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## The Mainstream Again Admits – HIV Does Not Exist as a Unique Particle

🕒 November 26, 2011    📁 Human Endogenous Retrovirus - HERV, Methylation, The Tests    👤

webmaster

by *Liam Scheff*

Dear voyagers of dark and dusky paths; of shattered highways, of broken dreams. Willkommen!

How good it is to see you in the old haunt, reading the medical journals, looking for clues this Century's greatest lie (well, since 2001 we're competing with some whoppers...but).

Have a stroll through this mainstream review of all things retroviral nonsense. This paper provides a basis of understanding the AIDS mess deeply from their point of view (the point of view of bad science in many labs reaching consensus agreement).

So, please read along. Take your time. Take some dramamine, if needed, or ginger tea. Many of the leaps of logic will induce vertigo...

<http://www.retrovirology.com/content/6/1/40> [cached]

### Understanding AIDS theory goes like this:

**A.** It is a sexually transmitted particle – Yes or No.

**B.** It or They Kill T-Cells – Yes or No.

**C.** It is a Distinct particle with a distinct shape, size, (morphology) and physical characteristic – Yes or No.

(Why? because that's (supposedly) how infectious particles in the body are supposed to work – like jigsaw puzzle pieces. The need a particular size, and physical features to do their jobs).

For "A", see Padian, see this: <http://reducetheburden.org/?p=206>

175 couples. Doing it. In, out, up, down, front, back, six years of study time (plus all that came before): Zero 'conversions.' No negs became pos. Why? Because they weren't shooting drugs – meaning, they didn't raise their antibody count to a rancorous level, so as to tick off the touchy non-specific antibody tests.

For B?

Easy. See the mainstream’s defense of how they can’t figure out if/how anything is even remotely affecting T-Cells. Read “[The Happy Exosome](#).”

From the inception of the paradigm in 1984, to the present, the answer is the same: “We don’t know, but keep sending money”:

- “We are **still very confused** about the mechanisms that lead to CD4 T-cell depletion, but at least now we are confused at a higher level of understanding.” — *Dr. Paul Johnson, Harvard Medical School (Balter 1997)*
- “We still **do not know how**, in vivo, the virus destroys CD4+ T cells.... Several hypotheses have been proposed to explain the loss of CD4+ T cells, some of which **seem to be diametrically opposed**.” — *Joseph McCune, immunologist (McCune 2001)*
- “Despite considerable advances in HIV science in the past 20 years, the reason why HIV-1 infection is pathogenic is still debated... There is **a general misconception** that more is known about HIV-1 than about any other virus and that all of the important issues regarding HIV-1 biology and pathogenesis have been resolved. On the contrary, what we know represents only a **thin veneer on the surface** of what needs to be known.” — *Mario Stevenson, virologist (Nature Medicine 2003)*
- “Twenty-five years into the HIV epidemic, a complete understanding of what drives the decay of CD4 cells – the essential event of HIV disease – **is still lacking**. ... The puzzle of HIV pathogenesis keeps getting more pieces added to it.” — W. Keith Henry, Pablo Tebas, and H. Clifford Lane (*Henry 2006*)

So, it’s still a “puzzle” to the mainstream. Do T-Cells die when in the presence of “HIV” DNA? (Which is always different!) No. Or, “*We’re confused at a much higher level of understanding*” is the official answer.

For question C? That’s what this is about...See below. See [this](#) – with pictures even. No distinct shape or size.

**1.** There is no single thing called “HIV” (“it” is never the same, because “it” is a “they” – and they knew it from the start) – The crap we’re finding is “Extremely Variable.”

**“Every ‘isolated’ strain was different from the other also when obtained from the same individual but at different times.”**

Is that a particle? No, it’s a fishing expedition with genetic re-assembling (later PCR), and culturing techniques. They call different things by one name. Have a glance:

*“The ground for the feud was the following.  
Montagnier sent his first isolate LAV/BRU to Gallo in  
July of 1983. In May of 1984 Gallo’s coworker*

*Sarngadharan brings one of Gallo's five HIV strains (HTLV-IIIB) that grew well in a continuous cell line to Montagniers laboratory in Paris. In July of 1984 Montagnier sends Gallo a second sample of LAVBRU since Gallo had complained that the first didn't grow well at NIH. Gallo then found and reported[33] that **HIV was extremely variable; every isolated strain was different from the other also when obtained from the same individual but at different times.*** “ — Genomic diversity of the acquired immune deficiency syndrome virus HTLV-III: different viruses exhibit greatest divergence in their envelope genes. Proc Natl Acad Sci U S A. 1985 Jul;82(14):4813-7.

Right. Get it? Nothing is ever the same? Even when it's the same thing?

*“Converging lines of research have linked human T-cell lymphotropic virus type III (HTLV-III) to the pathogenesis of the acquired immune deficiency syndrome. A characteristic feature of this virus is its genomic heterogeneity, which occurs to varying degrees in different viral isolates.” – Hahn BH, Gonda MA, Shaw GM, Popovic M, Hoxie JA, Gallo RC, Wong-Staal F.*

Right! It's always different! (Wrong. It's not the same bloody thing, you bleeping morons). What they're doing is fishing out different bits of genetic stuff from the 'redundant' genome – they used to call it 'junk DNA,' now it's 'important epi-genetic DNA,' now it's “exosomal DNA.”

Here's how that works: <http://reducetheburden.org/?p=2714>

## 2. Highly Variable – Because “HIV” is not a Single Entity. “It” is a “They,” and “They” are HERVS or Now, “Exosomes”:

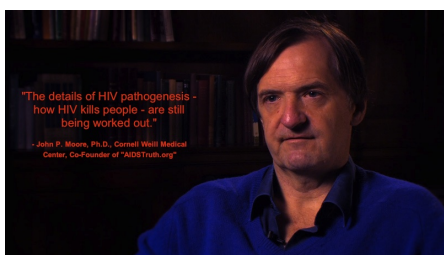
### *H-I-V is really H-E-R-V*

*AIDS researchers have been forced to admit **time and again** that their “HIV” is morphologically identical to “HIV-like particles” they find in “HIV negative persons.” This is true even though their “HIV” has the bad habit of having no standard size or physical quality – it can be too small or too large, and still be “HIV” to determined true-believing AIDS researchers.*

*This is why AIDS patients can “suppress” or stop the production of “HIV” **by taking Selenium** and other pro-methylating micronutrients. Why? Because returning cells to healthy levels of methyl production is good for bringing order back to loose and disordered DNA. Methylation stops or slows the production of these transposable elements. These are mistakenly thought of as “viruses,” but “HIV” is “LAV,” which is and always was human endogenous retroviral expression in stressed and damaged cells.*

*And this is at least part of the reason why HIV tests are so lousy.\* Humans and animals produce HERVs*

*under stress and illness, and so “HIV tests” are really “HERV tests,” and react with proteins produced by people who are suffering from almost any illness, drug abuse, vaccination, or, of course, pregnancy – because HERVs are expressed like wildfire in the placenta. \*(The other reason is because Gallo’s HIV test slurry came from so many different people, mixed with so many different chemical and biological elements, it’s really impossible to know what they’re testing for).*



*Finally, this is why “HIV” (LAV or HTLV) doesn’t kill T-Cells. This has been the central claim of the AIDS paradigm – but it was proven false from the start. Robert Gallo invented the idea of slow T-Cell depletion by a scavenging, ravaging retrovirus in order to package his product with enough fear and anxiety, covered by pseudo-scientific technobabble, so pure belief would make it stick. Gallo sold his mixed cells containing HERVs (or HTLV-III, which was both as functionless and fraudulent as his other “human lymphotropic viruses”) to Abbott Labs. But he sold them in... ready? T-Cells. His “HIV” Grows in T-Cells. It’s called*

