How, indeed, is it possible that a virus could spread so much more rapidly **by heterosexual contact in Africa** than anywhere else in the world? It is here as in so many other aspects of AIDS research, **that racist beliefs about the sexual propensities and promiscuity of Africans** conflict with scientific evidence, and in such a confrontation **belief is almost invariably the victor**.

**Researchers had originally proposed that AIDS was an “old disease of Africa” that had reached the West via recent intercontinental travel ,(3) a rather curious notion given the enforced intercontinental travel of up to 100 million Africans in previous centuries (32). [Slavery].** As this hypothesis became increasingly untenable attention was diverted to the possibility of **a monkey origin of the virus**. Such ideas cohabit easily with racist notions that **Africans are evolutionary closer to sub-human primates**. Dr. Robert Gallo and his co-workers were among **the pioneers** of this line of research, both for HTLV-I and HTLV-III (later renamed HIV). (5,33,34) Two of Gallo’s colleagues, Kanki and Essex, reported the isolation of a virus similar to HTLV-III in macaque monkeys who were suffering from an **AIDS-like** illness, and labeled it simian T-lymphotropic virus type III (**STLV-III**) of macaques. (35) For those who were arguing an African origin of the AIDS virus, **an Asian monkey** like the macaque was not a suitable source, but less than six months later the same researchers reported **finding the virus** in “wild-caught” **African green monkeys** from Kenya and Ethiopia.(36) This research, like most other research on AIDS in Africa, was motivated only **by a desire to prove an African origin** of the disease, and was greeted with enthusiasm by the Western scientific community. Discussion quickly moved on to the question of how the virus crossed the species barrier, and two AIDS “experts” from St Mary’s Hospital in London even offered this explanation:

“Monkeys are often **hunted for food** in Africa. It may be that a hunting **accident of some sort, or an accident in preparation for cooking**, brought people **in contact** with **infected** blood. Once caught, monkeys are often kept in huts for some time before they are eaten. **Dead monkeys are sometimes used as toys by African children** (37).”

Are we seriously to believe that African parents are so desperate for toys for their children that **they give them putrefying carcasses of dead animals**? More fantastic suggestions were published in *The Lancet*:

“Sir: The isolation from monkeys of retroviruses closely related to HIV strongly suggests a simian origin for this virus... Several unlikely hypotheses have been put forward... In his book on **the sexual life of people of the Great Lakes area of Africa** Kashamura writes: “pour stimuler intense, on leur inocule dans les cuisses, **la region du pubis** et le dos du sang preleve sur un singe, pour un homme, sur une guenon, pour ne femme” (to stimulate a man or a woman **and induce them to intense sexual activity, monkey blood [for a man] or she-monkey blood [for a woman] was directly inoculated in the pubic area and also the thighs and back**). These magic practices would therefore constitute **an efficient** experimental transmission model and could be responsible for the emergence of AIDS in man.” (38)

This came in for particular **derision** at the conference on AIDS in Africa held in Naples in October 1987:

When queried regarding the plausibility of a premise put forth in a letter to *The Lancet* suggesting that a bizarre tribal ritual of injecting monkey blood into the pubic region of young African men and women to stimulate intense sexual activity could be responsible for the emergence of AIDS in man, researchers from Zaire, Congo and Belgium were unanimous in declaring it to be **preposterous...**(39).

*It is hardly surprising that western AIDS researchers have become persona non grata in many African countries.*<sup>6</sup>

According to conversations I had with Dr. Gallo, his work with Dr. Montagnier was open and collaborative, and Gallo believes that his work established that AIDS and Montagnier's group's pre-AIDS from "Patient One" exhibited consistent molecular markers or profiles [2]. These markers were first called lymphadenopathy-associated virus (LAV-BRU and LAV LAI), Human T-cell-lymphotropic virus type III (HTLV-IIIB), AIDS-related virus (ARV) and, finally, Human Immunodeficiency Virus ("HIV").

The importance of these initial observations in shaping the AIDS era cannot be overestimated. From a later *NEJM* retrospective article written jointly by Gallo and Montagnier we learn that [2]:

*The story began in an unfavorable environment: during the late 1970's many people thought that epidemic diseases caused by microbes, including viruses, no longer posed a threat in industrialized countries.*

**Other prevailing beliefs were that viruses did not cause any human cancer and that there was no such thing as a retrovirus that infected humans.** Some of these beliefs were justified, since attempts to find tumor viruses and, in particular, retroviruses in cancers and other diseases in humans had a troubled history, and many of the groups that had the greatest expertise in the study of retroviruses had turned their efforts toward research on oncogenes. Luckily, and rather amazingly, however, the conceptual and technical tools arrived in our hands just before the first patients with AIDS were identified in 1981. In addition, there remained a few heretical or "old-fashioned" groups-among which were our two laboratories-that persisted in searching for retroviruses in human cancers, particularly breast cancers and leukemias. **This search finally paid off with the discovery of human T-cell leukemia virus types 1 and 2 (HTLV-1 and HTLV-2), the first of which was shown to cause an unusual T-cell leukemia.** This discovery was made possible by 15 years of basic research on leukemogenic retroviruses in animals, including the design and development of highly sensitive biochemical assays that were based on reverse transcriptase-the enzyme that is present in all retroviruses, which was discovered in 1970 by Temin and Baltimore.

An additional important contributor was the development of methods for growing T lymphocytes in culture for a period sufficient to allow the expression of putative latent retroviruses. This effort was helped greatly by the isolation of specific factors-in particular, the T-cell growth factor (now called interleukin 2) in Bethesda, Maryland. The role of interferon in repressing the production of retroviruses in mouse cells was demonstrated in Paris, and this discovery led to the use of anti-interferon serum in the search for human retroviruses. **Thus, at the beginning of the 1980's, we had the essential tools required to search for a retrovirus in this new and menacing disease called AIDS.** But why search for a virus, and specifically a retrovirus, in AIDS? The answer was far from obvious in 1982.

At that time, AIDS had already appeared as a long-lasting disease, with an extremely long lag time between exposure to the agent (through blood or sexual activity) and the profound state of immune suppression characterized by the occurrence of opportunistic infections or cancers. Many factors-fungi, chemicals, and even an autoimmunity to leukocytes-were invoked at that time as possible causes. **However for us, there were clues.** First, the various manifestations of AIDS were unified by a biologic marker: a decrease in the levels of a specific subgroup of T cells that harbored the CD4 surface antigen. CD4 and other CDs had been identified only a few years earlier with the use of specific monoclonal antibodies, thanks to the work of

*Milstein and Kohler. The findings regarding the T cell subgroup suggested an agent that specifically targeted CD4+ T cells, and HTLV (the putative cancer causing virus) was one such agent. Moreover there were animal models in which lymphotropic retroviruses caused not only leukemias or lymphomas, but also an AIDS-like wasting syndrome. Furthermore, HTLV was transmitted through blood and sexual activity, as well as from mother to infant, which was consistent with some of what we learned early on about the epidemiology of AIDS. Finally, the Centers for Disease Control and Prevention (CDC) reported cases of AIDS in patients with hemophilia who had received only filtered clotting factors, which seemed to eliminate the possibility that the agent was a microorganism larger than a virus.*

This set of arguments convinced us, as well as **Max Essex** in Boston, each independently to start a search for **an HTLV-like virus** in patients with AIDS. We began conducting this research at the National Institutes of Health in Bethesda and at the Pasteur Institute in Paris. **The theory that a retrovirus caused AIDS was correct, but the hypothesis that it was a close relative of HTLV proved to be wrong.** In Bethesda, an earlier survey involving the use of molecular and immunologic probes **seemed** to favor a variant similar to HTLV-1 (**the putative cancer virus**). In fact, some patients with AIDS were **doubly infected** with HTLV-1 and the new agent, which complicated **the interpretation** of the nature of the virus causing AIDS. In January of **1983**, **the CDC met with blood banking organizations** in Atlanta to discuss proposals to screen out individuals at high risk for AIDS from the blood donor pool. Self-identification **through questionnaires or interviews** was proposed. In March of 1983, the CDC published guidelines adopted by the PHS **requesting members of groups** having increased **risk for AIDS to refrain** from donating blood [1]. Through such self-identification and questionnaires, the blood supply **was declared**, “cleaned up.”

By May of 1983, the research group of Luc Montagnier published that a molecular marker they called LAV was **associated** with **lymphadenopathy** [3]. The Barre-Sinoussi et al. paper was at first **rejected**. However, the paper was **accepted** and published **with the help of Robert Gallo**, who shepherded the paper through to publication (personal communication), and who as a collaborator maintained close contact with the Montagnier group both before and after the rejection of the paper, as they shared samples, technical training (Barre-Sinoussi received some brief training in Gallo’s lab-Gallo, personal communication), laboratory protocols, and hypotheses regarding AIDS and retroviruses.

Gallo and Montagnier also claimed in their co-written 2003 NEJM article that [2]:

In **early 1983**, a **clear-cut isolate** was obtained **in Paris**, **with the help of interleukin-2** and anti-interferon serum, **from cultured T-lymphocytes derived from a lymph-node-biopsy specimen from a patient with lymphadenopathy**, a syndrome that **was considered** to be a precursor of AIDS. **This virus proved to be different from HTLV in terms of antigenicity and morphology, but it could be propagated only in fresh cultures of T lymphocytes and not in permanent T-cell lines**, which impeded its **full** characterization. The idea that the **causative** agent of AIDS should be sought in swollen lymph nodes **was partly right**, since we now know that lymph nodes are the main site where the virus **hides during the presymptomatic phase**. At this early stage, **it seemed more likely that the isolate was causative than that it was opportunistic**, since the immunosuppression **was very mild**. In some ways, however, it was also a misleading idea that delayed the full characterization of the virus and its mass production for seroepidemiologic studies, **because only some viral isolates from patients with fully developed AIDS grow quickly in permanent cell lines**, as we would soon learn.

*This technical breakthrough was first achieved in late 1983 in Bethesda. Among a few strains in the Bethesda laboratory that grew in continuous cell lines, one came, unbeknownst to both of us, from the third isolate from a patient with Kaposi’s sarcoma in Paris. The origin of the HIV strain with a very high capacity for growth that could readily overcome other HIV strains in culture-and which contaminated cell cultures in several laboratories, beginning with both of ours-was unraveled only in 1991, thanks to the use of the polymerase-chain reaction-technique.*

**“PATIENT ONE” HAD MANY DISEASES BUT NOT AIDS. MONTAGNIER’S “LAV-BRU=”ARC” ASSOCIATION**

## IGNORED COULTER'S HIDDEN LINK WITH SYPHILIS

The Pasteur Institute's *Patient One* had "pre-AIDS." Patient One had sought medical consultation for swollen lymph nodes, muscle weakness **without fever or weight loss**, and for at least two episodes of gonorrhea, **cytomegalovirus**, Epstein-Barr virus, herpes viruses I and II. The year before, he **was treated for syphilis**, which for decades, has been known as "the great imitator:"

The patient will complain of rashes, fever, itching, sore throat, headache, malaise, vertigo, sweating, insomnia, nausea, prostration, weight loss, loss of hair, or aching in the bones and joints. Some have hypertension, kidney disease, swollen liver, or swollen spleen; others have a subacute meningitis with cranial nerve involvement. This stage of syphilis is often confused with such conditions as infectious mononucleosis, iritis, neuroretinitis, lichen planus, cancer, nephritis, dementia, lymphomas, psoriasis and other skin eruptions, and even drug reaction. For this reason secondary syphilis is called the great imitator [4].

The *Treponema pallidum* acts specifically against the thymus gland. The thymus dependent parts of the lymphatic system deteriorate, and there is consequent decrease in the numbers of T-lymphocytes. **The T-helper cells are particularly affected by this: there is a decline in their number and the ratio with the T-suppressor cells is reversed.** Consequently, a long-term effect of syphilis is loss of, or decline in, the system of immunity, and lowering of the individual's capacity to defend himself against other infectious conditions [4].

Although syphilis and AIDS have some completely overlapping symptoms, AIDS was considered a distinct disease entity regardless of the fact that in both syphilis and AIDS:

The T-helper cells are particularly affected by this: there is a decline in their number and the ratio with the T-suppressor cells is reversed [4], and ...the various manifestations of AIDS were unified by a biologic marker: a decrease in the levels of a specific subgroup of T cells that harbored the CD4 surface antigen [2].

The Pasteur Institute's Patient One had also tested positive for antibodies to three different viruses: cytomegalovirus (**CMV**), Epstein-Barr virus, and Herpes [3]. Because of these symptoms, "Patient One" was diagnosed with pre-AIDS or ARC, and not what would be described later as full-blown AIDS. The 1983 Barre-Sinoussi et al. paper describing the characterization of "Patient One's" sera included biochemical data, in vitro culturing data using stimulated primary lymphocytes exposed to Patient One's serum that naturally harbor HERV's (Human Endogenous Retroviral (like) particles), electron microscopy of these co-cultures exhibiting the molecular marker(s) of "HIV" and viral-like particles normally present in umbilical-cord-isolated lymphocytes, and serum derived from "Patient One." The characterization of the sera from this ARC patient (AIDS-related complex) was principally carried out using PHA and IL-2-stimulated primary lymphocyte cultures to which fresh, "uninfected" cord lymphocytes that harbor these HERV's were serially added. This strategy was employed in order to propagate the production of a possible lymphocyte-tropic virus reverse transcriptase signal.

This method to propagate "LAV's" molecular signature was deemed "inefficient" by Gallo, and, during the year, the Gallo group experimented with, and advanced the idea that permanently growing infected **cancer cell lines** would be a more efficient way to amplify the minute molecular signature that Montagnier's group sometimes could detect as their "LAV-BRU's" trace and hardly detectable molecular signature **in their primary lymphocyte cultures** that harbored retroids or HERV's.

By November of 1983, the CDC published formal recommendations for the protection of laboratory and clinical personnel having contact with AIDS patients and clinical specimens. The recommendations were based on those **for hepatitis B** [1].

This is how Luc Montagnier recounted this period in retrospect, as he disclosed in his Nobel acceptance speech in 2008:

"This presentation will be obviously focused to one new epidemic, AIDS, but we should not forget that there are other persisting and life endangering epidemics, especially in tropical countries such as malaria and tuberculosis. Moreover, other new epidemics should not be excluded as human activities generate more favouring factors: > Lack or loss of **hygiene** habits > lack of **water** > **globalisation** and acceleration of exchanges, travels > **atmospheric and chemical pollution** leading to **oxidative stress** and immune depression > **malnutrition, drug abuse, aging**, also leading to immune depression > **global warming** leading to new ecological niches for insect vectors > **changes in** sexual behaviours. This last factor and immune depression caused by **malnutrition, drug abuse** and increased **co-infections**, are **probably** the causes of emergence of AIDS as a global epidemic, affecting most if not all continents including recently Polynesia islands. **The causative agent existed in Africa before the emergence of the epidemics in Central Africa and North America in the 1970s.** As there exists related viruses **apparently well tolerated in non-human primates, it is tempting to consider AIDS as a zoonosis, resulting of the transmission to human of related viruses infecting primate species without causing disease.** But let us first recall the circumstances of HIV discovery in my laboratory at the Pasteur Institute. AIDS as a pathologic distinct entity was first identified in June 1981 by members of the CDC (particularly James Curran) after reports received from two medical doctors, Michael Gottlieb in Los Angeles and **Alvin Friedman-Kien** in New York, of clusters of opportunistic infections and Kaposi sarcoma occurring in young gay men which had **related** sexual intercourse [**Gottlieb said they were unrelated**]. Following publication of this report in the CDC Bulletin, similar cases were described in Western European countries and particularly in France by a group of young clinicians and immunologists led by **Jacques Leibovitch** and **Willy Rozenbaum**. It was soon recognized that **a similar disease**, characterized at the biological level by a profound depression of cellular immunity and clinically by infections previously described in **chemically or genetically immunodepressed patients, also existed in haemophiliacs and blood transfused patients.** The case of haemophiliacs was giving a clue as to the nature of **the transmissible agent**: these AIDS patients had received purified concentrates of factor 8 or 9, made from pools of blood donors which had been filtrated by **bacteriological** filters. This purification process **should have** eliminated any soluble toxic compound [**except foreign proteins-my emphasis**] and the filtration should have retained bacterial or fungal agents: only viruses [**or foreign non-self proteins-my emphasis**] could be present in the preparations given to patients. This is why I become interested in a search for **viruses**; but what kind of viruses? Many viruses have immunodepressing activity, in order to persist in their hosts. This is particularly the case of herpes viruses (cytomegalovirus) and retroviruses. A putative candidate was the Human T Leukemia virus (**HTLV**) described by R.C. Gallo **and** Japanese researchers. Having more expertise on retroviruses, we embarked on the search for an HTLV-like virus, at the suggestion of the French working group and also incited by the Institute Pasteur Production, **an industrial subsidiary of the Institute, producing an hepatitis B vaccine from pool of plasmas from blood donors.** Knowing that retroviruses are **usually** expressed in activated cells, I have set up **classical conditions** to grow in culture **activated** lymphocytes, using first a bacterial activator of both T and B lymphocytes, Protein A, since I ignored in which subset of cells the virus was **hiding out.** The reasoning at that stage was that we should look first in lymphocytes from swollen lymph nodes, supposedly the site where viruses accumulate in the early phase of infection. I received in January 3 a biopsy of a patient with cervical adenopathy, a symptom already recognized as an early sign of AIDS. After dissection of the sample into small pieces and their dissociation into single cells, the lymphocytes were cultured in nutrient medium in the presence of Protein A and anti-interferon serum. In fact, after addition of Interleukin 2, only T lymphocytes were multiplying well and produced **a small amount** of virus detected **by its reverse transcriptase activity**, measured by my associate **Françoise Barre-Sinoussi**. Only some 9 months later could I show also growth of the virus in B-lymphocytes **transformed** by Epstein-Barr virus. The viral growth **ceased** as the cellular growth started declining, but we could propagate the virus in cultures of lymphocytes **from adult blood donors as well as in lymphocytes from chord blood.** This allowed characterization of the virus, and showed for the first time that **it was different from HTLVs. A p24-25 protein could be immuno-precipitated by the serum of the patient and not by antibodies specific of the p24 gag protein of HTLV1, kindly provided by Dr R.C. Gallo.** Electron microscopy of sections of **the original lymph node biopsy**, as well as those from **infected cultured lymphocytes, showed rare viral particles with a dense conical core, similar to the**

**retrotransposons of animals** (infectious anaemia virus of horse, Visna virus of sheep, etc.), **but different from HTLV**. Unlike the case of HTLV, we could **never see emergence of permanent transformed lines** from the infected lymphocyte cultures. These results were published in a Science paper in May 1983, **together with two papers by Gallo and Essex groups in favour of HTLV being the cause of AIDS**. During the following months, more data accumulated in my laboratory showing that this new virus **was not a passenger virus**, but was really the best **candidate** to be the cause of AIDS. (1) The same type of virus was isolated from patients of different origins: gay men with multiple partners, haemophiliacs, drug abusers, **Africans**. (2) Besides immune-precipitation of viral proteins (p25, P18), serums from patients with lymphadenopathy syndrome and **a fraction of the serums from patients with advanced AIDS, were positive in an ELISA test using proteins from partially purified virus**. (3) In vitro, the virus was shown to infect **only CD4+ T lymphocytes** and not the CD8+ subset. (4) **A cytopathic effect** was observed **with isolates made from patients with late symptoms of AIDS**. Particularly the third isolate made from a young gay man with Kaposi Sarcoma (Lai) caused the formation of large syncytia, presumably due to the fusion of several infected cells. **Attempts to grow the first isolate Bru in T cell lines isolated from patients with leukaemia or lymphoma were unsuccessful**. However, we discovered later that **the Bru isolate was contaminated with the Lai isolate, which** by contrast could be grown **in T cell lines (CEM, HUT78)** in laboratories which received our Bru isolate on their request. In fact, a few laboratory isolates were shown to grow in mass quantities in T cell lines, facilitating analysis of the virus and its use for detection of antibodies by commercial blood tests. Our data which I presented **in September 1983 at a meeting on HTLV in Cold Spring Harbor** were met **with skepticism** and only in the Spring of 1984, the description of **a quasi identical virus under the name of HTLV III** by the group of R.C. Gallo **convinced** the scientific community that this **new retrovirus was the cause** of AIDS. The group of Jay Levy in San Francisco also isolated **the same kind of virus**, followed by many other laboratories.”

## **GALLO BECOMES A SHEPHERD AND SCIENTIFIC PEER REVIEW IS AVOIDED**

Despite an initial claimed concordance between the “LAV” and “HTLV-3” signatures in isolates from what were then defined as ARC or AIDS patients, because by accident “a different contaminated isolate” was sent from The Pasteur Institute to Bethesda in 1983 which had “infected” the cultures of the Gallo group, of Robin Weiss’s group in England, and of at least several other laboratories on both sides of the Atlantic, research in these labs was confounded until at least the next year (Robert Gallo, personal communication). Despite the fact that Gallo *shepherded* through the 1983 Barre-Sinoussi et al. paper through to publication because it didn’t pass initial review, the American-French collaboration and patent on the blood test was to be plagued by numerous accusations of stealing, deception, misconduct, and fraud for over a decade.

Shepherding of papers occurs frequently among cooperating scientific groups, and such papers are not rigorously “peer-reviewed.” The interests of the U.S. and French governments regarding the patenting of a blood test, fear of contagion, religious views regarding morality, sex, and drugs, and politics, began an era that perhaps should be characterized as the most politicized disease in medicine’s history. And even though it was finally concluded that there was no misconduct in the Gallo/Papovic cases, the numerous accusations, and the lengthy and costly investigations of misconduct and blame only served to polarize those involved and those critical of them, and the charges of wrong doing only fueled bitter rivalry and criticism that has lasted for more than two decades. Robert Gallo recounted how the Nobelist and Director at that time of the Pasteur institute, Jacques Monod, warned him in a French café that if the patent royalties brought by the test kits wasn’t shared with the Pasteur, that there might be hell to pay, as disclosed in Nikolas Kontaratos’s ghost-written account mentioned earlier (<http://dissectingadiscovery.com/>) (also see <http://exlibhollywood.blogspot.com/2009/05/doctors-without-boundaries.html> ).

## **GALLO’S “36% CAUSALITY:” “HIV” CAUSES AIDS!**

An articulate analysis was presented in retrospect by *The New AIDS Review*,<sup>7</sup> on the 1984 May 4<sup>th</sup> Gallo Science paper: “**Frequent**



detection and **isolation of cytopathicretroviruses** (HTLV-III) from patients with AIDS and at risk for AIDS...” As reviewed by Michael Geiger, Executive Director of HEAL San Diego (Health Education AIDS Liason):

*Gallo didn't find HIV in 48 out of samples from 48 AIDS patients, as he keeps implying. He found whatever he took as the signature or signal for it in 26 out of 72 samples of AIDS patients, which is a rather different 36%. The rest of the 48 came from **pre-AIDS** patients (who may have had nothing but **a cold**) and three mothers (who were perfectly **well**) and **1 clinically well gay man out of 22** (who later developed AIDS). **119** in total from which **48** scored positive.*

*Robert Gallo's claim that HIV is the cause of AIDS was first put forward on the basis of four papers he published in Science; May 4 1984 issue.<sup>8</sup> A press conference at the time, arranged and conducted by Margaret Heckler, then Secretary of Health and Human Services for **the Reagan administration**, claimed that his work in the soon-to-be-published papers had shown that HIV was the “**probable**” cause of AIDS.*

*The New York Times (EIS-Epidemiology Intelligence Service trainee Lawrence K. Altman) reported the news of the claim but **his article contained six or seven caveats to the effect that the claim might not bear out.***

*When the papers were published for all to see, it turned out to be insufficient to demonstrate the claim. Gallo had found the virus in too few of the AIDS patients with actual AIDS symptoms – only 26 out of 72, or **36%** – to substantiate his claim. He was unable to demonstrate the presence of the virus in two thirds – 64 per cent – of the AIDS patients sampled.*

*Here are the figures as shown in Table 1 of the paper “**Frequent Detection and Isolation of CytopathicRetroviruses (HTLV-III) from Patients with AIDS and Risk for AIDS**”, Robert C. Gallo, Syed Z. Salahuddin, Mikulas Popovic, et al, Science, May 4, 1984: 224:500-502):*

**Group Diagnosed: Number positive for HTLV-III/Number tested/Percent positive:**

**Pre-AIDS: 18/ 21 85.7%**

**Clinically normal mothers of juvenile AIDS patients: 3/47 5.0%**

**Juvenile AIDS: 3/ 8 37.5%**

**Adult AIDS with Kaposi's sarcoma: 13/ 43 30.2%**

**Adult AIDS with opportunistic infections: 10/ 21 47.6%**

**Clinically normal homosexual donors: 1/ 22 4.5%**

**Clinically normal heterosexual donors: 0/115 0%**

*Or as noted in the article:*

*As summarized in Table 1, we found HTLV-III in 18 of 21 samples from patients with pre-AIDS, from three of four clinically **normal** mothers of juvenile AIDS patients, 13 of 43 adult AIDS patients with Kaposi's sarcoma, and 10 of 21 adult AIDS patients with opportunistic infections.*

This result partly veiled the stark failure of the sampling to identify persuasively HIV as a cause of AIDS. For the sum total of AIDS patients with symptoms of AIDS – the groups in bold in the table above – was that in ONLY 26 (3 +13 +10) out of 72 (8 + 43 + 21) cases was the Gallo lab able to show HTLV-III virus detected and isolated.

26 of 72, or **36%**, was insufficient to demonstrate that HIV was the plausible cause of the AIDS symptoms or their underlying immune deficiency. If anything, the testing demonstrated that HTLV-III was certainly **not a plausible cause** of AIDS.

‘These studies of HTLV-III isolates from patients with AIDS and pre-AIDS and from healthy individuals at risk for AIDS provide strong evidence of a causative involvement of the virus in AIDS.’

Thus contrary to subsequent headlines the paper did not state firmly that HTLV-III (later renamed Human Immunodeficiency Virus) was the cause or even a “probable” cause of AIDS, only that there was evidence of a “causative involvement” of the virus in AIDS.

In another paper of the four (Sarngadharan MG, Popovic M, Bruch L, et al. Antibodies Reactive to Human T-Lymphotropic Retroviruses (HTLV-III) in the Serum of Patients with AIDS. *Science* 1984;224:506-508), however, the claim was bolder:

‘The data presented here and in the accompanying reports suggest that **HTLV-III is the primary cause** of AIDS.’

**The low figure** of only **36%** of AIDS patients with symptoms that had HTLV-III virus present **was excused** in an accompanying news report in a *Science Research News* column by Jean Marx as possibly due to **deterioration** of the samples.

When the investigators calculated the percentage, they used the total of all the AIDS samples sent to them, **even though some had deteriorated** to the point where they were of questionable value for analysis.

The paper itself had noted:

The incidence of virus **isolated** reported here probably underestimates its true incidence since many tissue specimens were not received or handled under what we now recognize as ideal conditions. **This is particularly so for the samples received from late stage AIDS patients.**

That is to say, 26 of the 72 with AIDS tested positive for the virus, 22 of the 47 who did not have AIDS (asymptomatic or pre-AIDS- i.e mild and non AIDS specific symptoms), suggesting that **HTLV-III positivity was a poor guide as to who would develop AIDS symptoms. Later, this problem was solved by counting as AIDS only those who were HTLV-III positive.**

To put it even more bluntly, the paper’s landmark finding is the reason why Gallo is forced to leave the number “forty-eight” standing out there in his letter all by itself, not saying 48 out of how many, because in fact he **found the virus in blood** from 48 individuals out of 119, and 22 of the 48 did not have AIDS, rather than all of them being “individuals who had AIDS.”

Despite these interpretations of the Gallo et al. results, it still wasn’t clear and certainly not explained why only a minority of patients with “full blown AIDS” exhibited the “HIV” molecular markers, but, as Gallo and Montagnier stated [2], it could have been, in addition, because, as it was claimed:

“...only some viral isolates from patients with fully developed AIDS grow quickly in permanent cell lines, as we would soon learn,”

suggesting that other strains might grow slowly, or not at all, depending on if primary lymphocytes were used, or cancer cells, or perhaps other cells. Again we find that “the soil” may be more important than “the seed,” in many cases.

## **THE GONDA LETTER SAID THERE WAS NO “HIV” (HTLV-I, II, OR HTLV-III) IN THE POPOVIC/GALLO SAMPLES**

Thus many scientists, doctors, and leaders of gay organizations were initially highly critical of the strength of Gallo’s group’s correlation of the complex disease syndromes with which Montagnier’s group and Gallo’s group claimed these minute traces of greatly amplified molecular markers were associated. Not only that, but the month preceding Science’s April publication of Gallo’s 4 “etiologically” publications he claims established the link between “HIV” and AIDS, the first of many attempts to demonstrate the so-called “extremely fragile” “HIV” viral particles failed, as was revealed in an NIH interdepartment memo, brought out into the open during later Congressional misconduct hearings, Health and Human Services hearings, and Office of Scientific Integrity investigations of Gallo’s relationship to the Pasteur group’s published findings, and which was sent to Dr. Gallo by the head of the EM facility, Mathew Gonda. This document is particularly significant because it was apparently sent to Dr. Gallo in the week(s) before the Heckler-Gallo Press release and the Papovic/Gallo et al. publications in Science:

Dr. Mica Papovic March 26, 1984 (Received March 27):

Laboratory of Tumor Biology

NIH

Building 37, Room 6B22

Bethesda, Maryland, 20205

Dear Mica

I am sending you 4 extra copies of results requested by Betsy Read. She said Dr. Gallo wanted these micrographs for publication because they contained HTLV particles. If this assumption is based on the cultures being antigen positive, I would like to point out that **the “particles” in micrograph 0905 are in debris of a degenerated cell.** No other extracellular “virus-like particles” were observed free between cells anywhere in the pellet. The small extracellular vesicles in 0904 are at least 50% smaller than HTLV mature particles seen in type I, II, or III. **Again, these vesicles can be found in any cell pellet. I do not believe any of the particles photographed are HTLV I, II, or III.**

cc Robert Gallo Best regards,

Betsy Read Mathew Gonda, Ph.D

Head, Electron Microscopy Laboratory

It should be pointed out, however, that in the Kontaratos book Gallo sent to me, Gonda is portrayed in a positive light, as the electron microscopist who first accurately named “HTLV-3B” (“HIV”) a lentivirus.

In November, of 1984, the Pasteur Institute investigators published the genetic sequence of LAV [1],

### **ENTER GOVERNMENTS, PATENTS, ALLEGATIONS OF MISCONDUCT AND FRAUD**

In April of **1984**, amidst a political climate of Reaganomics, Gay Liberation, extreme urgency, SDI (The Strategic Defense Initiative) and fear, and to assure the public that government scientists were attempting to protect the Nation’s blood supply from spreading a potential Gay Plague or African-derived microbe, HTLV-III (later called “HIV-1”) was introduced at a press conference given by Margaret Heckler, as being “a variant of a known human **cancer virus**,” and it was announced that “HTLV-III” was “**the probable cause of AIDS.**” In May, and in the months following the press release, four papers from Dr. Gallo’s laboratory claiming that the HTLV-III retrovirus was the cause of AIDS were published in Science. These are the 4 papers that established that “HIV’s” signature was present in “bona-fide AIDS patients” rather than only ARC patients, and constituted the experimental basis of the hypothesis that “HIV” causes “AIDS.”

The initial description and press release by Margaret Heckler of “HIV” as **a known variant of a cancer virus** in retrospect was perhaps unfortunate, because as stated by Gallo and Montagnier [1], the molecular profile that was associated with “HIV,” was derived using the same techniques employed to characterize the molecular signatures of Gallo’s previous **putative cancer viruses**, “HTLV-I” and “HTLV-II.” But the Gallo team claimed that “HTLV-III (“HIV”) was distinctly different from his leukemia-associated “cancer causing” retroviral signatures of “HTLV-I” and “HTLV-II.”

The patent and intellectual property issues of the U.S. and French governments surrounding the creation of an “AIDS” blood test to protect the blood supply also proved to be an unfortunate series of confusing and hateful events that made possible accusations against the American-French collaboration that virtually insured suspicion of the “HIV=AIDS” hypothesis, and served, it could be argued, to derail progress toward defining Gottleib’s identification of anergy, and profound immune suppression.

### **THE FAILURE OF FEAR, THE BLOOD SUPPLY, CLOTTING FACTORS.**

By **1985**, it was published that transmission of AIDS amongst health care workers was not a major public health concern because no transmission of AIDS was reported [5]. Health care workers aren’t good “soil” either, and don’t exhibit full-blown AIDS very often. Or maybe it was all statistical artifacts?

Also, in **1985**, after a year of “HIV” testing, it was published that “**68% to 89% of all repeatedly reactive ELISA (HIV antibody) tests represent false positive results among sperm donors**” [6].

Several other important events that occurred in 1985 included the fact that in January, the **NCI scientists and their collaborators published the genome of HTLV-III in Nature**, and in March, the first AIDS antibody test, an ELISA-type test, was released. In June, the CDC revised the case definition of AIDS to include additional specific disease conditions and to exclude people as AIDS cases if they had a negative result on testing for serum antibody to HTLV-III/LAV. In July, United Press

International reported that actor **Rock Hudson** had AIDS, and in September, Indiana teen **Ryan White**, a hemophiliac said to be suffering from “AIDS,” **was refused entry to school**. Also in September, the U.S. military services began testing for the AIDS virus among its personnel, and in October, Rock Hudson died. He was the first major public figure to die of AIDS, and Public fear about AIDS increased dramatically. That December, the publication of a finding that the AIDS virus is present in saliva increased public fears even more regarding AIDS [1].

## **DRUGGING BEGINS WITH SURAMIN, HUNDREDS OF DRUGS ARE SCREENED, AND COMPOUND “S” (AZT) IS FIRST USED IN 1985, AND PUSHED THROUGH BY SAM BRODER.**

News stories from the mid-1980’s provide valuable accounts pertaining to the growing iatrogenic effects of hurried drug trials. Two stories by reporter Marilyn Chase of the *Wall Street Journal* are worth citing as she writes of the AIDS’ drug experiments in 1985. Her *Wall Street Journal* articles (August 5, and September 26, 1985) document in detail, in addition to Dr. Abrams San Francisco accounts, how AZT and many other fiercely toxic drugs were in NCI trials in mid-1985 in clinics on their campus in Bethesda, Maryland. Chase’s articles cite how AZT, code named “Compound S” and supplied by Burroughs Wellcome, was first tried experimentally in 1985 on suspected AIDS patients. This “push” to dose gay men with a largely untested by failed cancer drug was directed by Dr. Sam Broder in a secret meeting (attended by former Nature Biotechnology Founding Editor Dr. Harvey Bailly, personal communication), who from the first was one of the most prominent supporters of drug therapy for AIDS patients throughout the growing epidemic.

Martin Delaney, the founder in the early 1980’s of the AIDS group *Project Inform*. espoused fast drug treatment trials, and is said to have smuggled AIDS drugs into the U.S. illegally to distribute to AIDS patients, and he Delaney recorded that 5000 people being supplied with AZT (Compound S) in 1985 by the National Cancer Institute employing the Treatment IND approach (please see our correspondence in the DIALOGUES WITH THE PROMOTERS OF AIDS). Thus it is documented that AZT was being ingested long before its actual FDA approval in 1987 and its now undisputed terrible toxicity unwittingly spurred on the panic:

**Subject: Will AIDS Drug Will Be Cleared for Tests in U. S. in Fall? Date: Published: 7/30/85, Source: Wall Street Journal.**

*French Firm Hopes **Possible** AIDS Drug Will Be Cleared for Tests in U. S. in Fall. By Marilyn Chase, Staff Reporter*

*A French drug company said it hopes to begin “limited clinical trials” this fall **in the U.S.** of the experimental drug HPA-23, **which has drawn ailing film star Rock Hudson and other Americans to Paris for treatment** of acquired immune deficiency syndrome.*

*The FDA said it granted HPA-23 “**orphan drug**” status July 17, a designation given when a company believes a drug may be **unpatentable, unprofitable or hard to test**. This confers **special tax incentives** for research and also gives the manufacturer “**a patent-like, exclusive marketing position,**” an FDA spokesman said.*

*Meanwhile, the National Cancer Institute said that in recent months it has screened more than 100 drugs for possible testing on AIDS patients. **Testing on humans has begun for two of the most potent drugs: suramin, produced by Bayer AG of West Germany, and a new drug code-named “compound S,” made by Burroughs Wellcome Co.** U. S. researchers so far, however, have **almost no first-hand knowledge of the drug, how it works, or its possible toxic side effects.***

***Murray Gardner**, chairman of pathology at the University of California at Davis and the only U. S. scientist known to have conducted research on HPA-23, described his experience with the drug as “**inconclusive.**” Dr. Gardner said he received a small amount of the drug from the Pasteur Institute last year and was able to perform only a very brief test **on rhesus monkeys with***

**AIDS. “We didn’t cure the animals,”** he said. “We don’t know whether it was because we didn’t have enough drug **or what.**”

Such uncertainties among U. S. researchers, however, haven’t stopped Paris from becoming a mecca for AIDS victims seeking treatment with the substance. More than **11,000 cases** of AIDS have been reported in the U. S. since the disease was first identified in 1981, and **about half of those patients have died [1981 was 4 years before this article]**. AIDS, which breaks down the body’s immune system, occurs most frequently in homosexuals, intravenous drug users, and hemophiliacs. The disease is spread through **sexual contact and** contaminated blood.

HPA-23 inhibits the enzyme needed for the AIDS **virus to reproduce**. In a paper published months ago, **Luc Montagnier and Jean-Claude Chermann of the Pasteur Institute reported on four patients in whose blood the AIDS virus couldn’t be detected after treatment with HPA-23**. One of the patients, **a teen-age hemophiliac with AIDS, was reported to have felt well enough to return to school after treatment**.

But Drs. Montagnier and Chermann **also reported that three of the four patients developed thrombocytopenia**, a potentially deadly side effect in which the blood’s platelet count plunges, **sometimes** requiring that treatment be discontinued.

The chief antiviral drug currently being tested in the U. S. is **suramin**, a drug used for decades against **African sleeping sickness**. Suramin is being tested in about 40 patients at the National Cancer Institute and in experiments at several university medical centers. Samuel Broder, associate director for clinical oncology at the cancer institute, has said that suramin, like HPA-23, blocks the enzyme needed for the AIDS virus to reproduce. So far, **suramin has lowered the level of the AIDS virus in the blood, but without curing clinical symptoms of the disease**.

Also, the cancer institute recently began the first phase of safety studies of Burroughs Wellcome’s “compound S.” So far, only three patients have received the drug. Burroughs Wellcome, a pharmaceutical concern based in Research Triangle Park, N. C., is a unit of Wellcome Foundation Ltd. of London. The substance, developed as part of a collaboration **between government and industry**, is believed to work by blocking reproduction of **the virus**.

**“HIV” IS VERY “FRAGILE: BUT HEMOPHILACS GET AIDS ANYWAY DESPITE SOAP, DEHYDRATION, AND CRYOPRESERVATION?**

By **1986**, CDC and other agencies were continuing to explore how infectious “HIV” could be – if it could be transmitted casually, through insect bites, or through saliva, or simply from a drop of saliva on a kitchen table. From all of these studies it was concluded that **even huge amounts of “HIV” are very fragile**, and its activity is completely abrogated by detergent exposure for a few seconds, even:

*...with an initial infectious titer of approximately  $7 \log_{10}$  tissue culture infectious, dose (TCID<sub>50</sub>) per milliliter is undetectable and reduced more than  $7 \log_{10}$ TCID<sub>50</sub> within one minute 0.5% solution of nonidet-P40[7].*

Nonidet-P40 is a weak detergent used widely in many laboratories, and this implied that simple methods of sterility and precautions could be used so as to not become infected in laboratories, or in nosocomial (hospital) situations.

These edicts clashed, however, with the assumed infective potential of factor concentrates for Hemophiliacs. And although Gallo claimed in his NEMJ article he wrote with Montagnier that **(2)**:

...the Centers for Disease Control and Prevention (CDC) reported cases of AIDS in patients with hemophilia who had received only filtered clotting factors, which **seemed** to eliminate the possibility that the agent was a microorganism larger than a virus... it was

not clear how “HIV” could indeed maintain its infectivity after the freezing and thawing and lyophilization procedures used to make Factor VIII and IX concentrates, and because it was claimed by many “advocates” that “HIV” couldn’t be preserved and photographed in an electron microscope because – it was so fragile.

In retrospect, and after thousands of PCR “isolations” in the 1990’s, it is likely that the apparent “fragility” of “HIV” found in tests designed to test its infectivity after freezing, thawing, drying, exposure to light, etc., precluded that its infectivity couldn’t possibly survive the Factor XIII and IX preparation procedures of that day. Nevertheless, “HIV=AIDS” proponents suggested that the reason these concentrates might still be pathogenic after months or years of drying, freezing, and thawing, and cause “HIV-infections” in hemophilic Factor XIII and IX recipients, is because “HIV” was imagined to be **concentrated** in these pooled sera derived from tens of thousands of blood samples (Robert Gallo, personal communication).

However, simple logic based on diffusion and dilution argued against the idea that the virus was concentrated in pooled factor preparations precisely because factor XIII and IX concentrates were pooled from thousands of blood donations. If it is assumed that one “HIV-positive” blood sample may contaminate thousands of non-infected ones, even if that one “infected” sample has “high viral load” amongst the pooled several thousand negatives, then by pooling the blood, any “HIV” would be diluted, rather than concentrated. This contradiction needed to be addressed, but wasn’t, in order to explain how so many hemophiliacs who were said to become “HIV-positive” through factor XIII and IX concentrates, acquired their immune-suppressive states. The Public Health Services ignored (and still ignore) the fact and logic, that the factor concentrates, because they were pooled from so many individuals, were loaded with foreign proteins from thousands of different human beings, constituting perhaps an ideal concoction to provoke autoimmune conditions in many recipients. Foreign proteins recognized and attacked by the recipients’ immune systems has even been used as a simple strategy to immune suppress transplant patients in order to avoid highly immunotoxic drugs such as cyclosporin used to prevent transplant rejection. Also, despite the filtering of the concentrate, scientists assumed that only particles the size of viruses can pass through these filters. However, a far more serious threat to factor VIII and IX recipients were the potential “cytokine-storm”-provoking foreign proteins. This hypothesis would be consistent with the fact that whatever the agent was that is associated with the transfer of “HIV’s” molecular signature under such harsh conditions of freezing, drying, thawing, etc., it could survive freezing and drying in a vacuum, and it was small enough, like proteins, to pass through the filters during the clotting factor preparation process.

## **ROBIN WEISS SAYS “HIV” ATTACKS BRAIN CELLS?**

By **1986**, another key paper appeared in the prestigious journal *Cell*, suggesting that “HIV” tropism wasn’t directed only at CD4 lymphocytes, as Gallo and Montagnier had proposed in their “HIV=AIDS” hypothesis. Instead, it was suggested that the expression of the normal cellular protein, T4, by any and all human cells, was exploited by “HIV” as a receptor molecule that enabled entry of the virus into any cell expressing the normal endogenous Human T4 protein. This included microglia, perhaps brain cells, and epithelial cells (skin cells) that expressed the T4 protein, and this molecular species was suggested to explain such diverse effects as immune suppression and AIDS dementia. As stated by Paul Jay Maddon, Angus G. Dalgleish, J. Steven McDougal, Paul R. Clapham, Robin A. Weiss, and Richard Axel (*Cell*, Vol 47, 333-348, 7 November 1986):

The T4 gene encodes the AIDS virus receptor and is expressed in the immune system **and the brain**.

*The isolation of clones encoding the human surface protein T4, and the expression of the T4 gene in new cellular environments, have enabled us to examine the role of this protein in the pathogenesis of AIDS. Our studies support a mechanism of AIDS virus infection that initially involves the specific interaction of the AIDS virus with T4 molecules on the cell surface. This association can be demonstrated on T4+ transformed T and B lymphocytes as well as epithelial cells. **Furthermore, the presence of T4 on the surface of all human cells examined is sufficient to render these cells susceptible to AIDS virus infection. Our data suggest that the T4-AIDS virus complex is then internalized by receptor-mediated endocytosis. Finally, we***

*find that the T4 gene is expressed in the brain as well as in lymphoid cells, providing an explanation for the dual neurotropic and lymphotropic character of the AIDS virus. In this manner, a T lymphocyte surface protein important in mediating effector cell-target cell interactions has been exploited by a human retrovirus to specifically target the AIDS virus to populations of T4+ cells.*

This claim by Weiss's group freed the "HIV=AIDS" hypothesis from the somewhat limited constraints of retrovirological thought, in that no longer was cell activation and then cell division needed, or indeed any specific cellular receptor or cell cycle dependence associated with immune cells (such as the CCR5 receptor), if indeed the T4 receptor was yet another gate through which "HIV" passed into all T-4-expressing cells of the body, do it's retroviral dirty work in glia or perhaps even neurons that express this receptor to cause "AIDS dementia" (which is indistinguishable from dementia due to late-stages syphilis and other forms of dementia). Montagnier's PHA-stimulated yet "inefficient production" of "LAV's" molecular profiles, and Gallo's tumor-cell "amplifications" of what he believed was "HIV" both depended upon activated, cycling, dividing lymphocytes or lymphoma H9 cells In Vitro, respectively. If indeed neurons (or glia) were susceptible to "HIV infection" through receptor-mediated endocytosis, as Weiss claimed because of his finding of "the T4 receptor's" role in this process, cell division could not be the means to propagate the pro-retroviral genome of "HIV" from cell division to cell division in the neuronal population, because human central neurons don't divide (except for some in the olfactory lobe it is claimed), and glia divide only rarely. In theory, only glial cells could therefore propagate a 5-10 year dormant and latent infection from cell generation to cell generation, since they do continue to divide occasionally. "HIV" supposedly, according to retrovirology experts, requires cell division to propagate, because after the "virus" inserts its genetic code after reverse transcription into target cell DNA, it therein stays put for about a decade as a latent gene sequence and replicates as the DNA of the cell is synthesized. That non-dividing neuronal brain cells or rarely dividing glial cells now became the targets of the effects of this seemingly supernatural virus through a common ubiquitous T4 receptor meant that the nucleic acid sequence of the "HIV" DNA, once incorporated, sleeps for maybe 5-10 years, and then the nucleic acid sequence becomes completely and mysteriously unmasked at some point years after infection to produce viral proteins, and all of the components necessary for productive cell-to-cell infection. The years long "dormancy" of a virus thought to require cell division, together with its supernatural ability to "wake up" and produce the necessary proteins for viral production years after infection, requires that the supernatural virus has essentially two pathways in its life cycle: one that propagates along with cellular DNA, and another that, once incorporated into the host cell DNA, becomes completely unmasked (demethylated perhaps?) to produce all of the proteins required to produce a horizontal infection that is pathogenic and infectious among non-dividing neurons and other cells. Similar difficulties would later be postulated with other AIDS-indicator syndromes such as anemia, another "AIDS-indicator" syndrome in which it was thought "HIV" played a significant role by exploiting yet another cell type (the human red blood cell) known not to contain a nucleus once it was differentiated. Yet the expert "virologists" ignored a century or more of basic cell biology as they would assume that in addition to neurons, the "HIV" DNA could become incorporated into a cell without a nucleus. And according to these expert virologists, another cell type supposedly affected pathologically by "the AIDS virus," were skeletal (striated) muscle cells which also don't divide after they form muscle (but regenerate using satellite stem cells), to exhibit what the AIDS industry calls "wasting syndrome" or what had been known for decades in Africa as "slim disease." Once the door was opened by the postulation and acceptance of the T4 receptor's role in "HIV's" receptor-mediated endocytosis that is ubiquitously associated with many cell types, the horse was out of the barnyard gate with respect to "HIV's" supernatural abilities. Who could now believe, as Socrates suggested, in horses activities without also believing in horses?

#### **HAROLD VARMUS CALLS MONTAGNIER'S "LAV-BRU" AND GALLO'S "HTLV-IIIB "HIV."**

In **1986**, Harald Varmus chaired the scientific advisory committee that proposed the name human immunodeficiency virus (HIV) for the etiologic agent of AIDS.

Also in **1986**, a disinformation campaign became associated with "Patient Zero." The idea disseminated in the mass-media that a



young, gay Canadian airline steward named Gaetan Dugas first introduced the AIDS virus into the homosexual population of Manhattan, and was first advanced by the late San Francisco writer Randy Shilts and given further credence by coverage on the CBS television news program, 60 Minutes.

In his book, *AIDS and the Doctors of Death*, Hollywood dermatologist and cancer researcher Dr. Alan Cantwell states that a large number of homosexual men in New York, City had the disease before Dugas was ever diagnosed. “As evidence for this, we know that Cladd Stevens and her group from the New York City Blood Center have traced the first and earliest known ‘positive’ AIDS virus antibody test back to young Manhattan gays who were injected with the hepatitis experimental vaccine at the Blood Center beginning in November 1978.”

*“Re-examined blood specimens taken during 1978-1979 show ‘positive’ AIDS virus antibodies in 6.6% of those men injected with the experimental hepatitis vaccine (Journal of the American Medical Association, Volume 255, pp.2167-2172,1986).*

### **CASE SHAKES THEORY OF AIDS ORIGIN**

Chicago Tribune (CT) – Sunday October 25, 1987, John Crewdson, 1987 Chicago Tribune. *ST. LOUIS* –

*Long before Robert R. finally entered the hospital, his body had begun to fail him in many ways.*

***For nearly two years his lower legs and genitals had been swollen. Since then the black teenager had grown thin and pale, fatigued and short of breath, and now his bloodstream swarmed with the microbe called Chlamydia.***

*Just when Robert’s condition seemed to have stabilized, his breathing became more labored and his white blood cell count began to plummet. He developed a fever, went into a convulsion and died.*

*The parade of doctors who examined the young man in life, who poked and prodded and photographed him for their archives, agreed that Robert’s immune system had somehow ceased to function. But none of them could offer a clue as to why.*

*None, that is, until Dr. William Drake, the pathologist who performed the autopsy, discovered something odd: a small, purplish lesion on the boy’s left thigh, and several similar growths in the soft tissue inside his body.*

*In his autopsy report, Drake concluded that the lesions were a malignant tumor called Kaposi’s sarcoma, a rare brand of cancer once confined mostly to elderly Jewish and Italian men.*

*According to contemporary diagnostic criteria, Kaposi’s sarcoma in a patient younger than 60 **is almost certain to signal a case of acquired immunodeficiency** syndrome [unless one is an old Jew or Italian man-emphasis mine]. But on May 16, 1969-the day that Robert died-nobody had ever heard of AIDS.*

*The doctors who attended Robert R., (and who agreed to talk about the case in exchange for an agreement to withhold his last name) and for whom his case has presented a continuing puzzle, now believe the **15-year-old youth from the St. Louis ghetto** was infected with the same human immunodeficiency virus (HIV) that has since been linked to AIDS.*

*If they are correct-and laboratory evidence obtained just last week indicates strongly that they are-it means the AIDS virus has existed in this country **for at least two decades**, a full 10 years before the first cases of AIDS-related Kaposi’s sarcoma began showing up in white, male homosexuals in New York City.*

The implications of such a conclusion are profound, for the length of time that the AIDS virus has been present may not only determine how many Americans have been exposed to it but reveal much that so far is unknown about the past and future course of the disease.

At the moment, however, the case of Robert R. raises more questions than it answers. From whom did he acquire the AIDS virus, and how? To whom might he have passed it? Most important of all, when did the AIDS virus arrive in this country, and where did it come from?

Before he died, Robert was unable to contribute much to the solution of the mystery that surrounds him. "He was the typical 15-year-old who is not going to talk to adults, especially when I'm white and he's black," said Dr. Memory Elvin-Lewis, a microbiologist at Washington University in St. Louis who followed Robert's decline for more than a year.

"He was not a communicative individual. He knew the minute I walked into the room that I wanted something more from him—more blood, more lymph fluid, more something."

Between extractions and injections, **Robert did tell his doctors a few key facts: that he had been born in St. Louis and had never traveled outside the Middle West, much less the country. Nor, he said, had he ever received a blood transfusion.**

He also admitted having had **heterosexual** relations; according to his autopsy report, "the patient dated his physical disability from an instance of sexual relations with **a neighborhood girl.**"

Robert was never asked about the possibility of homosexuality, but **circumstantial evidence suggests that he may have been the recipient of anal sex**, the variety of intercourse believed most likely to transmit HIV.

"We knew from the very first that he wouldn't let us do a rectal examination on him," recalled Dr. Marlys Hearst Witte, a professor of surgery at the University of Arizona who was closely involved with the case of Robert R.

"We knew that he had **genital edema and severe proctitis**, which is an unusual problem in a 14-year-old boy—the stigmata, **almost, of homosexuality.** At autopsy he had Kaposi's sarcoma of the rectum and anus, which is an unusual place for Kaposi's sarcoma to be.

"So if you're asking me, do I think this boy lived in an environment or engaged in practices that one would now associate with transmission of AIDS, I would say I think that was rather likely. **He could have been a male prostitute. He certainly lived in the environment where that was possible.**"

However Robert acquired the virus, he must have gotten it from **someone, since no viruses can exist for long outside the human body.** And whether he passed it on or not, the presence of HIV in this country as early as 1968 raises important questions about the current thinking on the genesis of the virus.

**Most researchers** now believe that HIV assumed its present shape somewhere **in Central Africa** and arrived in this country during the middle 1970s. **The theory is bolstered by the discovery, two years ago, of HIV antibodies in a blood sample dating from 1959 in Kinshasa, the capital of Zaire.**

Because the incidence of AIDS **in Haiti** is high, and because some of the first cases in this country occurred among Haitian emigres **in Florida**, **it has been assumed** that the virus probably passed through that island nation on its way **from Africa** to

the United States.

One theory suggests that French-speaking Haitians, imported to Zaire and other French-speaking African nations **as servants** during the 1960s and 1970s, brought the virus back to Haiti, where it was picked up by **vacationing American homosexuals** in the mid-1970s.

Another holds that HIV first came ashore in south Florida with the successive waves of **Haitian boat people** who began landing there in 1978.

But however it is constructed, there are a number of gaps in the Africa- Haiti theory. **One is that the per capita incidence of AIDS in other Caribbean nations, including the Bahamas, Barbados and Bermuda, is even higher than in Haiti.**

Another is the question of why, **since nearly equal numbers of Haitian men and women appear to be infected with HIV**, the virus was not also acquired by heterosexual American tourists in Haiti-or, for that matter, in Miami.

If some other explanation for the passage of HIV to the United States must be constructed on the strength of Robert R's case, an explanation will also have to be found for the fact that white male homosexuals, who make up two-thirds of all AIDS victims, did not begin to sicken and die in large numbers until the late 1970s.

So perplexing was the case of Robert R. that two of the doctors who attended his autopsy took samples of his blood and tissue back to their laboratory freezers, along with the faint hope that science might someday tell them what to look for.

One was **Dr. Elvin-Lewis**, then a newly minted Ph.D. who had just finished a doctoral dissertation on a little-known sexually transmitted disease named, like the microbe, Chlamydia.

"He was my first patient," Elvin-Lewis recalled in a recent interview, "and I couldn't believe what I was seeing. He was a bag of producing Chlamydia. His antibodies were so low that nobody could understand it."

**"The case sure was consistent with some kind of virus knockdown of the immune system,"** said Drake, the pathologist, who is now retired. "The Chlamydia, for instance, shouldn't have been in his blood. Chlamydia should stick to the site where it enters the body."

Another who watched Robert's progress with great interest was Marlys Witte, then a young thoracic surgeon who, with her physician husband, Charles, had become intrigued by the apparent **obstruction** of the boy's lymphatic system.

"When he died, Marlys and I just stood there and took everything," said Elvin-Lewis. "Blood and lymph and tissue and you-name-it."

For two decades the samples were kept in cold storage, some of them in Elvin-Lewis' laboratory in St. Louis, the others by Witte at the University of Arizona.

The case remained sufficiently perplexing that Elvin-Lewis, the Wittes, Dr. William Cole and some of the others involved reported the enigma in a 1973 medical journal article. And there it might have ended, as the co-authors followed their separate career paths to teaching and research.

Elvin-Lewis became chairman of the microbiology department at the Washington University Dental School. The Wittes moved to

Tucson. Cole gave up his post as chief of surgery at Barnes Hospital in St. Louis to open a small- town practice in Sedalia, Mo., and Drake went on doing autopsies.

*But the case of Robert R. stayed in the back of Marlys Witte's mind. "I'm not someone who's devoted my whole life to AIDS," she said in a recent interview. "This was an incidental patient, coming in with something I deal with on a regular basis-lymphedema. But I have always thought this was an important case, and I did the best thing. I saved everything."*

*In 1984, as AIDS was moving to the forefront of American medical research, Witte decided that some of Robert R.'s samples should be thawed and tested.*

*Antibodies were found to **Herpes simplex, Cytomegalovirus and Epstein-Barr disease**, three viruses that, **along with bacterial Chlamydia, are common among homosexual men, especially those with AIDS.***

*But there was then no way to test for antibody to HIV, which represents **nearly irrefutable** evidence of **exposure** to the virus.*

*"I thought that I would just sit until techniques were better, so that I would have my best shot at really documenting it," Witte said. "We felt we had so little fluid that we were going to save what we had and do everything at once."*

*Early **last year**, after sensitive HIV **antibody** tests had become available, Witte called Elvin-Lewis to ask whether she had saved any of the samples she took from Robert R.*

*"She turned out to be as much of a pack rat as I am," Witte said. "So I said, 'Send me everything you've got.'"*

*In June, Witte sent a half-teaspoon of Robert's blood and a few specks of tissue to Dr. Arthur Gottlieb, a friend and colleague who heads the microbiology department at the Tulane University Medical School in New Orleans.*

*"I thought that things were at a stage where, if there was going to be something to be found, we would be able to find it now," Witte said.*

*The decision to test Robert's remains for HIV was first disclosed by Witte, who **is secretary general of the International Society of Lymphology**, in the opening address to a special AIDS seminar last month at the 11th International Congress of Lymphology in Vienna.*

*There she told an astonished audience, "We are currently testing body fluids and tissues preserved for nearly 20 years for evidence of HIV or related retroviruses, to see whether this perplexing case was actually HIV infection before its time. **Preliminary determinations are suspicious.**" Antibodies and viruses are mainly made up of proteins, long chains of amino acids that have a biochemical attraction for one another.*

*In the human body, molecules of antibody produced in response to an invasion by a specific virus **fight off the disease by binding themselves to, and then killing, virus molecules in the bloodstream.***

***If a blood sample is mixed with several of the major proteins that make up HIV, and if the sample contains HIV antibody, the same kind of binding will occur in the laboratory.***

*At Tulane, the samples from Robert R. were given over for testing to Dr. Robert Garry, an associate professor of microbiology and colleague of Gottlieb.*

The test chosen by Garry to search for the presence of HIV antibody was the Western Blot, the most specific and sensitive of the antibody tests now in general use.

*The Western Blot is so sensitive that the Pentagon, which is testing millions of new recruits and in-service personnel for AIDS, requires evidence of antibody to just two of the nine main viral proteins before rendering a positive diagnosis. The Red Cross insists on three.*

**The blood of Robert R. contained antibodies to every one of the nine HIV proteins used in the test.** “We found very good reactivity,” Garry said. “We also got positive reactions to two of the tissue samples from his spleen.”

Gottlieb agreed that “there’s no question that it’s positive,” but he said the test would be repeated this week out of an abundance of scientific caution.

Steve Alexander, director of immunology for Biotech Research Laboratories of Rockville, Md., the firm that manufactures the test, said positive reactions for all nine HIV proteins made the presence of HIV **antibody** virtually certain.

“The only alternative would be if someone contaminated the sample,” he said. But he added that in cases of contamination it was unlikely that antibodies **to all nine viral proteins** would be detected.

Asked whether the age of the blood sample might make the possibility of a false positive more likely, Dr. Alexander said he had used the Western Blot on some 15-year-old blood samples “and as long as they’re preserved, they’re okay.”

In **1987**, The London Times (May 11, 1987) reported that the incidence of AIDS infections in Africa coincides exactly with the locations of the W.H.O. smallpox vaccination program in the mid-1970’s. Some 14,000 Haitians then on UN secondment to Central Africa were also vaccinated in this campaign. Personnel actually conducting the vaccinations may have been completely unaware that the vaccine was anything other than what they were told.

## **REVERSE TRANSCRIPTASE AND BAD DRUGS ARE EVERYWHERE, AND NOT SPECIFIC TO RETROVIRUSES.**

In **1987**, an important review article appeared in *Scientific American*, written by Nobelist, and former NIH director, Harold Varmus, entitled *Reverse transcription* [8], which claimed that reverse transcriptase is a normal protein found in the uninfected cells of yeasts, insects and mammals, which is important because it contradicted the views of Gallo and Montagnier that RT’s presence could be taken as proving the presence of retroviruses.[2] Yet they continued to assert:

***This search finally paid off with the discovery of human T-cell leukemia virus types 1 and 2 (HTLV-1 and HTLV-2), the first of which was shown to cause an unusual T-cell leukemia. This discovery was made possible by 15 years of basic research on leukemogenic retroviruses in animals, including the design and development of highly sensitive biochemical assays that were based on reverse transcriptase—the enzyme that is present in all retroviruses...and: Thus, at the beginning of the 1980’s, we had the essential tools required to search for a retrovirus in this new and menacing disease called AIDS (i.e. the detection of RT).***

## **PETER DUESBERG’S PASSENGER VIRUS “HIV” ACTUALLY EXISTS.**

In **1987**, the National Academy member and noted retrovirus researcher Peter Duesberg wrote an encyclopedic review article in which a series of experimental outcomes of experimental putative cancer causing viruses in animals was presented, in an attempt to help determine if indeed a virus actually could be associated with cancer, or immune suppression, or not [9]. In this review article,

which portrayed retroviruses as carcinogens and pathogens, Duesberg argued for the retroviral reality of “HIV’s” molecular signature as advanced by the Gallo and Montagnier groups. However, in the article Duesberg also theorized that the presence of “HIV” in AIDS patients, in people who had received transfusions, or in IV drug addicts, or in non-human primates, constituted a harmless passenger virus, challenging Gallo’s and Montagnier’s hypothesis: (note above what Gallo and Montagnier wrote in 2003: “it seemed more likely that the isolate was causative than that it was opportunistic”). Duesberg has always maintained that “HIV” was indeed a real retrovirus, but he generated doubt about the pathogenicity of retroviruses in the development of human cancer and human AID (Acquired Immune Deficiencies).

In the 1987 *Cancer Research* paper about retroviruses [9], and with his careful and painstakingly difficult-to-read logic, Duesberg, perhaps more than anyone else, raised the issue that if “HIV” was indeed a retrovirus, it could not possibly cause AIDS. Duesberg’s objection to the fact that “HIV” causes AIDS” emerged from a comparison of associations of non-specific **virally-attributed** molecular signatures in natural populations, and observable disease. As necessary evidence for “infectious cancers,” for example, Duesberg demonstrated how it was possible to go beyond Koch’s postulates with respect to viruses, by confirming if molecular markers associated with disease seen under experimental circumstances in genetically weakened animals or highly inbred animals, also occurs among wild, or natural populations of animals (and humans).

Koch’s postulates are perhaps more readily applicable to bacteria and other free-living exogenously-derived pathogens or parasites (Robert Gallo, personal communication), but certainly not to all or even most of them. Because bacteria are free-living organisms, and viruses are not, because every molecule of a virus ultimately is made by cells, including its template through Watson and Crick base pairing after entry into the cell, isolation such as Robert Koch had demonstrated on solid agar with tuberculosis was not possible for viruses, and in fact, improbable. Every molecule of a virus is made without exception by molecules of the cells they infect – so in effect, one could in reality never **materially** isolate the molecules of a cell from that of an infecting virus.

Because of Duesberg’s exhaustive and rigorous comparison of statistics that documented observable disease symptoms to disease markers of cancer in wild populations compared to that statistical relationship observed among weakened laboratory or highly inbred domestic animals, he clearly demonstrated that among natural populations, that markers assumed to be cancer viruses rarely, and only under very special circumstances, were associated with observable disease.

Duesberg’s analysis [9], in addition, detailed a century or more of virological research and cancer, and described in great detail the dynamics, speculative mechanisms of genetic integration, and timing of virological infections that do cause cytotoxic and cytotoxic damage associated with some types of neoplastic transformation seen in laboratory Petri dishes, and in some genetically weakened species of animals. A few examples might convey a small part of Duesberg’s hypothesis (*Italic references are from the Cancer Research paper*):

Avian lymphomatosis virus was originally isolated from leukemic chickens (29). However subsequent studies proved that **latent infection** by avian lymphomatosis viruses occurs **in all chicken flocks** and that by sexual maturity most birds are **infected** (30-32). Statistics report an annual incidence of **2-3% lymphomatoses** in some flocks. Yet these statistics include the more common lymphomas caused by Marek’s virus (a herpes virus) (33,34).

*Viremia has a fast proliferative effect on hemopoietic cells and generates lymphoblast hyperplasia (Fig.1)(32,36,37). Hyperplasia appears to be necessary **but not sufficient** for later leukemogenesis because it does **not** lead to leukemia in tumor-resistant birds (36).*

*The murine leukemia viruses were also originally isolated from leukemic inbred mice (9) and subsequently detected **as latent infections in most healthy mice** (8,13,16,17, 38). Indeed, about **0.5%** of the DNA of a normal mouse is estimated to be proviral DNA of **endogenous retroviruses**, corresponding to **500 proviral equivalents per cell** (18). Nevertheless, leukemia in feral*

mice is apparently **very rare**. For instance low virus expression, **but not a single leukemia was recorded in 20% of wild mice** (38) probably because wild mice restrict virus expression and thus never become viremic and leukemic. However in an **inbred** stock of feral mice predisposed to lymphoma and paralysis, **90%** were viremic from an early age, of which **5%** developed lymphomas at about 18 months (3).

Experimental infections of newborn, inbred mice with appropriate strains of murine leukemia viruses induce chronic viremias. Such viremic mice develop leukemias with probabilities of **0-90% depending on the mouse strain**. However, if mice that are susceptible to leukemogenesis are infected by the time they are immunocompetent or are protected by maternal antibodies if infected as neonates, **no chronic viremia and essentially no leukemia are observed** (although a latent infection is established) (41).

The evidence that mammary carcinomas are transmissible by a milk-borne virus, MMTV, indicates that the virus is an etiological factor (51,52). However, the same virus is also **endogenous but not expressed** in most healthy mice (16, 53). Since **no** mammary tumors have been reported in wild mice, the natural incidence must be **very low**, but in mice bred for high incidence of mammary carcinomas it may rise to **90%** (13,16,54,55).

Feline leukemia virus was originally isolated from cats with lymphosarcoma (59) and subsequently from many **healthy** cats. It is estimated that at least **50-60%** of all cats become naturally infected with feline leukemia viruses at some time during their lives (60,61).

The bovine and human retroviruses associated with acute leukemias are **always biochemically inactive or latent**. **Viremia**, which is frequently associated with a leukemia **of congenitally or experimentally infected** domestic chickens, cats, or inbred mice, **has never been observed in the bovine or human system**. Accordingly, bovine and human leukemia viruses could be isolated from certain leukemic cells **only after** cultivation in vitro **away from the suppressive immune system of the host** (71,72).

**HTLV-I or ATL**V was originally isolated from a human cell line derived from a patient with T-cell leukemia (71). It replicates in **cycling** (emphasis mine) T-cells (27) and also **in endothelial cells (76) or fibroblasts (77)**. The virus was subsequently shown, using **antiviral antibody** for detection, to be endemic as **latent, asymptomatic infections in Japan and the Caribbean (27)**. **Since virus expression is undetectably low not only in healthy but also in leukemic virus carriers, infections must be diagnosed by antiviral antibody or biochemically by searching for latent proviral DNA**. Due to the complete and consistent latency, **the virus can be isolated from infected cells only after activation in vitro when it is no longer controlled by the host's antiviral immunity and suppressors**. Therefore **the virus is not naturally transmitted as a cell-free agent** like other pathogenic viruses, but **only** congenitally, sexually, or by blood transfusion, that is, by contacts that involve exchange of infected cells (13, 27).

It is often pointed out that functional evidence for the virus cancer hypothesis is difficult to obtain in humans because experimental infection is not possible and thus Koch's third postulate cannot be tested. However, this argument does not apply here **since naturally and chronically infected, asymptomatic human carriers are abundant**. Yet **most infections never lead to leukemias and none have been observed to cause viremias**. Moreover, **not a single adult T-cell leukemia was observed in recipients of blood transfusions** from a virus-**positive** donor (13, 78, 79), although recipients developed antiviral antibody (81).

The incidence of adult T-cell leukemia among Japanese with antiviral immunity is estimated to be only **0.06%** based on **339** cases of T-cell leukemia among **600,000** antibody-positive subjects (78).

*In conclusion, the tumor risk of the statistically most relevant group of retrovirus infections, namely the latent natural infections with antiviral immunity, is very low. It averages less than 0.1% in different species, as it is less than 1% in domestic chickens, undetectably low in wild mice, 0.04% in domestic cats on an annual basis, 0.01 to 0.4% in cattle, and 0.06% in humans. Thus the virus **is not sufficient** to cause cancer.”*

Duesberg's 1987 Cancer Research review article also discussed integration sites of viruses, tumor resistance genes, mathematically calculated probabilities of transformation, the evidence of chromosomal abnormalities being the only transformation-specific markers of retrovirus-infected tumor cells, how heterogeneity among the karyotypes (chromosome numbers) of individual human or murine leukemias of the same lineage suggest that chromosome abnormalities are coincidental with rather than causal for transformation, the oncogene hypothesis of Huebner is discussed and dismissed because the hypothesis was based on viral studies only in highly inbred strains of mice and domestic chickens, the hypothesis that latent cellular cancer genes are activated by provirus integrations is discussed and challenged because of the monoclonality Duesberg believed is the hallmark of tumors induced by oncogenes. The hypothesis that latent cellular cancer genes are transactivated by viral proteins was discussed and challenged, and an argument was made that these viruses might indirectly cause cancer through hyperplasia that may be induced by the carcinogenic or irritant properties of these “viruses,” and that understanding the initiation of cancer will result when a better understanding of hyperplasia and host resistance genes are forthcoming – a valid point that is still awaiting clarification by the cancer establishment today.

