BACK

The "HIV" GENOME

Eleni Papadopulos-Eleopulos Nuclear Physicist and Biophysicist, Perth, Western Australia

Valendar F. Turner Emergency Physician, Perth, Western Australia

John M Papadimitriou Emeritus Professor of Pathology, Crawley, Western Australia

Barry A. P. Page Physicist, Perth, Western Australia

David Causer Physicist, Perth, Western Australia

www.theperthgroup.com

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The "HIV" experts including Robert Gallo agree that to prove the existence of a retroviral RNA (genome) it is absolutely necessary to have electron microscopic evidence for purified retroviral particles.[1] Neither Montagnier nor anyone else has published such proof. A detailed discussion of the "HIV" genome can be found in a paper published in *Continuum* in 1996,[2] "The isolation of HIV: Has it really been achieved? The case against". In regard to Montagnier's claim of "Molecular cloning of lymphadenopathy-associated virus",[3] here is the relevant extract from this paper (with minor editing).

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6.1 MINIMUM EVIDENCE REQUIRED TO PROVE THE EXISTENCE OF HIV DNA
If "HIV DNA" is the genome of a unique retroviral particle then the most basic requirement is proof for the existence of a unique molecular entity "HIV DNA", that is, unique fragments of DNA identical in both composition and length in all infected individuals. The claim that a stretch of RNA (cDNA) is a unique molecular entity which constitutes the genome of a unique retrovirus can be accepted if and only if it is shown that the RNA belongs to a particle with the morphological, physical and replicative characteristics of a retroviral particle. Proof of these properties can only by obtained by isolating the putative viral particles, that is, by obtaining them separate from everything else, extracting the nucleic acids and demonstrating that such particles are identical (their constituents including their nucleic acids are identical) and infectious. The correct procedures, now having been used for over half a century to achieve this proof, require demonstration that:

1. In "infected" cell cultures (co-cultures) there are particles with a diameter of 100-120nM containing "condensed inner bodies (cores)" and surfaces "studded with projections (spikes, knobs)".[4]
2. In sucrose density gradients the particles band at a density of 1.16 gm/ml.
3. At the density of 1.16 gm/ml these is nothing else but particles with the morphological characteristics of retroviral particles.
4. The particles contain only RNA and not DNA and that the RNA consistently has the same length (number of bases) and composition no matter how many times the experiment is repeated.
5. When the particles are introduced into secondary cultures, but mindful of the critical caveat discussed below:
    (a) the particles are taken up by the cells;
    (b) the entire RNA is reverse transcribed into cDNA;
    (c) the entire cDNA is inserted into the cellular DNA;
    (d) the DNA is transcribed into RNA which is translated into proteins;
6. As a result of 5 the cells in the secondary cultures release particles into the culture medium.
7. The particles released in the secondary cultures have exactly the same characteristics as the original particles, that is, they must have identical morphology, band at 1.16 gm/ml and contain the same RNA and proteins.

The caveat is that while the introduction of the majority of infectious particles into cell cultures and subsequent release of similar particles is proof that such particles are indeed infectious, this is not the sufficient case for retroviruses. The basis of this exception is the fact that "one of the most striking features that distinguishes retroviruses from all other animal viruses is the presence in the chromosomes of normal uninfected cells, of genomes with those of infectious viruses".[5] In fact, a cell may contain the genome of many retroviruses. As far back as 1976 retrovirologists recognised that "the failure to isolate endogenous viruses from certain species may reflect the limitations of <u>in vitro</u> cocultivation techniques".[6] In other words, the finding of a retrovirus in both the primary and secondary "infected" cultures/co-cultures is not proof that the cells have been infected with an exogenous retrovirus.

