

**IS THE DISSIDENT SCIENCE “HIGHLY QUESTIONABLE”,
“EMBARRASSING” AND “DAMAGING”?**

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September 2010

Please note: This document was written before the Vienna meeting but we hesitated to send it because we thought we would most likely be accused yet again of “fuss, screaming, bitching, criticism”. However, since Christian Fiala accused us of not talking we decided to send/post it.

DE HARVEN AND HIS CLAIM THAT ELENI STOLE STEFAN LANKA’S WORK

As Crowe puts it, de Harven’s “great status” in retrovirology is based on his electron microscopy work, including an electron micrograph of purified particles of the so-called “Friend Leukaemia Virus”, a “virus” which neither he nor anybody else has proven to exist. (See our analysis at <http://www.tig.org.za/Friend.pdf>, indexed at Anthony Brink’s website on RA: <http://www.tig.org.za/RA.htm>).

De Harven became a dissident after:

- (i) Peter and we published our most important papers on “HIV” and AIDS; following Peter’s claim for the *Continuum* prize and our response: “The isolation of HIV: has it really been achieved? The case against”;
- (ii) we commented on the significance of the Bess et al and Glushankof et al *Virology* papers;
- (iii) Djamel Tahi’s interview with Montagnier, the first question commencing: “A group of scientists from Australia argues that nobody up till now has isolated the AIDS virus, HIV...”.

He announced his “dissidency” with a short note to Eleni and two short publications in *Continuum* entitled “Pioneer deploras “HIV” (*Continuum* Vol. 5, No. 2, Winter 1997/98) and “Remarks on methods for retroviral isolation” (*Continuum* Vol. 5, No. 3, Spring 1998).

The first was published in the same issue as Djamel’s interview and our commentary on Montagnier’s responses. In his article de Harven describes the method he used to purify the “Friend leukaemia virus”, together with a picture of his purified “virus”. “HIV” is mentioned only in the penultimate sentence: “It is only in 1997 after fifteen years of intensive HIV research, that elementary EM controls were performed, with the disastrous results recently reviewed in *Continuum*”. No references were provided.

The third sentence in his second publication reads: “Still, according to E Papadopoulos et al¹ and S Lanka², isolation of hiv from fresh plasma of aids patients has never been achieved under any circumstances”.

Here he describes the method used to prove purification of animal viruses, which he applied to purify the Friend leukaemia “virus” in 1965, and again he published the EM of his purified “Friend Leukaemia Virus”. But he added: “However sedimentation in sucrose density gradients, at the density of 1.16g/ml soon became the most popular method for retrovirus purification⁸.” Ref. 8 is the Sinoussi (Barre-Sinoussi) Chermann and associates paper published in *Spectra* in 1973, a difficult to obtain publication introduced by us to the HIV/AIDS debate and which we had already discussed extensively. He then repeats some of the evidence we had already published regarding “HIV” isolation and “it’s” markers and concludes: “In conclusion, and after extensive reviewing of the current aids research literature, the following statement appears inescapable: neither electron microscopy nor molecular markers have so far permitted a scientifically sound demonstration of retrovirus isolation directly from aids patients. This conclusion fully confirms the recent reports published in *Continuum* by E Papadopoulos and by S Lanka”.

He substantiates his claim to have conducted “extensive reviewing of the current aids research literature” by citing 15 references. Four of them are his own publications on “Friend leukaemia virus” and two are ours, both wrongly cited. He claims that in Ref. 1 we presented evidence that “isolation of hiv from fresh plasma of aids patients has never been achieved under any circumstances”. Incredibly, instead of citing any of our many papers in which we presented such evidence, including:

1. the 1988 *Medical Hypothesis* paper entitled “Reappraisal of AIDS: Is the oxidation induced by the risk factors the primary cause”, which he considers to be “classical”;
2. “The isolation of HIV: has it really been achieved?”, which he claims to have repeatedly read;
3. our commentary on Djamel’s interview;

he cited papers where the existence/non-existence of “HIV” is not even mentioned. His reference 2 is apparently an article published by Stefan in a German newspaper, which we still have not seen. He also cites the Bess and Gluschkoff papers in *Virology* in March 1997 which we were the first to analyse, and he failed to mention this fact or our analysis.