*an ‘eternal line’ of production, in T-Cell leukemia. It never dies. Because HERVs don’t kill T-Cells.*

*Do you know what does cause T-Cells to suffer? De-methylation of DNA **by exposure to pharmaceuticals and toxins.***

*<http://liamscheff.com/2010/05/an-onoff-switch-for-retroviruses-can-it-be-that-simple/>*

### **3. What Kind of ‘Virus’ is ‘HIV’? It’s as many as they need it to be. A type-C a type-D, etc.**

It’s supposed to be a “C.” Or. Well. Here, in Jay Levy’s lab, it’s a “D”:

*Isolation of lymphocytopathic retroviruses from San Francisco patients with AIDS.*

*Levy JA, Hoffman AD, Kramer SM, Landis JA, Shimabukuro JM, Oshiro LS.*

*Abstract*

**Infectious retroviruses have been detected in 22 of 45** randomly selected patients with acquired immune deficiency syndrome (AIDS) and in other individuals from San Francisco. The AIDS-associated retroviruses (ARV) studied in detail had a **type D morphology**, Mg<sup>2+</sup>-dependent reverse transcriptase, and cytopathic effects on lymphocytes. The viruses can be propagated in an established adult human T cell line, **HUT-78**. They cross-react with antiserum to the lymphadenopathy-associated retrovirus isolated from AIDS patients in France. Antibodies to ARV were found in all 86 AIDS patients and in a high percentage of 88 other homosexual men in San Francisco. This observation indicates the widespread presence of these lymphocytopathic retroviruses and their close association with AIDS.

But for everybody else, it was a “B.” No, a “C.” No. Well... nobody really knew. Or cared.

If they could outsmart themselves, they could certainly outsmart the public.

#### **Let’s Look at the Numbers:**

From their mouths to your ears: 22 out of 45 ‘AIDS’ patients have ‘infectious retroviruses.’ Pardon? Where’s the 100 percent correlation for infection? Answer – they don’t care. It’s never to be found.

Where do the proteins come from? They were and are Propagated (grown, made) in an adult HUMAN T-CELL LINE.

What is “HIV” supposed to do?

### **Kill T-Cells.**

Where does the mainstream GROW ‘immortal’ lines of ‘HIV?’

### **In T-Cells.**

*Can we all go home now, and get on with our humping?*

Back to their numbers...

Antibodies to this ‘specific, never-the-same’ retroid were found in ... 86 AIDS patients and in a high percentage of 88 other homosexual men.

Good news. I guess you can find it anywhere...

### **4. Don’t Worry About “HIV” Bothering Anyone...It’s “Fragile”**

**“HIV is an enveloped virus and hence fragile. Most certainly they had lost the virus envelope in their purification of the virus.”**

You can find the Perth researchers citing AIDS theory originator, Robert Gallo, saying that MOST lose their envelopes AFTER or DURING budding. So it’s all a very, very fragile soup of non-uniform, never-the-same crap. Poor dears! I bet they cried during “Girl, Interrupted.” (I surely did).

*“In the same issue of Science where Montagnier and his colleagues published their study Gallo pointed out that “the viral envelope which is required for infectivity is very fragile, it tends to come off when the virus buds from infected cells, thus rendering the particles incapable of infecting new cells”. Because of this Gallo claimed that “cell-to-cell contact may be required for retroviral infection”. — Marx JL. Human T-cell virus linked to AIDS.” Science 1983;220:806-809.*

<http://www.theperthgroup.com/CONTINUUM/epeondjamel.html>

**But, what's this? A Cure Already! Way back in the Eighties!**

Gallo also said, years ago (and this was new to me) that AIDS was curable with 'chemokines':

*“Gallo brings more than his reputation. He already has several promising projects on the fast track. Last fall, he identified what he called **chemokines** – naturally occurring molecules that suppress HIV in vitro. These could prove a powerful treatment for AIDS. He’s following up on the vaccine research of Jonas Salk and Daniel Zagury, and trying to develop a “vector vaccine,” one that uses the smallpox virus to deliver particles that might trigger an immune response against HIV. He’s developing a treatment for Kaposi’s sarcoma, the deadly skin cancer seen in many AIDS patients.*

*Any of these paths could lead to a blockbuster product. “AIDS will soon drive the whole biotechnology field,” Gallo predicts. “It will be worth ten times ten our efforts here.”*

<http://www.virusmyth.com/aids/hiv/vcgallo.htm> |

<http://en.wikipedia.org/wiki/Chemokine>

Good news, because the redoubtable wikipodium says that they are ‘found in all vertabrates,’ so I guess the spinal column has cured AIDS.

### **5. HIV Proteins are not HIV Proteins, Are Only Sometimes or Later, or Perhaps Another Time Important (or not), Depending...**

In HIV-ology, proteins with numbers (daltons – microscopic weight) are very important. The good news is you can find these “HIV specific proteins” in everybody. In pregnant women, in their children, and certainly in sick people, arthritic, alcoholic, whatever, poor, starving, etc. They occur everywhere, in animals too.

*“The French group **did not detect gp41 in their immune precipitation studies using purified LAV. Their inability to detect this protein in their ELISA or immune precipitation experiments is probably the main reason that their positive scores with AIDS and pre-AIDS sera were so low.**”*

**Hey, isn’t ‘gp41’ the capo da tutti** of all HIV proteins? But, well. Bah. Besides **p24, which shows up everywhere**. So. You know. Screw it, sure it’s important. But, you know, we don’t want to be anal-retentive!

Nope, You don’t have to find any “HIV” proteins in “HIV.” Monty found a p25, not a p24 (but I’m sure that was just an accounting error); and he didn’t find any very important p41...

And what’s this: “Scores were so low?” This means, yes, the tests SUCK.

### **6. HIV Occurs in SOME AIDS Patients...**

Or, really “reverse transcriptase” occurs in some, or alot of AIDS, and also non-AIDS patients. Because when they say, “HIV,” they mean, *“We found this enzyme, and we’ll say it’s a virus. We know it’s not, but you’re never going to figure that out.”*

Reverse Transcriptase is an enzyme process, (many enzymes) which ‘copy’ material from RNA to DNA.

They used to think this “backward copying” was a big deal, because it contradicted DNA-wonderbrats Watson and Crick, (who were wrong about almost anything, anyway, except they figured out where to put the phosphates – on the inside. Linus Pauling put them on the outside, so he didn’t discover DNA, and they ‘did.’)

The mainstream used to get so excited about RT (reverse transcriptase), that they liked to imagine that it only occurred in Tumor Viruses (which no longer are said to really exist as such, so they were relabeled retroviruses, which are now being rechristened, ‘exosomes.’)

But the HIV quacks spend their time and your dollars looking for RT.

That is, You don't have to find anything anywhere consistently, as long as the NIH is paying your tab (and you're spending their (I mean, the taxpayer's) dough). How many AIDS patients 'have HTLV/LAV/LAI/HUT9' etc??

So here you can find RT in....

The 48 HTLV-III isolates [Yes, they don't really mean "isolate," they mean reverse transcriptase] were obtained from –

- 18 of 21 tested patients with unexplained lymphadenopathy and leukopenia, with an inverted T4/T8 lymphocyte ratio (designated pre-AIDS), [No, it was probably not 'unexplained,' it's just not politically correct to talk about poppers]
- 3 of 4 clinically normal mothers of juvenile AIDS patients,
- 3 of 8 juvenile AIDS patients,
- 13 of 43 adult AIDS patients with Kaposi sarcoma,
- 10 of 21 adults AIDS patients with opportunistic infections, and
- 1 of 22 clinically normal homosexual donors.

**10 out of 21. 13 out of 43.** With their version of "HIV" (reverse transcriptase). And they sell THIS to the public? What a sick bunch of ... right. Research dollars are needed to help these poor lab jockeys along.

