Link to the Perth Group

March 6th 2010

The link for this file is http://leederville.net/links/SullivanMar062010.pdf

The Perth Group response to Kevin Sullivan's post # 21

Re the RAK papers you wrote: "I reviewed some of the information that you provided. One of the key points to your argument that HIV testing is flawed comes from the papers on the RAK antigen".

You did not say where you obtained "the information we provided" but in regard to "HIV" testing, we have never argued our case using "the RAK antigen" or RAK antigens or any other data in Rakowicz-Szulczynska's papers. Indeed, our principal paper on "HIV" antibody testing was published several years before Rakowicz-Szulczynska's published her findings. What we argued then and subsequently is the absence of proof the "HIV" antibody tests are specific for "HIV" infection. Hence, in framing our arguments it was not only impossible to use Eva Rakowicz-Szulczynska's data, there was no need to. The only way to prove the specificity of the antibody tests is to compare the presence or absence of a positive test with the presence of absence of "HIV" – as determined by isolation/purification. That is, to use "HIV" itself as the gold standard. This has never been reported, making it impossible to know how many, if any, seropositive individuals are infected with a retrovirus "HIV". Biotechnology companies are well aware of this deficit and it is regularly asserted as a disclaimer in their antibody test kit packet inserts.

You wrote: "It is true that HIV does make proteins p24, p41(gp41) and p120(gp120)".

This is not true. As Montagnier and Gallo point out, the only way to prove the proteins are "HIV" is to purify the "HIV" particles. Sylvia offered to provide the evidence for purification but has not delivered. In fact she cannot because it has not been done.

"You wrote: "The HIV envelope protein gp160 is a polyprotein that is cleaved into gp120 and gp41".

Not true. To the contrary, gp160 and gp120 are polymers of gp41 which, according to Montagnier, is actin.

You wrote: "The [Rakowicz-Szulczynska's] lab used PCR to detect HIV-1 like sequences in cancer cells. There are a number of problems with their finding. First they amplify a 142 base pair (bp) fragment to make conclusions from. HIV is on the order of 10,000 bases this represents less than 1.5% of the HIV sequence".

HIV experts also utilise short "HIV" DNA fragments "to make conclusions from". When they find a particular fragment in their patients they claim it is

proof for the presence of the whole “HIV” genome. Similarly, Rakowicz-Szulczynska did not search for the whole “HIV” genome in her patients yet you conclude or at least imply that her patients harboured “less than 1.5% of the HIV sequence”. Do you imagine if the same “1.5% of the HIV sequence” was found in a gay man with PCP there would be any dispute about the nature of this DNA or that his cells contained the whole “HIV” DNA sequence?

Perhaps you think finding “less than 1.5% of the HIV sequence” proves Rakowicz-Szulczynska did not find “HIV DNA” at all. Perhaps you think the unexpected nature of this finding adds weight to this conclusion. If this is the case let us acquaint you with one of several emails sent to us from Brian Foley, custodian of the Los Alamos HIV Database.

“12/01/2008
Below is a BLAST result, querying the entire GenBank database with one of Eva’s [Rakowicz-Szulczynska] 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”.

“HIV” DNA sequences have not been reported from normal, healthy individuals. The question we must consider is why they are present in all Rakowicz-Szulczynska’s breast cancer patients (and the majority of her other cancer patients). There are two possible explanations.

1. They are the result of a viral infection with “HIV” or an “HIV-like” retrovirus, which is highly associated with breast and other common cancers.
2. The sequences are not of viral origin but the result of factors associated with neoplastic change. Note: These DNA sequences were absent in adjacent non-cancerous tissue taken from the organ that contained the cancer.

Since breast, ovarian and prostate cancers are not infectious (transmissible), the only conclusion possible is (2). This means that “HIV” DNA cannot be unique. If the factors leading to breast cancer result in the appearance of “HIV” DNA in cancer cells then similar factors may also cause the appearance of the same DNA in other diseases.

You wrote: “From the best of my knowledge the work of this author was not followed up on by others”.

In the United States a women is diagnosed with breast cancer every three minutes. Given (i) no one accepts human breast cancer is transmissible; (ii) the annual spectre of 175,200 women discovering they harbour “HIV” DNA;

(iii) the additional problems this would cause their families and their doctors; (iv) the problems it would cause the army of "HIV" experts and their retroviral theory of AIDS; it is no wonder no scientist has followed up Rakowicz-Szulczynska's research. Even though her research constituted a promising development for the early detection of common cancers, including the highly lethal ovarian cancer with an incidence of 21,550 cases and 14,600 deaths in the USA in 2009.