Then, using similar kinds of considerations, a relatively short section entitled “Retroviruses and AIDS” was presented by Duesberg toward the end of the 1987 Cancer Research paper, the information Duesberg presented was subjected to the same kind of considerations as the “cancer viruses” discussed above, and it is in this context of cancer viruses and oncogenes and “HIV” that his arguments should be considered. Moreover, reading the “HIV-AIDS” section of this paper in the context of his extensive examination of “cancer viruses” and their behavior, is a subject that has never been addressed by Duesberg's many critics, yet considering “HIV” in this context generates a completely different view of what Duesberg actually was criticizing about the “HIV=AIDS” hypothesis.

The first section of the Retroviruses and AIDS section is a synopsis of the epidemiology of “AIDS” and “HIV” as it was typically presented by The Public Health Service in the year 1987 and several years before he wrote this paper. Duesberg made no claims that weren't widely disseminated in CDC official records, and other available surveys of the time. However, in retrospect, it is interesting to note that Duesberg presented the following statements after his extensive treatment of cancer-associated viruses:

Because of the nearly complete correlation between AIDS and immunity against the virus, the virus is generally assumed to be the cause of AIDS (13, 27). Accordingly, detection of antiviral antibody, rather than virus, is now most frequently used to diagnose AIDS and those at risk for AIDS (27, 217-224). **This is paradoxical, since serum antibody from AIDS patients neutralizes AIDS virus (225-227) and since antiviral immunity or vaccination typically protects against viral disease.** It is even **more paradoxical** that a low antibody titer is equated with a low risk for AIDS (228, 229).

Here, Duesberg raised issue with what he considered to be paradoxical viral effects: namely that if testing positive for “HIV” with antibody detection kits are accurate, and if this antibody has been shown by McDougal et al., Gallo et al., Weiss et al., (references 225-227 in the Cancer Research Paper) to neutralize “HIV,” then how could this virus possibly cause disease once anti-viral immunity was established? This question still goes unanswered today, and is still a good question that goes unanswered in the context of other microbial diseases (Hepatitis B, C, HPV, syphilis, etc).

Duesberg continued his argument by stating:

**Unlike all other retroviruses,** AIDS viruses are thought to be direct pathogens **that kill** their host cells, namely T-lymphocytes (13, 27), **and possibly** cells of the brain (230, 255). This view is compatible with the phenotype of AIDS, the hallmark of which is a



defect in T-cells (13, 27, 215), and with experimental evidence that many **but not all** viral isolates induce cytopathic fusion of T-lymphocytes **under certain conditions in vitro** (Section D). Further it is **incompatible** with neurological disease (231, 232, 255). However, cell killing is incompatible **with the obligatory requirement of mitosis for retrovirus replication** (16, 25) **and with the complete absence of cytotoxic effects in all asymptomatic infections in vivo** (Section D).

Duesberg presented other concerns about “HIV’s” molecular signature and AIDS as well:

*Infections with no risk and low risk for AIDS indicate that the virus is not sufficient to cause AIDS.*

*On the basis of this particular epidemiology, it was concluded that the virus is not transmitted as cell-free agent like pathogenic viruses but only by contacts that involve exchange of cells (13, 27).*

In these virus-infected groups the annual incidence of AIDS was found to average 0.3% (224) and to reach peak values of 2 to 5% (218, 223, 233). However even in these groups there are many more asymptomatic than symptomatic virus carriers.

Other infected groups appear to be at no risk for AIDS. **In Haiti** and in certain countries **in Africa** antibody-positive individuals range from 4 to 20% of the population, whereas the incidence of AIDS is estimated at less than 0.01% (223, 229, 234). Several reports describe large samples of children from Africa who were **20 (228) to 60% (221)** antibody positive and of female prostitutes who were **66 to 80% antibody positive (221, 235), yet none of these had AIDS**. Among male homosexuals and hemophiliacs of Hungary about 5% are **AIDS virus** positive, **yet no symptoms of AIDS were recorded** (161). Among native **male and female Indians of Venezuela 3.3 to 13.3% have antiviral immunity, but none have symptoms of AIDS (236)**. Since these Indians are totally isolated from the rest of the country, in which only one **hemophiliac** was reported to be virus positive (236), the asymptomatic nature of their infections is not likely to be a consequence of a recent introduction of the virus into their population. Thus **it is not probable** that these infections will produce AIDS after the average latent period of 5 years (Section B).

Again, the references and data Duesberg gives were not disputed. In fact, many of these quoted references were produced by key AIDS officials including Montagnier, Curran, Weiss, Biggar, Gallo, and others, and were published in Science, The Lancet, Cancer Research, JAMA, and in other journals of some notoriety.

Each of the issues in the AIDS section of the paper that Duesberg raised, was simply a rehashing of arguments he raised concerning the non-compatibility of long latent periods and cancer initiation among the so-called onc or non-onc gene-containing viruses (as seen in cell culture dishes in transformation experiments, or in genetically weakened animals given toxic carcinogenic infusions before their immune systems have been given a chance to develop). What is perhaps most striking about Duesberg’s Retroviruses and AIDS section in retrospect, is that he advocated that “HIV” might generate a mononucleosis-like illness for all the same reasons that timing of replication and biochemical activity of high virus titres may play a role in oncogenic and non-oncogenic viruses. In other words, through his knowledge of the so-called and supposed retroviral cancer virus-induced effects in genetically weakened animal strains, he separated the immediate short-term effects of viral replication before antibody immunity becomes established, from long latency viral diseases such as those induced by what he believed constituted a hyperplastic (overstimulated) augmentation of cell proliferation and aneuploidy, that is typically associated with cancers that form in genetically weakened strains of animals who harbor the molecular signature of viruses that do not contain onc genes.

The eclipse period of AIDS virus replication in cell culture is on the order of **several days**, very much like that of other retroviruses (238). In humans virus infection of a sufficient number of cells to elicit an antibody response appears to take **less than 4 to 7 weeks**. This estimate is based on an accidental needle-stick infection of **a nurse**, who developed **antibody** 7 weeks later (239), and on reports describing 12 (240) and 1 (232) cases of male homosexuals who developed antibody **1 to 8 weeks** after **infection**. During this period a mononucleosis-like illness associated with **transient** lymphadenopathy was observed. In contrast to AIDS

(see below), this illness appeared 1 to 8 weeks after infection and lasted only 1 to 2 weeks **until antiviral immunity was established**. The same early mononucleosis-like disease, associated with lymphocyte hyperplasia, was observed by others in primary AIDS virus infections (234). This is reminiscent of the direct, early pathogenic effects observed in animals infected with retroviruses prior to the onset of antiviral immunity (Part I, Section B).

This short latency period was set in contrast to what Gallo and Montagnier believed was the latency for their hypothesized syndrome of the development of “AIDS” 5, 10 or more years after infection, as Duesberg pointed out:

By contrast the lag between infection and the appearance of AIDS is estimated from transfusion-associated AIDS to be **2 to 7 years** in adults (220, 223, 241, 242) and **1 to 2 years** in children from infected mothers (220, 223). The most likely mean latent period was estimated to be **5 years** in adults (220, 223). Unexpectedly, most of the AIDS virus-positive blood donors identified in transfusion-associated AIDS transmission did **not** have AIDS when they donated blood and were reported to be in good health **6 years** after the donation (220). Likewise there is evidence that individuals shown to be antibody positive since 1972 have not developed AIDS (228). Further 16 mothers of babies with AIDS did not have AIDS at the time of delivery but three of them developed AIDS years later (276). This indicates that the latent period may be longer than 5 years or that AIDS is not an obligatory consequence of infection.

Duesberg’s principal and cogent argument therefore was that cells or viruses do not wait 5, 10 or 20 years to generate their pathogenic effects, nor in humans who develop a mononucleosis-like condition weeks after they exhibit the molecular signature of “HIV,” when a positive test and anti-viral immunity become established.

### **DRUGS-AIDS, FOREIGN PROTEIN-AIDS, OR MALNUTRITION-AIDS.**

That recreational drugs, prescription drugs, foreign proteins infused into the bloodstream, and malnutrition can cause profound immune suppression was not advanced by Duesberg in the 1987 *Cancer Research* paper. It was advanced in other papers Duesberg wrote later in an attempt to explain how advanced acquired immune suppression may be caused if not by a harmless passenger virus, “HIV.” At numerous public lectures and in several papers published in the next several years, Duesberg communicated these alternative known causes of immune suppression to the scientific community and to the general public, in an attempt to provide rational and alternative possible reasons why a growing group of young men in Los Angeles, San Francisco, and New York appeared to be developing a strange cluster of rarely observed 3rd World-like diseases and syndromes. Instead of being caused by “HIV,” he argued that the fatal syndrome called AIDS probably had nothing to do with a “passenger virus,” but instead was the result of numerous assaults on the immune system by behaviors such as acute or long-term heroin addiction, multiple transfusions, factor concentrates used by hemophiliacs causing foreign protein autoimmune conditions, and by severe malnutrition due to, in many cases, profound illicit or prescription drug abuse, or in Africa, by lack of food, clean water, and common diseases of poverty.

Nutritional deficiency and prescription and illicit drug reactions and their association with various immune deficiencies and other syndromes had been described in the writings of numerous scientists and physicians for more than a century, long before Duesberg was born. It was common knowledge, for example, that malnutrition or exposure to foreign proteins are all phenomena which are known to induce many of the hallmarks of “AIDS,” such as T-cell depletion, and other syndromes, as is the chronic long-term use of illicit and certain prescribed drugs. For instance, prescription steroids were said to iatrogenically cause of AIDS, as was published years before in 1975-6 by Anthony Fauci, who later became the NIAD Czar of AIDS funding. Doctors caused immune suppression, Fauci claimed, if they subject their patients to multiple transfusions, transplant surgery, or corticosteroid administration, as these drugs and treatments can non-specifically induce AIDS-specific drops in T-cells with high frequency [10, 11].

Fibrosis of the lung due to heavy crack cocaine or PCP use, opium use or Ecstasy, also were considered all to be potent inducers of the most commonly seen AIDS-defining illness, by Fauci and others before the AIDS era. Duesberg, in his book, *Inventing the AIDS*

*virus* attempted to correlate hospital emergency room statistics about drug overdose frequency following the Vietnam era, and the appearance of AIDS amongst heavily drug-using individuals whether or not they were gay or straight. These qualifications served to undermine the “HIV=AIDS” hypothesis, because A (“HIV”), does not generate B (immune suppression), that leads to C (full-blown AIDS) because iatrogenically applied glucocorticoids, transfusions, blood factor concentrates in hemophiliacs, lack of nutrition, and other factors such as chronic crack cocaine use may induce a precipitous drop in B, and consequently lead to C. What this meant, at least to those who care about patients, Fauci warned, is that if not careful, doctors can cause profound immune suppression through too much steroid administration.

As the investment by the government and government scientists was exponentially increasing and becoming institutionalized according to the design and predictions of the “HIV=AIDS” hypothesis of Gallo and Montagnier, “the AIDS epidemic” likewise began to increase as a function of increased testing, and Duesberg’s questions regarding the causal connection between the molecular markers thought to be diagnostic of “HIV” and “AIDS” were perceived as a threat. Bitter attacks were launched against Duesberg with accusations that he erected a drugs-AIDS hypothesis “on the basis of no real research,” or because he was “jealous” of the success of the oncogene hypothesis advanced by others such as Varmus, Bishop, Baltimore, Gallo, and others. As his critics were and are so often heard to retort, “There are no published studies that show that drugs and drug effects are consistently associated with the development of AIDS.” But Anthony Fauci’s observations did. Also, Harry Haverkos, who worked for the CDC during these early days of the AIDS era, published that drug addicts should be considered to be non-AIDS patients, but merely immunosuppressed, because of chronic drug abuse [12].

“HIV=AIDS” hypothesis proponents, advocacy groups, as well as the press and certain members of the gay community held a different view of Duesberg’s contributions. For example, despite his National Academy member status and prestigious Fogarty award for his pioneering work on oncogenes and retroviruses, Duesberg received no federal funding following the appearance of his 1987 *Cancer Research* paper, and he has been repeatedly denied public debates or publication of his ideas. His writings were repeatedly rejected by powerful journal editors such as John Madox of *Nature*, and others. At least once a year, it is still not unusual to see the following kinds of portrayals of Duesberg’s thinking and hypotheses by journalists:

*The Trouble with Duesberg’s Theory, By Marc B Haefele, Editor, Los Angeles Alternative Times:*

*Peter Duesberg isolated the first cancer-causing gene from a virus at 33, got tenure at Berkeley at 36, joined the National Academy of Sciences at 49. So why is he in bad odor in the scientific and gay communities? In small part because, on the basis of no real experimental research, he proclaimed in a 1988 article that AIDS wasn’t caused by the HIV virus but by bad nutrition and recreational drugs. As the Scientific American put it “his idea died as most failed theories do: never fully disproved but convincingly rebutted by the National Institute for Allergic and Immune Diseases—and ultimately ignored by nearly everyone working in the field.”*

*And largely because Duesberg, a non-physician, kept peddling his theory as fact. As the San Francisco based Project Inform noted “He offers an easy, comforting approach to AIDS and has been able to influence people to trust him and reject their doctor’s advice.” **He fronted** a “well financed” campaign to get AIDS patients to reject medical care. Most recently, he succeeded – temporarily – in getting South African President Thabo Mbeki to reject the viral cause of AIDS. It’s not typical scientific procedure to promote a debunked theory at the probable cost of thousands or millions of lives. But that’s what Duesberg has been doing.*

Similarly, not only journalists, but establishment scientists and physicians perpetually criticize Duesberg’s ideas and hypotheses. To quote Marc Wainberg, the 2006 Chair of The Toronto International AIDS Conference—who possesses several “HIV” drug patents such as lamivudine (3TC), and grants from GlaxoSmithKlein, Bristol-Myers Squibb and Boehringer-Ingelheim):

*As far as I’m concerned, and I hope this view is adequately represented, those who attempt to dispel the notion that HIV is the*

cause of AIDS are perpetrators of death. And I would very much for one like to see the Constitution of the United States and similar countries have some means in place that we can charge people who are responsible for endangering public health with charges of endangerment and bring them up on trial. I think that people like Peter Duesberg belong in jail. (Quoted from the documentary, "The Other Side of AIDS" which won a special Jury Prize at the AFI Los Angeles International Film Festival) [13].

As a result of Duesberg's comparison(s) of putative cancer-inducing retroviruses and symptomatic versus asymptomatic disease(s), in the cancer community, Duesberg provided us with a new logic to assess potential causal relationships, if any, between retroviruses and cancers in populations of wild-type individuals, or if it could be shown, between "HIV" and AIDS.

Perhaps this belief in the reality of retroviruses and "HIV" can best be seen in a statement Duesberg made later in the 1990's, when he tried to claim an award offered to find "the missing [HIV] virus."<sup>9</sup> In all the literature, it is a statement, perhaps, that stands as the most persuasive argument that the molecular signature of "HIV" corresponds to a unique retrovirus .

*Here I take up these challenges. I will argue that **HIV exists, and has been properly identified** as a unique **retrovirus** on the grounds that (i) **it has been isolated – even from its own virion structure** – in the form of an infectious, molecularly cloned HIV DNA that is able to induce the synthesis of **a reverse transcriptase** containing virion, and (ii) that HIV-specific, viral DNA can be identified **only in infected, but not in uninfected human cells**. In view of this I can base my claim for the isolation of HIV on the most rigorous method available to date, i.e. **molecular cloning of infectious HIV DNA**, rather than only on the much less stringent, traditional "rules for isolation of a retrovirus ... discussed at the Pasteur Institute, Paris, in 1973" that were stated criteria of isolation in Continuum's missing virus reward. Indeed I will show that molecular cloning of infectious HIV DNA exceeds the criteria of the old "Pasteur rules.*

#### *(I) Isolation of HIV*

**The existence of the retrovirus HIV predicts that HIV DNA can be isolated from the chromosomal DNA of infected cells.** This prediction has been confirmed as follows: Full-length HIV-1 and HIV-2 DNAs have been prepared from virus-infected cells and cloned in bacterial plasmids (13-15). **Such clones are totally free of all viral and cellular proteins, and cellular contaminants that co-purify with virus. These clones produce infectious virus that is neutralized by specific antisera from AIDS patients.** For example, virus produced by infectious HIV-2 DNA is neutralized by antiserum from HIV-2 but not from HIV-1-infected people.

**Since infectious HIV DNA has been isolated from infected human cells that is free of HIV's own proteins and RNA as well as from all cellular macromolecules, HIV isolation has passed the most vigorous standards available today. In other words these infectious DNA clones meet and exceed the isolation standards of the traditional "Pasteur rules." Isolation of infectious HIV DNAs is theoretically the most absolute form of isolation – it is the equivalent of isolating the virus' soul, its genetic code, from the virus' body, the virus particle.** Thus HIV isolation based on molecular cloning exceeds the old standards defined as "Pasteur rules" by Continuum.

#### *(II) Identification of HIV*

The existence of HIV predicts that infected cells contain a unique, virus-specific DNA of **9150 nucleotides that cannot be detected in DNA of uninfected human cells**. The probabilities that cellular DNA and other viral DNAs would contain the same sequence of 9150 nucleotides is 1 in  $4E9150$ , or 1 in  $10E4500$  – extremely close to zero! Since the odds that a given nucleotide of any DNA is either A, G, C or T are in 1 in 4, the odds that any DNA has the same sequence of 9150 nucleotides as HIV-1 or HIV-2 are only 1 in  $4E9150$ .

Thanks to the **outrageous interest** in HIV as the hypothetical cause of AIDS, many investigators have sought specific HIV DNA in humans with and without AIDS in an effort to confirm **that rather unreliable HIV antibody-test**.

**But because only 1 in 100 T-cells are ever infected in humans, virtually all such studies use Kary Mullis' polymerase chain reaction**, a technique that is designed to amplify a DNA-needle into a DNA-haystack. Such efforts have confirmed the existence of HIV-specific DNA **in most** (not all) antibody-positive persons with and without AIDS – but not in the DNA of antibody-negative people. For example, Jackson et al have tested blood of **409 antibody-positives** including **144 AIDS patients and 265 healthy people**. In addition **131 antibody-negatives** were tested. HIV-specific DNA subsets – defined in size and sequence **by HIV-specific primers** (start signals for the selective amplification) – were found in 403 of the 409 antibody-positives, but in none of the 131 antibody-negative people.

**The high sequence specificity of HIV DNAs is translated into the specificity of their proteins**, eg. antibodies against HIV-1 do not neutralize HIV-2 and vice versa.

### **In Conclusion**

HIV has been isolated by the most rigorous method **science** has to offer. **An infectious DNA of 9.15 kilo bases (kb) has been cloned from the cells** of HIV-antibody-positive persons, that – upon transfection – induces the synthesis of an unique retrovirus. **This DNA “isolates” HIV from all cellular molecules, even from viral proteins and RNA**. Having cloned infectious DNA of HIV is as much isolation of HIV as one could possibly get. The retrovirus encoded by **this infectious DNA** reacts with the same antibodies that cross-react with Montagnier's global HIV standard, produced by immortal cell lines in many labs and companies around the world for the HIV-test. This confirms the existence of the retrovirus HIV.

The uniqueness of HIV is confirmed by the detection of HIV-specific DNA sequences in the DNA of most antibody positive people. **The same DNA is not found in uninfected humans**, and the probability to find such a sequence in any DNA sample is 1 in 4E9500 – which is much less likely than to encounter the same water molecule twice by swimming in the Pacific ocean **every day** of your life.

These statements serve to show only that Duesberg believed in the reality of retroviruses and “HIV” in particular, and also felt that the tools of molecular virological reductionism such as cloning were and are unequivocal, with statements such as:

*Such clones are **totally free** of all viral **and cellular** proteins, **and cellular contaminants** that co-purify with virus; Since infectious HIV DNA has been isolated from infected human cells that is free of HIV's own proteins and RNA as well as from all cellular macromolecules, HIV isolation has passed the most vigorous standards available today.*

It could therefore be argued that one of Duesberg's principal contributions to the AIDS era was to argue for “HIV” constituting a unique exogenous retroviral signature that Gallo and Montagnier believed they had isolated from AIDS (or ARC) patients.

But was this certainty by the retrovirologists such as Peter Duesberg proof that “HIV” has been purified free of all contaminating cellular debris? Others would later show that “HIV” as an exogenous retrovirus had not been isolated from the cellular debris caused by cell fractionation (breaking up cells to “isolate HIV”) let alone that isolation of “HIV's” proteins, lipids, and nucleic acids had been achieved. Nevertheless, Duesberg championed the certitude of cloning technology of his day to isolate specific molecular components with near 100% certainty, and perhaps to this day, this belief has continued to serve as a foundation for all of the work upon which retrovirology as a science is based.

**THE RNA BASIS OF REVERSE TRANSCRIPTASE AND AIDS SCIENCE: MODERN CELLULAR CHROMOSOMES**

## **CAME FROM RETROELEMENTS, CAULIFLOWER VIRUSES DON'T ENTER THE NUCLEUS, AND VIRUSES REALLY WEREN'T AND AREN'T CELLULAR PARASITES BUT THEIR STRUCTURE WAS CREATED BEFORE PROTEIN SYNTHESIS.**

Despite the fact that the entire idea of retroviruses flew in the face of biology's central dogma that DNA makes RNA, after the acceptance of the reality of "HIV," molecular clonologists came up with ever more fantastic ideas and hypotheses. These ideas were ushered in, in part, by the growing belief in power of the primary nucleic acid sequence to explain all things, and that the constant exceptions encountered by "HIV-scientists" could then be borrowed or exploited to form the bases of other equally speculative molecular sciences regarding molecular evolution, "retroviruses," "retroelements," and their natural hosts. "The genomic tag hypothesis," for example, was advanced to suggest an alternative view of the origin and evolution of retroviruses as fossils that reveal the diversity of ancient replication pathways (see Wintersberger and Wintersberger 1987; Reference: *Bull.* 196: 327-330. June 1999). Such ideas advanced the proposal that viruses **have not devised novel and subversive** replication strategies; rather, they have conserved the useful features of more ancient forms of **cellular** chromosomal replication. Why? The following types of arguments and reasoning provided some of the basis of the belief in "HIV" and retroviral reverse transcriptases, which lay at the basis of both retroviral causes of cancer and AIDS science, as well as a phylogenetic basis upon which to ignore phylogenetic similarities between "SIV," "HIV," and other retroviruses in primate evolution, while confusing these ancient gene sequences with exogenous, infectious, and constantly mutating retroviruses. The following section briefly recounts the thinking that became almost universally accepted and taught, without critical examination:

*No single molecule, product, or function is common to all viruses, so there is no universal standard for evolutionary comparisons that is useful **in quite the same way as small subunit ribosomal RNA sequences have been for organismal phylogenies**. Furthermore, the interchangeability of **functionally similar viral modules can undermine** viral phylogenies based on a single molecule **such as reverse transcriptase** (but see Xiong and Eickbusch 1990; Eickbusch 1997; Nakamura et al. 1997). Simple assumptions **cannot** be made about the regularity of viral **molecular clocks** (see Wilson and Sarich's history in PART 1), because generation times are short, burst sizes are large, and **there is no single typical genome**, but rather a population of **quasispecies and defective interfering particles** all propagating simultaneously (Eigen et al. 1981).*

**Retrotransposons are descendants of autonomous transitional genomes** which had both RNA and DNA replicative forms (see their imagined phylogenetic sequence below Fig. 6). **Modern eukaryotic DNA chromosomes would then have been built by stepwise assembly of these smaller independent genomic elements.**

**The attitude that viruses are cellular parasites, assembled in relatively recent evolutionary time from preexisting parts of the cell (Benner and Ellington 1988), is difficult to reconcile with data showing that many aspects of viral life cycles are stable over evolutionary time.** In fact, the genomic tag hypothesis suggests an alternative view of viruses, as fossils that reveal the diversity of ancient replication pathways (see also Wintersberger and Wintersberger 1987). Considered in this way, **viruses have not devised novel and subversive replication strategies**; rather, they have conserved the useful features of more ancient forms of cellular chromosomal replication, even as cellular replication strategies continued to evolve.

**TRANSLATION:** In other words, in this view, the idea is advanced viruses didn't arise from cells during recent evolution, but instead represent fossils that reveal and still use ancient replication strategies of cellular chromosomal DNA and RNA.

*Stunning and completely unanticipated results from the groups of Lambowitz (Kuiper and Lambowitz 1988; Akins et al. 1989; Chen and Lambowitz 1997), Saville and Collins (1990), Blackburn (for review, see Blackburn 1991), and Cech (Lingner et al. 1997) subsequently provided key **molecular fossil evidence** for genomes and genomic tags **in transition from** single-stranded RNA to double-stranded DNA.*

## **Telomerase: A Genomic Tag Carried by the RNA Component of a Reverse Transcriptase**

*When we first suggested that the 3-terminal CCA motif of tRNA-like genomic tags was related to the nearly universal C<sub>m</sub>An motif of **eukaryotic nuclear telomeres** (Weiner and Maizels 1987), **we were unable to discern any hints in the molecular fossil record regarding the mechanism or chain of events by which a 3-terminal CCA motif in RNA could be transformed into a 5-terminal motif in DNA.***

***Another missing link in the molecular fossil record emerged from detailed characterization of the Tetrahymena telomerase.** This enzyme, first described by Greider and Blackburn (1985, 1989), possesses an unusual terminal nucleotidyltransferase activity that adds the species-specific TnGm repeats, one nucleotide at a time, to an appropriate TnGm primer (for review, see Blackburn 1991). Remarkably, telomerase is **a ribonucleoprotein**, and the RNA component is in fact an internal template for synthesis of the species-specific TnGm repeat. (For example, the telomeric repeat in the ciliate Tetrahymena is T<sub>2</sub>G<sub>4</sub>, and the internal template sequence in the Tetrahymena telomerase is 5-CAACC- CCAA-3.) **One of the protein components of telomerase, known in Saccharomyces cerevisiae as EST2 (ever shorter telomeres), is homologous to retroviral reverse transcriptases (Lingner et al. 1997).***

***Telomerase can thus be seen as a specialized reverse transcriptase with an internal tRNA-like template** (for review, see Blackburn 1991 and this volume).*

*The historical roots of modern DNA chromosomes in such transitional genomes would then be apparent in two conspicuous molecular fossils: RNA serves as primer for the initiation of DNA synthesis (at least in eubacteria), and RNA serves as template **for the completion** of chromosomal replication (eukaryotic telomeres).*

*A friendly amendment might be that involvement of RNA **in any DNA transaction** (replication, recombination, repair, or modification) suggests that the process is ancient, **dating back to an RNA World or a world in transition from RNA to DNA genomes.***

**TRANSLATION:** This was quite a leap in logic on the basis of no evidence—that the mere presence of RNA and ribonucleoprotein measured in any eukaryotic process involving nucleic acid replication, recombination, repair, or modification means that that process dates back to a mythical “RNA world” that preceded DNA’s role as the central and stable molecular repository of genetic instructions, and that even protein synthesis itself had to have occurred differently in the RNA beginning though the employment of such molecules as polyamines, which not known then, universally condense both prokaryotic and eukaryotic DNA, making protein synthesis impossible. To accommodate ideas involving the topological conundrum of the modern eukaryotic cell’s postulated telomere, and the nearly impossible to detect “retroviruses” or “retroelements” like “HTLV-I,” “HTLV-II,” and “HIV,” the following type of story was constructed on this basis, and as shown here below, became the phylogenetic sequence of events believed to have occurred by using this reasoning:

*Figure 6 A phylogenetic tree for replication strategies based on conservation of tRNA-like structures in the initiation of genomic replication.”*

- 1. **tRNA as template for RNA synthesis.***
- 2. **Neurospora mitochondrial plasmids** (tRNA as template for cDNA synthesis).*
- 3. **Cauliflower mosaic virus** (tRNA as primer for cDNA synthesis).*

4. **Integrating retrovirus** (*tRNA as primer for cDNA synthesis*).

5. **Eucharyotic chromosome** (*rRNA as a template for telomere synthesis*).

Upon what evidence was this sequence of evolution of retroelements and modern chromosomes based? It is based on the following kinds of ideas and beliefs:

The historical roots of modern DNA chromosomes in such transitional genomes would then be apparent in two conspicuous molecular fossils: RNA serves as primer for the initiation of DNA synthesis (at least in eubacteria, and RNA serves as template for the completion of chromosomal replication (eukaryotic telomeres).

The genomic tag hypothesis **suggests** that aminoacylation initially conferred a replication advantage on molecules carrying a genomic tag. This could have occurred **in any** of several ways. Aminoacylation **might have** facilitated binding of the replicase to the 3' end of the genome, perhaps simply by countering the net negative charge of the RNA replicase with a positively charged (basic) amino acid. Aminoacylation **could also have served** as a regulatory mechanism for withdrawing a genomic RNA from the replicative pool by blocking binding of the tag to the replicase. A third possibility, suggested by Wong (1991), is that aminoacylation **might be seen** as a form of RNA modification (like methylation, thiolation, isopentenylolation) that would broaden the structural or catalytic range of the RNA bearing it. “

*Our model for the origin of protein synthesis is unique in postulating that key components **of the translation apparatus**—*tRNA and tRNA aminoacylation activity*—**first evolved as essential components of the replication machinery before the advent of protein synthesis.***

*With these two key components of the translation apparatus in place, the scene was set for the **interdependent** coevolution of replication **and** templated protein synthesis.*

***Perhaps at first**, random condensation of aminoacylated tRNAs generated short **polycations**. These simple **polymers** could have facilitated RNA-catalyzed reactions in a manner analogous to modern **polyamines** (Jay and Gilbert 1987; Maizels and Weiner 1987; Weiner and Maizels 1987).*

***Or they could have** stabilized or promoted a particular RNA structure, **much as the tract of basic amino acids within the HIV Tat protein shapes the structure of the TAR element** (Puglisi et al. 1992).*

***The ability of polyamines to regulate translational frameshifting on the ornithine decarboxylase antizyme mRNA** also provides additional, **albeit less direct, evidence** that short polycations might have been useful in an RNA World (Matsufuji et al. 1995).*

#### **TRACING GENOMIC TAGS FROM RNA PHAGE TO MODERN CHROMOSOMAL TELOMERES: THE NOTION OF TRANSITIONAL GENOMES**

*Although we **suspected** that tRNA-like genomic tags survived from an RNA World into a DNA World **and were, in the process, transformed into the tRNA primers of retrovirus reverse transcription and the terminal CmAn motifs of modern chromosomal telomeres** (Weiner and Maizels 1987), **we were unable to make these connections explicit because there appeared to be one or more missing links in the molecular fossil record.***

*Thus, as long as the tRNA recognition domain of reverse transcriptase is flexibly tethered to the active site, the enzyme **might***



readily evolve from using tRNA as template to using it as primer. **Although HIV reverse transcriptase is only distantly related to the Neurospora enzyme, the ability of the HIV enzyme to form a binary complex with primer tRNA (Barat et al. 1989) is consistent with the idea that separation of tRNA recognition and the polymerase module may be a general phenomenon.**

Use of tRNA as primer also **necessitates RNA helicase activity**, because the enzyme would have to melt the top half of tRNA (a coaxial stack of the acceptor stem on the T U c arm (Figures 1 and 2) in order to allow the primer to base-pair with the genomic primer-binding site (see Fig. 6).” However, this helicase activity would already be in place, because any enzyme that uses a genomic tag as template must possess an RNA helicase activity that can melt the top half of tRNA once initiation has occurred on the 3-terminal CCA. Extensive complementarity between the tRNA primer and the template (Isel et al. 1995; Lanchy et al. 1996) may be a later refinement to assure a unique site of initiation and to stabilize the initiation complex.

And here is the proof and basis of the RNA science upon which “HIV’s” and chromosomal telomere reverse transcriptase’s behavior was believed to have occurred:

### **Caulimoviruses Are Non-integrating One-LTR Retroviruses**

We have discussed several reasons for thinking that mitochondrial retro-plasmids may represent the ancestral form of **modern retroviruses**, but there is yet another missing link between duplex DNA plasmids and the prototypical integrating retrovirus with long terminal direct repeats (LTRs). **A plant retrovirus, cauliflower mosaic virus (CaMV), may provide this connection. The CaMV genome is an extrachromosomal circular duplex DNA that lacks direct repeats and never integrates into chromosomal DNA.** Transcription of viral DNA generates a full-length genomic RNA, but because the polyadenylation site for this transcript is located 180 bp downstream from the viral promoter, the transcript contains a 180-nucleotide terminal redundancy (Hohn et al. 1985; Covey and Turner 1986; also see Fig. 6).

These similarities in genomic structure and replication strategy **imply that modern integrating retroviruses descended from an extrachromosomal retroviral element resembling CaMV.**

The **obvious** advantage of chromosomal integration is that it assures perpetuation of the element.

The existence of **two LTRs in a prototypical retroviral provirus could then be interpreted** as an invention that preserved the established replication strategy **by counterfeiting the circular topology of the ancestral genome.** “

**The key question, however, is the direction of time’s arrow.**

Did **linear** HeT-A-like elements give rise to **circular** retroelements, **or** did autonomous **circular** elements generate multimers (perhaps by homologous recombination or runaround transcription) that then acquired the ability to retropose **as linears**? We favor the view that large DNA chromosomes were assembled stepwise from smaller autonomously replicating units carrying one or a few linked genes.

### **CONCLUSION**

We originally conceived of the genomic tag hypothesis to explain **the origin of protein synthesis** (Weiner and Maizels 1987), but with time it became clear that this hypothesis had **equally distinct implications for the evolution of replicative**

**mechanisms, beginning in an RNA World and continuing to the present day.** *The new evidence that we have discussed for the central role of genomic tags in the evolution of RNA to DNA genomes strengthens the case that tRNA-like genomic tags arose early in an RNA World.*”

“ Translation today is a complex and sophisticated process, involving at least 2 ribosomal RNAs, more than 50 ribosomal proteins, 20 synthetases, tRNAs, initiation factors, elongation factors, etc. **It is clear** that the translation apparatus **must have arisen stepwise**, but it has been difficult to imagine how **any** single component could be useful by itself, or how additional components could each individually confer a further selective advantage. Central to the genomic tag hypothesis is the suggestion that the two key components of the translation apparatus—tRNA and tRNA aminoacylation activity—first evolved as essential components of the replication apparatus, and were subject to selection **before** the advent of protein synthesis. Once in place, these two key components of the translation apparatus could be coopted for other purposes, with the result that replication and templated protein synthesis were fated to coevolve forever after.

Thus it was with these kinds of totally speculative chemical arguments that the entire basis of retrotransposons, retroelements, reverse transcriptase, telomerase, and retrovirus evolution was based.

### **THE FATHER OF TRANSFER RNA AND RIBOSYME TECHNOLOGY DR. PAUL ZAMESCNİK SAYS HIS RIBOSYMES WILL CURE “HIV,” (BUT LATER CHANGED HIS MIND).**

An associate professor working with Gallo’s group that Paul Zamesnik had known named Prem Sarin. Dr. Sarin had tried in Gallo’s lab to inhibit “HIV” in vitro, but couldn’t consistently measure “HIV’s” so-called reverse transcriptase signal(s) in his cultures lacking anti-sense oligonucleotides, and was thereby rudely treated by Gallo. As rumor had it, Sarin had been kicked out of the lab. Prem Sarin eventually was arrested and convicted of siphoning about 20 K of AIDS charity funding, and he was sentenced to 3 months of community service. By 1989, Dr. Zamecnik’s Worcester-based group had published on “HIV” work and, in collaboration with Dr. Gallo’s Bethesda group, they had published several papers with such titles as:

Inhibition of replication and expression of human TA-cell lymphotropic virus type III in cultured cells by exogenous synthetic oligonucleotides complementary to viral RNA. Paul Zamecnik, John Goodchild, Yoshi Taguchi, and Prem S. Sarin. *PNAS* Vol. 83, No. 12 June 15, 1986.

Inhibition of Human Immunodeficiency Virus Replication by Antisense Oligodeoxynucleotides. John Goodchild, Sudhir Agrawal, Maria P. Civeira, Prem S. Sarin, Daisy Sun and Paul C. Zamecnik. *PNAS* Volume 85, No. 15, Aug. 1 1988.

Phosphoramidate, Phosphorothioate, and Methylphosphonate Analogs of Oligodeoxynucleotide : Inhibitors of Replication of Human Immunodeficiency Virus. Sudhir Agrawal <sup>1</sup>; John Goodchild <sup>1</sup>; Maria Civeira <sup>2</sup>; Prem S. Sarin <sup>2</sup>; Paul C. Zamecnik. <sup>1</sup> Affiliations: <sup>1</sup> Worcester Foundation for Experimental Biology, Shrewsbury, MA; <sup>2</sup> Laboratory of Tumor Cell Biology, National Cancer Institute, Bethesda, MD. *Nucleosides, Nucleotides and Nucleic Acids*, Volume 8, Issue 5 & 6 , pages 819 – 823, 1989.

Most of these discussions at that time focused on how to stabilize Zamecnik’s anti-sense molecules using other molecules like thiols, so they would not be degraded by intracellular nucleases that chewed them up. Dr. Zamecnik conceded that the “HIV” responses they saw were nothing but “In Vitro” phenomena.”

### **THE SUCCESS OF HIGH DOSE AZT**

**1987** was the year of the publication of the results of the first AZT trial, and FDA approval of the drug was obtained only after only 4

months of study. The Fischl Phase II trial was the first to have claimed to demonstrate the efficacy and safety of AZT, upon which FDA approval was obtained, and it is claimed that it was the only trial in US history to show in a record 4 months that a drug (AZT) was “worthy” of FDA approval [14, 15, 16]:

The licensing study of AZT, performed in 1987 by the NIH in collaboration with the drug’s manufacturer Burroughs Wellcome in the US, is the primary placebo-controlled study set-up to test the ability of AZT to reduce the mortality of AIDS. The study showed that, after 4 months on AZT, 1 out of 145 AIDS patients died, whereas 19 out of 139 died in the placebo group. The study interpreted this result as evidence for reduced mortality by AZT. **However, this interpretation failed to consider that among the 4-month-survivors of AZT, 30 could only be kept alive with multiple blood transfusions because their red cells had been depleted by AZT below survivable levels. Thus, without lifesaving transfusions 30 more AZT-recipients would have died from anemia.** In addition many AZT recipients had developed life-threatening **bone marrow suppression, neutropenia, macrocytosis, headaches, insomnia and myalgia, that augured poorly for their future survival** (Richman D D, et al and the AZT Collaborative Working Group 1987 The toxicity of azidothymidine (AZT) in the treatment of patients with AIDS and AIDS-related complex; N. Engl. J. Med. 317 192-197). Indeed, the low mortality of 1/145 reported for the first 4 months on AZT, could not be maintained in a follow-up study, which found the “survival benefits” of AZT rapidly declining after the original 4-month period. **By 21 months, 42% of the original AZT group had died and 35% of the control group, which by then had also received AZT for 12 months on a “compassionate” basis:** Thus the placebo-controlled, licensing study did not prove that AZT reduced AIDS mortality by more than 80% compared to the untreated control.

Fischl et al. noted a transient rise in T-cells, but by 21 months, 42% of the original AZT group had died and 35% of the control group, which by then had also received AZT for 12 months on a “compassionate” basis. Control groups were abandoned in this trial for the reasons alluded to above, which is important because it was the trial that led to the FDA approval of AZT in an unprecedented 4 months. This allegation of collusion by pharma and the CDC was unprecedented in FDA history, especially in light of accusations that the trial became unblinded after 1 week because the toxicity of AZT was apparent to everyone, especially the patients [15]. The FDA’s recommendation for the approval for AZT was also beyond all logic or common sense because it was discovered that a series of patients were switched over from the control arm to the experimental arm, and it was disclosed that 30 patients in the experimental group required life-saving transfusions to stay alive. These facts, in addition to the Boston arm of the trial being cited for “fraudulent record keeping,” were all obtained by the Freedom of Information Act and published by Lauritsen [16].

It is interesting to note that the document Lauritsen obtained through the FOI act and presented in his book is riddled with black deletions of text, as if the contents of the study were describing a new nuclear weapons technology rather than a new drug. The document is difficult to read because of so many deletions, yet creative record keeping was evident. Also, in the context of prematurely terminating antiretroviral human trials and switching experimental arms to placebo arms “for compassionate reasons,” one must also factor in the violations of human rights and complete lack of informed consent accompanying this practice. But these sorts of violations were to become legion in all future AIDS drug clinical trials, versus the practice of conducting controlled, and complete experiments that are not changed in mid-stream, and in which patients are given full disclosure about the risks and uncertainties of an experimental drug.

In 1987, The London Times (May 11, 1987) reported that the incidence of AIDS infections in Africa coincides exactly with the locations of the W.H.O. smallpox vaccination program in the mid-1970’s. Some 14,000 Haitians then on UN secondment to Central Africa were also vaccinated in this campaign. Personnel actually conducting the vaccinations may have been completely unaware that the vaccine was anything other than what they were told.

**CHANGING THE DEFINITION OF AIDS TO MEAN “HIV-INFECTED” WELL PEOPLE WITH THE MOLECULAR MARKER OF “HIV:” “HIV” BECOMES A MOLECULAR DISEASE IN “HEALTHY SICK PEOPLE.”**

Several other events of importance occurred in 1987. In March President Ronald Reagan and French Prime Minister Jacques Chirac announced a joint agreement settling the dispute arising from the discovery of the AIDS virus, the first international agreement relating to a biomedical research issue to be announced by heads of state. In April FDA approved the first Western blot blood test. In June NHLBI awarded a contract to maintain a colony of 50 chimpanzees for studies of post-transfusion HIV infection and AIDS, and most importantly, in August, the CDC revised its definition of AIDS to place a greater emphasis on counting AIDS patients as those who are healthy but exhibit the molecular signature of "HIV." This "greater emphasis on "HIV infection" status served to dramatically increase the number of AIDS cases overnight, because healthy persons who presented with the molecular markers of "HIV" were in future to be counted as AIDS patients. In December, the CDC released the results of a study on the prevalence of HIV infection in the United States, indicating a shifting emphasis toward defining AIDS as "infection with HIV" rather than by defining particular "indicator diseases" that characterized late-stage AIDS [1].

At that same time, it became increasingly more apparent to many who were studying Apartheid and the devastation of African lives due to colonialist policies of the past, that Africa had been devastated by the intervention of Western medicine and medical practices such as routine vaccination and revaccination, although no mechanism was proposed for this carnage:

London Times Edition 1 MON 11 MAY 1987 **Smallpox vaccine 'triggered Aids virus'** BY PEARCE WRIGHT, SCIENCE EDITOR

***The Aids epidemic may have been triggered by the mass vaccination campaign which eradicated smallpox. The World Health Organization, which masterminded the 13-year campaign, is studying new scientific evidence suggesting that immunization with the smallpox vaccine Vaccinia awakened the unsuspected, dormant human immuno defence virus infection (HIV). Some experts fear that in obliterating one disease, another disease was transformed from a minor endemic illness of the Third World into the current pandemic. While doctors now accept that Vaccinia can activate other viruses, they are divided about whether it was the main catalyst to the Aids epidemic. But an adviser to WHO who disclosed the problem, told The Times: 'I thought it was just a coincidence until we studied the latest findings about the reactions which can be caused by Vaccinia. Now I believe the smallpox vaccine theory is the explanation to the explosion of Aids.' 'In obliterating one disease, another was transformed.' Further evidence comes from the Walter Reed Army Medical Centre in Washington. While smallpox vaccine is no longer kept for public health purposes, new recruits to the American armed services are immunized as a precaution against possible biological warfare. Routine vaccination of a 19-year-old recruit was the trigger for stimulation of dormant HIV virus into Aids. This discovery of how people with subclinical HIV infection are at risk of rapid development of Aids as a vaccine-induced disease was made by a medical team working with Dr Robert Redfield at Walter Reed. The recruit who developed Aids after vaccination had been healthy throughout high school. He was given multiple immunizations, followed by his first smallpox vaccination. Two and a half weeks later he developed fever, headaches, neck stiffness and night sweats. Three weeks later he was admitted to Walter Reed suffering from meningitis and rapidly developed further symptoms of Aids and died after responding for a short time to treatment. There was no evidence that the recruit had been involved in any homosexual activity. In describing their discovery in a paper published in the New England Journal of Medicine a fortnight ago, the Walter Reed team gave a warning against a plan to use modified versions of the smallpox vaccine to combat other diseases in developing countries. Other doctors who accept the connection between the anti-smallpox campaign and the Aids epidemic now see answers to questions which had baffled them. How, for instance, the Aids organism, previously regarded by scientists as 'weak, slow and vulnerable,' began to behave like a type capable of creating a plague. Many experts are reluctant to support the theory publicly because they believe it would be interpreted unfairly as criticism of WHO. In addition, they are concerned about the impact on other public health campaigns with vaccines, such as against diphtheria and the continued use of Vaccinia in potential Aids research. The coincidence between the anti-smallpox campaign and the rise of Aids was discussed privately last year by experts at WHO. The possibility was dismissed on grounds of unsatisfactory evidence. Advisors to the organization believed then that too much attention was being focussed on Aids by the media. It is now felt that***

doubts would have risen sooner if public health authorities in Africa had more willingly reported infection statistics to WHO. Instead, some African countries continued to ignore the existence of Aids **even after US doctors** alerted the world when **the infection spread to the United States**. However, as epidemiologists gleaned more information about Aids from reluctant Central African countries, clues began to emerge from the new findings when examined against the wealth of detail known about smallpox as recorded in the Final Report of the Global Commission for the Certification of Smallpox Eradication. **The smallpox vaccine theory would account for the position of each of the seven Central African states which top the league table of most-affected countries; why Brazil became the most afflicted Latin American country; and how Haiti became the route for the spread of Aids to the US. It also provides an explanation of how the infection was spread more evenly between males and females in Africa than in the West and why there is less sign of infection among five to 11-year-olds in Central Africa. Although no detailed figures are available, WHO information indicated that the Aids league table of Central Africa matches the concentration of vaccinations. The greatest spread of HIV infection coincides with the most intense immunization programmes, with the number of people immunised being as follows: Zaire 36,878,000; Zambia 19,060,000; Tanzania 14,972,000; Uganda 11,616,000; Malawai 8,118,000; Ruanda 3,382,000 and Burundi 3,274,000. Brazil, the only South American country covered in the eradication campaign, has the highest incidence of Aids in that region. About 14,000 Haitians, on United Nations secondment to Central Africa, were covered in the campaign. They began to return home at a time when Haiti had become a popular playground for San Francisco homosexuals. Dr Robert Gello, who first identified the Aids virus in the US, told The Times: ‘The link between the WHO programme and the epidemic in Africa is an interesting and important hypothesis. ‘I cannot say that it actually happened, but I have been saying for some years that the use of live vaccines such as that used for smallpox can activate a dormant infection such as HIV. ‘No blame can be attached to WHO, but if the hypothesis is correct it is a tragic situation and a warning that we cannot ignore.’** Aids was first officially reported from San Francisco in 1981 and it was about two years later before Central African states were implicated. It is now known that these states had become a reservoir of Aids as long ago as the later 1970s. Although detailed figures of Aids cases in Africa are difficult to collect, the more than two million carriers, and 50,000 deaths, estimated by the World Health Organization are concentrated in the Countries where the smallpox immunization programme was most intensive. The 13-year eradication campaign ended in 1980, with the saving of two million lives a year and 15 million infections. The global saving from eradication has been put at dollars 1,000 million a year. Charity and health workers are convinced that millions of new Aids cases are about to hit southern Africa. After a meeting of 50 experts near Geneva this month it was revealed that up to 75 million, one third of the population, could have the disease within the next five years. Some organizations which have closely studied Africa, such as War on Want, believe that South Africa’s black population, so far largely protected from the disease, could be most affected as migrant workers bring it into the country from the worst hit areas further north. **The apartheid policy, they predict, will intensify its outbreak by confining the groups into comparatively small, highly populated towns where it will be almost impossible to contain its spread.**

In **1988**, several important events occurred. In January, The CDC updated the International Classification of Diseases codes for HIV infection for use with U.S. morbidity and mortality data [1], and in June, **the brochure “Understanding AIDS,” prepared by Surgeon General C. Everett Koop in collaboration with the CDC, was mailed to every household in the United States**. In August, NIAID’s AIDS Vaccine Evaluation Units initiated their first study of an experimental AIDS vaccine. In September, NIDR investigators reported that saliva inhibits transmission of HIV, and in October, AIDS protestors, demanding a quicker approval process for drug treatments, shut down the FDA. In October, NIAID established three programs: the Centers for AIDS Research (CFARS) to improve the diagnosis, treatment, and prevention of AIDS; the Pediatric AIDS Clinical Trials Units (Pediatric ACTUs), a network of clinical sites to test experimental drugs on children; and the Programs for Excellence in Basic Research (PEBRA), to develop novel strategies to determine how HIV causes disease [1].

**PAPADOPULOS-ELEOPULOS – AIDS MAY BE CAUSED BY OXIDATION AND REDOX IMBALANCE**

By **1987-88**, a new hypothesis was published based on how “HIV” culturing techniques and test kits like the ELISA and WESTERN blot tests were seriously flawed, coupled to the idea that attempted to explain the pathogenesis of AIDS as the result of a disturbance in cellular reduction/oxidation reactions as had been demonstrated by many groups In Vitro [17]. As hypothesized by Papadopoulos-Eleopoulos and The Perth group:

There are good reasons to doubt that HTLV-III/LAV can be regarded as the exclusive single variable in the pathogenesis of AIDS. There is therefore a spectrum of possibilities. Either it plays no role at all, is of minor significance or it contributes significantly but not exclusively to the disease. Be that as it may the one major significant variable is the concurrent exposure of the patients to oxidising agents including sperm, nitrites, opiates and factor VIII. If this is true then prevention, and possibly even cure, may be achieved with the use of appropriate antioxidants.

Part of the basis of this hypothesis derived from the fact that Gallo and Montagnier’s groups both had used PHA (phytohemagglutinin-a plant lectin), IL-2 (interleukin-2), and other oxidizing chemicals believed to stimulate T-cells to divide several times, which was necessary to induce lymphocytes or continuous cell lines to generate the molecular signature of “HIV” after exposure to AIDS patient sera, or after exposure to supernatants from cells induced to express “HIV’s” molecular signature. AIDS patients, Perth argued, also are highly oxidized, through over-exposure to various things such as sperm, drugs, and foreign proteins. Also Papadopoulos-Eleopoulos et al. (The Perth Group) were the first who rigorously questioned that isolation of “HIV” had been achieved independently of cellular debris and the virus-like particles that artificial oxidation or other stimulation of T-cells causes.

Papadopoulos-Eleopoulos et al. argued that although electron micrographs and computer-generated models of what have been identified as “HIV” viral particles can easily be found on numerous public access sites as well as in the CDC’s information websites, the Big Picture Book of Viruses, from text books, and from numerous other information sources, they look different in every picture. They either do or do not have surface projections, they either are or are not harboring a nucleic acid core, they are or are not of similar sizes, and they are always accompanied by excessive quantities of cellular debris or junk which is the most specific hallmark of viral isolation preps that claim to harbor the molecular signature of “HIV,” and importantly, on WESTERN blots, their proteins are present and can be seen as faint bands in samples from supposedly non-infected sources.

Thus the PERTH group more than anyone else, first championed, and subsequently provided rigorous testable arguments that “HIV” has not been isolated, and therefore claimed that the evidence for its non-existence is far greater than for its existence. This analysis can be seen in many of the impressive papers they have published (all of which can be obtained on line at <http://www.virusmyth.net/aids/find.htm>), and in articles and letters of theirs that were censored, all of which can be found on the website: “virusmyth.com” and on Perth’s own website.

‘Reappraisal of AIDS’ (1988)

Kaposi’s Sarcoma and HIV’ (1992)

‘Oxidative Stress, HIV and AIDS’ (1992)

‘Is a Positive Western Blot Proof of HIV Infection?’ (1993)

‘Has Gallo Proven the Role of HIV in AIDS?’ (1993)

‘AIDS in Africa: Distinguishing Fact and Fiction’ (1995)

‘HIV Seropositivity and Mortality in Persons with Haemophilia’ (1995)

'Factor VIII, HIV and AIDS in Haemophiliacs' (1995)

'A Critical Analysis of the HIV-T4-Cell-AIDS Hypothesis' (1995-this one is my personal favorites)!

'AIDS in Africa?' (1995)

'AIDS in Thailand' (1995)

'Isolated Facts About HIV – A Reply' (1996)

'The Isolation of HIV; Has It Really Been Achieved?' (Reply Duesberg I) (1996)

'Do HIV Antibody Tests Prove HIV Infection?' (1996)

'What Is the Evidence for the Existence of HIV?' (1996)

'The AIDS Virus: Matter or Metaphor ?' (1997)

'HIV Antibodies: Further questions and a plea for clarification' (1997)

'Comments on Montagnier Interview' (1997)

'Where Have We Gone Wrong' (1998)

'Patient to Patient Transmission of HIV in a Surgeon's Private Rooms' (1998)

'HIV Antibody Tests and Viral Load' (1998)

'Geneva Presentation' (1998)

'The Yin and Yang of HIV' (1999)

'A Critical Analysis of the Pharmacology of AZT and its Use in AIDS' (1999)

'The Last Debate' (1999)

'HIV Testing and Surveillance' (2000)

Reply to Durban Declaration (2000)

Correspondence With Prof. William Makgoba (2000)

'Mother to Child Transmission of HIV and its Prevention With AZT and Nevirapine' (2001)

Presentation on Nevirapine (2002).

**Here's a list of some of the censored Papandopulos-Eleopulos et al. work:**

Gallo's experiments and a plea for clarification

Perth Group reponse to Nature and its Durban Declaration

gp120 and HIV infection

Commentary on the Ho and Wei Nature papers

Concerning TB in Africa

Commentary on AZT

Anti-HIV antibodies, ARVs and informed consent

HIV transmission from chimpanzees

A critical analysis of HIV serology does not affirm a retroviral infection

A critical appraisal of the evidence for the existence of HIV

Commentary on the Gallo/Montagnier HIV as the cause of AIDS paper

Response to Declan Butler

Letter to Lancet in response to the WHO

Letter to NEJM July 2004

Letter to BMJ

Letter to Nature Medicine

Letter to Lancet

Letter to Structure

The HIV Genome

**RESEARCHER OF VIRAL BUDDING AND MOUSE RETROVIRUSES ASSERTS THAT "HIV" HASN'T BEEN ISOLATED, ACCORDING TO STANDARDS USED TO ESTABLISH THE REALITY OF OTHER VIRUSES**

In agreement with Papadopoulos-Eleopoulos et al., and in a short piece entitled, "*Remarks on methods for retroviral isolation,*" Dr. Etienne de Harven who is an emeritus Professor of Pathology, University of Toronto, and who worked out the ultrastructure of "retroviruses" throughout his professional career of 25 years at the Sloan Kettering Institute in New York and for 13 years at the University of Toronto, vociferously objected to the standards used to claim that "HIV" viral particles had been convincingly isolated. In 1956 he was among the first to show electron microscope images of the Friend virus in murine (mouse) leukemia, and in 1960, studied viral "budding" assembly on cell surfaces. He also delivered a speech at the 12th World AIDS Conference in Geneva (June



28-July 3) at the session entitled, “HIV-testing: Open Questions about Specificity,” and in his concluding remarks said:

*In conclusion, and after extensive reviewing of the current AIDS research literature, the following statement appears inescapable: neither electron microscopy nor molecular markers have so far permitted a scientifically sound demonstration of retrovirus isolation **directly from AIDS patients.***

In Continuum de Harven also had written:

*I published the following picture in 1965 in a paper entitled Viremia in Friend Leukemia: the electron microscope approach to the problem which appeared in Pathologie-Biologie, vol. 13, pp. 125-134. Transmission electron microscopy was used **to verify the success of a method for virus purification** which I had developed when working at the Sloan Kettering Institute in New York.*

In the reference he cites it is stated:

*The first successful purification of a murine leukemia virus from the blood of leukemic mice was reported by Moloney and Dalton. The technique described by these authors has been recently combined with density gradient centrifugation and a very successful purification of the Rauscher viruses has been achieved. Our method of purification of the Friend virus was originally derived from that recommended by Moloney and Dalton. The critical point of the method is **to take advantage of the existence of a “free” plasma viremia rather than of the “cell-associated” viremia.***

In another letter, de Harven reiterated the Perth’s group’s reasoning that: (1) The human placenta is loaded with HERVs, (2) Lymphocytes from the umbilical cord blood are therefore very likely to carry the same HERVs, (3) Such lymphocytes were added to the mixed cell cultures of Barre-Sinoussi and Montagnier at Pasteur in 1983, (4) The EM picture in the 1983 Barre-Sinoussi et al. paper simply demonstrate that, under PHA and TCGF stimulation, these placental lymphocytes express [viral-like particles], by “budding” off their HERVs, (5) This observation has nothing to do with the innoculum from an AIDS patient and is no proof of the exogenous infection of these lymphocytes by hypothetical retroviruses originating from outside the organism.

## **CELIA FARBER, AZT, AND THE SINS OF OMISSION**

In 1989, the investigative journalist, Celia Farber published the controversial piece, “Sins of Omission,” in *Spin Magazine*, which emphasized the toxicity and the rushed FDA approval of AZT with statements like:

*But there were tremendous concerns about the new drug. It had actually been developed a quarter of a century earlier as a cancer chemotherapy, but was shelved and forgotten because it was so toxic, very expensive to produce, and totally ineffective against cancer. Powerful, but unspecific, the drug was not selective in its cell destruction.*

*Drug companies around the world were sifting through hundreds of compounds in the race to find a cure, or at least a treatment, for AIDS. Burroughs Wellcome, a subsidiary of Wellcome, a British drug company, emerged as the winner. By chance, they sent the failed cancer drug, then known as Compound S, to the National Cancer Institute along with many others to see if it could slay the AIDS dragon, HIV. In the test tube at least, it did.*

*At the meeting, there was a lot of uncertainty and discomfort with AZT. The doctors who had been consulted knew that the study was flawed and that the long-range effects were completely unknown. But the public was almost literally baying at the door. Understandably, there was immense pressure on the FDA to approve AZT even more quickly than they had approved thalidomide in the mid-60s, which ended up causing drastic birth defects.*

Everybody was worried about this one. To approve it, said Ellen Cooper, an FDA director, would represent a “significant and potentially dangerous departure from our normal toxicology requirements.”

*Flash forward: August 17, 1989. Newspapers across America banner-headlined that AZT had been “proven to be effective in HIV antibody-positive, asymptomatic and early ARC patients,” even through one of the panel’s main concerns was that the drug should only be used in a last-case scenario for critically-ill AIDS patients, due to the drug’s extreme toxicity. Dr. Anthony Fauci, head of the National Institutes of Health (NIH), was now pushing to expand prescription.*

*Burroughs Wellcome stock went through the roof when the announcement was made. At a price of \$8,000 per patient per year (not including blood work and transfusions), AZT is the most expensive drug ever marketed. Burroughs Wellcome’s gross profits for next year are estimated at \$230 million. Stock market analysts predict that Burroughs Wellcome may be selling as much as \$2 billion worth of AZT, under the brand name Retrovir, each year by the mid-1990s – matching Burroughs Wellcome’s total sales for all its products last year.*

*As it happened, AZT was rampantly prescribed as soon as it was released, way beyond its purported parameters. The worst-case scenario had come true: Doctors interviewed by the New York Times later in 1987 revealed that they were already giving AZT to healthy people who had tested positive for antibodies to HIV.*

*AZT was singled out among hundreds of compounds when Dr. Sam Broder, the head of the National Cancer Institutes (NCI), found that it “inhibited HIV viral **replication In Vitro.**”*

***The last surviving patient from the original AZT trial, according to Burroughs Wellcome, died recently. When he died, he had been on AZT for three and one-half years. He was the longest surviving AZT recipient. The longest surviving AIDS patient overall, not on AZT, has lived for eight and one-half years.***

*The news that AZT will soon be prescribed to **asymptomatic people** has left many leading AIDS doctors **dumbfounded and furious.** Every doctor and scientist I asked felt that it was **highly unprofessional and reckless** to announce a study **with no data to look at**, making recommendations with such drastic public health implications. “This simply does not happen,” says Bialy. “**The government is reporting scientific facts before they’ve been reviewed? It’s unheard of.**”*

“It’s beyond belief,” says **Dr. Sonnabend** in a voice tinged with **desperation.** “I don’t know what to do. I have to go in and face an office full of patients asking for AZT. I’m terrified. **I don’t know what to do as a responsible physician.** The first study was ridiculous. Margaret Fishl, who has done both of these studies, obviously doesn’t know the first thing about clinical trials. I don’t trust her. Or the others. They’re simply not good enough. We’re being held hostage by second-rate scientists. We let them get away with the first disaster; now they’re doing it again.

## **SHYH-CHING LO’S MYCOPLASMA INCOGNITUS FOUND IN 22/34 AIDS PATIENTS AND IN ZERO NON-AIDS PATIENTS**

In **1989-1990**, a series of articles published by Shyh-Ching Lo of the Armed Forces Institute of Pathology was discussed in AIDS TREATMENT NEWS No. 095 – January 26, 1990, written by John S. James:

*MYCOPLASMA INCOGNITUS: Newly Discovered Treatable Opportunistic Infection?*

*Researchers at the U. S. Armed Forces Institute of Pathology (AFIP) in Washington, D. C., and the Warren Grant Magnuson Clinical Center at the National Institutes of Health, have found compelling evidence that a previously unrecognized opportunistic*

infection — one potentially treatable with antibiotics — may be a major cause of illness in people with AIDS. Many infections of organs including the brain, spleen, liver, or lymph nodes — as well as some systemic infections — might be caused by the newly-discovered organism, called **Mycoplasma incognitus**. Until now, these infections would be counted among the many which cannot be diagnosed. While the first report of the organism now known as *Mycoplasma incognitus* was published over three years ago, most of what is now known was learned later and published last year. And only in the last few weeks has the AIDS research community paid serious attention.

Until recently the new organism was mistakenly believed to be a virus, and its discovery seemed to have little immediate relevance to treatment. Then a series of five articles by Shyh-Ching Lo and others in the *American Journal of Tropical Medicine and Hygiene*, between February and November 1989, showed: (1) The new organism is a mycoplasma — which is potentially treatable. *Mycoplasma*, a form of life between bacteria and viruses in complexity, was discovered about 100 years ago. Some species are known to cause human diseases.

The published articles only hint that the new organism might be treatable with antibiotics. But scientists at AFIP tested 15 common antibiotics against the *Mycoplasma incognitus* in the laboratory. A detailed report is being prepared for publication, but because of the public-health importance of the information, AFIP released a list of the drugs and their effective concentrations in a separate document. Doxycycline, tetracycline, clindamycin, lincomycin, and ciprofloxacin were found to be effective against *Mycoplasma incognitus*. **But erythromycin, the antibiotic most commonly used to treat mycoplasma infections, was not effective — and penicillin, streptomycin, gentamicin, and others also had no effect. (2)**

*Mycoplasma incognitus* was found in the thymus, liver, spleen, lymph node, or brain of **22 of 34** persons who **had died of AIDS**. The patients who were selected for this autopsy study had all had evidence of organ failures. (3) In a separate study with different patients, the mycoplasma was found **in seven of ten persons** with AIDS. Also, a much earlier study had found *Mycoplasma incognitus* in blood lymphocytes of **12 of 23 living persons with AIDS — but in none of 22 healthy blood donors used as controls**. (4) The mycoplasma was also found in six HIV-negative patients (with no sign of AIDS) from different parts of the world, who had died in one to seven weeks of an undiagnosed infection. No one knows how the organism spreads, but evidently it is not by casual contact, as family members of infected persons have not become infected themselves. (5)

**Four monkeys were injected with *Mycoplasma incognitus*; all died in seven to nine months.** The organism was found in the **spleens** of all the monkeys, and in some other organs as well. It was not found in a fifth monkey tested as a control. (6)

Extensive evidence **from electron-microscope examinations**, from specially designed PCR tests to look for the DNA of *Mycoplasma incognitus*, and from immunologic tests, showed that **the organism was concentrated in lesions in affected organs**. *Mycoplasma incognitus* **is unusual** in that it often infects and kills tissue **without** causing an inflammatory reaction, suggesting that it disables or evades part of the immune system.

The publication of this evidence, much of it in November 1989, led to a meeting between Dr. Anthony Fauci, director of NIAID (the National Institute of Allergy and Infectious Diseases) and other AIDS experts, with Dr. Lo and his colleagues at AFIP. The meeting, on December 14, 1989 in San Antonio, was chaired by Dr. Joel B. Baseman, chairman of the Department of Microbiology at the University of Texas Health Sciences Center in San Antonio, an expert on mycoplasma. An article in *THE WASHINGTON POST* (January 5) quoted Dr. Baseman as saying that Lo's mycoplasma "might be a significant agent for many infectious diseases, not just AIDS. There is enough information to say that this agent is real." The same article quoted Dr. Fauci as saying that *Mycoplasma incognitus* "may be an important opportunistic infection ...If it's real, it could have an important impact on how doctors look at AIDS patients with unexplained problems." An in-depth history of the discovery of *Mycoplasma incognitus* and its early dismissal by parts of the scientific community was published in *THE NEW YORK TIMES*, January 16, 1990. What Should Be

Done Now? Awareness of the new importance of *Mycoplasma incognitus* has not yet spread far in the medical community. The biggest problem is that there is no readily available test for the organism; at this time, there may be only one research lab which can do the test reliably. Other mycoplasmologists are becoming involved, however, and a much easier blood test is being developed. In addition, clinical trials are now being planned. **The AIDS physician and patient community should help make sure that trials proceed quickly.** There may also be immediate uses of the new information, for example: (1) Empirical Use of Antibiotics Several months ago, before *Mycoplasma incognitus* was known, Dr. Nathaniel Pier mentioned that he had found good results trying **doxycycline** for patients who had an apparent infection which could not be diagnosed. (Doxycycline is the antibiotic most often discussed as a possible treatment for *Mycoplasma incognitus*; however neither it nor any other antibiotic has yet been tested for treating this infection in humans.) Incidentally, the next physician we asked about empirical use of antibiotics preferred erythromycin, which would not be effective against the mycoplasma. The discovery of *Mycoplasma incognitus* provides an additional rationale for trying doxycycline (or one of the other antibiotics found effective against this organism in the laboratory) for certain patients, such as those with signs of undiagnosed infection, especially in the central nervous system, spleen, or certain other organs. Patients should know that antibiotics can cause side effects — some of which, such as overgrowth of *Candida*, might be more severe in persons with HIV.

For some reason, the recommendations never were developed whereby, **“The AIDS physician and patient community should help make sure that trials proceed quickly”**.

In **1989**, Harold Varmus and Stephen Bishop shared the Nobel Prize in Physiology or Medicine “for their discovery of the cellular origin of retroviral oncogenes.” Harold Varmus, in his Nobel lecture, described the ability of scientists up until that time to distinguish between viral and cellular oncogenes, and viral versus cellular reverse transcriptases [18]. He also published a mini-review in the prestigious journal, *Cell*, describing reverse transcriptase in bacteria [19].

#### **A MACARTHUR FELLOW SPECULATES: ANTI-HLA ALLO-IMMUNIZATION CAUSES AIDS.**

In **1990**, the noted historian of science, immunologist, and MacArthur Fellow, Robert Root-Bernstein wrote an article in *Perspectives in Biology and Medicine* [20]:

*Most investigators believe that acquired immunodeficiency syndrome (AIDS) is caused solely by human immunodeficiency virus (HIV) (1,2). However several puzzling facts cast doubt on this conclusion: about 5 percent of all AIDS patients have been tested for seroconversion (3); seroconversion may not indicate active infection but, rather, a successful immunological response to HIV, at least in some cases (47); a single T lymphotropic virus cannot explain the simultaneous immunosuppression of T cells, natural killer cells, B cells, and macrophages that characterizes the immune system of AIDS patients (47); several other immunosuppressive viruses and bacteria are as highly correlated with the syndrome as is HIV (8); Koch’s postulates have not been satisfied, nor have chimpanzees infected with HIV displayed any of the typical symptoms of AIDS (67); and seroconversion following HIV exposure is so varied (anything from seroconversion after a single unprotected sexual contact with an HIV carrier to no seroconversion after hundreds of unprotected encounters) that even HIV proponents are admitting that there must exist some “as yet unexplained biologic variation in transmissibility or susceptibility” to HIV infection (9). Indeed, between 30 and 100 hemophiliacs may use the same lot of clotting factor concentrates, and yet there are no reported cases of more than one hemophiliac developing AIDS from an AIDS-donorcontaminated lot (10, 11).*

Furthermore, there is a logical problem that is often overlooked by uncritical HIV proponents: AIDS patients die **of previously identified diseases**, not of HIV infection per se. **That is why AIDS is a syndrome, not a distinct disease entity.** Thus, the putative role of HIV is solely to cause the immunosuppression that sets the stage for subsequent fatal opportunistic infections. But before we can accept HIV as the sole cause of immunosuppression characteristic of AIDS patients, it is necessary to assure ourselves

that alternative explanations of the data do not exist. After all, theories, just like experiments, need controls; for just as experimental artifacts are reproducible, so can a theory explain existing data and yet, as Aristotle's theory of falling bodies demonstrates, still not be the best explanation. In the present context we must, therefore, before accepting HIV as the sole cause of the immunosuppression typifying AIDS, demonstrate directly that HIV actually does cause immunosuppression in animals or human beings and also assure ourselves that other immunosuppressive agents cannot explain the etiology of AIDS. In other words, we must determine that the HIV theory is necessary and sufficient to explain AIDS and that no other theory is necessary or sufficient. Are there, for example, individuals who are immunosuppressed whose sole infection is HIV? If so, then we can assure ourselves that HIV is sufficient to cause immunosuppression. Do AIDS patients in general have any identified immunosuppressive risks other than HIV? If so, are these sufficient to explain the immunosuppression associated with AIDS in the absence of HIV, or not?