So, here they're working on improving HIV tests, because they come up pos for everyone who's sick in any way, and they want the protein reactions to focus on the drug addicts and gay men:

In a second accompanying paper...The number of sera that gave positive scores in the ELISA were:

- **43 of 49 (88%)** of patients with AIDS (two of whom had developed AIDS after blood transfusion)
- 11 of 14 patients with pre-AIDS, ["I am your doctor. I regret to inform you that you have....Pre-AIDS... You'll have to wear a condom on your face, because HTLV-iii is very fragile, and you don't want to break it, do you?"]
- 3 of 5 intravenous drug users (of which one positive ways also homosexual),
- **6 of 17** homosexual men.
- Out of 186 controls only one scored positive in the ELISA (1 of the 164 normal subjects). ["Normal subjects," ie, not drug addicts. Here they're getting better at gearing the tests to people with drug and other-related antibody production]

Pretty good, huh? They're getting the tests to work a liiiitttle better on sick people. 88% of their AIDS patients now click the tests. Impressive. You've just identified that 88% of the people are sick. And also everybody else.

Now, here are retro-antibodies showing up **in all immune illness:**

- "The controls also included 3 patients with hepatitis B virus infection, 1 with rheumatoid arthritis, 6 with systemic lupus erythematosus, 4 with acute mononucleosis, and 8 patients with lymphatic leukemias. Of the latter some were positive for HTLV-I."

Wow! More people are 'positive' for more fake retroviruses, based on a finding of non-specific antibodies and reverse transcriptase...

How's this for an hypothesis:

**Sick people produce more retroviral proteins and reverse transcriptase than very healthy people. As do pregnant women, drug users (file under 'sick,'), people starving to death and riddled with parasites (see 'sick people'), and, well. You get the gist.**

#### **7. HIV Proteins Can Be Added to the Consensus at Any Time, if Someone Important Says So.**

The mainstream worked in waves, with different cell cultures, different cancer T-Cells, different labs. They all got different results.

They welded them together to create a consensus idea of "HIV".

*"None of these 22 control patients scored positive in the ELISA or Western blot. Of note, in Western blot the antigen most prominently and commonly detected among all of the sera from AIDS patients had a molecular weight of 41,000 (now designated gp41).*

*It was presumed that this is a virus envelope protein (which later turned out to be correct). Others, including myself, have later confirmed that gp41 is extremely reactive in ELISA of sera from HIV infected individuals. In fact we have found that an ELISA having as only antigen a peptide with the amino acids GKLICT, representing an epitope of gp41, reacts positively with the majority of sera from HIV infected individuals."*

It was presumed! And so it was. And Don Francis, and Jay Levy, and all the other members of the goon squad made some dollars too, by adding more crap proteins to the mix, that didn't show up elsewhere. How great for all of them, to find different crap proteins in different people that they all

added to the consensus agreement bitch's brew; that was then sold to Abbott labs, to make fake HIV tests.

*Let's have a holiday in their honor.*

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And we're back where we started. p41 is important, except when it isn't. Like in the premiere Nobel-prize winning papers on "LAV" (I mean...HT...L.....whatever, you get the point....

And the proteins get added after the fact, and there's no there there.

Anyway, read the paper. It's illuminating, especially in the deep criticisms of Montagnier, the chronic ad hoc additions; the papier mâché nature of the whole thing... built from failure, from nothing, with each group finding nothing like the previous, and improvising a theory out of it – "HIV."

Really, improvising:

"We found a 'new protein' to your thing; sure you never found it, but that's science – it's a mystery!! Let's add it into the consensus model!"

**And extra credit to anyone who wants to figure this one out:**

*"It is noteworthy that B.R.U.'s serum reacted with 90–100% of the co-cultured cells from B.R.U and the healthy donor since we know that only the CD4 positive cells should be infected. The B.R.U.'s serum also reacted with 90–100% of the HTLV-I producing cells! If this were to be due to a possible double infection with HIV and HTLV-I again only CD4 positive cells should be positive. More likely something unrelated to either HIV or HTLV-I was detected by the B.R.U. serum, **in my opinion most probably mycoplasma, a common contaminant in cell culture.**"*

How cross-contaminated can something be and still be "pure?" And yet it's still proof..of.....???

*“The 0.5 to 2% positive infected **umbilical cord lymphocytes** may indicate retrovirus-infected cells. However, the lack of reactivity with the p19 and p24 sera with these cells is not a proof that the B.R.U. virus was not HTLV-I. The few percentages of possibly positive cells could simply have been missed with the specific antibodies but detected with the patient’s sera containing antibodies to all viral proteins. The paper does not present any photos of the fluorescent cells.”*

They grow their proteins in contaminated cultures – umbilical cells, cancer T-Cells. Which contain... what...?

Anybody? Anybody...

Reverse transcriptase; retroviruses. All the stuff they’re looking for. They’re always going to find what they’re looking for – which is cellular detritus.

Happy hunting. Interesting paper, most definitely worth reading, especially for the ‘fragile,’ ‘never the same,’ and the list of percentages...

### *3 thoughts on “The Mainstream Again Admits – HIV Does Not Exist as a Unique Particle”*

★ webmaster November 26, 2011 at 10:18 pm

And here is the paper, also linked in the body of the article:

A historical reflection on the discovery of human retroviruses

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#### Abstract

The discovery of HIV-1 as the cause of AIDS was one of the major scientific achievements during the last century. Here the events leading to this discovery are reviewed with particular attention to priority and actual contributions by those involved. Since I would argue that discovering HIV was dependent on the previous discovery of the first human retrovirus HTLV-I, the history of this discovery is also re-examined. The first human retroviruses (HTLV-I) was first reported by Robert C. Gallo and coworkers in 1980 and reconfirmed by Yorio Hinuma and coworkers in 1981. These discoveries were in turn dependent on the previous discovery by Gallo and coworkers in 1976 of interleukin 2 or T-cell growth factor as it was called then. HTLV-II was described by Gallo's group in 1982. A human retrovirus distinct from HTLV-I and HTLV-II in that it was shown to have the morphology of a lentivirus was in my mind described for the first time by Luc Montagnier in an oral presentation at Cold Spring Harbor in September of 1983. This virus was isolated from a patient with lymphadenopathy using the protocol previously described for HTLV by Gallo. The first peer reviewed paper by Montagnier's group of such a retrovirus, isolated from two siblings of whom one with AIDS, appeared in Lancet in April of 1984. However, the proof that a new human retrovirus (HIV-1) was the cause of AIDS was first established in four publications by Gallo's group in the May 4th issue of Science in 1984.

#### Background

Unfortunately the omission of the American scientist Robert C. Gallo from the 2008 Nobel Prize in Medicine or Physiology for the discovery of HIV by many has been viewed as a final scientific verdict handed down by the Nobel committee of the Karolinska Institutet on an old controversy between the Institute Pasteur and NIH and that previous settlements were for political reasons only. Also, the decision to omit Gallo has resulted in the resurrection of false allegations in the media that Gallo and coworkers at NIH had rediscovered or even stolen the French HIV isolate previously sent to them from

the Pasteur Institute. Thus, it could be interpreted as if the Nobel committee finally had put right an unjust settlement previously obtained between the French and American scientific groups. There is no doubt or controversy about the fact that the French group was first to isolate this new virus. This is what the Nobel committee chose to award.