One way which will suggest but will not prove that the cells acquired virus from the outside (exogenously acquired retrovirus, infectious retrovirus) and have not assembled a retrovirus from information already existing in normal cells (endogenous retrovirus), is to conduct experiments that use controls, that is, to run in parallel with test cultures/co-cultures control cultures/co-cultures. The only difference between the test and control cultures should be the introduction of particles into the test cultures. In other words, apart from the introduction of particles, in every other respect control cultures must be dealt with identically.
For example:
    (a) because detection of RT and retroviral genetic sequences and release of retroviral particles depends on the metabolic state of the cells, the physiological state of the cells used in the control cultures should be as close as possible to those of AIDS patients;

(b) because the mere act of co-cultivation alone may lead to release of endogenous retroviral particles, if test cells are cocultured, so should the cells used in control experiments; [7]
(c) extracts, even from normal unstimulated cells, when added to the cultures may increase endogenous retroviral expression.[8] Because of this, when cells are cultured with "HIV" (supernatant or material which bands at 1.16 gm/ml), the controls must be cultured with similar material from cell cultures originating from sick individuals with illnesses similar to AIDS, that is, matched individuals who are immunosuppressed;
(d) the appearance of endogenous retrovirus can be accelerated and the yield increased a million fold by stimulating the cultures with mitogens,[9] mutagens, chemical carcinogens and radiation.[10 11] If test cultures are exposed to or employ such agents so should the controls;
(e) since AIDS patients and those at risk of developing the syndrome are exposed to strong oxidising agents,[12 13] the control cells should also originate from such patients;
(g) to avoid observer bias and in the best interests of science, blind examination of test and control cultures/cocultures should be performed.

6.2 EVIDENCE FOR THE EXISTENCE OF "HIV DNA"
6.2.1 In 1984, in the first of two papers, Montagnier and his colleagues described the following experiment: "Because LAV can induce T-cell fusion and because EBV [Epstein Barr virus] is known to have fusion activity in B cells, we performed co-infection experiments of unfractionated lymphocytes (B and T) with both viruses. It was hoped that stable hybrids of LAV-infected T cells and of EBV-transformed B cells would be formed and that such hybrids would be able to continuously produce LAV. Several regimens were tried. The one that gave rise to continuous productive infection of LAV was the following. Whole lymphocytes of F. R. were first stimulated for 24 hours with Protein A and then infected with and EBV strain, M81, derived from a nasopharyngeal carcinoma. Five days later, half of this culture was infected with LAV as described (1) and then divided in two subcultures: one was cultured in medium lacking T-cell growth factor (TCGF: interleukin-2), the other in medium containing TCGF. As expected, the TCGF-fed culture produced LAV as detected by a peak of RT activity appearing between day 12 (day 6 after LAV infection) and day 21 in the supernatant. In contrast, the cells cultured in the absence of TCGF did not yield any detectable RT...On day 19, at the time of decline of LAV production, a subculture of the TCGF-fed cells received fresh T cells from the same donor: these T cells had been activated for 3 days with phytohemagglutinin (PHA)...Six days later (day 25), a new peak of RT appeared, but contrary to the first infection, it was not transient...At the time of the second LAV infection, large round cells transformed by EBV could be readily seen in this culture, as well as in the control culture not infected with LAV, indicating that immortalization of the B cells by EBV had already occurred. The immortalized B-cell line was termed RF8".[14] (Reference 1 to which Montagnier refers, is the 1983 paper in which Montagnier *et al* described the first "isolation" of HIV[15]).

In the second study, 200 ml of supernatant from the "HIV infected" FR8 cells were banded in sucrose gradients, "Virus containing fractions were pooled" and centrifuged. (It is not

stated how they determined the existence of "virus", in which band(s) (fraction(s)) "virus" was found, how many bands if any were found to have particles, or why there were more bands than one (1.16 gm/ml) containing the "virus"). The pellet was incubated with several substances, dATP, dGTP, dTPP, dCTP including [α-^{32}P]dCTP and an oligo(dT) primer. DNA/RNA hybrids were obtained and the ^{32}P DNAs were said to be the cDNA of the HIV genome. From the DNAs, cDNAs, thus obtained, three clones "pLAV13, 75 and 82, carrying inserts of 2.5, 0.6 and 0.8 kilobases (kb), respectively, were characterized further. All three inserts have a common restriction pattern at one end, indicative of a common priming site. "The 50-base pair (bp) common HindIII-PstI fragment was sequenced and shown to contain an oligo(dA) stretch preceding the cloning dC tail. The clones are thus copies of the 3' end of a poly(A) RNA. The specificity of pLAV13 was determined in a series of filter hydbridization experiments using nick-translated pLAV13 insert as a probe". Firstly, "using an adapted spot-blot technique" they tested the pellet obtained from the supernatant of "LAV infected" normal lymphocytes and CEM cells as well as non-infected lymphocytes. The "infected" pellets were positive and the non-infected negative. "Second, the probe detected DNA in the Southern blots of LAV-infected T lymphocytes and CEM cells. No hybridization was detected in DNA from uninfected lymphocytes or from normal liver". No details are given regarding the method used to produce "infection", but it would appear that the normal cells and the CEM cells were cultured with supernatant from the FR8 cells, that is, the same supernatant they used to obtain the probe! They concluded: "Together, these data show that LAV pLAV13 DNA is exogenous to the human genome and detects both RNA and integrated DNA forms, derived from LAV-infected cells. Thus, pLAV13 is LAV specific".[3]