You wrote: "As a friendly suggestion I would not use this series of papers as a cornerstones to your argument in the future".

We appreciate friendly suggestions but this suggestion is arcane. If these DNA sequences are, as Dr. Foley says, "clearly [HIV] subtype B, which is typical for North America", but are not caused by infection with "HIV", then what are they? How did they arise? If they are "clearly [HIV] subtype B, which is typical for North America", but not of viral origin, then we repeat, the notion that "HIV" DNA is unique cannot be substantiated. There must be ways of acquiring "HIV" DNA which are not a retroviral infection. This is a significant problem for the HIV theory of AIDS. Is this why you are recommending we do not use it? Do you intend to simply ignore these data?

Re morphology, you wrote: "Your group claims that because HIV has morphological characteristics of both class C and D retroviral particles". Your next sentence reads "In your literature you equate this difference to mistaking an attractive model for a chimpanzee".

The first sentence makes no sense. We have never said "HIV has morphological characteristics of both class C and D retroviral particles". What we have said is that in the published scientific literature different HIV experts have classified the "HIV" particle as a type C, (not class, there is no class at the species level), and a type D particle, and a lentivirus.

You wrote: "In your literature you equate this difference to mistaking an attractive model for a chimpanzee".

Incorrect. We used a taxonomical analogy to illustrate the difference between type C and type D particles. To say that at one and the same time "HIV" is a type C and a type D particle is no different from saying an object is simultaneously an attractive model and a chimpanzee. And given that "HIV" is also classified as a lentivirus it must now rate as a "Holy Trinity".

You wrote: "First if everything were classified by morphology based on grainy black and white images that lack the resolution for specific detail…"

If this is your assessment of electron microscopy and its many skilled professional practitioners then nothing we can say will alter your viewpoint. Perhaps you should seek counselling from Professor Hans Gelderblom at the Koch Institute in Berlin. Or Professor John Moore who has collaborated with him. During the Parenzee court case in 2007 Gallo discussed the critical significance of the morphology of "HIV" when he referred to a paper published

by him and his electron microscopist, Matthew Gonda. Gallo stated, "…although I am a co-author, I contributed really nothing to that paper. It was Gonda that recognised the lenti/retrovirus nature, not me. He corrected a mistake from lack of the structural familiarity by almost all retrovirologists with this family known by veterinarians. That is the answer. Montagnier, of course, did not know". So Gallo acknowledges the role that electron microscopy , that is, morphology, plays in determining the taxonomy of "HIV". You do not share Gallo's view.

You wrote: "Sequencing their DNA would reveal their true classification. This is also true of viruses".

In the same court case Gallo testified that "HIV" is a lentivirus and added "it is clear even by its genomic analysis". However, commenting on Gonda and Gallo's paper, Montagnier in his 2000 book *Virus* wrote: "*Science* published an article by his [Gallo's] group that showed similarities of sequences between HTLV-I, -II, and -III, and then, more curiously, between these and the lentivirus prototype, the Visna sheep virus. The two sets of findings proved entirely false, and nothing from these two articles holds any longer. We were beginning to seriously wonder whether *Science* was not starting to compete with the *Journal of Irreproducible Results*! The Pasteur team, to clear the matter up, also unraveled the Visna virus sequence in record time…there was no significant homology (similarity) between the Visna and LAV ["HIV"] sequences". True classification?

You wrote: "Lack of a spike protein".

There is unanimous agreement that the "spike protein" is of critical importance to "HIV" and the "HIV" theory of AIDS.

According to:
1. Weber and Weiss, "The first step in any viral infection is the binding of the virus particle to a component of the host cell's membrane...For some time it has been known that the binding takes place when CD4 interacts with an "envelope" protein of the virus called gp120" (Weber & Weiss, 1988);
2. Moore *et al*, "HIV infection of CD4+ cells is initiated by an interaction between its surface glycoprotein, g120, and the cellular antigen CD4" (Moore & Nara, 1991);
3. Mortimer, "The gp120 surface protein interacts with CD4 receptors on T4 cells so that the viral RNA can be injected into the cell" (Mortimer, 1989);
4. Matthews and Bolognesi, "First gp120 binds to the CD4 receptor on an uninfected cell; then gp41 becomes anchored in the adjoining membrane; next the two membranes begin to fuse, and the virus spills its contents into the cell" (Matthews & Bolognesi, 1988);
5. Redfied and Burke, "Infection begins as a protein, gp120, on the viral envelope binds tightly to a protein known as the CD4 receptor on the cell surface" (Redfield & Burke, 1988);
6. Rosenberg and Fauci, "The initial event in the life cycle of HIV is the high-affinity binding of the HIV envelope glycoprotein (gp120) to CD4 that is present on the surface of cells" (Rosenberg & Fauci, 1990);