Two years ago I had the privilege to painstakingly and thoroughly go through all the literature related to the discovery of HIV. Since the motivation for the Prize by the Nobel Committee is very limited and the fact that the Committee members cannot comment on how they came to their decision, I think it is important that the medical community gets the correct historical facts about this important discovery. Therefore, I have written this article. I would say that what I present below is a fair and accurate account on the events and work that led to the discovery of a new virus as the cause of AIDS. Regarding whom should get the credit for the discovery of HIV, this review should enable the reader to come to his or her own conclusion. Mine, however, is different from that of those of my fellow faculty members that presently make up the Nobel Committee for the Nobel Prize in Physiology or Medicine. I will here show that by going through the literature it is evident that Gallo's group was not only first to show that HIV is the cause of AIDS but that the French group had not been able to discover this new virus without the active assistance of, as well as, previous work by Gallo. It will also be evident that Gallo and his associates had no reason to "steal" any French isolate. Last year this journal published another account of the 2008 Nobel Prize [1].

Paving the way for the discovery of HIV

Isolation of a virus means infection, propagation and (usually cell free) transmission of an infectious agent in cultured cells. New viruses, for which there are no susceptible cells in culture, have lately also been detected solely by molecular techniques, e.g. hepatitis C virus by using a random-primed complementary DNA library from an infected patient (Michael Houghton) and subtypes of human papilloma viruses by using hybridization under low stringency and subsequent DNA cloning (Harald zur Hausen).

The difficulty in isolating a new virus is choosing the right cell culture and detection systems and to obtain specimens containing the virus. With a susceptible cell culture system and a detection system available, isolation of a new virus is not only possible but also rather straightforward. In the case of HIV, before the successful isolation of the first human retrovirus (human T-cell leukemia virus, now human T-cell lymphotropic virus type I; HTLV-I) by Robert C. Gallo [2], neither was at hand.

After the discovery of reverse transcriptase from animal oncogenic RNA viruses (then called oncornaviruses and now called retroviruses) a large number of publications on putative discoveries of retrovirus detections in human malignancies appeared in the early 1970-ties. However, they were all either owing to contaminations in the laboratories with animal retroviruses or the mitochondrial DNA-polymerase  $\gamma$ , the latter when the reports were based on reverse transcriptase activity alone. DNA-polymerase  $\beta$  is a normal cellular DNA polymerase which uses RNA as a primer but not as a template. Therefore, like reverse transcriptase, the activity of DNA-polymerase  $\beta$  was sensitive to a ribonuclease treatment [3-5]. This cellular enzyme was not known at the time. In 1972 Gallo's group [3] reported that stimulated normal human lymphocytes contained a ribonuclease sensitive DNA polymerase distinct from viral RNA-directed DNA polymerase, an enzyme that Gallo's group characterized further in a number of publications. The enzyme prefers  $Mn^{2+}$ . Unlike DNA polymerases  $\alpha$  and  $\beta$ , the preferred primer-template for DNA-polymerase  $\gamma$  is  $(dT)_{15} \cdot (A)_n$  over  $(dT)_{15} \cdot (dA)_n$ ! This third cellular DNA polymerase was independently from Gallo discovered by Art Weissbach and they later named it DNA-polymerase  $\gamma$  [6].

From the numerous and erroneous reports on retroviruses in various human cancers, the notion of human cancer viruses became in ill repute and rather than talking of “human tumor viruses” people in science talked of “human rumor viruses”. In fact, as narrated by Gallo in one of his reviews[7], when Gallo first submitted their report on HTLV-I to Journal of Virology it was rejected right away by the editor Robert Wagner “insisting that they should cease, and not continue to perpetuate the controversy, strongly implying that we all know human retroviruses do not exist”.

In his quest to find a human retrovirus in lymphoma/leukemia Gallo developed sensitive and generalized techniques for the detection of reverse transcriptase to discriminate it from cellular DNA polymerases [8,9].

To isolate T cell lymphotropic viruses one needs to be able to culture T lymphocytes. Working with conditioned medium to grow lymphocytes, Gallo together with two of his post doctoral fellows Doris Morgan, Frank Ruscetti discovered T cell growth factor (TCGF) later named interleukin 2 (IL-2). Hence, the first report of IL-2/TCGF was by Robert Gallo was published in 1976 [10]. The first paper by Kendal A. Smith on IL-2/TCGF did not appear before 1978[11].

I sincerely doubt that anyone would have been looking for a retrovirus as the etiological agent for AIDS had HTLV-I not previously been isolated. I will therefore shortly recapitulate the history of the discovery of this virus.

The discovery of the first human retrovirus

Reverse transcriptase activity was detected by Gallo’s group in a T-cell line established (using IL-2) from a patient diagnosed originally with mycosis fungoides in 1979. To show that this was indeed a new human retrovirus Gallo and coworkers set out to show that the same virus could be isolated from primary tissue samples of the same patient by culturing primary T-cells with IL-2; demonstrate that the virus was novel, i.e., not any of the known animal retroviruses; show it could infect human T cells in vitro; demonstrate specific antibodies to the virus in the serum of the patient; demonstrate that proviral DNA could be found integrated in the DNA of the cells from which the virus was isolated; and provide evidence that this was not a one-time affair by showing serological evidence of specific antibodies not only in the patient but in some others as well.

Most or all these results were obtained by the time Gallo submitted the first paper to PNAS allowing it to quickly be followed with several other reports[12]. The paper on the first isolation of HTLV-I [2] was submitted (communicated) for publication in PNAS on August 4th 1980 and appeared in the December issue of the same year. The second paper from Gallo and his group (especially Bernard J. Poiesz, another post doctoral fellow) on the isolation of HTLV-I now from fresh cultured cells from a patient with Sezary T-cell leukemia was submitted to Nature on May 1st 1981 and appeared in the November 19th issue of that journal[13]. In the February 19th 1982 issue of Science (submitted October 6th 1981) Gallo’s group [14] reported that five of six tested ATL patients in Japan had antibodies to HTLV-I (only HTLV at the time).

On the 26th of June 1981 (six months after the Poiesz et al. paper from Gallo’s group was published) Hinuma et al. submitted (communicated) a paper to PNAS showing antibodies against an antigen in a T-cell line, MT-1 from a patient with adult T-cell leukemia (ATL), in all 44 patients with ATL examined and in 32 of 40 patients with malignant T-cell lymphomas using indirect immunofluorescence[15]. The antibodies were also detected in 26% of the healthy adults examined from ATL-endemic areas but in only a few of those examined from ATL-non-endemic areas. Extra-cellular type C virus particles were detected in pelleted cells of the MT-1 T-cell line. Hinuma called this virus adult T-cell leukemia virus (ATLV). Characterization of the virus as a retrovirus was published in the March issue of PNAS,

submitted (communicated) November 23rd 1981. In this paper[16] also proviral DNA was detected in fresh peripheral lymphocytes from all of five patients with ATL but not in those from healthy adults. This paper was submitted more than a month later than the Gallo paper showing antibodies to HTLV-I in Japanese ATL patients.

On July 13th of 1981 Miyoshi et al. (Hinuma last author) submitted a paper to Nature (published December 24th 1981) on the transmission of virus from MT-1 cells (female) to cord blood cells of a male infant transforming (immortalizing) the latter cells[17].

In the November 4th 1982 issue of Nature Gallo's group together with the Japanese colleagues Nakao, Miyoshi, Minowada, Yoshida and Ito reported that HTLV-I and ATL was one and the same virus[18] and decided to call both viruses HTLV-I.

As is evident from the above Gallo was truly the first to isolate the first known human retrovirus and to report it. In 1982, Gallo and co-workers reported the discovery of the second human retrovirus, HTLV-II, in a patient with hairy cell leukemia. However, no malignancy or other disease has yet been clearly linked to the infection of this virus.

The isolation of what is now called HIV-1 (will also be referred to as HIV, LAV, IDAV-1, IDAV-2, LAV-1, HTLV-III and ARV) and the demonstration of this virus as the cause of AIDS

In May of 1983 Françoise Barré-Sinoussi et al. published a paper in Science [19] describing the isolation of a putative new human retrovirus from the lymph gland of a patient suffering from persistent generalized lymphadenopathy, which is regarded as a precursor condition of AIDS. They called this new virus LAV (later LAVBRU) for lymphadenopathy virus and BRU from the first three letters of the patient's last name. Since this has been viewed as a seminal paper for the discovery and characterization of HIV, I will here describe this paper in detail.