6.2.4 SUMMARY AND DISCUSSION

It is obvious that although Montagnier, Gallo and Levy and their respective colleagues refer to virion or virus particles purification or isolation, none of these groups have presented evidence for the isolation of retrovirus particles or even the isolation of virus-like particles, the first and absolutely necessary step in proving the existence of a retroviral genome. (At the time of writing, neither has any other group of HIV/AIDS researchers). Finding some RNA which bands at 1.16 gm/ml, selecting from it a poly(A) rich fraction, or a fragment of a given length, even if always found to be the same length and sequence, and referring to it as HTLV-III, LAV, ARV does not constitute such proof. It must be stressed that even if the RNA is incorporated in a particle which in sucrose density gradients bands at 1.16 gm/ml, this is still not proof that it is retroviral RNA. According to John Coffin, one of the best known experts on the retroviral genome, there are particles "with a full complement of viral proteins, but the particles contain a collection of cellular RNAs and only about 1% genomic RNA...assembly of particles does not require the genome...in its absence other RNA molecules may be substituted".[5] It is important to note that although all groups, Montagnier's, Gallo's and Levy's refer to the material from the culture supernatants which in sucrose density gradients bands at 1.16 gm/ml as viral particles, virions, and to the RNA and proteins at that density as "particle-associated" RNA or proteins, not one of the groups presented evidence for the existence at this density of any particles, retroviral-like or otherwise, pure (isolated) or otherwise. Instead these researchers cultured lymphocytes from AIDS patients and stimulated (activated) them with a wide variety of agents. Reverse transcription of A(n).dT$_{15}$ in the culture supernatant was considered proof

for infection with a retrovirus or even proof of isolation. Supernatants from these cultures were introduced into cultures of leukaemic or transformed cell lines. With the supernatants from these cultures they performed two types of experiments:
(a) The supernatants were banded in sucrose density gradients. At the 1.16 gm/ml band (and sometimes at other band(s), at least in Montagnier's group experiments, this is not made clear), they found fragments of RNA of certain lengths (although no two had the same length) or were rich in adenine, (poly(A) rich fragments), and called these "HIV RNA", the "HIV genome". Using a (dT) primer the "HIV RNA" was transcribed into a complementary DNA (cDNA);
(b) The supernatants were introduced into another set of the transformed and leukaemic cell lines as well as into stimulated cultures of normal T-cells. The DNA from these cells, as well as the DNA from the cultures to which no supernatant was added, were hybridised using probes from the cDNA. Positive results were obtained only with the DNA from the cells to which the supernatants were added. This evidence was interpreted as proving that the "HIV DNA", the retrovirus, originated from the AIDS patients and in fact that these patients acquired it from the outside, that is, the retrovirus was exogenous.