Reference 15 is an interview contemporaneously given to Paul Philpott in which he repeated everything we said regarding “HIV” isolation but attributed all to himself. (When we questioned him about this he blamed Paul). Incorrectly citing our papers, not citing us, and implying he is the first person to show that “HIV” has not been isolated has become the norm for de Harven. He has tried to convince everybody that, unlike us, he is a great retrovirologist; he developed a method for retroviral purification; he purified the first murine retrovirus (the “Friend leukaemia virus”); and he coined the term “budding”. None of which is true. (<http://www.tig.org.za/Friend.pdf>). On more than one occasion he even tried to convince us that if we wanted our science to have any weight we should re-write it and include him as a co-author. In fact he has used every trick in the book, each more

unethical than the other, behind our backs to marginalise us. Some examples of this behaviour are mentioned in *Rethinking AIDS and the Perth Group* <http://www.tig.org.za/RA&RAConference&PGSep1609.pdf>

De Harven's incredible effort culminated last year when he claimed that the "HIV does not exist" idea is not the Perth Group's, it was that of Stefan Lanka in 1994(?). And the Perth Group swiftly appropriated it!!!!". What surprised us the most was (a) this time he attributed the "idea" to Stefan and not to himself; (b) with very few exceptions the dissidents remained silent. Among the few who did speak up were David Crowe and Celia Farber. Crowe thought de Harven was correct and Celia congratulated him for clarifying the matter. The same Celia who was writing about Eleni before Stefan wrote a word about "HIV"/AIDS, in fact before anybody had ever heard of him. The same Celia who once told us how Harvey Bialy had warned her off writing about "HIV" isolation despite her fervent wish to do so. The same Celia who said we had wronged Neville Hodgkinson in August 1993 (regrettably true, to his and our detriment), by not providing a picture of our group to accompany the front page article he wrote about our work in regard to "The virus that never was" in the London *Sunday Times*. And the same Celia who cannot see that such an article would not have been possible if we had stolen Stefan's work.

Among the very few people who supported us was one of de Harven's best friends, Anthony Brink. Anthony asked de Harven to apologise. De Harven responded: "I am perfectly willing to apologise if it is proven that I did a mistake! I spent late hours, last night, reviewing, in "Virusmyth" all the long papers by the PG. I found many papers, mostly in Continuum, in the 1996-1998, explaining in extreme details how difficult, close to impossible it has been to isolate and purify HIV. They NEVER, incidentally, quoted my early work (1965...) on murine retrovirus purification!!!! Still, my method was recognised as the best by many!! The PG totally ignored it! And I could never find, in PG's papers, a statement to the non-existence of HIV. I found that statement in Lanka's 1995 paper in Continuum. If you can give me a PG reference (1995 or earlier) that you feel I missed, send it to me right away!". (De Harven was personally made aware by us and others, that in the 1988 *Medical Hypotheses* paper, which de Harven calls "classical", we wrote: "It must be emphasised that unlike other viruses HTLV-III/LAV has never been isolated...By isolation of the virus, in fact, it is meant transient detection of...[which] are non-specific...HTLV-III/LAV has never been isolated from fresh AIDS tissues").

In his response on 30 July Anthony wrote:

"My dearest Etienne,

It pains me to convey the explanation for why the PG never 'quoted [your] early work (1965...) on murine retrovirus purification...recognised as the best by many'.

The PG aren't among those many, and the reason they aren't among those many is that they think it's no good. No good at all.

I attach their critical analysis of it.

As for your charge that the PG 'swiftly appropriated' Lanka's work, which is to say stole it like thieves in the night without even hesitating to do so, you'll need to look wide of the virusmyth website for the facts.

Perhaps in the course of your investigation of the matter, to find supporting evidence for the terrible guilty verdict that you have already pronounced on a charge of the

gravest capital crime that it is possible to commit in science, you should telephone Eleni to establish the facts. And after that, perhaps sound out Lanka. Since you have pronounced Eleni a scientific criminal, based on your reading of some materials you found on the internet, you'll agree that the gravity and urgency of this matter can hardly be exaggerated".

In a further email on 2 August Anthony wrote:

"Please pardon my insensitive suggestion that you telephone Stefan Lanka and Eleni Papadopulos-Eleopulos to investigate the veracity of your claim that Eleni 'swiftly appropriated' Stefan Lanka's discovery that 'HIV' hasn't been shown to exist: I was about to email you their telephone numbers when I recalled your trouble hearing over the phone...

The international AIDS dissident community knows that the almost universally accepted and implemented HIV-AIDS model is wrong.

Until the other day we all knew that the author of the most piercing, radical critique of this model is the Australian physicist Eleni Papadopulos-Eleopulos.

When to my stunned amazement you proclaimed her a common thief.

I'm sure you'll agree that it's profoundly important that the history of the priority of the missing virus discovery be recorded correctly.

And that there should be no unfounded and unwarranted controversy muddying it...

Your stealing accusation, Etienne, is consequently extremely grave, and it must be resolved promptly, either with proof tabled in support of it, or with an unequivocal retraction coupled if possible with a due apology.

This matter cannot be left floating undetermined.