Cells from a lymph node of patient B.R.U. was cultured under the conditions described by Gallo[2,13], i.e. culture medium with T-cell growth factor (TCGF or IL-2), and were stimulated with phytohemagglutinin (PHA). They also added antiserum to human  $\gamma$ -interferon to neutralize possible endogenous interferon. (The latter is not necessary, and is not used by others.) After three days, the culture was continued in the same medium without PHA. After 15 days in culture reverse transcriptase (RT) activity was detected in the culture supernatant, using the protocol by Gallo[2,13]. Importantly, the ionic conditions were the same as for isolating HTLV-I previously described by Gallo (1 and 2; in contrast to other animal retroviruses HTLV-I has a  $Mg^{2+}$ -dependent and not  $Mn^{2+}$ -dependent reverse transcriptase). Virus production continued for 15 days and decreased thereafter, in parallel with the decline of lymphocyte proliferation. A standard and routine procedure in clinical virology when trying to isolate a virus is to passage the cells to fresh ones, usually when the original cells start to die, and particularly if they do not yet show any signs of being infected. Hence, to show virus transmission, cells from patient B.R.U. after three days in culture were also co-cultured with lymphocytes from a healthy donor of the Blood Transfusion Center at the Pasteur Institute. Also with these co-cultures, RT could be detected after 15 days of culture (not before) and amounts of RT remained stable for 15 to 20 days. Transmission of cell-free supernatants from the original culture of B.R.U. cells was successfully obtained using 3-day-old cultures of T lymphocytes from two umbilical cords. There is no mentioning of cytopathic effects in any of the cultures or that fresh T lymphocytes from healthy donors were added to make the virus isolation possible or to save the virus isolate except for the virus transmission experiment described above.

The virus isolate had a density of 1.16 (same as HTLV-I) in a sucrose gradient. Electron micrographs of the virus from the umbilical cord lymphocytes were reported to be of typical C-type virus, i.e. with a

spherical core (same as HTLV-I). Of note, HIV is a lenti retrovirus having a conical or cylindrical core structure and does not have type C virus morphology.

Two experiments were performed to distinguish the new isolate from HTLV-I. The first was by immunofluorescence using serum from the patient as well as a goat anti HTLV-I p24 (capsid protein) and mouse monoclonal anti HTLV-I p19 (matrix protein). The two latter anti-sera, as well as two HTLV-I producing cell lines were from Robert C. Gallo as acknowledged by the French group in the paper. The sera were tested against two different cultures of normal blood lymphocytes, against the two lines of HTLV-I producing cells, and against virus producing cells from the co-culture of T lymphocytes of patient B.R.U. and the healthy donor and against infected cord blood lymphocytes. In addition cells from a lymph node from a person (patient 2) who presented with multiple adenopathies and who had been in close contact with an AIDS case was also tested. No RT activity was detected in the latter patient's cultured lymphocytes. The anti-HTLV-I sera from Gallo (anti p19 and p24) reacted with the HTLV-I producing cell lines only. Serum from patient B.R.U. reacted with 90–100% of the HTLV-I producing cell lines and with 90–100% of the co-cultured cells from B.R.U and the healthy donor, as well as, the cells from patient 2. The B.R.U. serum reacted with only 0.5 to 2% of the infected umbilical cord lymphocytes. It is noteworthy that B.R.U.'s serum reacted with 90–100% of the co-cultured cells from B.R.U and the healthy donor since we know that only the CD4 positive cells should be infected. The B.R.U.'s serum also reacted with 90–100% of the HTLV-I producing cells! If this were to be due to a possible double infection with HIV and HTLV-I again only CD4 positive cells should be positive. More likely something unrelated to either HIV or HTLV-I was detected by the B.R.U. serum, in my opinion most probably mycoplasma, a common contaminant in cell culture. The 0.5 to 2% positive infected umbilical cord lymphocytes may indicate retrovirus-infected cells. However, the lack of reactivity with the p19 and p24 sera with these cells is not a proof that the B.R.U. virus was not HTLV-I. The few percentages of possibly positive cells could simply have been missed with the specific antibodies but detected with the patient's sera containing antibodies to all viral proteins. The paper does not present any photos of the fluorescent cells.

The other experiment performed to distinguish the new virus from HTLV-I was immunoprecipitation of lysates of infected cord lymphocytes, as well as, virus released from the infected cells with the same sera used for immunofluorescence and in addition serum from patient 2. Serum from B.R.U. and patient 2 (whose lymphocytes were RT negative) precipitated a protein of the apparent size of 25,000 from extracts of the infected cord lymphocytes and from the supernatants of these cells. Serum from a healthy donor did not precipitate this protein, nor did the anti HTLV-I p19 or p24 sera. The sera from B.R.U. and patient 2 did not precipitate the p25 protein from an extract of one of the HTLV-I producing cell lines. However, neither did the goat anti HTLV-I serum from what I can determine from the figure presented! Thus, there is no positive control indicating that they indeed had an HTLV antigen to precipitate. If the goat antiserum indeed precipitated a p24 protein from the HTLV-I producing cell extract, the band of p24 precipitated was extremely weak, indicating that the serum was not very good at precipitating HTLV-I p24, at least not in the hands of Barré-Sinoussi and coworkers. The HTLV-I producing cells were all infected as opposed to only 0.5 to 2% of the cord blood cells. So, if the cord blood cells were infected with HTLV-I and not a new virus, the goat antiserum would still have had a hard time precipitating any protein! An appropriate control would have been a serum from a HTLV-I infected individual. The size of the protein they precipitated is in fact 24,000, the same as that of HTLV-I. The core protein of both HIV and HTLV-I is 24,000.

In reality, in my view there is no evidence whatsoever in this paper that a new human retrovirus has been isolated! With the data presented, the virus they isolated could well have been HTLV-I or in particular HTLV-II. The paper was obviously written in haste, as acknowledged by Montagnier[20], and contains numerous errors and omissions in the figures legends.

After having a first manuscript being rejected by Nature, Gallo suggested to Montagnier to send it to Science and even strongly endorsed the paper to the journal (the book of Nikolas Kontaratos shows a facsimile of Gallo's letter to the editor of Science[21]).

A more thorough description of the French isolate LAV from patient BRU and two new retrovirus isolates from two patients with AIDS was given at a Cold Spring Harbor meeting held in September of 1983. A proceedings, however not peer reviewed, from the meeting was published not until September of 1984[22]. The oral presentation by Luc Montagnier at this meeting is to my mind the first report on a new third human retrovirus, in that electron micrographs on the isolate LAV from patient BRU clearly showed virus with conical cores. A selective tropism of LAV to CD4 positive T-cells (as is the case for HTLV-I) was also demonstrated.

The first publication in a peer reviewed journal indicating the isolation of a new retrovirus, distinct from HTLV-I and HTLV-II, isolated from two siblings with hemophilia B of whom one with AIDS, appeared in Lancet in April of 1984 and was written by the French group[23]. Again the immunological and molecular characterization of the isolated virus does not convincingly separate the isolated virus from HTLV-I. However, an electron micrograph clearly depicts a virus with a lenti retrovirus type morphology having a cylindrical or conical core, distinctly different from the larger spherical core of HTLV-I, and HTLV-II. The paper, however, fails to conclusively link the new virus as the causative agent of AIDS.