7. Montagnier *et al*, “The gp120 is responsible for binding to the CD4 receptor” (Gougeon *et al*, 1993);
8. Haseltine and Wong-Staal: gp120 is “crucial to HIV’s ability to infect new cells” (Haseltine & Wong-Staal, 1988);
9. Callebaut *et al*, “The human immunodeficiency virus (HIV) infects lymphocytes, monocytes, and macrophages by binding to its principal receptor, the CD4 molecule, through the viral envelope glycoprotein gp120. The V3 loop of gp120 is critical for HIV infection” (Callebaut *et al*., 1993).

Thus, there is general agreement that the HIV envelope protein gp120 is crucial for HIV infection. However, agreement also exists that “gp120 is easily shed by virus and virus-infected cells” (Bolognesi, 1990). Gelderblom and his colleagues at the Koch-Institute in Berlin who have conducted the most detailed electron-microscopy studies of “HIV particles” have shown that the knobs on the surface of the particles, where the gp120 is found, are only present in immature (budding) particles, which are “very rarely observed”. “Mature”, cell-free particles do not have knobs, that is, gp120 (Hausmann *et al*., 1987). (These references are in our paper at http://theperthgroup.com/SCIPAPERS/ephemophilia.html

How many spikes (knobs) does the “HIV” particle possess? Here the “HIV” experts do not agree.

In *Virus*, Montagnier wrote: “Particles of HIV are shaped like little spheres, each with roughly 80 rounded projections shaped like pegs”.

In the 2005 textbook co-authored by Niel Constantine (who appears in the *House of Numbers*) and Professor Elizabeth Dax, Head of the Australian National Serology Laboratory and International Expert on HIV testing, there are “72 knobs or spikes of the external envelope of HIV”.

According to Hans Gelderblom and John Moore, immediately after being released from the cell membrane “HIV particles” possess an average of 0.5 knob per particle which are rapidly lost, but also pointed out that “it was possible that structures resembling knobs might be observed even when there was no gp120 [knobs] present, i.e. false positives”. That is, there may not be any knobs at all.

In 2003 Kuznetsov and his colleagues reported a study utilising atomic force microscopy that contradicted what all “HIV” experts claim. They reported “We suggest that spikes, knobs, observed by negative-staining electron microscopy may be an artifact of the penetration of heavy metal stain between envelope proteins. Indeed, the term “spike” appears to have assumed a rather imprecise, possibly misleading definition, and might best be used with caution”. In other words, this posits zero knobs on the so-called “HIV” particle.

Hence the “HIV” scientific consensus “HIV” particle knob count is roughly 80, exactly 72, 0.5 (on average), possibly zero and actually zero.

Significantly, the electron micrograph http://theperthgroup.com/Nobel/MontagnierEMNobel.pdf projected during

Montagnier's 2008 Nobel lecture shows a few particles devoid of knobs and possessing barely any of the principal morphological features of retroviruses, much less, a lentivirus. This is not the EM that appears in the version of his Nobel lecture recently published in *Virology*.

We are still waiting for an EM showing purified "HIV" with all the morphological characteristics "HIV" experts claim the virus particles possess.

In regard to "Reverse Transcriptase and retroviruses" you wrote: "Over the course of discussion your group argues against the existence or the uniqueness of the enzyme reverse transcriptase".

Partially true. We have never claimed reverse transcriptase (and reverse transcription) do not exist. However, in regard to uniqueness we are in good company. Temin, Baltimore, Montagnier, Gallo and Weiss. Or read Bruce Albert's book *Molecular Biology of the Cell* or one of the latest publications on this topic "Retrotransposons, reverse transcriptase and the genesis of new genetic information". *Gene* 448 (2009) 180–186.

You wrote: "Without an enzyme performing this function there could be no retroviruses".

True but so what?

You wrote: "Based on many statements made by your group one would include that you do not only deny the existence of HIV but also of all of the retroviruses".