For instance, it would never be acceptable for me to publicly accuse you of paedophilia, and then respond casually when challenged: 'Well that's how it looked to me from some things I read on the internet, even though no one else in the world has drawn the same appalling conclusion, but I'll gladly withdraw my accusation that you're a paedophile if you show me the proof that I'm wrong.'

It doesn't work that way, Etienne...To get to the bottom of this, you can write to Eleni...Let's settle this quietly!".

We have never had a word from de Harven. Perhaps we should let Stefan talk.

In "HIV, Reality or Artefact", 1995, Stefan wrote: "In 1993 a research group from Perth, Australia succeeded in publishing a paper on the HIV test. Since then anybody could have read for him or herself that no AIDS test could ever work, because HIV has never been isolated nor even shown to exist. Since AIDS research and the media have largely ignored any critique of HIV=AIDS, especially the essential question of whether HIV really does exist, it is time to call again for a reappraisal of the whole HIV/AIDS hypothesis".

In "Rethinking HIV", 1996, Stefan wrote: "The distinguished Australians led by Dr Eleopulos-Papadopulos have already provided a detailed reply to the Duesberg claim, so I shall endeavour to explain how the erroneous concept of retroviruses brought about the present situation...So one can only guess why molecular biologist Peter Duesberg refers to such a standard experiment as proof of the existence of "HIV". As the group around Eleni Eleopulos et al has shown (3) neither he nor anybody else has shown that the genetic pieces of "HIV" used in the transfection experiments he cites (9) were isolated out of a virus".

Anyone who's read Stefan's two articles, which are posted on Virusmyth, and our responses to Peter, including "The isolation of HIV: has it really been achieved? The case against", will realise that Stefan does not claim anything original.

One of the first things de Harven did after becoming a dissident was to attend the Geneva International AIDS Conference. Describing the conference's events, Alex Russell wrote in *Continuum*, Vol. 5, No. 4, late summer 1998: "Once the first speaker had begun, Stefan Lanka and I went up to the empty panel and handed the somewhat 'isolated' Montagnier a document entitled: 'Three Open Questions to Prof. Dr. Luc Montagnier' which he then began to read looking nervous and bewildered. Here is an extract to which he did not respond: "In 1993 Eleni Papadopoulos-Eleopoulos published a study about HIV-antibody tests in the journal *Bio/Technology*. It is claimed that prior to publication the study had been approved by the Pasteur Institute in which you are working...Papadopoulos-Eleopoulos draws the scientific conclusion that the existing HIV-antibody tests, due to lack of complete isolation are not reliable...Professor Montagnier, is it true that your institute had approved the study of Papadopoulos-Eleopoulos prior to publication or is this wrongly claimed? (Dr Stefan Lanka)"" .

Joan Shenton wrote: "Virologist Dr Stefan Lanka moved to the dais next. In view of what had been said by the Perth scientists, he called for the World AIDS Conference programme to be changed there and then. He said all HIV testing should be banned forthwith; discussion of the evidence from Perth should be put high on the conference agenda, and a review of current antiviral treatments should take place, with new non-toxic treatment options based on reconstituting the immune system taking the place of the damaging combination cocktails".

In an email (February 1999) de Harven wrote: "I mainly wanted to tell you about the telephone calls I got from Stefan Lanka. All I wrote to him was that, in my views, alleged isolation of HTLV1 was as scientifically unsound as that of HIV. Then, he called back asking me to develop that argument in a more formal way, which I was hesitating very much to do...He is a very difficult man to follow! You give him one inch and he eats up your full arm!! I also understood, from the "Open Letter to P.D." written by M Nitsche (a student from Berlin, masterminded by S Lanka!), how narrow a view Lanka has about virology...I really doubt he ever read a single virology paper from before 1970?". (Apparently, Stefan thought that there were only two retroviruses, HTLV-I and HTLV-II, he did not know that retroviruses were said to exist long before 1970, the year when reverse transcription was discovered, but were known as oncoviruses).

In his numerous telephone calls (we used to joke that Stefan's telephone bill would "break the Bundesbank") over many years following the publication of our *Bio/Technology* paper Stefan repeatedly urged us to write a paper on HTLV-I and HTLV-II and put the question: do HTLV-I and HTLV-II exist?

On one occasion Eleni responded: Stefan, what does Gallo say, which is the most studied virus, the virus we know more about than any other? To Stefan's response, "HIV", she said: If this is the case, and if, as we say, the presently available evidence does not prove the existence of "HIV", how is it possible for anybody to claim there is proof for the existence of HTLV-I and II? Subsequently, Stefan began to claim that no retroviruses exist. From what we hear, including from the person who best knows

the PG/Stefan story and who helped Stefan in many ways in his *Continuum* publication, Stefan remains an honest scientist. It's a pity that some other dissidents do not follow his example.