In conclusion, by April of 1984 the Pasteur group headed by Luc Montagnier had reported on a new human T-lymphotropic retrovirus distinct from HTLV-I and HTLV-II as judged by morphology and which was present in a few patients with AIDS and lymphadenopathy, as well as, in people at risk of acquiring AIDS. The virus infected CD4-positive T-lymphocytes, the very cells affected in AIDS. Although clearly associated with AIDS, they had not yet shown that the new virus was an etiological agent, and the only one at that, of this new disease.

On May 4th of 1984 four papers by Robert C. Gallo's group were published in Science describing a new human retrovirus virus as the probable cause of AIDS. All four papers were submitted the 30th of March 1984. One paper[24] describes the isolation of the new virus from cultured lymphocytes obtained from 48 different individuals. The culturing technique was what had previously been described by Gallo and which Montagnier's group also used. The new cytopathic (large multinucleated cells) virus isolates were collectively designated HTLV-III and was characterized by having a Mg<sup>2+</sup>-dependent reverse transcriptase, being transmittable by co-cultivation of T cells with irradiated donor cells or with cell free fluids, having distinct morphology by electron microscopy, and by expressing specific viral antigens (indirect immune fluorescence) using a serum obtained from a patient with pre-AIDS (described in an adjoining papers 25 and 26; this serum did not react with cells infected with HTLV-I or HTLV-II), as well as, antisera prepared against purified, whole disrupted HTLV-III. The 48 HTLV-III isolates were obtained from 18 of 21 tested patients with unexplained lymphadenopathy and leukopenia, with an inverted T4/T8 lymphocyte ratio (designated pre-AIDS), 3 of 4 clinically normal mothers of juvenile AIDS patients, 3 of 8 juvenile AIDS patients, 13 of 43 adult AIDS patients with Kaposi sarcoma, 10 of 21 adults AIDS patients with opportunistic infections, and 1 of 22 clinically normal homosexual donors. Importantly, this homosexual donor, from whom HTLV-III was isolated, developed AIDS six month after the virus isolations were performed. This means that these isolations were performed not later than September of 1983. HTLV-III could not be isolated from any of 115 clinically normal heterosexual donors.

In a second accompanying paper [25] antibody reactivity to HTLV-III antigens in patients with pre-AIDS and AIDS was determined by an enzyme-linked immunosorbent assay (ELISA) as well as a Western

electrophoretic blotting technique using a lysate of sucrose gradient purified HTLV-III from a cell line continuously producing HTLV-III. [26] as antigen. The number of sera that gave positive scores in the ELISA were: 43 of 49 (88%) of patients with AIDS (two of whom had developed AIDS after blood transfusion), 11 of 14 patients with pre-AIDS, 3 of 5 intravenous drug users (of which one positive was also homosexual), 6 of 17 homosexual men. It is noted in the paper that these homosexual men had been seeking medical assistance; they probably were not representative of the homosexual population. Out of 186 controls only one scored positive in the ELISA (1 of the 164 normal subjects). The controls also included 3 patients with hepatitis B virus infection, 1 with rheumatoid arthritis, 6 with systemic lupus erythematosus, 4 with acute mononucleosis, and 8 patients with lymphatic leukemias. Of the latter some were positive for HTLV-I. None of these 22 control patients scored positive in the ELISA or Western blot. Of note, in Western blot the antigen most prominently and commonly detected among all of the sera from AIDS patients had a molecular weight of 41,000 (now designated gp41). It was presumed that this is a virus envelope protein (which later turned out to be correct). Others, including myself, have later confirmed that gp41 is extremely reactive in ELISA of sera from HIV infected individuals. In fact we have found that an ELISA having as only antigen a peptide with the amino acids GKLICT, representing an epitope of gp41, reacts positively with the majority of sera from HIV infected individuals.

The French group did not detect gp41 in their immune precipitation studies using purified LAV. Their inability to detect this protein in their ELISA or immune precipitation experiments is probably the main reason that their positive scores with AIDS and pre-AIDS sera were so low. HIV is an enveloped virus and hence fragile. Most certainly they had lost the virus envelope in their purification of the virus.

Taken together, these two papers from Gallo's group for the first time convincingly demonstrated that AIDS was caused by a new human retrovirus distinct from HTLV-I and HTLV-II. It also provided with a blood test (ELISA) by which blood donors could be screened and a confirmation assay (Western blot) for those who tested positive in the ELISA. The authors speculate that the virus they found could well be the same virus that was previously detected by the French group, but direct comparisons had not yet been performed.

A third paper. [26] describes the establishment of cell lines continuously producing HTLV-III. A total of 51 single cell clones (designated H1 to H51) were obtained from a neoplastic aneuploid T-cell line (HUT-78). The clones were tested for susceptibility to concentrates of HTLV-III. All clones were susceptible and permissive for the virus, but virus yields and cell proliferation varied considerably. The best clones (H4 and H9) were used for the long-term propagation of HTLV-III from patients with AIDS and pre-AIDS. Five different isolates using the H4 or H9 clones are presented. Four were obtained by co-cultivating the patients T-cells with the H4 cells and one by infecting H9 cells with a cell free concentrated culture fluid harvested from T-cell cultures from a patient (W.T.) who had lymphadenopathy. One was from an AIDS patient from Haiti (R.F.) and four were from the US. In the paper they also report that some of the 48 isolates described in the accompanying paper[24] also could be propagated in the H4 and H9 clones. The importance of this paper is that for the first time it was shown that one could propagate HIV in large quantities as a source for antigen in a blood test, as well as, for in depth characterization of the virus. It was this paper, and the patent which was based upon it, that later caused the controversy between the NIH and the Pasteur Institute. It turned out that the HTLV-III producing H9 clone selected for the blood test was in fact a pick-up of a French HIV isolate sent to Gallo in September of 1983. This will be discussed later.

The fourth of the Gallo Science papers [27] describes a first attempt to serologically characterize HTLV-III using Western blot and sera from AIDS and pre-AIDS patients. The paper describes for the first time

a virus protein of approximately 130,000 (in fact it is 120,000 and now designated gp120). Also a protein of 55,000 (p55) is described and correctly concluded to be a precursor protein for the capsid protein p24.

Lastly, a photomontage of electron micrographs of HTLV-I, HTLV-II, and HTLV-III with budding virus particles, immature virus particles and mature virus particles is shown. Although the budding and immature virus particles are very similar for all three viruses, the mature HTLV-III viruses are distinctive from those of HTLV-I and HTLV-II.

Three more papers on antibody reactivity to LAV/HTLV-III in patients with AIDS or pre-AIDS were published in the summer of 1984. June 9th, Montagnier's group[28] published an ELISA based on purified virus particles. The presented results were: 18/48 (37.5%) of AIDS patients, 38/51 (74.5%) of pre-AIDS patients and 8/44 (18%) of homosexual men without pre-AIDS, but only one of 100 unselected blood donors were positive. In a note added in proof they claim that by modifying their assay now 75% of AIDS patients and 90% of pre-AIDS patients scored positive. In the Lancet issue of June 30th Gallo's group[29] publish their second report (the first being the one in Science above) on ELISA and Western blot confirmatory assay in a double-blind seroepidemiological study. The composite result of the two assays gave: 34 of 34 AIDS patients were positive (100%), 16 of 19 (84%) of lymphadenopathy (pre-AIDS), 3 of 14 (21%) at risk for AIDS, and none of 14 controls were positive. Lastly, Kalyanaraman et al. [30] published a paper the 20th of July in Science submitted May 4th 1984. This paper was from Donald Francis group at the Center for Disease Control, Atlanta, in collaboration with Montagnier's group at Pasteur. The assay they used was based on immuno-precipitation. The positive scores were: 51 of 125 (41%) of AIDS patients; 81 of 113 pre-AIDS patients, 0 of CDC workers, and 0 of 189 random blood donors. Of 100 blood samples collected in 1978 from homosexual men in San Francisco, only one was positive as opposed to 12 of 50 such sera collected in 1984.