So a retrovirus "HIV" exists because "all of the retroviruses exist"?

You wrote: "However, inconsistently you argue that HIV has morphology that is reminiscent of type C and D retroviruses".

We argue no such thing. We cite evidence that HIV experts have classified "HIV" as a type C and type D particle and a lentivirus. Neither they nor we use the word "reminiscent".

You wrote: "This statement lends credence to the fact that retroviruses do indeed exist. This in turn validates the existence of the enzyme. Hepatitis B viruses carry a DNA genome when they are released from the cell. This virus passes through an RNA intermediate in the infected cell that is reverse transcribes by a virally encoded RT. I would suspect that your group would not deny the existence of hepatitis B".

So, again, "HIV" exists because reverse transcriptase exists and hepatitis B virus exists"? The enzyme does not exist because retroviruses exist and *vice versa*. Since you accept that hepatitis has a reverse transcriptase then how can you conclude the detection of RT or RT activity is proof for infection with "HIV". Especially since so many individuals in the AIDS risk groups are

infected with hepatitis B, including infection of their T-lymphocytes, as Weiss pointed out.

You wrote: "Your group chooses not to believe that HIV exists".

This is wrong and it is not a matter of belief. It is the lack of evidence in the scientific literature which had led us to conclude there is no proof for the existence of "HIV". Witness Sylvia Piccinotti's problem. She claimed she would provide explanations about our "errors" and when we took her at her word, she disappeared. Is it possible that Sylvia, like us, could not find the evidence that proves the existence of "HIV"? This would at least make her disappearance rational. Could the same happen with others who take a dispassionate look at the evidence?

You wrote: "Where do viral sequences come from then? They do not exist in the human genome or the genomes of any other species"

What is your evidence they are viral? Obviously you do not appreciate how the "HIV" sequences were obtained. Maybe you'll be surprised when you take the trouble to find out. All "HIV" experts agree the only way to prove the existence of the "HIV" genome (RNA) is to purify the virus. This has not been done. Instead, "HIV" RNA is a poly(A) RNA banding at the density of 1.16 g/ml. The material in the 1.16 g/ml sucrose density gradient was obtained from cell cultures/co-cultures which are exposed to a plethora of agents (chemical and irradiation) including transforming agents. For further information please read
http://theperthgroup.com/REJECTED/GENOME1f.doc and
http://theperthgroup.com/CONTINUUM/pgvsduesbergreward.html

It appears you are stuck on the notion that, because some particular DNA sequence is not found in normal healthy humans this proves the sequence must be exogenously acquired. You, like the "HIV" experts, seem to believe in an invincible, unalterable DNA hiding behind a force-field the envy of Darth Vader. You do not seem to appreciate that a new DNA sequence could arise *in situ, in vivo, de novo*. Your view is not shared by eminent biologists.

Although more than half a century has passed since the Nobel laureate Barbara McClintock discovered the phenomenon of transposition, which can lead to the appearance of new genotypes and phenotypes, at present it is still generally accepted that any time one finds a particular stretch of RNA in a cell, for example, in a T-lymphocyte, unless RNA or DNA has been introduced from outside, all T-cells, regardless of their physiological state or stresses to which they are subject, will contain a corresponding stretch of DNA. In other words, the DNA (genes) in a cell are invariant and all RNA molecules in the cell are subservient to a matching length of DNA. However, according to McClintock, the genome can be restructured and not only by transposition. In her Nobel lecture of 8th December 1983, she said, "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. Homeostatic adjustments to various accidents would be required if these accidents occur frequently. Many such mishaps and their adjustments would not be detected unless some event or observation directed attention to them...Unquestionably, we will emerge from this revolutionary period with modified views of components of cells and how they operate, but only however, to await the emergence of the next revolutionary phase that again will bring startling changes in concepts".

In your posting you imply the "HIV" genome exists in AIDS patients and those who have a positive antibody test. Your assumption is questionable. In 1984 Gallo reported "...the observation that HTLV-III sequences are found rarely, if at all, in peripheral blood mononuclear cells, bone marrow, and spleen provides the first direct evidence that these tissues are not heavily or widely infected with HTLV-III in either AIDS or ARC". And in a 1994 meeting held in Washington DC sponsored by the US National Institute of Drug Abuse, Gallo admitted "We have never found HIV DNA in the tumor cells of KS...In fact we have never found HIV DNA in T-cells".