How much reliance can one place on a person who, on the one hand, declares we have misappropriated the work of Stefan Lanka but on the other, is on public record as saying "There is no question that your group in Perth was the first one to strongly call the attention of the scientific community on the fact that HIV had never been properly isolated and probably didn't even exist...with every best wishes. Etienne, April 10, 2000".

FURTHER CLAIMS OF THEFT BY THE PERTH GROUP

Not long after Crowe became a dissident he edited an article entitled "A note to the scientific and medical community. Could it be because she is just a woman?". In this article one reads: "People in the general public seem to at least recognise the name Peter Duesberg, while almost none Eleni-Eleopulos. Why is the public familiar with Peter Duesberg, and so few people have heard of Eleni Papadopulos-Eleopulos? We can rightfully say that Ms Eleopulos has scientifically proven that Robert Gallo never isolated a communicable virus, now called HIV...And I believe that it is because of her gender that we have never heard a bit of praise on her behalf".

Not long after he edited this piece, and subsequent to his becoming the "leader" of over 2500 dissidents, including the Perth Group, the first thing he did was to dictate what we all must do. Our participation in the RA Board "can't happen until the existing Board members can conclude your participation will be co-operative". Since becoming a "leader" he has spared no effort to discredit the Perth Group. His most obvious and best known being his sabotage of the Parenzee case. Since then he has not stopped claiming there is no scientific basis for our claim that semen is toxic, suggesting that the basis of our claim is not scientific. It is just homophobia.

There is no doubt that Crowe has done a lot of harm to the Perth Group. But the harm he has done us is insignificant compared to the harm he has done the dissident movement, to Peter himself and to those at risk of AIDS.

Crowe has repeatedly said that Duesberg is wrong, there is no evidence that the existence of "HIV" has been proven. At present it appears that virtually all dissidents accept there is no evidence that proves the existence of "HIV". That is, Peter has made an error of interpretation of the scientific evidence. This being the case, and if Crowe really respects Peter, as he wants us to believe, then he should put all his efforts into trying to correct Peter. Instead he has done everything possible to propagate Peter's error.

Although Crowe claims Peter is wrong in regard to the existence of "HIV", he has never mentioned that the Perth Group showed this to be the case. In fact Crowe refused our request to post the summary of our scientific contribution to the "HIV"/AIDS debate on the RA website. Instead, he claims he has been questioning "HIV" for a long time and by reading the literature he concluded that "HIV" does not exist.

In an email to us copied to many other dissidents Crowe wrote: “From a perspective of AIDS, Duesberg’s theory is virtually identical to yours. Duesberg says that drugs, malnutrition and exposure to foreign blood products cause AIDS. So do you. You have identified oxidative stress as a common denominator, which is important but the only additional factor you introduce is semen”.
http://www.tig.org.za/Crowe_responds_to_the_Perth_Group’s_reply_to_Fabio_Franchi.htm

In other words, the Perth Group stole the drug theory of AIDS from Peter Duesberg and just added semen to it. Let us quote from a letter Eleni wrote to Peter in May 1988: “...apart from my usual filing, I have a file (admittedly thin), with papers by authors who, to me, appear to be people who can look critically and objectively at their own work as well as that of others and who put scientific principles above their personal interests. In early 1985 one of the papers filed there was, “Activated Proto-Onco Genes: Sufficient or Necessary for Cancer?” When last year I read your article, “Retroviruses as Carcinogens and Pathogens: Expectation and Reality” I was delighted, firstly, because my initial impression of you was confirmed and secondly, because it appears that we share some scientific ideas. Since then every paper I have read by you or about you (Bio/Technology, New Scientist, The Listener, California Monthly), has increased my delight...In late 1985 – early 1986 I wrote the attached paper entitled, “Reappraisal of Aids – is the Oxidation Induced by the Risk Factors the Primary Cause?”. In March 1986 I sent it for publication to Nature and was rejected on the grounds of being too speculative and too long. Subsequently I sent the paper to Medical Hypotheses. Dr Horrobin found it “extremely interesting and well thought out”, but rejected it on the grounds of the African evidence for heterosexual transmission. I sent it back together with a draft (attached) entitled “Aids in Africa and its Heterosexual Transmission”, which rebutted the referee’s criticism. The paper was accepted and at last appeared in print in March this year”.

Anyone, even a non-scientist, who reads Eleni’s 1988 *Medical Hypotheses* paper, and manuscript (part of which is reproduced in “Looking back on the oxidative stress theory of AIDS”) and which were sent to Peter, will have no problem realising we were the first dissidents to attribute AIDS in Africa to poverty and its sequelae and a unified drug theory of AIDS in the other AIDS risk groups. (John Lauritsen and Michael Callen preceded us in regard to AIDS in gay men). We have no doubt that Peter (our views of Peter have not changed during the last two decades) will be the first person to accept this. It is a salutary fact that, in his seminal 1987 paper, while Peter argued that “HIV” is not the cause of AIDS, he presented no alternative theory to explain the AID syndrome.