In the July 6th issue of Science (submitted April 6th 1984), Donald Francis' group in collaboration with Montagnier's group reported on the isolation of a retrovirus from a blood donor-recipient pair with AIDS [31]. In an elegant experiment, using a competition radioimmunoassay they clearly show that the viruses they isolated were closely related to LAV but not to HTLV-I or HTLV-II. This is the first paper to show transmission of HIV-1 from one patient to another. This is also the first time, beside the electron microscopic pictures of LAV, Montagnier convincingly shows that LAV is antigenic distinct from HTLV-I.

On August 24th 1984 Jay Levy in San Francisco[32] published a paper in Science (submitted May 31st) reporting that using the Gallo protocol they had isolated a retrovirus with lenti retrovirus type morphology designated ARV for AIDS associated retrovirus in 22 of 45 patients with AIDS. Positive virus cultures were also received from 5 of 10 patients with lymphadenopathy (pre-AIDS), 3 of 14 male sex partners of AIDS patients, 2 of 9 clinical healthy homosexual men, and 1 of 23 clinically healthy heterosexual men. When tested in immune fluorescence with slides containing acetone fixed cells infected with ARV, HTLV-I or LAV, 78/86 (91%) of AIDS patient's sera were positive to ARV infected cells, 22 of 40 (55%) to LAV, and 8 of 60 (13%) to HTLV-I. None of 56 controls reacted to any of the virus-infected cells. The fixed ARV infected cells were from a cell line (HUT-78) successfully established to continuously produce ARV.

The LAV/HTLV-IIIB contamination story and the patent feud between the Pasteur Institute and NIH

Right before the first public announcement by Barré-Sinoussi at a conference at Cold Spring Harbour in May of 1983 of the Pasteur group's findings, the Pasteur Institute filed a patent for the virus they had isolated. Before going public with the four Science papers, the NIH filed for a patent for the blood test described in one of the papers to be published in May of 1984. The United States patent office quickly

allowed the American patent, shortly to be followed by allowances from European patent offices, and a number of American companies started to produce and sell blood tests. The approval of the Pasteur patent was delayed, principally because the French had not reduced their patent to practice, i.e. showed that they had a working blood test in the patent application. This led to a patent feud between the NIH and the Pasteur Institute starting in August of 1985. To solve this feud, the governments of both countries had to become involved. The patent fight came to an end on March 31st, 1987, when President Ronald Reagan and French Prime Minister Jacques Chirac signed an agreement to settle the arguments. The financial outcome of the agreement, however, did not turn out to the satisfaction of the French, and when it became clear that the US patented blood test was based on a laboratory contamination of a French virus the deal was re-negotiated in 1994. It should be stated right away that neither of the scientists at the time stood to gain from respective patent.

The ground for the feud was the following. Montagnier sent his first isolate LAVBRU to Gallo in July of 1983. In May of 1984 Gallo's coworker Sarngadharan brings one of Gallo's five HIV strains (HTLV-III<sub>B</sub>) that grew well in a continuous cell line to Montagnier's laboratory in Paris. In July of 1984 Montagnier sends Gallo a second sample of LAVBRU since Gallo had complained that the first didn't grow well at NIH. Gallo then found and reported[33] that HIV was extremely variable; every isolated strain was different from the other also when obtained from the same individual but at different times. However, the two strains LAVBRU (received in July of 1984) and HTLV-III<sub>B</sub> isolated on either side of the Atlantic Ocean were strikingly similar. Gallo's reaction to this was that Montagnier must have contaminated his cultures with the American isolate, i.e. that the Pasteur group had had a so-called "pick-up" of HTLV-III<sub>B</sub> into his poorly replicating LAVBRU. Gallo gave Montagnier a call, but the latter denied that this could have happened in his laboratory. Since the LAVBRU obtained by other laboratories, before HTLV-III<sub>B</sub> had been introduced to the Pasteur laboratory, had a genome more or less identical to the French isolate, it was concluded that the contamination must have happened in Gallo's laboratory. Gallo found this very strange, since LAVBRU replicated very poorly and could not be transferred to a continuously producing cell line like they had achieved with HTLV-III<sub>B</sub>. On Sunday, November 19th, 1989, the Chicago Tribune published a 16 pages account by journalist John Crewdson of the discovery of HIV. The article concludes that HTLV-III<sub>B</sub> is LAVBRU. Crewdson implied in not so subtle words that Gallo had stolen the virus from the French. This started three separate investigations for scientific misconduct by Popovic and Gallo that didn't end until November of 1993.

The explanation came in 1992. Sequencing the original isolate LAVBRU received in NIH in 1983, Gallo found that it was different from the LAVBRU received in July of 1984. The original LAVBRU was as expected of the slowly replicating CCR5 co-receptor using genotype whereas LAVBRU from 1984 was of the rapidly replicating CXCR4 using genotype. In fact the 1984 LAVBRU was identical to LAVLAI. Thus, the contamination had originally occurred in the Pasteur laboratory. According to Montagnier at least six other laboratories received the LAI sample (under the name BRU) from his group and experienced the same contamination. [34]. Montagnier speculates that this was due to *Mycoplasma pirum* contamination of the cultures infected with LAVLAI. In his review "A history of HIV discovery" in Science. [34] Montagnier writes: "This physical association makes a fraction of the LAI virus highly infectious, and, in fact, this fraction can be neutralized with antibodies against *M. pirum*. As mycoplasmas are common contaminants of cultured cells, an infectious pseudotype virus (LAI associated with *M. pirum*) may have caused several contaminations between 1983 and 1984 in different laboratories".

In Gallo's laboratory LAVBRU had contaminated a pool of viruses from different AIDS patients. Pooling viruses was the idea of Mika Popovic in order to get the "survival of the fittest" to grow out in the continuous cell line H9, a subclone of the HUT-78 cell line. The virus that grew best was named III<sub>B</sub> because it was from the B-pool of two pools in culture.

Many probably thought that the finding that a contamination had taken place already in the Pasteur laboratory was “convenient” for Gallo. However, to “steal” the French virus Gallo must have had a motive. One would have been that they couldn’t culture any virus from AIDS patients and were becoming desperate. However, Gallo had already recognized that the French group was first to isolate a new retrovirus from AIDS patients. More so he endorsed the publication of the first Pasteur paper, which (although in my opinion erroneously) claimed so. Secondly, Gallo’s group already had 48 (sic!) isolates many growing short term in their laboratory of which five were growing in continuous cell lines[24,26]. Gallo’s misfortune was that he decided to choose the IIB for the blood test and for further characterization of the virus. Had he chosen a Haitian strain, which he also had growing long term in the laboratory at the time, we now know that he would have chosen a virus, which was as much of a prototype strain for HIV-1.

It has been questioned whether Gallo indeed had all those isolates. Considering that Gallo published this and that he and his laboratory was scrutinized for almost five years by three different investigations, had Gallo not had those isolates he would for sure have been found guilty of scientific misconduct and expelled from the NIH. Going through 13 foot high pile of Gallo’s lab records including laboratory note-books, some 10,000 man hours of interviews with laboratory personal and other witnesses, all the Office of Research Integrity (ORI; a non-scientist office of government consisting of lawyers and administrators) could come up with in criticism was for Mika Popovic that he wrote “ND” in two occasions in one published table (in paper 25), and found him therefore guilty of scientific misconduct. The table legend didn’t define “ND” and it was the ORI’s understanding that “ND” meant, “not done” and that Popovic indeed had performed the experiment. However, Popovic insisted that by “ND” in his notebook he meant, “not determinable”. The paper was written while Popovic was on holiday back in Czechoslovakia. Following Popovic’s appeal to the Research Integrity Adjudications Panel the decision of ORI’s was reversed. Gallo was temporarily criticized by the ORI for having written a sentence in the discussion of the same paper that LAVBRU had not yet been growing well enough to make possible comparisons with IIB when in fact a technician of Popovic’s had done so. These charges were later dropped by the ORI. In its decision on the Popovic case the Departmental Appeals Board’s Research Integrity Adjudications Panel writes: “One might anticipate that from all this evidence, after all the sound and fury, there would be at least a residue of palpable wrongdoing. This is not the case”. It is safe to say that whatever Gallo claimed he had, he had.