Existing data do not, as yet, allow us to establish HIV as the unequivocal cause of immunosuppression in AIDS. No nonhuman animal other than the chimpanzee appears to be infected by HIV, and HIV-infected chimpanzees do not display longterm immunological abnormalities (6,7). Moreover, all AIDS patients do have multiple, well-established causes of immunosuppression prior to, concomitant with, subsequent to, and sometimes in the absence of, HIV infection. These immunosuppressive agents are of seven basic types: chronic or repeated infectious diseases caused by immunosuppressive microorganisms; recreational and addictive drugs; anesthetics; antibiotics; semen components; blood; and malnutrition. **While no AIDS patient is likely to encounter all of these agents, all AIDS patients encounter several.** Healthy heterosexuals and lesbians rarely encounter more than one. Therefore, the conclusion that HIV is the sole cause of immunosuppression in AIDS, and the sole factor differentiating AIDS patients from non-AIDS patients, cannot be maintained, and alternative hypotheses remain possible?

Intravenous drug abusers also share an immunosuppressive risk factor with hemophiliacs and blood transfusion recipients: **they receive other people's blood.** I am unable to find any data concerning the immunological effects of small doses of untyped blood such as drug abusers might encounter repeatedly by sharing needles: however, **it is a well established principle of immunology that repeated injections of very small amounts of almost any antigen eventually result in suppression of the immune response (72).** Given that these small, repeated blood injections will include a proportion of leukocytes, it is likely that immunosuppression to various HLA types will eventually occur. This mechanism of immunosuppression has previously been suggested for semen, which also contains small numbers of leukocytes."

**Anti-HLA alloimmunization** has already been observed **in multiply transfused patients (74,75).** Repeated use of anti-clotting factors results in **abnormal** suppressor: helper T-cell ratios even among otherwise healthy hemophiliacs and even in countries like Australia, in which HIV is virtually nonexistent (76, 78). Almost every hemophilic also contracts **hepatitis (79)** and presumably various other viral agents that are transmissible in blood, such as **CMV and EBV (80, 82, 84).** Physicians have known for over a decade that blood transfusions **depress** the immune response **effectively enough to facilitate the acceptance of organ transplants and to increase significantly the risk of death from cancer (85, 89).** This immunosuppression is **dose related**, and it is therefore significant that the average transfusion-related AIDS patient receives blood from **16 to 21 donors** – five or more times that of the average **surgery patient** (a statistically significant difference) (90, 91). Although the exact mechanism of transfusion induced immunosuppression **is unknown**, T cells are certainly **a primary target, and B cells and macrophages are also involved.** Thus, recent studies show that anyone receiving multiple transfusion or blood-derived products such as clotting agents-hemophilacs, those with sickle cell anemia (92), trauma patients (93), and surgery patients (94)-are at high risk for developing the lymphadenopathy, low helper T cell: suppressor T cell ratio, and low-grade fever associated with AIDS related complex (ARC). These symptoms generally precede HIV seroconversion.

**"HIV" FOUND IN NON-INFECTED ALLOIMMUNIZED MICE, NORMAL HUMAN PLACENTAS, AND IN 1/250 HUMAN SUBJECTS INCLUDING BERGALIS AND GERTZ, IN NON-INFECTED MONKEYS AND CHIMPS, AND IN**

## HUMANS AFTER HEPATITIS B VACCINATION.

In **1991**, Science published a paper claiming that mice exposed to alloimmune serum or cells from other mice said not to be “HIV” positive will test positive for p24 and GP120, two supposedly “HIV-specific” molecules. In the same year, the so-called specific marker of “HIV,” p24, was reportedly found in normal (non-“HIV-infected”) human placentas [21].

Also In **1991** the CDC announced that five patients had likely become infected in the course of treatment by Dr. Acer. Ms. Bergalis’s case sparked a firestorm of controversy and intense public policy debate concerning HIV testing in health care settings and possible strictures on practicing by HIV-positive health care workers. Ms. Bergalis, who died from AIDS in December 1991, blamed her illness on public health officials in a highly publicized letter published in Newsweek (Kantrowitz, 1991). In media appearances and in congressional testimony, the Bergalis family launched a campaign calling for mandatory testing of health care workers and disclosure of test results.

*The issue of disclosure of caregivers’ HIV status to patients is a critical policy concern. In a Gallup poll conducted among 1,014 adults in May 1991, 87 percent of the general public believed that doctors and dentists should be tested for AIDS, and 84 percent believed that nurses should be tested. In another study, more than one-half of patients surveyed said they would seek care elsewhere if they found out their physicians were caring for people with HIV disease (Gerbert et al., 1989).*

*In July 1991, largely as a result of the Bergalis case, the CDC published new guidelines reiterating the need for strict adherence to universal precautions and infection control procedures (Centers for Disease Control, 1991). The guidelines also stated that health care workers who perform “exposure-prone invasive procedures” should know their HIV status. Infected health care workers were directed not to perform such procedures unless they sought counsel from an expert panel as to the circumstances, “if any,” under which it would be appropriate to do so. A few days before the guidelines were issued, an additional clause was added at the behest of Senator Orrin Hatch (R-Utah) and Health and Human Services Secretary Louis Sullivan (Gautier, 1991): the “informed consent” provision in the CDC guidelines requires that HIV-infected health care workers disclose their seropositivity to prospective patients undergoing exposure-prone, invasive procedures.<sup>8</sup>*

*On August 16, 1991, the CDC had announced the initiation of a process to develop a list of exposure-prone, invasive procedures to serve as a guide for local review bodies. Although the CDC anticipated completing the development of such a list by November 15, 1991, in order to clarify what it wanted the States to do, a November 4 meeting revealed widespread discontent among the medical and public health community with CDC’s approach (Altman, 1991). **Virtually all medical and professional associations except the American Medical Association refused to cooperate in the list-making endeavor because they were not convinced that there existed sufficient scientific evidence on which to base such a list.** In October 1991 Congress passed an appropriations bill that included an amendment requiring states to institute the CDC guidelines or their “equivalent” **or risk the loss of Public Health Service funds** (Treasury, Postal Service, and General Government Appropriations Act, 1992, P.L. 102-141 Sec. 633 (1991)). However, on July 30, 1992, the National Commission on AIDS recommended **against** both mandatory testing for doctors and mandatory notification of patients because the risks of infection are so remote and disclosure (National Commission on AIDS, 1992; Leary, 1992).*

Peter Duesberg had a different take on the Acer-Bergalis affair: From: With Therapies Like This, Who Needs Disease?—of the book Inventing the AIDS Virus by Peter Duesberg. 1996. From: The Stories of Those Who Believed in AZT.

*Kimberly Bergalis: The story began in late 1986, in the small town of Stuart on Florida’s Atlantic coast. David Acer, a dentist who had begun his private practice five years earlier, felt a bit under the weather and saw a physician. Acer was also **accused** of being an active homosexual, a fact that led him to seek an HIV test. The result came back positive. Although disturbed by the news, he still felt reasonably healthy and saw no reason to stop his dental practice, nor apparently his fast-track lifestyle.*

One year later he experienced worsening symptoms and a visit to his doctor confirmed the diagnosis: full-blown AIDS. A Kaposi's sarcoma covered the inside of his throat and his T-cell count had fallen dangerously low. Both symptoms suggested the extensive use of poppers and other drugs so common in the homosexual bathhouse scene. Acer could see his life slowly wasting away. He continued practicing dentistry while remaining discreet about his sexual life and failing health, making sure to follow the standard guidelines for protecting his patients from infection.

That December, in 1987, he pulled two molars from a nineteen-year-old college student, Kimberly Bergalis. At the time he had no idea the business major would one day be touted as his hapless victim.

The story picks up again in May 1989, when Bergalis developed a transient oral yeast infection. Later that year, during the emotional stress of preparing for an actuarial exam for the state of Florida, she felt some ongoing nausea, and she became dizzy during the test itself. Afterward, the symptoms disappeared. But a brief pneumonia that December sent her to the hospital, where the doctor decided out of the blue to test her for HIV. As chance would have it, she had antibodies against the virus.

Up to this point, none of her occasional diseases differed from the common health problems many HIV-negative people encounter. But the positive HIV test changed her whole attitude, as well as her medical treatment. Within three months the CDC had heard of her case, possibly aided by the presence of several EIS members in the Florida health department, and sent investigators to probe further. The CDC team included such EIS members as Harold Jaffe, Ruth Berkelman, and Carol Ciesielski. Bergalis denied any intravenous drug use or blood transfusions and insisted she was a virgin. **During the prolonged examination, the CDC officers stumbled across David Acer's positive HIV status and made the connection to Bergalis.** Before the HIV hypothesis of AIDS, no medical expert in his right mind would ever have entertained the slightest thought that a dentist with a Kaposi's tumor and a patient with a yeast infection had anything in common. But in the era of AIDS, doctors tended to discard common sense. That the dentist and patient both carried a dormant virus was enough.

Excited by its discovery, the CDC boldly advertised its results in its weekly newsletter, the same one that nine years earlier had broadcast the first five AIDS cases. The July 27, 1990, issue prominently featured their amazing leap of logic—that the dentist must somehow have infected Bergalis. Naturally, the CDC's speculation leapt straight to the front pages and prime-time television news broadcasts.

Acer died in early September 1990. Bergalis meanwhile sought medical care at the University of Miami, where she was treated with an unidentified "experimental" method. Certainly this was the appropriate place for such therapies. Margaret Fischl, the head of the Phase II AZT trial, worked at that medical center, which had served as one of the twelve facilities sponsored by Burroughs Wellcome for the study. So Bergalis was prescribed AZT.<sup>110</sup>

Suddenly she started a precipitous decline in health. In an angry letter, she partly acknowledged her symptoms resulted from the toxic drug:

"I have lived through the torturous ache that infested my face and neck, brought on by AZT. I have endured trips twice a week to Miami for three months only to receive painful IV injections. **I've had blood transfusions.** I've had a bone marrow biopsy. I cried my heart out from the pain."<sup>111</sup>

This represented only the beginning. Her yeast infection worsened and became uncontrollable, she lost more than thirty pounds, her hair gradually fell out, her blood cells died and had to be replaced with **transfusions**, and her muscles wasted away. Her fevers hit highs of 103 degrees, and by late 1990 her T-cell count had dropped from the average of 1,000 to a mere 43. She looked just like a chemotherapy patient—which she now was.

**The CDC saw its golden opportunity in the Bergalis case.** It publicized a second report on the Bergalis case, announcing its belief that four of Dr. Acer's other patients had also been infected by him, and even surveyed the patients of other HIV-positive doctors and dentists—suggesting that all HIV-positive patients had also been infected by their doctors. Such CDC-funded organizations as Americans for a Sound AIDS Policy (see Chapter 10) aggressively promoted public fear with these speculations. A media feeding frenzy resulted, with every major television talk show, and every national magazine, running scare stories.<sup>112</sup> The CDC's relentless publicity had its expected effect: **By mid-1991, more than 90 percent of the public believed HIV-positive doctors should be forced to inform their patients of their status, and a clear majority favored banning such doctors from medical practice.**<sup>113</sup> Many doctors, angered by the publicity campaign, “accused the federal Centers of Disease Control of unduly alarming the public.”<sup>114</sup>

The CDC certainly had an agenda behind its campaign. In July of 1991, the agency issued a set of proposed rules that would require doctors to follow extraordinarily burdensome measures, supposedly to protect their patients from HIV infection. By hyping up the Bergalis case, the CDC had created enough public panic and backlash to favor its proposed regulations. To dramatize the point, Bergalis was brought in to testify before a stunned Senate in October of 1991. Her muscles largely destroyed by AZT, she had to be brought in a wheelchair. Her furious testimony, whispered into the microphone, made a powerful emotional impact on the attentive congressmen and the television audience.

Congress soon passed a new law requiring the states to adopt the CDC guidelines—**or else begin losing federal funds.** When the medical profession resisted the new rules, the Occupational Safety and Health Administration (OSHA), which works closely with the CDC, stepped in with parallel rules of its own. **On threat of criminal prosecution, laboratory and medical workers must now follow incredibly restrictive regulations on their practices and equipment, and must deal with extra bureaucratic red tape.**

Blaming her deteriorating condition on the latent virus supposedly passed on by her dentist, Bergalis sued the Acer estate. She received a \$1 million award, plus unannounced compensation from the dentist's insurance company. She parceled out the money to a variety of friends, family members, and AIDS organizations, and told her father to purchase “a new, red Porsche and deliver it to my aunt with a large bow on top.”<sup>115</sup> Had she known better, she could have instead sued Burroughs Wellcome.

Bergalis died in December 1991 at twenty-three years of age, having taken AZT for up to two years. Her death became the ultimate symbol of the deadly powers of HIV. No one pointed out that, according to the HIV hypothesis, the virus should take ten years to kill its victims, particularly someone like Bergalis with no other risk factors. She had died within four years of her initial visit to Dr. Acer. As her symptoms would indicate, AZT must have killed her instead.

In December 1992, another former patient of Dr. Acer tested positive for HIV, but had no symptoms. Two months later, eighteen-year-old Sherry Johnson began taking AZT. She has since begun wasting away, admitting she periodically feels sick.

The CDC continued to exploit the Bergalis story as proof of the risk of doctor-to-patient HIV transmission. Some eleven hundred of Acer's two thousand former clients volunteered for HIV tests. Seven of these were positive, including Bergalis, two of them having standard risk factors for AIDS. That left five people who supposedly caught the virus from Acer. Expanding its search, the CDC tested almost sixteen thousand total patients of some thirty-two HIV-positive doctors around the country, finding eighty-four infected patients. Though admittedly baffled by how HIV could pass from doctors and dentists to the patients, the CDC nonetheless advertised the alleged threat. Curiously, when confronted with an unexpected outcome for an unproved test, the CDC did not proceed with caution. It published its findings in July 1990 without further verification.

Apart from HIV being a harmless virus, the evidence that this virus has ever been medically transmitted remains dubious. Based on their own research, insurance companies concluded that the HIV strains in the five patients were different from that in Acer,



meaning each caught it from a different source.<sup>116</sup> A study out of Florida State University has backed this conclusion.<sup>117</sup> Even the CDC acknowledged this evidence, though it still preferred to believe the dentist had infected Bergalis. **But the CDC's own numbers give away the reality. An estimated 1 million Americans have HIV, in a total population of 250 million. Thus, 1 in 250 Americans have the virus. Five HIV-infected patients of Dr. Acer, out of 1100 tested, comes to 1 in 220, virtually identical to the national average. So does the proportion of HIV-positives from the patients of the 32 doctors, which works to 1 in 188.** These HIV-positive patients merely represent random samples from the general population.

And where did these people get the virus? **HIV is probably transmitted much as other retroviruses, from mother to child during pregnancy.** There is no evidence that Kimberly Bergalis' mother has ever been tested for HIV antibodies, nor that the mothers of Dr. Acer's other patients were tested. Perhaps Kimberly carried the harmless virus for twenty-three years.

The CDC's theory that AIDS was transmitted from Dr. Acer to his patient began to crumble in the mainstream press in 1994 when an investigative reporter researched the alleged victims of Dr. Acer. "He found weak evidence shoddy science, and the work of a very accomplished malpractice attorney."<sup>118</sup>

The report first casts doubt on the time course of AIDS transmission from Dr. Acer to his patients. "She developed AIDS just two years after the surgery, and only 1 percent of HIV positive patients develop the full-blown disease that quickly."<sup>119</sup> The investigation disclosed that one of the six other patients that Acer presumably infected had visited the dentist's office only once **for a cleaning by a hygienist**, not by Acer himself.<sup>120</sup> The report further calls into question the exclusive reliance of the CDC and the malpractice attorney of the "Acer six" on the DNA fingerprinting technique to match Acer's virus with those of his patients. This same technique had also been used to determine that the NIH researcher Gallo had claimed HIV obtained from his French rival Montagnier as his own. Several experts have directly challenged the DNA fingerprinting that linked Acer to his patients, claiming that instead Bergalis's virus matched other HIV strains much more closely.<sup>121</sup> In view of this, a writer in the New York Times commented, "The CDC owes it to the public to reopen [Acer's] case."<sup>122</sup>

The re-investigation of the "Acer six" provides unknowingly yet another reason why the "CDC owes it to the public to reopen [this] case": It supports the hypothesis that AIDS is caused by recreational drugs and AZT. Only three of the "Acer six" have developed AIDS, and every one of them was on drugs: Bergalis was on AZT; a thirty-year-old male was involved with "drug dealers, and a homosexual relationship"; and another male was a "notorious crack head."<sup>123</sup>

While on AZT, Bergalis once told a reporter she hoped to also get dideoxyinosine (ddI), another experimental AIDS drug. This drug and ddC, two products of cancer chemotherapy research, work in precisely the same way as AZT. Chemically altered building blocks of DNA, they enter the growing chain of DNA while a cell is preparing to divide and abort the process by preventing new DNA building blocks from adding on (see Figure 1). So, like AZT, ddI and ddC kill dividing cells and have similar toxic effects. They destroy white blood cells and therefore can cause AIDS. The only difference between ddI, ddC, and AZT lies in how easily each is absorbed into the body; people who absorb one evidently may not be equally affected by the other.

## **HIV=AIDS CONTROVERSY: AZT AND THE DEATH OF ALLISON GERTZ**

From:—With Therapies Like This, Who Needs Disease?—of the book *Inventing the AIDS Virus* by Peter Duesberg. 1996. The Stories of Those Who Believed in AZT.

*Alison Gertz: Both ddI and ddC have begun to claim their victims. In 1988, twenty-two-year-old New York socialite and aspiring graphic artist Alison Gertz entered the hospital for a fever and diarrhea. At some point the doctor decided to test for HIV and found antibodies against HIV. Gertz's transient illness was rediagnosed as AIDS. She had not injected drugs, although her wilder*

days at Studio 54 bespoke the cocaine and other free drugs available to patrons. A process of elimination traced her infection to a one-night stand with a bisexual male—six years earlier. The announcement left her feeling depressed, but she began a lecture circuit at high schools and colleges, admonishing students that AIDS could come from a single sexual encounter. Television talk shows followed, as did the cover of *People* magazine and *Woman of the Year* for *Esquire*. Even the World Health Organization circulated a documentary featuring her story.

Gertz started AZT treatment in 1989. The 1990 *People* magazine profile recounted the consequent disaster: Last October she was hospitalized with a severe allergic reactions to AZT. When doctors called for a lung biopsy, Ali balked. “I told them if they put me to sleep, I’d never wake up,” she says. “My strength was gone.” Released after 17 days, she recuperated at home, where her mother and girlfriends took turns nursing her around the clock. “They’d help me to the bathroom, feed me, see that I didn’t fall in the shower,” says Ali. “My knees were so bony, I had to sleep with a pillow between them.”<sup>124</sup>

The doctors switched her to the still-experimental ddI, which Gertz apparently did not absorb as well and thus allowed her partly to recover. She mixed the powder in her drink twice every day. Her immune system and general health declined, though more slowly. “Gertz remains susceptible to infections like thrush, a fungus that frequently affects the mouth,” stated the *People* article. “She has lost 30 lbs. since last summer, naps each afternoon and continues to visit her doctor every 10 days.”<sup>125</sup> Ultimately, the ravages of the chemotherapy took her life in August 1992, the news media advertising her death as AIDS-related. She was only twenty-six.

In **1992**, it was reported that “HIV-sequences” exist in normal in human, chimpanzee, and rhesus monkey DNAs” [22]. That same year, it was also reported that the hepatitis B vaccine causes false positive “HIV” test results [23].

**THE VETERANS AFFAIRS COOPERATIVE STUDY CONCLUDES THAT  
AZT KILLS MORE HEALTHY PATIENTS THAN SICKER PATIENTS,  
AND DISPROPORTIONATELY HARMS BLACKS AND HISPANICS  
AND PROVIDES NO BENEFIT**

In **1992**, The Veterans Affairs Cooperative Study Group reported that:

*In symptomatic patients with HIV infection, early treatment with zidovudine delays progression to AIDS, **but did not improve survival**, and was associated with MORE side effects. There were 43 deaths, 23 in the early-therapy group, and 20 in the late-therapy group. The medium time from the diagnosis of AIDS to death was **16** months in the early therapy group, and **19** months in the late-therapy group.*

*The racial and ethnic groups appeared to respond differently to the timing of zidovudine therapy. Fewer minority (African American and Hispanic) patients died in the late therapy group (two deaths) than in the early-therapy group (nine deaths), but the difference was not significant. Among non-Hispanic white patients, early therapy significantly delayed the onset of AIDS but **had no effect upon survival**. Minority patients were much more likely than white patients to be intravenous drug users (40% vs. 10%). After two years of follow-up, we found **no difference** in survival between the two treatment groups.*

Therefore, the Hamilton et al study concluded:

*AZT kills more healthy patients (early group) than sicker (late treatment) patients, and in addition, AZT disproportionately*

*harmed Blacks and Hispanics, and provided no benefit to the quelling of advancing immune suppression in Caucasians [24].*

In **1993**, Defer et al. in a paper entitled, “Multicentre quality control of polymerase chain reaction [viral load] for detection of HIV DNA” claimed that:

“False-positive and false-negative results were observed in all laboratories (concordance with serology ranged from 40 to 100%). In addition, the number of positive PCR results did not differ significantly between high and low-risk seronegatives. The use of crude cell lysates in DNA preparation produced the same PCR results as phenol-extracted DNA. Discrepancies between laboratories indicated that factors other than primer pairs contributed strongly to laboratory variability. [25].

### **HALF OF INFANTS SEROREVERT BY 18 MONTHS**

In **1993**, it was reported that half of infants that test “HIV” positive at birth serorevert (reverse) their “HIV-positive status within 18 months [27].

### **ARTHUR ASHE, RUDOLF NUREYEV, AND RANDY SHILTS DIE FROM “LIFE SAVING” AZT.**

**1993** *Arthur Ashe: This lesson almost saved the life of the late Arthur Ashe, the tennis star and one-time Wimbledon champion who died in 1993, supposedly of AIDS. Ashe’s medical problems surfaced in 1979 with a heart attack, despite his young age of 36. In December he underwent quadruple-bypass surgery. His chronic heart condition continued plaguing him, and by 1983 he had double-bypass surgery. **A blood transfusion during either one of the operations may have carried HIV.***

*His heart condition and its complications nagged him for several years. Then in 1988 he entered the hospital for toxoplasmosis, a protozoal disease relatively uncommon in humans. The germ resides in cattle and household pets, **and in 17 percent to 50 percent of the U.S. population**, but most people never succumb to the disease because of healthy immune systems. This also happens to be one of the many diseases **on the AIDS list**, so the doctor tested and found Ashe to be HIV-positive. Although his toxoplasmosis soon disappeared, Ashe was pronounced an AIDS victim. His disease was retroactively blamed on HIV, not on his heart condition.*

*Yet his condition hardly seemed contagious. **Neither his wife nor his daughter, born three years after his second transfusion, ever developed any AIDS conditions.** Indeed, his immune system must have neutralized HIV quite effectively, **as Ashe never transmitted the virus to his family.***

*His daily medicine intake expanded to a virtual pharmacy. He continued to take several drugs for his heart problems, one to lower cholesterol **by interfering with liver function**, another **to slow down the heartbeat**, and three others, including nitroglycerin, **to lower blood pressure**. To these his doctors added **a spectrum of antibiotics**, all with mild to serious side effects, to prevent the possibility of opportunistic infections. Ashe took Cleocin **to fight further toxoplasmosis**, nystatin **to slow down yeast infections**, and toxic pentamidine **to stave off Pneumocystis pneumonia**. Two other drugs were prescribed against **possible brain seizures**. Eventually his daily regimen included some thirty pills, only a few of them vitamins.*

*But just as soon as Ashe received his AIDS diagnosis in 1988, his doctor pushed him into taking AZT. He started on an unbelievably high dose, **nearly double the seriously toxic levels used in the Phase II trial**. His doctor only gradually lowered the dose over the next four years. “I refuse to dwell on how much damage I may have done to myself taking the higher dosage,” Ashe later admitted.<sup>127</sup>*

In early 1992 he established an acquaintance that came close to rescuing him. A close friend arranged a series of meetings with Gary Null, a New York-based radio talk show host and nutritionist. Null introduced Ashe to the evidence of AZT's toxicity and against the HIV-AIDS hypothesis, desperately trying to convince him to halt the therapy. For the next ten months, Ashe "wrestled with the possibility of breaking away from the medical establishment to seek alternative treatment for AIDS," according to one columnist. Ashe never met Peter Duesberg, but became familiar with his arguments. "He read everything; he studied what we gave him and asked lots of questions," recalled Null.<sup>128</sup> In October, Ashe announced the lessons he was learning in a column he wrote for the *Washington Post*: "The confusion for AIDS patients like me is that there is a growing school of thought that HIV may not be the sole cause of AIDS, **and that standard treatments such as AZT actually make matters worse.** That there may very well be unknown cofactors but that the medical establishment is too rigid to change the direction of basic research and/or clinical trials."<sup>129</sup> But psychological pressure stopped Ashe short from rejecting AZT. As Null stated, "He wanted to do it, but he would say, '**What will I tell my doctors?**'"<sup>130</sup>

In his 1993 book, *Days of Grace: A Memoir*, Ashe openly acknowledged his interest in alternative AIDS hypotheses:

*But AZT was controversial in other ways. A gift from heaven to many desperate people, it was poison to others. Developed for use in cancer chemotherapy to destroy cells then in the process of actively dividing, AZT was only later applied to AIDS. Some scientists believe that AZT, which relentlessly kills cells but cannot distinguish between infected and uninfected cells, is as harmful as AIDS itself. After all, HIV is present in only 1 of every 10,000 T-cells, which are vital to the immune system; but AZT kills them all. Dr. Peter Duesberg, the once eminent and now controversial professor of molecular and cell biology at the University of California, who bitterly disputes the notion that HIV causes AIDS, has called AZT "AIDS by prescription."*

*Dr. Duesberg argues that the use of recreational drugs, not sex led to AIDS. It is well known that many gay men used—and many of them continue to use—drug stimulus in sexual activity or to facilitate intercourse. "Natural and synthetic psychoactive drugs," he has argued (drugs such as cocaine, amphetamine, heroin, Quaaludes, and amyl nitrates and butyl nitrates, or "poppers"), "are the only new pathogens around since the 1970s and the only new disease syndrome around is AIDS, and both are found in exactly the same populations."<sup>131</sup>*

*Ashe faithfully summarized the main points against the HIV hypothesis and for the drug-AIDS hypothesis and explained the deadly effects of AZT and the flaws of its Phase II trial. "Some tolerate [AZT] for a while, then must give it up. Still others cannot tolerate it at all," wrote Ashe. "To my relief, I tolerate AZT fairly easily."<sup>132</sup> With that rationalization, he sealed his fate.*

*During 1992, his doctors placed him on ddI. Each morning he sprinkled the powder on his cereal, in addition to the AZT pills he swallowed throughout the day. By this time he was wasting away rapidly, his underweight frame hidden by loose clothes. He began rotating in and out of the hospital. January of the following year brought more bad news: Now he had a serious case of *Pneumocystis pneumonia* that his poisoned immune system could no longer fight off. He never recovered. On February 6, 1993, he breathed his last.*

*The list of celebrity AIDS patients who died on AZT for their belief in medical authority includes ballet star **Rudolf Nureyev**, who died in 1993, **Randy Shilts**, the author of the bestseller *And the Band Played On*, who died in 1994, and many more.*

*As a thoroughly politicized epidemic, AIDS began with a falsehood and ended in tragedy. **Virus hunters** in the CDC-directed public health movement first made the new syndrome **appear** contagious. Virus hunters in the NIH-funded research establishment then blamed AIDS on a retrovirus. And virus hunters in the NIH, CDC, FDA, and pharmaceutical industry exploited the situation by resurrecting failed cancer chemotherapeutic drugs for AIDS treatment. In the crisis atmosphere created by the CDC, which allowed no time to think before acting, such toxic drugs as AZT, ddI, and ddC could bypass the normal review procedures and achieve a sanctified monopoly status. The final results have been an unnecessary death toll and an artificially*

expanding AIDS epidemic.

*To make all this possible, the virus hunters from all fields first had to join forces. They have used their combined influence, often behind the scenes, to mobilize the government, media, and other institutions behind a global war on AIDS. Few outsiders have realized just how coordinated the whole strategy has been.*

## **HIV CAUSES 15 “CANCERS” OR PROBABLY ALL OF THEM(which are a spectrum of diseases characterized by too many cells rather than too few cells)?**

In **1993**, cervical cancer was included in the revision of the surveillance case definition for AIDS. The suggestion that cervical cancer is caused by “HIV” brought the number of cancers either caused directly or indirectly by “HIV” to include six different cancers, if antibodies, proteins, glycolipids, or genomic fragments indirectly associated with “HIV” are detected in a cancer patient.

At the first international meeting devoted to AIDS and cancer sponsored by the National Cancer Institute 4 years later, the reporter and Epidemiological Intelligence Service agent, **Lawrence Altman** of the New York Times wrote the following:

*AIDS Patients Face Growing Threat From Cancer. New York Times May 6, 1997:*

*As more people with AIDS survive longer, many are confronting yet another serious health problem: cancer.*

*Malignant tumors, particularly Kaposi’s sarcoma and certain cancers of the lymph system, have been common among AIDS patients since the disease was first recognized in 1981. But **now** the array of cancers **is broadening in mysterious ways**. Once rare cancers **are appearing more often**. Some kinds of cancer that **were expected to increase** in frequency have not. And strange geographical patterns of certain cancers are emerging.*

*It is a  **baffling situation**, yet scientists **see hope** in it. Researchers are looking at the puzzling trends with the aim of identifying and using biological clues to unlock the secrets of the myriad cancers that afflict people who are not infected with HIV, **the virus that causes AIDS**, and to devise new ways to treat them.*

***Like everyone else**, people with AIDS can develop **any kind** of cancer. And to **a doctor**, a slide of a specific tumor looks **the same** under a microscope, **regardless** of whether it came from a person infected with HIV.*

*Now, though, **the new patterns of disease** have raised the possibility that further studies of subtle differences might eventually help researchers improve cancer therapy.*

***An emerging theory** is that **most AIDS-related cancers** are **somehow** linked to **viral infections**. But doctors **do not know** if HIV enhances the ability of other viruses to produce certain cancers, scientists said here last week, at the first international meeting devoted to AIDS and cancer. The meeting was sponsored by the National Cancer Institute.*

*Dr. Richard Klausner, the director of the federal institute, said that at least **30 percent** of people **with AIDS also developed cancer**. The frequency is estimated from a variety of studies and cancer registries using information collected from hospitals and death certificates.*

*Dr. Valerie Beral of the University of Oxford in England said that studies had **definitely** linked **five cancers** with HIV infection: [1] **Kaposi’s sarcoma** (skin and internal organs); [2] **non-Hodgkins lymphoma** (lymph system); [3] **squamous cell cancer of the conjunctiva** (eye); and in situ (early stage) carcinomas of the [4] **cervix** and [5] **anus**.*

Three other cancers are probably related to HIV. [6,7] Two affect the lymph and blood system (**Hodgkins disease and plasmacytoma**) and [8] one affects muscle (**leiomyosarcoma**). Once rare, leiomyosarcoma has become **the second** most common cancer in **HIV infected** children but, **for unknown reasons**, not in adults.

Studies have also suggested an increase in [9] **lung**, [10] **lip**, [11] **testicular** and **other** cancers. Although the overall incidence of breast cancer is not increased in AIDS patients, several cases of unusual types of **aggressive** [12] **breast cancers** have been reported. But their link to AIDS remains **uncertain**.

Most perplexing is why HIV-infected people **are not** developing **liver** and certain other cancers. **For example, many HIV-infected people are also infected with either the hepatitis B or C viruses, which increase the risk of liver cancer, yet they are not unusually prone to liver cancer.**

The meeting took place at a time of **optimism** about AIDS. Combinations of newer anti-HIV drugs like protease inhibitors, and older ones **like AZT**, can suppress the virus to levels **below the limits of detection by the latest laboratory techniques**.

But whether the new therapies will reduce **or increase** cancer rates among people with HIV **is not known**.

...But there was little formal discussion about the adequacy of the current techniques to track cancer among people infected with HIV and to detect what proportion, **if any, might be caused by the new combination anti-HIV drugs themselves**. In hindsight, members of the program committee said in interviews, **they should have added this problem to their agenda**. The cancer institute's system to track cancers among AIDS cases relies heavily on studies and registries that link the two diseases. Two officials at the institute, Dr. Ellen Feigal and Dr. James J. Goedert said the existing system should be able to detect an increase in cancer rates among long-term survivors of HIV infection, though the information may be delayed.

The Centers for Disease Control and Prevention also tracks AIDS-related cancers through its own system. It includes information about certain cancers if they are detected at the time AIDS is diagnosed, said Dr. Harold W. Jaffe, an AIDS expert at the federal agency in Atlanta.

But if an AIDS patient develops a cancer after the initial report, he said, "we probably do not know that, and we need to do a better job of getting that information in a timely way."

**If the frequency** of AIDS-related cancers does increase or **turns out to be linked to anti-HIV drugs**, it would be important to get the word quickly to patients and their doctors, who are now **being urged** to begin **combination** therapy **early** in the course of HIV infection.

Dr. **Alexandra M. Levine**, an AIDS and cancer **expert** at the University of Southern California, said she was "**extremely worried** that we will see more and more cancer" among HIV-infected patients.

Treatment of HIV infection in its early stages might prevent major damage to the immune system, though it could still leave an individual with a slightly impaired immune system. "**If therapies prolong lives, the immune system may be more effective at combating HIV than in controlling cancer cells**" because **different components** of the immune system **may be involved** in fighting the two diseases, Levine said.

Earlier this **year**, a panel appointed by the National Institutes of Health urged careful long-term follow-up **of all children whose mothers took AZT during pregnancy**. The use of AZT in pregnancy has been **one of the most striking successes** in the AIDS story because it **greatly reduces** maternal transmission of HIV to a newborn. **But a study has shown**

**development of tumors among the offspring of mice that received very high daily doses of AZT during pregnancy.**

**It was the sudden appearance of the Kaposi's sarcoma cancer in large numbers of gay men in New York City that led doctors** to recognize what is now called AIDS. Until then, Kaposi's sarcoma had been rare, and few experts suspected that it was related **to a virus.**

Then in late 1994, Dr. Patrick S. Moore and his wife, Dr. Yuan Chang, working at Columbia University without a federal grant, used a new technique to discover **human herpes virus 8 (HHV-8).** Further studies **suggested** that **infection** with HHV-8 **was needed** before Kaposi's sarcoma developed. Scientists have also linked HHV-8 to a rare type of **lymphoma** in people **infected** with HIV. **But scientists do not know how HHV-8 virus causes normal cells to become cancerous or precisely how it is transmitted, and they continue to debate the possible ways.**

Might other viruses help produce other cancers? Scientists are now **looking hard.** For example, **virtually all** lymphomas arising in transplant recipients are related to **the Epstein-Barr virus.** But **only about 50 to 75 percent** of the lymphomas arising in AIDS patients are linked to Epstein-Barr.

Dr. **Robin Weiss** of the Institute of Cancer Research in London said **he was among those who had sought but failed to detect a new virus in the lymphomas linked to Epstein-Barr among people with AIDS.**

**One enigma** is why HIV-infected women have about four times the risk of developing in situ carcinoma of the cervix **yet do not go on to have an increased incidence** of the more deadly invasive cervical cancer.

"It **defies** logic," **Levine** said.

**Experts** said **they doubted the explanation** that it was because Pap smears and other tests were preventing the progression of the cervical cancers. Invasive cervical cancers **are not being detected in Africa** and other areas where such health care is not available.

**Another enigma** concerns the Burkitt's type of lymphoma, which can produce grotesque tumors of the face and neck. **Before the AIDS epidemic,** Burkitt's **was common** in central Africa, but rare in developed countries. Now an estimated 2 percent of AIDS patients in developed countries get Burkitt's lymphoma, said Dr. Alan Rickinson of the University of Birmingham in England.

Rickinson said he had assumed that doctors in Africa, where there is **an epidemic of HIV infection,** "should see a flood of Burkitt's coming in." But Rickinson said **he was astonished to learn last week from doctors at a cancer registry in Kampala, Uganda, that the incidence of Burkitt's had not increased among Africans infected with HIV.**

"This is **completely counterintuitive,**" Rickinson said. Yet, he added, "They tell me that the spectrum of AIDS-associated malignancies in **Africans** **different from** that which we see in **the West.**"

Burkitt's has historically occurred in regions of Africa where **malaria** commonly infects children. Rickinson **speculated** that HIV infection, by **mimicking the chronic effect of malaria** through **an unknown mechanism** on the immune system, might somehow protect against Burkitt's lymphoma.

If scientists can identify such protective mechanisms **and other viruses** that help produce HIV-related tumors, then they can

target them in developing new strategies to prevent Burkitt's and other cancers. For example, several scientists reported using such an approach in treating Epstein-Barr-related lymphomas.

According to a recent revised 05/12/2008 update from the American Cancer Society, the current establishment thinking about "HIV" and cancer is as follows:

*Certain types of cancer occur **so often** in people with AIDS that they are considered AIDS-defining conditions – that is, their presence in a person infected with HIV is a clear sign that full-blown AIDS has developed. They are also called AIDS-related cancers and include the following:*

*\* Kaposi sarcoma*

*\* lymphoma (especially non-Hodgkin lymphoma and primary central nervous system lymphoma)*

*\* invasive cervical cancer*

*Other types of cancer that may be more likely to develop in people with HIV infection are invasive anal cancer, Hodgkin disease, lung cancer, cancer of the mouth, cancer of the testicles, and skin cancers, including basal cell, squamous cell, and even malignant melanomas. Of course, people without HIV or AIDS can also have all of these types of cancer, even the ones that are better known as AIDS-related. **They are only called AIDS-related cancers if they develop in people with HIV infection.***

*In developed countries, about **4 people in 10** with AIDS develop cancer at some time during their illness. However, the cancer picture in HIV is changing. Kaposi sarcoma and non-Hodgkin lymphoma have decreased **as anti-retroviral treatment has become more common**. Most other types of cancer do not seem to be **slowed** by HIV treatment, and have the same risk factors as those in people without HIV. For instance, **people who smoke and have HIV are more likely to have cancers of the lip, mouth, throat, and lung than people who have HIV and don't smoke.***

*The relationship between HIV and these other cancers **is still not completely understood**. However, it is **believed** cancers **can grow quickly** because people with HIV have weakened immune systems. Unfortunately, cancer in people with HIV can be harder to treat. This is partly because of the decreased immune function caused by HIV and the lower white blood cell count that can result from HIV infection. **Having AIDS can make it hard for a person to take chemotherapy because the bone marrow**, which is needed to make new blood cells, is **sometimes** already damaged **by the HIV infection**. People with this problem **often can't take the full dose** of chemotherapy **without serious harm**. The introduction of highly active anti-retroviral therapy or HAART in the late 1990s led to a decrease in **some** types of cancer among those with HIV and better survival with anti-cancer treatment. **It has also allowed full chemotherapy doses** to be used for treatment. Other types of cancer treatment, such as monoclonal antibodies and stem cell transplant, are being studied in people with HIV.*

*Non-AIDS -related cancers*

*...With more widespread use of anti-retroviral drug treatment, AIDS-related cancers are happening less often. **But** as people with HIV are living longer, **they are developing cancers that are not generally linked to HIV, such as lung, [13] throat, [14] liver, intestinal, and anal cancers as well as Hodgkin disease and [15] multiple myeloma**. Most of the time, treatment includes ART **along with cancer treatments that have worked** for people without HIV. At the same time, any other needed treatments for HIV (such as antibiotics to prevent infections) are used.*

**GALLO CONSULTS JUDAH MOSES FOLKMAN AND KAPOSÍ'S SACOMA AND THUS HALF THE AIDS-**



## **INDICATOR ILLNESSES NO LONGER CAUSED BY “HIV.”**

Dr. Judah Moses Folkman, the world’s best respected promoter of anti-angiogenesis therapies and his vascular task force spent much of their efforts managing young children who had developed hemangiomas, which are typically non-metastasizing tumor-like growths which tend to occur more frequently in low birth-weight children and for unknown reasons as well. Often deadly vascular anomalies were also managed, along with diseases such as Graft Versus Host Disease as well as cancers. Hemangiomas in particular appeared to be highly angiogenic in the way and abundance with which they supply themselves with neovasculature, although they do not metastasize like cancer, and, it is likely that many of Dr. Folkman’s views regarding the immune system and cancer were predicated on these clinical experiences and other important morbid syndromes such as Kassabach-Merritt syndrome, Kaposi’s sarcoma, and other contexts in which neoangiogenesis appears robust. In the context of Kaposi’s and the complex role of immunity in cancer, and as is noted in Dr. Robert Gallo’s book (*Virus Hunting—AIDS, Cancer, & the Human Retrovirus: A Story of Scientific Discovery*. New York: Basic Books, 1991. page 267), Dr. Gallo consulted Judah Folkman to help find an explanation as to why Kaposi’s sarcoma itself, as one of the first two AIDS-indicator illnesses in substance abusing young men, could not possibly have anything to do with a virus, as it was known that Kaposi’s spindle-shaped cells have characteristics other than endothelial cells, as Dr. Folkman educated him about (Robert Gallo, personal communication). Eventually, after the “tat-transgene” experiments of Jay failed to show any “tat” protein within Kaposi lesions, another virus was soon blamed (HHV8), and thus Kaposi’s, as one of the first categories of AIDS-indicator illnesses was no longer directly or even indirectly due to the molecular signatures associated with “HIV.” Dr. Folkman would often comment regarding the role of the immune system and cancer:

*“Although embryos do not have developed immune systems, they do not typically develop cancers, nor do the majority of SCID mice. Unless we place tumor cells in them, they do not, like embryos lacking any immune system, typically develop cancers. The lack or presence of a robust immune system may modulate cancers, but does not cause them to occur.”*

*The burning question amidst all of these considerations became, could blood vessel recruitment to growing tumors be blocked with the same endogenous molecules that are believed to stop new blood vessel growth in non-cancer contexts, such as menses, without harming the patient? Because they weren’t tumor cells believed to “mutate around drugs or drug cocktails,” no resistance to inhibitory compounds should occur among endothelium, it was reasoned.*

## **MULTIPLE-ANTIGEN-MEDIATED –AUTOIMMUNITY THROUGH IDIOTYPIC-ANTI-IDIOTYPIC REACTIONS.**

In 1993, Robert Root-Bernstein published the book, *“Rethinking AIDS; The Tragic Cost of Premature Consensus”* [26], in which he outlined an hypothesis he called Multiple Antigen Mediated Autoimmunity (MAMA) to explain how an idiotypic and anti-idiotypic immune mechanism could create an autoimmune syndrome like AIDS; given that the molecular mimicry of the profile attributed to the presence of “HIV,” and the anti-idiotypic moieties that normally shut down the production of those antigens stimulated by “HIV’s molecular signature interact, which might cause an autoimmune disease that destroys the immune system.

*Root-Bernstein (Excerpt from Five Myths at virusmyth.com).*

*(A) significant proportion of people repeatedly exposed to HIV become PCR positive but remain antibody negative and healthy. Other people repeatedly exposed to HIV remain antibody negative, are PCR-negative, but demonstrate T-cell activation toward HIV antigens, strongly suggesting that they have previously mounted an effective T-cell response to HIV (or some cross-reactive alloantigen [Ston (or Stott), 1991; Kion & Hoffman, 1991]). In short, HIV is controlled by the T-cell response, and **antibody positivity to HIV is negatively correlated with control of HIV infection.***

*These data strongly suggest that the primary line of defense, and the only effective one against HIV is a T-cell response (Clerici et al., 1992; Clerici & Shearer, 1993; Salk et al., 1993). T-cell regulation is reasonably common for non-cytolytic encapsulated viruses,*

and antibody enhancement of infection is relatively common for these viruses as well (see below). Antibody is only produced when T-cell immunity fails to control viral replication and a sufficient amount of free virus is present in blood, lymph, or tissues to activate B cells. Clearly, as the cases of sero-**reversion** prove, this antibody is short-lived in the absence of active, ongoing **infection**, and does not remain to protect against further **infection**. This phenomenon of rapid loss of antibody is also consistent with a primarily T-cell regulated immune response, rather than a primarily B-cell regulated immune response.

In fact, there exists a class of infectious diseases, most of which consist of non-cytopathic encapsulated viruses (of which HIV is one), **the pathological effects of which are actually exacerbated by the presence of antibody**. One example is **Lymphocytic choriomeningitis virus (LCMV)** infection in **mice**, which has many similarities to **hepatitis B virus** and **HIV infection in man** (Oehen, Hengartner & Zinkernagel, 1991). In LCMV, as in many other viral infections **in which the immune response, rather than the virus infection itself causes lymphocyte death, survival is dependent on a successful T-cell response rather than upon antibody production. Production of antibody, whether naturally occurring or as a result of vaccination, is highly associated with death of the animal** (Oehen, Hengartner & Zinkernagel, 1991). Dengue virus infection in human beings presents **a similar picture**. The severe hemorrhagic fever associated with the virus is almost always a result of an **anamnestic or secondary antibody response**. Presence of antibody has been found to be a strong predictor of severe disease following reinfection with a variant strain of the virus (Halstead, 1988; Kliks et al., 1989). Since **HIV mutates very quickly**, and an extremely large number of HIV strains are known, probability of antibody-mediated enhancement of secondary infection with variant HIV strains in AIDS becomes a very likely event, which may, indeed, explain the long latency found in the syndrome.

**LCMV and dengue virus are only two of many examples antibody-enhanced disease**. Porterfield (1986) and Burke (1992) have summarized the relevant data for a very wide range of non-cytopathic viruses, including dengue, Japanese encephalitis, yellow fever, tick-borne encephalitis, Sindbis, respiratory syncytial virus, rabies, reoviruses, murine cytomegalovirus, corona viruses, and lentiviruses (e.g. the visna virus of sheep). In some cases, vaccines (usually live attenuated strains) against these viruses have been very effective, but in others, such as respiratory syncytial virus, measles virus, and visna virus, **vaccination with formalin inactivated whole virus dramatically increased the probability of severe or life-threatening infection among recipients (Burke, 1992)**. Burke has concluded that antibody-dependent enhancement of infection is a general in vitro property of **all** enveloped viruses, and that this in vitro activity is more often than not mirrored by physiological enhancement of infection as well, particularly when humoral immunity is not complete.

**Particularly frightening in this respect is significant data that antibody-dependent enhancement of HIV infection occurs in vitro and possibly in vivo as well (reviewed in Burke, 1992)**. The problem is exacerbated by **the mimicry between HIV proteins and HLA proteins of lymphocytes** that results in immunologic **crossreactivity** between HIV and lymphocyte cellular receptors (Golding et al., 1988; Vega, Guigo & Smith, 1990; Garry et al., 1991; Bjork, 1991; Kion & Hoffman, 1991; Ston (or Stott), 1991; Clerici et al., 1992; Dalgliesh et al., 1992; Susal et al., 1993; Root-Bernstein & Hobbs, 1991, 1993; Root-Bernstein & DeWitt, 1994).

Thus, a recent study of the immunological effects of recombinant HIV gpl60 resulted in 3 of 5 human volunteers developing **anti-idiotypic antibodies that cross-reacted with their CD4 protein**. The study concluded that such **vaccine-induced anti-CD4 antibodies potentially may: (1) limit the use of vaccines which elicit them; (2) contribute to the immunodeficiency occurring in HIV-1 infected individuals, and (3) provide evidence of HIV-1 infection during the period when anti-HIV-1 antibodies are not detectable, (Keay et al., 1992; see also Sabin, 1988)**.

**DR. ANTHONY PINCHING WOULD HAVE BEEN BOOED OFF THE STAGE BUT HE WAS APPLAUDED HEAVILY WHEN HE SAID, " WE HAVE NO IDEA WHICH DRUGS WORK. WE HAVE NO IDEA WHAT WE'RE DONG IN**

## TREATMENT, AND IT'S TIME TO RETURN TO THE USE OF PLACEBO TRIALS.”

**1993** From Peter Duesberg: The Stories of Those Who Believed in AZT.

*A backlash is now rising against the toxic and irrational treatment approaches to AIDS. In 1993, during the Ninth International AIDS Conference in Berlin, Germany, medical reporter Laurie Garret was interviewed on the MacNeil-Lehrer News Hour. She described the growing discontent among scientists and patients alike:*

*Most drug trials were terminated early. The AZT trial was terminated early, ddI, ddC, and so on, and people were allowed as soon as there was any sign that something showed promise to jump out of the placebo arm and get into the treatment arm...*

*Dr. Anthony Pinching, who was really the leader of most of the clinical research related to AIDS in the United Kingdom, gave a very important speech this morning. I think if he had given this precise same speech **a year ago, he would have been booed off the stage**, and this morning, he was applauded heavily. And what he basically was saying was **we have no idea what drugs work. We have no idea what we're doing in treatment, and it's time to return to the use of placebo trials.** He went a step further and said that at least in Europe a lot of AIDS activists and patients now agree, because they're shocked to find out that the drugs they've been taking, thinking they would be helpful, might even be hurtful.<sup>126</sup>*

## THE CLINTON YEARS START ROLLING: THE YEAR OF CONSTANT TERRORISM!

In **1993**, President William Jefferson Clinton nominated Herald Varmus as director of the National Institutes of Health, the nation's premier research and funding institution in the biomedical sciences. The first Nobel laureate to head NIH, Varmus strengthened its commitment to basic research while balancing demands from advocacy groups for more targeted research on particular diseases, most notably AIDS. Here are some of these 1993 reports of this critical period of AIDS metastasis (only a few of the propaganda pieces from the end of June through the beginning of July that golden Clinton White House year are presented):

**Man Infected With H.I.V. by Florida Dentist Dies, New York Times** (06/28/93), P. A8.

*A man who **contracted** HIV infection from his Florida dentist has died of an AIDS-related illness at the age of 33. The man, Richard Driskill, died of **pancreatic failure** on Saturday, according to his lawyer, Robert Montgomery. Driskill was one of six patients known to be **infected** by dentist Dr. David Acer, who died of AIDS in 1990. Driskill worked as a foreman at a citrus plant, and was referred to Dr. Acer by his insurance company. Once another patient of Acer's, Kimberly Bergalis, publicly disclosed her **infection**, Driskill underwent testing for HIV **infection**. He tested positive and subsequently sued the insurance company, CIGNA Dental Health Plan of Florida, for an undisclosed amount [\$1,000,000). Even though Dr. Acer tested positive for HIV in 1986 and developed AIDS-related symptoms, he continued to treat patients. Researchers at the Centers for Disease Control determined that the strain of HIV carried by the six patients **matched** Acer's strain. Public health officials claim that Dr. Acer is the only doctor or dentist known to have infected a patient with the virus. Since Kimberly Bergalis died in 1991 at the age of 23, 57 other health-care professionals have told the authorities that they are HIV-positive, and **19,000** of their patients have been tested. **Not one** patient has contracted HIV from being treated by an infected health-care worker, aside from the Acer cases.*

Washington Post (06/27) P. A14; Philadelphia Inquirer (06/27) P. B5. **Panel Is Probing Early Abbott AIDS Test.** Wall Street Journal (06/28/93), P. B6C, Burton, Thomas M.

*An HIV test made by Abbott Laboratories in 1985 **has been found to be inaccurate** in detecting the virus in donated blood, according to **congressional** investigators. As a result, dozens of unsuspecting patients may have received **HIV-contaminated blood**, say investigators for the House Subcommittee on Oversight and Investigations. In addition, investigators are critical of the*

American Red Cross' continued use of the test for months throughout 1986 despite evidence that other recently introduced tests were more accurate. The Red Cross says it was faced with a dilemma. Although it was aware of the difficulties in **the Abbott blood test**, it believed any switch to a different test could have led to more problems. Staff members of the House panel, headed by Rep. John Dingell (D-Mich.), have said that the subcommittee hopes to publish a report on the HIV test this fall that calls into question the roles of Abbott and the Red Cross. Also, several lawsuits have been filed against Abbott, and at least one against the Red Cross, by people who **allegedly** contracted HIV through blood transfusions while the Abbott test was in use. The congressional inquiry found internal documents from Abbott and the Red Cross that show that a **fierce internal debate emerged in 1986 at the Red Cross over the Abbott blood test**, which the Red Cross later decided to continue using. It was not until January of 1987 that Abbott introduced a refined HIV test that detected **early phases of virus** more effectively than had the old test. The Red Cross immediately adopted that version of the test.

**Experts Change Guides to Using Drugs for H.I.V.** New York Times (06/27/93), P. 1. Altman, Lawrence K.

An independent panel of federal AIDS experts has recommended a major shift in the strategy for treating people with HIV infection. The new recommendations, released Friday night after a three-day meeting at the National Institutes of Health, **allow for more flexibility** than existing ones. While the new guidelines **are not** official federal health policy, they are expected to **strongly influence doctors' decisions**. The new **rules** reflect recent research such as **the Concorde study**, and stress that **patients and doctors should decide together about when and how to treat HIV by concentrating on the patients' views of drug treatment, personal health, and other factors**. The guidelines **acknowledge** that individuals respond to AZT **differently** and that before starting AZT therapy in each case **there should be a full discussion of benefits and risks**. The panel said that **treatment with AZT is no longer necessarily advised** for people **with a low count of CD4 immune cells**. Patients with CD4 counts between **200 and 500** should start taking AZT, according to the panel. Careful monitoring **without drug therapy** is also recommended, the new guidelines say, and AZT treatment can begin if a patient's condition changes **or laboratory tests show a depletion of the immune system**. The panel said that AZT should be **the first-line drug to attack HIV**. For AIDS patients who experience progression of the disease **despite** AZT use, the panel recommended switching from AZT to **ddI**. The experts also **cautiously** advocated the use of **more than** one anti-HIV drug, **either in combination or in sequence**.

**Canada Court Allows Wife to Sue Bisexual Husband Over AIDS Risk.** United Press International (06/26/93)

An Ontario provincial court judge decided that a suburban Toronto woman can sue her estranged husband for more than \$1 million **for putting her at risk of contracting HIV** because **he neglected** to disclose his **homosexual activities**. Justice Alvin Rosenberg on Friday refused to dismiss a suit filed by Sophia Bell-Ginsburg of Mississauga against Norman Myron Ginsburg. The husband sought dismissal, saying the suit was frivolous. Sophia Bell-Ginsburg said her husband, whom she married in 1986, **deliberately hid** his homosexual activities from her. The judge said the husband **"knew** his sexual practices put him in a **high-risk** category for contracting AIDS yet he deliberately concealed the nature and extent of his **sexual practices** from Sophia." The couple are currently involved in divorce proceedings, **and Sophia has tested negative for HIV**. Rosenberg said the actions of the husband may prevent Sophia **"from establishing a normal relationship for the rest of her life."** The husband was described by Rosenberg as **"in ill health**, the particulars of which are known only to him."

**HIV Overlooked in Women.** Health (07/93) Vol. 7, No. 4, P. 18.

HIV is **three times less likely** to be detected in infected women than in infected men who are admitted to an emergency ward, according to a study conducted by researchers from the Albert Einstein college of Medicine in New York, N.Y. In an attempt to determine the rate of HIV infection in the emergency room at North Central Bronx Hospital, **Drs. Ellie Shoenbaum and Mayris Webber tested blood from 852 patients** during three weeks in 1989. A total of **90** of the patients **were infected**

with HIV, but **only 27 were identified** after being evaluated by medical staff. About **40 percent of the infected men were detected**, whereas **only 17.5 percent of the HIV-positive women were identified**. Dr. Shoenbaum said, "It was astonishing. For everything we looked at, women were disadvantaged." Physicians were more apt to identify HIV infection by the AIDS-related symptoms exhibited, which mostly occurred in men. Men were more inclined than women to be asked about **risky drug use**, even though **most patients weren't asked about risk factors at all**. **HIV infection was detected in men of all ages, but in women, it was identified only between the ages of 25-44, possibly because most infected women in New York City fall within that range**. The study indicates that more than men, HIV-positive women may not be **recognized** during emergency care unless they already have full-blown AIDS. **However, if they'd been detected sooner, the symptoms could have been delayed by earlier drug therapy, concluded Shoenbaum.**

**Agenda: Something Rotten in the Air.** Advocate (06/29/93) No. 632, P. 17

American Airlines' employee health plan **discriminates** against people infected with HIV, according to recent protests by AIDS activists. The company's health plan **denies coverage** to any employee hired after Dec. 1, 1986 who has a preexisting medical condition or any illness arising from that condition. **Activists say that American's health policy intentionally excludes HIV-positive individuals.** "When you have a policy like American's, it's tantamount to having no coverage for HIV infection," said Mike Isbell, an attorney for **the gay group Lambda Legal Defense and Education Fund**. Jacques Chambers, benefits program manager for AIDS Project Los Angeles, said, "If an insurance company tried this, it would be struck down by the regulatory authorities, but American can get away with it "because it is self-insured." Chambers said his organization has intervened on behalf of at least one HIV-infected employee of American. However, American's spokesman Gus Whitcomb said **the policy is not discriminatory because it applies to all preexisting medical conditions, not only HIV infection. A Supreme Court ruling last year mandated that self-insurers, such as American, are permitted to alter or restrict their health coverage at will.**

**AIDS Panel's Final Report Repeats Pleas for Action.** New York Times (06/29/93), P. B7. Hilts, Philip J.

The National Commission on AIDS issued its final report, after four years of work, which reemphasizes its **request for the federal government to establish a national plan to combat the AIDS epidemic**. The commission ended its work yesterday and said **it hoped that the Clinton administration would do a better job of handling the epidemic than previous administrations**. It also commended the appointment of Kristine M. Gebbie last Friday as the White House AIDS Coordinator. In addition, the commissioners mentioned that the administration had asked for the most significant increase in funds to fight AIDS—**to \$2.7 billion from \$2.1 billion**. However, the report said, "To date, no opportunity has yet been found to discuss recommendations sent to President Clinton upon his taking office." It also said, "**Our nation has continued on its shortsighted course**. Sadly, we must continue to report that America is still doing poorly." The report indicated that the panel is still advocating recommendations it made two years ago. Most of the recommendations have not been followed, with some exceptions: **the government has expanded the criteria for full-blown AIDS to include people who were previously considered to only be HIV-positive, and the White House has just appointed an "AIDS czar."** The commission advised the government two years ago to devise a single plan for the nation to combat AIDS, **provide treatment for drug abuse to all who need it, abolish any laws that prevent drug users from obtaining clean needles and bleach, and provide medical coverage to all citizens with the cost of prescription drugs included.**

**U.S. Will Relax Disability Rules in H.I.V. Cases.** New York Times (06/29/93), P. A1. Pear, Robert.

**The Clinton administration will release new regulations this week to facilitate the process in which HIV-positive people receive federal disability benefits, contradicting a decade of more restrictive policy.** The rules, being distributed Tuesday to Social Security offices nationwide, come partly **in response to a lawsuit filed three years ago by 19**

**New York state residents.** The suit is pending before Federal District Judge Miriam G. Cedarbaum in Manhattan. **The plaintiffs, who are seeking class-action status to allow the suit to include thousands of people, have complained that they had been improperly denied benefits even though they were unable to work because of HIV-related illnesses.** The new rules would allow for many people who were denied benefits in the past to be eligible automatically. AIDS advocates predict that 10,000 more HIV-positive people may qualify under the new standards. More than **55,000 people** with HIV now receive monthly cash benefits, at a cost of more than **\$300 million** a year. The new criteria for evaluating HIV infection look for many conditions specific to women, **like pelvic inflammatory disease and cervical cancer.** Also, under the rules, people **infected with HIV will be able to qualify for benefits if they are found to have serious illnesses like bacterial pneumonia and tuberculosis.** In addition to the specific criteria, there is **a new catchall category** for people who show “repeated manifestations of HIV infection,” as well as a significant restriction in routine activities of daily living or difficulties in completing work in a timely manner **because of inability to concentrate on a specific task.**

**Government Panel on HIV Finds the Prospect for Treatment Bleak.** New York Times (06/29/93), P. C3. Altman, Lawrence K.

Because of new findings about **the efficacy** of AZT, the outlook for treating AIDS is **disheartening.** At a meeting at the National Institutes of Health last week, **most experts agreed with the European “Concorde” study’s conclusion that AZT is not effective** in treating early HIV infection. **Dr. Merle A. Sande, the chairman of the meeting and an AIDS expert at the University of California–San Francisco, said regarding beliefs that AIDS would gradually become treatable like other chronic diseases, “Somehow, it clearly hasn’t happened.”** He added, “We are still quite a ways back, in the infancy, I hope, of being able to treat this disease **effectively.**” AIDS experts said one of the reasons the outlook for AIDS therapy is **bleaker** than previously thought is that they have been **hampered by too many variables in evaluating studies of AZT** and its pharmacological cousins, **ddI** and **ddC.** **The studies frequently have different criteria for entry, test different amounts of drug for people at different stages of HIV infection and AIDS, vary in the lengths of follow-up of volunteers, and set different criteria for stopping the trials.** **Dr. Douglas D. Richman, an infectious disease expert at the University of California–San Diego, said, “All the studies are right, and the problem is how to put all the pieces together into a clear picture,” which is impossible, “because all the pieces do not exist.”**

**UCSF Will Be One of First California Sites to Participate in National Study of AIDS in Women.** Business Wire (06/28/93).

(San Francisco, CA) The University of California–San Francisco is one of four sites announced Monday to take part in a national study of the effect of HIV infection in women. The Women’s Interagency HIV Study will last four years and will research signs and symptoms in HIV-positive women. It will also investigate the pattern and rate of depletion of their immune systems, and **the potential co-factors** that may affect HIV disease progression. The research at UCSF will be conducted by **Dr. Ruth Greenplatt,** assistant professor of medicine with the AIDS clinic. The other study sites named on Monday also include the University of Southern California, Bronx-Lebanon Hospital Center, and Georgetown University. The National Institute of Allergy and Infectious Disease will appropriate \$5 million to fund the project. Researchers involved in the study will collect information from about 1,700 HIV-positive women and 375 women who are HIV-negative **but at high risk** of contracting the disease. Health and Human Services Secretary Donna E. Shalala made the announcement about the project on Monday. She said, “AIDS is now **the sixth leading cause of death for women** aged 25 to 44 in the United States.” Approximately **11.4 percent** of the U.S. AIDS cases reported by March 31 in people **older than 13** were among women, **according to the Centers for Disease Control.**

**Trends in HIV Prevalence Among Disadvantaged Youth.** Journal of the American Medical Association (06/09/93) Vol. 269, No. 22, P. 2887. Conway, George A.

HIV prevention strategies should be targeted at the hard-to-reach group of **adolescents** who are out of school **and indigent**, write George A. Conway et al. of the Centers for Disease Control in Atlanta, Ga. The researchers conducted an analysis of demographic and geographic trends of HIV infection among Job Corps students aged 16 to 21 years from January 1988 through December 1992. Among the **269,956** Job Corps students screened, **812 (0.3 percent)** tested HIV-positive. Seroprevalence of HIV for young men **decreased from 3.6 per 1,000 in 1988 to 2.2 per 1,000 in 1992**. The rate of HIV among young women increased from **2.1 per 1,000 in 1988 to 4.2 per 1,000 in 1990**, with seroprevalence remaining stable from 1990 through 1992. **The decreasing trends in the rate of HIV among men and increasing trends among women were primarily a result of changes in the seroprevalence in African-American students.** The substantial rise in HIV rates among female Job Corps students provides proof of the increasing risk of infection for socioeconomically disadvantaged young women. **Reasons for the declining trend in the rate of HIV among male Job Corps students are unclear.** Anti-HIV interventions that increase access to HIV counseling and testing, drug counseling and treatment, and employment opportunities will be essential in reducing the impact of HIV on high risk adolescents in the United States, the researchers conclude.

**Rules Are Set for Granting Benefits on AIDS Virus.** Wall Street Journal (06/30/93), P. B.

The Social Security Administration disclosed rules that are aimed at expediting the procedure in which benefits are given to people infected with the HIV virus. As with the interim regulations issued by the outgoing Bush administration, **the new rules list symptoms connected with aids that will automatically qualify individuals for disability benefits. Included in them list are several gynecological conditions and serious infections like tuberculosis.** In addition, the rules add a new category that **qualifies people who suffer from “functional limitations”** from HIV that restrict their day-to-day activities at home and at work.

**Don't Ask to Be on AIDS Coordinator's Staff.** Washington Post (06/30/93), P. A19

The nation's new AIDS coordinator, Kristine M. Gebbie, formerly Washington state's health official, revealed to the **Fox network** on Tuesday that her staff will be “very small, four or five people at the maximum.” Gebbie also said that **she approved of needle exchanges for drug addicts** as a means of stemming **the spread** of the disease, **but did not encourage condom distribution in schools.** She explained that condoms should be accessible to “**anybody who is sexually active,**” but that the question of school distribution is **a local decision.**

**Victims Reject Blood Deal.** Toronto Globe and Mail (Canada) (06/29/93), P. A7. Picard, Andre

People in Quebec who were infected with the AIDS virus because of **Canada's tainted-blood scandal** have strongly **rejected a \$10- million** provincial compensation plan. **Claire Desrosiers**, executive director of the Quebec division of the Canadian Hemophilia Society, announced yesterday that 95 percent of the 172 people who responded to a questionnaire rejected the proposal earlier in June. **Over 1,000 people became infected** with the HIV virus through blood and blood products before compulsory testing of donated blood began in 1985, following an eight-month delay. **Approximately 100 of those people are now deceased.** So far, only Quebec and Nova Scotia have implemented compensation programs. **But whereas the Nova Scotia plan provides victims with \$30,000 tax-free each year, as well as reimbursement for AIDS drugs not covered under the provincial health plan, the Quebec plan will give victims and their families less than \$13,000 per year, and will be provided only if the victims are deemed in “desperate need.”**

**Nation Editorial Sampler: What Newspapers Are Saying.** United Press International (06/30/93)

President Clinton's willingness to support **the politically touchy recommendations** from his AIDS-policy coordinator, Kristine Gebbie, will determine her success in the position, write the editors of the *Seattle Times*. While Gebbie will encounter a

number of obstacles, she has the training, experience, and personality for the job. Gebbie, **trained as a nurse**, was Washington state and Oregon's **top public-health officer**, and served on **the Reagan administration's national AIDS commissions**. In order to succeed in her present position, Gebbie needs more than Clinton's general support **for greater AIDS research funding**. Clinton, in order to support Gebbie and make headway against the disease, must prepare for controversies over spending, research, needle exchanges, and advertising campaigns. Clinton's commitment to fighting AIDS will determine Gebbie's success, the editors conclude.

**Researchers Debate Timing of AZT Treatment.** American Medical News (06/21/93) Vol. 36, No. 23, P. 4

Staver, Sari.

Even though there is **new evidence** indicating that early AZT treatment **does not prolong the onset of full-blown AIDS**, two leading AIDS researchers claim that **it may be preferable to start treatment with the drug even earlier than previously recommended**. At a recent American Medical Association symposium on early HIV intervention, **physicians** debated the clinical implications of the new data, which come from two of the longest trials comparing early AZT treatment with delayed monotherapy. **The controversial trials were the "Concorde,"** a collaborative effort between researchers in the United Kingdom and in France, **and the U.S. Veterans Administration's Study 298**. Both studies compared early and delayed AZT treatment, but the Concorde study researched asymptomatic patients at all CD4 levels, while the VA study included only symptomatic patients with CD4 levels of 200 to 500. **But Dr. Robert Walker**, of the National Institute of Allergy and Infectious Diseases, **said he personally believes HIV-positive patients should consider beginning therapy immediately upon diagnosis**. He mentioned several recent laboratory studies that show **active HIV replication takes place from the earliest stages of infection**. In addition, **Dr. Paul M. Volberding**, director of the AIDS program at San Francisco General Hospital and a principal investigator of a large trial of early AZT treatment, said he would **"be tempted"** to begin antiviral therapy in a patient with an acute HIV infection. He said the benefits of **early treatment include lower toxicity rates, less chance of developing resistant virus, and longer duration of benefit from the antiviral drugs**.

**Surge in First-Quarter AIDS Cases Not Expected to Continue.** AIDS Alert (06/93) Vol. 8, No. 6, P. 81.

Although there was a **204 percent increase in AIDS cases reported since the implementation of the new federal AIDS surveillance definition**, it is not expected to continue, say health experts. The number of AIDS cases reported in the United States during the first quarter of 1993 rose to **35,779, a 204 percent increase from the 11,770 cases reported for the same period last year**. But health experts say the large and sudden increase was anticipated and should have little effect on clinical care. **The CDC implemented the new definition on Jan. 1, 1993. It added CD4 counts of less than 200 and three diseases: pulmonary tuberculosis, recurrent pneumonia, and cervical cancer, to the list of criteria. The CDC estimated a total of 85,000 to 90,000 new cases in 1993, or a 75 percent overall increase over 1992**, dropping to 10 percent to 20 percent in 1993. The agency estimated that about **50 percent of the new cases reported in 1993 would come from the expanded definition**. If the rate of cases reported in the first quarter were sustained throughout the year, **about 140,000 new cases would be reported**, increasing the cumulative number of AIDS cases reported since 1981 by 50 percent. But Dr. John Ward, director of AIDS surveillance for the Centers for Disease Control, says the rate will decrease substantially, and the 75 percent annual projection will be more out of line.