In a posting entitled “Can we learn from Parenzee?” on Crowe’s “Alberta Reappraising AIDS Society” website, “Copyright Tuesday, April 24, 2007: Alberta Reappraising AIDS Society and Dr Henry Bauer”, Bauer gives several reasons why we lost. Let us leave the reasons and instead consider the strategy he advocates.

“Irrespective of the above, the [Judge] Sulan decision underscores the need to identify exactly what is necessary to *establish sufficient doubt* about the HIV = AIDS dogma.

In my opinion, to accomplish this it is not necessary to establish that HIV does not exist, it should suffice if one can establish that HIV is not sexually transmitted so efficiently that it could be responsible for the epidemics of AIDS claimed to be

ravaging Africa now and those that ravaged within a few years several relatively isolated communities of fast-lane gay men in metropolitan areas of developed countries. The evidence for lack of efficient sexual transmission exists in ample amount in official data on the prevalence of HIV (more accurately, the prevalence of positive HIV-tests) over the past quarter century; for example, studies of transmission have invariably delivered probabilities on the order of only 1 per 1000”.

Doesn’t Bauer know that the “HIV” experts claim the 1 per 1000 “delivered” probability is only for heterosexuals in the developed world? That, according to the “HIV” experts, there are many reasons for it to be much higher in African heterosexuals?

Bauer continues: “As to whether any danger is associated with possibly transmitting HIV, the fact that HIV does not cause AIDS is evident from the known thousands of HIV-negative AIDS patients-especially those afflicted with Kaposi’s sarcoma-and from the known thousands of HIV-positive people who have remained healthy for as much as two decades”.

Doesn’t Bauer know that not only we (*Medical Hypotheses* 1992) but even the “HIV” experts accept that KS is not caused by “HIV”? Doesn’t he know that not everybody who is infected with microbes becomes sick?

Bauer continues: “If there is indeed the need to present an alternative theory, I point without false modesty to the conclusions reached from my collation of HIV-test data [*]:

1. A positive HIV-test is an entirely non-specific indication of a reversible stimulation of the immune system (a stimulation that remains to be fully understood, but which quite possibly reflects oxidative stress, as the Perth Group have argued);
2. The likelihood that a given stimulation of the immune system will produce an “HIV-positive” response is mediated by an individual’s age, sex, and race. [So what? A positive test for syphilis and gonorrhoea is also “mediated” by an individual’s age and race. In the developing countries (poverty stricken) from where the vast majority of positive “HIV” tests are reported, positivity does not depend on sex].

Qualifying his [*] Bauer wrote: “*Literature citations documenting the evidence referred to above, as well as fuller discussions, are in the book published 27 April: Henry Bauer, *The Origin, Persistence and Failings of HIV/AIDS Theory*, McFarland”.

And what does Bauer say in his book? (Note: Bauer uses the term “F(HIV)” to mean “the frequency of positive HIV-tests”, presumably antibody tests).

“The manner in which F(HIV) differs between various tested groups, and the circumstances of newborns [what are these circumstances?], are evidence that F(HIV) in some way reflects a general, *non-specific* challenge to health; a positive HIV-test means only that the immune system is reacting in some fashion, not that it is reacting to some specific virus, HIV, nor that the test is detecting antibodies to such a virus.

The degree to which one's immune system is likely to react in this way varies with age, sex, and racial ancestry. In other words, an HIV-test says "positive" in the presence of some level or type of physiological stress; it is analogous perhaps to an inflammation, or a fever, or the release of adrenaline in response to some environmental challenge, or the release of histamine as an allergic response".

Bauer continues: "That is rather like what has been argued for some two decades by researchers and physicians in Australia, the so-called "Perth Group" (www.theperthgroup.com; www.virusmyth.net/aids/index/epapadopoulos.htm; both accessed 18 January 2006), namely, that HIV tests detect signs of "oxidative stress." That "oxidative" processes can be bad for one's health, that they can lead to ill health, is nowadays widely accepted and known (hence the marketing of a large variety of dietary supplements described as "*anti-oxidants*"). At the same time, a certain degree of oxidative stress is not uncommon and not necessarily a serious threat".

Bauer continues: "A great variety of reported observations that present puzzles under the HIV-causes-AIDS theory are accommodated by this hypothesis".

In other words: (i) it is he, Bauer, who put the hypothesis and proved it (without any evidence) that a positive antibody test "in some way reflects a general, *non-specific* challenge to health"; (ii) The Perth Group has been arguing for some time "that HIV tests detect signs of "oxidative stress"", but this may or may not be true. Be this as it may, everybody knows that "oxidative processes" are detrimental to health, that is, according to Bauer, the Perth Group just repeats what everybody else says and any claim to the contrary is theft.