If you are fully acquainted with the literature you will point out these studies are based on Southern blot hybridisation (SBH), and no doubt claim this all changed with the advent of PCR technology. And you are right. PCR did save the "HIV" genome and thus "HIV" and the "HIV" theory of AIDS. Yet one cannot help wondering why it is possible to detect the "HIV" sequences with PCR but not SBH. According to Gallo, SBH can detect one infected cell in a million. As far as the PCR is concerned, researchers from several institutions from the USA pointed out in 1996: "To evaluate the sensitivity and specificity of PCR, investigators must ascertain whether study participants are infected with HIV. Typically, a new test is compared with a superior reference (or gold standard) test…The lack of an appropriate reference test (for HIV PCR) substantially complicates evaluation". Even if a gold standard did exist, the specificity of the PCR still could not be determined for the simple reason, that this test, as the same researchers point out, is not standardised". "The criteria for determining when PCR gave positive results varied among the studies". Even when the lack of standardisation is ignored and totally unsuitable gold standards are used, such as the antibody test, the specificity of PCR varies from 40% to 100% leading researchers from different institutes in the USA to conclude "Our investigation produced two main findings. First, the false-positive and false-negative rates of PCR that we determined are too high to warrant a broader role for PCR in either routine screening or in the confirmation of diagnosis of HIV infection. This conclusion is true even for the results reported from more recent, high-quality studies that used commercially available, standardised PCR assays…We did not find evidence that the performance of PCR improved over time".

You wrote: "Why is it only found in patients who according to the establishment has HIV?"

First, this is not true as evidenced by Rakowicz-Szulczynska's's findings. Second, in studies of "HIV"/AIDS no researcher reports the use of proper controls. Proper controls means running parallel experiments which include tissues obtained from patients who are ill with diseases similar to but not AIDS, and in whom there are similar in not identical laboratory findings. For example, abnormal indices of immunological function and cellular oxidation. When performing experiments such tissues must be treated in exactly the same manner as the tissues from AIDS patients. In addition, for the sake of scientific accuracy, the experiments must be done blindly.

You wrote: "Some of the strongest proof that HIV exists comes from the fact that there is documented evidence of viral sequences sharing the most similarity to the partner that passed the virus to the other partner".

Please explain exactly how phylogenetic analysis proves the existence of "HIV"? How do you know the similar DNA shared by two people is that of a retrovirus?

The "evidence" that "HIV" is sexual transmitted is obtained solely from epidemiological studies. All sexually transmitted agents are transmitted from the active to the passive partner and *vice versa*. This translates to transmission between men and women in heterosexual sex and between the passive and exclusively active male in gay sex. No epidemiological study has proven that "HIV" (a positive antibody test or AIDS) is bidirectionally transmitted.

In 2007 Laura Geretti from the Department of Virology, Royal Free Hospital, London UK, wrote an article entitled "The use of phylogenetic analysis as evidence in criminal investigation of HIV transmission". This paper made it quite clear it is impossible to use DNA sequences data, including phylogenetic analysis, to prove two individuals harbour an identical "HIV" genetic sequence. Geretti and her colleagues concluded "Consequently, even with the appropriate controls, phylogenetic analysis cannot 'prove' transmission".

You wrote: "Your arguments about the Montagnier paper twist what the intent of the experiments were".

Montagnier's intent was to prove the existence of a new virus which Montagnier claimed he did. Do you have another reason for Montagnier's experiments?

You wrote: "In the case of retroviruses it is accepted by your group that they possess reverse transcriptase".

Yes. RT is the "*sine qua non*" of a retrovirus. That is, its presence is necessary. However, it is not specific and Gallo was well aware of this problem at the beginning of the 1970s. Later, in 1976, he wrote: "Release of

virus-like particles morphologically and biochemically resembling [containing RT] type-C virus but apparently lacking the ability to replicate have been frequently observed from leukaemic tissue".

You wrote: "Looking for this enzymatic activity (which is rare in human cells and only occurs under a defined set of conditions) is much easier than looking for something unknown by PCR".

Two problems here: 1. RT activity is not rare in human cells. 2. You cannot look for something new by PCR before you have the primers and probes you need to look for it. These can only be obtained by first purifying the viral particles. Please give us the proof that anyone has purified the "HIV" particles.

You wrote: "Your group is 100% right, poly T is not specific in any way for viral RNA, it will pick up any cellular mRNA in addition to vRNA".

Is poly T a typing error? Do you mean poly A or RT? If so, you cannot claim RT activity or poly(A) RNA are retroviral and not cellular. And without such proof there can be no proof for the existence of "HIV".