**Researchers on AIDS Find a Use for Thalidomide.** New York Times (07/01/93), P. A13. Altman, Lawrence K.

**The once-banned sedative thalidomide is now being investigated for use in treating AIDS, tuberculosis, and other maladies.** Thalidomide was banned in the early 1960s after it was found to cause deformed limbs in the children of women who took it early in pregnancy. But in a report published today **in the Proceedings of the National Academy of Sciences**,



researchers said that **thalidomide could inhibit the growth of HIV in test tubes and that in very limited tests of humans it seemed to alleviate some of the severe symptoms of AIDS.** The researchers reported that thalidomide works in laboratory experiments against HIV by selectively suppressing a natural substance produced in the body. The substance, **tumor necrosis factor, also called cachectin,** defends against infection, and **is believed to play a key role in inciting the progression to full-blown AIDS.** Cachectin is released by white cells during infection and **serves as a hormone** in the immune system by acting on other cells **to combat an invading organism.** **Dr. Gilla Kaplan,** who headed the team of researchers from Rockefeller University and New York Hospital-Cornell Medical Center, said that in the laboratory, **thalidomide significantly slowed the growth of HIV when added to cultures of the virus** taken from patients at Bellevue Hospital and New York Hospital. **The patients did not take thalidomide.** In another study, the drug was given to patients with TB, some of whom had HIV infection. **Due to the apparent relief of AIDS-related wasting symptoms,** Dr. Kaplan said her team had started another study to give patients thalidomide **for two weeks, then stop** for a period, and later **resume** treatment.

**Novello Satisfied With Fight She Waged Against AIDS.** Washington Times, Philadelphia Inquirer (07/01) P. A8.(07/01/93), P. A4. Price, Joyce.

Dr. Antonia Novello, who resigned Wednesday as U.S. surgeon general, claims that she made **significant** steps in controlling the AIDS epidemic, and **becomes irritable when anyone indicates that she was not as aggressive as her predecessor, C. Everett Koop.** “We were counting patients with AIDS when Dr. Koop was surgeon general, and there were **29,000,**” said Dr. Novello. “As of today, there have been **289,000** people with AIDS, and we’re caring for many patients. There’s much more **treatment** today,” she added. **In 1989, President Bush appointed Dr. Novello, a pediatrician,** to a four-year post as surgeon general. At the time she was nominated, she served as deputy director of the National Institute of Child Health and Human Development. While in that position, **one of Dr. Novello’s biggest concerns was AIDS.** In addition, she said that **the disease was fast becoming one of the leading killers of children.** However, she left after three years and three months to take a job with UNICEF and to allow Dr. Joycelyn Elders, **another pediatrician,** to take over. Dr. Novello released what she considers a “timely, accurate, and sensible” report on AIDS three weeks ago, which was presented at the Ninth International Conference on AIDS in Berlin. It recognizes that **“sex and drugs are facts of life,”** and **provides instructions on condom use and the need for intravenous drug users to keep their needles clean.** Regarding her AIDS report—the first by a surgeon general in seven years—Dr. Novello said, **“I must prevent if I cannot cure. And in 1993 there is no magic bullet, no magic cure for AIDS. But what I can do is to prevent the [HIV] infections of the 130 people who are becoming infected each day”** in the United States.

**Burroughs, Rivals Clash as AZT Trial Opens.** Investor’s Business Daily (07/01/93), P. 35.

The role of government researchers in the development of the **anti-AIDS drug AZT** was disputed yesterday by attorneys for Burroughs Wellcome and two companies fighting to enter the market for the drug. The debate emerged after opening statements in a **patent-infringement lawsuit that could lead to less-expensive generic version of the drug that Burroughs Wellcome now sells exclusively.** Thomas Curnin, lead attorney for Burroughs, told a federal jury that **researchers from the National Institutes of Health did little under the authority of company scientists.** But Dan Webb, representing Barr Laboratories Inc., said **Burroughs researchers would not have known that their research on AZT was successful without the help of NIH scientists. Tests on mouse viruses yielded clues that would have led to a dead end if doctors Samuel Broder and Hiroaki Mitsuya of the NIH hadn’t risked infection and tested AZT on live samples of HIV,** said Webb. **Determining when a “definite and permanent” concept of using AZT against HIV emerged and who developed the concept is crucial to deciding the legitimate inventors.** Burroughs filed a lawsuit against Barr and Novopharm Ltd. after the two generic drug companies applied for federal permission to manufacture AZT. **The generic drug firms allege that AZT was developed with the assistance of public funds and research, while**

**Burroughs contends that its patents give it a monopoly on the drug until the year 2005.**

**Transmission of Drug-Resistant Virus Raises More Questions.** AIDS Alert (06/93) Vol. 8, No. 6, P. 84.

**Researchers have reported a sexually transmitted case of drug-resistant HIV infection for the first time. Even though it is too early to know whether this is a common occurrence, the new finding intensifies the debate over early antiretroviral treatment and emphasizes the virulence of the mutated virus, say researchers. The case was reported in the New England Journal of Medicine in April by University of Minnesota-Minneapolis researchers who describe a 20-year-old homosexual man infected by an HIV-positive partner. He soon developed a three-month, acute retroviral infection that did not respond to AZT treatment. Alejo Erice, lead author of the study and associate professor of medicine at the University of Minnesota--Minneapolis, said, "This confirms that resistant viruses are pathogenic—that is, they are able to produce disease." Keith Henry, director of the HIV program at St. Paul-Ramsey Medical Center in Minnesota, said, "So far it seems safe to say it doesn't appear to be real common," adding that most HIV-infected Americans were infected before AZT became available in 1987. The virus isolated from the patient after he was administered AZT had a significantly higher level of resistance than the virus obtained before he was given the drug. That led researchers to speculate that AZT may have selected drug-resistant viruses and hastened progression of the disease. Both Erice and Henry agree that their findings will incite additional study. The Minnesota researchers have already requested that a nationwide study be conducted by the AIDS Clinical Trials Group.**

**FDA Moves to Strengthen Blood Bank Quality Control.** Washington Post (07/02/93), P. A2. Schwartz, John.

*The Food and Drug Administration announced yesterday that it will implement new regulations to ensure the safety of the blood supply. The rules are designed to enhance quality control for blood banks and to set new standards for record keeping so that potential HIV-positive donations can be tracked and identified more easily. The guidelines call for blood banks and other suppliers of blood and blood products to develop strong quality assurance programs and employee training and competency evaluation programs. A new requirement that the guidelines incorporate is that blood centers must use a consistent "look back" policy. Blood centers currently have to retrieve and quarantine all blood from repeat donors found to be HIV-positive. The new regulations would facilitate communication between different institutions about problem donors. The rules would also force hospitals to inform patients who received blood from donors who subsequently tested HIV-positive. Food and Drug Commissioner David A. Kessler said the new rules are designed to provide enough layers of protection to make the system safer than it already was. He said, "The days are long gone when collecting and providing blood was a simple operation. The bottom line is a safer product and better assurances of quality." After the guidelines are published in the Federal Register, there will be a 60-day period for evaluation and public comment. If the FDA does not take any additional action, the rules will be put into effect in September.*

**AIDS is Claiming More Young Lives.** Baltimore Sun (07/02/93), P. 10A.

*AIDS has become the second-leading killer of young men in the United States, surpassing heart disease and cancer, according to a report released Thursday by the Centers for Disease Control. The federal agency said in its weekly report that AIDS is also closing in on unintentional injuries, such as car accidents, as the leading cause of death in men ages 25 to 44. Last month, the CDC reported in the Journal of the American Medical Association that AIDS was the No. 1 killer of young men in 64 American cities and five states. The CDC said yesterday that in 1991, the most recent year for which data are available, young black men died at three times the rate of whites as a result of AIDS. The death rates among Hispanics was twice that for whites. Dr. Peter Drotman, assistant director of public health in the CDC's AIDS division, said the agency is not sure whether the death rate among ethnic minorities indicates significantly higher infection rates, lack of access to health care, or both. "This study is based on data taken from death certificates,*

**which doesn't tell us much about socioeconomic status,"** said Dr. Drotman. "I'm sure if we had **a study** that measured ability to obtain insurance and access to medicine, we'd have better measures of that," he added. The CDC reported that AIDS deaths were **drastically increasing** during 1990 and 1991, even though most leading causes of death among young men and women remained relatively constant. Moreover, **among women, it seems AIDS will move from the sixth-leading cause of death in 1990 to the fourth in 1991, surpassing suicides and homicides and trailing behind only cancer, unintentional injuries and heart disease.**

**Calling Attention to the Need for AIDS Prevention and a Cure.** PR Newswire (07/01/93).

(New York, NY) The nine-member team of Princeton University students who began a climb of Alaska's Mt. McKinley on June 17 as a means of raising AIDS awareness is making steady progress to the summit of 20,320 feet. The "Climb for The Cure" was delayed for two days after there was a shift in weather patterns. But the effort has already achieved one of its goals—drawing attention to the need for AIDS prevention **among 13- to 24-year-olds**. In the past two years, the U.S. government has reported **a 62 percent increase in HIV transmission among this age group**. The climb is sponsored in conjunction with the American Foundation for AIDS Research (**AmFAR**). Participants had set their sights on raising \$250,000 for AIDS **research and education**, and so far more than \$200,000 has been raised. The team set up base camp June 14 on the Cahiltinah Glacier, and by June 21 had reached 11,000 ft. despite light snow, dense fog, and freezing temperatures. They reached 17,000 ft. by June 29, and expect to make the summit any day. In addition to the "Climb for The Cure," a symbolic movement called "Hopes and Prayers for The Cure to AIDS" has been initiated. Personal letters from **Elizabeth Taylor**, founding national chairman of AmFAR, and **Dr. Mathilde Krim**, AmFAR's chairman, **were sent to hundreds of world leaders**, asking for their "Hopes and Prayers for The Cure." Once the climbers return, other "Hopes and Prayers" that have been received will be encoded onto laser disks over the year, **placed in a time capsule, and buried within the shadow of Mt. McKinley** during a special dedication ceremony scheduled for the spring of 1994.

**Disease Detection International Inc. (DDI) Announces Brazilian Approval to Sell Rapid, On-Site, AIDS Test.** PR Newswire (06/30/93).

(Irvine, CA) Disease Detection International (DDI) recently received preliminary approval and specific recommendation from Sao Paulo, Brazil, to sell its SeroCard HIV-1 **rapid whole-blood HIV test**. In laboratory research performed at the Adolpho Lutz Institute in Sao Paulo, **the SeroCard HIV-1 test was found "suitable for use as a screening test to detect the presence of antibodies to the AIDS virus."** Other tests performed **within the state prison system** incited similar enthusiasm for DDI's **patented format**, which uses whole blood from a simple prick of the finger to perform the world's first **"whole-blood ELISA test in under eight minutes."** DDI's president and CEO, H. Thad Morris, said, "This enthusiastic endorsement was particularly significant in light of the fact that the state of **Sao Paulo, with a population of more than 33 million people, has more than three times the rate of infection than the nation of 150 million people as a whole.**" DDI will also formulate a joint venture that will be named Disease Detection International do Brazil and will be headed by Joao Carlos S. Bento, **a Brazilian businessman** who has been working with the company and Brazilian health officials to obtain approval of SeroCard. Bento said, "The possibility of having such a rapid and convenient test available within our health care system will be a **true asset** for our state and country, where **recent studies report up to 30 percent of donated blood may go untested for AIDS** due to the lack of sophisticated, expensive instrumentation and highly trained personnel in satellite regions."

**On Stage and Off: Anthem Against AIDS.** New York Times (07/02/93), P. C2, Collins, Glenn.

The American Foundation for AIDS Research (**AmFAR**) has established as its official anthem the song "The Day After That" from the musical "Kiss of the Spider Woman." In the play, the song, performed by Anthony Crivello, tells of a political revolution. However, when Liza Minnelli saw the show, she believed the song was "about a lot more than revolution." She added, "It's about

hope, about fighting despair.” With the approval of AmFAR and the song’s authors, Minnelli will record “The Day After That” for a video to be presented on World AIDS Day on Dec. 1.

**Walking Wounded.** Los Angeles Times–Washington Edition (07/01/93), P. B10, Natale, Richard.

**A neglected group of people affected by the AIDS epidemic** are the HIV-negative friends, families, or lovers of those who have died of the disease. These people witness successive deaths of friends and loved ones which can lead to grief, guilt, and subsequently to bouts of hypochondria, clinical depression, anxiety, insomnia, patterns of self-destructive behavior **and anhedonia**, an inability to derive a sense of pleasure from life. **Dr. Mark Katz, an HIV specialist** and member of the Los Angeles County AIDS Commission, says there is a significant other, friend, or family member for each of the approximately 40,000 HIV cases in Los Angeles to date. **The emotions felt by these people have been compared to the guilt experienced by survivors of the Holocaust, to the post-traumatic stress disorder that affects those who endure natural catastrophes like earthquakes and tornadoes, and to the battle fatigue syndrome soldiers withstand.** The difference with AIDS is that the epidemic continues to **ravagethrough** society, leaving no delay to complete the normal grieving process. “It’s like suffering post-traumatic stress but without **the post**,” said Los Angeles therapist Don Alonz. **Survivors of the AIDS epidemic often think of suicide or self-harm and frequently attempt to suppress their feelings with alcohol and drugs. Moreover, it’s a behavior that can lead to unprotected sex.** Peter Nardi, a sociologist at Pitzer College in Claremont, Calif., said **a pattern of unsafe sex among survivors may have less to do with fatalism than with feelings of monumental powerlessness.** Dr. Mark Katz said, “Safe-sex education is not enough. **We must encourage them to feel grief and pain** and to find things to rejoice about.”

**HIV Also Kills Developing White Blood Cells.** Science News (06/26/93), P. 406. Pennisi, E.

**Not only** does HIV kill mature white blood cells, but two research teams have found that **the virus also eradicates young immune-system cells before they have a chance to develop.** The reports were published in the June 24 issue of *Nature* and support the belief that HIV is most destructive in lymphoid tissues. **Grace M. Aldrovandl and colleagues from the University of California–Los Angeles School of Medicine treated a dozen SCID-hu mice with either no virus, inactivated virus, HIV from children with AIDS, or HIV cultured in the laboratory.** They subsequently evaluated the T-cells in each thymus implant by staining and sorting through these cells. **In the mice that received no active virus, 80 percent of the stained T cells demonstrated both CD4 and CD8 receptors, indicating immature cells. Mice with HIV from children had lost most of the CD4 cells,** the researchers reported. **The scientists next injected a much lower dose of different viral strains into additional mice. The T cells disappeared more slowly, and it seemsthe immature cells were destroyed first, followed by CD4 cells,** says **Jerome A. Zack**, who heads the UCLA group. He said that analyses of **viral genetic material** in these different cell types showed that **the immature cells were between five to 10 times more likely to harbor as much virus per cell as other cells.** The other research group from **Systemix Inc.,** in Palo Alto, Calif., found that **HIV infection spread through the thymus destroying most CD4 and immature cells within five weeks.** Both research teams agree that the findings **may help** in studying HIV in living organisms and **may help** scientists understand **how** HIV destroys the immune system.

**Legions of Life Scientists Will Be Called to the Front, as War on AIDS Intensifies.** Scientist (06/28/93) Vol. 7, No. 13, P. 1, Lewis, Ricki.

Although the AIDS epidemic continues to spread, **a growing amount of skilled AIDS researchers is expected,** according to experts. **While gauging the current exact amount of AIDS researchers is nearly impossible, the number is probably in the tens of thousands,** according to several estimates and predictions. Many offices contacted at the Centers for Disease Control in Atlanta **could not** provide precise numbers of AIDS researchers **at the CDC.** But a recent issue of the agency’s

Morbidity and Mortality Weekly Report indicated that CDC researchers **are indeed hard at work** on AIDS issues and problems in the fields of **behavioral studies, public health, and epidemiology**. Scientists say that after considering the number of AIDS researchers working in government agencies, academia, and industry, **the AIDS research work force is enormous**. However, right now most new hiring of scientists for AIDS-related research is **in academia and government**. Nevertheless, **once the epidemic is eradicated there will still be a need for legions of AIDS researchers**. Kenneth Bridbord, a **pediatrician** who is chief of the international studies branch of **the Fogarty International Center** at the National Institutes of Health, where he coordinates the training of foreign AIDS researchers in the United States, said, “The skills [for studying AIDS] are easily transferred to biomedical and public health issues that are emerging, and also to reemerging microbes and diseases.” He adds, “A glut of scientists with a broad spectrum of skills? I’d be glad to face that dilemma!”

## **PAPA GALLO’S SECRET SERVICE**

In her book *‘Fear of the Invisible’* (2008, 2009), Janine Robert’s investigation suggested that it was “President Clinton that gave the NIH a new director, Dr. Harold Varmus the orders to protect Gallo, since he had been called in to rename “LAV” and “HTLV-III” as “HIV.” And in June 1993 the Chicago Tribune reported that:

*...”the government’s long-running case against its star AIDS researcher, Dr. Robert C. Gallo, has been expanded to include a broader range of misconduct surrounding his decade-old claim to have discovered the cause of AIDS. [1]*

*“The ORI by now had drawn up a powerful Indictment (‘Offer of Proof’) against Gallo and Popovic. This it presented to the Department of Health’s lawyer-based ‘Research Integrity Adjudication Panel’. It was broad ranging and powerful. Here are some excerpts:*

§ *‘Research process can proceed with confidence **only if** scientists can assume that the previously reported facts on which their work is based are correct. If the bricks are in fact false...then the scientific wall of truth may crumble...Such actions threaten the very integrity of the scientific process.’*

§ *‘In light of the groundbreaking nature of this research and its profound public health implications, ORI believes that the careless and unacceptable keeping of research records [for proving HIV the cause of AIDS by Gallo and his team] ...reflects **irresponsible laboratory management** that has permanently impaired the ability to retrace the important steps taken.*

§ *[This] ‘put the public health at risk and, at the minimum, severely undermined the ability of the scientific community to reproduce and/or verify the efforts of the LTCB [Gallo’s ‘Laboratory for Tumor Cell Biology’] **in isolating and growing the AIDS virus.**’*

§ *‘Gallo’s failings as a Lab Chief are evidenced in the Popovic Science paper, **a paper conspicuously lacking in significant primary data and fraught with false and erroneous statements.**’*

§ *Gallo ‘repeatedly misrepresents distorts and suppresses data in such a way as to enhance his own claim to priority and primacy in AIDS research.*

§ *‘The [lead] Science paper contains numerous falsifications... the paper was replete with at least 22 incorrect statements concerning LTCB research, at least 11 of which were falsifications amounting to serious deviations from accepted standards for conducting and reporting evidence.’ Some of the captions to micrographs, descriptions of experiments and enclosed tables were ‘false and misleading’ [2]*

§     ‘The absence of virtually any assay data for the parent cell line is simply unbelievable. [Especially since this was used to develop **and patent** the HIV antibody blood test.]’

§     Gallo, **‘in violation of all research protocols**, impeded scientists wanting to follow up on his research ... imposed on others the condition that they did not try to repeat his work.’

“These are only a few selections from those indictments:

*“The Adjudication Panel, to which this indictment was submitted for action, was made up of lawyers not scientists. It decided to first consider the case of Popovic – and came to an amazing conclusion. They fully accepted that Popovic had published careless inaccurate and deceptive research, but still deemed him ‘innocent’ since the ‘intent to deceive’ had not been proved. They finished by astonishingly praising*

*Popovic’s research as published in Science in May 1984 as important for all time.*

*“This utterly shocked the scientists who had helped produce the ORI report. Their indictment had been supported with the testimony of over 100 scientists, and they had been expressly directed not to try to prove ‘intent’ in their indictment. How could the Panel now absolve Popovic from blame on the grounds they had not tried to prove ‘intent’? How could they absolve him of responsibility while accepting their conclusion that the key research he did on HIV was deeply flawed, contained false statements, and might have sent AIDS research off in the wrong direction? Furthermore, how could an Adjudication Panel made up solely of lawyers conclude by praising this research, when they as scientists had condemned it? They wondered darkly just who had advised the lawyers?*

*“The Panel was next to consider the case of Robert Gallo – but in face of the decision on Popovic, the ORI in disgust felt it had no choice but to drop its attempt to find Gallo guilty of scientific misconduct since they had been misdirected over the need to prove ‘intent’. They nevertheless declared their ‘fundamental disagreement’ with the Panel’s understanding of ‘the importance of clarity, accuracy and honesty in science,’*

*“But Gallo was not yet clear. **The Secret Service now presented the evidence they had unearthed to the Dingell Inquiry. They had been charged to examine for fraud the laboratory documents that Gallo had presented as legal evidence. They had discovered that many were ‘fixed’ before being presented. Documents written on different dates were changed on the same day. They found incriminating overlapping imprints of the changes on the enclosing folders.***

*“This was the clearest evidence of criminal fraud and was immediately presented to the State Attorney General in January 1994 in the expectation that a criminal prosecution would now be ordered, but he ruled it was **‘out of time’**. Too long had elapsed **under the five-year Statute of Limitations** since the fraud was carried out. Gallo thus may have escaped prosecution on a technicality. [3]*

*“But the investigators were not content to leave it there. Hadley and others went to see Varmus, the new Director of the NIH, to present the new damning evidence, **including more now produced by the Inspector General’s Inquiry on fraud in the Patent application for the HIV test.** The Inspector General had even expressed doubts on whether the related experiments were ever done! The Patent Examiner also now acknowledged ‘had she been aware of (the French AIDS test research) at the time she examined the blood test application of Gallo, she would have suspended Gallo’s application.’ [4]*

*“Varmus was persuaded – and had to act. In June 1994 Gallo was given a choice: prepare to leave the NIH – or face a new*

investigation that might be harder to escape from unscathed. **He decided to leave – in a year’s time.** It was then headlined on July 12th that: ‘US, France settle AIDS virus dispute. The NIH will give up millions in profit from Test Patent.’ The Financial Times reported: ‘US climb down in feud with the French over AIDS research.’ The NIH had at last acknowledged that there was justice in the French claim against them as the employer of Gallo. “

[1] Chicago Tribune June 6, 1993

[2] The Office of Research Integrity – Offers of Proof Report 1993.

[3] It was reported that ‘Federal prosecutors decided earlier this year not to bring Criminal charges against Gallo, citing what the Inspector General’s summary calls ‘several obstacles, jurisdiction concerns and procedural rules governing criminal prosecution’ including the five-year statute of limitations. Chicago Tribune 26 June 1994

[4] Chicago Tribune *U.S. INQUIRY DISCREDITS GALLO ON AIDS PATENT DIAGNOSTIC TEST. CLAIMS WERE RIDDLED BY HOLES, PROBE SAYS.* June 19, 1994.

But these kinds of charges miss the true nature of Gallo’s motivations, and especially, the faith-based scientific enthrallment surrounding the erection of the AIDS paradigm.

## **RIVAL SCIENTISTS QUESTION RESEARCH ON AIDS LESIONS**

Gallo critics say tests of drug may be invalid. By John Crewdson, Chicago Tribune 9 June 1994

*A major medical journal has taken the highly unusual step of publishing an article questioning the veracity of research appearing in a rival journal, raising tempers as well as new questions about what had appeared to be **one of the few hopeful developments** regarding AIDS.*

*The article, published in Thursday’s issue of the Journal of the American Medical Association (JAMA) by a little-known research group from the University of Arizona, is critical both of the journal Science, where the original article appeared, and of its senior author, Dr. Robert C. Gallo, one of the biggest names in AIDS research.*

*The JAMA article contains some of the most pointed language ever seen in the normally **well-mannered** scientific prints, dismissing a central conclusion of the Gallo piece as “highly dubious” and declaring that “the validity of the peer review process and self-correcting nature of scientific inquiry are also called into question.”*

*Gallo’s Science article, published two years ago, concerns Kaposi’s sarcoma, or KS, an often-fatal, cancer-like skin condition whose origins and frequent appearance in gay men (**but not women or heterosexual men**) with AIDS represent **one of the enduring mysteries** of that disease.*

*Gallo’s report, that a drug named SP-PG appeared to inhibit the development of Kaposi’s sarcoma in mice, was seen as welcome news by KS patients and by physicians desperate for better AIDS treatments. “It **really prevents** the development of lesions,” Gallo said at the time.*

*The Arizona group states flatly, “**Serious systematic errors and omissions flaw the original study, and we cannot replicate some of the pivotal findings.**” The group emphasized, however, that its findings did not necessarily mean the drug is ineffective against KS.*

Two weeks before the Science article was submitted, in July 1992, Gallo was ordered by Dr. Samuel Broder, director of the National Cancer Institute, to review all “primary data” for **every** manuscript published by Gallo’s laboratory.

A spokesman declined to say whether Gallo had done so in this case, saying only that Gallo had “personally reviewed” the manuscript before it was submitted to Science. He said Gallo planned to send JAMA a response to the article “within the next few days.”

The National Cancer Institute, where Gallo heads a major research laboratory, struck a commercial development deal with Daiichi Pharmaceuticals, the Japanese company that owns the U.S. patent on SP-PG, which quickly began laying **plans to try the drug on Americans** with AIDS.

The NCI declined to disclose the amount of money Gallo’s laboratory received from Daiichi to support Gallo’s research on SP-PG, but sources put the figure at \$75,000. After the deal was announced, Gallo told the Wall Street Journal he did not stand to earn anything personally.

“As to what Daiichi could earn, I don’t know,” Gallo said. “I’m not an economist. But if it helps patients, that’s what I’ll get out of it.”

Daiichi’s stock gained 6 percent on the Tokyo exchange **the day the Gallo article appeared in Science.**

Despite the initial fanfare, **the SP-PG compound, produced by a strain of bacteria found in soil**, currently is being tested in only **seven patients** at the University of Southern California. The researchers in charge of those trials reported recently that several subjects had experienced **unexpected bleeding and other side effects**, and that only two of the seven appeared to have improved since taking the drug.

“We pursued this because it’s clinically important,” said Dr. Marlys H. Witte, a professor of surgery at the University of Arizona College of Medicine and the principal author of the JAMA article, which also is signed by her husband, Dr. Charles L. Witte, and four co-workers.

“This article seemed to be offering some drug treatment that might be useful. When we read it, many important questions were raised. **The answers that we were given were not legitimate or relevant.**”

Marlys Witte explained in the article that she and her colleagues had turned to the Chicago-based JAMA **only after Science and Gallo refused to publicly acknowledge their criticism.**

“More than 2 years have passed since the (Gallo) article was published and our efforts to address the validity of the data and conclusions in an open scientific forum were initiated,” she wrote. “No follow-up studies by these authors have appeared in print that might clarify the points in question.”

**Daniel E. Koshland Jr., the editor of Science**, said in a statement that he still believed “our actions were justified” in not publishing the Witte group’s original criticism of Gallo. Koshland also noted that the JAMA article contained new information reflecting the Witte group’s unsuccessful attempts to reproduce Gallo’s results.

The deputy editor of JAMA, Dr. Drummond Rennie, said he had made **the unusual decision** to publish an article critical of work appearing in a rival journal only after Witte’s group “provided us with ample evidence that Science had absolutely finished with them.”



Rennie added that the Witte group's questions about Gallo's research "seemed valid" to him, and that an earlier letter Witte sent to Science raising those questions "**had been rejected for reasons that didn't hold scientific water.**"

The third major biomedical journal in this country, the New England Journal of Medicine, suffered the same treatment last year when JAMA published a critique of one of its more important research articles. "I think it is an odd thing to do, and **it's certainly something we wouldn't do,**" said **Dr. Jerome Kassirer**, editor of the New England Journal.

When the Science article appeared it was viewed with particular interest by Witte and her Arizona group, one of the few in the country then concentrating on Kaposi's sarcoma. In particular, Witte's attention was focused on photographs the article contained **of four mice**, their skins splayed and pinned back to reveal their insides.

A central point of the Gallo article was the ability of SP- PG to prevent whatever "growth factors" might cause Kaposi's sarcoma lesions from leaking out of the blood vessels and forming lesions in the surrounding tissues. Hoping to demonstrate this, Gallo's assistants inoculated some of the mice with varying doses of SP-PG and others with different drugs or none at all.

They next infected the mice with Kaposi's sarcoma cells, waited **a few hours**, then injected them with blue dye through a vein in the tail. When the amount of blue dye that had leaked from the blood vessels in each mouse was measured, the Science article said, it was greatest in those mice that received no SP- PG and least in those that got the most.

When the Wittes examined the pictures closely they saw something they thought odd: the tails of the untreated mice in which the most dye apparently had leaked through the vessel walls were an intense blue color, while the tails of mice treated with SP-PG were not.

**To the Wittes, the difference suggested that Gallo's assistants had made a serious mistake, injecting dye into the tissues of the untreated mice as well as into the tail vein.** If the Wittes were right, the increased leakage in the mice that did not receive SP-PG would actually have been caused **by a botched injection.**

A month after the Gallo article appeared in Science, the Wittes and their co-workers sent a letter to Science. Entitled "**The Tell-Tale Blue Tail,**" the letter described how the Arizona group had achieved an effect similar to one reported by the Gallo group **via just such a "botched" injection of blue dye into the tail vein of a mouse.**

The letter also questioned Gallo's claim that a sizable amount of dye-containing mouse blood had leaked into a small Kaposi's lesion in just a few minutes. "Even a localized scalding burn of that tiny dimension," the Wittes wrote, would not cause the loss of so much blood in so short a time.

The Wittes also questioned Gallo's assertion that the blue dye, which they termed "notoriously difficult" to extract from soft tissue, had been removed by Gallo's assistants from the mouse lesion overnight.

They also expressed "surprise"-the scientific equivalent of the raised eyebrow-at the "smoothness" of the graph in the article showing that blood vessel leakage decreased in step with increases in the dosage of SP-PG.

**Science sent the Wittes' letter to Gallo, who dismissed their criticism as "an extraordinary waste of time and effort."** Gallo noted that the Science article contained other claims for the effectiveness of SP-PG that the Wittes had not addressed, and he included a picture of another mouse that he said disproved the Wittes' theories.

Science asked four other scientists, whose identities it did not disclose, to read the Wittes' letter and Gallo's response and decide

whether they should be published in tandem.

*All said no, even though two reviewers shared the Wittes' surprise that so much blood had leaked from the blood vessels in so short a time, and that Gallo's lab had been able to extract the blue dye so easily. The most important shortcoming in the Wittes' arguments, a third reviewer said, was their failure to have tried to reproduce Gallo's experiments themselves.*

*The Wittes were not satisfied. Gallo's response, the new picture and the comments of the anonymous reviewers, they told Science, **"not only fail to answer our original questions but actually raise additional disturbing issues."***

*The Wittes sent Science a lengthy written analysis from a reviewer of their own, Dr. Hugh J. Carroll of the State University of New York, who found much merit in the Wittes' claims. "The editors of Science were remiss," concluded Carroll, "in foreclosing the opportunity for exploration and debate on a topic that I perceive to be still unsettled."*

*Science sent the expanding collection of criticisms and rebuttals to yet another scientist, identified only as "someone in whom we have the utmost confidence." Like previous reviewers, this one pointed out that while some of the Wittes' assumptions **were reasonable**, "for all their concern and criticism" the Wittes had not tried to test them by conducting the experiments described by Gallo.*

*In the fall of 1992, Witte and her group set out to do what their critics suggested, **beginning from scratch to replicate Gallo's experiments with the mouse and dye, even persuading Gallo to send them some of the special Kaposi's sarcoma cells he used to generate the lesions in his mice.***

*"Perhaps the most serious aspect of the whole matter," the JAMA article concludes, **"has been the reticence and obstacles encountered to public airing of our questions and the inability of the peer review process to correct itself once errors and inconsistencies were pointed out and bolstered by further experimentation."***

## **THE CONCORDE STUDY CALLS INTO QUESTION THE VALUE OF T-CELL MEASUREMENTS, AND DAVID ACER IS EXONERATED OF COMMITTING HOMICIDE.**

**1994**, the Concorde study, which was up until then the longest, largest, and most carefully controlled AZT trial reported:

*"The results of Concorde do not encourage the early use of zidovudine in symptom-free HIV-infected adults. They also call into question the uncritical use of CD4 cell counts as a surrogate endpoint for assessment of benefit from long-term antiretroviral therapy" [28].*

Also in 1994, the details came out of why Dr. David Acer was accused of committing homicide. It was discovered that on the basis of mistaken charges that he spread "HIV" to his patients [29], which the CDC later exonerated him of doing (after his so-called AIDS death). The CDC could find no evidence the dentist's HIV-positive patients contracted their infections from him because their virus' DNA **did not match his**, and also concluded the dentist's patients did not contract the virus from one another — in effect, that unclean dental implements **did not** act as conduits, one of the patients was **never** in the room with him and hygienists had cleaned the teeth of others...

## **CANADIANS URGE THEIR "HIV-POSITIVE" WOMEN TO GET ABORTIONS.**

In 1994, the Canadian government advised: *One might expect that women found to be seropositive would opt for an elective*

abortion. (Screening for HIV antibody. Health, Canada, 1994).

### “HIV-NEGATIVE CHILDREN HAVE “HIV’S” CAPSID PROTEIN P24.

In **1994**, p24 is found in the thymus gland of “HIV-negative” children [37]:

*Abstract: An immunopathologic study of normal and severely atrophic thymuses (STA) was undertaken in order to evaluate the expression of human retrovirus (envelope and core) molecules in thymic epithelial cells (TEC) in HIV negative children. Both normal and STE thymuses disclosed p19, p24, p39, p45 and p55 viral core proteins as well as gp46, gp63 glycoprotein of envelope origin. No evidence of gp160, gp120 and gp41 molecules were observed in TEC which suggested endogenous lack of receptor molecules for HIV. The results are discussed in the context of possible thymus oriented autoimmune reaction in HIV and non-HIV bearing patients and in consequence, severe injury of TEC forming microenvironment.*

### FLU VACCINES CAUSE “HIV-POSITIVE” TEST RESULTS IN “UNINFECTED” PEOPLE.

In **1995**, it was reported that flu vaccines cause false positive “HIV” test results [30]. It was suggested later that this false positive rate was believed to have been the result of bad “HIV” tests, rather than the possibility that “HIV’s” molecular signature includes epitopes that cross react with those of influenza viruses. In 1995, it also was confirmed **again** that about **67% of infants that test “HIV” positive at birth serorevert** (reverse) their “HIV-positive status by 18 months [31].

### PCR INVENTOR AND NOBELIST KARY MULLIS SAYS, THERE ARE TOO MANY VIRUSES AND NO VACCINE IS POSSIBLE: AIDS IS A CHEMICAL CHAIN REACTION!

In **1995**, the Nobel Laureate and developer of The Polymerase Chain Reaction used to detect viral load against his warnings that PCR cannot detect viral load, Kary Mullis, published a new hypothesis attempting to explain how immune collapse **need not be due** to any particular virus, but by an immunological chain reaction:

*If previously latent virus with a distinct epitope would provoke a new immune response, every immune response would be perpetually generating new immunogens. The immune system so infected would be perpetually generating new immunogens. As the frequency of infection increased such an immune chain reaction would be progressively more debilitating for the stability and effectiveness of immune function [32].*

This hypothesis was important because it predicted that a vaccine against any specific virus would be ineffective against AIDS:

*If correct, then an experimental animal model of AIDS should be induced in laboratory animals by infecting them at a low multiplicity with a very large number of diverse viruses. One way of doing this would involve collecting the blood from a large number of wild mice from geographically distant locations, mixing it together and injecting it into healthy mice. The number of mice that would be required to produce such a lethal injection or series of injections is not predicted by this hypothesis, although from the numbers suggested by the behavior patterns of the human victims of AIDS, the number of individuals whose viruses must be pooled might be quite high.*

Mullis’s hypothesis also suggested that:

1. Some level of **diverse infection** would cause AIDS-like malfunction of the immune system to appear rapidly, and that this **could not be reproduced** by simply isolating a particular infectious species and infecting similar animals with only this species.

2. Blood from a single human AIDS patient should be capable of transferring the appropriate level of **diversity of infection** to another organism, given that the recipient organism contains a functional human immune system.

3. Aliquots of an appropriate dilution of the blood from a single AIDS patient injected into a large number of experimental animals **with a human immune system** would **not** be able to produce AIDS-like immune dysfunction in any one of them.

### **OFFICE OF TECHNOLOGY ASSESSMENT SAYS THERE HAVE BEEN 30 “HIV-VACCINE” TRIALS TO DATE: SOME CAUSE CANCER, AND OTHERS CAUSE “ORIGINAL SIN.”**

In **1995**, the conclusions of The Office of Technology Assessment Book (1995 Congress of the United States: Office of Technology assessment. Adverse Reactions to HIV Vaccines: Medical, Ethical, and Legal Issues. Roger C. Herdman, Director) were presented to the 1995 Congress of the United States by the AIDS Research Advisory Committee (ARAC) of the National Institute of Allergy and Infectious Diseases (NIAID).

These conclusions recommended that Phase III clinical trials with enveloped vaccines

**should not proceed in the United States because of scientific, political, and ethical issues, and because of the significant level of scientific uncertainty about the wisdom of immediate trials. Some of the conclusions included:**

*Vaccines may cause a false-positive HIV screening testing test resulting in discrimination against vaccine recipients in, for example, military service, health insurance, life insurance, employment, and travel.*

*Participation in an HIV vaccine trial, itself, may result in stigmatization, as others may assume that all vaccine trial participants are members of groups, such as injection drug users and men who have sex with men, who are at increased risk for HIV infection.*

*Vaccinees, relying on the protection afforded by an experimental vaccine, may engage in behaviors that increase their risk for HIV infection.*

*There is the potential for the viruses to be inadequately attenuated, for an adequately attenuated viral vaccine to cause disease in immunocompromised individuals (**read AIDS patients**), and for an adequately attenuated virus to revert to virulence. There is also concern that a live attenuated vaccine could induce **tumors**.*

**From page 19: (Product liability)”**

**“Almost 30 candidate vaccines have been in clinical trials” (before 1995).**

**From page 21: (“Development of Cancer”):**

*“There has been speculation that, because HIV is a **retrovirus**, an HIV vaccine might cause cancer many years after vaccination. Although a manufacturer is **not** liable for injuries caused by **unforeseeable** dangers in its **products**, there may be **some** question as to whether a manufacturer adequately investigated a suggested risk (i.e. induction of cancer). Given **THE NEED** (EMPHASIS MINE) for an HIV vaccine, it appears **unlikely** that a manufacturer would be held responsible for distributing a vaccine with a risk (causing cancer) that **could not be verified** at the time it was released.”*

Toward the end of the Congressional document, the term “original antigenic sin” is advanced to describe:

A) When a vaccinated individual is exposed to a non-cross-reactive strain of HIV that induces the production of antibodies specific

**for the vaccine strain that are unable to neutralize the newly encountered strain** (in other words when a vaccine doesn't work).

B) *The fixing of an immune response in a “non-adaptive pattern.”*

C) **When vaccinated individuals may be no worse off than unvaccinated individuals** because unvaccinated individuals also have a lag in generation of antibody to HIV because their immune response has not been “primed” by vaccination.

#### **NO CONSISTENT CELL CULTURE MODEL OF “HIV’S PATHOGENIC EFFECTS.**

Also in **1995**, a critical analysis of the HIV-T4-Cell-AIDS hypothesis appeared in *Genetica* by Papadopulos-Eleopulos et al., that reviewed the problematic issues associated with culturing “HIV” in vitro, and questioned whether the in vitro models can model anything like the AIDS syndrome [33]. The work of Laurent-Crawford et al. (of the Pasteur group) was particularly criticized because of documented variations in cell culturing techniques they had described for “HIV” production In Vitro that resulted in unbalanced signals, apoptosis, syncytial formation, or persistent non-pathogenic viral production. Papadopulos-Eleopulos et al., attributed these various effects to oxidation, and not differences due to lymphocytes being “immature or “mature,” as suggested by Laurent-Crawford et al., or whether, as Gallo et al. had achieved, the molecular signature of “HIV” had been amplified using continuous cancer cell lines which express reverse transcriptase, the enzyme once thought to be specific to retroviruses.

#### **DALAKAS PUBLISHES IN NATURE MEDICINE THAT ARVS KILL MITOCHONDRIA.**

Also in **1995** Lewis Dalakas published an important paper in *Nature Medicine* entitled: Mitochondrial toxicity of antiviral drugs. [Nat Med. 1995 May;1(5):417-22].

*“Clinical manifestations of ANA [Antiviral Nucleoside Analogs, such as AZT] toxicity: It is **self-evident** that ANAs, like all drugs, have side-effects. However, the prevalent and at times serious ANA mitochondrial toxic side-effects are particularly broad ranging with respect to their tissue target and mechanisms of toxicity: **Haematological toxicity, myopathy, cardiotoxicity, hepatic toxicity, peripheral neuropathy.**”*

#### **NEW ENGLAND JOURNAL PUBLISHES FIRST CDC WORKING GROUP SURVEY OF INFANT PCP: CLAIMS THAT TREATMENT WASN'T GIVEN, AND WHEN IT WAS, IT FAILED TO REDUCE TRANSMISSION.**

**1995** Medical records of 300 children given a diagnosis of PCP between January 1991 and June 1993 were analyzed to determine why treatment according to the 1991 guidelines for prophylaxis against PCP either was not given or failed to prevent the disease. **CONCLUSION: In the United States the incidence of PCP among HIV-infected infants has not declined.** (N Engl J Med. 1995 Mar 23;332(12):786-90). Prophylaxis against *Pneumocystis carinii* pneumonia among children with perinatally acquired human immunodeficiency virus infection in the United States. *Pneumocystis carinii* Pneumonia Prophylaxis Evaluation Working Group. Simonds RJ, **Lindegren ML**, Thomas P, Hanson D, Caldwell B, Scott G, Rogers M. Division of HIV/AIDS, Centers for Disease Control and Prevention, Atlanta, GA 30333. Comment in: N Engl J Med. 1995 Aug 3;333(5):320-1).

#### **SEXUAL TRANSMISSION STUDIES SHOW NO SEXUAL TRANSMISSION OF “HIV.”**

In July of **1995**, The Journal of Clinical and Infectious disease published a paper claiming that:

*We studied **50 sexually active couples with discordant antibody results** [one positive, one negative]...seronegative partners continued to have **negative results** in all tests for a mean follow-up period of 17 months despite ongoing sexual*

*relations with their seropositive partners....approximately one-half of each group reported some instances of unprotected intercourse...intercourse with outside partners was uncommon in both groups, as was current illicit drug use.* (Clin Infectious Disease. July, 1995; 211).

In April **1996** study in Nature Medicine focused on 24 hetero- and homosexual men who've remained HIV negative despite "histories of multiple high-risk sexual exposures to HIV-1," including "sex with multiple HIV-1-infected partners," or "long-term relationships involving unprotected sexual intercourse over many years [with] predominantly a single HIV-infected partner." "**All subjects were HIV-1 negative,**" even though "several [of their] partners succumbed to AIDS." (Nature Medicine. 1996 2(4)).

### **TOMMY MORRISON, TWO-TIME HEAVYWEIGHT CHAMPION ACQUIRES "HIV" WITHOUT ANY TEST RESULTS OR PROOF.**

**1996.** Tommy Morrison was a boxer who was on top of the world in 1996 who refused to "lay down in the ring." Because of a worthless "HIV" testing program that has failed to incorporate modern advances in the evolving biomedical conundrum of AIDS, he became another victim of the medical, pharma-ceutical, and government "HIV" agenda. While his boxing successes were legion and while he continues to box in states that don't subject boxers to the witchcraft of "HIV-testing" in order to qualify to fight, Morrison's life and career nevertheless have been ruined by "HIV/AIDS" stigma and ignorance. In recent years, Morrison also has and is being deliberately attacked by "the silent war" warriors, and so-called "AIDS activists," who militate against human health, reason, and human rights-and by the propaganda war that is being waged by a malicious element in our medical industrial complex known as The AIDS establishment, or what can be described as a McCarthyesque *AIDStruth* cabal.

As of 2012, March, Tommy Morrison never has been shown his "HIV" testing results with a correct date of birth, social security number, or his own doctor's signature ordering the results. Contrary to doctors' assessments 15 years ago regarding their prophetic severity of his "HIV disease," even suggesting that soon he soon would be unable to open even a car door due to rapid AIDS illnesses setting in, Morrison has obtained and defended two heavyweight championship belts, he has had two "HIV-negative" sons, with two "HIV-negative" wives, he has led a productive, healthy life, and recently, he has petitioned every federal agency responsible for the HIV-AIDS scam to show him any evidence of a real, infectious, or pathogenic virus in his blood (or lymph nodes), while continuing to make his living coaching disadvantaged youth to become young boxers.

If you watch Youtube-posted interviews with Mr. Morrison, it is difficult to see a person sit-

ting before you who appears to suffer from "mental defects," someone who appears hopelessly

drug addicted or alcoholic, or who exhibits the demeanor of a gun runner, or advanced "hepatitis C disease," and, by the way, someone who is an advanced 15-year "HIV/AIDS" victim, as Morrison has been portrayed repeatedly in the press. Instead, you would find one of the most charming, personable, humorous, soft-spoken, and healthy two-time heavyweight champions of the world that you will ever meet. For more than a decade, his goal has been to get back in the ring and fight, while he continues to train young boxers. What you are told and led to believe about "HIV/AIDS," or about

this two-time heavyweight world champion by mainstream media and the Federal Government-backed drug salesmen that direct the NIH, CDC, and other so-called Public Health Service

institutions, is nothing less than what should be regarded as human rights violations of the worst

kind.

On April **1996**, Abbott Laboratories published their test kit insert which claimed that:

*In low prevalence populations the predictive value [of an HIV test] was 11.1% , while in populations with known HIV-1 infection, the predictive value was 97.1% .*

In **1997**, Advances in HIV Testing Technology- AIDS Education and Prevention published that:

*In a high risk group, “the positive predictive value (PPV) of a home screening test would be 67%; 33 of 100 would be false positives. With a lower prevalence the PPV drops to 17%, and 83 out of 100 positive tests would be false.*

**EVEN THOUGH CONDOMS AREN'T USED BY 25% OF 175 SERO-DISCORDANT COUPLES, NANCY PADIAN BECOMES THE WORLD'S BEST AIDS-PREVENTION COUNSELOR AND REPORTS ZERO SEROCONVERSIONS IN A 10-YEAR STUDY IN NORTHERN CALIFORNIA.**

In **1997**, it was also reported that “**no** seroconversions” were observed among 175 HIV-discordant couples (where one partner tests “HIV” positive, one “HIV” negative), for a total of approximately 282 couple-years of follow up in a 10- year study [34]. The reason for no conversions advanced by Padian et al. to explain why “HIV” was not transmitted by anyone during her 10 year study was because the people in her study were instructed as to how to have sex and how not to have sex with each other, and not because “HIV” isn't dangerously infectious, reinforcing the notion that if people would only listen, and behave properly, there wouldn't be an AIDS pandemic.

**THE PROBLEM WITH VIRAL LIKE PARTICLES AND THE BASIS OF “HIV's” MOLECULAR MARKERS.**

Also in **1997**, two teams of investigators, one consisting of a French-German collaboration [35], and another whose investigators were involved in the AIDS Vaccine Program, SAIC, National Cancer Institute-Frederick Cancer Research and Development Center, Maryland [36], reported that PHA (phytohemagglutinin) and IL2 (interleukin-2) stimulated healthy cells to produce Human Immunodeficiency “viral like particles” and the molecular signatures of “HIV” only when stimulated with PHA and IL-2. They also claimed that microvesicles were a source of contaminating cellular proteins found in purified HIV-1 preparations, as their titles of their papers suggest:

Cell membrane vesicles are a major contaminant of gradient-enriched human immunodeficiency virus type-1 preparations (Gluschankof et al.,1997) [35].

*Microvesicles are a source of contaminating cellular proteins found in purified HIV-1 preparations (Bess et al., 1997) [36].*

This work not only underscored the problem that there are no tissue or cell culture models of “HIV's pathogenic effect as raised by Papadopoulos-Eleopoulos et al. in their Genetica article [33], but these facts raised issue regarding the basis of Gallo's and Montagnier's isolation, and challenged Duesberg's claims that the nucleic acids have been uniquely cloned, discrediting the hypothesis that the signature of “HIV” is due to a specific virus.

In the Bess et al. paper, cellular debris was not distinguishable from other objects in the electron micrographs, and the Bess et al. authors specifically emphasized that:

Identification and analysis of the virus are complicated by the presence of cellular membrane vesicles which COPURIFY (my emphasis) with the virus.

We recently reported a proteolytic procedure (Ott et al., 1995b) that effectively removes greater than 95% of proteins associated with these membrane vesicles. This procedure has allowed us to demonstrate that the cytoskeletal proteins, **actin, ezrin, moesin, and cofilin are located in the interior of virions.**

Such “advancements” in “HIV isolation” raised questions such as: If the cell’s cytoskeletal proteins, actin, ezrin, and other proteins are located INSIDE the virions, how can one tell if p24, for instance, which is a faint band on most cellular gels that come from non-“HIV-infected” cells in most labs, and the other molecules thought to represent the specific molecules of “HIV,” aren’t also proteins of cellular origin?

Other issues the Bess et al. paper raised also revisited the issues that Papadopulos-Eleopulos had raised in their Genetica article [33]:

*PHA activated human PBLs were also shown to produce microvesicles that incorporated cellular proteins (Fig. 6).*

**DIVISION OF AIDS (DAIDS) SAYS EVERYBODY’S GOT “HIV’S CAPSID PROTEIN IN THEIR CELLS, BUT WANT TO CONVICT ONLY THOSE WITH MORE THAN 30pg/ml P24 PROTEIN.**

In 1997, The DAIDS official “HIV” culturing manual was published presenting a series of standard protocols for culturing “HIV.” From the Reporting Results Section (section VII), a rationale was presented to unequivocally identify “HIV-infected” cells as truly “infected” by “HIV” cell culturing labs if:

*Two **consecutive** HIV p24 antigen VQA CORRECTED values of > 30 pg/ml (from a healthy donor source), of which the second value is at least four times greater than the first value or out of range” (O.D.>2) or*

*Two **consecutive** HIV p24 antigen VQA CORRECTED values (from a healthy donor source) that are out of range” (Optical density.> 2); or*

*Three **consecutive** HIV p24 antigen VQA CORRECTED values of > 30 pg/ml (from a healthy donor source), where neither consecutive value is > **four times** the previous sample, but the third value is at least four times greater than the first.*

**TRANSLATION:** Thus, under DAIDS recommendations, if someone should have sex without informing the other participant(s) of their more than 30pg/ml p24 status, the “HIV-positive” person can be arrested by the State, thrown in jail, and tried and convicted for murder, if pregnant, a woman may be urged or even forced to abort, or to have a hysterectomy, as in the Serrano case (see **DIALOGUES WITH THE FIGHTERS OF AIDS**). But if one has **less than** 30pg/ml p24 (let’s say one has 29pg/ml of p24 “HIV-capsid protein”), one can donate blood or is free to sleep with anybody or everybody one can without telling them their less than 30 pg/ml status, which constituted p24 protein discrimination, rather than a positive “HIV” test result indicating the presence of an exogenous retroviral infection.

**“HIV’s” MOST IMPORTANT PROTEIN MOLECULE, REVERSE TRANSCRIPTASE IS FOUND IN CHICKEN VACCINES, AND IS DESTROYED WITH DNASE.**

In 1998, as a substitute for viral isolation and purification, and although during the AIDS era, the enzyme reverse transcriptase (RT) was the first of many surrogate molecular signatures used to detect “HIV,” The Center For Biologics Evaluation and Research Advisory Committee on Vaccines and Related Biological Products claimed in November in a chapter regarding the Update On Reverse Transcriptase Activity In Chicken Cell Derived Vaccines, by Dr. Arifa Khan (pages 13-15), that:



Initially Boni et al. (1996) published that low level reverse transcriptase activity was detected in ALL chicken cell derived vaccines using a highly sensitive PCR-based reverse transcriptase assay called PERT, which can detect **one to ten virions** which was reported to the WHO, and then additional studies were done by several laboratories in Europe, as well as the U.S., including the NIBSC, the CDC, as well as labs in the FDA to confirm this initial finding. However, after further work, it was discovered that this **reverse transcriptase activity could be eliminated by treatment of extracts with DNAase**, and that using **Alu-based EAV sequence integration studies**, that no integration of anything derived from the chicken cell supernatants was detected in Human PBMC cells.

**TRANSLATION:** This claim is absurd because reverse transcriptase, supposedly a ubiquitous molecule present to help telomeres replicate, is a protein, and DNAase should not disturb its structure or presence after treatment(s) of vaccine extracts with this nucleic-acid destroying enzyme.

Also in **1998**, the head of the Swiss and European Blood Banks, the late Dr. Alfred Hassig (1921-1999) and his associates, including Dr. Hans Kremer (a specialist in drug addiction medicine), and a virologist (Stephan Lanka), made a series of claims suggesting that:

The question of the specificity of the anti-HIV antibody test has to be re-evaluated as it was shown that the viral enrichment obtained from co-cultivations of patients lymphocytes with fetal cord blood by Barre-Sinoussi et al. and leukaemia cells by Gallo et al., exclusively consisted of proteins of the cell types used in the cell culture. This precludes a clear separation of presumed retroviral and cellular proteins or extracellular matrix proteins. In this context it was shown that the anti-HIV antibody test detects autoimmune antibodies directed against cytoskeletal proteins e.g. the liver cells. Strongly augmented **anti-actin** autoantibodies is considered close to pathognomonic for chronically active hepatitis. **The original assumption that reverse transcription from RNA to DNA is evidence for the existence of retroviruses was wrong.** In fact reverse transcription is a vital mechanism for the maintenance of the genome. The decrease in numbers of circulating lymphocytes can be explained by **a stress-induced hypercortisolism. Up to date, direct HIV-mediated destruction of CD4 lymphocytes could not be proved.** The same is true for the measuring of the **“viral load.”** Shortcomings of the applied method to quantify the “viral load” do not permit definitive conclusions. Possibly, it may be taken as an expression of a stress induced weakening of the cellular immune reactions, in the course of which, **nucleoside fragments resulting from the current cell turnover are inadequately eliminated.** Furthermore, the treatment of patients with nucleoside analogs has a toxic effect on both the genome of the cell nucleus **and the mitochondria.** The latter, therefore, may produce insufficient amounts of ATP, causing **organ failure** and, eventually, **death.** The synthetic protease inhibitors used these days are associated with serious side-effects. Therefore, it seems worthwhile, in these patients, to bring back the catabolic situation of whole body inflammation to homeostasis by administering anabolic phyto-polyenolic compounds.

In March, **1998** an “AIDS Alert” article on rapid tests appeared claiming:

*Whether the tests will perform as well in the United States as they have abroad is still unknown, experts add. For one thing, using a single rapid test **in a low-prevalence population** will give a lower positive predictive value....That error rate **won't matter much** in areas with a high prevalence of HIV because **in all probability** the people testing false-positive **will have the disease.** But if the same test was performed on 1,000 white, affluent suburban housewives – a **low-prevalence** population – in all likelihood **all positive results will be false**, and positive predictive values **plummet to zero.** (Coming to your clinic: Candidates for Rapid Tests. Aids Alert, March 1998).*

**THE JOURNAL, ‘AIDS’ REPORTS RISK OF DEATH IN ARV-TREATED INFANTS IS RAPID AND INCREASES DURING FIRST 3 YEARS OF LIFE.**

In **1999**, it was published in The Journal, AIDS, that children born to ZDV-treated mothers

*“are more likely to have a rapid course of HIV-1 infection compared with children born to untreated mothers, as disease progression and immunological deterioration are significantly **more rapid** and the **risk of death** is actually increased during the first 3 years of life” [38].*

## **GOAT AND COW PROTEINS CAUSE “HIV-POSITIVE TEST RESULTS.**

In **1999**, it was also known that goat and cow proteins cause “HIV-positive” reactions [39].

## **DR. HOWARD URNOVITZ TESTIFIES BEFORE THE COMMITTEE ON GOVERNMENT REFORM THAT HE DOESN'T WANT TO VACCINATE PEOPLE AGAINST THEIR OWN GENES TO CAUSE ADVERSE EVENTS WITH “HIV-SUBTYPE “O.”**

On August 3, **1999**, written testimony of Dr. Howard B. Urnovitz to the Committee On Government Reform and Oversight was presented claiming that:

*House of Representatives I am grateful to this committee for allowing me to address the issue of vaccine safety. I am Dr. Howard B. Urnovitz. In 1979, I received my doctorate degree in Microbiology and Immunology from the University of Michigan, where I studied vaccines. I am testifying today as the Scientific Director of the Chronic Illness Research Foundation. For the record, I am also the chief science officer of a biotechnology corporation.*

*My testimony will describe the insights of recent scientific studies into the health consequences of exposing individuals to both toxic and foreign biologic materials, particularly multiple bacterial and live virus vaccines. **The conventional wisdom concerning the use of vaccines needs to be reconsidered, taking into account the adverse medical effects that vaccines can have on the human body.** Vaccine science must evaluate not only acute adverse side effects, but also possible associated chronic illnesses such as learning and behavior disorders, Autism Spectrum Disorders, intussusception, arthritis, cancer, diabetes, chronic fatigue syndrome, multiple sclerosis, autoimmune thyroiditis, and other chronic health problems. These chronic illnesses are increasingly costly to society in both human and financial terms.*

*By year's end, the Chronic Illness Research Foundation and its research colleagues will have published four **peer-reviewed papers on the genetic basis of four different chronic diseases: vaccine associated human cancers, Gulf War Syndrome, multiple sclerosis, and AIDS.** The implications of these findings for vaccine safety are:*

- 1. the human body retains a genetic memory of the foreign substances to which it has been exposed, including viral and bacterial vaccines;*
- 2. each individual responds to foreign substances differently, based on his or her own unique genetic background;*
- 3. there appears to be a limit on how much foreign material to which the human body can be exposed before some level of genetic damage occurs and a chronic disease initiates.*

*It is known that our genetic blueprints for life, received from our mother and father, create new genetic material, allowing each individual to cope with toxic environmental exposures. **Research needs to focus more intensely on precisely how the body handles the unprecedented level of gene-damaging substances in our air, water, food and even some medicines.** These substances range from infectious agents, both natural and vaccine-related; pesticides, herbicides, petroleum byproducts and other synthetic chemical hazards; and physical hazards such as radiation. Regarding vaccine safety, I suggest the initiation of serious inquiries into the following research areas:*

1. How do genes change in response to vaccines, and what are the chronic consequences of these changes?
2. What are the acceptable limits of dose, age, timing, and combinations of vaccines that the body can handle? (Not only with respect to their ability to create an immune response to the infectious agent, but also with respect to their acute and chronic health effects).
3. How might we minimize vaccine adverse effects on our genome through life style, diet, and pharmaceutical intervention?
4. How can we repair or minimize the effects of genetic damage?

Today, we are beginning to understand the indirect mechanisms that link toxic exposures and chronic disorders. **Unfortunately, efforts by scientists to explore fully the possible negative effects of vaccines mandated by public policy has been met with stiff resistance by public health agencies.**

Let me give you two examples of vaccine programs that are underway that lack a solid scientific foundation. First, several of my colleagues and I currently have a peer-reviewed paper in a major medical journal due out in September that contains the medical profile of a woman who died from a mysterious case of AIDS. Over several years, her laboratory tests showed a consistent pattern of negative or indeterminate HIV-1 blood antibody tests.

However, when an alternative fluid test was used, she was HIV-1 antibody positive in her urine. **The virus was eventually isolated** from this woman and **sequenced**. This HIV-1 variant came to be known as HIV-1 Group O. Analyses of the viral genetic material suggest that the virus originated, **in part, from genetic reshuffling of human chromosomal material**. HIV-1 could have serious consequences with respect to the initiation of autoimmune diseases. **To put it simply, are we embarking on a course that will vaccinate people against their own genes?**

The second example concerns the intensive effort to create a vaccine for the hepatitis C virus. If you read the literature very carefully, you will find that, while there is a strong marker for the disease, **there is no hard scientific evidence to support the existence of a hepatitis C virus**. Clearly, a non-A, non-B hepatitis disease exists, **but the science behind an associated virus is weak at best. As a scientist I am compelled to ask, how can we vaccinate people against a disease-causing agent that has not been fully characterized?**

Protecting the public against vaccine related chronic diseases is and will be a difficult task. Not only must researchers meet the scientific challenges, but increasingly **they also must battle the politics of science**. Research is showing that our understanding of chronic diseases, as illustrated by my two examples, often is seriously inadequate. Because the issue of vaccine safety involves both policy and science, the public needs to be better represented in the decisions made by public health agencies. In this realm, where science and politics collide, Congress should take a more active role in representing the public interest during the formulation of public health policies.

On the issue of **informed consent**: Had my mother and father known that the poliovirus vaccines of the 1950s were heavily contaminated with more than 26 monkey viruses, including the cancer virus SV40, I can say with certainty that they would not have allowed their children and themselves to take those vaccines. **Both of my parents might not have developed cancers suspected of being vaccine-related, and might even be alive today**. Government, industry, and medicine should embrace the ethical principle of informed consent about possible adverse reactions associated with vaccines.

I appreciate the opportunity to discuss with you my research findings that span a quarter of a century. I will continue to work with my colleagues to unravel the links between toxic exposures and chronic illnesses. While others seek to map the human

genome, our goal is to study the detours the human body's genes must take to survive in an increasingly toxic environment. I ask that the full text of my statement be submitted for inclusion in the record of this hearing.

Thank you.

## **THE LINEGREN STUDY CLAIMS THAT “THE WANG METHOD” CAN BE MODIFIED TO PREDICT HOW HUMAN EXPERIMENTS WILL TURN OUT WITHOUT ANY DATA TO REDUCE MTCT.**

JAMA. 1999 Aug 11;282(6):531-8. Trends in perinatal transmission of HIV/AIDS in the United States.

**Lindgren** ML, Byers RH Jr, Thomas P, Davis SF, Caldwell B, Rogers M, Gwinn M, Ward JW, Fleming PL. Division of HIV/AIDS Prevention: Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention, Centers for Disease Control and Prevention, Atlanta, GA 30333, USA. mll3@cdc.gov Comment in: JAMA. 1999 Aug 11;282(6):577-9.

In the 1999 Lindgren study, the populations monitored were selected **using antibody tests**, and were also selected using a **maximum likelihood procedure**, which predicts when and how things will happen later during an experiment or study, before the results are in.

*The 29 states that conducted surveillance for HIV infection among children including perinatally exposed children, accounted for approximately one third of AIDS cases and one third of births to HIV-infected women in 1994. **These states monitor perinatally exposed children as they are HIV-antibody positive**; update records with HIV tests, AIDS-defining conditions, and vital status; and assess receipt of care. We analyzed data on zidovudine (AZT) use by HIV-**infected** mothers and their infants born in 1993 through 1997 from 14 of the 29 HIV-reporting states that had very complete ascertainment of the number of HIV-infected mothers giving birth, **as estimated** from the Survey of Childbearing Women (SCBW), an anonymous serosurvey **of births (39)** (not of infection or morbidity-real clinical endpoints-emphasis mine).*

*“Reporting delays **were estimated with a maximum likelihood procedure**, which takes into **account the effect of factors such as demographics and HIV exposure groups on delay distributions, as well as the use of “antibody tests” {presumably for mothers}**, and updated records with “HIV” tests.”*

As for the statistical projections in the 1999 Lindgren study, the authors claimed that:

### ***“Incidence Trends by Birth Cohort***

*“To evaluate trends in incidence by birth cohort, **we modeled observed AIDS incidence from AIDS surveillance data reported to the CDC by means of the Wang procedure, a nonparametric method for estimating the birth incidence of HIV-infected children.** Details of the method have been published. **The Wang procedure simultaneously adjusts perinatal AIDS surveillance data for reporting delays and progression time to AIDS for HIV-infected children who have not met the AIDS case definition.** Because national AIDS surveillance does not include HIV-infected children without AIDS, **we modified the Wang procedure by using a parametric estimate of the distribution of progression time to AIDS based on data on HIV-infected children in the Pediatric Spectrum of Disease (PSD) project, a multicenter, active-surveillance project.**”*

**TRANSLATION:** In other words, the cohorts in the Lindgren study were arrived at through a series of statistical and “demographic” (racist) assumptions and mathematical manipulations.

Moreover, **because it is known that markers of a positive test for infants are different than from adolescents or adults, because maternal antibodies persist in infants for 9-14 months on average**, protein-based tests cannot be used on infant subjects to test for assumed “HIV” surrogate markers. If antibody tests are used to diagnose infants, as is indicated by the authors of the Lindegren study, then it should be taken into account that **seroreversion** (loss of what are thought to be “HIV”-specific markers) have been reported in as much as 60% of 14 month old initially seropositive infants. This is even worse than flipping a coin.

These considerations specifically raised issue with the Lindegren study. If 60% of infants serorevertant after initially testing positive for antibodies at birth, and if the Lindegren study used data from “antibody tests” as they claim (ignoring for a moment the flawed design of the study using “the Wang” procedure), does this mean that the transmission of the deadly AIDS virus was blocked by 67% by AZT in the Lindegren study as they reported? It means that at least 60% of the infants counted by Lindegren et al. as seropositive probably seroreverted by 14 months as maternal antibodies became expended. The following studies cited below would argue for this second interpretation, rather than support the claim that AZT blocked transmission by 67%:

Parekh BS, Shaffer N, Coughlin R, et al. Dynamics of maternal IgG antibody decay and HIV-specific antibody synthesis in infants born to seropositive mothers. The NYC Perinatal HIV Transmission Study Group. *AIDS Res Hum Retroviruses*. 9:907-12, 1993; CDC. Current Trends Classification System for Human Immunodeficiency Virus (HIV) Infection in Children Under 13 Years of Age. *Morb Mortal Wkly Rep*. 36:225-30, 235-6, 1987; CDC. 1994 Revised Classification System for Human Immunodeficiency Virus Infection in Children Less Than 13 Years of Age. *Morb Mortal Wkly Re*. 43 (RR-12):1-10, 1994; Chantry CJ, Cooper ER, Pelton SI, Zorilla C, Hillyer GV, Diaz C. Seroreversion in human immunodeficiency virus-exposed but uninfected infants. *Pediatr Infect Dis J*. 14:382-7, 1995.

Finally, it is not clear if the 1999 Lindegren study selected their infant population principally on the basis of statistical projections to identify infants they followed in the study using “The Wang procedure” (Lindegren et al. *Trends in Perinatal Transmission of HIV/AIDS in the United States*. *JAMA*. 282:531-538, 1999), or both. What is clear is that like most studies advocating the use of AZT for the prevention of MTCT, is that the Lindegren et al. studies were terminated prematurely, did not measure clinical endpoints of wellness, sickness, and death, but only measured surrogate markers, or hypothetical statistical projections of surrogate markers.

#### **“UNINFECTED” WOMEN TEND TO TEST POSITIVE:**

In **2000**, it was reported that pregnant women test positive for “HIV” at high frequency [**40**], and the appearance of the so-called specific and unique antigens of an “HIV’s” molecular signature thus shares common molecular epitopes (molecular configurations) with those detected during pregnancy of certain women.

In September, **2000** a study was published in *The Archives of Family Medicine* about false positive and indeterminate HIV test results in pregnant women:

*As the number of women being screened has increased, the proportion of false-positive and ambiguous (indeterminate) test results has increased....”(Archives of Family Medicine, Sep/Oct 2000).*

#### **THE SUCCESS OF GENITAL LESION-CAUSING MICROBICIDES ON AFRICANS.**

Also in **2000**, regarding one microbicide trial funded by The Gates foundation, it was claimed that spermicide doesn’t actually reduce any risk of transmission of “HIV’s” molecular profiles, but actually increased the rate of the appearance of its profiles. This finding ran contrary to what is known about how safe sex practices, including condoms and spermicide (Maggie Fox. Spermicide worsens HIV risk, study finds. *Reuters* 12 July 2000):

South Africa — Researchers hoping to find a way for women to protect themselves from AIDS have said they were dismayed to find that a product they thought may prevent infection actually increased the risk.

The product, a spermicide called nonoxynol-9, did not protect prostitutes in Benin, Ivory Coast, Thailand and South Africa from infection with HIV, a team of U.N.-sponsored researchers said.

We were **dismayed** to find out that the group using the N-9 gel **had a higher rate of HIV infection** than the group using a placebo,” Dr Joseph Perriens, who heads the UNAIDS microbicide effort, told an AIDS conference Wednesday.

They tested nearly 1,000 women and found 59 of those who used the spermicide became infected with HIV, compared to 41 of those who used a dummy gel.

### **THE SUCCESS OF GENE SEQUENCING IN “HIV” RESEARCH: THE RACE TO PATENT HUMAN GENES COULD LEAD TO SHODDY SCIENCE AND PROFITEERING IN AIDS RESEARCH.**

On Tuesday, March 21, **2000**, The Los Angeles Times (Paul Jacobs, Peter G. Gosselin, Times Staff Writers) reported that: *Scientists have uncovered **what they believe are glaring errors in a patent issued last month to Human Genome Sciences Inc.** for a human gene that plays a crucial role in AIDS. Errors Found in Patent for AIDS Gene, Scientists Say; News comes amid concerns that genomics race could lead to shoddy science and profiteering.*

*The potential setback comes amid concerns that the race to patent human genes could lead to shoddy science and profiteering. Indeed, Human Genome Sciences’ shares have fallen along with the rest of the biotech industry’s after a spate of negative news recently.*

*The company’s description of the chemical makeup of the gene contains **at least four significant mistakes**, according to research scientists, an allegation that legal experts say could allow the company’s competitors **to attack** the patent’s validity.*

*But Human Genome Sciences officials say that because the company isolated the gene first, any errors in describing it won’t matter—**it is still entitled to royalties from anyone using the gene to discover new treatments.***

*The scientific challenge to the company’s claim is likely to be one of many in the industry, as the race to be first with genetic breakthroughs inevitably leads to challenges to U.S. patent policy.*

*“This is a perfect example of the rush to sequence [human genes],” said Christopher C. Broder, a former member of a National Institutes of Health team that did pioneering work on the role of the gene in AIDS. “They get it wrong. **They don’t know the function [of the gene].** That is what I have problems with: the whole notion of the rush to patent genes.”*

*Academic scientists such as Broder are particularly angry that companies such as Human Genome Sciences have been able to isolate and analyze genes **by the tens of thousands and have won hundreds of patents for their efforts.***

***When Human Genome Sciences announced last month that it won its patent on the AIDS gene, its stock price soared,** though it has since dropped.*

*Company officials say they are confident the patent issued last month by the U.S. Patent and Trademark Office will withstand legal attack. Any errors in the company’s description of the gene and the protein it produces **are of no consequence**, they contend.*

The description of the gene, also called its sequence, is spelled out in the patent as a series of letters representing the order of the chemical compounds in its DNA. However, the patent also refers to an actual copy of the gene in a living cell that Human Genome Sciences deposited in the American Type Culture Collection in Virginia.