Bauer claims that unlike him, in the Parenzee hearing we did not have an "alternative theory" for a positive test (in fact the false prosecution claim, repeated by the Judge, was that we did not have an alternative theory for AIDS, not for a positive test).

Let us quote from a few sources which we know Bauer read before he published his book on 27th April, 2007 and the Alberta Reappraising AIDS Society comments, "Copyright Tuesday, April 24, 2007...", and let the reader decide if we had or did not have an alternative explanation for positive "HIV" antibody tests.

In the monograph "Mother to Child Transmission of HIV and its prevention with AZT and Nevirapine. A Critical Analysis of the Evidence", published in 2001 (thus well before Bauer became a dissident), in section 1.6.2.3 page 14 under the subtitle "Why there is a relationship between a positive test and the appearance of AIDS" one reads: "The explanation [for a positive antibody test] may not be that curious if one realises, as many do, that there are many non-specific but nonetheless useful laboratory tests employed in clinical medicine. ("[C]urious" because of an editorial written by Timothy Dondero and James Curran on the Mulder paper (*Lancet*, 1994): "Although most reasonable observers do accept that HIV causes AIDS, even sceptics cannot fail to acknowledge the high prevalence of antibody to HIV in Africa. If there are any left who will not even accept that antibody to HIV indicates infection with the virus, their explanation of how HIV seropositivity leads to early death must be curious indeed").

The explanation of how a positive antibody test may predict early deaths is far less curious than the predictions engendered by an increased erythrocyte sedimentation rate (ESR). The ESR, discovered in 1918 by Fahraeus while seeking an early test for pregnancy, is a common but non-specific test which, when elevated, “is a measure of the presence and intensity of morbid processes within the body”. Like a positive “HIV” antibody test, an elevated ESR also has the capacity to predict “a likelihood of death within the next several years far above” a normal ESR. A common cause of elevated ESR is infection and “Elevated ESRs are also seen with pregnancy, malignancy, collagen vascular diseases, rheumatic heart disease, and other chronic disease states, including human immunodeficiency virus infection”.¹⁴³ Even asymptomatic, non-anaemic HIV positive individuals may have an increased ESR¹⁴⁴ and the test may be predictive for disease progression.¹⁴⁵ In HIV positive children a correlation exists between seropositivity, hypergammaglobulinaemia and an elevated ESR.¹⁴⁶ As far back as 1988 researchers from the Institut National de Transfusion Sanguine, Paris, France, found that: “An increased ESR in HIV-seropositive subjects seems to constitute a predictive marker of progression towards AIDS before the decrease of the CD4 count”.¹⁴⁷ ...Given that the “HIV” proteins are likely to be normal cellular proteins, cellular proteins with new antigenic epitopes or newly induced cellular proteins, and that individuals who test positive have high levels of auto-antibodies and/or antibodies to many “non-HIV” antigens all or some of which may cross-react with cellular proteins, “HIV” seropositivity, curiously or not, like the ESR, may represent nothing more than a non-specific indicator, serendipitously discovered in 1983/84, of altered homeostasis connoting a propensity to develop particular diseases”.

Responding to Brian Foley in the BMJ debate June 20th 2003 we wrote: “Not surprisingly, patients are about as interested in the arcane and ludicrous distinctions between being “officially” or “unofficially” infected as they are in being “partially pregnant”. They just want to know if they’re infected. Is the patient one of the lucky ones whose “elevated levels of some antibody or antibodies that bind to HIV proteins” are not HIV antibodies? Given that AIDS patients are characterised by hypergammaglobulinaemia, that is, they have “elevated levels of some antibody or antibodies”, because of antibody cross-reactivities it is more than likely that many and perhaps 100% of the antibodies which “bind to HIV proteins” are not HIV antibodies at all.²⁻⁴ Note this would not negate a correlation between a positive test and AIDS. That is a completely different matter and we trust Brian is cognisant of the distinction. Correlations between diseases and non-specific tests are well recognised and are of great clinical utility. Both in diagnosis, prognosis and predicting the success or failure of treatments. For example, the humble temperature measurement. Or, more attuned with antibody tests, measurements of the erythrocyte sedimentation rate (ESR).⁵”.

As far as the Perth Group not having an alternative theory for a positive test in the Parenzee case is concerned, please read Val’s evidence in chief on our website and then Bauer’s book and tell us what new and significant evidence one can find in his book.