There are many problems associated with these experiments and their interpretation. Among the many questions their conclusion raises the most obvious are:

1. HIV is said to be a retrovirus and retroviruses are particles which contain among other things, RNA. How then is it possible to claim that the RNA which banded at 1.16 gm/ml, "HIV RNA", is the genome of a retrovirus without proof that it is a constituent of a particle, viral or non-viral which bands at this density?
2. RT is not specific to retrovirus and in fact A(n).dT_{15} can be reverse transcribed by all cellular DNA polymerases α, β and γ. Is it possible then to consider reverse transcription of A(n).dT_{15} as proof for HIV isolation or even detection of a retrovirus? Even if the process of reverse transcription is specific to retroviruses, can the detection of a process ever be considered proof for the isolation of an object, in this case, retroviral particles?
3. Cell culture supernatants will contain both DNA and RNA including some enclosed in cellular debris (fragments) especially if cellular viability is not one hundred percent as is the case in cultures used by the three groups. The RNAs may include messenger RNA (which is adenine rich), as well as high molecular weight heterogenous nucleic RNA. These RNAs, in addition to having high molecular weight and heterogeneity in size, also have poly(A), with the poly(A) attached at the 3' end of the molecule, and may be RNAase resistant. Actinomycin, inhibits its synthesis and also interferes with its proper processing and breakdown.[16] From animal virology it is also known that non-retroviral RNA and DNA also bands at 1.16 gm/ml.[17] How is it then possible to claim that just because an RNA bands at 1.16 gm/ml and is adenine rich or has a certain length, it is "HIV RNA"? If this RNA is "HIV RNA", then what is the other RNA and the DNA which also bands at this particular density? If the latter are cellular why not the poly(A)RNA as well?
4. By definition, retroviruses are infectious particles which contain only RNA. When they enter a cell the RNA is reverse transcribed into DNA, which is then integrated into cellular DNA as a provirus, which means that "HIV DNA" will be present only in the cell and nowhere else. Yet many HIV experts including Gallo have shown that both the supernatants of "infected" cell cultures and the "HIV particles", that is, the material which

bands at 1.16 gm/ml, contains "HIV DNA" which "may integrate directly into the host chromosomal DNA".[18-20] The question then arises, is the "HIV DNA" the result of "HIV RNA" reverse transcription or is it *vice versa*?

5. It is accepted that the HIV RNA is localised in a condensed core surrounded by a "lipid-bilayered envelope derived from the cellular membrane of the host cell, studded with virally encoded gp120 and myristylated protein, p17. The so-called core-envelope link (CEL) attaches the core to the envelope".[20] One of the best know facts in biology is that condensed cores (chromatin) is transcriptionally inactive. This is one of the reasons why viruses, including retroviruses, to multiply, must first enter cells where their chromatin is decondensed. However, in a paper published in 1993 by Hui Zhang and colleagues including Poiesz, from Suny Health Science Center at Syracuse, New York, wrote: "We have shown that in the absence of detergent, large amounts of DNAase-resistant viral DNA can be synthesized within intact HIV-1 virions, indicating that this phenomenon is not dependent on perturbation of the viral envelope. [Not to mention decondensation of chromatin]. Nascent viral DNA synthesis also occurred in purified virions incubated at 37°C in cell-free human physiological fluids including seminal plasma, breast milk, and fecal fluids".[20] This means that either (i) the "intact HIV-1 virions" perform a function that no other biological system with very condensed chromatin can perform or (ii) the "HIV RNA" found in the supernatants or in the "purified virions" is present in an unembodied form or (iii) the "HIV RNAs" are *de novo* synthesised in the cell cultures (see 6.3.5).

6. At present there is ample evidence that any RNA or DNA present in the supernatant, irrespective of its origin, especially when cells are stimulated by polycations and oxidising agents, will be taken up by the cells (see 7.1). How is it then possible to claim that a positive hybridisation signal in cells cultured with the same "HIV DNA" containing supernatant as the supernatant from which the "HIV DNA" probe originated but not in other cells is proof that the "HIV DNA" is the genome of an exogenous retrovirus?

7. The first, absolutely necessary step in proving that the "HIV DNA" originated from the lymphocyte cells of AIDS patients and those at risk, is to perform hybridisation experiments using the DNA of their fresh, uncultured lymphocytes and the "HIV DNA" as a probe. It is hard to understand why neither Montagnier's nor Levy's group reported such experiments. Gallo's group did and the results were negative (see 6.4.4). How is it then possible to claim that "HIV DNA" is the genome of an exogenous retrovirus which originated from AIDS patients and those at risk?

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Again, for a detailed discussion of the “HIV” genome please see reference 2, “The isolation of HIV: Has it really been achieved? The case against”. Here it suffices to mention several facts which have come to light since 1996.