Conclusion

There is no doubt that Luc Montagnier’s group at the Pasteur Institute in Paris was the first to isolate the causative agent of AIDS. Montagnier, however, got the idea to try to isolate a retrovirus indirectly from Robert Gallo and Myron Essex. The protocols he used for virus isolation and RT detection were developed by Robert Gallo and the reagents he used to discriminate the new virus from HTLV-I and HTLV-II were obtained from Robert Gallo. Moreover, it is well known that Françoise Barré-Sinoussi had spent time in Robert Gallo’s laboratory to learn to culture lymphocytes. Robert Gallo was the first to convincingly show that the new human retrovirus (HIV) was the causative agent of AIDS, and the only one at that. He also was also the first to provide a blood test to screen blood donors for HIV infection. The rapid implementation of the latter in the US and Europe probably saved hundreds of thousands of lives.

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Alex Villasenor November 27, 2011 at 1:14 am

Hi Liam,

Excellent job here ... Thanks so much for providing us with glimpses or summaries of some of the craziness that is driving "HIV" research ... I like to use your site as a reference when discussing "HIV"/aids with friends and/or colleagues because most of the other sites seem to be large repositories of articles and research that are a bit difficult to navigate.

-Alex

Cal Crilly November 28, 2011 at 9:23 am

HOW TO TRICK THE TEST

"Three recombinant proteins from HIV-1 (p24, gp41 and gp120)".....these are the antigens to knock off....

New membrane assay for the detection of antibody to HIV-1 and HIV-2 using recombinant proteins.  
<http://gateway.nlm.nih.gov/MeetingAbstracts/ma?f=102199042.html>

I think you can try Bromelain, Turmeric, Olive Leaf Extract and and Green Tea, Sage or Lemon Balm to block the antigens.

Maybe not Sage a week before the test as it has estrogens which in theory could stimulate retroviruses.

Green tea definitely is needed.

Eat enough veggies like the brazil nuts, broccolli garlic for selenium, lemons and all the rest of the yum stuff with a folate and B12 supplement, that's for the viral load test.

Eat good amounts fat (: that keeps T-cells up and get enough sun and sleep so you're hormones stay normal.

Cod liver oil if it rains or gets cloudy for days as low Vitamin D makes viruses express.

When you stay in front of a computer at night you destroy the melatonin in your pineal gland and that effects things too.

So get good sleep.

Fish may be the best, it also has decent lysine levels, milk and red meat have a molecule we are allergic to and can cause inflammation.

You may get through one day.

[editorial note on the abstract below – the words “isolates” is not only misleading, it is fictional. By an “isolate,” AIDS researchers mean a cancer or other cell line in which they've been cultivating proteins for some time, and which gives rise to reverse transcriptase enzyme activity. And it's hard to say what the budding and virus indicates, but they forced some HERVS or “exosomes” out of cells by stimulating them. Which is normal, and happens all the time. AIDS folk just call these normal bits “HIV” when they're getting paid to do so. No, really. That's how it works.]

“CD44 was concentrated in two locations: in clathrin coated pits and at the base of budding HIV virions. For many (although not all) virions, CD44 co-located with budding HIV and appears to form a “collar” at the base of the budding virus. We saw no differences in CD44 localization between HIVLAI/III and HIVBAL isolates, nor differences in HIVLAI/IIIB and HIVBAL gp120gp41 “spike and ball” morphology. No significant immunogold staining of ICAM-1 was seen in these same experiments although low expression of ICAM-1 on the cell surface was shown by indirect immunofluorescence and flow cytometry. However, by using AA5 cells with high expression of ICAM-1 on the cell surface, an even distribution of ICAM-1 both HIVLAI/IIIB-infected and uninfected AA5 cells was observed. We saw only rare association of ICAM-1 molecules with HIV virions. Co-localization of CD44 with HIV virions at the site of virus budding supports the hypothesis that CD44 may be involved in HIV budding.”

Co-localization of CD44 expression with HIV budding in HIV-infected cells.

<http://gateway.nlm.nih.gov/MeetingAbstracts/ma?f=102222881.html>

I think and suspect Bromelain may have an effect on p24 but I may be wrong, I'll work out the maths later

But it stops HIV budding in CD44....

Be careful with bromelain being a blood thinner, it's good to be on lysine first and make sure you have vitamin k in the diet to not bleed.

CD44 cell surface proteins are involved in leukocyte binding to endothelium and the metastatic spread of tumor cells. Using flow cytometric analysis (FCMA), we investigated the effects of the proteases bromelain, papain, trypsin, and chymotrypsin on the density of CD44 molecules present on human leukemia Molt 4/8 cells.

Bromelain was found to be most active in reducing CD44 receptor density.

Bromelain proteinases modulate the cd44 expression on human molt-4/8 leukemia and sk-mel-28 melanoma-cells in-vitro.

<http://www.ncbi.nlm.nih.gov/pubmed/21559602>

High levels of the adhesion molecule CD44 on leukemic cells generate acute myeloid leukemia relapse after withdrawal of the initial transforming event

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3072510/>

Recent experimental study found that OLE (olive leaf extract) has anti-HIV activity by blocking the HIV virus entry to host cells

Computational study of bindings of olive leaf extract (OLE) to HIV-1 fusion protein gp41

<http://www.ncbi.nlm.nih.gov/pubmed/17537437>

Binding of the green tea catechin, epigallocatechin gallate, to the CD4 receptor on human immune cells resulting in inhibition of HIV-1-gp120 binding and HIV-1 infectivity

[http://www.jimmunol.org/cgi/content/meeting\\_abstract/178/MeetingAbstracts/S69-c](http://www.jimmunol.org/cgi/content/meeting_abstract/178/MeetingAbstracts/S69-c)

Extracts from lemon balm (*Melissa officinalis* L.), peppermint (*Mentha × piperita* L.), and sage (*Salvia officinalis* L.) exhibited a high and concentration-dependent activity against the infection of HIV-1 in T-cell lines, primary macrophages, and in ex vivo tonsil histocultures with 50% inhibitory concentrations as low as 0.004%. The aqueous Lamiaceae extracts did not or only at very high concentrations interfere with cell viability. Mechanistically, extract exposure of free virions potently and rapidly inhibited infection, while exposure of surface-bound virions or target cells alone had virtually no antiviral effect. In line with this observation, a virion-fusion assay demonstrated that HIV-1 entry was drastically impaired following treatment of particles with Lamiaceae extracts, and the magnitude of this effect at the early stage of infection correlated with the inhibitory potency on HIV-1 replication. Extracts were active against virions carrying diverse envelopes (X4 and R5 HIV-1, vesicular stomatitis virus, ecotropic murine leukemia virus), but not against a non-enveloped adenovirus.

Aqueous extracts from peppermint, sage and lemon balm leaves display potent anti-HIV-1 activity by increasing the virion density

<http://www.retrovirology.com/content/5/1/27>

and keep up the turmeric defo as this is used in leukemia and any thing that stops leukemia naturally stops a so called “leukemia retrovirus” like HIV and while you’re at it use coconut oil in the curries

Tomatoes are fab too cos of lycopenes, artichoke pastas... actually all the foods with flavonoids have effect... berries etc..

Mushrooms I believe should thrown in a plenty due to wound healing properties and the sulphurs.. all of them  
hope this helps