“When we file a patent, we don’t claim the sequence as the invention,” said **William A. Haseltine**, chairman and chief executive of the Rockville, Md., company, which has filed about **7,500** gene patents. “The invention we claim is the gene we deposit with the ATCC. **We know that our sequence and most sequences are not perfect.**

Genomics companies such as Human Genome Sciences and its competitors Celera Genomics and Incyte Pharmaceuticals are able to cast a broad net and haul in genes and gene fragments by the tens of thousands, using automated machines to spell out genetic code and sophisticated software to make **reasonable guesses** about their function.

Haseltine insists that his company is different from its rivals—that it does not rely solely on software **but does experiments** to determine the role played by the genes it seeks to patent.

“You would think that all we did was find a gene and use a computer to find its function,” he said. “We made **a real invention** and we made it first.”

The controversy started quietly enough five years ago, when researchers at Human Genome Sciences isolated a particularly **interesting** gene and quickly determined it was a member of a class that produces protein receptors. These substances sit on the surface of cells like antennas, ready to pick up chemical signals from the body. Haseltine said company scientists showed that the gene was a receptor for **chemokines**, which appear to play a role in inflammatory diseases such as arthritis.

**He acknowledged that the company had no idea that this particular gene and receptor played a role in AIDS.**

In the months after the company filed its patent, however, other researchers at several academic centers, including the Aaron Diamond AIDS Research Center in New York and the NIH, made a remarkable series of discoveries about how HIV, **the virus that causes AIDS**, enters living cells, the first step in an **infection**.

Unaware of the company’s patent application, these scientists found and isolated a protein that the virus requires for entry—**the CCR5 receptor**. And they isolated the gene that carries the instructions for building the receptor. A drug that can block the protein could be a new weapon against AIDS.

Haseltine complains that several of the government-funded groups that made the discoveries have applied for patents of their own. “**The fight going on now is not a disinterested fight about who gets credit for it, but who gets money for it,**” **Haseltine said.**

The academic scientists counter that it is simply unfair to award ownership rights to a gene to a company that had no idea of its function in disease and that did not even spell out the gene and protein sequence correctly in its patent.

Broder, now on the faculty of the **Uniform Services** University in Bethesda, Md., discovered the errors in the Human Genome Sciences patent **the day it was announced.**

Broder said he did a quick comparison of the amino acid building blocks of the CCR5 protein described in the company’s patent and the protein that had been filed in a public database. **He noted that four out of the 352 amino acids in the protein were incorrectly identified in the patent**—each error corresponding to a mistake in **the genetic code**. The protein described

in the patent, he said, would not fold in the same way as the CCR5 receptor identified by academic researchers and **would most likely be useless as a tool for developing new AIDS therapies.**

**Broder pointed out the discrepancy to John P. Moore, whose team at the Aaron Diamond AIDS Research Center helped discover the role of the gene in AIDS.**

Such **fundamental errors** show that the work **should not** earn the company a patent, **Moore said** in a recent interview. **“It’s like patenting an airplane that doesn’t have a tail,” he said. “They know it won’t fly, but they’ll stop everyone who has an airplane with a tail.”**

Moore talked to a former postdoctoral fellow in his lab, Tanya Dragic, who agreed to put the patent to the test. Using **standard** methods, Dragic, now at the Albert Einstein College of Medicine in New York, **plans** to reproduce the gene described in writing in the Human Genome Sciences patent. Then she will insert it into cells and determine if a working receptor protein appears on the cell surfaces and does indeed interact with HIV.

The process should take **a few weeks**, she said.

**20% TO 25% OF PATIENTS WHO DIED HAD PLASMA HIV RNA LEVELS BELOW DETECTION LIMITS WHILE END-ORGAN FAILURES INCREASED, AND WHILE DOCTORS HAD MORE CONTROL OVER HIV REPLICATION AND THE PRESERVATION OF IMMUNE SYSTEMS: LIVER DISEASE WAS CITED AS LEADING CAUSE OF DEATH OF HOSPITALIZED “HIV/AIDS” PATIENTS BY INFECTIOUS DISEASE SOCIETY OF AMERICA.**

In **2001**, the Infectious Diseases Society of America claimed that **“HIV/AIDS” treatment is improving but more patients are dying** who have **undetectable “viral load”** because of *“treatment-related toxicities, and end-organ failure in patients with HIV disease:”*

### **Abstract**

We analyzed the deaths in an outpatient human immunodeficiency virus (HIV) care clinic at University Hospitals in Cleveland from January 1995 through December 1999. The number of annual deaths **decreased** progressively, from 112 in 1995 to 32 in 1999. The median final CD4+ cell count before death increased progressively from 10 cells/L in 1995 to 90 cells/L in 1999 ( $P < .01$ ); **20%25% of patients who died from 1997 through 1999 had plasma HIV RNA levels below detection limits.** From 1995 through 1998, deaths due to infection, to end-stage acquired immune deficiency syndrome, and to malignancies decreased, **whereas the proportion of deaths due to end-organ failures and of uncertain relationship to HIV infection increased.** The spectrum of mortality in HIV disease has changed recently; although **opportunistic infections** cause death less frequently, deaths are occurring **in people who have control of HIV replication** and with **some preservation** of immune function. These observations underscore the need to monitor the etiologies of HIV-associated mortality and to better our understanding of the relationships among immune defenses, **treatment-related toxicities**, and **end-organ failure** in patients with **HIV disease.**

In the journal, Clinical Infectious Diseases (2001;32:492-497), this emerging clinical phenomenon was stated in the following way:

**“End-stage liver disease is now the leading cause of death in our hospitalized HIV-seropositive population.”**

**FDA RECALLS P24 ANTIGEN TEST KIT, AND NUCLISENS IS NOT INTENDED TO BE USED AS A SCREENING OR DIAGNOSTIC TEST:**



February, **2001**. FDA recalls HIV p24 Antigen Test Kit.

### ISIS Report – July 19, 2001-GM AIDS Virus More Deadly

Researchers have been creating one deadly virus after another in the laboratory, and the latest is ‘SHIV’, a hybrid between the human and monkey AIDS virus containing human interleukin genes that **suppress immune response against viruses**. At the same time, GM crops engineered with interleukin genes are being grown in **open field** trials.

Nov. 13, **2001**. NucliSens(R) HIV-test kit package insert was published. <sup>10</sup>*The NucliSens(R) HIV-1 QT assay is not intended to be used as a screening test for HIV-1 nor is it to be used as a diagnostic test to confirm the presence of HIV-1 infection.*

On November 9, 2001, the CDC publishes revised guidelines for “HIV” counseling, testing, and referral:

*When a preliminary, positive rapid test is explained to clients, phrases like “a good chance of being infected” or “very likely infected” can be used to indicate the likelihood of HIV infection and qualified **based on the HIV prevalence in the setting and the client’s individual risk.**” (CDC: “Revised Guidelines for HIV Counseling, Testing, and Referral” November 9, 2001).*

### ISIS Report – July 29, 2001-AIDS-Vaccines Trials Dangerous

*The embattled OECD Conference in Genoa announced a \$1.2 billion package to help combat AIDS in the Third World. Vaccine developers and United Nation agencies are pushing for large-scale clinical trials of AIDS vaccines in vulnerable Third World populations ravaged by the AIDS pandemic. AIDS virologists point to evidence that **the vaccines are not only ineffective but dangerous.** Dr. Mae-Wan Ho reports.*

*“The culprit viral gene”*

*“The intended vaccines all contain gp120, a glycoprotein (protein decorated with side-chains of carbohydrates) belonging to the envelope of the human AIDS virus, HIV-1. The candidates include recombinant HIV proteins and peptides (subunit vaccines), HIV-1 or SIV (the monkey AIDS virus), killed or ‘attenuated’, ie, rendered harmless by successive passage in cultured cells, and a wide range of recombinant viral, bacterial and plasmid vectors expressing HIV proteins. HIV researchers Dr. Veljko Veljkovic and his colleagues in Belgrade Yugoslavia, have shown that the **gp120, is similar to the part of human immunoglobulin (antibody) proteins (Ig) involved in binding foreign antigens, a crucial step in the immune response.** Thus, any AIDS vaccine containing the gp120 glycoprotein or the gene coding for it **could strongly interfere** with the immune system and make the host **more vulnerable** to the virus. And in the longer term, it could accelerate disease progression in HIV patients that do not yet have symptoms. But the gp120 gene has other properties that pose an even greater threat to the vaccinated population. It contains ‘recombination hotspots’ similar to those in bacteria and viruses such as Haemophilus influenzae, Mycobacterium tuberculosis, hepatitis B virus and herpes simplex virus, that often co-infect with the HIV, **and also similar to recombination elements found in immunoglobulin genes and oncogenes (genes associated with cancer) in the human host.** Recombination hotspots are breakpoints at which genetic exchange or recombination occurs much more frequently than usual. Recombination of HIV with bacteria and viruses would generate new pathogens. **Within the human host, recombination with human genes would promote chromosomal rearrangements and formation of abnormal immunoglobulins, thus undermining immune responses. HIV-1 sequences integrated into the genome can act as retrotransposons (jumping genes) that can mutate genes by jumping into them, and some of the mutations may trigger cancer [1].”***

*“Dr. Veljkovic’s team, in collaboration with researchers in UK, Italy and US, already found evidence of recombination between gp120 and a gene from Haemophilus influenzae [2]. Recombination between an HIV gene and Mycoplasma fermentans has been*

implicated in 'Gulf war syndrome' [3] affecting a high proportion of soldiers from United States and the United Kingdom who served in the Gulf war. A new subtype of HIV-1 may also have resulted from recombination between HIV- 1 and SIV [4].”

“The proponents of the AIDS vaccination trials argue that the desperate situation precipitated by the AIDS epidemic justifies acceptance of the ‘small risks’ involved. But Veljkovic and his colleagues have written a monograph documenting **the lack of efficacy of the vaccines and the enormous risks involved** [5].”

“Not effective and dangerous”

“**In 1994, the AIDS Research Advisory Committee of the US National Institutes of Health (NIH) recommended that phase III clinical trials of gp120 vaccines should not be conducted ‘at this time and in this country’.** The reasons, according to Dr. A. Fauci, director of National Institute of Allergy and Infectious Diseases (NIAID), were that the vaccines were ineffective; and there was a remote chance that the vaccines **would compromise the immune system and make the recipient more vulnerable to infection** [6]. The possibility that a vaccinated individual runs a greater risk of developing an established infection, or of progressing to disease more rapidly once infected, **was confirmed subsequently** [7]. The recombinant gp120 subunit vaccine tested in HIV-negative individuals was ineffective in protecting them against infection. **Those who became infected during or after vaccination actually had in their blood sera significant levels of antibodies against the vaccine before they became infected, but those antibodies failed to protect them from infection.** On the contrary, the vaccine appeared to have acted as a decoy to fool the immune system into mounting an attack on it, while allowing the HIV itself to slip through the host defence to get established. **This subunit vaccine is due to go on Phase III clinical trial in Thailand.**”

“The safety concerns for the individual is bad enough. But it is the effect on vulnerable populations that really worry Veljkovic and his colleagues, especially from the live recombinant viral and bacterial vector vaccines (see box).”

### **viral vector vaccines**

*Vaccinia virus*

*Canarypox virus*

*Fowlpox virus*

*Influenza virus*

*Polio virus*

*Venezuelan equine encephalitis virus*

*Rabies virus*

*Adenovirus*

*Hepatitis B virus*

*Herpes simplex*

*Epstein-Barr virus*

*Coxsackie virus*

*Atenuated HIV-1*

*Vesicular stomatis virus*

*Alfaalfa mosaic virus*

### **bacterial vector vaccines**

*Escherishia coli*

*Sallmonela*

*Shigella*

*Mycobacterium bovis BCG*

*Streptococcus gordonii*

*Listeria monocytogenes*

*“Many viral and bacterial pathogens are being used as vectors, and a number are currently considered promising AIDS vaccines. **But they are also promising candidates for generating new infectious agents.**”*

*“The Salmonella vaccine to be trialed in Uganda”*

*“The AIDS vaccine based on live Salmonella vector was developed by the International AIDS Vaccine initiative (IAVI) in partnership with the US- based **Institute of Human Virology (IHV) of the University of Maryland** (Gallo’s group) and the Uganda Ministry of Health. The development of the ‘disarmed’ Salmonella vector expressing HIV-1 gp120 and gp120- derived peptides was started in the early 1990s.”*

*“The Salmonella vector expressing HIV envelope proteins has been tested in **37** people in a phase I trial by NIAID. Uganda will be the first country in Africa to host a clinical trial of this vaccine. The only safety concern, it seems, was to ensure that the vaccine did not induce Salmonella disease (ie, diarrhea) in participants [8]. Whereas, Veljkovic stressed, the right safety question should be: ‘Is the probability for transfer of HIV’s genetic material from recombinant Salmonella vector to other pathogens equal to zero?’ To which the answer is an emphatic no. Salmonella has the same kinds of recombination hotspots (called ‘Chi’) that are present in gp120, and is known to exchange blocks of genes with E. coli and other bacteria. **The potential is rife for generating new pathogens by recombination between the Salmonella vaccine and diverse endemic infectious bacteria in Africa.**”*

*“The Venzuelan equine encephalitis vaccine trialed in South Africa An AIDS vaccine based on the live Venezuelan equine encephalitis (VEE) virus vector, developed jointly by South Africa and the United States, was due for phase I clinical trials early this year, moving on to large trials lasting several more years. The country hoped to make a vaccine against AIDS generally available by 2005. According to a spokesperson of the Medical Research Council of South Africa, a successful vaccine has the potential to save 20 million lives during its first decade of use. The VEE vaccine was developed by the University of North Carolina*

at Chapel Hill with five-year federal funding from NIAID totally more than **\$12 million.**”

“The stated advantages of the VEE vaccine, according to the developers, are that it targets cells in the lymph nodes, and that ‘unlike vaccinia virus, poliovirus, adenovirus, herpesviruses and influenza virus-based vaccine vectors, most of the human population have never been exposed to VEE. Therefore immunisation to HIV with a VEE-based vector would not be restricted by preexisting immunity to the vector itself’ [9]. **Unfortunately, that is not the case.** VEE virus is carried by **arthropods**, and it is endemic in northern South America, Trinidad, Central America, Mexico and Florida; and eight different VEE strains have been associated with human disease. These agents also cause disease in horses, mules, burros and donkeys. Natural infections are acquired by bites from a wide variety of **mosquitoes**. **The same virus was also developed as a biological weapon by the US in the 1950s and 1960s.**”

“A herpes simplex viral vaccine shows promise in non-human primates A modified herpes simplex virus (HSV) that invades host cells and expresses protein from the SIV has been developed by researchers in Harvard University into a live attenuated AIDS vaccine, which show promise in non-human primates. They claim that ‘HSV vectors show great promise for being able to elicit persistent immune responses and to provide durable protection against AIDS’ [10]. The same research team has also developed an HSV-2 vector based on another herpes virus responsible for genital herpes, with the expectation that this vector could serve a double role as vaccine for HIV as well as genital herpes.”

“Unfortunately, the HSV genome contains the greatest number of Chi recombination hotspots of all the microorganisms listed. It also contains Ig class-switch sequences (also recombination hotspots) and other sequences involved in the genetic rearrangements that take place in producing human immunoglobulin genes in blood cells. High levels of recombination have already been identified in the HSV genome associated with these hotspots.”

“A vaccinia virus vaccine led to disease and death among the first AIDS vaccine with live viral vectors which was tested in humans was a recombinant, highly attenuated vaccinia virus expressing HIV-1 proteins. The vaccinia-gp160 vaccine was developed by Bristol-Myers-Squibb who performed the preclinical study in the period 1985-1988. The phase I/II research began in 1988 and was dropped in 1993, then continued for an additional year. These studies combined the vaccinia-p160 vaccine with gp160 or gp120 vaccine developed by MicroGeneSys, Chiron, Genetech, and Immuno AG. **Unfortunately, a recombinant HIV-vaccine virus arose from the attenuated live vaccine, which was harmful for the immune compromised individuals, producing symptoms of progressive vaccinia and death** [11]. There was also the danger that the recombinant virus could spread and harm other persons with AIDS.”

“A canary pox vaccine trialed in Uganda, Haiti, Trinidad and Brazil Another poxvirus was used, the canarypox virus. When the canarypox virus carrying HIV genes infect human cells, the cells make proteins from the genes and package them into HIV-like particles called **pseudovirions that are non-infectious**. These trigger the host immune response against HIV. The first such canarypox viral vaccine carrying the HIV-1 gp160 gene was developed by Pasteur-Merieux-Connought and, in combination with Chiron’s gp120 construct. It entered phase II trial in the US in 1997.”

“The first phase I trial of a canarypox vaccine in Africa was launched early in 1999. It was tested for safety and immunogenicity in Ugandan volunteers, and to reveal the extent to which immunized Ugandans have cytotoxic lymphocytes that are active against the subtypes A and D of HIV, which are prevalent in Uganda. The vaccine was planned to enter phase I/II trials in Haiti, Trinidad, and Brazil during 2000. Is canarypox virus safer than vaccinia virus? Most probably not. Both are orthopox viruses and are rich in recombination hotspots. This family of viruses is widely distributed, and recombination between different poxviruses can readily take place. **Recombinants have arisen that are more virulent than either parent, and it is impossible to predict the fate of released canarypox vaccine with HIV genes.** The use of these vaccines in Africa where monkeypox is endemic is likely to generate recombinants with unpredictable pathogenicities. Monkey pox is transmitted from human to human,

but the natural virus is relatively harmless. Could a recombinant virus arise that may be as virulent as the smallpox virus?"

"AIDS vaccines in plants could generate recombinant viruses that switch hosts from plant to animal"

"Finally, AIDS vaccines based on HIV antigens produced in plants are also being developed. The tobacco mosaic virus, TMV, has been used as a vector to express recombinant coat protein of alfalfa mosaic virus (AIMV) containing antigenic peptides from the rabies virus and HIV-gp120 [12]."

"Vaccine trials in breach of UNAIDS ethical, scientific and safety standards"

"According to the WHO report 2000, more than 90% of all AIDS cases are in developing countries. UNAIDS and NIH are the two most important organizations involved in developing AIDS vaccines. UNAIDS Executive Director Peter Piot has declared, 'It is our collective responsibility to ensure that all vaccine trials are conducted under the strictest possible ethical and scientific standards.' But Dr. Veljkovic has shown that current vaccines based on HIV-1 gp120 can harm the immune system of individuals and, on account of its recombinogenic tendencies, has the potential to generate deadly viruses and bacteria that can spread through the vaccinated populations and to wild life. **The intended vaccine trials are in serious breach of ethical, scientific and safety standards.**"

"AIDS, more so than other diseases, cannot be addressed simply by vaccinations, even if efficacious and safe vaccines could be found. More than drugs and vaccines, we need to end poverty, malnutrition and environmental destruction, to reinstate social equity and free access to primary healthcare and education."

"A UN body to monitor and control GM experiments Dr. Veljkovic is calling for the formation of an 'organization which could pick up information concerning all laboratories performing GM experiments, like [the] International Atomic Agency which control all nuclear experiments and activities around the world'. That should be the task of the International Biosafety Clearing House."

See Superviruses and Superbugs from AIDS vaccines, ISIS News 9/10 ISSN1474-1547(print), ISSN1474-1814 (online).

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## **MANY CASES OF NO “HIV” TRANSMISSION AMONG SERODISCORDANT COUPLES, AND OTHER TEST KIT RECALLS.**

January 17, **2002** the Journal of Infectious Disease featured an article about 17 women who remained “HIV” uninfected, despite a history of heavy exposure to HIV through repeated, unprotected sexual contact with an infected partner, and 12 of their regular, male HIV-positive partners.

Also in **2002**, the FDA recalled BioRad Genetic systems HIV Types 1 &2 Synthetic Peptide containing test kits.

Also in **2002**, it was reported in The Journal of Virology, that saquinovir and other protease inhibitors are severely toxic to T-cells in the absence of “HIV” infection [41].

## **ISIS Report – 1 May 2002-Doubts Deepen over Safety of AIDS Vaccines**

**“Another key AIDS vaccine is abandoned before phase III trial.** This latest setback comes at the end of a string of failures in developing vaccines that may be worse than useless.” Dr. Mae-Wan Ho reports.

*“The US government abandoned a controversial AIDS vaccine trial and announced it will combine the work of two federal institutions, the National Institutes of Health and the Department of Defence. Both institutions had proposed trials to test a combination of similar vaccines – a dose of canarypox virus engineered to carry HIV-1 proteins with a booster shot of the HIV protein gp120. The trial was designed to compare the types of immune responses the vaccine evoked with the protection it provided. **That required the vaccine to produce an immune response in at least 30 per cent of volunteers. But analysis of the data suggests the response did not come up to scratch. “It didn’t even come very close,” said Anthony Fauci, director of the NIH’s National Institute of Allergy and Infectious Diseases. The cancelled NIH trial, which would have involved 11,000 volunteers, was anticipated to cost \$60 to \$80 million dollars. The Department of Defence trial, which was designed to test only the efficacy of the vaccine, will still go ahead. But that may be a grave mistake.**”*

“The canary pox vaccine was Aventis Pasteur’s ALVAC-HIV (vDP1452), and the booster, VaxGen’s AIDSVAX B/B. **The ALVAC vector failed to provoke a strong immune reaction**, and new evidence from Harriet Robinson’s team at Emory University, announced at the 9th Conference on Retroviruses and Opportunistic Infections (Seattle, 24-28 February 2002), demonstrated that adding a gp120 booster to another vaccine **actually reduced the vaccine’s efficacy rather than improving it**. These **disappointing results were to be expected**, according to AIDS virologists, among whom, Dr. Veljko Veljkovic, who have been studying the problems of AIDS vaccines for years.”

“More bad news comes from vaccine trials in non-human primates.”

“Vaccines that induce only cellular immunity through cytotoxic lymphocytes (CTL) – immune cells that destroy cells infected with virus – without circulating antibodies in the plasma gave only partial protection. In a study carried out by the Merck Research Laboratories in Pennsylvania, the Center for Aids Research in Duke University Medical Center and Division of Viral Pathogenesis in Harvard Medical School, **two out of 15 immunised macaque monkeys became ill with AIDS-related symptoms six months after being challenged with the pathogenic HIV-SIV hybrid virus (SHIV)**.”

“The best vaccine based on an adenovirus vector “greatly attenuated” viral infection, but did not prevent it. Despite that, the authors claim that the vector was “promising” for development of an HIV-1 vaccine. Though they admitted that **the relevance of their model system to human HIV-1 infection is not firmly established, and “cannot be extrapolated” to predict what would happen in human beings**. This assessment must be done in clinical trials, they stated. The SHIV hybrid virus, routinely used in such studies, is an especially virulent form of the AIDS virus that kill victims in weeks, and its safety has been strongly questioned.”

“Another study by researchers based in Harvard Medical School, Northwestern University of Chicago, Duke University Medical Center and the Southern Research Institute in Maryland is less optimistic. **One out of eight immunised rhesus monkeys died as the challenge virus mutated and escaped from the CTL**. Such **mutations** in the virus that escape immune recognition have been described in more than a dozen reports in the literature in trials involving both humans and non-human primates. The authors conclude that such viral escape from the immune system “may be a major limitation of the CTL-based AIDS vaccines that are likely to be administered to large human populations over the next several years.”

“Some **virologists** have been warning for years that the entire class of AIDS vaccines based on the HIV gp120 gene or protein **is not only ineffective, but also dangerous** for the recipients and the human populations. **There is evidence suggesting that gp120 can interfere with and undermine the immune system** and can readily recombine with viruses and bacteria (used as vectors) to generate new pathogens.”

“The envelope glycoprotein, gp120 of HIV-1, **is similar to the region of human immunoglobulins** that binds antigen, a crucial feature of the immune response. Thus, any AIDS vaccine containing the gp120 could interfere with the immune system and make people more vulnerable to the virus. And in the long term, **it could accelerate disease progression in HIV patients that do not yet have symptoms**.”

“Recombinant viruses expressing gp120 could also be a source of potential new pathogens. The gp120 gene contains genetic elements that stimulate recombination or are ‘recombination hotspots’. These elements are similar to certain ‘Chi’ (pronounced ‘kye’) sequences found in bacteria and viruses such as Haemophilus influenzae, Mycobacterium tuberculosis, hepatitis B virus and herpes simplex virus that often co-infect with the HIV, and are also similar to immunoglobulin recombination elements in the human host. Recombination of HIV with bacteria and viruses mediated by Chi sequences would generate new pathogens, and such recombinants **have been found**.”

*“Within the human host, recombination with human genes would promote chromosomal rearrangements and formation of aberrant immunoglobulins, **leading to inadequate immune responses**. Furthermore, HIV-1 sequences integrated into the genome **have the potential to initiate a wide variety of diverse genetic effects caused by all mobile genetic elements, especially mutations of genes due to random insertion, some of which might trigger cancer.**”*

*A recent **theoretical** study adds further fuel to their warning. Partially effective vaccines that inhibit the growth of the pathogen, such as the AIDS vaccines described here **may leave death rates unchanged, or worse, increase deaths with the level of vaccination** (see “Health warning over partially effective vaccines”, this issue). But a company in Texas, Prodigene, is putting gp120 into GM maize as a cheap, edible oral vaccine against HIV as announced in the internet journal, AIDScience. This will surely lead to widespread contamination of our food crops with disastrous consequences. Not only is this extremely hazardous for human beings. It will affect all organisms in the food chain and multiply the opportunities for this gene to recombine with bacteria and viruses in the environment, of which 99% cannot be cultured and are hence completely unknown. An edited version of our response was published in the internet journal ([www.aidsscience.com](http://www.aidsscience.com)).*

**DESPITE SOMETIMES BEING ABLE TO REDUCE HIV RNA IN THE BLOOD TO UNDETECTABLE LEVELS, PERIPHERAL CD4+ COUNTS IN 25% OF PATIENTS DO NOT CORRELATE WITH VIRAL LOAD AND, LYMPH NODE FIBROSIS PRIOR TO HAART INITIATION IS PERHAPS A BETTER INDICATOR OF A PATIENT’S ABILITY TO RECOVER PERIPHERAL CD4+ CELLS FOLLOWING HAART.**

**2003** (January) Lymph node fibrosis impedes peripheral [CD4.sup.+] T-cell count; fibrosis could be better predictor of ability to recover after HAART. (*The Scientist* | January 27, 2003 | Roberts, Josh P).

*Because HIV preferentially targets [CD4.sup.+] T cells, their numbers, along with other metrics like HIV RNA levels, traditionally are used to indicate **the infection’s** severity. Moreover, clinicians use these numbers to predict the efficacy of future immunological reconstitution treatment in first-time patients undergoing antiretroviral therapy.*

*But a recent finding shows that **highly active antiretroviral therapy (HAART) failed to markedly increase the peripheral [CD4.sup.+] count in 25% of patients, despite sometimes being able to reduce HIV RNA in the blood to undetectable levels**. This finding has brought into question the utility of these factors as recovery predictors. (1)*

*“There **must be** factors **beyond** suppression of viral replication,” says Timothy Schacker, associate professor of medicine, University of Minnesota.*

*Schacker and his colleagues contend that the amount of **lymph node fibrosis prior to HAART initiation** is perhaps a better indicator of a patient’s ability to recover peripheral [CD4.sup.+] T cells following HAART. (1) The damage to the lymph nodes, where nearly all HIV replication takes place in the activated [CD4.sup.+] cells that reside there, has already occurred before therapy has even started.*

*An ever-expanding body of work—much of it undertaken by the once-lone voice of the National Institutes of Health’s Steven Shaw (2,3)—is **showing the importance of lymph node architecture in providing a suitable microenvironment for the immune processes**. Here, T cells interact with B cells, antigen-presenting cells, stroma, and each other, as well as receive soluble messages through cytokines and other growth factors. **So, if the structure is compromised, the lymph nodes’ ability to support a viable immune system may be severely compromised as well.***

*PERPETUAL INFLAMMATION Schacker and colleagues examined the T-cell zones of lymph nodes from treatment-naïve patients at various stages of HIV infection, from presymptomatic to full-blown AIDS. **The number of [CD4.sup.+] T cells found there***



**did not correlate with either peripheral [CD4.sup.+] cells or with detectable amounts of viral RNA in the plasma. But the nodes had considerably more collagen deposition than HIV-negative controls.** The collagen showed an inverse relationship to the nodal [CD4.sup.+] T-cell population; the number of [CD4.sup.+] cells decreased as the amount of fibrosis increased. Similarly, the potential for immunological reconstitution as measured by the peripheral [CD4.sup.+] T-cell count **after** therapy showed an inverse relationship with the amount of nodal collagen deposition.

The investigators speculate that lymph nodes are likely damaged because of perpetual inflammation. “In the long struggle between immune defenses and HIV-1 that partially controls replication, the immune system is maintained in a state of chronic activation,” they write.

The model is not unprecedented. **“The situation is analogous to what happens to the liver in a chronic hepatitis infection,”** says ...

## **Mye-Wan HO ISISAIDS Vaccines Worse Than Useless?**

**17th June 2003**

The US administration is offering **AIDS-ravaged nations** support for fighting AIDS tied to the purchase of GM products. The main anti-AIDS strategy is a class of vaccines that carries its own risks. **Prominent AIDS researchers have called repeatedly for a moratorium as evidence of hazards accumulates.** Dr. Mae-Wan Ho reports.

“George Bush has taken Europe to the World Trade Organisation over Europe’s de facto moratorium on GM imports. In the week of the G8 summit in Evian, France, Bush blasted Europe for perpetuating starvation in Africa by blocking US food aid with anti-GM policies, and announced his pledge of \$15bn to combat AIDS globally, especially in Africa.”

“The UN Population Division reported earlier this year that by 2050, the population of the hardest hit nations will have risen by 400 million less than previously estimated because of AIDS. **“This estimate could be the first sign that HIV-1 will cause extinction of human beings in this millennium unless an effective AIDS vaccine is developed,”** said a commentary by Veljko Veljkovic and colleagues in the *Lancet*, published in February.”

“The only AIDS vaccine to have progressed past phase 3 trial, made by VaxGen, took 5 years and involved 5108 gay men and 309 women. Unfortunately, it proved ineffective, and may even be harmful.”

“In the 3003 white and Hispanic volunteers who received VaxGen’s vaccine, **a higher proportion suffered breakthrough infections than in the 1508 controls: 6% vs 5%.** Although the difference is not significant, it could indicate a dangerous trend. **But the company is not releasing further details on the trial results.**”

“A few days after Bush announced the AIDS package, US Congress was denounced for tying support for anti-AIDS research programmes in 50 countries to their acceptance of GM products. This accusation came from Julio Sanchez, representative of Mesoamerican Trade. Introducing GM food to hungry, malnourished nations ravaged by AIDS is bad enough in terms of health risks, but AIDS research programmes are heavily concentrated towards vaccine development with a strategy that introduces its own health hazards, as is becoming increasingly clear. During the past decade, a number of AIDS researchers, among whom Veljkovic and his team in Yugoslavia, have been studying the properties of the human immune deficiency virus, HIV-1, especially its envelop glycoprotein, gp120, which features in most of the AIDS candidate vaccines.”

“The gp120 protein is strongly immunogenic, which is why it is widely used in vaccines, in the hope that the body will produce

antibodies against the protein and hence protect against the virus. But there have been many worrying signs that this may have just the opposite effect.

*“For although the body mounts a strong immune reaction against the protein, and produces antibodies against it, those antibodies fail to protect against the virus. **One main reason is that the virus is very mutable, and can readily mutate to escape immune detection.** In addition, the immune reaction mounted against the original gp120 undermines the effectiveness of the immune system by over-stimulating it, **so that it is less effective to cope with new infections.**”*

*“A recombinant gp120 vaccine tested in HIV-negative individuals in phase I/II trials, was not effective in protecting against the disease. Not only that, **participants in the trials had significant levels of circulating antibodies against the vaccine before they became infected, and came down with AIDS disease.**”*

*“The vaccine could also be dangerous. A vaccine based on the gp120 from the strain SF2, actually suppressed the production of antibodies that could neutralise the later infecting virus, while boosting the production of useless antibodies that were specific for the vaccine strain, SF2. In other words, gp120 acts as a molecular decoy to disarm the body’s antiviral response, leaving it more vulnerable, and increasing the likelihood of rapid disease progression in those vaccinated that later became infected. This phenomenon is called “deceptive imprinting” of the immune system.”*

*“Were those effects predictable in advance of the clinical trials? Veljkovic and his colleagues answer a definite yes. “*

*“First of all, the part of the gp120 molecule that plays the dominant role in provoking an immune response is the V3 loop. The V3 loop and flanking regions are similar in base sequence and structure to the antigen-binding region of the human immunoglobulin (Ig) (antibody protein). And it has been proposed since the early 1990s that this immunoglobulin-like domain in gp120 may interfere with the immune regulatory network. This is strongly supported by later observations that the anti-V3 and anti-Ig antibodies of healthy individuals are similar in structure, and that antibodies reacting to V3 are present in sera that are HIV-negative.”*

*“In 1999, Howard Urnovitz and colleagues identified a mysterious case of AIDS in a French woman with no risk factors. Analysis of the isolated HIV viral envelope showed that **it had homology to sequences found on at least 14 different human chromosomes.** This opened a whole new can of worms. Was this rare strain of HIV-1 the result of genetic recombination (reshuffling) in the human genome, similar to that found in veterans suffering from Gulf War syndrome (see “Dynamic genomics”, this series)? Antibodies to human **endogenous** retroviruses were found in the urine of patients with clinical AIDS. Thus, vaccinating against HIV-1 may be tantamount to vaccinating people against their own genes (see “Endogenous retroviruses & chronic disease”, this series). Does that mean genetic reshuffling and retroviral elements in the human genome may have a key role to play in AIDS disease, as in Gulf War Syndrome and other chronic disease?”*

*“Another piece of evidence implicating genetic recombination is that the V3 loop and its flanking regions are located between recombination signals similar to those found in human immunoglobulins, and also similar to the Chi recombination hotspots found in many viruses and bacteria. Consequently, the immunologically dominant region of gp120 may be involved in recombining with human immunoglobulin genes resulting in autoimmune responses, and may also recombine with co-infecting viruses and bacteria to generate new pathogens. Evidence of such recombination has subsequently been found in the sera of AIDS patients.”*

*“Many other observations have linked gp120 with auto-antibodies that react against the body’s own cells and enhance the infectivity of HIV-1, and those researchers have also issued warnings against AIDS vaccines.”*

*“In fact, warnings against AIDS vaccines go back to Albert Sabin, one of the most prominent viral vaccine developers of the 20th*

century. **“The available data provide no basis for testing any HIV vaccine in human beings either before or after infection,” Sabin stated.**”

*“The current issue of Vaccine carries an article evaluating the long-term safety of a range of AIDS vaccines involving 3189 HIV uninfected, healthy volunteers who were enrolled into 51 NIAID (NIH) – sponsored Phase I and II clinical trials. It concluded that there were no adverse effects. Veljkovic remarks, “This conclusion was based on analysis of many important parameters ....Unfortunately, the key information – comparison of the health status between breakthrough infected vaccinated volunteers and control subjects who participated in these trials – was not reported, just as it was not reported by VaxGen in the results of their Phase III clinical trial.”*

**“Unless this information is reported, says Veljkovic, the companies and institutions that organized these clinical trials are in danger of committing a scientific and ethical misconduct.”**

*“It is pertinent to point out that transgenic DNA in GM food and feed also carry recombination hotspots, such as the ones associated with the CaMV 35S promoter and the left and right borders of the Agrobacterium T-DNA used as vector to introduce transgenic DNA into the plant genome. These recombination hotspots enhance horizontal gene transfer and recombination. Furthermore, as Veljkovic said, the recombination hotspots in transgenic DNA may interact with the recombination signals flanking the V3 loop of the gp120 gene in AIDS vaccines to generate yet more exotic viruses.”*

*“Veljkovic and his colleagues have repeated their call for **an immediate moratorium** on the current clinical trials of HIV-1 gp120/160 vaccines.”*

In December, **2003**, At Kenyatta National Hospital [Kenya] ...out of 31 couples tested, 23 were discordant [one positive, one negative]. It was reported that some of them stayed in a sexual relationship with the infected partner **for more than six years** without the infected one passing the virus to the other. And when these discordant couples brought their children for testing, **all of them were free of the virus...**” (Horizon Magazine, December 18, 2003).

Also in December 19, **2003**, COBAS AmpliScreen HIV-1 Test package insert, version 1.5 was published (<http://www.fda.gov/cber/label/hiv1roc121903LB.pdf>): **“This test is not intended for use as an aid in diagnosis.”**

#### **“HIV” INJECTED CHIMPS ARE GIVEN RETIREMENT HOMES:**

In the New York Times story appears entitled, “For Retired Chimps, a Life of Leisure,” by Stolberg, describing how it has been about 22 years since chimps were injected with both sera and “isolates” of “HIV” obtained from AIDS patients, but have yet to become ill, as they grow old in their new 27 million dollar retirement homes.

In **2004**, the FDA **recalls** bioMerieux NucliSens automated isolation reagent(s), DiaSorin’s HIV-1 – HIV-2 Plus O EIA Testing Software, and Roche’s Amplicor HIV Monitor test.

In 2004, it was reported in the New England Journal of Medicine that **vitamin supplements** can ward off progression to AIDS in the absence of HAART (Highly Active Anti-Retroviral Therapy) [42].

#### **THE SUCCESS OF THE GP120 AIDS VACCINE.**

Also in **2004**, the failure of AIDS VAX, the 120 million dollar effort to vaccinate against “HIV” was announced in the journal Science [43]. Shortly thereafter, it is announced that VAXGEN, Donald Francis’s company that performed the failed “HIV” trial, would

receive more than 800 million from the military budget to make a new anthrax vaccine.

### **RED CROSS SAYS THERE ARE 2 OR 12 FALSE POSITIVES OUT OF 37 MILLION NEGATIVES:**

In **2004**, the Red Cross also reported that even after repeated testing using different test kits, “low-risk” populations, such as blood donors (or military recruits) will typically yield **12 (PCR) positive or 2 (ELISA) positive results out of 37,000,000 samples**, leaving potentially 10 out of 12 false positives [44]. In follow-up, it was noted that 6 of the 12 PCR-positive subjects tests seroconverted within several months, thereby obtaining a consistent “HIV” molecular profile in 8/12 cases, out of 37 million negatives.

### **EDMOND TREMONT LIKES TO REWRITE SAFETY REPORTS FOR GEORGE W. BUSH, AND FIRE WHISTLEBLOWER SAFETY OFFICERS TO PROMOTE PEPFAR:**

That same year (**2004**) it was announced that the government’s chief of AIDS research, Dr. Edmond Tremont, rewrote a safety report on a U.S.-funded drug study of nevirapine to change its conclusions and delete negative information, and later, ordered the research resumed over the objections of his staff, so that George W. Bush’s \$500 million dollar plan to distribute nevirapine to African women would proceed, even though the drug’s approval was withdrawn in the U.S. because of excessive toxicity, its association with liver failure, and deaths [45]. The Institute of Medicine later pardoned Tremont’s changing of the safety data, while Johnathan Fishbein, the NIH safety officer who blew the whistle was subsequently fired from his position as safety officer for the Nevirapine trials that Tremont, his boss, rewrote [46].

### **DENTISTS DO NOT GET OCCUPATIONAL AIDS: AN OPEN LETTER TO THE PROFESSION AND AN EVIDENCE-BASED STUDY ON THE AIDS EPIDEMIC IN DENTISTRY:**

By E. J. Neiburger DDS, Director -Center for Dental AIDS Research. This article was published in the January ’04 issue of the Journal of the American Association of Forensic Dentists, vol. 26, no. 1-3, 2004

*The greatest impact that dentistry has experienced in the last decades of the 20th century has been concerns about infection control.*

*This was primarily due to fears about the occupational transmission of HIV/AIDS.*

*Billions of dollars and millions of person-hours were devoted to this issue because of extrapolations of approximately 100 medical (not dental) HIV transmission cases reported world wide.(1)*

*As a result, thousands of allergic emergencies and some deaths (e.g.latex anaphylaxis) have resulted from staff and patient exposures to protective devices recommended for the prevention of transmission of this single terrifying disease.*

*With all the panic and publicity surrounding the great FAIDS (fear of AIDS) epidemic of the late 1980’s and early 1990’s one critical fact is often missed.*

*There are (and never have been) any documented cases of dental workers getting occupational HIV/AIDS. (1, 2). Our profession has spent billions of dollars and person-hours on questionable disposables, research, training, legislation, regulation and litigation in an effort to prevent a disease that has never occurred occupationally in dental workers.*

*There are, however, a reported seven “possible” non-documented cases of occupationally acquired dental HIV/AIDS which are*

continuously referenced as the only “solid” evidence that HIV/AIDS is a serious concern for dentistry.

*This paper will examine the scientific aspects of these cases and how “soft” this “solid” evidence really is .(1)*

### **PANIC—THE DR. ACER CASE**

*The one issue that threw the nation into a panic and damned dentistry in the mind of the public was the Dr. David Acer case where an AIDS infected Florida dentist ( using recommended Universal Precautions) was alleged to have transmitted the virus to 5 (later 6) of his dental patients. (3-5).*

*The Centers for Disease Control (CDC), a division of the U.S. Public Health Service under the Secretary of the U.S. Department of Health and Human Services, mishandled the scientific, statistical and media aspects of this case causing wide spread confusion. (4,5).*

*The “infected patients”, were finally identified with high risk behaviors and in a following governmental investigation, the U.S. General Accounting Office (GAO) reported:*

*“...CDC could not identify, on the basis of its investigation, exactly how HIV was transmitted to the 5 patients.” “...this case provides little specific information to advance an understanding of how to prevent such occurrences in the future.”*

*Litigation, big-buck settlements, unremitting media publicity and panic muddied the issue and established the public’s perception (as well as many in the profession) that dental care could easily transmit HIV/AIDS.(1-5)*

*Serious questions were asked about the conclusions the CDC made in this case but they fell on deaf ears.(3-6)*

*The GAO and other agencies recommended that the Acer case be considered an anomaly and not be used for policy decisions. Unfortunately the “horse was out of the barn” and the Acer case became the symbol of AIDS dangers; not the exception that it really was.*

*As time went on, the public and dental media expanded the concept that “AIDS is everywhere”.*

*Numerous gay rights and AIDS organizations, in an effort to avoid the stigma and discrimination surrounding AIDS being a “gay only disease,” fostered, with the help of the government and a few dental groups, the faulty concept that AIDS could affect everyone equally; heterosexuals and homosexuals alike.*

*As the FRAIDS panic spread, bizarre predictions appeared such as with TV host Oprah Winfrey’s 2-17-87 “Women living with AIDS” show where Oprah stated “by 1990, twenty percent of heterosexuals will be dead of AIDS”.*

*A 1991 Gallup Poll reported that Americans (and their political representatives) believed that AIDS (which killed approximately 25,000 that year) was eight times more important than cancer (which killed 900,000+ people in 1991).*

*This alarmist climate resulted in heavy pressure on the dental profession to show that the public was “safe” in the dental office and numerous laws, regulations and procedures were enacted to give this appearance.(1-7)*

*Many dental journals and supply manufacturers saw a boom in disposables advertising and sales. Self proclaimed “experts” and infection control organizations proliferated, generating millions of dollars in educational schemes.*

*Dental offices were awash in latex, wrappers and sterilants.*

*The U.S. Surgeon General, C. Everitt Koop publicly stated, "Getting AIDS from a Health Care worker is essentially nil." Using a few occupational seroconversions among the world's non-dental health care workers as a rationale, the CDC supported draconian governmental regulatory measures which gave an opposite message.(1-5)*

*The Surgeon general's advice was ignored by the media and public.(7)*

*Gradually the panic diffused and dissipated as FRAIDS fatigue and clearer minds prevailed. The constant media attention became old and boring. The public saw that, in spite of the doomsayers and activists' predictions, very few people were going to die of AIDS; especially middle class, heterosexuals.*

*AIDS was not a disease of average Americans.(3-7)*

*Serious questions about the Acer case, the effectiveness of Universal Precautions, the CDC's accuracy, rampant fraud/waste in many AIDS organizations and the obvious miniscule dangers of AIDS transmission caused many exhausted people to calm down and take a second look at the situation(3-5).*

*In the 1990's annual AIDS case numbers began to significantly fall.(1,5,8)*

*AIDS was clearly identified as a preventable and treatable, chronic disease predominately affecting homosexuals, IV drug users and their sex partners.*

*New medications made AIDS a "tolerable" disease, cleared out hospital wards and allowed many of the infected, who otherwise would have quickly died, to live relatively comfortable, productive lives.*

*The epidemic was over and dentistry, with the exception of the Acer case, had not been implicated.*

### ***POLITICS AND MONEY WARP SCIENCE***

*The Atlanta based CDC is the nation's main broker for AIDS epidemiology data and related health information.*

*It is, by its nature and history, a politically involved government organization.(4-6,9)*

*The CDC made serious errors in the analysis of the Acer case (4,5,10,11).*

*The organization routinely "amends" their statistics on HIV/AIDS and in some cases, exaggerates the dangers.(11,12)*

*For example, the CDC, in its main publication, Morbidity and Mortality Weekly Report (MMWR), published the total of AIDS cases for 1995 as 68,367 (MMWR (1-12-96 p.23), then published 71,547 ( MMWR 8-20-96 p.749) and 71,210 (MMWR 1-1-97 p.1138)...all three sets of data for the same year (1995).*

*The CDC treats AIDS as its golden child. No other disease has its cumulative, multi-decade case totals routinely published nor has the "data tortured" classification of the "25 to 44 year old group" which was selected to show the worse statistical expression of the AIDS epidemic.(13) It is not used for any other human disease category.*

*This lacks scientific reliability.*

*In 1996, the CDC was taken to task in Congressional hearings accusing the organization of exaggerating the risks of AIDS and inflating case numbers in order to increase funding.(4,/a>9)*

*In one exchange, the U.S. Department of Health and Human Services Director, Secretary Shalala, was asked by a Congressional investigator (Mr. Istook),*

*“But I still don’t understand why you were telling this committee about an increase in AIDS and trying to dramatize increases when actually the reports from the CDC show fewer cases and that the increase you talk about is due to a change in definition.”(14)*

*The Secretary responded by stating, “I deny my testimony was inaccurate”.(14)*

*Incidentally, it was Secretary Shalala who in a news conference in 1984, announced: the discovery of the AIDS virus by NIH sponsored Dr. Gallo, that HIV was the sole cause of AIDS and a vaccine would be ready by 1986.*

*None of these statements proved true.*

*The CDC has often been involved in shady situations involving money and scandal.*

*The famous head of the CDC, Surgeon General C. Everett Koop, invented Universal Precautions (recommending glove, mask and eye ware for health care workers during all patient contacts).(5)*

*It was based on the Hadler Hepatitis B infection report (a case about an oral surgeon who transmitted Hepatitis B to patients) which was later found to be scientifically flawed (incorrect HBV incubation periods were used). (15)*

*In late 1999, Dr. Koop was exposed in what was reported as a million dollar “financial arrangement” with a latex glove maker (WRP Corp), the attempted suppression of government action responding to the erupting latex allergy epidemic and a failing web site (Dr. Koop Life Care Corp.) which sold stock to the public.(16,17)*

*Recent CDC scandals over misuse of funding (18), the unexpected resignation of its director (19), the retraction of its recommendation for an anti-AIDS cream, nonoxynol-9, (it increased the AIDS transmission rate, not reduced it (20), the feeble attempt to boost AIDS case numbers with a new AIDS designation (AIDS-Opportunistic Illnesses) (21) and the latest Surgeon General’s condemnation (after the 9-11 and anthrax attacks) that the “Atlanta labs are a national disgrace” (22), placed a cloud over the integrity of the policies and scientific methodology used at the CDC.*

*In an effort to reduce criticism in an often no-win situation, the CDC began a program that exerted great efforts to avoid embarrassing questions and admissions.*

*One way of doing this was to use “unpublished data” to substantiate “scientific conclusions/recommendations and when questioned, to refuse researchers requests to examine the non-referenced data by claiming coverage under the Public Health Service Act. Section 301(d) of the Act allows the organization to avoid releasing data under the guise of protecting individuals’ privacy.(23)*

*It is important for health care providers to carefully examine the scientific basis of governmental mandates and recommendations and not blindly follow edicts that may be more politically than scientifically inspired.*

**THE MANY DEFINITIONS OF AIDS: CONFUSION**

*The AIDS of 1984 is different from the AIDS of 2004. It differs by definition which has changed numerous times.*

*AIDS is truly a “political” disease.*

*The definition of AIDS differs from country to country.*

*In the U.S. there were major changes in the definition in 1987, 1992, 1993 and 2000. Each of these changes resulted in the inclusion of increasingly more ill individuals to the point that AIDS is really a collection of 25+ immunodeficient diseases.(5)*

*The 1993 definition change caused an almost doubling of yearly AIDS case numbers in one week.*

*After a year or so the case numbers came crashing down and in a fit of spin doctoring, the CDC refers to the episode as a “temporary distortion”. (24)*

*In 1987 the CDC defined AIDS as:*

*“Human Immunodeficiency Virus (HIV), the virus that causes acquired immunodeficiency syndrome (AIDS), is transmitted through...”(25)*

*By 1998 the CDC changed its definition:*

*“Acquired immunodeficiency syndrome (AIDS) is a group of diseases or conditions which are indicative of severe immunosuppression related to infection with the Human Immunodeficiency Virus (HIV).”(26)*

*These definitions all related to serologic HIV testing.*

*A different set of classifications were reserved by the World Health Organization (WHO) for third world countries without the means to do accurate lab HIV testing.*

*In 1992, WHO devised a definition of AIDS involving a combination of major (weight loss, diarrhea, fever, etc.) and minor signs (cough, dermatitis, herpes zoster, etc.).*

*If you had two major and one minor sign, you had “AIDS”.(27)*

*Unfortunately these signs are also present in TB, malaria, cancer, malnutrition, parasite infestation and a whole host of other “natural” background diseases that occur in many of the poor folk in third world countries.(28)*

*You do not have to be HIV positive to have “AIDS”.(5,29)*

*Since AIDS receives more funding than the above diseases, there is a strong financial pressure for impoverished health departments to diagnose more cases of “AIDS”.*

*Thus we are faced with the CDC and WHO, political organizations with an unimpressive record of counting statistics and some serious deficiencies in the analysis and interpretation of AIDS data. It is unfortunate, but this is the best epidemiology we have today. We must be very careful in what data we accept as accurate and factual.*

**PEOPLE LIE: AIDS RESEARCH IS OFTEN BASED ON BAD DATA**



*Much of AIDS epidemiology is unreliable.*

*It depends on patient interviews where carefully positioned questions attempt to get truthful responses.*

*Most AIDS data relies on the accuracy and truthfulness of those interviewed.(2-5)*

*Unfortunately, people lie. They especially lie about their sex-lives (5,30-35 ) and illegal activities (e.g. IV drug use)(36,37). Some even lie so that they can get to participate in vaccine trials.(38)*

*Numerous studies have shown that that people initially lie but often recant upon pressure.(30,31,33,35,37)*

*Some people do not.*

*A number of studies illustrate these phenomena. Castro et al. found that 75% of HIV positive individuals reporting no high risk behavior later admitted that they lied.(31). In a CDC study of heterosexually acquired AIDS patients, 9% later admitted they were homosexuals.(33). Cochran and Mays found 47% of individuals with sexually transmitted disease lied about their behavior: 20% said they would lie about being HIV positive.(35)*

*In a U.S. government study of 12,329 AIDS patients claiming “undetermined” risk factors, follow up interviews discovered that all but 491 individuals (3.9%) really participated in high risk behavior.(30) Healthcare workers were found to be no more truthful in telling the facts about their private activities.(37)*

*Why would someone lie that they caught HIV/AIDS occupationally when, in truth, it was from high risk behavior? The answer is simple. If you claim to have been infected with HIV/AIDS occupationally, you get sympathy from your family and community, disability payments, legal protection and other secondary benefits. If you admit your AIDS came from high risk behavior (e.g. anal intercourse with homosexual men, drugs) you get thrown out of the house, divorced, jailed, fired from your job and generally stigmatized. That is why people lie about AIDS and we should be very suspicious of any stories claiming non-risk sources of occupationally involved AIDS infection. In many of these cases, the CDC took subjects’ claims at face value in absence of other scientific facts.(4-6 ) This “soft” data forms the basis of the CDC’s determinations in the Seven possible dental (occupational) AIDS transmission cases.*

### **LIMITED TESTING ACCURACY**

*AIDS is diagnosed in the industrial nations with a series of blood tests. Usually an ELISA survey test and, if needed, a confirming Western Blot test. Both tests require a sophisticated lab and well trained technicians.*

*Even though tests are considered accurate, false positives do occur. Kleinman, in a study of 5 million samples, found a 4.8% false positive rate for HIV (Western Blot) tests when compared to the much more accurate (and expensive) HIV-1RNA PCR test.(39)*

*The study found HIV tests to have a specificity of 100% and a sensitivity of 98%.*

*Another study found that numerous conditions like liver disease, drug abuse, pregnancy, hemodialysis, transfusions, etc. will give a false positive HIV test results.(40 )*

*Thus it is possible to be diagnosed as being HIV positive and having AIDS yet never be sick from the disease. This may explain the numerous HIV positive “non-reactors” who, unless they take the toxic antiviral drugs, have no observed problem with their*

health.(5,41 )

*Because of these reasons, dentists must be skeptical of anecdotal reports and cautious in extrapolating rare reports of occupational HIV/AIDS transmission “cases”.*

### ***THE SEVEN DENTAL WORKERS WITH “POSSIBLE” OCCUPATIONALLY ACQUIRED HIV***

*The CDC, in several years of “HIV/AIDS Surveillance Report” issues, stated that there were seven dental workers who are “possible” cases of occupational HIV/AIDS transmission. (42).*

*The designation, “possible” is defined as, “These healthcare workers have been investigated and are without identifiable behavioral or transfusion risks: each reported percutaneous or mucocutaneous occupational exposures to blood or body fluids or laboratory solutions containing HIV, but HIV seroconversion specifically resulting from an occupational exposure was not documented.” (42-44) In this often quoted data, there are no sources referenced.*

*The last possible occupational case was recorded in 1995.(44)*

*With no further cases reported, the CDC stopped publishing this category of health care “infection” in 2001.(1)*

*In 1999, the CDC changed the total number, removing one case; thus reporting a new total of 6 “possible” cases of dental worker occupational exposure. (45).*

*Dr. H. Gayle, Director of the CDC’s National Center for HIV, STD and TB Prevention explained that this change was because, “...CDC surveillance data are always presented as ‘provisional’ in these reports... further investigation showed the dental worker had other (behavioral or transfusion-related) risk factors...”.(46)*

*The subject had lied to investigators.*

*After several years of inquiry through innumerable phone calls, Freedom of Information Act (FOIA) requests, litigation and Congressional/government inquiry (9,14,46,47-52), the following data describing the “possible” occupational transmissions in dental workers was received from the government and is presented:*

*Of the seven (six) dentists classified as “possible” occupational HIV/AIDS transmission, three were general practitioners, one a periodontist, one a pedodontist and two were dental students.*

*Five had AIDS, two were HIV positive but had no symptoms.(52)*

*Three dentists were mentally impaired. The seven performed 22,134 procedures on 6,740 patients with no HIV/AIDS being transferred to or from the operators (DNA studies).(52)*

*Dentist 1. The first case was reported by Klein et al and used by OSHA to extrapolate the dangers of AIDS transmission to dental workers.(2,5,48). Klein found a male dentist who tested HIV positive and denied high risk behavior in a survey of 1,309 dental staff. He lived among and treated New York City “village” patients; a high AIDS risk population. He intermittently used protective equipment. His wife refused to be tested. HIV exposure could not be documented and the CDC authors freely made an assumption; that if the dentist did contract HIV occupationally, then Universal Precautions would have prevented transmission.(48)*

*The problem with this study is that it was based on an unproved assumption (the dentist got HIV occupationally from his patients)*

with no other supporting evidence concerning false positive testing or other high-risk causes (e.g. bisexual contacts, drugs, etc.). Investigators took his word as fact. OSHA based its decision to include dental workers in its 1991 Blood Borne Pathogen Rule on this one case describing it as proof of...”a risk of dental professionals acquiring HIV”.(2) There is no science supporting this conclusion. It was a guess.

*The 12-6-91 Federal Register (Bloodborne Pathogen Rule p.64021) contains one reference of “further evidence” involving two seroconverted dental workers, among a group of 69 health care workers, with no identifiable risk for infection. OSHA considers these cases “less complete” and states, “it is reasonable to assume that at least some of them resulted from occupational exposure.” but gives no scientific references to support this claim.(2)*

*A 1992 report in MMWR mentions these two dentists and states they worked in a correctional facility (treating high risk patients), experienced needle sticks from equipment used on unidentified patients and died before HIV –DNA studies and in depth interviews could be done.(49) Since there was little information on these two dentists (e.g. their potential high risk behavior), occupational transmission could not be ruled out by CDC staffers and thus they were classified as “possible”.(2,,(49),50)*

*This “possible” designation is problematic because “possible” is often extended to “probable”, then “most likely” and finally being assumed as “actually happened” classifications: data torturing often seen in other government publications with a political bias.(4)*

*Dentists 4 ,5 & 6 (including perhaps dentists 1-3). The CDC, after years of numerous calls and an ignored FOIA request from the American Association of Forensic Dentists, reconsidered its decision and provided more data on “possible” occupational seroconversion cases in 1996 and later, 2003. This change of heart may have been encouraged by pressure of a high ranking Congressional committee chairman (John Porter, MC) during funding hearings. The CDC provided a single “scientific” document in the form of a short abstract from the 1995 meeting of the American Association of Public Health Dentistry. This was the “hard scientific” data the CDC supplied to Congress (and the FOIA requests) on the “possible” dental occupational seroconversions.(51)*

*The objectives were “To describe demographic characteristics and exposure to HIV among dental workers (DW) reported to the CDC through 1994.” The summary of the report stated:*

*“Six Dental Workers (DW) reported without a specific risk had occupational exposures that were possibly associated with HIV transmission: three of those reported percutaneous exposure to patient’s blood or body fluids, although the patients were not known to be HIV-infected. Conclusions: Almost all of the DWs reported to the CDC with AIDS had behavior risks for HIV infection. Adherence to universal precautions by DWs is recommended.”(52)*

*This report states that “almost” all the possible cases of DWs seroconverting had high risk behavior, a proven source of HIV/AIDS infection unrelated to dentistry. The first dentist would not admit high risk behavior. There were no examples of individuals who did not have this probable cause of infection. When asked how accurate this data was in supporting the “possible” designation, one CDC official stated, “the scientific evidence is not very ‘hard’”. There are no documented cases of occupational HIV/AIDS transmission. There are no “probable” cases and the six dentists classified in the “possible” designation appears arbitrary, lacking any scientific veracity.*

*So where do we stand on the potential of dental workers (dentists, assistants, etc.) of acquiring HIV/AIDS professionally? AIDS/HIV seroconversion rates of dentists have been studied for over 20 years. There is no dependable scientific evidence to substantiate that dental workers are or have been in ANY danger. The historical odds of a dental worker acquiring HIV/AIDS occupationally is zero. This is supported by the facts that in billions of dental patient contacts there have never been any documented cases of occupational HIV/AIDS infection in dentistry anywhere in the world since AIDS was discovered. It appears*

that the CDC's proposed seven (six) possible cases of dental worker infection are based on scant, unscientific, poorly substantiated and unreliable/data.

*Because of the politics, panic, exaggerations, denials, scandals, redefinitions and unscientific epidemiology which form the basis of the governments dental-related recommendations/ regulations (not to mention an ignorant and fear crazed populace), dentistry has spent billions of dollars, person hours and lives lost on infection control schemes addressing the prevention of a disease that does not affect dental personnel.*

*Because of the lack of demonstrative infection transmission over the 20-plus years of AIDS (before and after the advent of Universal Precautions), we are faced with one humbling conclusion. The dental profession has been duped. Dental workers do not get occupational HIV/AIDS.*

*The FRAIDS epidemic in dentistry fueled an extreme infection control movement that was not warranted nor supported by the alleged science identifying a hazard. It has not significantly reduced the already small infection transmission rates of other diseases. Vast resources were diverted from the population's health care and livelihoods to address a "chicken little" disaster that never existed. Now that mythology and fear has somewhat abated, our profession should carefully re-examine the research and evidence available and produce clear, practical standards on disinfection, sterilization and patient treatment that more accurately reflect the objective scientific realities of HIV/AIDS hazards in dentistry. We should be skeptical of any alarmist's tales. Dentistry should not continue the fear and hype that has been embarrassing the dental community and enriching hucksters and false prophets since the 1980's.*

*I would recommend the following measures:*

- 1. Cease confusing the CDC's "six possible" occupational dental cases as fact. It is at best, an unsubstantiated guess. Carefully investigate the CDC's data and publicize the scientific findings.*
- 2. Do not believe everything government tells you. Require the CDC to provide full documentation (e.g. web) on all its data and decision making processes. Be skeptical and demand hard scientific proof for regulations.*
- 3. Allow the dental workers the option of choosing what protective equipment and measures they will use on a case by case basis utilizing their professional judgment. The existing broad governmental mandates (e.g. Universal-Standard Precautions are unsupportable.*
- 4. Establish a mechanism to insure accuracy in future infectious disease reporting and recommendations outside of the CDC (e.g. independent review panel, firing untruthful employees).*
- 5. Insist on objectivity, accuracy and balance in dental organizations and publications.*
- 6. Don't be so gullible and easily lead.*

*It is time for a change.*

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### **ISIS Report 03/04/04-“Pink Panacea,” at last a vaccine against AIDS?**

Sam Burcher reports on an **unconventional** vaccine that **could** provide treatment for AIDS

*A company based in Thailand has developed an oral vaccine against HIV/AIDS. The makers of a pink pill called “V1”; claim striking success in the treatment of HIV/AIDS symptoms [1].*

*Immunitor Corporation Company and V1’s creators are clinical researcher Dr Aldar S. Bourinbaiar and pharmacist Vichai Jirathitikal who have put V1 through a placebo-controlled phase II study. The results showed **significant** improvements in CD4 and CD8 cell counts, weight gain, decreased viral load and survival of end-stage AIDS patients. It also suggested that the V1 could **reverse the progression of AIDS without concurrent toxicity** [2].*

*Immunitor and Dr Orapun Metadilogkul, an independent physician who heads the Thailand Association of Occupational and Environmental Medicine Physicians, claim that **27** patients diagnosed with HIV/AIDS **have seroconverted from HIV positive to HIV negative after treatment with V1**. A phase III trial application has been submitted to the Thai Food and Drug Administration (FDA) aimed at demonstrating the beneficial properties of V1 on associated symptoms of AIDS such as wasting. If approved, trials will take place at the largest public hospital in Bangkok under Dr Metadilogkul.*

*V1 is said to be **a therapeutic vaccine** comprising “HIV antigens from **pooled clinical isolates** from HIV infected donors”. **These antigens are made into pills taken orally** that do not degrade in the digestion process of the stomach, **but trigger***

## **immune responses in the underlying mucosa-associated immune cells in the lining of the small intestine.**

*Some 90% of the immune cells in the human body are made up of lymphocytes and monocytes in the lining of the mucosal surfaces. So there are ten times more CD4 and CD8 lymphocytes residing in the mucosal lining than in the blood where only 2% of lymphocytes are present. According to Immunitor, these intestinal cells are the front-line defence against HIV and are the first to be destroyed or disabled by the virus.*

*Once mucosal immunity has failed, the common and often fatal symptoms of HIV/AIDS appear, such as diarrhoea and respiratory infections. V1 works on the premise that HIV/AIDS is a disease of mucosal immunity, so targeting antigens at mucosal surfaces is a valid clinical approach.*

*Public opinion on V1 came sharply into focus when Thai clinics began giving out the pink pills free of charge to AIDS patients at mass rallies organised in schools, police stations, sport stadiums and Buddhist temples. There was opposition to these actions by a number of workers in conventional medical practices, despite the fact that they have no effective treatment to offer to patients with HIV/AIDS.*

*V1 is extensively subject to toxicity studies both in vitro and in vivo. So far, studies by the Thai government and independent private laboratories have proved it exhibits no toxicity. Five mammalian cell lines tested at the highest dose of 10mg/ml showed no sign of cytotoxicity. The extrapolated dose of V1 that would cause death in humans is 2 200 pills per day, as against the recommended daily dose for adults of one or two pills per day.*

*Recently published data from Immunitor shows that 40 AIDS patients on a six month trial of V1 treatment increased their CD4 and CD8 cells counts by a mean average of 51 cells (19%) per microlitre of blood. Increase in body weight was 2.2 kg on average. But some patients' weight increased by as much as 30 kg, which is an important gain in the treatment of AIDS.*

*These encouraging results led Bourinbaiar and Jirathitikal to evaluate V1 therapy in the treatment of terminally ill AIDS patients in intensive care wards in Thai hospitals. They approached 117 patients and 53 decided to take V1 while 64 declined treatment. All patients were bedridden and had been receiving palliative care. None of the patients had access to conventional anti-retroviral drugs, but some had been treated with antibiotics. All the patients in the non-V1 group were dead by week 9. In contrast 30 out of the 53 in the V1 group were alive and able to resume normal activity.*

***After 20 months** on V1, 18% of patients who started **with almost zero CD4 counts** were still alive. It was also noted that patients receiving V1 seldom developed opportunistic infections, which further suggests that V1 improves mucosal immune responses to infections.*

*A retrospective analysis by Bourinbaiar and Jirathitikal of 650 HIV positive patients who had taken V1 for an average of twenty-three weeks showed significant results. In total, 496 (76%) were able to increase their body weight or at least maintain weight on the V1 regime; 389 (59%) gained 4.2 kg, while 107 (17%) remained unchanged and 159 (24%) lost weight. Everyone participating in the trial was able remain on it and suffered no serious side effects [3].*

*A further study took place whereby V1 was administered to the HIV-negative relatives of terminally ill AIDS patients over a median period of twenty-four days. Their blood was then transfused into the AIDS patients who experienced an improvement in their health. Results showed that increases in CD4 and CD8 counts were statistically significant [2].*

*V1 is currently licensed as a food supplement by the Thai Food and Drug Federation (FDA) and is produced for R&D purposes. A **one months supply costs around \$20-£30 per person**, but it is given freely to poor patients in public hospitals wherever*



possible. So far 65 000 Thais infected with HIV have been given the treatment.

Costs for V1 **contrast strikingly** with those of more established combination therapies or “cocktails” consisting of three-drug antiretroviral treatments of HIV/AIDS. A recent HIV Cost Services Utilization Study Consortium Analysis estimates that in the USA, 33 500 HIV infected adults seen twice a year for medications and blood tests spend **\$6.7 billion or \$22 000 per patient per year** [4].

Apart from the economic viability of V1, there may be other advantages when considering its use as a safe therapy for the developing world. It has broad-spectrum activity against many HIV subtypes and is stable in ambient tropical temperatures for three years, making refrigeration unnecessary. And no special skills or syringes are needed to administer the pill [5].

Immunitor is not disclosing the medicinal properties of V1, but instead recommend a cocktail of V1 and certain generic drugs as alternative and inexpensive treatments for HIV/AIDS. They cite examples of five compounds: gramicidin (the first antibiotic to be isolated), cimetidine (Tagamet), warfarin, (a common anti-coagulant), levamisole (an animal de-wormer), originally developed for animal use, but latterly became a useful drug in treating colon cancer in humans, and acetaminophen (Paracetamol). Immunitor says these unapproved drugs are all highly effective against HIV/AIDS and are incredibly cheap in comparison to approved combination therapies [6]. (See *Alternative AIDS Therapy from Cheap Generics*, this series)

Clinical trials of V1 are ongoing and phase III trials are scheduled for Africa with results pending. It is registered in Ghana and licenses have been applied for in several other African states. Immunitor hopes to build a vaccine plant to supply large amounts of V1 to Africans at low cost. This would meet a critical demand for affordable and available HIV/AIDS treatment in the Continent.

Much attention is centred on the high rates of infection and death, 95%, caused by HIV/AIDS in the developing world. But until recently the intimate association between the pandemic and poverty has been played down in the application of strategic approaches for HIV/AIDS. In his recent letter to *The Times* newspaper Prof. Kenneth Stuart, the medical advisor to the Commonwealth Secretariat, highlighted the need to recognise the role of poverty in effective treatments for HIV/AIDS. He says “The more the gap widens between rich and poor the greater the number of people who are left stranded in the backwaters of progress.” So not only are people in poverty traps deprived access to helpful technologies and medicines their ability to acquire knowledge is diminished along with their human rights [7].

The report “*Thailand Social Monitor: Poverty and Public Policy*” says 16 per cent of the country’s population, or about 10 million people, are now living on less than the minimal income of Bt900 per person per month, which constitutes the country’s poverty line [8].

“Poverty is re-emerging as one of the nation’s most serious problems,” said Ian Porter, the World Bank’s country director for Thailand at the launch of the new report, which was jointly prepared by the National Economic and Social Development Board, the Thailand Development Research Institute and international experts [9]. An international AIDS conference will be held in Bangkok in July 2004.

## **AIDS CAN BE PREVENTED AND TREATED WITH AEROBIC EXERCISES.**

### **2004 (JUNE) ISIS Report 06/04/04-Can Exercise Help Prevent & Treat AIDS?**

Dr. Veljko Velkovic presents evidence on how exercise may help treat and prevent AIDS, and if so, the simplest, most widely available and affordable natural ‘vaccine’ is being ignored.