In a paper published in *Science* 20th October 1984,[21] one reads “as the source of virus, the original strain of LAV (LAV-1) grown on stimulated lymphocytes of an adult healthy donor (F.R.) was used at the beginning of these studies”. This means that the F.R. culture was infected with BRU’s “HIV”. However, as Montagnier admitted in his 1997 interview, in what he called BRU’s “purified” virus, there were no retroviral particles.[22] Hence, whatever was added to the F.R. cultures, it could not have been “HIV”. If as

Montagnier claimed in 1983, he obtained purified retroviral particles, why did he not take the RNA from these particles, obtain the cDNA and then proceed with the cloning and characterisation of the HIV genome? Why did Montagnier have to take such elaborate and indirect steps to obtain the HIV genome? Even if he had proof of a retroviral genome, given the drastic manipulations of the cell cultures/co-cultures, what made him claim that he characterised the genome of a retrovirus whose ultimate origin was BRU?

In her Nobel Lecture of December 8th 1983, Barbara McClintock stated “"rapid reorganisation of genomes may underline some species formation. Our present knowledge would suggest that these reorganizations originate from some "shock" that forced the genome to restructure itself in order to overcome a threat to its survival...Major genomic restructuring most certainly accompanied formation of new species". The "genomic shock" which leads to the origin of new species may be "either produced by accidents occurring within the cell itself, *or imposed from without such as virus infections, species crosses, poisons of various sorts, or even altered surroundings such as those imposed by tissue culture*. We are aware of some of the mishaps affecting DNA and also of their repair mechanisms, but many others could be difficult to recognize”[23] (emphasis ours and see this reference for examples).

In the 27 month long BMJ Online debate,[24] Dr. Brian Foley, custodian of the Los Alamos HIV Database, agreed with us that no proof exists that the “HIV” RNA, the “HIV” genome, originated from material which contained nothing else but “HIV” particles. He also agreed that poly(A) RNA is not specific to retroviruses. [25] Nonetheless he insisted that the existence of the “HIV infectious molecular clone” proves that the RNA (cDNA) is “HIV” and thus the “HIV” genome and “HIV” exists. However, despite repeated requests, Brian Foley could not give us even one reference that proves the existence of the “HIV infectious molecular clone”. (Please note: there is a difference between cloning of the “HIV” DNA and cloning of the virus. The former means introducing the cDNA into a suitable vector and obtaining the same DNA. The latter means introducing the cDNA and obtaining a retrovirus particle containing an RNA complementary to the introduced cDNA).

Despite the fact there are no published studies on the HIV genome which include controls, much less proper controls, HIV experts claim that the HIV genome is unique and found only in AIDS patients and those at risk. However, in 1998 researchers from the USA led by Dr. Eva Rakowicz-Szulczynska published evidence showing that nucleic acid sequences similar to the "HIV" env and gag genes were found in 95% of breast and gynaecological cancer in women and prostate cancer in men. “The DNA fragments amplified in seven blindly selected breast cancer samples were sequenced. The breast cancer DNA sequences showed at least 90% homology to the HIV-1 gene for p41”. In 1999 they reported “DNA sequences with 90 to 96% homology to HIV-1 Env were recently detected by PCR in breast cancer and in prostate cancer cells…The results obtained strongly suggest that the long-postulated breast cancer virus may, in fact, be related to HIV-1”.[26-29]

According to one Australian HIV expert, Professor David Gordon, the 10% difference in the nucleic acid sequences between the “cancer virus” in breast, gynaecological and

prostate cancer on the one hand and HIV-1 on the other, makes them two distinct retroviruses. "In particular, perhaps they might be 90% similar, which might sound quite a lot but in genetic terms that's very distinct. So the difference between you and me is one in one thousand of our nucleic acid bases is different, so 10% is enormous. The difference between humans and chimpanzees is probably one to two per cent". (T1096)[30]

In regard to the 10% differences we asked Brian Foley "What is the typical and range of variation (least to greatest) between these [HIV-1] genomes?" He provided us with the following data:

> "Percent DNA Sequence Identity between
> HIV-1 M group subtype B isolate HXB2 vs:
> HIV-1 O group isolate ANT70 63.82%
> HIV-1 N group Isolate YBF30 68.03%
> HIV-1 M group subtype G Isolate 03GH175G 81.77%
> HIV-1 M group subtype C Isolate 02ZM108 82.51%
> HIV-1 M group subtype B Isolate 05CSR3 88.25%"

We also sent Brian Foley copies of Dr. Rakowicz-Szulczynska's papers and sought his views on her data. Below are his responses.