The most unexpected accusation of stealing by the Perth Group came from Eugene Semon in his House of Numbers posting “Why I’m not a Perthian”. While Bauer stated that we have not said anything new about “oxidation”, that is, any claim to the

contrary amounts to stealing, and Crowe similarly claimed that the oxidative “idea” was Montagnier’s, Eugene also implies we stole it:

“2. I do not accept that PG has claims of “priority” with the “oxidative stress” idea. It strikes me as a form of America bashing, rewriting history, etc. Is it a matter of PG followers ‘taking it too far’, for “mitotic theory” paper, and the 1988 Med Hyp paper (which of course are great contributions to progress)?

It might be why American scientists and lawyers, who know better about history of “redox and excess pro-oxidant” breakthroughs by “anti-aging” researchers of the 70’s, will be “primed” to dismiss PG. If full-blown AIDS is anything, it’s accelerated aging and premature activation of the “death program”, where the “silent infections” take over”.

Eleni’s redox theory of cellular function and structure in general and pathology in particular (cancer, cardiovascular diseases among others) was first presented at a meeting in Colorado in 1979. A concise version dealing specifically with cancer was published in *Speculation in Science and Technology* in 1980. The more detailed version was published in *the Journal of Theoretical Biology* in 1982, under a somewhat insubstantial title, “A Mitotic Theory” (a far more fitting title would have been “The redox and its oscillations theory of cellular structure and function”. Experimental proof on cardiovascular function was published in the 1980s).

Eugene, please give us a single paper published before 1982, if not before 1979, with a redox theory of cellular function and structure and cancer in particular even remotely resembling Eleni’s (since then there have been a few which curiously are extremely similar).

The paper with the redox theory of AIDS was first submitted to *Nature* in 1986, and was published in *Medical Hypotheses* in 1988. Please give us one single paper published before 1988, if not before 1986, with a similar redox theory of AIDS. (We urge you to find even one paper among the thousands published to date on “oxidative stress” and AIDS which remotely resembles our redox AIDS theory).

The free radical theory of aging was put forward in 1956 by Denham Harman from the Donner Laboratory of Biophysics and Medical Physics. In the decades which followed, many researchers became interested in this theory. Perhaps in America the best known is Richard Passwater. As Passwater pointed out, by the beginning of the 1970s there were laboratories around the world which, based on this theory, worked “to slow, stop, or reversed the human aging process”. In the American Laboratory, May 21-26, 1971, Passwater wrote: “A summary of the aging process was given in the preceding issue of American Laboratory. In essence, we pointed out that aging was a condition resulting from diminished body reserve caused by the loss of cells. The loss of reserves diminishes the body’s ability to combat stress; loss of cells results from **free-radical attack**, radiation-related events, and poor nutrition” (emphasis ours).

Our redox theory of cellular function and structure has very little in common with the free-radical theory of aging. Only someone who is ignorant of one or both would

consider them the same. In our theory the free-radicals are the result and not the cause of cellular oxidation.

Eugene please read:

- (i) “A mitotic theory”, Cancer and epigenetic reversion--the fundamental role of redox. *Am J Pathol* 2007;171:1726-7 and also “The Depletion of Nuclear Glutathion Impairs Cell Proliferation in 3+3 Fibroblasts”, PLoS, 2009; 4: 1-14 and ref. 6, 7, 8, 9, 11, 50, 51 and 52 within and tell us, if there is a misappropriation who is misappropriating from whom;
- (ii) Barry Page’s correspondence with Professor Prabhat Goswami (see Appendix 1 annexed) and tell us why, in your view, his second email remains unanswered;
- (iii) Reappraisal of AIDS: Is the oxidation caused by the risk factors the primary cause?”, 1988, then do a Medline search on “oxidation and AIDS” or “oxidation and HIV” and tell us who is misappropriating from who.

Apparently Eugene agreed with de Harven that we stole the “non-existence idea” from Stefan. When others pointed out this is wrong, he replied: “Our disagreement over my interpretation/extension of Lanka’s review in the paper “HIV, Reality or Artifact” considering Gallo et al’s isolation of 70S RNA via sedimentation velocity centrifugation.

Excerpts from Lanka on the origin of HIV nucleic acids (<http://www.virusmyth.com/aids/hiv/slartefact.htm>)”.

In fact, to obtain the “HIV” RNA Gallo did not use sedimentation velocity/centrifugation but banding in density gradients. The poly(A)-RNA which banded at the density of 1.16g/ml was defined as “HIV” RNA.

One of Eugene’s quotes from Stefan was the following: “Choosing a desired probe. Since no DNA from HIV existed to hybridise with the prepared DNA, Gallo and Montagnier simply used stretches of DNA from what they said was specific to HTLV-I, a retrovirus Gallo had earlier claimed to have discovered, and which deemed suitable for this purpose. The DNA detected in this was replicated and certain stretches of it cloned and declared to be the DNA of HTLV-III (later to be called HIV)”.