## Introduction

The most effective way to control the HIV/AIDS pandemic would be the development of a safe and effective HIV vaccine. Unfortunately, despite enormous scientific and financial resources being deployed worldwide over the past 15 years, no vaccine candidate is on the **immediate** horizon [1,2] (but see “Pink panacea, an AIDS vaccine?” this series). There are strong indications, besides, that the AIDS vaccines currently tested in humans **are not only ineffective but also harmful** [3-5]. In addition, current medical therapy of HIV disease is **extremely toxic**, with multiple **side effects** and **drug interactions** (see “AIDS & HIV?” this series). It is also very expensive, and carries risks of developing **drug-resistant** HIV strains.

It is clearly desirable to pursue other less toxic, inexpensive, non-drug approaches in order to slow the spread of HIV infection and to decrease **the burden of HIV infection** and treatment.

The answer **may come from** certain antibodies that appear to be directly involved in controlling HIV disease progression [6,7]. These antibodies have **specific affinity, or cross reactivity**, to the HIV-1 envelope protein (gp120 surface antigen, **residues 280-302**, designated peptide NTM); but **may be naturally occurring auto-antibodies** (antibodies generated against the individual's own antigens) against a small protein molecule that acts **to dilate the blood vessels in the intestine**, the vasoactive intestinal peptide (VIP) [8,9].

It so happens that aerobic exercise training stimulates the formation of these anti-VIP/NTM antibodies [10] in both normal and HIV-positive individuals, and **perhaps** both **could benefit** from such exercise [9].

Increased levels of anti-VIP/NTM antibodies induced by exercise **may have** two beneficial effects. First, in HIV-negative individuals, the anti-VIP/NTM antibodies **could bind** HIV particles **in circulation** and prevent them from reaching their target cell, thereby, **reducing the risk of infection** with HIV and decrease **the transmission** of the disease. Second, in HIV-positive individuals, increased levels of anti-VIP/NTM could slow HIV disease progression **and** reconstitute the damaged immune system.

Aerobic exercise [like flapping your arms and flying] may be an important, inexpensive, non-toxic, widely available front line defence and therapy against HIV/AIDS. By acting as an immune stimulant (for both HIV positive and HIV negative individuals), it creates a type of “natural vaccine” that, if widely adopted, could contribute to **a worldwide** slow-down of the AIDS pandemic.

### **HIV and AIDS disease (Should at least be two diseases-Kaposi's and OI's, right?)**

The first step in HIV infection **involves** the gp 120 on the **outer envelope** of the virus binding **to receptors** on the cell surface of the host, allowing the HIV virus to enter the cell. The central portion of the gp120 molecule has **an immunoglobulin-like** structure, which facilitates participation in the immune network. Therefore, immediately after infection, HIV **tries** to produce a fit to the host idotype (individual type) by producing **thousands of variants of gp120**. This process of adaptation usually takes years, and during this time, the host immune system is more or less able to control the HIV disease.

In some HIV-infected persons, this period is short, and in others it can be quite long, giving rise to the designations “slow” and “fast” disease progression. After this latent period, a separate fraction of viruses will be established whose gp120 carries the host idotype [11-13]. This population of HIV becomes accepted by the host immune system as ‘self’ and therefore protected from the host's immune attack. Even worse, these gp120 molecules will be included in regulation of the immune network, destabilizing its vital components and accelerating progression of disease [14]. In this way, HIV may escape from the latent period and progressively destroy the immune system of the infected person.

The gp120 protein represents the key component of all AIDS vaccine candidates that are currently in clinical trials. These vaccines

may have an important flaw in that they produce antibodies that neutralize only the HIV variants, which carry vaccine-like gp120. As the variants of gp120 are produced by the HIV infection, the vaccine antibodies may have the effect of disarming the immune system's antiviral response and thus, increasing the likelihood of rapid disease progression [1,4,5]. This phenomenon has been seen in gp120 vaccine volunteers who later became infected with HIV [15,16], and would certainly reduce the utility of an HIV vaccine in AIDS prevention.

### *Useful auto-antibodies in HIV disease*

If an Achilles' heel exists in HIV, it might be in the central portion of gp120 (residues 280-302, RSANFTD NACTIIVQLNESVEIN, designated as peptide NTM [6]). (The letters stand for different amino acids: R=Arginine, S=Serine, A=Alanine, N=Asparagine, F=Phenylalanine, T=Threonine, D=Aspartic acid, C=Cystine, I=Isoleucine V=Valine, Q=Glutamine, L=Leucine, E=Glutamic acid) This portion of the molecule is highly conserved in all known HIV variants and appears to be crucial for viral infectivity. In fact, researchers have demonstrated that minimal changes in this sensitive peptide region will completely abolish HIV infectivity. Unfortunately, this part of gp120 is not immunogenic in humans [17] because the immune system treats this part of gp120 as 'self', possibly due to peptide's similarity to several human proteins [18]. However, an antibody, found in both HIV positive and HIV negative individuals, seems to have reactivity to this region of the gp120 molecule.

A computer-assisted search of the Swiss-Prot database reveals vasoactive intestinal peptide (VIP) as the best match to NTM among currently analyzed human proteins [8,9]. The antibodies reacting to NTM may therefore be auto-antibodies against VIP.

VIP is a small naturally occurring peptide, which plays several important roles in the human body as a vasodilator (dilates blood vessels), neurotransmitter, and modulator of the immune system.

VIP stimulates natural killer (NK) cells in the immune system (among the first line of defence against infection) and also the production of cytokine, a hormone that influences the activity of other cells in the immune system. VIP therefore has a very important role in modulating the immune system.

The HIV protein gp120 is sufficiently similar to VIP to serve as a molecular mimic and interfere with its function. The main consequence of this mimicry is to undermine the NK cells, making them dysfunctional, which is common in HIV-infected subjects. As Peruzzi and co-workers demonstrated, gp120 inhibits the ability of NK cells to kill infected cells, and this inhibition affects also the production of the pro-inflammatory cytokine IFN-gamma [19], which enlists the help of other cells in the immune system to fight the infection.

Thus, increase in circulating VIP can counteract the effects of the HIV gp120, by overcoming the latter's inhibition of NK cells, and by stimulating the production of VIP auto-antibodies which can also bind gp120 and prevent it from binding to NK cells.

Finally, both VIP and the peptide NTM has been previously identified as possessing sequence characteristics responsible for the interaction between HIV and the CD4 receptor [20], which represents the first step in process of infection.

It has also been demonstrated that sera from **HIV-negative asthma patients** contains high levels of natural anti-VIP antibodies with peptide NTM reactivity. A recent study on sera from 393 HIV- blood donors found that approximately 5% (21/393) contain significant levels of the anti-VIP/NTM antibodies, corresponding to two standard deviations above average.

For HIV+ individuals, the amount of anti-VIP/NTM antibodies available appears to strongly correlate with progression of HIV disease, suggesting that the immune system is attempting to overcome the infection. HIV patients in the first stage of illness (characterized by CD4 lymphocytes count greater than 500/ml), when the immune system is efficiently controlling HIV, have very

low levels of anti-VIP/NTM reactive antibodies, similar to levels in normal HIV- people [21].

*The level of these antibodies significantly increases in disease stages corresponding to CD4 values between 200 and 500/ml. Below that CD4 level (less than 200/ml), however, the amount of anti-VIP/NTM antibody sharply decreases. In the terminal stages of AIDS disease, NTM-reactive antibodies in sera of HIV+ patients appear to be significantly decreased.*

*Neurath and co-workers have also reported differences in the spectrum of antibodies against HIV gp120 in two groups of HIV-infected individuals, those who remained healthy for at least 10 years, and those who developed AIDS within 5 years of the onset of infection [6]. They found antibodies recognizing the peptide 280-306 of HIV-1 gp 120 (overlapping NTM) significantly more prevalent in asymptomatic carriers than in AIDS patients. Thus, the absence or disappearance of these antibodies may be a possible factor contributing to the development of AIDS [6].*

#### *Exercise as a natural source of VIP/NTM reactive antibodies*

*A unique method to produce high titers of VIP/NTM reactive antibodies may be available to both HIV- and HIV+ individuals. An article by Paul and Said in 1988 [10] showed that auto-antibodies to VIP were present in plasma from 29.6% of healthy (HIV-) human subjects who habitually performed aerobic muscular exercise, compared to 2.3% of healthy subjects who did not. The exercise involves running, cycling, swimming, aerobic dancing, and/or weight training, three or more workouts per week for a year or more prior to the study. The antigenic stimulus for the formation of these auto-antibodies could not be identified from their data. However, acute exercise has been shown to be associated with a brisk increase in plasma levels of VIP [22,23]. It is possible therefore that the antibodies may have been produced in response to increased VIP levels during exercise.*

#### *Effect of aerobic exercise training on HIV-positive individuals*

*Several studies on aerobic exercise training in HIV-positive individuals have demonstrated that it is safe, effective, and has a number of beneficial outcomes [24-38]. The aerobic exercise fitness improvements include a 10-25% improvement in lactic acidosis threshold (a sign of fatigue) and 5-10% increase in maximal oxygen uptake depending on the exercise training intensity. In addition, despite concerns about the stress of aerobic exercise on already damaged immune systems (specifically, increases in infections, morbidity, or mortality), there have been no documented adverse effects of aerobic exercise training in HIV-positive patients at either moderate or heavy exercise training levels [25]. The available literature clearly supports the idea that aerobic exercise is well tolerated by HIV-positive individuals.*

*With regards to immunologic improvement with aerobic exercise training, CD4 counts or viral loads may or may not improve during the exercise intervention in exercise (see below), although skin test reactivity to Candida antigen has been shown to improve with moderate exercise. The quality of life outcomes, however, were found to have improved significantly with aerobic exercise training relative to a non-exercising control group.*

*There are indications that exercise can stabilize CD4 cell count in HIV-infected individuals. Studies showed that people with CD4 cells between 200 – 500 /ml seemed to benefit the most from an exercise program. A pilot study performed by Olson and co-workers found that the mean change in CD4 percentage over the 24 months interval for weight lifters was -3.1% compared with -5.9% for runners [26]. According to these researchers, among HIV infected patients motivated to and capable of regular strenuous exercise, weight training may offer a salutary benefit superior to intense running. The same authors have also reported a case of a long-term survivor (12 years HIV+) of a tri-athlete with a rigorous daily exercise regimen demonstrating very low viral burden as reflected in non-detectable HIV RNA quantitative PCR and increase in CD4 during 6 years from 3 to 50 /ml (usual CD4 cell count is > 800 /ml) [27]. There was a report that exercise facilitated a return of the CD4 cell count to more normal levels [28].*

*In a large study involving 415 individuals (156 HIV positive and 259 HIV negative) Mustafa and co-workers demonstrated that exercising 3 – 4 times/week had a more protective effect than daily exercise [29]. Exercise in the HIV positive group covered by this study showed an increase in CD4 count during a year by a factor of 7%. It should be noted that some authors have reported moderate training can be sustained without any large change in CD4 cell count [30-32].*

### *Exercise for the masses*

*Aerobic exercise training has been shown to be a promising, non-toxic, non-drug adjunct therapy to improve physical fitness, increase quality of life, and potentially improve the immune status (as indicated by reactivity to Candida skin test) of HIV-positive individuals. If aerobic exercise training can also be shown to increase the titer of anti-VIP/NTM antibodies in normal individuals (potential to decrease the risk of HIV transmission) and in HIV-positive individuals (potential to slow disease progression), it would strengthen its case to serve as a widely available and affordable intervention that is non-toxic and free of drug interactions. It would be applicable worldwide, in both developed and developing countries.*

In January of **2005**, the FDA **recalled** Procleix HIV-1 – HCV Assay.

### **FORMAL PROPOSALS TO TEST EVERYBODY.**

On February 10th, **2005**, articles began to appear in the New England Journal of Medicine advocating that it would be timely and cost effective to test every man, woman, and child for “HIV” at least once in their lifetime.

***In all but the lowest-risk** populations (?), routine, voluntary screening for HIV once every three to five years is justified on both clinical and cost-effectiveness grounds. One-time screening in the general population may also be cost-effective [47, 48, 49].*

The authors of these articles did not define with precision who should be selectively biased “in all but the lowest risk populations,” but they recommended testing for children, and monogamous adults in addition of course to “high risk” people of color, drug addicts, pregnant women, and men who have sex with men.

In February, **2005**, the FDA **recalls** Globus Media Rapid HIV test kits.

In March **2006**, an article was published in the New England Journal of Medicine warning that:

*A case-control study of 101 blood donors (Simonson et al., 1995) who had been vaccinated against influenza and 191 matched controls showed that recent inoculation with any brand of influenza vaccine was significantly associated with **a false positive screening assay for HIV antibodies**. Guidelines of both Johns Hopkins and the New York State Department of Health list influenza vaccination as a known cause of indeterminate results on Western blotting for HIV antibodies (Reasons for false-positive, false-negative, and indeterminate results in assays for the detection of antibodies against HIV) [50]. However, it was subsequently suggested by “experts” that the false positives were not due to the flu vaccine, but instead were due to problems with “HIV” testing.*

**BRIAN WILLIAMS OF THE WHO SAYS PEOPLE ON ARVS HAVE SAME CLINICAL OUTCOME AS PEOPLE NOT ON ARVS DESPITE HIGH OR LOW T-CELL COUNTS. BETWEEN 3 TO 5% OF “HIV-NEGATIVE PEOPLE” NATURALLY HAVE CD4 COUNTS BELOW 350.**

In November, 11, **2006**, it was announced that “HIV positives that started AIDS drug treatment with low T cell counts had the same survival outcomes as HIV positives that began treatment with high T cell counts (From New Scientist, Nov. 11, 2006):

*“In cash-starved regions of the world, deciding who should get anti-retroviral drugs for HIV is a tough call. Now it seems that one of the main tools for making that decision may be **less reliable** than it appeared.*

**World Health Organization guidelines recommend** starting anti-retroviral drugs when someone’s CD4 cell count has fallen below 350 cells per microlitre, an indicator of HIV infection, or for people with symptoms of AIDS whose CD4 count has dropped to below 200.

Brian Williams of the WHO and his colleagues studied HIV-positive and HIV-negative populations **in eight African countries** including Ethiopia, South Africa, Uganda and Zambia. They found that **between 3 and 5 per cent of HIV-negative people had CD4 counts below 350.**

What’s more, when people with low pre-infection cell counts did contract HIV, and received anti-retrovirals, they survived for about nine years – **the same as people with high counts** (*Journal of Infectious Diseases*, vol 194, p 1450).

The new findings **call into question** just how much **we** understand about CD4 cells and their interaction with HIV, says Williams. “Generally, if you have high CD4 counts you can be considered to be doing pretty well and if you have very low counts, you’re in trouble,” says Williams.

**But CD4 counts can vary a lot naturally** so if you follow the WHO guidelines to the letter, then **some people started on anti-retrovirals would not even be infected with HIV, he concludes.**“

**“IT’S THE GUT STUPID.”**

In 2005, an article advancing how the pathogenesis of “HIV” works appeared in *Science Magazine* with the introductory sentence reading, “It’s The Gut, Stupid,” (4 March, 2005: Vol 307, p. 1395), by Jon Cohen, “an AIDS journalist” who covered the 12th International conference on Retroviruses and Opportunistic infections.

The meeting Cohen covered, as he described, “*was attended by some 3900 “HIV/AIDS researchers from 72 countries.*” In the *Science* article, a picture was presented showing a Kampalan man standing in front of a billboard, which said, Condoms. Abstinence, Faithfulness, and a caption under it stating that “New data ascribe Uganda’s AIDS success to condom use rather than the abstinence and faithfulness (or to abstinence and monogamy, as claimed by The Bush Administration). In his synopsis of this meeting, claims by David Ho, Daniel Douck, Ronald Desrosiers, were presented stating that “HIV” destroys the gut lymphoid tissue of recently infected people, and essentially “wipes clean” the lymphoid tissue lining the gut shortly after infection. The *Science* article also suggested that “**HIV**” **fibrosis** might be treatable with a new cancer drug and abl receptor inhibitor, **Gleevec**, which is supposedly an inhibitor designed for leukemia, but they suggest **it could stop fibrosis** and make AIDS a more survivable disease than cancer. The conclusion that “HIV” kills the lymphoid tissues of the gut in a few days to months **was at variance** with the CDC’s long-standing contention that “HIV” has an average latency of **5-10 years** before progression to AIDS [(CDC, National Prevention Information Network Website, Frequently Asked Questions (FAQs) About HIV and AIDS (<http://www.cdcnpin.org/hiv/faq/virus.htm>); CDC, *Morbidity and Mortality Weekly Report* (MMWR), Vol. 47, no. RR-5, April 24, 1998, p. 4)].

In 2005, The CDC published that rapid HIV testing should routinely be made available for the mother or her newborn” (National HIV Testing Resources; “HIV Test FAQ” 2005).

**VAXGEN GETS AN 877 MILLION DOLLAR REWARD FOR “BLOWING UP” ITS “HIV VACCINE,” BUT HAS FUN WITH ANTHRAX.**

In 2006, The Project On Government Oversight (POGO) presented an investigative report that examined The Government's procurement procedures for vaccines **under Bioshield**, and revealed intricate conflicts of interest as former high level government officials "continue to wield influence after moving to the manufacturers of two controversial anthrax vaccines – Bioport and **VaxGen**." <sup>11</sup>

## **Of Drugs and Dollars**

*Newsweek* reports that VaxGen, a little-known California biotechnology company, will start its first delivery of **its anthrax vaccine** to the government six months later than originally slated. The company was awarded an **\$877.5 million** contract to produce and manufacture the vaccine, which was developed by the U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID). The company blames the delay on "**regulatory questions and production issues**" (the *Newsweek* article, this *Wall Street Journal* piece and this *Forbes* article from the summer delve into the problems).

**Seventy five million doses of VaxGen's vaccine are to be procured for the Strategic National Stockpile under Project Bioshield**, a joint Department of Homeland Security (DHS) and Department of Health and Human Services (HHS) initiative **to stimulate the creation of a domestic biodefense industry**. Five million doses of VaxGen competitor Bioport's vaccine were procured earlier this year in response to Bioport's aggressive lobbying and anti-VaxGen campaign (pdf) (see this pdf for VaxGen's rebuttals to Bioport's claims). VaxGen's vaccine has **not** been approved by the Food and Drug Administration. Bioport's vaccine, which has been used by the Defense Department, has been controversial **because of its side effects** and its FDA approval has been disputed. POGO pointed out in a recent blog that Jerome Hauer, the former head of HHS's biodefense program, is now on the Board of Emergent Biosolutions, Bioport's parent company. VaxGen's counterpart to Bioport's Hauer is Eve Slater, former Assistant Secretary for Health at HHS, who just joined VaxGen's Board. And as a *Forbes* article suggests, VaxGen may have had help from **a well-positioned friend** in the government when it obtained the Fort Detrick-based USAMRIID vaccine:

*As its AIDS work was **blowing up** (as the AIDSVAX vaccine was **clearly failing**), VaxGen was working to get access to Fort Detrick's anthrax vaccine technology. It had friends. VaxGen Chief Gordon is a long-time acquaintance of Philip Russell, the former chief of Army medical research. Both sit on the board of **the Albert B. Sabin Vaccine Institute** in New Canaan, Conn. Fort Detrick gave VaxGen the license to its anthrax technology in October 2003. Russell, then an adviser to HHS, stepped in to settle a fight between government bureaucrats over whether VaxGen would pay royalties to the government. He said, "Dammit, I don't care what you do, but settle it—don't leave this company in the lurch," recalls Gordon.*

*A year later HHS awarded VaxGen the BioShield award. Gordon and Russell adamantly deny their relationship had any influence on VaxGen's selection. "I scrupulously stayed away from talking to him, to the point where I felt terrible about it," says Gordon. The company now quotes Russell in its media kit: "We have a lot of **faith** in this vaccine, and we believe it's the right way to move forward to protect **the country** against anthrax."*

*In a CNN/Money piece, Jeffrey Marshall, an analyst for Fairview Capital Group, said VaxGen has the "closest ties with the government" versus competitors for other Bioshield contracts. Which given the Russell and Slater connections may be true. But examination of VaxGen and its CEO and President Lance Gordon's history lends even more weight to Marshall's opinion.*

*Reminiscent of the Darleen Druyun-Boeing tanker lease scandal last year, the *Forbes* article notes:*

*In 1999 [VaxGen] won an **\$8 million government contract** to test its AIDS vaccine, but the official negotiating the contract for the Centers for Disease Control got in trouble for simultaneously talking to VaxGen about a job.*

And in 1998 the New York Times reported that **OraVax's** vice president (OraVax was then headed by Gordon) may have influenced a high-level government meeting to shape the US government's **biodefense strategy** to benefit Oravax financially, while not **adequately** disclosing his ties.

Is VaxGen capitalizing on its ties to the government in order to score hundreds of millions of dollars in government contracts?

With a reorganization and expansion of Project Bioshield being contemplated in Congress **and President Bush proposing to spend \$7.1 billion on avian flu preparations, including vaccines**, the government should learn from what it's already doing with vaccine procurement. **There may not be time to make anymore mistakes.**

Nick Schwellenbach, Investigator, Project On Government Oversight (POGO) <http://www.pogo.org>

## **MANDATORY TESTING OF ALL INFANTS BEGINS.**

January 19, **2006**. Illinois House Committee Approves Mandatory HIV Testing Bill:

Springfield, IL-The House Human Services Committee voted this morning 9-3 in support of House Bill 4306 mandating HIV testing of newborns **when the mother's HIV status is unknown**. The measure, sponsored by State Representative Mary Flowers (D-Chicago), now advances to the full House for its consideration.

The AIDS Foundation of Chicago (AFC) opposes House Bill 4306 on the grounds that it is unnecessary and discriminatory toward pregnant women. **Fully 98% of new mothers and infants** discharged from Illinois birthing hospitals **have received HIV testing** as a result of legislation enacted in 2003 expanding voluntary HIV counseling and testing for pregnant women and newborns. Thanks to passage of the 2003 law, championed by AFC and a broad-based coalition of groups, testing rates among expectant mothers and newborns **have risen from 73% in 2004 to 98% at the end of 2005**. **Because newborn testing only shows the HIV status of the mother-an infant's own immune system develops several months after birth-mandated newborn testing is in fact mandated testing for new mothers**. H.B. 4306 **would deny** new mothers the informed consent to testing which is afforded to virtually all other populations.

In 2006, the FDA **recalls** BioMerieux, Vironostika HIV-1 test kit, and Abbott's Home access HIVAG-1 Monoclonal EIA Test Kit.

## **AFTER 20 YEARS, "HIV" BEGINS TO CHANGE ITS SIZE BY A FACTOR OF THREE**

Tuesday, 24 January **2006**, The BBC released a story about researchers claiming that "HIV" varies in size from virus particle to virus particle by a factor of three:

*3D structure of HIV is 'revealed'*

*Aids affects **40 million** people across the world. Scientists have created a map showing the 3D structure of the virus which causes AIDS. **The variable size and shape of HIV** has made it **hard** to map, the team said in the journal Structure. So the UK-German team took hundreds of images of viruses, that are **60 times** smaller than red blood cells(?), and used a computer program to combine them. Oxford University's Professor Stephen Fuller said the 3D map would assist in understanding how the virus grows.*

## **Unusual features**



*He told the BBC: "You say can you show me the structure of the HIV virus **and the question is which one.**"*

*"HIV is **very** variable. It varied in diameter by a factor of three."*

### **BARRE-SINOUSSE SAYS "15 THERAPEUTIC HIV VACCINES ARE NO GOOD."**

In **2006** at the Toronto International AIDS conference, Barre-Sinoussi said:

*It is not clear if **therapeutic vaccines** might be useful, since 15 trials to date have not demonstrated definitive evidence of improved outcomes.*

From U.S. Congressional records produced from meetings with vaccine makers in 1995, Barre-Sinoussi's statement came 11 years after there were a total of at least 30 different trials for "HIV" vaccines on record (many of them Phase I or Phase II trials, without a single claim for induction of antibodies against "HIV," cellular immunity, stimulating of cytotoxic T-cells, or mucosal immunity). [51].

### **"VIRAL LOAD" CAN'T PREDICT WHEN 94% TO 96% OF PEOPLE WILL PROGRESS TO AIDS, BUT CAN PREDICT WHEN 4% TO 6% OF THOSE WHO "TEST POSITIVE" FOR "HIV" WILL:**

In **2006**, it was announced that viral load is only able to predict **the rate** of progression to disease in 4% to 6% of any HIV-positives studied, challenging much of the basis for current AIDS science and treatment policy for any individual who tests "HIV" positive [52, 53].

### **THE SUCCESS OF CIRCUMCISION IN PREVENTING TRANSMISSION OF "HIV" IN AFRICAN STD CLINICS: BIBLICAL SLAVE-MARKING PRACTICE OF THE EGYPTIANS AND HEBREWS WINS OUT OVER PHARMACEUTICAL TECHNOLOGY:**

On December 14, **2006**, a New York Times editorial article appeared, entitled, "Rare Good News About AIDS":

The announcement yesterday about the results in two African studies of male circumcision **may be** the most important development in AIDS research **since the debut** of antiretroviral drugs more than a decade ago. The National Institutes of Health **halted** studies in Uganda and Kenya when it became **overwhelmingly clear** that circumcision significantly reduces men's chances of **catching** H.I.V.

A Kenyan AIDS trial was interrupted because a 53 percent reduction in acquisition of "HIV" among circumcised men was observed. Out of 2,784 men studied in the trial, 69 men were "HIV" positive: 22 of these were circumcised, and 47 uncircumcised. **Many, if not all 65 of them** had received prior (or concurrent) treatment **for penile infections**, and 28 of the 69 **had detectable serologic syphilis** at the outset. A year before, it was claimed that a trial of 4,996 HIV-negative men in Rakai, Uganda, showed that HIV acquisition was reduced by 48 percent in circumcised men. **Yet**, other prior studies regarding the ability of circumcision to protect against "HIV" acquisition in both males and females yielded conflicting data regarding the ability of circumcision to block acquisition of "HIV," and the role that other STD's or medical conditions play as cofactors in acquiring "HIV." Uncertainties existed because: data had been acquired at STD clinics or from trial participants with genital ulcer disease (GUD) or other infections, and the relative roles (if any) of biological versus cultural practices that influence "HIV" acquisition were challenged by the WHO. Uncertainties regarding **the damage done by microbicides** also existed at that time, **which** apparently **increased the frequency** of reported genital lesions and the **feared spread** of "HIV." The ability or inability to neutralize "HIV" by washing with mild or concentrated detergents was **in question**, and the transmission of "HIV" from human to human by providing evidence of

seroconversion was yet to be provided in a form that constitutes as careful a study as the 10 year study that followed 175 serodiscordant couples for 10 years that found no seroconversions in “HIV”-serodiscordant couples [34]:

*We followed up 175 HIV-discordant couples [one partner tests positive, one negative] over time, for a total of approximately 282 couple-years of follow up...(it was a 10 year study-emphasis mine)- **No transmission** [of HIV] occurred among the 25% of couples who did not use their condoms consistently, nor among the 47 couples who intermittently practiced unsafe sex during the entire duration of follow-up...” “We observed **no seroconversions** after entry into the study [nobody became HIV positive]...This evidence argues **for low infectivity** in the absence of either needle sharing and/or other cofactors.*

Uncertainties also existed because of the vastly different rates and efficiency of transmission said to be associated with heterosexual, homosexual, and IV drug use in different regions, and, because of the ability of gamma globulin in neutralizing “HIV” among well-nourished and healthy individuals. Uncertainties also exist especially because of the validity (and invalidity) of different test kits to identify “HIV- positive” participants, and the role (or non-role) of T-cells in progression to AIDS is also still in question.

The role of circumcision in preventing transmission of “HIV” and acquisition of AIDS in Africa was further complicated by compelling evidence from a series of recent studies by Gisselquist et al., that identified nosocomial (hospital and doctor-medicated) “HIV” transmission as the single most critically important factor for the spread of AIDS in Africa, and which accounts for many anomalies and conundrums that cannot be explained by a sexual transmission hypothesis. For example, many studies reported HIV infections in African adults with **no sexual exposure** to “HIV,” **and in children with “HIV-negative” mothers**. Transfusion was also known to be a very high risk factor, and unexplained high rates of “HIV” incidence were observed in African women **only during** antenatal and postpartum periods. In the years preceding the release of this “rare good news about AIDS” there had been many studies that claimed to show that 20%-40% of “HIV’s” molecular profile in African adults is associated with injections **at clinics**. From the 1950s into the 1980s, unsafe injections may have contributed to the silent spread of “HIV’s” molecular signature **in Africa** in much the same way that other types of vaccination campaigns, including injections for schistosomiasis and other treatments in Egypt, established “hepatitis C” as a major blood-borne molecular profile (not a virus). Evidence for hospital transmission of “HIV’s” molecular profile continued to accumulate since the long established fact that hepatitis B and flu vaccines cause “HIV” positive tests in some individuals.

Despite all the money invested, 22 years after the AIDS era began, nobody could have guessed that it would be the age-old biblical slave-marking practice of the Egyptians and Hebrews who lived in ancient Egypt, that circumcision would be superior to results obtained from “HIV” vaccines, microbicides drug roll-out experiments executed on millions of people with HAART, AZT, nevirapine, integrase inhibitors, microbicides, or condom crusades. Thus, in 1996, because of the circumcision results of African males who visited STD clinics, all of whom were said to have previous STD’s, victory was claimed at last over the AIDS pandemic with this appearance of African circumcision studies that now claimed to show success in diminishing the transmission of “HIV’s” molecular signature of African males who circumcise.

## **STEPHEN LEWIS WANTS TO SMEAR AFRICAN GENITALS, AND THE SUCCESS OF THE UN.**

Also in **2006**, a documentary appeared [54] in which CNN’s Chief International Correspondent Christiane Amanpour was filmed walking through villages in Kenya, remarking about the fact that the impoverished orphaned children don’t have enough watered down gruel to drink down the drug nevirapine, and other drugs such as AZT. In the documentary, Ms. Amanpour also provided the viewer with an interview with Stephen Lewis about his perspectives as the UN Secretary-General’s Special Envoy for HIV/AIDS in Africa, a post he held since June 2001. As Commissioner for the World Health Organization’s Commission on the Social Determinants of Health, as Senior Advisor to the Mailman School of Public Health at Columbia University in New York, as Director of the Stephen Lewis Foundation, and recipient of many awards and 22 honorary degrees including a Companion of the Order of Canada, Maclean’s Magazine 2003 “Canadian of the Year,” and as Time Magazine’s April 2005 100 most influential people in the

world, Lewis is an important figure in the AIDS establishment.

In a speech Lewis gave at the closing session of the XVI International AIDS conference in Toronto, he presented a list of issues regarding AIDS in the world and especially in Africa. In his speech, Lewis spent one of the sessions vilifying The South African Minister of Health for advocating foods that are important for nutrition and health.

And the sixth most important issue regarding AIDS, according to Lewis, was:

Number **6**: It is now accepted as unassailable truth that people in treatment need nutritious food supplements to maintain and tolerate their treatment. And yet, there is a growing clamour from People Living with AIDS that decent nutrition simply isn't available, leaving them in a desperate predicament. The World Food Programme released a study at this conference calculating the cost of food supplementation at 66 cents a day for an entire family; what madness is it that denies the World Food Programme the necessary money?

The growing "clamour" from people living with AIDS, as Mr. Lewis described it, was another way of saying "we are hungry- we are starving for food and water," and it was made clear in the documentary that the people filmed couldn't possibly be receiving the amount of protein, in either vegetable or animal form, to sustain immunity. When considering assays in human patients which diagnose "AIDS" by quantifying the number of lymphocytes/ml, patients are not considered to have an AIDS-defining illness if they have suffered from chronic starvation, as these individuals are known to possess a helper T-cell ratio in the AIDS-defining range or even lower (< 250 cells/ml), and can present with as much as a 90% reduction in their normal T-cell number which is reversible upon nutritional supplementation and a normal diet [55, 56]. Both in Amanpour's documentary, and in Lewis's lofty speech before the International AIDS conference where he lists food as number 6 in importance (following of course more important things like circumcision and microbicides to smear on African people's genitals), an intractable paradox was presented whereby it is difficult to conceive how all the African children could have received highly toxic immune suppressive drugs such as nevirapine and AZT, when they didn't have enough food and water to wash down the pills.

#### **"HIV" IS CAUSED BY CIGARETTES, BUT CIGARETTES DON'T CAUSE AIDS.**

On Thursday, Sep 21, 2006, as reported in Reuters, it was announced for the first time that the probable cause of increased "HIV's infection had been found to be.....**smoking cigarettes, but 9 of 10 other studies failed to find a link between smoking and progression to AIDS:**

LONDON (Reuters) – Smoking, already linked to several illnesses, may also increase the risk of infection with HIV, **the virus that causes AIDS.**

*In a review of studies that looked at the association between smoking and HIV, British **doctors** said five of the six studies they analysed showed smokers had a higher chance of becoming **infected.***

**Nine of 10 other studies** in the review that tracked the progression from HIV to AIDS found no link with smoking.

*"The studies identified in this systematic review indicate that while smoking might be independently associated with acquiring HIV **infection**, it does not appear to be related to progression to AIDS," said Dr Andrew Furber, of the South East Sheffield Primary Care Trust.*

*Furber and his colleagues, who reported the findings in the journal Sexually Transmitted Infections, said tobacco smoke may increase susceptibility to HIV infection **by modifying a variety of immune system responses.***

Research has shown that smoking is a **leading cause** of preventable death. It increases the risk of heart attack and stroke, respiratory problems, lung and other types of cancer. The researchers suggest in the study that public **health measures that encourage smokers to quit could also improve the effectiveness of HIV/AIDS prevention programmes.**

### **AIDS PATIENTS CAN LIVE ON AVERAGE 24 YEARS IF THEY PAY \$385,000 DOLLARS.**

In **2006**, Dr. Bruce R. Schackman, chief of health policy at Weill Cornell Medical College in New York and lead author of a paper appearing in Medical Care in 2006, a journal published by the American Public Health Association claimed that “...patients can live average 24 years, if they pay \$385,000.”

### **AFTER FIRST DECADE OF HAART IT IS REPORTED THAT IMPROVEMENT IN “VIRAL LOAD” MEASUREMENTS ARE OBTAINED BUT NO IMPROVEMENT IN MORTALITY IS ACHIEVED: THERAPY WAS A SUCCESS BUT THE PATIENTS DIED.**

Methods: We analyzed data from **22,217 treatment-naïve HIV-1-infected** adults who had started HAART and were followed in one of **12** cohort studies. The probability of reaching 500 or less HIV-1 RNA copies per mL by 6 months, and the change in CD4 cell counts, were analyzed for patients starting HAART in 1995-96, 1997, 1998, 1999, 2000, 2001, and 2002-03. The primary endpoints were the hazard ratios for AIDS and for death from all causes in the first year of HAART, which were estimated using Cox regression.

*Interpretation: Virological response after starting HAART **improved** over calander years, **but such improvement has not translated into a decrease in mortality.** [The Antiretroviral Therapy (ART) cohort Collaboration-www.thelancet.com Vol 368, 451-58, August 5, 2006].*

### **HOW BETTER MICROBICIDES WERE DEVELOPED BY JOHN MOORE AND OTHERS.**

Also in **2006**, Dr. John Moore of Weil Medical College, one of the featured speakers in the 2006 International Toronto AIDS Conference described his work, and claimed that his “**Hail Mary**” experiments hold great promise and could solve the “AIDS apocalypse in Africa and elsewhere. He alluded to the fact that multiple inseminations with his monkeys are necessary in order to model the frequent sexual activity that goes on in these 3rd World Nations. To rigorously prove his “**SIV-fighting**” microbicide worked, his Hail Mary experiments involved inseminating macaques 4-5 times after smearing his microbicides on their genitals.

February 1, **2007**, Tests of Drug to Block H.I.V. Infection Are Halted Over Safety: The Conrad Trial. By Lawrence K. Altman:

*Efforts to develop a topical microbicide to prevent H.I.V. infection during sex suffered a **surprising** setback yesterday when researchers announced that **they had stopped two full-scale trials for safety reasons.***

*The trials, in Africa and India, involved a chemical, cellulose sulfate or Ushercell, and were the second failure of a potential microbicide in a full-scale trial in recent years. In one of the latest trials, a standard check by an independent scientific committee found **an increased risk of H.I.V. infection** among women who used cellulose sulfate compared with those who used a placebo gel.*

*In 2000, a large full-scale trial showed that the only other microbicide candidate, nonoxynol-9, was **unsafe** when it had been expected to be effective. Subjects in that trial developed **a higher incidence** of H.I.V. infection, presumably through ulcers caused by chemical irritation.*

*Yesterday, AIDS researchers at the World Health Organization, the United Nations AIDS program and other organizations*

expressed **hope** that at least one of three other potential microbicides undergoing full-scale testing would prove to be safe and effective. The others are Pro 2000 by Indevus Pharmaceuticals, BufferGel by ReProtect and Carraguard, whose trademark is held by **the Population Council**.

“While the closing of these trials is a profound disappointment for the microbicide field, we cannot let it paralyze us,” said Dr. **Zeda Rosenberg**, chief executive of the nonprofit International Partnership for Microbicides in Silver Spring, Md.

In the absence of an AIDS vaccine, specialists say development of a microbicide is a public health priority, mainly to protect the many women in poor countries whose partners **refuse** to use condoms. Such protection could take the form of a gel, cream, film, tablet or sponge that could be inserted into the vagina or rectum.

The study that led to stopping the trials involved **1,333** participants in Benin, South Africa and Uganda. Conrad, a health research organization in Arlington, Va., conducted the study.

Conrad said the independent committee found more new H.I.V. infections among those who used cellulose sulfate than among those who used an inactive gel, **but did not report any numbers**. Final numbers are expected in March, a spokeswoman for Conrad said.

Family Health International of Research Triangle Park, N.C., conducted the second trial involving 1,700 participants in Nigeria. The study found neither a benefit in preventing H.I.V. infection nor an increased risk of developing it.

So, given the adverse findings in the Conrad trial, “the responsible course of action was to halt our study” also, said Dr. Vera Halpern, the principal investigator of the Family Health International trial.

An ideal microbicide would work in three ways. First, it would kill H.I.V. in the vagina and cervix. Second, the microbicide would prevent any virus that escaped from attaching to a woman’s cells, the way the virus starts to infect. Third, for any virus that did enter cells, the microbicide would block an enzyme, **reverse transcriptase**, that the virus needs to replicate.

The Bill and Melinda Gates Foundation and the Agency for International Development paid **\$20 million** for the two latest studies.

In speaking at the 16th International AIDS Conference in Toronto in August, Bill and Melinda Gates were **enthusiastic** about the prospects of developing a microbicide.

Yesterday, Dr. Nicholas Hellmann, acting director of the foundation’s H.I.V. and tuberculosis program, affirmed **the optimism**.

“We remain hopeful that a safe and effective microbicide will be developed,” Dr. Hellmann said, adding that the foundation was still committed to supporting research on microbicides and other prevention methods.

The new findings were surprising, researchers said, because 11 smaller trials of more than 500 women conducted since 1999 showed that cellulose sulfate was safe. The chemical, which was developed as UsherCell by Polydex Pharmaceuticals in Toronto, was active against H.I.V. in laboratory tests.

Dr. Peter Piot, the executive director of Unaid in Geneva, said the new findings were puzzling because there appeared to be no biological explanation for the failure of cellulose sulfate, as there was in the case of nonoxynol-9 and the ulcers associated with its use.

*Finding new drugs like a microbicide often can be a process of trial and error, and requires scientifically rigorous trials, Dr. Piot said.*

*He speculated that one of the antiretroviral drugs used to treat AIDS might be needed for an effective microbicide. The world needs a microbicide because “the stakes are so high,” Dr. Piot said.*

### **PEDIATRICIANS SUGGEST FORCED COMPLIANCE USING GASTROSTOMY TUBES IN INFANTS TO FORCE THEM TO TAKE AS MANY AS 7 BLACK-BOX LABEL DRUGS:**

**2007**, February. A report from American Academy for Pediatrics publishes:

*“Gastrostomy Tube Insertion for Improvement of Adherence to Highly Active Antiretroviral Therapy in Pediatric Patients With Human Immune Deficiency Virus.”*

### **ROGER ENGLAND SAYS “TOO MUCH MONEY IS BEING SPENT ON AIDS:”**

**2007** February. Roger England says too much money is being spent on “AIDS.”

*Too much money for AIDS-Roger England Press releases Saturday 17 February 2007.*

*Billions of pounds are being spent on the fight against AIDS in developing countries. In this week’s BMJ, two experts go head to head over whether we are spending too much. HIV is receiving relatively too much money, with much of it used inefficiently and sometimes counterproductively, argues Roger England, Chairman of Health Systems Workshop.*

*Data show that 21% of health aid was allocated to HIV in 2004, up from 8% in 2000. It could now exceed a quarter. Yet HIV constitutes only 5% of the burden of disease in low and middle income countries as measured by disability adjusted life years lost (DALYs). It causes **2.8 million deaths a year** worldwide – fewer than the number of stillbirths, and much less than half the number of infant deaths. More deaths are attributable to **diabetes** than to HIV.*

*Furthermore, HIV interventions are not cost effective enough to justify this disproportionate spending, he writes. Much HIV money could be spent with more certain benefits on, for example, bed nets, **immunisation**, or **family planning**. Money is also wasted in areas that reflect **the interests of those on the AIDS industry payroll more than evidence**.*

*He believes that the money could be more effective if used to strengthen public health systems rather than focusing on disease-specific programmes. AIDS is widely acknowledged as a public health crisis and current spending is **woefully inadequate**, argue Paul de Lay and colleagues at the Joint United Nations Programme on HIV and AIDS (UNAIDS).*

*Resources currently pledged **are only half** of what is needed for a comprehensive response. For instance, in 2006, **\$9bn** was available for the AIDS response but the real need was estimated at **\$15bn**. Poor coordination between different **stakeholders** in affected countries also impedes effective spending. This is compounded by weak institutions and regulatory policies, poor governance, and **in some cases** corruption.*

*They argue that the response to AIDS needs to be seen in the context of international commitments to the millennium development goals, which also call for progress across many other developmental priorities. HIV threatens many of these goals, especially those related to poverty and health.*

The cost of inaction against AIDS is huge, far greater than for any other public health crisis, they say. Current costs are so high because of **the inadequacy of previous investments**, but they **will be** higher tomorrow if we continue to underinvest.

## **SIX HEALTH CARE WORKERS BEFORE A FIRING SQUAD FOR INFECTING 426 CHILDREN IN LEBANON, BUT THE BLACK PEOPLE ARE BLAMED AND THE HEATH-CARE WORKERS ARE FREED.**

Six Bulgarian health care workers (The Tripoli Six) were to be executed by firing squad in Libya for their alleged role in supposedly transmitting “HIV” to 426 Libyan children. In the summer of 2007 the 6 health care workers were exonerated of spreading “HIV” to the 426 children because of the efforts of Luc Montagnier and others, who shifted the blame for the 426 infections on **black persons** coming to work at the hospital **from sub-Saharan Africa**:

### ***Epilogue in Libya: A spreading AIDS epidemic***

*By Elisabeth Rosenthal*

*Thursday, July 26, 2007*

*ROME: Five Bulgarian nurses and a Palestinian doctor landed in Sofia this week, freed of a death sentence after eight years in Libyan prisons, an apparent victory of diplomacy at long last.*

*Officially, two visits to Libya by Cécilia Sarkozy, the French president’s wife, precipitated the release of the six medics who had been found guilty – not once, but twice – of **infecting** more than 400 children with HIV as part of a plot **by the Israeli secret service**.*

*Sarkozy’s visit was only the latest in countless pilgrimages by diplomats and scientists to the Libyan leader, Muammar el-Qaddafi, to plead the medics’ cause. Recent visitors included the U.S. secretary of state, Condoleezza Rice, the European Union’s external relations commissioner, Benita Ferrero-Waldner, and Richard Roberts, a Nobel laureate, who represented more than **100 Nobel Prize winners**.*

*But the drawn-out drama also reflects a complex structure of Libya’s internal politics that prevented an obvious solution from being reached, experts in the case said. And the **sad epilogue** will be in Libya, too: **an AIDS epidemic** that has never been fully acknowledged and that continues **to spread**, as well as the 426 children dependent on treatment in a system ill-prepared for the task.*

*It was completely clear scientifically since 2002 that they were not guilty,” said Vittorio Colizzi, a **renowned** AIDS expert who was invited by the Qaddafi family to study the hospital in Benghazi where the infections took place and was given wide access to wards and medical records. “But the nurses suffered for years from the incapacity of diplomacy and politics to free them in a timely manner.*

*He and another expert, **Dr. Luc Montagnier**, the French virologist whose team discovered HIV, concluded that the AIDS virus was present in the hospital before the nurses arrived, **probably brought to Libya by guest workers from countries in sub-Saharan Africa**.*

In other words, the infection of the 426 children was attributed to the presence of Africans near the children, if this quote from Montagnier is accurate.

## **THE SUCCESS OF NEVIRAPINE EXPERIMENTATION ON 875,000 AFRICAN BLACK WOMEN AND THEIR INFANTS, AND 33-87% VIROLOGICAL FAILURE AFTER A SINGLE DOSE:**

In 2007, virological failure or drug resistance became widely used technical terms among “HIV-AIDS” promoters that have come to mean that an anti-retroviral drug doesn’t work (fails to suppress virus), or that disease progression is more rapid in those that take a particular drug. In the New England Journal of Medicine, it was reported (and despite its known toxicity and withdrawal from the U.S. several years ago):

Well **over 875,000 women and infants** have received a single dose of nevirapine. A single dose of nevirapine is the cornerstone of the regimen recommended by the World Health Organization (WHO) to prevent mother-to-child transmission among women without access to antiretroviral treatment and among those not meeting treatment criteria. However, nevirapine resistance is detected (with the use of standard genotyping techniques) in **20 to 69%** of women and **33 to 87%** of infants after exposure to a **single**, peripartum dose of nevirapine. Among 60 women starting antiretroviral treatment within 6 months after receiving placebo or a single dose of nevirapine, no women in the placebo group and **41.7% in the nevirapine group had virologic failure** ( $P < 0.001$ ). Women who had received a single dose of nevirapine had significantly higher rates of virologic failure on subsequent nevirapine-based antiretroviral treatment than did women who had received placebo. This **apparently deleterious effect** of a single dose of nevirapine was concentrated in women who initiated antiretroviral treatment **within 6 months** after receiving a single dose of nevirapine. We did not find that a previous single dose of nevirapine compromised the efficacy of subsequent nevirapine-based antiretroviral treatment in women who started antiretroviral treatment 6 months or more **after** delivery. Among the **30 HIV-infected infants**, a single dose of nevirapine (one each to mother and infant) as compared with placebo **was associated with significantly higher rates of virologic failure and smaller CD4+percentage increases** in response to subsequent nevirapine-based antiretroviral treatment [57].

## **THE SUCCESSES OF BREAST FEEDING DISSUASION COUNSELING TO PREVENT “HIV” TRANSMISSION, PROGRAMS OF FORMULA DUMPING ON THIRD WORLD NATIONS, AND 532 DEAD CHILDREN:**

On Monday, July 23, 2007, in Nkange, Botswana, it was reported by Craig Timberg, Washington Post Foreign Service, that in Botswana, steps to cut AIDS proves a formula for disaster:

*Doctors noticed two troubling things about **the limp, sunken-eyed children** who flooded pediatric wards across Botswana during the rainy season in early 2006: They were dying from **diarrhea, a malady that is rarely fatal here**. And few of their mothers were breast-feeding, a practice **once all but universal**.*

After the outbreak was over and at least **532 children had died** — **20 times** the usual toll for diarrhea — a team of U.S. investigators solved **the terrible riddle**.

A decade-long, global push to provide **infant formula** to mothers **with the AIDS virus** had **backfired** in Botswana, leaving children **more vulnerable** to other, more immediately lethal diseases, the U.S. team found after investigating the outbreak at the request of Botswana’s government.

April 2007, FDA recall of Combivir/Ziagen (lamivudine and zidovudine) Tablets, counterfeit labels.

## **ALL “HIV-POSITIVE PATIENTS TO BE TRACKED BY YEARS END:**

April 1, 2007 *HIV patient names to be tracked in all 50 states by year’s end*



(The Associated Press):

*CHICAGO: The names of people infected with HIV will be tracked in **all 50 states** by the end of 2007, marking a **victory** for federal health officials and a **quiet defeat** for AIDS advocates who wanted to keep patients' names out of state databases.*

*Vermont, Maryland and Hawaii, the last states not tracking the names of HIV-positive people, are quickly moving toward adopting names-based surveillance. Eight other states and Washington, D.C., began collecting the names of HIV patients last year, and Massachusetts switched in January.*

*The states are bowing to federal pressure **so they will not lose money for medications and health services** for patients.*

*This is the first year federal funding has been tied to names-based surveillance of HIV. More than \$1.4 billion (€1.05 billion) in federal money will be distributed this fiscal year based on new formulas that include **numbers of people with HIV** counted by states using names. In some states, including Illinois, **millions of dollars** are at stake.*

*That is why advocates say they have quit fighting — although they still worry that collecting names **will deter some people from getting tested and seeking treatment**, and about the possibility of names being released due to security breaches.*

*“I have patients who are very high-profile individuals — physicians in practice, people who are politicians” who don't want their real names reported, said Dr. Dan Berger, medical director of NorthStar Healthcare in Chicago's Lincoln Park.*

*In a 2005 security breach in Palm Beach County, Florida, **the names of 6,500 HIV and AIDS patients were mistakenly e-mailed to 800 county health workers**. Other security breaches have occurred in California and Kentucky.*

*Some worry that names-based reporting could have the greatest effect on whether minorities and the poor get tested and treated because they may be less likely to **trust** the government to keep their names **secret**.*

*In a low-income Chicago neighborhood 10 miles from Berger's office, **patients now are told they must release their names** to the state to get medications. Bruce Jackson, director of **the Gift House**, which **offers** HIV testing and counseling in southwest Chicago, said some clients are apprehensive, fearing their families or friends will find out they're **infected**.*

*Reporting names “can affect if (disadvantaged people) come back for care and it can affect how they describe to other people their experience of getting tested,” said Catherine Hanssens of New York's Center for HIV Law and Policy.*

*There are an estimated **40,000** new HIV infections annually in the United States.*

*Methods of tracking cases varied from state to state until recently. Some states, including Illinois, and the city of Philadelphia previously tracked HIV with identifying codes that preserved anonymity and were unique to each patient.*

*The U.S. Centers for Disease Control and Prevention rejected code-based systems after finding they could lead to **double-counting** and were cumbersome for health care providers. The CDC announced its support for names-based HIV reporting in 1999, and strengthened that **to a recommendation** in 2005.*

*“After many evaluations of code-based systems, it became clear that those systems do not meet CDC standards for HIV data,” said Dr. Timothy Mastro, deputy director of the Division for HIV/AIDS Prevention at the CDC. Diseases such as syphilis, tuberculosis and AIDS already were tracked by patient names, he said, making HIV the exception.*

Starting this fiscal year, the CDC's HIV numbers were used, along with AIDS case numbers, by the U.S. Department of Health and Human Services **to calculate funding** to cities and states **receiving formula grants through the Ryan White CARE Act**, the government's largest HIV/AIDS program. The formulas include only HIV data from states using names-based surveillance.

AIDS advocates, who argued against collecting names of HIV patients in the 1990s, preferred code-based systems to protect the confidentiality of patients and said the CDC ignored evidence that codes could work.

"I've not so much changed my opinion as surrendered," said Ron Johnson, deputy executive director of Aids Action in Washington, D.C. "I still believe code-based reporting is valid and is preferable for HIV reporting. It, for all practical purposes, has become a losing battle." For now, public health officials are trying to reassure people who test positive for HIV that stiff security measures protect state databases of names.

In Illinois, staff members handling names take an oath of confidentiality and get special training. The names are in a stand-alone computer system, **behind locked doors**. "I've never been in that room where they're kept. The security is that tight," said Tom Hughes, a deputy director with the Illinois Department of Public Health.

Participating states strip names and other identifying information from their HIV reports before transmitting them in an encrypted form to the CDC, Hughes and other health officials said. **Berger, the Chicago doctor, said he has told some patients not to give him their real names.** He has enrolled patients anonymously in medical studies of HIV drugs without reporting their names, he said.

Health officials said **it is impossible to predict** how many doctors and patients are finding ways of keeping names out of databases, and whether HIV case numbers will show any decline that can be connected to names-based reporting.

With **better drugs** forestalling the progression of HIV to AIDS, people with **no apparent symptoms** face knowing their names will be on a state list for decades — protected by security measures, but nevertheless subject to exposure.

**"In many ways, it's a different world today than the world that motivated people to insist on anonymous systems for tracking HIV,"** said Suzanne Goldberg, director of the Sexuality and Gender Law Clinic at Columbia Law School in New York. "A lot has changed, but unfortunately not enough."

## **EIGHTEEN TO EIGHTY-PERCENT HAVE RESISTANT "HIV" TO FIRST LINE TREATMENT IN CHINA: A WINDOW OF OPPORTUNITY.**

In May 3, 2007. Bates Gill, Ph.D., and Susan Okie, M.D. published an article regarding "AIDS" in China in New England Journal of Medicine (Volume 356:1801-1805. Number 18) which claimed that *"the rate of resistance to first-line treatment was 18% in one small Chinese study but 45 to 80% in separate cohorts in another study:"*

### **China and HIV — A Window of Opportunity**

*Relying almost entirely on generic drugs produced in China, the country's first-line therapy regimens — zidovudine, didanosine, and nevirapine or zidovudine, stavudine, and nevirapine — have severe side effects, raising concerns about adherence and the emergence of drug resistance.*"The rate of resistance to first-line treatment was 18% in one small Chinese study but 45 to 80% in separate cohorts in another study.

## **THE SUCCESS OF MATHEMATICAL MODELING OF "THE AIDS EPIDEMIC."**

Saturday, 23 June 2007.

## **HIV infection theory** challenged

*T cells are lost at a slow rate. A longstanding theory of how HIV slowly depletes the body's capacity to fight infection **is wrong**, scientists say. HIV attacks human immune cells, called T helper cells. Loss of these cells is gradual, often taking many years.*

*It was thought infected cells produced more HIV particles and that this caused the body to activate more T cells which in turn were infected and killed. Modelling by UK and US researchers suggests that, **if that was true, cells would die out in months not years.***

*The study, led by Emory University in Atlanta and the Institute of Child Health in London, was published in the journal PLoS Medicine. **If the specific process by which HIV depletes this kind of white blood cell can be identified, it could pave the way for potential new approaches to treatment.** Professor Jaroslav Stark*

The researchers used a **mathematical model** of the processes by which T cells are produced and eliminated. Using this they showed that the current theory of an uncontrolled cycle of T cell activation, infection, HIV production and cell destruction – dubbed the **“runaway” hypothesis** – was flawed.

*They concluded that it could not explain the very slow pace of depletion that occurs in HIV infection. If the theory were correct, then T helper cell numbers would fall to very low levels over a number of months, not years.*

## **Lack of certainty**

*Researcher Professor Jaroslav Stark, from Imperial College London, said: “Scientists have never had **a full** understanding of the processes by which T helper cells are depleted in HIV, and therefore they’ve been unable to **fully** explain why HIV destroys the body’s supply of these cells at such a slow rate.*

*Our new interdisciplinary research has thrown serious doubt on one popular theory of how HIV affects these cells, and means that **further studies are required** to understand the mechanism behind HIV’s **distinctive slow process** of cellular destruction.*

*The researchers think one possible explanation could be that the virus slowly **adapts itself** over the course of the **infection**. But they stress that further analysis is needed to verify this alternative theory. Professor Stark said: “If the specific process by which HIV depletes this kind of white blood cell can be identified, it **could** pave the way for potential new approaches to treatment.”*

*Roger Pebody, a treatment advisor at HIV charity Terrence Higgins Trust, said: “**HIV is an incredibly complex virus** and research is ongoing to try and establish exactly how it works. We need **more studies** in this area before we can draw any **clear** conclusions.*

**21 MEDICS AND DOCS FOUND GUILTY OF KILLING 10 CHILDREN AND INFECTING 1198 CHILDREN AND INFANTS WITH “HIV:” ALL EXCEPT THE MINISTER OF HEALTH ARE GUILTY!**

## **2007, June 27. Kazakh HIV medics found guilty**

*Not all of the accused were given jail sentences. A court in the Central Asian state of Kazakhstan has found 21 medical workers guilty of causing an HIV outbreak which has so far killed 10 children. At least **119 children and babies** contracted **the virus***

after receiving **treatment** in hospitals in Shymkent.

The judge said that **the accused had acted recklessly**, and that corruption and malpractice led to **the outbreak**. The HIV outbreak was first discovered last year, but the number of cases is still rising. The night before the verdict, another child died. He was two years old.

This trial is over but the Shymkent HIV problem is not, says the BBC's Natalia Antelava in the town.

### **Unnecessary transfusions**

The judge announced that all 21 medical workers on trial were guilty. But for each defendant he announced a different punishment. Medical workers accused of trading illegal blood were sentenced to eight years in prison. Several doctors were sentenced from three to five years. But the former head of the regional health department and four of her deputies had their sentences suspended.

Mothers of the victims wailed and shouted as they heard that one woman, who many local people hold responsible for the outbreak, would not be jailed. Many said that this was not the kind of justice they were hoping for, and added that they would appeal.

An investigation into the outbreak found that many children had unnecessary and often multiple blood transfusions.

**Medical equipment was often not sterilised properly.** One boy, who is now aged two, contracted the virus after receiving a blood transfusion prescribed **to treat pneumonia**. The prosecutors alleged that the doctors were selling blood to make money. It is unclear why the suspected infected transfusions affected only children.

### **NEJM SAYS 99% OF RESIDENT STUDENT DOCTORS HAVE HAD NEEDLE-STICK INJURY, AND 53% OF THEM INVOLVED "HIGH RISK" SINNERS.**

**June 28, 2007.** New England Journal of Medicine documented the numbers of needle-stick injuries among student health care workers (Needlestick Injuries among Surgeons in Training. Martin A. Makary et al., M.D., M.P.H., Volume 356:2693-2699):

#### **ABSTRACT**

*Background* Surgeons in training are at high risk for needlestick injuries. The reporting of such injuries is a critical step in initiating **early** prophylaxis or treatment.

*Methods* We surveyed surgeons in training at 17 medical centers about previous needlestick injuries. Survey items inquired about whether the most recent injury was reported to an employee health service or involved a "**high-risk**" patient (i.e., one with a history of infection with human immunodeficiency virus, hepatitis B or hepatitis C, or injection-drug use); we also asked about the perceived cause of the injury and the surrounding circumstances.

*Results* The overall response rate was 95%. Of 699 respondents, 582 (83%) had had a needlestick injury during training; the mean number of needlestick injuries during residency increased according to the postgraduate year (PGY): PGY-1, 1.5 injuries; PGY-2, 3.7; PGY-3, 4.1; PGY-4, 5.3; and PGY-5, 7.7. **By their final year of training, 99% of residents had had a needlestick injury; for 53%, the injury had involved a high-risk patient.** Of the most recent injuries, 297 of 578 (51%) were not reported to an employee health service, and 15 of 91 of those involving high-risk patients (16%) were not reported. **Lack of time**

was the most common reason given for not reporting such injuries among 126 of 297 respondents (42%). If someone other than the respondent knew about an unreported injury, that person was most frequently the attending physician (51%) and least frequently a “significant other” (13%).

*Conclusions* Needlestick injuries are common among surgeons in training and are often not reported. Improved prevention and reporting strategies are needed to increase occupational safety for surgical providers.

#### *Source of Information*

*From the Center for Outcomes Research, Department of Surgery (M.A.M., C.G.H., J.B.S., D.S., M.M.G., P.J.P.), the Quality and Safety Research Group, Department of Anesthesiology and Critical Care Medicine (M.A.M., C.G.H., J.B.S., D.S., P.J.P.), and the Division of Infectious Diseases, Department of Medicine (M.S.S.), Johns Hopkins University School of Medicine; and the Department of Health Policy and Management, Johns Hopkins Bloomberg School of Public Health (M.A.M., J.B.S., P.J.P.) — all in Baltimore; and the Department of Plastic Surgery, Georgetown University School of Medicine, Washington, DC (A.A.).*

### **DANGEROUS CHEMICAL FOUND IN VIRACEPT.**

In **July, 2007**, and article appeared: “Low-Key **Recall** of AIDS Drug Hits World’s Poor,” by Elisabeth Rosenthal:

ROME, July 21 – A total recall of **an important AIDS drug widely used in developing countries** has disrupted treatment for tens of thousands of the world’s poorest patients, with no clear word from the manufacturer on when shipments **will resume**.

The recall of the drug, Viracept, by Roche Pharmaceuticals of Switzerland, went largely unnoticed in the developed world when it was announced in early June, after the company had discovered that some batches made at its Swiss plant contained a dangerous chemical. But the recall has caused growing concern among global health officials and in AIDS programs in many poor nations. They say the company did an inadequate job of informing patients and officials about the potential risks and helping them find affordable access to newer alternative drugs.

### **MICROCHIPS MULLED FOR HIV CARRIERS IN INDONESIA’S PAPUA**

**July 24, 2007** 03:57 AM US/Eastern

[http://www.breitbart.com/article.php?id=070724075657.4w2f978g&show\\_article=1](http://www.breitbart.com/article.php?id=070724075657.4w2f978g&show_article=1)

*Lawmakers in Indonesia’s Papua are mulling the selective use of chip implants in HIV carriers to monitor their behaviour in a bid to keep them from infecting others, a doctor said Tuesday.*

*John Manangsang, a doctor who is helping to prepare a new healthcare regulation bill for Papua’s provincial parliament, said that unusual measures were needed to combat the virus.*

*“We in the government in Papua have to think hard on ways to provide protection to people from the spread of the disease,” Manangsang told AFP.*

*“Some of the infected people experience a change of behaviour **and can turn more aggressive** and would not think twice of infecting others,” he alleged, saying lawmakers were considering **various sanctions** for these people.*

*“Among one of the means being considered is **the monitoring** of those infected people who can pose a danger to others,”*

Manangsang said.

*“The use of chip implants is one of the ways to do so, but only **for those few who turn aggressive** and clearly continue to disregard what **they know** about the disease and spread the virus to others,” he said.*

*A decision was still a long way off, he added.*

*The head of the Papua chapter of the National AIDS Commission, Constant Karma, reportedly slammed the proposal as a violation of human rights.*

*“People with HIV/AIDS are not **like sharks** under observation so that they have to be implanted with microchips to monitor their movements,” he told the Jakarta Post on Tuesday.*

*“Any form of identification of people with HIV/AIDS violates human rights.”*

*According to data from Papua’s health office cited by the Post, the province has just over 3,000 people living with HIV/AIDS. Some 356 deaths have been reported. Papua has a population of about 2.5 million.*

### **LESS DEADLY “HIV” IS BETTER KILLER?**

**On July 31, 2007**, a report appeared which described how scientists probe how HIV infection turns into AIDS by devising a new model that somehow could point to better drug targets:

*FRIDAY, Aug. 3 (HealthDay News) — The common scientific wisdom on how HIV infection proceeds to full-blown AIDS **might be wrong**, two U.S. researchers say. They hope that their new insights, if proven, will lead to **exciting new treatment targets** down the line.*

*Working from a complex mathematical model of viral replication and immune cell death, the researchers now suspect that AIDS begins when one especially fast-killing strain of HIV gains the upper hand over a less-lethal, but more prolific, strain. “This throws into question a lot of the notions that have been accepted about the evolution of the virus” within a typical infected human, explained study co-author Dominik Wodarz, associate professor of biology at the University of California, Irvine. He and another researcher, David Levy, of New York University, published their findings in the July 31 issue of the Proceedings of the Royal Society.*

*Since its first recorded appearance nearly three decades ago, **HIV infection has followed the same deadly path: a short, weeks-long period of acute flu-like symptoms** followed by **years** of asymptomatic **dormancy**, and then symptoms of immune system breakdown that herald the emergence of AIDS.*

*But what is it that tips asymptomatic, low-level infection into AIDS?*

*The common dogma among scientists has long been that **various strains of HIV battle a silent war within the body** over time until **the fittest** — defined as the strain that reproduces itself the most — wins. That strain then goes on to overwhelm the body’s immune cells and destroy the host’s defenses against disease.*

*To test that theory, Wodarz and Levy constructed **a complex mathematical model** that took into account two factors about HIV: how fast the various strains replicate and how fast they kill cells (not always the same thing, the researchers noted). They*

also **factored in** human immune system responses to HIV.

What the two scientists found surprised them. According to the **new model**, AIDS **actually begins** when a **less fit variety of HIV wins** the day. This strain kills immune system cells extremely widely and quickly, but, in doing so, also limits the number of copies of itself it can produce. “**It basically kills its own habitat, its house**,” Wodarz explained.

However, because this form of HIV is very good at quickly killing large numbers of immune cells, “once these **less-fit** strains emerge, they can plunge the patient into AIDS,” Wodarz said.

In **many** cases, two or more strains of the virus can co-infect the same immune system cell, he added. If a fast-killing variety is one of those strains, it kills the cell before slower — but better-replicating — versions can go to work making **millions of new viral particles**.

“But without this **ganging up** on the same cell, the killer virus [that leads to AIDS] would go extinct, because evolution would select against it — because it is less fit and replicates less,” Wodarz explained.

That means that — according to **the model** — one way of keeping AIDS at bay might be to **make sure that only one type of HIV invades a cell** at any given time. **Specific cellular mechanisms** do allow a second or third viral particle to enter a cell, and a medicine that thwarted these “**party crashers**” might keep the deadliest form of HIV from ever emerging, Wodarz speculated.

He pointed to wild monkeys that are infected throughout their lives with HIV-like simian immunodeficiency virus (SIV) **but never get sick**. “Some of them have a lot of **the virus**, and it evolves a lot, **but it does not cause AIDS, ever**,” Wodarz said. **He suspects** the monkey’s immune cells **may have** evolved to block secondary viral entry and thereby keep the most dangerous strain of SIV at bay.

**Not everyone is convinced by the new model, however.**

**Dr. Benigno Rodriguez** is assistant professor of medicine at Case Western Reserve University in Cleveland, and a specialist in the evolution of HIV disease. He called Wodarz and Levy’s paper “an interesting concept,” but said it contained a few significant **flaws**. First of all, he said, most of the available data suggests that HIV does get better at forming copies of itself as AIDS progresses. And Rodriguez believes the two scientists have left another important factor out of their model — the fact that most AIDS patients’ immune cells **are not killed off** by the virus **directly** but are destroyed by so-called “**bystander**” mechanisms that accompany AIDS.

“In an individual with advanced disease, if you look at the number of cells that are **actually infected** [with HIV], **we are talking less than 1 percent**,” he said. “But, in reality, that individual may have lost 20, 30, 50 percent of his immune cells.”

Rodriguez also questioned the importance of multiple strains of HIV infecting the same immune cell. “The data that we already have in hand shows that multiple infection is relatively infrequent,” he said.

The bottom line, according to the Cleveland expert: As with any mathematical model, this one needs to be tested out in the laboratory.

Wodarz **agreed** that experimental verification is necessary, but he said mathematical disease models **more often than not prove to be right**. In fact, he said, it was just **such a model that led scientists to discover that HIV never stops**

**evolving in the body** — even during **infection's** years-long asymptomatic phase. “In HIV, mathematical models have led to great progress before,” Wodarz said.

## **NEJM SAYS KAPOSÍ'S IS ASSOCIATED WITH HIGH CD4 COUNT AND LOW VIRAL LOAD:**

2007, September. Publication in the NEJM that Kaposi's sarcoma is associated with **high** CD4 count and **low** viral load (NEJM 357;13 p 1352, Sept. 2007).

## **SUCCESS OF THE MERCK “HIV” VACCINE TO PREVENT DEVELOPMENT OF AIDS AND STIMULATE ANTI-HIV ANTIBODIES AFTER THE STEP TRIAL.**

**In September, 2007**, Merck released the results of its best “HIV” vaccine trial to date (more than 30 had been carried out on record as of 1995):

*In a major setback, one of the leading experimental AIDS vaccines not only failed to prevent test subjects from becoming infected with HIV, but it didn't offer any indication it might delay the onset of full-blown AIDS, which had been a key hope.*

*The collapse of the trial leaves Merck & Co., which had spent a decade developing the vaccine, with **no** remaining prospects in the global hunt for an AIDS immunization. The vaccine was tested in a network **funded by** the National Institutes of Health. “We've been kicked in the teeth,” said Bruce Walker, a veteran AIDS researcher at Harvard University who wasn't involved in the study. Lawrence Corey, a leader of the NIH-funded HIV Vaccine Trials Network, said he was “mourning.”*

*The results are particularly disappointing **because it is widely agreed that only a vaccine could end the epidemic.** Last year, more than four million people world-wide contracted HIV, **the virus that causes AIDS**, and nearly **three million** died, according to **United Nations** estimates. **Almost 40 million** people are currently living with HIV.*

*But researchers cautioned against overreacting. **Merck's vaccine is one of many in or heading into clinical trials**, and different types of vaccines are known to stimulate different kinds of immunity. For example, an experimental immunization now in human trials that was developed by the HIV Vaccine Research Center of the NIH had shown **more-promising results in monkey trials** than did the Merck vaccine.*

*“It isn't the end of the line,” said Mitchell Warren, executive director of the AIDS Vaccine Advocacy Coalition, a New York group advocating prevention. Merck's data “aren't the answers we wanted, **but they will help improve our other vaccine candidates.**”*

*Tuesday, **the trial was stopped early** by independent overseers known as the Data & Safety Monitoring Board. Comparing two groups — those who received the vaccine and those who received a placebo — the overseers determined there was virtually no statistical difference in **infection** rates between them, indicating the vaccine wasn't working. Also, **the amount of HIV in the blood** of those who did get infected, a predictor of how fast a person will get full-blown AIDS, was virtually the same in each group.*

*The ultimate fear among researchers is that the whole theory underlying the Merck vaccine might be flawed, which, if true, could doom an entire class of experimental vaccines.*

*Most **classical vaccines**, such as those against smallpox or polio, **stimulate the body to produce antibodies that ward off infection.** But stimulating antibodies that neutralize a broad range of HIV strains has been notoriously difficult, so*



researchers focused on the other arm of the immune system: killer T-cells, which attack and kill cells that HIV has already infected. Such vaccines have been considered less likely to prevent someone from getting infected; instead, it was hoped they would enable an infected person to suppress the virus and so delay, perhaps for many years, the onset of disease.