You wrote: "They are looking for the enzyme. It happened to sediment exactly where one would predict viral particles to settle. Is that a coincidence? Now if it were cytoplasmic or membrane bound RT one would not get a finite band of activity in the middle of the fractions. This indicates whatever was sedimented was mostly uniform".

Since you accept it was Montagnier and his colleagues who proved the existence of HIV let us take a close look at his evidence.

1. In a culture containing cells from BRU they detected reverse transcriptase activity;
2. The same activity was detected in a co-culture, that is, a culture of BRU cells to which were added T cells from a healthy donor;
3. The same activity was detected in umbilical cord T-lymphocyte cultures to which supernatant from the co-culture was added.

In the last culture, using the electron microscope, they also detected retrovirus-like particles. The finding of reverse transcriptase activity in the three consecutive cultures was considered proof that BRU was infected with a retrovirus which was transmissible to T-lymphocytes. Nonetheless, it appears that Montagnier and his colleagues were not 100% sure of their findings. This is small wonder since at least ten years earlier they knew that RT activity and virus-like particles are not retroviral specific. Hence, in 1983 they reported: "That this new isolate was a retrovirus was further indicated by its density in a sucrose gradient, which was 1.16 [g/m]". At the 1.16g/ml band, which they claimed was their "purified" virus, they found:

(i) RT activity, and not the enzyme reverse transcriptase as you claim. (The cellular DNA polymerases γ and β can also reverse transcribe RNA into DNA);

(ii) three proteins, p80; p45 (p41); p25 (p24) which reacted with antibodies in BRU's serum.

Then, for no reason whatsoever, they ignored the p80 protein and the antibody which reacted with it. In the case of p45 (and presumably the antibodies which reacted with it) they claimed it was cellular actin (the molecular weight of actin is 41,000). The p24 protein was said to be "HIV" and the antibodies which reacted with it, "HIV" antibodies. Because none of the proteins which sedimented "exactly where one would predict viral particles to settle" (the 1.16 g/ml band), did not react with an antibody to HTLV-I p24, they claimed to have discovered a new retrovirus, consisting of only one protein, p24.

Fourteen years later, in July 1997, during a videotaped interview conducted by the French journalist Djamel Tahi, Montagnier stated: "analysis of the proteins of the virus demands mass production and purification. It is necessary to do that". Then, astonishingly, he said that in 1983 he did not purify the virus: "I repeat we did not purify". And, unbelievably, in what they called "purified, labeled virus" they were unable to find any particles with "the morphology typical of retroviruses". In a similar interview conducted in December 2005, Charles Dauget, the Pasteur Institute electron microscopist, confirmed that in the "purified" virus, they found only cellular debris. In other words, Montagnier accepted that his 1983 evidence did not satisfy his own criteria necessary to prove the existence of a new retrovirus, or even a retrovirus. Obviously, since Montagnier found RT activity in a "finite band" which had cellular debris but no retrovirus particles, the enzyme can only be cellular. And it can have nothing to do with a retrovirus and much less with transmission of a retrovirus, even if detected in an infinite number of consecutive cultures.

Similarly, any protein present in the "finite band", irrespective of its reaction with antibodies present in BRU's serum, including p24, must be cellular. Ditto for any RNA including poly(A) RNA and primers and probes based on this particular RNA. In conclusion, in 1997 Montagnier admitted that his 1983 evidence did not satisfy even his own criteria necessary to prove the existence of a new retrovirus, "HIV".

Our questions to you are:

1. Do you agree with our analysis of Montagnier's data – yes or no?
2. If no, could you please explain where and why we are wrong?
3. If yes, don't you think that like us, you, as a scientist, must question the generally accepted view that in 1983 Montagnier discovered a new retrovirus, "HIV"? And thus, like us, become a dissident?

You wrote: "I truly believe that people are entitled to believe and practice whatever they want provided it does not cause others harm. I truly believe your group preaches views that do harm others and society as a whole".

We do not “preach”. We write scientific papers based on our research and interpretation of the HIV/AIDS literature. If you find errors, or do not agree, do what true scientists do, write the editors of the journals where we have published and defend your thesis.

The fact that you accuse us of harming “others and society as a whole”, means you have either not read or understood our papers. There is no scientific basis to your claim. Public health policy based on our views is more stringent than that recommended by the HIV experts. People who do not understand this fact reveal their ignorance of our views.