Email from Brian Foley January 11th 2008

> "My take is that she had false positive PCR reactions. The sequences she came up with were HIV-1 M group subtype B. She states in at least 1 of her papers that she used HIV-1 samples as positive controls for the primers she used. So we know she had not only HIV-1 in her lab, but also that she had HIV-1 PCR product in her lab.
>
> If she is correct, and an exogenous retrovirus (she always claims this is an exogenous virus, and claims that the sequences could not be amplified from normal tissue, only from some cancer tissues), then people should be very alarmed about a cancer epidemic".

(Indeed, "Every three minutes a woman in the United States is diagnosed with breast cancer" [1]. The combined annual number of new cases of breast, ovary and prostate cancer in the US in 2007 was 419,027 cases [2]. That is, a new case every 75 seconds).

1. breastcancer.org.
http://www.breastcancer.org/about_us/press_room/press_kit/cancer_facts.jsp?gclid=CKKGIIv40pMCFQvFbwodNRgcig#statistics
2. American Cancer Society. Cancer Statistics 2007 Presentation.
http://www.cancer.org/docroot/PRO/content/PRO_1_1_Cancer_Statistics_2007_Presentation.asp
3. CDC. HIV/AIDS in the United States.
http://www.cdc.gov/hiv/resources/factsheets/PDF/us.pdf

Email from Brian Foley January 12th 2008

"Below is a BLAST result, querying the entire GenBank database with one of Eva's sequences. In this region of the Envelope gene, the typical inter-patient distance between any two sequences is between 88% and 95% identity within one subtype (in this case, HIV-1 M group subtype B). Between subtypes Such as comparing Eva's sequence to a subtype C virus, the percent identity is between 84% and 90% identity. This region of Envelope is rather hypervariable, so there is typically almost as much diversity within a subtype as between subtypes in this region. But these sequences of Eva's are still clearly subtype B, which is typical for North America".

There is no doubt that Dr. Rakowicz-Szulczynska has extensively studied what she calls her "cancer virus" and has shown it present in the neoplastic cells of 100% of breast cancer patients and the vast majority (90-95%) of gynaecological and prostate cancer cells but not in the non-cancer cells of these patients or the cells of healthy individuals. According to Brian Foley, Dr. Rakowicz-Szulczynska's "cancer virus" is "clearly…HIV-1 M group subtype B…which is typical for North America".

If 36% differences between HIV-1 genomes mean one and the same virus, then less than 10% differences, no matter where and in whom they are found, represent the same virus, as Brian Foley points out.

If 10-36% differences between genomes do represent the same virus, and if "HIV" DNA is unique, then, as we have pointed out to a BBC journalist who recently became interested in our research, every 75 seconds a man or a woman in the United States is infected with HIV. This is to be contrasted with the accepted HIV incidence of 40,000 cases annually, that is, one infection every 13 minutes.

On the other hand, since cervical cancer is accepted to be caused by HPV and the other gynaecological and prostate cancers are not infectious, and since the "cancer virus genome" is found only in diseased cells, the only alternative explanation must be that the "HIV genome" is non-specific. And in fact may be the result and not the cause of the pathogenic process, as we have postulated from the very beginning (see "Montagnier, T4 cells (acquired immune deficiency and our oxidative theory of "HIV"/AIDS").[31] Be this as it may, as far back as 1975 the well known retrovirologist John Bader wrote "Molecular hybridization techniques, employing DNA synthesized on a viral RNA template and RNA extracted from tumor cells, have been used in attempts to identify viral nucleotide sequences in tumor cells. The validity of such studies depends upon the absolute purity of the virions, or on the purity of the viral RNA template used in the synthesis of the putative viral DNA".[17] Since today nobody has purified HIV, molecular methods cannot be used to prove HIV infection.

BACK

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