“Given that this study was the leading edge” of research on T-cell based HIV vaccines, said Mark Feinberg, vice president for medical affairs and health policy in Merck’s vaccine division, “there was great disappointment.” “There is nothing on the horizon” at Merck, he said. “We don’t have any other vaccine candidates we’ve identified as promising enough to advance into clinical studies.” Dr. Feinberg added that Merck is “committed to finding ways to share information accumulated over two decades to facilitate the broader effort” to develop an AIDS vaccine.

The Merck vaccine did stimulate the immune system’s T-cells — a notable development — but **not in a way that helped infected test subjects control the virus**. Now, researchers will try to figure out why.

Merck’s shares, reflecting downplayed hopes that such an early vaccine would work, **were up 44 cents** to \$51.82 at 4 p.m. Friday in New York Stock Exchange composite trading.

*Warning Is Sent to AIDS Vaccine Volunteers: South Africans Among Recipients Who May Be at Higher Risk of Contracting Virus. October 25, 2007; Thursday, Page A20, By Craig Timberg.*

JOHANNESBURG, Oct. 24 — South African AIDS researchers have begun warning hundreds of volunteers that **a highly touted experimental vaccine they received in recent months might make them more, not less, likely to contract HIV** in the midst of one of the world’s **most rampant epidemics**.

The move stems from the discovery last month that an AIDS vaccine developed by Merck & Co. **might have led to more infections than it averted** among study subjects in the United States and other countries. Among those who received at least two doses of the vaccine, **19** contracted HIV compared with **11** of those given placebos.

## **MORE SUCCESS WITH ANIMAL MODELS OF “HIV/AIDS.**

Although SIV has always been a better model of “HIV, because the chimps that were injected with “HIV” or AIDS patient sera more than 20 years ago never became ill, as they were recently built new retirement homes, at least one group of investigators recently challenged this long-standing belief:

Sodora’s paper provides evidence, using the sooty mangabey SIV natural host, that **virally induced CD4 T-cell depletion, by itself, is not sufficient to induce AIDS in a natural host**. “When we first observed the dramatic CD4 depletion in all the tissues we examined in these monkeys, we were concerned that they might begin to exhibit clinical signs of AIDS,” said Jeffrey Milush, Ph.D., lead author on the paper. “But after more than **six years, we are sure that CD4 depletion by itself** does not necessarily result in progression to AIDS”.

Sodora contributed to a second paper, with senior author Guido Silvestri, M.D., of the University of Pennsylvania. In a study of disease free SIV–infected **sooty mangabeys**, Silvestri proposes that these **African monkeys** preserve immune function **despite a major loss of mucosal CD4 T-cells** as a result of **an evolutionary adaptation** to reduce immune activation in response to virus replication.

The third of the three papers published this week in The Journal of Immunology, with Ivona Pandrea, M.D., Ph.D., and Cristian Apetrei, M.D., Ph.D., from Tulane University as lead authors, shows that a severe loss of **intestinal CD4 T-cells** in another natural

host, the African green monkey, is also **not predictive** of SIV virulence.

## **DOCTORS WITHOUT BORDERS NEEDS MONEY FOR PLUMPYNUT: SAY CHILDREN NEED FOOD, NOT DRUGS:**

**Oct. 21, 2007.** Doctors Without Borders Briefing Paper: Food Is Not Enough: African children need food, not drugs (See the video free at: <http://www.cbsnews.com/stories/2007/10/19/60minutes/main3386661.shtml>).

*Plumpynut is cheap, nutritious and needs no refrigeration. It is saving starving children in the developing world and could save more ... **if there were more of it.***

*You've probably never heard a good news story about malnutrition, but you're about to. Every year, malnutrition kills **five million** children — that's **one child every six seconds**. But now, the Nobel Prize-winning relief group "Doctors Without Borders" says it finally has something that can save millions of these children.*

*It's cheap, easy to make and even easier to use. What is this miraculous cure? As CNN's Anderson Cooper reports, it's a ready-to-eat, vitamin-enriched concoction called "Plumpynut," an unusual name for a food that may just be the most important advance ever to cure and prevent malnutrition.*

*"It's a revolution in nutritional affairs," says Dr. Milton Tectonidis, the chief nutritionist for Doctors Without Borders.*

*"Now we have something. It is like an essential medicine. In three weeks, we can cure a kid that is looked like they're **half dead**. We can cure them **just like an antibiotic**. It's just, boom! It's a spectacular response," Dr. Tectonidis says.*

*"It's the equivalent of penicillin, you're saying?" Cooper asks.*

*"For these kids, for sure," the doctor says.*

*No kids need it more than a group of children 60 Minutes saw in Niger, a desperately poor country in West Africa, where child malnutrition is so widespread that most mothers have watched at least one of their children die.*

*Why are so many kids dying? Because they can't get the milk, vitamins and minerals their young bodies need. Mothers in these villages can't produce enough milk themselves and can't afford to buy it. Even if they could, they can't store it — there's no electricity, so no refrigeration. Powdered milk is useless because most villagers don't have clean water. Plumpynut was designed to overcome all these obstacles.*

*Plumpynut is a remarkably simple concoction: it is basically made of peanut butter, powdered milk, powdered sugar, and enriched with vitamins and minerals. It tastes like a peanut butter paste. It is very sweet, and because of that kids cannot get enough of it.*

*The formula was developed by a nutritionist. It doesn't need refrigeration, water, or cooking; mothers simply squeeze out the paste. Many children can even feed themselves. Each serving is the equivalent of a glass of milk and a multivitamin.*

*To see the impact it's having, 60 Minutes drove for 12 hours from Niger's capital to a remote village, where every week Doctors Without Borders hand out Plumpynut. After sleeping in a field under mosquito nets, Cooper and the team awoke at sunrise to find mothers emerging from the fields. Many had walked for hours in the dark, along treacherous paths, avoiding scorpions, spiders and poisonous snakes.*

Rivers of women flowed into the site and within minutes there were more than a thousand of them, all waiting to get packets or tubs of Plumpynut. In a land where plastic bags are a luxury, they carry the food home in their scarves, their hands, or simply stacked on top of their heads.

“When you see some of these kids they don’t look sick. They don’t look malnourished. They don’t have bloated bellies or little stick arms,” Cooper remarks.

“The ones that we’re used to seeing on TV, that’s the worst of the worst of the worst. It’s the tip of the iceberg. And then below that, there’s the iceberg. So, there’s a whole spectrum of malnutrition,” Dr. Tectonidis says. “And when we go and check these kids, well, they’re way off in height or in weight. They’re way off.”

Niger has become Plumpynut’s proving ground. A daily dose costs about **\$1**; small factories mix it here and in three other African countries. Tectonidis says other companies could make similar products wherever children need them.

“There’s many countries in Africa now saying, ‘We want a factory. We want a factory.’ Well let’s give it to them,” he says. “We just have to focus on these areas. We don’t have to feed the whole world. We have to go for the jugular. Where are they dying? Where are they **wasted**? That’s where we have to intervene. If you feed them well until they’re two or three years old **it’s won**. They’re healthy, they can get a healthy life. **If you miss that window, it’s finished.**”

In Niger, most children need help now during what’s called the “hunger season,” just before the new harvest. Old food supplies have run out and about all that’s left is millet, a basic grain women pound for porridge. But millet doesn’t have enough nutrients to keep kids alive; in America we use it as **birdseed**.

Normally a children’s hospital 60 Minutes visited would have more patients than beds. But now, thanks to Plumpynut, **it has empty beds**. Dr. Susan Shepherd, a pediatrician from Butte, Mont., runs Doctors Without Borders in Niger.

She says children that would have been hospitalized in the past can now be treated at home. “The reason we can do that is because we can give children Plumpynut here in the ambulatory center, and they take a week’s ration home. Moms **treat** their children at home and come back every week for a weight check,” Dr. Shepherd explains.

That’s what Sahia Ibrahim has been doing. **She’s already lost four children to malnutrition**. Now her six-month-old twins, Hassana and Hussein, are malnourished and she’s worried they might die too. So she’s been coming to the hospital for Plumpynut. Hassana, at six months old, weighs only seven pounds. While that’s what a newborn should weigh, the little girl has put on a pound in just a week thanks to Plumpynut.

Children are weighed and measured at the distribution sites. They’re also examined to make sure they don’t have any **serious infections**. **Malnutrition destroys a child’s immune system**, so they’re more **susceptible** to diseases and less capable of recovering from them. “Often these kids aren’t even hungry. It’s the opposite. They are anorexic because of the deficiencies they have. They lose their appetite,” Tectonidis explains.

That’s what happened to Mansour Miko and Maroufee Mazoo. Less than a year old, they had stopped eating and became listless and weak — so weak that when their mothers brought them to get Plumpynut, the nurse put them in a van and sent them straight to the hospital. Three days later however, they were smacking their lips on Plumpynut, almost ready to go home.

“Have you seen kids who were on the brink of death brought back by Plumpynut?” Cooper asks.

“Oh, yeah, for sure. Again and again and again and again,” Dr. Shepherd says.

But not always. Sometimes parents wait too long before bringing their child **to doctors**. 60 Minutes found Rashida Mahmadou in intensive care, barely clinging to life. Rashida’s condition was very serious. Her skin was literally peeling away — **one side effect of malnutrition**, as skin becomes thin, pliable, cracks easily, **and bacteria invade**.

Just two hours later, Rashida’s little heart stopped beating. She was just 19 months old. “She died of severe, acute **malnutrition**,” says Shepherd, who says she sees this happening every day. Asked how she deals with so many kids dying, Shepherd tells Cooper, “It breaks your heart. It can break your spirit. It can ruin your confidence in your ability to be a **good doctor**. And it is sad. And I carry memories of many, many children with me and I’ll carry them with me for my entire life. But you certainly cannot indulge yourself in that kind of sadness. We need to do something about this.”

CBS) If Plumpynut is the answer, how come kids are still dying? “The answer is getting to kids earlier,” Shepherd says. “Once children are as sick as she is, Plumpynut is not gonna save her.”

Rashida was buried in a nearby cemetery. The grave digger, Salifu Ibrahim, told 60 Minutes he used to dig graves for about **seven children a day**, but now, on most days, he digs **only one**. Asked why he thinks fewer children are dying, Ibrahim says, “It is God’s will.” God’s will and Plumpynut.

Two years ago this region had the highest malnutrition rate in Niger. But now, after widespread use of the Plumpynut, it has the lowest. Dr. Shepherd told Cooper they’ll be able to treat more than **120,000** kids this year, up from just **10,000** children three years ago.

What about peanut allergies?

“**We just don’t see it**,” Shepherd says. “In developing countries food allergy is not nearly the problem that it is in industrialized countries. It’s hard to imagine a less industrialized country than Niger. On a list of 177 developing countries, the United Nations ranked Niger dead last — least developed. More than 70 percent of the people don’t know how to **read**. Most work in the fields and earn less than a dollar a day. Nomadic goat herders still roam this land — their children and their kids travel by camel. Goats seem to be the main garbage disposal, but clearly the goats are falling behind. You can still spot a skinny guard dog, but we were told all the cats have been cooked.

In the countryside, where 85 percent of people live, girls start marrying as young as **11** years old. By the age of **15** most are wed, and by **16** most have already become mothers. The average woman here will give birth at least **eight times** in her lifetime. But largely because of malnutrition, one in five of their children will die before they reach the age of five. Of those who survive, **half** will have stunted growth and never reach full adult height.

But now, with Plumpynut, more children are surviving and thriving.” And kids are doing better. Moms say their child’s skin is brighter. Their appetites are better. And they’re less sick. You know, what more could you ask for,” Shepherd remarks.

Doctors Without Borders is asking for more of this type of food. Their success in Niger proves, they say, that fortified ready-to-eat products, like Plumpynut, save children’s lives. Dr. Tectonidis says **if the United States and the European Union were willing to spend part of their food aid on this**, more companies will start making it.

“Even by taking a miniscule proportion of the global food aid budget, they will have a huge impact, huge impact!” Tectonidis says. “**We’re not even asking for billions**. It will solve so much of the underlying useless death. So we gotta do that now.” It’s

useless death,” Cooper remarks.

“Wasted life. Just totally wasted life for nothing. Because they don’t have this product, little a bit of peanut butter with vitamins,” Tectonidis says. “What a waste.

## **CROSS PRESENTATION OF CASPASE-CLEAVED APOPTOTIC SELF-ANTIGENS IN HIV INFECTION**

In a recent study just published in no less a journal than Nature Medicine (**58**), it is claimed that the mechanism of “HIV’s” deadly assault on the human body is that, when cells die they release proteins that MIGHT HAVE a key role in the induction of self-reactive CD8+ T-cell responses. In other words, as cells die, they release certain proteins that other immune cells recognize and attack. As this is a generalized mechanism the body uses to clear out dying or dead cells, “HIV’s” molecular signature would not be in any way specific or unique in its ability to trigger such reactions. In fact, the recent claim, if true, suggests that the “HIV=AIDS” theory is wrong and should be rejected.

*We found that the proteome of apoptotic T cells includes prominent fragments of cellular proteins generated by caspases and that a high proportion of distinct T cell epitopes in these fragments is recognized by CD8+ T cells during HIV infection. The frequencies of effector CD8+ cells that are specific for apoptosis-dependent epitopes correlate with the frequency of circulating apoptotic CD4+ T cells in HIV-1-individuals. We propose that these self-reactive effector CD8+ cells may contribute to the systemic immune activation during chronic HIV infection. The caspase-dependent cleavage of proteins associated with apoptotic cells has a key role in the induction of self-reactive CD8+ T cell responses, as the caspase-cleaved fragments are efficiently targeted to the processing machinery and are cross-presented by dendritic cells. These findings demonstrate a previously undescribed role for caspases in immunopathology.*

## **“HIV” REALLY NEEDS 237 CELLULAR PROTEINS TO DO THE JOB! BACK TO THE IMPORTANCE OF THE HOST.**

January 10, 2008 A team funded by the Howard Hugues medical institute publishes that “HIV” needs more than 200 host cell proteins to carry out its life cycle, signaling a coming full circle back to the organism and cell, as opposed to the putative “virus.” This gentle shift in emphasis on the host has been what is needed to return the question of immune suppression back to the host instead of the suspected microbe. As if science itself is self-correcting, such a radical departure now of course on the one hand frees the AIDS establishment to apply for and get at least 237 new types of grants (one could target each of these proteins one at a time until you found the essential one or perhaps several). On the other hand, it places the onus on “tweaking” host cells to somehow regulate one or many of these so-called “cellular provided proteins,” instead of on killing “the virus” with toxic meds:

### ***Host Cell Proteins Could be HIV’s Achilles’ Heel***

*Like all viruses, the human immunodeficiency virus (HIV) depends on its host cell to do much of its molecular **dirty work** as it promotes **infection**. Now, in a paper published online January 10, 2008, in Science Express, a team of researchers from the Howard Hughes Medical Institute (HHMI) has provided a detailed picture of just how extensively HIV exploits host cells’ proteins.*

*The new study, conducted by a team led by HHMI investigator Stephen J. Elledge of Harvard Medical School, identifies 273 host proteins that serve to keep the AIDS virus **healthy and happy** as it infects cells. **More than 200 of these were not previously known** to be needed by the virus during its life cycle. This new catalog of proteins could help researchers devise better treatment strategies to get around HIV’s **notorious propensity to develop resistance** to antiviral drugs.*

*“We wanted to get this information out to the field. We anticipate it will have a big impact — as what we’ve really done is provide a **hypothesis-generation set**. It will allow people to think **more deeply** about the **life** cycle of HIV and how to impede it.”*

Elledge and Abraham Brass, a post-doctoral fellow in his lab, collaborated with Judy Lieberman and other Harvard Medical School scientists on the study. The hope, Elledge said, is that their data will give scientists a more complete picture **of just how complicit the host cell is in the viral life cycle**. “We wanted to get this information out to the field,” Elledge said, noting that pharmaceutical companies are doing similar work privately. “We anticipate it will have a big impact — as what we’ve really done is provide a hypothesis-generation set. It will allow people to think more deeply about the life cycle of HIV and how to impede it.”

What’s more, the approach utilized by Elledge’s group **could be** used to expose previously hidden vulnerabilities in **other** viruses, such as **polio** or **hepatitis**, which might even rely on some of **the same** proteins identified in the new [“HIV” retroviral-emphasis mine] screen.

Because **the AIDS virus** makes only **a handful** of its own proteins, it must co-opt host cell proteins for successful infection. A key advantage to using these host proteins as therapeutic targets, Elledge emphasized, is that they confer essential abilities that the **AIDS virus** has not managed to accomplish on its own. “**If we could make drugs to host cell proteins**, they are unlikely to **be overcome** by HIV. **The virus would have to evolve a new ability**, and that’s not **very likely**.”

To identify the essential proteins, Elledge’s group used a technique known as **RNA interference** to turn off genes in human cells. They began their experiments by shutting off genes four at a time, then narrowed their focus to individual genes. They then exposed these cells to the AIDS virus to see how HIV fared in the absence of each host cell protein.

“There are a lot of proteins in cells that HIV doesn’t care about,” Elledge noted. “But it cares about some of them a lot. We knocked out known proteins one at a time to see which were required.” What they found was that 273 host proteins aided the virus in its **infection process**.

According to Elledge, the host cell proteins commandeered by HIV help the virus accomplish an array of critical tasks. Some of the host proteins implicated by the HHMI team abet the virus by helping it gain access to the cell, integrate into the cell’s genome, replicate, and exit the cell to renew the viral life cycle by infecting more cells.

“These proteins participate in a broad array of cellular functions and implicate new pathways in the viral life cycle,” according to Elledge. “Some of the proteins had been previously implicated in HIV biology, but had never been demonstrated to be required (by the virus).” Of the proteins identified by Elledge and his colleagues, 237 had never before been implicated as accomplices of HIV.

Many of the host proteins Elledge and his colleagues zeroed in on are more abundant in immune cells than other types of human cells. **The data help explain why HIV is so insidious**, taking over the very immune cells the body needs to thwart infection. “It suggests immune cells are good hosts because they have lots of the proteins HIV needs to function.”

Further work could offer new avenues to treating viral infections of all kinds, Elledge said. “We expect other people will be able to do similar work with other viruses. **A lot of viruses** have host factors that they need.”

**ABACAVIR AND DIDANOSINE INCREASE THE RISK OF HEART ATTACK IN ARV-TREATED PATIENTS BY 49% TO 90%:**

2008 FEBRUARY 7. <sup>12</sup>

A study to assess the adverse effects of anti-retroviral drugs shows that two widely-used HIV drugs are associated with an increased risk of heart attack/the formation of blood clots in the heart. With the use of Didanosine, the risk of developing a heart attack increases by **49%**, with Abacavir; the increased risk is **90%**. The effect is most pronounced in patients **with a high**

**underlying cardiovascular risk.** The research findings also show that the adverse effect is reversible, if patients discontinue use of these particular drugs.

*The side-effects associated with Didanosine and Abacavir are, naturally, most significant for HIV-infected patients who already have a high underlying cardiovascular risk. The drug effect increases an individual persons underlying risk by a factor of 1.9 for a person on Abacavir, and 1.49 for a person on Didanosine. For a person with a low underlying risk, this increase in risk is still **negligible**, but for someone with a high underlying risk, this could have serious consequences. The study shows, however, that **the risk of heart attack is removed once patients stop taking the drugs.** This seems to be the case, regardless of how long these drugs have been used by patients.*

## **TH-1/TH-2 RATIO IMBALANCE IS THE PRIMARY MECHANISM OF AIDS: JULIANA SACHER'S AND HEINRICH KREMER'S EXPERIENCE**

Dr. Juliane Sacher and her colleagues published three therapeutic studies conducted in connection with German drug rehabilitation clinics, following what they proposed many years ago, that the immune cell Th2/Th1 ratio is imbalanced in "AIDS" patients, and a consequent hypergammaglobulinemia characteristic of "HIV's" molecular signatures result. Although the information about these observations were suppressed and funding for the studies was withdrawn by the German government without explanation after early enthusiasm for the progress of Sacher and others (Juliane Sacher, Raum and Zeit, 2006), Dr. Sacher and her colleagues claim to have been curing AIDS patients with glutathione, alpha-lipoic acid, patient-specific amino acid profile restoration as well as vitamin, mineral, and trace element restoration in a patient-specific manner for 20 years. Also, cysteine, Ginkgo, proteolytic enzymes, mild aerobic exercise, and other non-toxic means also have been employed successfully in these German drug clinics for 20 or more years to reverse immune suppression, "AIDS," and "HIV-disease."

According to Dr. Sacher and colleagues, in these therapeutic trials, HAART has only occasionally been given for short periods of time successfully in some of the profound immune suppressed cases, because this deadly toxic cocktail antagonizes the imbalanced proliferation (disturbed TH1/TH2 ratio) of these cells, because they are rank cytotoxic poisons. Both in non-symptomatic "HIV" patients and in some profoundly suppressed "AIDS patients," HAART has the ability to kill replicating cells and dampen the molecular markers that these imbalanced sets of cells generate (and which are read as high viral load although no virus particles have been photographed or isolated). At higher doses HAART may antagonize raging bacterial infections, protozoa, and fungal infections. However, if given chronically, it is now well established that HAART will eventually wipe the immune system out and render the patient anergic and bone-marrow depressed, not to mention the toxic effects of the HAART regimens given chronically exert on the intestines, platelets, and other tissues that lead to mal-absorption disorders, neuropathies, and lypodystrophies, heart failure, and liver destruction. Dr. Sacher and her colleagues essentially have shown that relatively gentle treatments described above can in most cases reverse "AIDS-indicator illnesses," far more reliably, and completely than HAART.

Although the funding of Dr. Sacher, and her colleagues was abruptly ended without explanation years ago, even the AIDS Establishment now sees the merits of their approach. For example, one of the staunchest promoters of "HIV=AIDS" recently wrote:

*Immune Activation in HIV Infection More than Just Markers*

*By Richard Jefferys*

*"At the recent International AIDS Society conference in Sydney, Mike Lederman reminded attendees that abnormally high levels of immune activation were described in the first case reports of gay men with AIDS in 1981. The authors of those reports, led by Michael Gottlieb, specifically noted the "increased percentage of cells bearing the thymocyte-associated antigen T10." This antigen is now known as CD38, and an extensive literature—particularly the work of the late Janis Giorgi, an immunologist at UCLA—*

demonstrates that CD38 expression on CD8 T cells correlates strongly with the rate of disease progression in people with HIV infection” (in many instances, more strongly than viral load and peripheral blood CD4 T cell counts).

## **COCHRANE DATABASE INDICATES ARV’S DON’T BLOCK MOTHER TO CHILD TRANSMISSION IN MOST STUDIES:**

*Antiretrovirals for reducing the risk of mother-to-child transmission of HIV infection.*

*Volmink J, Siegfried NL, van der Merwe L, Brocklehurst P. <sup>13</sup>*

*SELECTION CRITERIA: Randomised controlled trials of any antiretroviral regimen aimed at decreasing the risk of mother-to-child transmission of HIV infection compared with placebo or no treatment. DATA COLLECTION AND ANALYSIS: Two authors independently selected relevant studies, extracted data and assessed trial quality. For the primary outcomes, we used survival analysis to estimate the probability of infants being infected with HIV (the observed proportion) at various specific time-points and calculated efficacy at a specific time as the relative reduction in the proportion infected. Efficacy, at a specific time, is defined as the preventive fraction in the exposed group compared to the reference group, which is the relative reduction in the proportion infected:  $1-(Re/Rf)$ . For those studies where efficacy and hence confidence intervals were not calculated, we calculated the approximate confidence intervals for the efficacy using recommended methods. For analysis of results that are not based on survival analyses we present the relative risk for each trial outcome based on the number randomised. No meta-analysis was conducted as no trial assessed the identical drug regimens.*

*MAIN RESULTS: **Eighteen trials** including **14,398** participants conducted in 16 countries were eligible for inclusion in the review. The first trial began in April 1991 and assessed zidovudine (ZDV) versus placebo and since then, the type, dosage and duration of drugs to be compared has been modified in each subsequent trial. Antiretrovirals versus placebo In breastfeeding populations, three trials found that:*

*ZDV given to mothers from 36 to 38 weeks gestation, during labour and for 7 days after delivery significantly reduced HIV infection **at 4-8 weeks** (Efficacy 32.00%; 95% CI 0.64 to 63.36), 3 to 4 months (Efficacy 34.00%; 95% CI 6.56 to 61.44), 6 months (Efficacy 35.00%; 95% CI 9.52 to 60.48), 12 months (Efficacy 34.00%; 95% CI 8.52 to 59.48) and 18 months (Efficacy 30.00%; 95% CI 2.56 to 57.44). ZDV given to mothers from 36 weeks gestation and during labour significantly reduced HIV infection at 4 to 8 weeks (Efficacy 44.00%; 95% CI 8.72 to 79.28) and 3 to 4 months (Efficacy 37.00%; 95% CI 3.68 to 70.32) **but not at birth.***

*ZDV plus lamivudine (3TC) given to mothers from 36 weeks gestation, during labour and for 7 days after delivery and to babies for the first 7 days of life (PETRA ‘regimen A’) significantly reduced HIV infection (Efficacy 63.00%; 95% CI 41.44 to 84.56) and a combined endpoint of HIV infection or death (Efficacy 61.00%; 95% CI 41.40 to 80.60) at 4 to 8 weeks **but these effects were not sustained at 18 months.***

*ZDV plus 3TC given to mothers from the start of labour until 7 days after delivery and to babies for the first 7 days of life (PETRA ‘regimen B’) significantly reduced HIV infection (Efficacy 42.00%; 95% CI 12.60 to 71.40) and HIV infection or death at 4 to 8 weeks (Efficacy 36.00%; 95% CI 8.56 to 63.44) **but the effects were not sustained at 18 months.***

*ZDV plus 3TC given to mothers **during labour only** (PETRA ‘regimen C’) with no treatment to babies **did not reduce the risk of HIV infection** at either 4 to 8 weeks or 18 months. In non-breastfeeding populations, three trials found that: ZDV given to mothers from 14 to 34 weeks gestation and during labour and to babies for the first 6 weeks of life significantly reduced HIV infection in babies at 18 months (Efficacy 66.00%; 95% CI 34.64 to 97.36).*



ZDV given to mothers from 36 weeks gestation and during labour with no treatment to babies ('Thai-CDC regimen') significantly reduced HIV infection at 4 to 8 weeks (Efficacy 50.00%; 95% CI 12.76 to 87.24) **but not at birth**. ZDV given to mothers from 38 weeks gestation and during labour with no treatment to babies **did not influence HIV transmission at 6 months**.

### **Longer versus shorter regimens using the same antiretrovirals**

One trial in a breastfeeding population found that: ZDV given to mothers during labour and to their babies for the first 3 days of life compared with ZDV given to mothers from 36 weeks and during labour (similar to 'Thai-CDC') resulted in **HIV infection rates that were not significantly different** at birth, 4-8 weeks, 3 to 4 months, 6 months and 12 months.

Three trials in non-breastfeeding populations found that: ZDV given to mothers from 28 weeks gestation during labour and to infants for the first 3 days after birth compared with ZDV given to mothers from 35 weeks gestation through labour and to infants from birth to 6 weeks significantly reduced HIV infection rate at 6 months (Efficacy 45.00%; 95% CI 1.88 to 88.12) but compared with the same regimen ZDV given to mothers from 28 weeks gestation through labour and to infants from birth to 6 **weeks did not result in a statistically significant difference in HIV infection at 6 months**.

ZDV given to mothers from 35 weeks gestation during labour and to infants for the first 3 days after birth **was considered ineffective for reducing transmission rates and this regimen was discontinued**.

An antenatal/intrapartum course of ZDV used for a median of 76 days compared with an antenatal/intrapartum ZDV regimen used for a median 28 days with no treatment to babies in either group **did not result in HIV infection rates that were significantly different at birth and at 3 to 4 months**.

In a programme where mothers were routinely receiving ZDV in the third trimester of pregnancy and babies were receiving one week of ZDV therapy, a single dose of nevirapine (NVP) given to mothers in labour and to their babies soon after birth compared with a single dose of NVP given to mothers only resulted in HIV infection rates that **were not significantly different at birth and 6 months**. However the reduction in risk of HIV infection or death at 6 months was **marginally significant** (Efficacy 45.00%; 95% CI -4.00 to 94.00).

Antiretroviral regimens using different drugs and durations of treatment In breastfeeding populations, three trials found that: A single dose of NVP given to mothers at the onset of labour plus a single dose of NVP given to their babies immediately after birth ('HIVNET 012 regimen') compared with ZDV given to mothers during labour and to their babies for a week after birth resulted in lower HIV infection rates at 4-8 weeks (Efficacy 41.00%; 95% CI 11.60 to 70.40), 3-4 months (Efficacy 39.00%; 95% CI 11.56 to 66.44), 12 months (Efficacy 36.00%; 95% CI 8.56 to 63.44) and 18 months (Efficacy 39.00%; 95% CI 13.52 to 64.48). In addition, the NVP regimen significantly reduced the risk of HIV infection or death at **4-8 weeks** (Efficacy 42.00%; 95% CI 14.56 to 69.44), 3 to 4 months (Efficacy 40.00%; 95% CI 14.52 to 65.48), 12 months (Efficacy 32.00%; 95% CI 8.48 to 55.52) and 18 months (Efficacy 33.00%; 95% CI 9.48 to 56.52). The 'HIVNET 012 regimen' plus ZDV given to babies for 1 week after birth compared with the 'HIVNET 012 regimen' alone **did not result in a statistically significant difference in HIV infection at 4 to 8 weeks**.

A single dose of NVP given to babies immediately after birth plus ZDV given to babies for 1 week after birth compared with a single dose of NVP given to babies only significantly reduced the HIV infection rate **at 4 to 8 weeks** (Efficacy 37.00%; 95% CI 3.68 to 70.32).

Five trials in non-breastfeeding populations found that: In a population in which mothers were receiving 'standard' ARV for HIV infection a single dose of NVP given to mothers in labour plus a single dose of NVP given to babies immediately after birth ('HIVNET 012 regimen') compared with **placebo did not result in a statistically significant difference in HIV infection**

**rates at birth and at 4 to 8 weeks.**

*The ‘Thai CDC regimen’ compared with the ‘HIVNET 012 regimen’ **did not result in a significant difference in HIV infection at 4 to 8 weeks.***

*A single dose of NVP given to babies immediately after birth compared to ZDV given to babies for the first 6 weeks of life **did not result in a significant difference in HIV infection rates at 4-8 weeks and 3 to 4 months.***

*ZDV plus 3TC given to mothers in labour and for a week after delivery and to their infants for a week after birth (similar to ‘PETRA regimen B’) compared with NVP given to mothers in labour and immediately after delivery plus a single dose of NVP to their babies immediately after birth (similar to ‘HIVNET 012 regimen’) **did not result in a significant difference in the HIV infection rate at 4 to 8 weeks.***

**KEVIN DE COCK SAYS WORLD’S HETEROSEXUAL AIDS EPIDEMIC OVER, EXCEPT IN AFRICA.**

2008, May. WHO: No AIDS pandemic for heterosexuals, from UK’s Independent *By Jeremy Laurance, Sunday, 8 June 2008.*

The opening day of the 61st World Health Organisation (WHO) assembly on May 19, 2008 in Geneva.

*Threat of world Aids pandemic among heterosexuals is over, report admits*

***A 25-year health campaign was misplaced outside the continent of Africa. But the disease still kills more than all wars and conflicts***

*A quarter of a century after the outbreak of Aids, the World Health Organisation (WHO) has accepted that **the threat of a global heterosexual pandemic has disappeared.***

*In the first official admission that **the universal prevention strategy promoted by the major Aids organisations may have been misdirected**, Kevin de Cock, the head of the WHO’s department of HIV/Aids said **there will be no generalised epidemic of Aids in the heterosexual population outside Africa.***

*Dr De Cock, **an epidemiologist** who has spent much of his career leading the battle against the disease, said **understanding of the threat posed by the virus had changed. Whereas once it was seen as a risk to populations everywhere, it was now recognised that, outside sub-Saharan Africa, it was confined to high-risk groups including men who have sex with men, injecting drug users, and sex workers and their clients.***

*Dr De Cock said: “**It is very unlikely there will be a heterosexual epidemic in other countries.** Ten years ago a lot of people were saying there would be a generalised epidemic in Asia – China was the big worry with its huge population. That doesn’t look likely. But we have to be careful. As an epidemiologist it is better to describe what we can measure. There could be small outbreaks in some areas.”*

*In 2006, the Global Fund for HIV, Malaria and Tuberculosis, which provides 20 per cent of all funding for Aids, warned that Russia was on the cusp of a **catastrophe**. An **estimated 1 per cent** of the population was **infected**, mainly through injecting drug use, the same level of infection as in South Africa in 1991 where the prevalence of the infection has since risen to **25 per cent.***

Dr De Cock said: "I think it is unlikely there will be extensive heterosexual spread in Russia. But clearly there will be some spread."

...

***Aids organisations, including the WHO, UN Aids and the Global Fund, have come under attack for inflating estimates of the number of people infected, diverting funds from other health needs such as malaria, spending it on the wrong measures such as abstinence programmes rather than condoms, and failing to build up health systems.***

***Dr De Cock labelled these the "four malignant arguments" undermining support for the global campaign against Aids, which still faced formidable challenges, despite the receding threat of a generalised epidemic beyond Africa.***

***Any revision of the threat was liable to be seized on by those who rejected HIV as the cause of the disease, or who used the disease as a weapon to stigmatise high risk groups, he said...***

***Critics of the global Aids strategy complain that vast sums are being spent educating people about the disease who are not at risk, when a far bigger impact could be achieved by targeting high-risk groups and focusing on interventions known to work, such as circumcision, which cuts the risk of infection by 60 per cent, and reducing the number of sexual partners.***

***There were "elements of truth" in the criticism, Dr De Cock said. "You will not do much about Aids in London by spending the funds in schools. You need to go where transmission is occurring. It is true that countries have not always been good at that."***

#### **NEJM SAYS ABRUPT WEANING OF BREAST FED INFANTS DOESN'T IMPROVE HEALTH OR SURVIVAL AND IS HARMFUL FOR "HIV-POSITIVE" INFANTS:**

2008 July 10. New England Journal of Medicine publishes an article by Kuhn et al., entitled "Early, Abrupt Weaning on "HIV" survival of children in Zambia (NEJM 359:2, July 10 2008), which concludes:

***Early, abrupt cessation of breast-feeding by HIV-infected women in a low-resource setting, such as Lusaka, Zambia, does not improve the rate of HIV-free survival among children born to HIV-infected mothers and is harmful to HIV-infected infants.*** (ClinicalTrials.gov number, NCT00310726.)

#### **ABBOTT LABS IS ATTACKED BY "DOGS" BECAUSE THEY ROBBED "AIDS" PATIENTS:**

**2008, August 04** Chicago Tribune.

*More AIDS drug suits dog Abbott.*

*By Paul Elias*

***SAN FRANCISCO — When Abbott Laboratories raised the price of a popular AIDS drug by 400 percent in 2003, executives prepared for the inevitable public relations hit but assured themselves the backlash would be brief.***

*Nearly five years later, the accusations against Abbott are still flying.*

*The North Chicago, Ill.-based drug company stands to lose **hundreds of millions** of dollars in several pending antitrust*

lawsuits. It settled only its first—and likely its cheapest one—last week.

The lawsuits, all filed in Oakland federal court, accuse Abbott of raising the price of the HIV-fighting Norvir to illegally stifle competition and boost sales of its own alternative, Kaletra. Embarrassing internal communications between executives plotting how to thwart their rivals in **the lucrative HIV drug cocktail market** have been made public in the process.

Executives acknowledged in 2003 internal memos that Abbott would appear as the “big, bad, greedy pharmaceutical company” if they implemented the Norvir price increase, according to court documents.

But it also led to a barrage of lawsuits filed by patients, drug wholesalers and one competitor.

Last week, Abbott agreed to pay \$10 million to \$27.5 million to charities to settle one of the smaller lawsuits filed in 2004 by HIV patients. The payout depends on how the company fares before the 9th U.S. Circuit Court of Appeals, which will be asked to settle several contentious antitrust questions raised in the case.

The payout in that lawsuit is expected to be small compared with potential settlements or losses in other cases because the amount was limited by federal law, which blocks “indirect” buyers — such as patients who buy the drug through pharmacies — from collecting damages in antitrust litigation.

Abbott does not enjoy that same legal protection in the remaining lawsuits, filed by pharmacies, drug wholesalers and competitor GlaxoSmithKline, which can argue to triple any antitrust awards they win. Those cases are pending before Oakland-based U.S. District Judge Claudia Wilken, who so far has generally ruled against Abbott.

The central allegation by all plaintiffs is that Abbott raised the price of its pioneering drug Norvir to illegally dominate the market.

Norvir is a key ingredient — a “booster” — in many multidrug cocktails made by rival companies that HIV patients take **to keep the disease at bay**.

When Abbott increased Norvir’s cost, it effectively raised the prices of its competitors’ drugs. At the same time, Abbott kept the price the same for its own multidrug pill, Kaletra, which contains Norvir.

Corporate documents produced in court showed Abbott executives hoped that the price increase in 2003 would boost sales of Kaletra at the expense of two rival drugs coming on the market that year. The documents also showed that executives were considering other options to persuade doctors to prescribe Kaletra rather than competing drugs.

**One option was to stop making Norvir pills and make only an unpalatable liquid form of the drug commercially available. The executives also considered halting all sales.**

Abbott spokeswoman Melissa Brotz said the proposals other than a price hike were “never seriously considered.”

She contended that Abbott increased Norvir’s price to reflect its unintended role as a booster drug rather than a stand-alone treatment.

The Food and Drug Administration approved Norvir in 1996 for use as a stand-alone AIDS treatment. Then Abbott scientists discovered that Norvir **worked wonders** in significantly lower doses as a booster with the rival drugs, and its popularity soared as a key ingredient to many multidrug cocktails.

## **DESPITE THEIR LARGE POPULATION OF HETEROSEXUALS, NEW YORK CITY STILL AIDS EPICENTER:**

2008 Aug 5. NYC Is Still the Epicenter of the U.S. HIV Epidemic. Walensky RP et al. Ann Intern Med; 149:153: AIDS, September 8, 2008.

## **MICE TURN INTO HUMANS AND CATCH AIDS:**

2008 Aug. RNA Interference Suppresses HIV Infection in “Humanized” Mice. Zier LS et al. Crit Care Med 36:2341

*RNA interference rendered mice with reconstituted human immune systems resistant to HIV infection. By Anthony L. Komaroff, MD August 26, 2008*

## **RAPID TESTS REVOLUTIONIZE EMERGENCY ROOM TESTING AND ARE SAID TO BE A SUCCESS DESPITE THE FACT THAT “THEY CAN’T RULE OUT OR CONFIRM” “HIV-INFECTION:”**

2008, September 8. Rapid HIV Testing in Emergency Departments; Beware of False Positives.

*“In a low-prevalence ED (Emergency Department), rapid tests often ruled out HIV, but, when reactive, **they often yielded false-positive results.**” By Carlos del Rio, MD September 8, 2008.*

*“In 2006, the CDC recommended routine HIV testing for all adults and adolescents in all healthcare settings, including emergency departments (EDs; AIDS Clin Care Oct 6 2006). This recommendation, coupled with the availability of rapid HIV tests, has resulted in a variety of efforts to increase HIV screening in EDs. However, many concerns remain (JAMA 2008; 300:945), such as the feasibility of testing in this setting, mainly because of the resources required” (AIDS Clin Care Jan 14 2008), and the specificity of rapid oral-fluid tests (AIDS Clin Care Jul 7 2008).*

*“In the present study, investigators evaluated the performance of the OraQuick Advance Rapid HIV-1/2 Antibody Test in the context of a clinical trial conducted in a busy ED in Boston. All rapid tests were performed using oral fluid, and all reactive tests were followed by a Western blot, an EIA, and measurement of both CD4-cell count and plasma viral load.”*

*“During an 8-month period in 2007, 2356 ED patients were invited to participate in the HIV testing trial; 1397 (59.3%) agreed. Of the 854 who actually underwent rapid testing, 849 had interpretable results. Thirty-nine patients (4.6%) had reactive tests. Of the 31 who agreed to confirmatory testing, only 5 were found to be HIV-infected, for an overall HIV prevalence of 0.6%; the others were catalogued as not infected. In this setting, the estimated specificity of the test was 96.9% (95% confidence interval, 95.7%; 98.1%), which was significantly lower than the specificity reported by the manufacturer” (99.8%; 95% CI, 99.6% ;99.9%).*

*JOURNAL EDITOR’S Comment: “This HIV testing program was considered a success, because many patients who otherwise would not have been tested left the ED knowing that they were not HIV-infected. However, **we clearly need better ways to confirm or rule out the presence of HIV infection in patients whose rapid tests are reactive.** The authors suggest including **viral-load testing** in the panel of confirmatory tests, along with Western blot and EIA.”*

## **NATURE SAYS THAT “HIV” AND “AIDS” WAS DEFINITELY, UNEQUIVOCALLY, AND HISTORICALLY, THE FAULT OF BLACKS, BECAUSE OF “THEIR ASSOCIATIONS” WITH CHIMPS WHEN THEY BUILT CITIES WITH THEM AND HAD “HIGH RISK BEHAVIORS” 125 YEARS AGO:**

2008, October. News Flash: HIV/AIDS Originated 125 Years Ago, Spread from Chimps to Humans

**TUCSON, Arizona, October 2, 2008 (ENS)** – New research indicates that the most pervasive global strain of HIV began spreading among humans as early as **1884**, suggesting that growing urbanization in colonial Africa through the early 1900s set the stage for the current HIV/AIDS pandemic. More than 25 million people have died of AIDS since 1981, and at least 30 million people are living with the disease today.

**The estimated period of origin**, much earlier than the previous estimate of **1930**, **coincides with the establishment and rise of urban centers in west-central Africa** where the pandemic HIV strain, HIV-1 group M, emerged.

**The growth of cities and associated high-risk behaviors may have been the key change** that allowed the virus to flourish, scientists believe.

The research, led by Michael Worobey, an assistant professor of ecology and evolutionary biology at the University of Arizona in Tucson, was co-sponsored by the National Institute of Allergy and Infectious Diseases, part of the National Institutes of Health, and the David and Lucile Packard Foundation. The findings are published in the current issue of the journal **“Nature.”**

Research shows that **the HIV virus spread from chimps to humans in southeastern Cameroon**. Worobey said the resulting HIV epidemic among humans correlates to the growth of urban centers near this area, principally the present-day city of Kinshasa in the Democratic Republic of the Congo, which began as a colonial center for Belgium.

Worobey and his team screened a number of tissue samples and uncovered the world’s second-oldest genetic sequence of HIV-1 group M, which dates from 1960.

They then used it, along with dozens of other previously known HIV-1 genetic sequences, to construct a range of plausible family trees for this viral strain.

The lengths of the tree branches represent the periods of time when the virus genetically diverged from its ancestors. **The timing and number of these genetic mutations enabled the scientists to calibrate the probable rates of evolution of HIV-1 group M**. Based on this range of rates, **the scientists projected back in time** to the period when the viral family trees most likely took root around the turn of the 20th century. This marks the probable time of origin of HIV-1 group M, according to Worobey and the other scientists.

Using newly developed techniques, the scientists recovered the 48-year-old HIV gene fragments from a wax-embedded lymph-node tissue biopsy from a woman in Kinshasa in the Democratic Republic of the Congo. The oldest known HIV-1 group M genetic sequence comes from a 1959 blood sample from a man, also from Kinshasa.

A comparison of the same genetic region in the 1959 virus and the 1960 virus provided additional evidence that the common ancestor of both viruses existed around 1900. The comparison revealed that the amount of genetic divergence between these two HIV sequences took more than 40 years to evolve.

**The virus responsible for HIV/AIDS jumped from chimpanzees to humans.**

Worobey, who teaches the evolution of infectious diseases and molecular phylogenetics at the University of Arizona, has spent several years studying how to recover the fragmented pieces of viral DNA and RNA from archival specimens, to track when the virus first **jumped** from chimpanzees to humans...

**By 1960** a large number of people in this region were infected with HIV, which shows as genetic diversity of the virus. From

there, events seeded the epidemic in different parts of the world. By 1981, people started realizing that something was happening **and the rest is history.**

Worobey said laying the technical groundwork for analyzing samples of HIV's ancient history was extraordinarily painstaking. "The DNA and RNA in these samples is in a **really sorry state. It's highly fragmented, so instead of a nice, pearl-strand of DNA or RNA, you have a jumbled mass that's all jammed together,**" he said. "It's been gratifying, but a ridiculous amount of work..."

"There's still a lot of interesting work we can do with these techniques. We have lots more samples to analyze and hopefully recover nucleic acids from and it's pretty exciting to be in that position," Worobey said. "I think the picture that has emerged here, where changes the human population experienced may have opened the door to the spread of HIV, is a good reminder that **we can make changes now that could help reverse the epidemic,**" he said.

**"If HIV has one weak spot, it is that it is a relatively poorly transmitted virus. From better testing and prevention, to wider use of antiretroviral drug therapy, there are a number of ways to reduce transmission and force this virus back into extinction,"** Worobey said. "Our results suggest that there are reasons for such **optimism.**"

**BARRE-SINOUSI, LUC MONTAGNIER, HAROLD ZUR HAUSEN RECEIVE NOBEL PRIZE FOR "HIV" AND HPV: GALLO SAYS THEY DESERVE IT:**

**2008** October 6. Montagnier, Barre-Sinoussi and zur Hausen Share Nobel

**Physiology or medicine prize recognizes work on HIV and human papillomavirus (HPV) linked to cervical cancer—but leaves out Robert Gallo** By Jordan Lite in *Scientific American*

A pair of French scientists who **isolated** the **AIDS-causing** human immunodeficiency virus (HIV) and a German scientist **who determined** that human papillomavirus (HPV) **causes** cervical cancer were awarded the [Nobel Prize](#) in Physiology or Medicine today.

The Nobel committee's decision to give the prize to Luc Montagnier and Françoise Barre-Sinoussi, who **isolated** [HIV](#) in 1983, caps a long, bitter dispute between the Pasteur Institute in Paris, where they made their discovery, and American scientist **Robert Gallo**, who **linked** HIV to AIDS **separately** but was [snubbed](#) by the Nobel committee.

Gallo, then a division chief at the National [Cancer](#) Institute, **made the connection** between HIV and AIDS in research published in 1984 in the journal *Science*. A year later, the Pasteur Institute **sued him** for allegedly using one of its samples of HIV to draw his conclusion.

In 1992 a review panel of the National Academy of Sciences determined that the sample Gallo used was contaminated with material from the Pasteur Institute, and accused him of "**intellectual recklessness of a high degree.**" That same year, Gallo was found **guilty of scientific misconduct** by the Office of Research Integrity of the U.S. Public Health Service.

Both determinations were [overturned on appeal](#). Gallo and Montagnier, 76, co-authored at least two papers, [one for Scientific American in 1988](#), and [another in Science in 2002](#) in which they credited each other with aspects of the discovery. Today, members of the Nobel committee said the award was given **to the right scientists.**

"These two persons awarded today made the discovery," Jan Andersson said at a Stockholm press conference broadcast online.

**“They provided the virus.”** Added Göran Hansson, another member of the committee: **“It’s completely evident the discovery was made in Paris. It’s quite clear if you go to the scientific magazines.”**

But Montagnier, remarking on the omission of Gallo from the prize, [told the Associated Press](#) that **“it is certain that he deserved this as much as us two.”**

Gallo [told the AP](#) that the snub was “a disappointment” but that the three winners deserved the prize.

“I am pleased my long-time friend and colleague Dr. Luc Montagnier, as well as his colleague Françoise Barre-Sinoussi, have received this honor,” Gallo, [co-director of the Institute of Human Virology at the University of Maryland School of Medicine](#), said in a statement afternoon. “I was gratified to read Dr. Montagnier’s kind statement this morning expressing that I was equally deserving. I am pleased that the Nobel Committee chose to recognize **the importance of AIDS** with these awards.”

Barre-Sinoussi, 61, had not yet been reached by the Nobel committee when they announced the prize at 11:30 A.M. Central European Time (5:30 A.M. Eastern time), and a spokeswoman for the Pasteur Institute, where she is director of the regulation of **retroviral infections unit**, said she was traveling. From Cambodia, Barre-Sinoussi told the AP that she and Montagnier hoped that identifying HIV would have curbed its spread. Some **33 million** people around the world are living with the virus, and **25 million** have died of AIDS.

### **The Nobel Prize in Physiology or Medicine 2008. The Nobel Assembly at Karolinska Institutet Press Release**

6 October 2008

[The Nobel Assembly at Karolinska Institutet](#) has today decided to award The Nobel Prize in Physiology or Medicine for 2008 with one half to **Harald zur Hausen** for his discovery of **“human papilloma viruses causing cervical cancer”** and the other half jointly to **Françoise Barré-Sinoussi and Luc Montagnier** for their discovery of **“human immunodeficiency virus”**

### **This year’s Nobel Prize awards discoveries of two viruses causing severe human diseases.**

Harald zur Hausen went against current dogma **and postulated** that **oncogenic** human papilloma virus (HPV) **caused** cervical cancer, the **second most common** cancer among women. He **realized** that **HPV-DNA could exist in a non-productive state in the tumours**, and should be detectable by specific searches for viral DNA. He found HPV to be a heterogeneous family of **viruses**. **Only some** HPV types cause cancer. His discovery has led to characterization of the natural history of **HPV infection**, an understanding of mechanisms of HPV-induced **carcinogenesis** and the development of **prophylactic vaccines** against HPV acquisition.

Françoise Barré-Sinoussi and Luc Montagnier discovered human immunodeficiency virus (HIV). Virus production was identified in lymphocytes from patients with enlarged lymph nodes **in early stages of** acquired immunodeficiency, and **in blood** from patients with late stage disease. They characterized this **retrovirus as the first known human lentivirus** based on its **morphological, biochemical and immunological** properties. HIV impaired the immune system because of **massive virus replication and cell damage to lymphocytes**. The discovery was one prerequisite for the current understanding of the biology of the disease and its antiretroviral treatment.

### **Discovery of human papilloma virus causing cervical cancer**



Against the prevailing view during the 1970s, Harald zur Hausen **postulated** a role for human papilloma virus (HPV) in cervical cancer. **He assumed** that the tumour cells, if they contained an oncogenic virus, should harbour viral DNA integrated into their genomes. The HPV genes promoting cell proliferation **should therefore be detectable** by specifically searching tumour cells for such **viral DNA**. Harald zur Hausen pursued this idea for over **10 years** by searching for different HPV types, a search **made difficult** by the fact that **only parts of the viral DNA were integrated** into the host genome. He found novel HPV-DNA in cervix cancer biopsies, **and thus discovered the new, tumourigenic HPV16 type in 1983**. In 1984, **he cloned HPV16 and 18 from patients** with cervical cancer. The HPV types 16 and 18 were consistently found in about 70% of cervical cancer biopsies throughout the world.

### **Importance of the HPV discovery**

The global public health burden attributable to human papilloma viruses **is considerable**. More than **5%** of all cancers worldwide are caused by **persistent infection** with this virus. Infection by the human papilloma virus **is the most common sexually transmitted agent**, afflicting **50-80%** of the population. Of the more than **100 HPV types** known, about **40** infect the genital tract, and 15 of these put women at **high risk** for cervical cancer. In addition, HPV is found in some vulval, penile, oral and other cancers. Human papilloma virus can be detected in **99.7%** of women with histologically confirmed cervical cancer, affecting some **500,000** women per year.

Harald zur Hausen demonstrated novel properties of HPV that have led to an understanding of mechanisms for papilloma virus-induced carcinogenesis and the **predisposing factors** for **viral persistence** and **cellular transformation**. He made HPV16 and 18 available to the scientific community. Vaccines were ultimately developed that provide **≥95 %** protection from infection by the **high risk** HPV16 and 18 types. The vaccines may also reduce the need for surgery and the global burden of cervical cancer.

### **Discovery of HIV**

Following medical reports of a novel immunodeficiency syndrome in 1981, the search for a causative agent was on. Françoise Barré-Sinoussi and Luc Montagnier isolated and cultured lymph node cells from patients that had swollen lymph nodes **characteristic of** the early stage of acquired immune deficiency. They detected activity of the retroviral enzyme **reverse transcriptase, a direct sign of retrovirus replication**. They also found **retroviral particles budding** from the infected cells. **Isolated virus infected and killed lymphocytes** from both diseased and healthy donors, and reacted with antibodies from infected patients. In contrast to previously characterized human **oncogenic retroviruses**, the novel retrovirus they had discovered, now known as human immunodeficiency virus (HIV), **did not** induce uncontrolled cell growth. Instead, the virus **required cell activation** for replication and **mediated cell fusion of T lymphocytes**. This **partly** explained how HIV impairs the immune system since the T cells are essential for immune defence. By 1984, Barré-Sinoussi and Montagnier had obtained several **isolates** of the novel human retrovirus, which they identified as a lentivirus, from **sexually infected** individuals, haemophiliacs, mother to infant transmissions and transfused patients. The significance of their achievements should be viewed in the context of a global ubiquitous epidemic affecting close to **1%** of the population.

### **Importance of the HIV discovery**

Soon after the discovery of the virus, several groups contributed to the definitive demonstration of HIV as the cause of acquired human immunodeficiency syndrome (AIDS). Barré-Sinoussi and Montagnier's discovery made rapid **cloning** of the HIV-1 genome possible. This has allowed identification of important details in its replication cycle and how the virus interacts with its host. Furthermore, it led to development of methods to diagnose **infected** patients and to screen blood products, which **has**

limited the spread of the **pandemic**. The **unprecedented development** of several classes of new antiviral drugs is also a result of knowledge of the details of the viral replication cycle. The combination of prevention and treatment has **substantially decreased** spread of the disease and **dramatically increased** life expectancy among **treated** patients. The cloning of HIV enabled studies of its origin and evolution. **The virus was probably passed to humans from chimpanzees in West Africa early in the 20th century**, but it is still unclear why the epidemic spread so dramatically from 1970 and onwards.

Identification of virus–host interactions has provided information on how HIV evades the host’s immune system by impairing lymphocyte function, **by constantly changing and by hiding its genome in the host lymphocyte DNA, making its eradication in the infected host difficult even after long-term antiviral treatment**. Extensive knowledge about these unique viral host interactions has, however, generated results that can provide **ideas** for future **vaccine development** as well as for therapeutic approaches targeting viral latency.

HIV has generated a novel **pandemic**. Never before has science and medicine been **so quick** to discover, identify **the origin** and provide treatment for a new disease entity. **Successful** anti-retroviral therapy results in life expectancies for persons with HIV infection now reaching levels similar to those of **uninfected** people.

In his Nobel lecture, Montagnier hedged about “HIV and AIDS” in the following way:

However, **a few opponents** led by P. Duesberg argued and are still arguing that **there is no real demonstration that the virus does exist and is the cause of AIDS** according to Koch’s postulates. In fact, the proviral DNA of the virus, renamed HIV (Human Immunodeficiency Virus) by an international nomenclature Committee, **was cloned and sequenced** (Alizon et al., 1984; Wain-Hobson et al., 1985; Ratner et al., 1985), showing **the classical gene structure of animal retroviruses** which Dr Duesberg helped himself to uncover at earlier times. But in addition, new genes (**Tat, Nef**), important in regulation of the expression of the viral genetic information, were recognized from the DNA sequencing, making the viral genome probably **the most complex known** in the retrovirus family. HIV and **its Primate Cousins** is therefore a well characterized entity only composed of DNA sequences **none existing in the human genome**. A posteriori, two facts should have provided to the few remaining skeptics final conviction that HIV is the culprit in AIDS: (1) Transmission of AIDS by blood transfusion has practically disappeared in countries where the detection of HIV antibodies in blood donors has been implemented. (2) The inhibition of virus multiplication by a combination of specific inhibitors of the viral enzymes (reverse transcriptase, protease), has greatly improved the clinical conditions of the patients. Mutations in the genome of HIV inducing resistance to **these inhibitors has led to relapses and aggravation of the patients’ condition**. In 1986, thanks to a collaboration with Portuguese colleagues, we isolated a second virus (which I named HIV2), from **West African** patients hospitalized in a Lisbon hospital. They **all** had the signs of AIDS **but had no antibodies against our first virus**. In fact, they had only antibodies **to the most variable protein of HIV, the surface glycoprotein**. **The patients had lost antibodies** against the well-conserved internal proteins of HIV2 which show common epitopes with their counterparts of HIV1, unlike the glycoprotein. **The isolation** of HIV1 and HIV2 viruses from AIDS patients **in Africa** made us realize that we were dealing with a large epidemic of **heterosexually transmitted viruses**. Evidence that HIV was not transmitted by **casual** contacts came from our study in a French **boarding school** where HIV infected haemophilic children were **in close contact**, day and night, with HIV negative non-haemophilic children: **none** of the latter was found HIV positive. The isolation of the virus causing AIDS allowed to implement **rational** prevention measures and also to start a search for efficient viral inhibitors. The first candidate, azidothymidine (AZT), was an efficient inhibitor of HIV reverse transcriptase **in in vitro experiments** (Mitsuya). However, its use **in AIDS patients** was soon recognized as **disappointing**. In fact, the treatment readily induced **mutants** of the virus resistant to AZT and did **not** extend the life span of the patients. The main obstacle of treatment with a single or two inhibitors was the capacity of the virus to **mutate**, which also impedes the design of an **efficient vaccine** and also explains the complexity of the pathophysiology of AIDS. **Only a combination of three inhibitors proved to be efficient on the clinical outcome**. Since 1996, clinicians are using HAART

(Highly Active Antiretroviral Therapy) to treat patients with high virus load and low CD4+ T cell number, preventing them **most of the time** from falling into lethal opportunistic infections.

### The HIV variability

In fact, in order to escape to the immune reactions of their hosts, most **viruses have a strategy** to change their immunogenic epitopes. In the case of HIV, a conjunction of several factors put it to an unprecedented level. I have listed below the factors which seem to be most responsible for this variability. (1) Errors of reverse transcription, (2) Genetic recombination, (3) Incomplete neutralization by Vif of the activity of the APOBEC3G cellular gene, (4) Oxidative stress. The first is that the replicative enzyme, **reverse transcriptase (RT)**, has no editing compensation, so that the transcription errors may reach **1/105 nucleotides**, far from **1/109** of the **cellular DNA polymerases**. However some other retroviruses, such as **HTLV**, **do not show this variation rate**, since once integrated, the proviral DNA remains replicated by the cellular DNA replicative machinery. The difference could be explained by the fact that **the HIV infected cells die**, so that the virus can maintain itself **only by many cycles of new infections** involving each time reverse transcription of its RNA into DNA. **However**, In Vitro infection of cell lines, also involving cytopathic effect and many cycles of re-infection, **the virus seems to be stable, in the absence of immunoselective pressure**. Another factor of variation is genetic recombination. The immune responses (humoral and cellular) against the virus are unable to prevent a second virus infection of the host (because of virus variability induced by the previous factor and other causes), so that **some cells could be** co-infected by **two viruses**: this will also **allow** genetic recombination between the two viral RNAs existing each in two copies. The result is a **“mosaic” virus** in which many sequences from the two original viruses are entangled, starting from “hot spots” of recombination. This is particularly visible **in Africa, probably because of repeated exposure to infection** of many patients. The mosaic viruses, because of their selective advantage, then disseminate in the infected population. The original subtypes called A B C D E G... defined by the sequence of their envelope gene are thus replaced by A/G, B/C, etc... depending on the geographic location. Moreover, two other factors have been more recently identified: In the lymphocytes are expressed a family of genes coding for enzymes able to convert guanosine into adenosine in the viral DNA, **fouling the viral genetic code (APOBEC3G)**. **However, the virus has evolved a gene, Vif, which can more or less counteract this effect, rendering viable the viral DNA without completely avoiding mutations**. A last factor of variability, **whose the importance has been probably overlooked, is oxidative stress**, a cause of RNA and DNA **mutations** (before integration of the proviral DNA): highly reactive molecules derived from oxygen can oxidize the bases, particularly guanine or deoxyguanine, thus modifying their coding capacity or inducing a wrong replacement in repair. A combination of these factors could explain both the intrinsic variability of the virus **in the host** during the long evolution of infection, and also **the increasing variability** of the circulating strains as the epidemic is spreading in various populations. We can at least act on this variability **by decreasing the viral multiplication rate** inside the host by antiretroviral treatment and also by neutralizing the oxidative stress. **The remaining problems: How HIV infection results in the destruction of the immune system**. In early years following the virus discovery, it was generally thought that the drop of CD4+ T cells was due to **their direct infection by a cytopathic virus**. **In fact, the viral isolates (like Bru) made in the early stage of the disease are not cytopathic**, they use after binding to the CD4+ receptor of activated lymphocytes, a co-receptor (CCR5) which is the receptor for a chemokine. **Only viruses isolated from patients at late stage of the disease are cytopathic (like Lai)** and their direct infection of the remaining T lymphocytes (by using another chemokine co-receptor CXCR4) could account for the final drop of these cells. In fact, the number of activated CD4+ T lymphocytes (the ones which only allow full replication of the virus), is **probably** a limiting factor of the initial infection, after the first contact with dendritic cells and monocytes **of genital or rectal mucosa**. **It is obvious** that inflammation and co-infections (bacterial, viral) **could** increase the number of activated T lymphocytes **and therefore could increase the risk of HIV infection**. Recently, the virus has been found associated with **the Peyer patches existing around the small intestine which constitutes a major source of activated T lymphocytes**. At the onset of infection, **the virus replication is high** in all the lymphatic tissues, **taking advantage of the delay of reaction of the immune system** (in time order, interferon, NK cells, CD8T cells, antibody response) and then

decreases while persisting in **somelymph nodes**. This is the beginning of the chronic phase which is **generally asymptomatic**, although the lymphadenopathy is **often** present. **It has been shown** that the virus replication continues in the lymph nodes, **despite the immune response**. This one starts declining, although there is a continuous renewal of T lymphocytes, both CD4+ and CD8+, which could last **for years**. During this period, we have found two phenomena which could help explaining **the indirect destruction** of the immune system: one biological: apoptosis one biochemical: oxidative stress. Apoptosis: my laboratory was the first to describe this program cell death in white blood cells cultured in medium **deprived of interleukin 2**. **All the subsets**, not only the CD4+ T cells were affected when taken from the blood of **asymptomatic** HIV patients as well as in patients presenting with full blown AIDS: CD8+ T cells, NK cells, B lymphocytes, monocytes. However, we found a good correlation between the drop of CD4+ T cells in patients and this In Vitro phenomenon. **We surmised** that in the In Vivo situation, **cells were still alive but in pre-apoptosis**. Indeed, we could detect in infected patients **a general phenomenon of immune activation**, which **has been now well recognized** as a major factor of AIDS pathogeny. At the biochemical level, we also showed that the lymphocyte population of **asymptomatic** patients (CD4+, CD8+, NK) displayed the biochemical signs of **oxidative stress** (excess of free radicals derived from oxygen): namely fast degradation of oxidized protein, carbonylation of some of their amino acids. In the patients' blood, we could detect similarly a hyper-oxidation of plasma lipids and oxidization of guanine. What could be the origin of this strong oxidative stress? At least one HIV protein may contribute to it. It was shown by C. Flores, Mc Cord and their collaborators that the **Tat** protein, among many functions, inhibits the expression in lymphocytes of the Mn-dependent superoxide dismutase gene. This enzyme is the key to transform the anion superoxide, highly oxidant into hydrogen peroxide. Tat has been shown to circulate in nanogram amounts in the blood of infected patients and to penetrate inside cytoplasm. In addition, bacterial and viral co-infectors can also induce oxidative stress. We have been studying the possibility that a "cold" persisting bacterial infection could co-exist in HIV infected patients. These studies were initiated because we observed that In Vitro **coinfection of lymphocytes with some mycoplasma species** (*M. pirum*, *M. penetrans*, *M. fermentans*) and HIV could greatly reinforce the cytopathic effect of the latter. Moreover, these small bacteria lack catalase, an enzyme able to convert hydrogen peroxide into water. Therefore they also generate oxidative stress and, furthermore, are activators of lymphocytes. **In summary, the pathophysiology of AIDS is complex. HIV is the main cause, but could also be helped by accomplices, and also have some indirect effects by wrongly activating the immune system through oxidative stress.**

## **GERMAN DOCTOR CURES CANCER AND ONE AIDS PATIENT'S POSITIVE STATUS WITH RADIATION, MUTATION, AND DAVID BALTIMORE'S DREAMS ABOUT GENE THERAPY:**

**2008**, NOVEMBER 7 The Wall Street Journal:

*A Doctor, a **Mutation** and a Potential **Cure** for AIDS*

*A Bone Marrow Transplant to Treat a Leukemia Patient Also Gives Him Virus-Resistant Cells;*

*By MARK SCHOOFS*

*The startling case of an AIDS patient who underwent a bone marrow transplant to treat leukemia is stirring new hope that gene-therapy strategies on the far edges of AIDS research **might someday** cure the disease.*

*The patient, a 42-year-old American living in Berlin, **is still recovering** from his leukemia therapy, but **he appears to have won his battle with AIDS**. Doctors have not been able to detect **the virus** in his blood for more than **600 days, despite** his having ceased all conventional AIDS medication. **Normally** when a patient **stops** taking AIDS drugs, **the virus stampedes through the body within weeks, or days**.*

Dr. Gero Hütter isn't an AIDS specialist, but he **'functionally cured'** a patient, who shows no sign of the disease.

"I was very surprised," said the doctor, Gero Hütter.

The breakthrough appears to be that Dr. Hütter, **a soft-spoken hematologist** who isn't an AIDS specialist, deliberately replaced the patient's bone marrow cells with those from a donor who has **a naturally occurring genetic mutation** that renders his cells immune **to almost all strains of HIV, the virus that causes AIDS.**

The development suggests a potential new therapeutic avenue and comes as the search for a cure has adopted **new urgency.** **Many fear that current AIDS drugs aren't sustainable.** Known as antiretrovirals, the medications **prevent the virus from replicating** but must be taken **every day for life** and are expensive for poor countries where the disease runs rampant. Last year, **AIDS killed two million people**; 2.7 million more contracted the virus, so treatment costs will keep ballooning.

While cautioning that the Berlin case **could be a fluke**, David Baltimore, who won a Nobel prize for his research on **tumor viruses**, deemed it **"a very good sign" and a virtual "proof of principle" for gene-therapy approaches.** Dr. Baltimore and his colleague, University of California at Los Angeles researcher Irvin Chen, have developed a gene therapy strategy against HIV **that works** in a similar way to the Berlin case. Drs. Baltimore and Chen **have formed a private company** to develop the therapy.

Back in 1996, when "cocktails" of antiretroviral drugs were **proved effective**, some researchers proposed that **all cells** harboring HIV might eventually **die off**, leading to **eradication** of HIV from the body — in short, **a cure.** Those hopes foundered on the discovery that HIV, which integrates itself into a patient's own DNA, **hides in so-called "sanctuary cells," where it lies dormant** yet remains capable of reigniting an **infection.**

But that same year, researchers discovered that **some gay men astonishingly** remained **uninfected** despite engaging in **very risky sex with as many as hundreds of partners.** These men had inherited **a mutation** from **both** their parents that made them **virtually** immune to HIV.

The mutation prevents a molecule called **CCR5** from appearing on the surface of cells. CCR5 acts as **a kind of door for the virus.** Since **most HIV strains** must bind to CCR5 to enter cells, **the mutation** bars the virus from entering. A new AIDS drug, Selzentry, made by Pfizer Inc., doesn't attack HIV itself but works by blocking CCR5. About **1%** of Europeans, **and even more in northern Europe**, inherit the CCR5 mutation from **both** parents. People of **African**, Asian and South American descent **almost never carry it.**

Dr. Hütter, 39, remembered this research when his American leukemia patient **failed** first-line chemotherapy in 2006. He was treating the patient at Berlin's Charité Medical University, the same institution where German physician **Robert Koch** performed some of his groundbreaking research **on infectious diseases** in the 19th century. Dr. Hütter scoured research on CCR5 and consulted with his superiors.

Finally, he recommended standard second-line treatment: **a bone marrow transplant** — but from a donor who had inherited the CCR5 mutation from both parents. Bone marrow is where immune-system cells are generated, so transplanting mutant bone-marrow cells would render the patient immune to HIV into perpetuity, **at least in theory.**

There were a total of **80 compatible blood** donors living in Germany. Luckily, on the 61st sample he tested, Dr. Hütter's colleague Daniel Nowak found one with **the mutation** from both parents.

To prepare for the transplant, Dr. Hütter first administered a standard regimen of **powerful drugs and radiation to kill the patient's own bone marrow cells and many immune-system cells**. This procedure, **lethal to many cells that harbor HIV**, may have helped the treatment succeed.

The transplant specialists **ordered** the patient to stop taking his AIDS drugs when they transfused the donor cells, **because they feared the powerful drugs might undermine the cells' ability to survive in their new host**. They planned to resume the drugs **once HIV re-emerged** in the blood.

But it never did. Nearly two years later, **standard tests** haven't detected **virus** in his blood, or in the brain and rectal tissues **where it often hides**.

The case was presented to scientists earlier this year at the Conference on Retroviruses and Opportunistic Infections. In September, the nonprofit Foundation for AIDS Research, or **aMFAR**, convened a small scientific meeting on the case. Most researchers there **believed** some HIV still **lurks** in the patient but that it can't ignite a **raging infection**, most likely because its target cells are invulnerable **mutants**. The scientists agreed that the patient is "**functionally cured**."

Caveats are **legion**. If enough time passes, **the extraordinarily protean HIV might evolve** to overcome the mutant cells' invulnerability. **Blocking CCR5 might have side effects**: A study suggests that **people with the mutation are more likely to die from West Nile virus**. Most worrisome: The transplant treatment itself, given only to late-stage cancer patients, **kills up to 30% of patients**. While scientists are drawing up research protocols to try this approach on other leukemia and lymphoma patients, **they know it will never be widely used to treat AIDS because of the mortality risk**.

There is a potentially safer alternative: Re-engineering a patient's own cells through **gene therapy**. **Due to some disastrous failures, gene therapy now "has a bad name," says Dr. Baltimore**. In 1999, an 18-year-old patient died in a gene therapy trial. Even one of gene therapy's greatest successes — curing children of the inherited "bubble boy" disease — came at the high price of causing **some** patients to develop leukemia.

Gene therapy also faces **daunting** technical challenges. For example, the therapeutic genes are carried to cells by re-engineered viruses, and they must be made **perfectly safe**. Also, most gene therapy currently works by removing cells, genetically modifying them out of the body, then transfusing them back in — a complicated procedure that would prove too expensive **for the developing world**. Dr. Baltimore and others are working on therapeutic viruses they could inject into a patient as easily as a flu vaccine. But, he says, **"we're a long way from that."**

**Expecting** that gene therapy **will eventually play** a major role **in medicine**, several research groups are testing different approaches for AIDS. At City of Hope cancer center in Duarte, Calif., John Rossi and colleagues **actually use HIV itself**, genetically engineered to be harmless, to deliver to patients' white blood cells three genes: one that inactivates CCR5 and two others that **disable** HIV. He has already completed the procedure on **four** patients **and may perform it on another**.

One big hurdle: doctors **can't yet genetically modify all** target cells. **In theory**, HIV would **kill off** the susceptible ones and, a victim of its own **grim success**, be left only with the genetically engineered cells that it can't infect. **But so far that's just theory**. All Dr. Rossi's patients **remain** on standard AIDS drugs, **so it isn't yet known** what would happen if they stopped taking them.

In 1989, Dr. Rossi had a case **eerily similar** to the one in Berlin. A 41-year-old patient with AIDS **and lymphoma** underwent radiation and drug therapy **to ablate his bone marrow and received new cells from a donor**. **It is not known if those cells had the protective CCR5 mutation, because its relation to HIV hadn't been discovered yet**. But after the

transplant, **HIV disappeared** from the patient's blood. **The patient died** of his **cancer 47 days after the procedure**. Autopsy tests from eight organs and the tumor revealed no HIV. (The Foundation for AIDS Research, which uses the acronym amFAR, is the name of the nonprofit group cited in this article. The name of the group was incorrectly given as the American Foundation for AIDS Research).

**THE MICROBICIDE, CELLULOSE SULFATE, INCREASES RATE OF “HIV”INFECTION AMONGST BLACKS IN BENIN AND UGANDA, WHO HAVE SEX 20 TIMES PER WEEK ON AVERAGE, AND WHO HAD 9 INFECTIONS WITH THE MICROBICIDE VERSUS 1 INFECTION WITH PLACEBO. BLACKS IN SOUTH AFRICA ONLY HAVE SEX 4 TIMES/WEEK AND ONLY ACQUIRED 12 INFECTIONS WITH THE MICROBICIDE AND ONLY 10 WITH PLACEBO**

**2008** NOVEMBER 8. New England Journal of Medicine (359;19, 2066-2068, 2008):

*To the Editor:*

*The results of the trial of cellulose sulfate as a vaginal gel for the prevention of human immunodeficiency virus (HIV) infection reported by Van Damme et al., (July 31 issue),<sup>1</sup> indicated that cellulose sulfate did not prevent sexual transmission of HIV and may have increased the risk of HIV acquisition, as compared with placebo. The cellulose sulfate and placebo gels had a pH of 7.5 and 4.4 respectively. The healthy human vagina provides a low pH, diminishing HIV infectivity and transmission of cell-associated HIV.<sup>2,3</sup> Therefore, microbicides should have a pH of approximately 4.5.<sup>3</sup> The apparently increased risk of HIV acquisition among women using the cellulose sulfate gel might be due to the disparity in pH between the active-treatment and placebo gels...It remains to be established whether candidate microbicides interfere with this defense mechanism and possibly increase the probability of HIV acquisition.*

*A. Robert Neurath, Ph.D.*

*Authors Reply:...*

*The potential **increased risk of infection** observed in our per-protocol analysis was driven by results from two sites (Benin and Uganda) where gel was reportedly used 20 times per week on average (**9** infections with cellulose sulfate and **1** with placebo). This frequency of use was dramatically higher than the four-times-per-week use reported in South Africa, where there was essentially no evidence of an effect (**12** infections with cellulose sulfate and **10** with placebo). Although not conclusive, these findings suggest that a mechanism related to very frequent exposure to cellulose sulfate is a more likely explanation for our results.*

*Lut Van Damme, M.D.*

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**DOZENS OF INFANTS AND SOME CHILDREN ARE GIVEN “HIV-INFECTION” BY GUILTY CENTRAL ASIAN DOCTORS AND HEALTH CARE WORKERS:**

**2008, NOVEMBER *Uzbek children in ‘HIV outbreak’* By Martin Vennard, BBC News.**

Children in Kazakhstan and Kyrgyzstan have also been affected. More than 40 young children have been **infected** with HIV at a hospital in Uzbekistan, officials have said. Health authorities told the BBC that an investigation into the infections of the mainly **new-born babies** was taking place in the eastern town of Namangan.

The United Nations says **Central Asia has one of the world's fastest-growing HIV infection rates. Unsafe blood supplies and contaminated equipment** are often blamed for spreading the **infection**.

*Stigma and secrecy*

*The infections in Uzbekistan are just the latest case of mass contamination in a health facility in the region. In August, a court in Kyrgyzstan convicted nine medical workers of infecting 24 children with HIV, while last year 21 medical workers in Kazakhstan were found guilty of infecting dozens of babies. The staff concerned pleaded not guilty, saying **poor hygiene conditions were to blame**.*

*The Uzbek cases were discovered in October, and have reportedly been referred to prosecutors. But they have not been reported in the local media, which is tightly controlled by the government. The officials who spoke to the BBC's Uzbek service were only prepared to comment on condition of anonymity. Aid workers say the stigma surrounding HIV/Aids and the atmosphere of secrecy means that many outbreaks of hospital-acquired infection do not get reported. In July this year, Jimmy Kolker, a senior UN official on HIV/Aids, called on Central Asian governments to record and share their information on cases. He was speaking at a meeting in Uzbekistan, which was discussing how to tackle a regional epidemic of HIV among women and children.*

#### **CIRCUMCISION DOESN'T STOP "HIV" TRANSMISSION BETWEENHOMOSEXUALS.**

**2008** Rebeca M. Plank, MD. JAMA 2008 Oct 8; 300:1698. A meta-analysis of observational studies does not provide evidence that male circumcision protects MSM against HIV or other sexually transmitted infections.

#### **DRUNK MONKEYS GET AIDS FASTER:**

**2008** <http://www.aidsmap.com/en/news/AF9B8471-7615-4DEB-BA46-8D6228A901A5.asp>

*"**Heavy drinking can accelerate time to AIDS** among **rhesus macaques** infected with simian immunodeficiency virus (SIV), researchers from Louisiana State University report in the October edition of *Alcoholism: Clinical & Experimental Research*. The monkeys were exposed to alcohol for **four days a week** at levels designed to simulate **`binge` drinking**, and compared with **a control group**."*

*"The key issue with alcohol consumption and HIV/AIDS is when to start individuals on **life-saving antiretrovirals** versus **the need to avoid their toxic effects that damage the liver and gut** along with the alcohol," added Kendall Bryant, Coordinator of HIV/AIDS Research at the National Institute on Alcohol Abuse and Alcoholism." How much did this study cost the American tax payer?*

#### **PREGNANT WOMEN GET SICK OR DEAD FASTER ON NEVIRAPINE:**

**2008, OCTOBER** <http://www.aidsmap.com/en/news/C1316A81-32F9-4AA9-A94F-93E3EB4E0DoE.asp>

*"Anti-HIV therapy based on the non-nucleoside (NNRTI) nevirapine (Viramune) is associated with an increased incidence of serious liver toxicities in HIV-positive pregnant women with a CD4 cell count above 250 cells/mm<sup>3</sup>, according to a study*



published in the July 1st edition of the Journal of Immune Deficiency Syndromes. The study was designed to compare the safety and efficacy of HAART regimens based on either the NNRTI nevirapine or the protease inhibitor nelfinavir (Viracept) in HIV-positive pregnant women, **however the study was stopped early because of a higher than expected incidence of serious side-effects in the nevirapine arm.** "Recruitment to the study was **stopped early** because of **a greater than expected incidence of severe liver side-effects in the nevirapine arm of the trial**, and because the manufacturers of nevirapine issued new prescribing information recommending caution if prescribing the drug to women with a CD4 cell count above 250 cells/mm<sup>3</sup>."

## **DRUG ADDICTS IN 1971-2 TEST "HIV-POSITIVE," THEN 18 YEARS LATER, NEGATIVE, DESPITE LACK OF DRUGS:**

2008 OCTOBER <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1580267#reference-sec>

*Serum specimens obtained from a nationwide sample of parenteral drug abusers (PDAs) during the period 1971-72 had previously been screened for human immunodeficiency virus (HIV) antibodies. Some specimens were considered to be positive to both ELISA and Western blot (WB) analysis. These findings have been a topic of controversy, since HIV was not thought to have penetrated at-risk populations at such an early date. This study was a follow up of those PDAs with apparent seropositivity to WB analysis. Of 10 persons followed, only one death (in 1985) was documented, and postmortem findings were inconsistent with HIV infection. **Eight of the remaining PDAs were traced and found to be alive and well in 1989.***

## **STOPPING AIDS DRUGS MAKES NO DIFFERENCE: TREATMENT BREAKS SET FOR A COME-BACK?**

**2008**, Gus Cairns, Wednesday, November 12, 2008<sup>14</sup>

*Two and half years after a major study of HIV treatment interruption was halted because of a higher risk of death in people who stopped treatment, results from a four-year Italian study reported this week suggest **no elevated risk of illness or death as a result of interrupting treatment.** The findings were presented this week at the Ninth Congress on Drug Therapy in HIV Infection in Glasgow.*

*The SMART study was a major international randomised trial of structured treatment interruption versus continuous antiretroviral therapy. Participants with CD4 counts above 350 cells/mm<sup>3</sup> on antiretroviral treatment were randomised to interrupt treatment until their CD4 counts fell below 250 cells/mm<sup>3</sup>, or take antiretroviral therapy continuously.*

*The study was halted after two years because people who interrupted treatment had an increased risk of death and HIV disease progression, and subsequent analysis showed that they also had a higher risk of **non-AIDS related serious adverse events including cancers and cardiovascular disease.***

*As a consequence of those findings, treatment interruptions, once popular, **have been warned against and discarded** as a potential strategy for managing long-term treatment.*

*So it was with some trepidation that Dr Franco Maggiolo of Bergamo, Italy, presented results from the LOTTI study, a lengthy Italian study of structured treatment interruption (STI). "My task this morning is not easy: it's a difficult thing to talk about Strategic Treatment interruptions (STIs) since the SMART Trial, but I'm here to convince you there are some good options for patients," he told the conference.*

**The LOTTI study showed that intermittent treatment of patients with CD4 counts over 350 produced clinical**

**results equivalent to continuous treatment.**

Alongside Maggiolo's presentation, a study from Dr Cal Cohen of the Community Research Initiative in Boston, USA found that **it was safe for patients to stop taking a regimen of Truvada (tenofovir+FTC) and efavirenz at weekends.**

The LOTTI study is a lengthy (four years follow-up and still continuing) study in which 329 patients were randomised either to receive continuous antiretroviral therapy (ART) or to stop treatment when their CD4 counts reached 700 and to resume when they fell below 350. In addition to having a baseline CD4 count of over 700, patients had to have a nadir (lowest-ever) CD4 count of over 200 and be on stable ART with a viral load below 50 copies/ml.

Half the patients had a baseline CD4 count over 500. The average age was 40 and 73% were male, with an even spread of **risk groups** (37% **heterosexual**, 23% **gay men** and 39% **injecting drug users**). The average time participants had been on ART was six years.

The study's primary endpoint was death from any cause, progression to AIDS or any condition requiring hospital admission. Secondary endpoints included any disease symptom not requiring admission and any grade 3 or 4 laboratory abnormality in tests.

**After four years, the proportion of patients reaching the primary endpoint was 12.1% of those on STIs and 11.6% of those on continuous treatment, a non-significant difference.** These results were achieved with the STI patients staying off therapy **two-thirds of the time** compared with less than 2% of the time in the continuous-therapy patients. Patients with CD4 nadirs exceeding 500 were able to stay off therapy **85% of the time**. After the presentation an audience member asked if Maggiolo's study just proved that **"those who should never have started can safely stop,"** but Maggiolo said that only a third of participants had a CD4 nadir over 350 cells/mm<sup>3</sup>, **so the majority had been eligible for ART under current guidelines.**

Maggiolo detailed the clinical endpoints observed in the trial. **Four patients on continuous treatment had cardiovascular disease requiring hospitalisation compared to none on STI, and six controls developed diabetes (a secondary endpoint) compared with none on STIs. In contrast, seven patients on STIs got bacterial pneumonia compared with no controls, and this was statistically significant.**

**There were significant differences in cardiovascular and metabolic events** (primary and secondary endpoints) between controls and people on STIs: they occurred in 10.5% of controls and 1% of STIs, an incidence of 0.2% a year in STI patients compared with 3.3% a year in controls. This contrasted with the SMART study which found an incidence of 1.1% per year each in both control and intervention groups for this combined endpoint.

Maggiolo commented that the average follow-up time in SMART had been just over a year compared with four years in his study and that if the LOTTI study had been stopped at the same timepoint as SMART, no difference would have been observed in cardiovascular and metabolic events either. His study demonstrated that **it took a long time for the benefits of therapy-sparing regimes to become evident.**

**He also said that in his study 95% of patients had CD4 counts over 350** compared with 65% on SMART and **0.5% below 250** compared with **8.6% on SMART**, which might explain the higher mortality and morbidity rate seen in patients off treatment in the latter.

Maggiolo commented that **doctors might like to see more evidence** before making STI's on the LOTTI model standard

clinical practice, but when asked what it would take to change clinical practice commented, **“It already is mine. It’s not for every clinician or every patient, but it’s a safe option for some”**.

Following Maggiolo’s presentation Cal Cohen presented results from the FOTO (Five On, Two Off) study which randomised 60 largely gay male patients either to continue treatment with Truvada plus efavirenz or to stop taking it at weekends.

The patients were 83% male and 77% of white ethnicity, with an average age of 47 in controls and 42 in the intervention arm and **an average CD4 count of 670**.

**After 24 weeks, 80% in the control arm and 83% in the intervention arm had a viral load under 50, a non-significant difference.** There were five dropouts in the FOTO arm and two in the control arm. Excluding these, a ‘on treatment’ analysis shows that **100% of the intervention arm had viral loads under 50 by week 24 compared with 85% in the control arm.** Most dropouts were due to the frequent monitoring required of participants, but one participant in the FOTO arm dropped out due to **efavirenz-related side effects** (dizziness and insomnia) and one before the study even began because **he was anxious about stopping his drugs**.

Eighteen patients – eight controls and ten FOTO patients – had viral load ‘blips’ during the study, defined as a single viral load between 50 and 500; the highest reading was 160 on FOTO and 465 on control.

**CD4 cells at week 24 were 705 in the control arm and 635 on FOTO, but this apparent difference, Cohen commented, has adjusted since then and the latest CD4 results are the same in both arms.**

Did patients **adhere** to the strategy? In the FOTO arm, three patients took over five doses a week at some point while eight took more than two days off, though none took more than three. **No patient who took three days off developed a detectable viral load** (viral loads were measured on Mondays). **Unlike the Maggiolo strategy, the FOTO protocol also kept patients undetectable, meaning that it did not entail the problem of raising the infectiousness of patients.**

However Cohen said he had not yet determined adherence rates in the control arm, which is obviously crucial to determining the feasibility and risks of the strategy.

Why do the strategy? Cohen asked. Apart from saving 29% of drug costs, patients much preferred it. **When asked on a scale of 0-10 whether they preferred stopping their drugs at weekends, where zero indicated total disapproval and 10 total approval, the average score was 9.5.**

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## CRIMINALIZING “HIV:”

**2008 NOVEMBER. Criminal HIV transmission and exposure laws spreading around the world ‘like a virus’**

Concern over the growing international trend towards the criminalisation of HIV transmission or exposure was documented in a Wednesday morning session at the XVII International AIDS Conference that highlighted “criminalisation creep” in Europe and Central Asia as well as the rapid spread of “**highly inefficient laws**” in West and Central Africa.

UNAIDS is so alarmed by these developments that this week they produced a new policy paper strongly suggesting that governments should repeal current laws that criminalise HIV transmission and exposure laws **with the exception of intentional transmission**.

With five major sessions over four days and least 20 different oral or poster presentations, the criminalisation of HIV transmission or exposure is one of **the burning topics** of the XVII International AIDS Conference agenda. And on Friday, South African **Supreme Court Justice Edwin Cameron** will deliver a plenary speech **calling for an unambiguous rejection of the use of criminal law to regulate the sexual behaviour** of those with and at risk of HIV.

Today, however, **the conference heard evidence that laws enabling prosecutions for HIV exposure or transmission – whether via sex, needle-sharing or from a mother to an unborn child or infant – are high on the agenda of many nations around the globe, despite the fact that there is no evidence these laws change behaviour, and growing evidence that they may inadvertently exacerbate the HIV epidemic.**

West African model law confusing and problematic. In a session entitled, ‘**To transmit or not to transmit: is that really the question? Criminalisation of HIV transmission**’, the conference heard that since 2005, Western and Central Africa has witnessed an explosion of national HIV-specific criminal exposure and transmission laws that threaten to make it one of the most legislated regions in the world for HIV.

So far, Benin, Guinea, Guinea-Bissau, Mali, Niger, Togo and Sierra Leone have passed laws in rapid succession and more Western, Central and Southern African countries are proposing similar laws, including Angola, the Democratic Republic of Congo, Malawi, Madagascar, Tanzania and Uganda.

**Most of these laws are based on the African Model Law**, created in September 2004 during a workshop by Action for West Africa Region– HIV/AIDS (AWARE–HIV/AIDS), in N’djamena, Chad. Richard Pearshouse of the Canadian HIV/AIDS Legal Network noted that **AWARE-HIV/AIDS receives USAID funding**, and is implemented by Family Health International **with additional funding from US-based organisations including Population Service International and the Constella Futures Group**. He suggested to conference delegates that they politely let these organisations know how they feel about this at their stands in the exhibition hall.

The model law comes in the guise of human rights legislation in order “to protect those who are infected and exposed to HIV,” and Mr Pearshouse pointed out that the model law does have some positive provisions, **including a guarantee of pre- and post-test counselling**; a right to healthcare services and medical confidentiality; and protection from discrimination when receiving healthcare and in the provision of goods and services.

However, Mr Pearshouse also pointed out that the model law contains a number of problematic provisions, such as the requirement that **someone newly diagnosed with HIV must disclose their status to a “spouse or regular sexual partner” as soon as possible and at most within six weeks of the diagnosis; mandatory HIV testing during antenatal care, following a rape charge, and “to solve a matrimonial conflict”;** and, most worryingly, the **extremely vague offence of “willful transmission” defined as transmission of HIV “through any means by a**

**person with full knowledge of his/her HIV/AIDS status to another person” including via sex, needle-sharing, and mother-to-child transmission.**

*He argued that the phrase “through any means” was imprecise and may end up criminalising all HIV-positive individuals, even those who practise safer sex regardless of disclosure and regardless of the actual risk of transmission.*

**Criminalising mother-to-child transmission is especially problematic, he said.** *The UNAIDS policy brief on criminal HIV transmission released this week argues that this is inappropriate because: **everyone has the right to have children, including women living with HIV**; when pregnant women **are counselled about the benefits** of antiretroviral therapy, **almost all agree to being tested and receiving treatment**; in the rare cases where pregnant women may be **reluctant** to undergo HIV testing or treatment, it is usually because **they fear that their HIV-positive status will become known and they will face violence, discrimination or abandonment**; **forcing women to undergo antiretroviral treatment in order to avoid criminal prosecution for mother-to-child transmission violates the ethical and legal requirements that medical procedures be performed only with informed consent**; and **often, HIV-positive mothers have no safer options than to breastfeed, because they lack breastmilk substitutes or clean water to prepare formula substitutes.***

***Do women really need these laws?***

*These new laws have arrived under the guise of protecting women – who have few legal or human rights in many African nations – noted Michaela Clayton of the AIDS & Rights Alliance for Southern Africa (ARASA), but, “is this what women really want?” she asked.*

*She said that **61%** of HIV-positive individuals in sub-Saharan Africa are women and that women are the often the first person in a couple to know their HIV status due to antenatal screening.*

*Women, she said, are then often blamed for “bringing HIV home” and consequently often feel unable to disclose their HIV status to their male partners due to a very real fear of physical harm and eviction.*

*In addition, due to power imbalances within relationships most women are unable to practise safer sex, since condoms are a male-controlled prevention method.*

***Under these laws, she said, it seemed likely that women as well as men will be arrested and prosecuted and suggested that these laws may deter women from accessing HIV testing and services aimed at preventing mother-to-child transmission of HIV.***

***“Criminalisation is bad public policy,” she concluded.*** *“Jurisdictions should not adopt criminalisation policies and those that have already done so should reverse course.”*

***”Criminalisation creep” in Europe and Central Asia***

*The same conclusion was asserted even more forcefully by HIV-positive advocate, Julian Hous, who presented results of an updated scan by the Global Network of People Living with HIV/AIDS (GNP+) and the Terrence Higgins Trust (THT) of **criminal HIV exposure and transmission laws in 53 countries in Europe and Central Asia.***

*A 2004 rapid scan, published in 2005, previously revealed that out of the 45 European countries surveyed, **in at least 36, the***

**actual or potential transmission of HIV can constitute a criminal offence.**

During his presentation, Mr Hows revealed that Sweden, Switzerland and Austria remained at the top of the prosecutions league table, followed by Denmark, Finland, France, Italy, Netherlands, **Norway, and the UK**. Only Albania, Bulgaria, Estonia, Luxembourg, and Slovenia had no existing or HIV-specific laws criminalising HIV exposure or transmission, **although reliable data were lacking for Greece, Monaco, Portugal, Spain, and Uzbekistan.**

However, Mr Hows pointed out that **data on prosecutions is difficult to obtain** in many countries and that **the data may “significantly underestimate prosecutions.”** He noted that **in the United Kingdom** although there have ‘only’ been **16 prosecutions and twelve convictions to date, there have been more than 100 police investigations that did not reach the court, “but which also had devastating effects on individuals, their families and their communities”** and suggested that this may also be the case in the other countries that currently prosecute HIV exposure or transmission. He added that **information on the enforced quarantine of HIV-positive individuals perceived to be a threat** – such as is the case in Sweden – is also difficult to ascertain.

**Since 2004, six countries have enacted or proposed laws that criminalise HIV exposure or transmission, including Albania, Moldova, Montenegro, Kyrgyzstan, Serbia and Turkey.** In addition, Malta, Poland, Kyrgyzstan and Serbia can now be added to the list of countries that have prosecuted at least one individual for HIV exposure or transmission. One **bright spot**, noted Mr Hows, is advocacy that has – or may have – a positive impact in four countries.

In Switzerland, the Swiss Federal AIDS Commission’s statement regarding **the lack of infectiousness of individuals on effective treatment may reverse the trend that has seen ten prosecutions and eight convictions in the past four years.**

However, last month the highest **Swiss court** ruled that **all people with HIV can be criminally liable for HIV transmission, even if untested.**

In the Netherlands, no prosecutions or convictions have taken place since 2005 due to two Dutch Supreme Court rulings in 2005 and 2007 following intense discussion between legislators, lawyers and civil society organisations. However, there has been one prosecution for intentional transmission of HIV by a needle filled with HIV-infected blood.

In the United Kingdom, new guidelines by the Crown Prosecution Service have clarified some of the uncertainties that have surrounded prosecutions for reckless HIV transmission, and given the high threshold of evidence required **it seems likely that prosecutions will become increasingly rare – in fact the last three attempts to prosecute reckless HIV transmission in England and Wales have failed.**

And, although there has been no change in law, the Ukrainian Network of People Living with HIV/ AIDS has had success in **highlighting the unreliability of phylogenetic testing** and have avoided several prosecutions being initiated.[To determine if “HIV” sequences are the same or not-phylogenetic testing].

Mr Hows concluded, however, that **“there is a slow ‘creep’ of increasing criminalisation across the countries being studied,”** and that **“laws are being introduced or being made more punitive without any regard or consideration for the evidence.”**

He noted that **“advocacy efforts to decriminalise where possible, mitigate where it is not, and ensure that laws are not introduced where there are none – are mainly noticeable by their absence”.**

## **UNAIDS argues only intentional transmission should be prosecuted**

*In an attempt to counter the growing trend of criminalising HIV exposure and transmission, UNAIDS this week published a new policy brief that strongly argues against all prosecutions for HIV exposure or transmission **with the exception of “cases of intentional transmission i.e. where a person knows his or her HIV positive status, acts with the intention to transmit HIV, and does in fact transmit it.”***

*The paper states that, **“there are no data indicating** that the broad application of criminal law to HIV transmission will achieve either criminal justice or prevent HIV transmission. Rather, **such application risks undermining public health and human rights.”***

*It argues that alternatives to criminal sanctions should be explored: “Instead of applying criminal law to HIV transmission, governments should expand programmes which have been proven to reduce HIV transmission while protecting the human rights both of people living with HIV and those who are HIV-negative”*

*Further, UNAIDS suggest that governments “strengthen and enforce laws against rape (inside and outside marriage), and other forms of violence against women and children; improve the efficacy of criminal justice systems in investigating and prosecuting sexual offences against women and children, and support women’s equality and economic independence, including through concrete legislation, programmes and services. These are the most effective means by which to protect women and girls from HIV infection and should be given the highest priority.”*

*The paper concludes with several important recommendations, including:*

*Governments should abide by international human rights conventions on equal **and inalienable** rights, including those **related to health**, education and social protection of all people, including people living with HIV.*

***Governments should repeal HIV-specific criminal laws, laws directly mandating disclosure of HIV status, and other laws which are counterproductive to HIV prevention, treatment, care and support efforts, or which violate the human rights of people living with HIV and other vulnerable groups.***

***General criminal laws should only apply to the intentional transmission** of HIV, and governments should audit the application of general criminal law to ensure it is not used inappropriately in the context of HIV.*

*Governments should redirect legislative reform, and law enforcement, towards addressing sexual and other forms of violence against women, and discrimination and other human rights violations against people living with HIV and people most at risk of exposure to HIV. Access should be significantly expanded to proven HIV prevention programmes (including positive prevention), and support voluntary counselling and testing for couples, voluntary disclosure, and ethical partner notification.*

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## **GETTING HIGH ON “HIV” DRUGS IN SOUTH AFRICA:**

December 2008 (<http://news.bbc.co.uk/2/hi/africa/7768059.stm>): BBC NEWS

Getting high on HIV drugs in South Africa, Alka Marwaha, BBC News

*“Anti-retroviral drugs used to treat HIV/Aids are being bought and smoked by teenagers in South Africa to get high.” “Reports suggest that the drugs are being sold by patients and even healthcare staff for money.” “Schoolchildren have been spotted smoking the drugs, which are ground into powder and sometimes mixed with painkillers or marijuana.”*

*“Aids patients themselves have been found smoking the drugs instead of taking them as prescribed.”*

## **THE PROBABLE CAUSE OF “HIV” HAS BEEN FOUND...IT IS ENDOGENOUS RETROVIRUS-LIKE SEQUENCES (HERVS) OR RETROIDS (RETROELEMENTS):**

The so-called template for the protein molecular signatures of “HIV” may derive from endogenous DNA sequences (coming from cellular origin instead of viral origin). These cellular proteins are expressed under certain conditions by normal uninfected yeast, insects, dogs, rhesus monkeys, chimps, and humans. “HIV” is said to have 9150 base pairs, but again, this template has not been purified without contaminating cellular nucleic acids. So, it is likely that “HIV’s” molecular markers could represent the by-products of our cells, which were incorrectly called HERV’s (**H**uman **E**ndogenous **R**etrovirus (**59**) nucleic acid sequences (incorrectly called HERV’s because they have not not proven to be viruses, or to cause human disease), or more likely, what is called a ‘retroid’ of one kind or another (**60**). That these hypothesis are possible has been shown again and again to be likely from studies with “HERV’s” such as “the Phoenix viruses,” that are produced by infecting cells with certain sequences of DNA, which then are packaged by the cells into viral-like particles.

Although the infectious or disease-causing ability of “HERV’s” or their viral nature haven’t been proven, modern analyses of the Human Genome Database reveal more than 120, 000 full-length retroids containing reverse transcriptase transcripts. It might be prudent to simply consider “HIV” markers as expression products of retroids that are now being discovered through genomic sequencing. Although the Promoters of “HIV=AIDS” are always saying the “HIV virus” reverse transcriptase sequence is mutating when patients die on anti-retroviral drugs that supposedly target this and other “HIV-enzymes,” genomic analyses show that reverse transcriptase is among the most stable transcripts that make up these retroids, and it is the sequence stability rather than the instability or mutability of the reverse transcriptase sequence itself that make these 120,000 retroids possible to classify.

The vast amount of evidence shows that the probable “cause of “HIV” are retroids and/or endogenous “HERV” sequences, that can be evoked, under stress conditions, or which may become expressed in healthy persons as part of a relatively rare genetic polymorphism. The science of genomics itself raises much doubt regarding the tacit assumption and arrogance that we know all there is to know about the human genome, or under what circumstances we may express novel but perhaps stereotypic gene sequences.

Someday, it may indeed be firmly established that there may be a relationship between “HIV’s” molecular markers and immune disorders in some individuals who harbor a plethora of different immunological conditions or diseases, but the ten million dollar



question science has not been permitted to ask about these individuals is: which comes first? Which is cause and which is effect, and what is the meaning of the presence of molecular markers of “HIV” in a healthy person who tests “HIV-positive”?

What is perhaps also the most remarkable fact about the history of AIDS, is that reverse transcriptase was once thought by all working in AIDS research who have promoted “HIV/AIDS,” to be specific to retroviruses, and this is the enzyme they first measured, and indeed some labs continue to measure, as evidence of “HIV infection.” However we are all made up partly of retroviral components, they are part of us. They are most accurately referred to as retroids. What they call “HIV” and what they have successfully branded as the most dangerous and infectious virus known to man that is said to confer a near 100% mortality rate like the Andromeda Strain of Hollywood “if not treated with life-saving AIDS drugs,” is simply expressed, or can be evoked in many of us, because it is made by our cells, and the expression of these markers can be submerged with ARV’s. The Promoters of “HIV/AIDS” have mistaken 237 endogenous normal cellular proteins for a “virus” for more than two decades now, the flawed technologies for detecting these markers have never been disclosed to the public, and without any of the sober analysis of what those tests are actually detecting or what “HIV’s” molecular signature means for a human being, millions of lives will continue to be ruined.

Other so-called “HIV-specific” sequences, such as those that give rise to the so-called GAG, PR, RT, ENV molecules are also found in the normal Human genome database. In gene bank searches, one can find 16 samples of spuma virus transcripts, 6 examples of snakehead virus, 16 samples of FIV (feline immune deficiency virus), 60 examples of detecting one or more HBV (hepatitis B virus) genes, and at least 11 cases of “HIV” sequences that are said to be scattered throughout the normal Human genome, according to the analyses of McClure and other Human Genome Database analysts.

Although Dr. Gallo and others have claimed that in a stadium full of “HIV-negative” people, not one molecule of “HIV” will be present, the DAIDS (Division of AIDS) culturing manual says that if “HIV-infected” cells from human blood express more than 30 units of “HIV-specific” p24 protein on 2 or 3 separate tests (30 pg/ml), one is considered “HIV-positive,” and if one sleeps with somebody without telling them they have these 30 or more units, one can be tried for attempted murder, one can’t obtain health insurance, one might be fired from his or her job, one might commit suicide, if pregnant one may be frightened into aborting her baby. If your cells express less than 30 units of this protein 2 or 3 separate times (pg/ml), then one is considered non-“HIV-infected” and is home free-one can donate blood, sleep with anyone he or she wants, without telling them his or her “less than 30 status,” etc. How could this be possible if there isn’t one molecule of “HIV” in a stadium full of “HIV-negative” people? Its an arbitrary measurement of a molecular signature that may have nothing to do with a virus or immune suppression that is arbitrarily being measured at more than 30 units for an “infected” person, and less than 30 units for a non-infected person.

P24, by the way, which supposedly is an essential “HIV” protein, is also found in the thymus gland cells of non-infected “HIV-negative” children.

The confusing thing may be that some of these endogenous cellular DNA or RNA sequences are only expressed rarely, or in response to physiological stresses: they aren’t infectious, and they may represent as much a 8% of the normal human genome according to some scientists.

“HIV’s” molecular signature has nothing to do with a specific exogenous virus: the molecular signature thought to be a virus may in fact be generated also in response to previously latent real viruses that at some point of physiological stress provokes a new and complex immune response, which is read as “HIV’s molecular signature. The immune system of a person so infected by multiple or numerous latent real viral infections could be perpetually generating new immunogens, which is read by AIDS scientists as an ever changing and mutating “HIV.” In theory, such an immune chain reaction caused by multiple real viral or bacterial or fungal infections would be progressively more debilitating for the stability and effectiveness of immune function, and, a vaccine against any specific virus or other pathogen would be ineffective against the development of AIDS. If this hypothesis is correct, then an experimental animal model of AIDS should be induced in laboratory animals by infecting them at a low multiplicity with a very large

number of diverse viruses, as was suggested one by Nobelist, and PCR-inventor, Kary Mullis, in a Genetica paper he wrote in 1995. The allo-immunization hypothesis of Root-Bernstein and the oxidation hypothesis of the Perth group also deserve careful investigation.

What is the probable cause of “HIV?” In the scientific community, there is increasing interest in how “HERV’s” may serve normal cellular functions like trophoblast fusion, and also how they may function in disease states. For example, reviews of this subject typically present information that equates so-called “viral” sequences to Human endogenous retroviral sequences as can be seen in references such as (61).

*Human endogenous retroviruses (HERVs) have a similar structure to the proviruses of infectious retroviruses but typically contain many inactivating mutations including point mutations (dark bands), frameshifts and deletions (particularly in env). Frequently, the entire central portion has been lost by homologous recombination, leaving behind a ‘solitary LTR’. Although almost all HERVs are defective, the LTRs may still be active, and **transcription of HERVs is common particularly in fetal tissue and in inflammatory disease and cancer**. In a few cases, coding competence has been retained for env even when adjacent viral genes **are heavily mutated**, suggesting that selective pressures have maintained these open reading frames **because they serve a cellular function**.*

### **HERVs and disease**

*HERVs have frequently been proposed as etiological cofactors in chronic diseases such as cancer, autoimmunity and neurological disease [13]. Unfortunately, despite intense effort from many groups, there remains little direct evidence to support these claims, and moreover some studies have served only to muddy the waters for others. One particular difficulty has been picking out the coding-competent subset of HERVs from the large background ‘noise’ of defective elements. The clinical heterogeneity of many of the associated diseases, such as lupus erythematosus, rheumatoid arthritis and multiple sclerosis, has also been a problem, since HERVs may be involved in specific subtypes of a particular disease and **such subtypes may not be recognized by current diagnostic criteria**. The availability of the human genome sequence will facilitate the identification of those HERV loci most likely to be involved in disease. In addition, **a deeper understanding of the genetic basis of these diseases should lead to a more precise definition of disease subtypes**. In turn, this may clarify the part played by HERVs.*

***Much of the evidence that links HERVs to disease comes from the detection of expressed retroviral sequences in patient tissue by degenerately primed PCR. For example, HERV-W was first identified in a search for retroviruses in people with multiple sclerosis [14], and the same HERV was recently detected in both cerebrospinal fluid and brain tissue from patients with schizophrenia [15]. The significance of HERV RNA expression in studies such as these remains unclear, because disease causation cannot be proved simply by the detection of virus expression, particularly for a ubiquitous sequence such as a HERV. In addition, although HERV RNA expression is known to be increased in several autoimmune diseases and cancers, there is usually activation of a range of class I and class II HERVs rather than expression of a single provirus.** In general, further validation of disease association for HERVs has not been described. An exception is the HERV-K(HML-2) subgroup, **which has been implicated in germ-cell tumors**. As noted above, this subgroup includes the youngest and most active HERVs, and **specific attention has focused on the ability of these elements to form virus-like particles in teratocarcinoma-derived cell lines [16].** Some of these particles are able to bud from the cell surface, **although it is doubtful whether they are infectious**. In addition, **patients with germ-cell tumors frequently have antibodies to HERV-KGag and Env proteins [17].** Recent work has shown that a regulatory protein produced by HERV-K(HML-2) can bind the transcription factor PLZF (promyelocytic leukemia zinc finger protein), which is required for spermatogenesis [18]. Impairment of spermatogenesis is associated with increased frequency of germ-cell tumors, and thus perturbation of PLZF function could provide a mechanism for the involvement of HERV-K in tumorigenesis.*

Clearly, many questions remain. Are HERV-K particles produced from a single intact provirus or does trans-complementation of proteins from several loci lead to the production of composite particles? Are any of these particles infectious and do they contribute to tumor formation? The human genome sequence may be able to help to address these issues by identifying those copies of HERV-K(HML-2) that are most likely to contribute to particle production. Comparison of these loci between patients with germ-cell tumors and controls may then reveal differences which could be the focus of further research.

**“HIV” “VIRAL LOAD” OR CD4-DEPLETION NO LONGER CAUSES AIDS LYMPH NODE FIBROSIS CAUSES AIDS.**

THE PUZZLE OF CD4-CELL DEPLETION DESPITE GOOD VIRAL SUPPRESSION:

[Abigail Zuger, MD](#)

Published in AIDS Clinical Care June 1, 2009

In **some** patients, CD4-cell counts **fail to rise as expected**. Could extensive lymph node **fibrosis** be responsible?

**We expect** that when combination antiretroviral therapy (ART) **suppresses** a patient's **HIV viremia [dampens retroviral production]**, a steady increase in CD4-cell count **will ensue**. In **some** patients, **however**, such increases **are minimal or fail to occur**, and in others, CD4-cell counts **plummet** after **an initial rise**, even though **viral load** [retroviral expression] remains undetectable. The combination of ddI and tenofovir has been **associated** with these **aberrant** CD4-cell responses, but the underlying mechanism is **unclear**, and the phenomenon is also seen in patients taking **other drugs**.

In a recent study, NIH researchers sought evidence to support **any of several hypothetical explanations** for the aberrant CD4-cell responses seen **in four patients** on combination ART whose CD4 counts had fallen from a median of 719 cells/mm<sup>3</sup> to a median of 227 cells/mm<sup>3</sup> **despite persistently** undetectable **plasma viral** loads [retrovirals]. Three of the four patients were receiving a regimen containing tenofovir and ddI.

**Residual replicating HIV did not seem to be the problem: Results of ultrasensitive PCR and assays for peripheral blood mononuclear cell-associated HIV RNA and proviral HIV DNA – and of assays for cell-associated HIV RNA and proviral DNA in mononuclear cells from inguinal lymph nodes – were similar to those obtained in other, successfully treated patients. No evidence of occult drug resistance sabotaging treatment was found. Changing ART regimens to avoid the tenofovir/ddI combination had little effect on CD4-cell counts during the follow-up period (median duration, 10 months).**

The single unusual finding was **a striking abnormality in inguinal lymph node architecture** in the four patients: From 24% to 34% of the T-cell zone was replaced by collagen. In contrast, collagen levels in six successfully treated patients have been reported to range from 2% to 12% (*J Clin Invest* 2002; 110:1133).

**Comment:** We do not know the exact pathogenesis of CD4-cell depletion in untreated HIV infection, so creating logical hypotheses to explain aberrant CD4 responses is especially challenging. These authors offer the intriguing suggestion that the unusual lymph node architecture documented in all four patients in this study may be related to (or even responsible for) the inadequate CD4-cell response – i.e., that CD4-cell depletion is independent of specific components of an antiretroviral regimen and is instead caused by lymph node fibrosis. They note that such architectural damage may well be “clinically irreversible with currently available interventions.”

**Citation(s):**

Nies-Kraske E et al. Evaluation of the pathogenesis of decreasing CD4<sup>+</sup> T cell counts in human immunodeficiency virus type 1–infected patients receiving successfully suppressive antiretroviral therapy. *J Infect Dis* 2009 Jun 1; 199:1648.)

**JOHNS HOPKINS SAYS THAT AS THEY EXPECTED, THE MAJORITY OF AFRICAN STD PATIENTS HAD SUBTYPE A OR D, AND NINE OF THEM (89%) HAD DEMENTIA BECAUSE OF SUBTYPE D, AS THIS AIDS VIRUS CAUSES MORE BRAIN INFLAMMATION THAN STRAINS A, B,C,E,F,G, H,I, J, AND K.**

2009 (August)

<http://www.newswise.com/articles/hiv-subtype-linked-to-increased-likelihood-for-hiv-subtype-linked-to-increased-likelihood-for-dementia>

HIV Subtype Linked to Increased Likelihood for Dementia

Released: 8/28/2009 3:20 PM EDT

Source: Johns Hopkins Medicine

Newswise —

*Patients infected with a particular subtype of HIV, **the virus that causes AIDS**, are more likely to develop dementia than patients with other subtypes, a study led by Johns Hopkins researchers shows. The finding, reported in the September *Clinical Infectious Diseases*, is the first to demonstrate that the specific type of HIV has any effect on cognitive impairment, **one of the most common complications of uncontrolled HIV infection**. HIV occurs in multiple forms, distinguished by small differences in the virus' genetic sequence and designated by letters A through K. Certain subtypes appear to cluster in particular areas of the world, and others have been associated with different rates of progression to full blown AIDS. Of the 35 million people living worldwide with HIV, the majority live in sub-Saharan Africa, where subtypes A, C and D dominate. Nearly half of patients with advanced HIV infections have at least mild cognitive impairments, and about 5 percent have the severe form of cognitive impairment known as dementia.*

*In earlier research, Ned Sacktor, M.D., and his colleagues found that about **31 percent of patients visiting an infectious disease clinic in the Ugandan capital, Kampala**, where subtypes A and D dominate, had dementia. The finding led him and his team to wonder whether patients with different subtypes had different rates of dementia. Sacktor, professor of neurology at the Johns Hopkins University School of Medicine and a clinician at the Johns Hopkins Bayview Medical Center, and his colleagues studied 60 HIV-infected patients from a Kampala clinic. **All of the subjects had been part of a different study testing the effect of anti-retroviral drugs on cognitive impairment, but had not begun taking the drugs.** After determining each patient's HIV subtype, they performed a battery of neurological and cognitive tests to assess each patient's brain function.*

*As expected, the majority of the patients had HIV subtypes A or D. Out of the 33 subtype A patients, the researchers determined that seven had dementia, or about 24 percent. However, out of the nine patients with subtype D, 8 had dementia, about 89 percent.*

*"We were amazed to see such a dramatic difference in dementia frequencies between these two subtypes," Sacktor says. "If this is the case in all of sub-Saharan Africa, HIV-associated dementia may be one of the most common, but thus far unrecognized, dementias worldwide."*

*The research suggests that some biological property of each subtype seems to influence the likelihood that infected patients will*

develop dementia, says Sacktor. He and his team hypothesize that subtype D may cause more inflammation and injury in the brain, a possibility they are currently investigating.

For more information, go to:

[http://www.hopkinsmedicine.org/neurology\\_neurosurgery/experts/team\\_member\\_profile/37D60115A5A2EF0013ECA0BB627B797A/Ned\\_Sacktor](http://www.hopkinsmedicine.org/neurology_neurosurgery/experts/team_member_profile/37D60115A5A2EF0013ECA0BB627B797A/Ned_Sacktor) : [http://www.hopkinsmedicine.org/neurology\\_neurosurgery/](http://www.hopkinsmedicine.org/neurology_neurosurgery/)

**THE WHO CRITERIA OF TREATMENT FAILURE ARE NOT SENSITIVE OR SPECIFIC IN HAITI: WAITING TO MEET THESE CRITERIA MAY DELAY THE RECOGNITION OF VIROLOGICAL FAILURE AND RESULT IN THE ACCUMULATION OF HIV-1 DRUG-RESISTANCE MUTATIONS.**

**2009** August) NEMJ Volume 361:828-829, August 20, 2009, Number 8.

***5-Year Survival of Patients with AIDS Receiving Antiretroviral Therapy in Haiti***

To the Editor:

*We report 5-year outcomes of 910 adults with human immunodeficiency virus (HIV) infection who consecutively initiated antiretroviral therapy according to guidelines of the World Health Organization (WHO) from 2003 through 2004. All the patients were followed at the Groupe Haïtien d'Etude du Sarcome de Kaposi et des Infections Opportunistes (GHESKIO) clinic in Port au Prince, Haiti, through May 1, 2009.<sup>1,2</sup> Combination therapy with zidovudine, lamivudine, and efavirenz was initiated in 428 patients (47%); therapy with zidovudine, lamivudine, and nevirapine in 381 patients (42%); and other regimens in 101 patients (11%). CD4 counts were measured every 6 months, and HIV type 1 (HIV-1) RNA levels were measured when patients met WHO clinical criteria or CD4 criteria for failure of antiretroviral therapy. The study was approved by the institutional review board at the GHESKIO Centers in Haiti and at Cornell University in New York.*

*Of the 910 patients, 70 (8%) were lost to follow-up, and 208 (23%) died. For 738 patients who were receiving care at 6 months, 587 (80%) had an adherence level of 90% or more. According to Kaplan–Meier analysis, 79% of the 910 patients were still alive at 60 months (Figure 1). The rate of death in the first 6 months (25 deaths per 100 person-years) was seven times the rate after 6 months (3.3 deaths per 100 person-years) ( $P<0.001$ ). Deaths during the first 6 months were associated with having an AIDS-related illness, a weight in the lowest quartile, and a CD4 count of less than 50 cells per milliliter ( $P<0.001$  for all comparisons). Deaths after 6 months were associated with an adherence rate of less than 90% ( $P<0.001$ ), an age of more than 50 years ( $P=0.009$ ), and a diagnosis of tuberculosis during the first 6 months of antiretroviral therapy ( $P=0.02$ ).*

*Of the 910 patients, 121 (13%) had a drug-related toxic effect that prompted a change in first-line therapy: anemia associated with zidovudine in 51 patients, central-nervous-system symptoms associated with efavirenz in 25 patients, gynecomastia in 20 men receiving efavirenz, rash associated with nevirapine in 15 women, and other effects in 10 patients. A total of 263 patients (29%) met the WHO clinical criteria or CD4 criteria for treatment failure. Of the 211 patients for whom data regarding HIV-1 RNA levels were available, 113 (54%) had a plasma HIV-1 RNA level of more than 50 copies per milliliter. The positive predictive value of clinical criteria (WHO stage III or IV HIV-related symptoms) for detectable HIV-1 RNA was only 48%. The positive predictive value of CD4 criteria was 77%. HIV-1 reverse-transcriptase genotyping was performed for 91 patients who had a plasma HIV-1 RNA level of more than 1000 copies per milliliter at the time of treatment failure, according to WHO criteria (see the table in the Supplementary Appendix, available with the full text of this letter at NEJM.org).*

*We also analyzed HIV-1 RNA levels in banked plasma from 405 control subjects in the same cohort who did not meet the WHO*

*criteria for treatment failure. Two control subjects were selected for each patient with treatment failure with the use of incidence density sampling, with matching for age, sex, and length of follow-up. Of the 405 control subjects, 63 (16%) had a plasma HIV-1 RNA level of more than 50 copies per milliliter. The sensitivity of the WHO clinical and CD4 criteria for predicting virologic failure was 113 of 176 patients (64%), and the specificity was 342 of 440 (78%).*

*This report documents the long-term sustainability of antiretroviral-therapy programs in resource-poor countries, with excellent rates of retention and adherence and a survival rate of 75% at 5 years. The WHO criteria of treatment failure are not sensitive or specific. Waiting to meet these criteria may delay the recognition of virologic failure and result in the accumulation of HIV-1 drug-resistance mutations.*

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**ONE WOMAN IN CAMAROOON CATCHES "HIV" FROM A GORILLA AND LIKE THE ANDROMEDA STRAIN, IT MUTATES IMMEDIATELY TO FORM A NEW, RAPIDLY GROWING, PROBABLY WIDESPRED, DEADLY PANDEMIC AIDS VIRUS.**

**Presented in its entirety in “Sacrifice of the Virgins:**

*“The **most likely explanation** for the new find is **gorilla-to-human transmission**, Plantier’s team said. But they added they cannot rule out the possibility that **the new strain started in chimpanzees and moved into gorillas and then humans, or moved directly from chimpanzees to both gorillas and humans.**”*

*“The 62-year-old **patient** tested positive for HIV in 2004, shortly after moving to Paris from Cameroon, according to the researchers. She had lived near Yaounde, the capital of Cameroon, but **said she had no contact with apes or bush meat**, a name often given to meat from wild animals in tropical countries. **The woman currently shows no signs of AIDS** and remains untreated, though she still **carries** the virus, the researchers said.”*

**SCIENTISTS HAVE DISCOVERED A NEW VIRUS CAUSING AIDS.** News: World by Nikolai Terziev January 17, 2010

*Chinese scientists have discovered a previously unknown virus that can cause a syndrome of acquired immune deficiency (AIDS). Until now it was believed that a person may develop AIDS only after infected with HIV.*

*But Chinese scholars argue that there is another virus that has yet to be studied and led to the development of the disease. “Thousands of patients who have AIDS have tested negative for HIV test,” said one of the doctors. Experts explain that this hypothetical mutation of HIV is more aggressive and dangerous when transmitted through sex.*

*Studies of the new virus began on Monday and still no clear date when results will be announced.*

**INNATE IMMUNITY TO “HIV” IS PROBABLY MEDIATED BY “TETHERIN,” WHICH CAN INHIBIT THE RELEASE OF HIV-1 FROM INFECTED CELLS.** NEW ENGLAND JOURNAL OF MEDICINE 362:6 February 11, 2010. John Guatelli, M.D 2010 (February)

*The innate immune response to viral infection includes an antiviral state induced by type I in-*

*terferons. What are the effector proteins of this antiviral state, and how do they work? A recent article by Perez-Caballero and colleagues<sup>1</sup> indicates that the interferon-induced protein **bone marrow stromal-cell antigen 2 (BST-2)**, known as **“tetherin”** in the human immunodeficiency virus (HIV) research community, prevents progeny virus particles (virions) from escaping from the surface of the infected cell through a direct mechanism that requires only the protein’s general structural features.*

*The name tetherin was coined 2 years ago, when BST-2 (also known as CD317 and HM1.24*

*antigen) was first recognized as a host factor that inhibits the release of enveloped virions from infected cells.<sup>2,3</sup> **This protein, previously best known for its high levels of expression on the surface of malignant cells in multiple myeloma,** was shown to be a target of the HIV type 1 (HIV-1) protein **Vpu**. Vpu had long been known to enhance the release of virions from infected cells, apparently by counteracting a cellular protein that inhibited this release. **Though expressed constitutively in certain cell types, this inhibitor was shown to be induced in others by type I interferons.**<sup>4</sup> The search for an interferon-induced protein that restricted virion release and was counteracted by Vpu led to the discovery of tetherin.<sup>2,3</sup> Once its antiviral activity was identified, **the known spectrum of action of tetherin expanded rapidly to include all retroviruses tested, as well as members of the filovirus (Ebola and Marburg viruses), arenavirus (Lassa virus), and herpesvirus (Kaposi’s sarcoma–associated herpesvirus) families.** Several of these viruses are now known to*

encode proteins that counteract tetherin, although these antagonists are distinct — both structurally and mechanistically — from Vpu.5

But how exactly does tetherin retain virions on cells? **A potential clue came from the protein's unusual topologic features: it binds the lipid membrane twice, once via a transmembrane domain and again via a glycosylphosphatidylinositol (GPI) lipid anchor (Fig. 1).** With these features, **tetherin could embed** one end in the virion lipid envelope and the other in the plasma membrane of the host cell, **tethering HIV-1 to the cell surface. Alternatively,** since the extracellular domain of the protein **probably** adopts an interactive “coiled-coil” structure (a twisted ropelike configuration), it could interact with itself to retain virions on cells. **Each of these models suggests that tetherin directly and autonomously restricts release, and each suggests that the protein is embedded wholly or in part within the envelope of the virion.** Perez- Caballero and colleagues show that this is indeed the case. Tetherin can insert itself into the **virion membrane** by way of either of its ends. It is appropriately situated along the cell surface to retain **nascent virions.** Each structural feature of the protein (i.e., its transmembrane domain, coiled-coil region, and GPI anchor) is required for antiviral activity. Remarkably, these structural features are all that is necessary for function: an “artificial tetherin” that the authors stitched together **from three different cellular proteins** had antiviral activity nearly equivalent to that of native tetherin itself. **However, HIV-1 Vpu did not counteract the effects of the artificial tetherin.**

What opportunities do artificial tetherin and the relationship of HIV-1 Vpu (and other viral antagonists) with native tetherin provide for antiviral therapy? **In principle,** artificial tetherin could be expressed in cells as a therapeutic molecule, **since it is likely that no virus would be able to counteract it.** More generally, the identification of tetherin as an antiviral effector protein of the innate immune response may invigorate interest in Vpu and other viral proteins that antagonize tetherin as drug targets. **“Accessory” viral proteins such as Vpu have not generated high enthusiasm** as drug targets, in part because — **unlike viral enzymes such as the HIV-1 reverse transcriptase, protease, or integrase — they are not necessary for viral replication in vitro.** **However, in vitro culture systems may not reflect the importance of such proteins in vivo.** Is tetherin an important host defense, worthy of an effort to sensitize viruses to it pharmacologically? The fact that viruses devote genetic information to encoding tetherin antagonists suggests that the answer is yes. **Inhibiting such viral proteins could in principle** enable the innate immune response to trap HIV and other enveloped viruses at their source.

From the University of California, San Diego, and the Veterans Affairs San Diego Healthcare System — both in San Diego.

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## CONCLUSIONS



The information and ideas I have presented in this brief History of AIDS are not an elaboration of the logic of the retrovirologist, Peter Duesberg's "harmless passenger virus hypothesis," but rest, in part, upon some of his early and correct criticisms/predictions of the "HIV=AIDS" hypothesis, especially the erroneous medical notions that Dr. Duesberg first pointed out (tuberculosis or cancer plus a positive "HIV" test equals AIDS, whereas tuberculosis or cancer minus an "HIV-positive test is plain old tuberculosis or cancer). Nor is the information and ideas presented here simply an elaboration of the logic and ideas elegantly developed by the Australian-based Perth Group regarding no proof for the existence of "HIV," but yet rests in part upon much of their correct thinking and demonstrations that revealed why an "HIV-virus" was never isolated as an exogenous virus in any lab. Because of the Perth's group's initial analyses, for instance, studies have demonstrated again and again why all "HIV" tests are flawed, especially those performed on so-called "low-risk groups," and why both "HIV" cell cultures and "AIDS patients" tend to exhibit imbalances in redox states.

The arguments of Duesberg and the Perth group are still not even discussed, or heard in the halls of academia or medicine, nor are they even considered important enough to address, according to the promoters of AIDS in the context of their mainstream beliefs. Every "educated person" **knows** for example, that when patients die of their cancer or their "HIV-infection," that it is the biological concept of mutation that is at the basis of failing to stop or reverse "mutating cancer cells" or mutating "HIV." In this context, if a metric ruler is used to measure some distance rather than a ruler etched with inches, there can be a complete disconnect between those who say an object is 2.54 centimeters long, and those that say it is one inch long, and no communication is possible. The molecular cloning tools, and the classical standards for viral isolation are such discordant measuring devices. Added to this confusion are new yard-sticks of diagnostic pathogenesis: the processes of tumor-erected matrices, lymph node fibrosis, and bacterial biofilms. If lymph node fibrosis, rather than "viral load," CD4+ T-cell counts, or the appearance of any of no less than 58+ AIDS-indicator illnesses now considered by those on the front line of AIDS clinical care to be the hallmark of true AIDS progression, than all the clinical trials conducted so far during the AIDS era need to be re-evaluated with this new yard-stick.

The modern vaccine paradox presented by the recent failed and halted "HIV" Thailand/military-monitored vaccine trial, and the 63 failed ones that preceded them, also must be explained using all measuring sticks, and these many failures can be viewed simultaneously through the glasses of molecular cloning and through the glasses of the classical rules of pathogen isolation and lymph node fibrosis if, and when, a new system of measurement, diagnosis, and treatment is considered that incorporates all of these detection methods. It is in this context that I hope progress and understanding here can be achieved, and significant progress in disease detection and treatment will proceed.

One primary assumption from which I derive my presentation to you here, is based on what might be described as a simple immunological "set and sub-set theory," erected upon the results of experimentally rigorous, current, and numerous experiments, conducted in the vaccinated human context (as opposed to limited experiments using animal models, culture models, arguments about isolation, electron microscopy findings, passenger viruses, etc., which form the foundation of the "Pasteur" rules and standards for pathogen isolation and proof that a pathogen will be associated with a disease state).

In this context, the failure to evoke an "HIV-specific" immunological reaction in a majority of vaccine recipients after 64 failed "HIV" vaccine trials could be interpreted in several different ways: even if an ill-defined slurry of mostly cellular proteins and debris, rather than a pure viral isolate had been used to make vaccines from material derived from persons suffering from immune suppression or who are healthy, and who express the markers of "HIV-disease," or from cultures made from such persons, true seroconversion amongst vaccine recipients should have occurred.

It also is in this context that it should be remembered that Peter Duesberg's critics once said that, if "HIV" was a harmless passenger virus, then he ought to inject himself with it and prove it harmless.

But for the many reasons I will elaborate upon, it became clear from the beginning that this [hypothetical] experiment was correctly

(and responsibly) seen by all to be impossible.

Even after “Roman” efforts by scientists, vaccine makers, genome analysts, and other organizations in many nations, “HIV” virus particles could not be isolated partially, or isolated at all, according to many who have published on this issue, without also a host of cellular nucleic acids and proteins such as actin, exrin, and perhaps as many as 237 other molecular markers that have since been linked to “HIV-disease.”

Also, the proposed Duesberg “self-injection experiment” was impossible because of the proposed “lag” time of this “new” “slow” “virus,” perhaps a wrongly framed idea in the first place, but an idea nevertheless that made such an experiment impossible to carry out to anyone’s satisfaction, regardless of their beliefs regarding the harmfulness, harmless, or non-existence or existence of a real “AIDS virus,” and amidst an almost conscious ignorance of the characteristics of so-called “high-risk” individuals.

Regardless of the fact that no pure isolates have ever been obtained in the case of “HIV” (and many other viruses), and no unequivocal link to causation of any specific virus to the syndrome of acquired immune suppression, fibrosis, or cancer, has been achieved in human populations, it should be clearly said that immunologically, purity of isolation shouldn’t matter if “HIV” were truly an exogenous virus, contrary to the Australian Perth group’s contention, even though their principal theoretical points of contention regarding true isolation have been strenuously and correctly argued in the literature and even in courts of law.

In the context of true seroconversion, or failure to truly seroconvert to a “true” “HIV-positive” pattern on a WESTERN blot in 64 failed “HIV”-vaccine trials, even after “HIV’s” so-called specific proteins were initially injected into the experimentally vaccinated so that inefficiency of exposure or route of exposure is not at issue, all truly specific and truly “foreign” proteins, lipids, or nucleic acids in the mixture should in theory be identified by the immune system, along with any proteins, lipids or nucleic acids that have become denatured during the vaccine making process, and a specific antibody directed against any and all of these foreign molecules or “epitopes” should be manufactured at some weeks or months after inoculation. Foreign antigens should be detectable as specific antibodies made against any and all foreign or denatured molecules. This is true by definition of current immunological antibody-antigen science.

In this sub-set, self versus non-self hypothesis, and when an immunologically competent population of injected humans, rabbits, rodents, or goats are challenged to exhibit the normal immunological process of antibody production against molecular foreigners, or against denatured self proteins or molecules, any foreign or denatured molecules should be detected and antagonized, even when only one or several or them are present in the ill-defined, mixed slurry of 6 or 10 or 25,000 self or non-denatured molecules.

This science and concept forms the basis of the biotech industry that provides biological laboratories with antibodies such as anti-tubulin, anti-fibronectin, anti-actin, anti-intermediate filament proteins, and indeed all antibodies we use on a daily basis. Even if the anti-tubulin in one manufacturer’s preparation wasn’t purified, and even if all of the accessory proteins such as tau, GTP-binding proteins, and a plethora of others isn’t removed adequately to isolate pure tubulin, we’d still see a signal under the microscope or on a WESTERN blot of the unique cow-brain obtained tubulin identified as a foreign protein, despite contamination with the others.

The failure to meet even the lowest standards of this immunological sub-set reaction hypothesis, also makes likely, or proves yet again, that “HIV” tests frequently identify false positives, and factors that are not due to an exogenous (AIDS) virus, “HIV.” In other words, and in the words of John Rappaport, who wrote “AID\$ Inc, Scandal of the Century,” no “HIV” vaccine recipient after the halted Thailand “HIV” trial of 2009, or the halted STEP trial of 2008, or the 62 trials that preceded them, would have needed to carry around a government issued letter as official evidence that their “HIV-positive status” was due to a vaccine, instead of risky behaviors. Why? Because no “HIV” vaccine recipient has truly seroconverted after 64 vaccine trials, in a manner that can be interpreted as immunologically constituting a true antigen-antibody conversion to “HIV’s” molecular profile, rather than simply a false positive “HIV” test result or for many other reasons.

If there is a high frequency of specific antibody reaction or seroconversion against even one or several truly foreign or denatured self-molecules in a vaccine mixture, despite contamination or co-existence with thousands of non-foreign or non-denatured proteins or substances, and despite the presence or absence of adjuvants, novel seroconversion against those foreigners or denatured molecules should occur.

An antibody not found in normal “uninfected contexts” should be evoked, even if by only one out several thousand that are initially injected and not foreign.

The foreign or denatured self-molecule, despite not being isolated from all other objects in the universe, should generate a different and novel antibody from all those derived from self which are not foreign or denatured, or which are not found in normal “uninfected” contexts.

Yet according to the vaccinology experts, there hasn't been one found yet.

In other words, the promoters of AIDS did a similar experiment to the one they wanted Dr. Peter Duesberg to perform on himself, but instead did it on thousands of people thus making the experiment far more rigorous: they injected many ill-defined molecules thought to be from foreign “isolated HIV” (proteins or GP 120, env, rev, or other moieties) into thousands of persons that have participated in at least 64 controlled trials that tested both experimental vaccines and placebo-containing vaccines. None of these have to date, according to them, been able to evoke “HIV's” complete or even minimally partial molecular profile in a single vaccinated individual, despite their claims that 33 million of us carry the same exact foreign “HIV” molecules in our bodies from “infection,” that constitute “HIV's” exogenous parts, or represent our body's reaction to them.

Of those few who did appear to partially seroconvert or perform differently on an “HIV” test after vaccination or booster vaccination in the recent Thai or STEP trials that were abruptly halted because **as many of the vaccinated “sero-converted” as the placebo group**-which is non-sense-because none of the numbers were statistically significant in the first place, then those persons who “seroconverted” were in no way significantly more numerous seroconverters than control vaccine recipients. This is a crucial point that points to the fact that “HIV” molecular profile is most likely represents what genome analysts now call retroids, and which a former generation called HERV's (Human Endogenous Retrovirus Sequences), together with non-specific molecular interactions as was argued by Perth and many others. Otherwise, why would a flu vaccine or hepatitis B vaccine cause an “HIV-positive” false test if this were not the case?

Doesn't this history show that with respect to understanding the significance of the molecular profile(s) of “HIV,” that it would be more prudent to adopt a point of view consistent with the cellular and immunological facts that we do know about immune suppression more generally? In other words, shouldn't we in the scientific community assume that profound immunosuppression is caused by a myriad of factors and that it is incorrect to group these syndromes under a single disease entity, “HIV/AIDS?” The consequences of this view also suggest that different treatment strategies are needed for the spectrum of the previously known “AIDS-indicator diseases,” rather than the toxic and dangerous pharmaceutical or vaccination approaches currently in place. With lymph node fibrosis becoming the new marker for profound unresponsive therapy in the absence of correlated viral load or CD4+counts, all clinical trials that have measured these correlates on patients placed on HAART need to now be evaluated for the frequency of this new marker.

Perhaps the scientists, doctors, public health officials, governments, and “activists,” and media magnates who continue to promote the “HIV=AIDS=death” paradigm (lets call them **The Promoters of “HIV/AIDS”**) will begin to someday ask themselves why “HIV” components aren't immunogenic or able to prevent AIDS progression in the immunosuppressed after the hundredth failed “HIV” vaccine trial, microbicide trial, breast feeding dissuasion trial, toxic drug trial, etc., and allow other scientists with different hypotheses (let us call them **The Fighters of “HIV/AIDS” and the “HIV/AIDS=death” paradigm**) to explore other

hypotheses, such as the biological basis of lymph node fibrosis, and provide funds for hypotheses that do explain the pathogenesis and rational reversal of severe immune suppressive illnesses? When will the Promoters of AIDS explain why a positive “HIV” test result is something to fear, since it is the same molecular signature that should be exhibited by “HIV-positive” individuals that they have been trying to evoke in their 63 failed “HIV” vaccine trials? A related question may be to ask, if “HIV” vaccines ever do evoke a consistent positive immune response (antibodies as evidence of immunization), then will millions upon millions of “HIV-immunized” people need to carry around letters to announce the fact that they have been vaccinated against “HIV” to avoid criminalization, so that their sexual partners, insurance agencies, and employers (and public health agencies, and such groups as ACIP advisory panels staffed with pharmaceutically-backed decision makers who are **The Promoters** of AIDS), won’t think an “HIV-positive” test result is due to acquiring “HIV” from a source other than a successful vaccination, and so their insurance policies, relationships, or jobs, happiness, and futures won’t be denied to them as they are currently because of the often deadly stigma of testing “HIV-positive?”

The relationship or non-relationship between “HIV” and “AIDS” has been questioned and in many cases rejected by highly praised individuals and leaders in science and other endeavors. Some of these include Nobelist and PCR test-kit inventor Kary Mullis (PCR is used to amplify “HIV’s” unique genome sequence despite Mullis’s warnings that his invention cannot be used to do quantify viral load), DNA foot-print inventor and Nobelist, Walter Gilbert (who said that *“the views of people like Duesberg are incredibly important”*), Alfred Hassig, former head of the European blood banks (who said that AIDS is caused by excessive oxidation and that the death sentence accompanying an AIDS diagnosis should be abolished because doctors aren’t prophets and that AZT causes AIDS), and Dr. Heinz Ludwig Sanger, Emeritus Professor of Molecular Biology and Virology, and former director of the Department of Viroid Research, Max-Planck-Institutes for Biochemistry, Munchen, and recipient of the Robert Koch Award in 1978 (who said that “HIV” hasn’t been isolated), as well as many others who have asked a question about the relationship between the molecular signature of “HIV” and “AIDS.” For instance, in a letter to *Suddeutsche Zeitung* (Oct. 2000), Dr. Sanger wrote:

During the past 20 years HIV-AIDS research has shown to a line of critical scientists again and again that the existence of HIV has not been proven without doubt, and that both from a aetiological (causal), and an epidemiological view, it can not be responsible for the immunodeficiency AIDS. In view of the general accepted HIV/AIDS hypothesis this appeared to me so unbelievable that I decided to investigate it myself. After three years of intensive and, above all, critical studies of the relevant original literature, as an experienced virologist and molecular biologist I came to the following surprising conclusion: Up to today there is actually no single scientifically really convincing evidence for the existence of HIV. Not even once such a retrovirus has been isolated and purified by the methods of classical virology.

Apparently as of June 2009, **“it”** still hasn’t been “isolated,” and as suggested in *AIDS Clinical Care*, “viral load” [the detection of retroviruses and HERV8] and T-cell depletion aren’t even as indicative of AIDS-morbidity as is fibrosis of the lymph nodes.

In this respect, for many of us who have followed every development in the AIDS debacle since it officially began here in the US in 1984, Wainberg’s statement about incarcerating Peter Duesberg, myself, or others who ask questions regarding the biology of AIDS, only serves as yet another pathetic example of the Public Health Service and media-censored McCarthyism regarding “HIV” and “AIDS.”

If women can’t pass the virus to their offspring through breast milk, even in populations that are supposed to have high rates of “HIV,” and have a much higher death rate of their infants if they don’t breast feed, then how could it be even considered a possibility that vaccine makers could inject some component(s) of “HIV” into a human and induce protection from immune suppression, or, in the case of the failed Merck STEP trial just announced, evoke “HIV’s” molecular signature in any significant number of vaccine recipients? In one arm of the recent Merck trial, for instance, it was reported that among 778 male volunteers, only 21 of those receiving the vaccine exhibited “HIV’s” signature compared with 9 in the placebo group. Most or all of the vaccinated should have at

least shown seroconversion if “HIV’s” components had been isolated and are immunogenic in human beings.

It is only appropriate in *THIS BRIEF HISTORY OF AIDS*, that because this history essentially has been an elaboration and crude burlesque pageant of a single hypothesis, The “HIV=AIDS=death” hypothesis fashioned and religiously monitored and protected by **The Promoters of AIDS**, that it should first end with a quote from Dr. Gallo and Dr. Montagnier: *Many lessons can be drawn from this early intense period, and most suggest that science requires greater modesty [2]*.

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