Commenting, Eugene wrote: “My point is that HTLV-I, according to a reasonable interpretation of it’s cDNA (Reitz et al; PNAS, March 1981, V78: 1887-1891), is not a “complete rag-bag”.

Firstly, let us repeat, neither Gallo nor Montagnier ever used DNA from what they said was specific to “HTLV-I” as probes for the detection of the “HIV” DNA. Apparently Stefan misunderstood what we said in “The isolation of HIV: has it really been achieved?”.

Secondly, Reitz et al did not have proof that the HTLV-I poly(A) RNA sedimented at 70S, but “about 70S”. Let us assume their poly(A) RNA sedimented at exactly 70S. So what? The only way they could claim that the RNA was retroviral was to prove that it originated from a mass of purified retroviral particles. They had no such proof. They wrote: “HTLV was purified from clarified media of HUT102 cells by

centrifugation...and then further purified by equilibrium density gradient centrifugation (see Results)". Under Results one reads:

"RESULTS

Characteristics of HTLV [³H] cDNA Probe. Concentrated, purified HTLV from the model K ultracentrifuge was rebanded to equilibrium in sucrose density gradients (22-55% sucrose in TNE) in an SW41 rotor (4°C, 16hr, 22,000 rpm). The gradient was assayed for DNA polymerase activity with oligo(dT)₁₂₋₁₈poly(A) and oligo(dT)₁₂₋₁₈poly(dA) as primer-templates. A peak of DNA polymerase activity was present at 1.16g/cm² with marked preference for oligo(dT)₁₂₋₁₈poly(A) over oligo(dT)₁₂₋₁₈poly(dA), a characteristic of reverse transcriptase (2). Most of the activity with oligo(dT)₁₂₋₁₈poly(dA), likely representing residual cellular DNA polymerase activity, bands at higher density. The peak fractions of reverse transcriptase activity were pooled, concentrated by velocity gradient centrifugation (100,000 x g, 1 hr), and used for the synthesis of [³H]cDNA as described above".

In other words the only proof they had that the 1.16 g/ml band was purified virus, was detection of reverse transcriptase activity.

DE HARVEN'S "ORIGINAL" ANALYSIS OF MONTAGNIER'S 1983 PAPER AND OUR IMAGINARY MEETING WITH PETER DUESBERG

De Harven's extensive correspondence with Anita Allen includes the following: "Back to Eleni! You are right: she frequently said and wrote that whatever the Pasteur group had in 1983 could not be a retrovirus! But she is wrong on that! Fig. 2 in this 1983 paper shows TYPICAL retroviruses budding on the surface of a lymphocyte. There is absolutely no doubt about that. Anybody with an "EM eye" will agree with me on that. These particles ARE RETROVIRUSES!...The interpretation I gave you about the famous Pasteur 1983 paper is, I believe, very specific, and I have never heard anybody suggesting exactly the same. I know that Eleni was never convinced by that paper, but I am sure the reasons for her rejection of that paper were similar to what I explained to you two days ago...Again, my key point is: (1) the human placenta is loaded with HERVs, (2) lymphocytes from the umbilical cord blood are therefore very likely to carry the same HERVs, (3) such lymphocytes were added to the mixed cell cultures, at Pasteur in 1983, (4) the EM picture in the 1983 paper simply demonstrate that, under PHA and TCGF stimulation, these placental lymphocytes express, by "budding" their HERVs, (5) this observation has nothing to do with the inoculum from an AIDS patient and is no proof of the exogenous infection of these lymphocytes by hypothetical retroviruses originating from the AIDS patient. If you can show me that Eleni presented an identical analysis, feel sure I shall be very glad to write to her immediately!!".

Anita gave him the evidence, but he never wrote either to Anita or Eleni.

To the contrary, at the December 2003 meeting at the European Parliament, de Harven presented his "original analysis" of the Montagnier paper and concluded that Montagnier did not prove the existence of "HIV" because he had no proof of purification, but he had proof for the existence of an endogenous retrovirus, despite the lack of purification. At the end of his talk he said: "Indeed, doubts concerning the very existence of HIV are nothing new, and were expressed by several dissident scientists several years ago (30,31) I completely share these doubts. Let us not forget

the title of Peter Duesberg's book (33) published in 1996: "Inventing the AIDS Virus".

Ref. 31 is Stefan's "HIV, Reality or Artefact?" and Ref. 30 is an article written by us. Of everything we wrote up till the end of 2003, de Harven could find only one article in which we expressed doubt regarding the existence of "HIV": "Papadopulos-Eleopulos E; "A brief history of retroviruses", Continuum, 1997; 5:25-29".

As the title suggests, we describe in this article, at pages 25-28 inclusive, the history of retroviruses and the methods used to purify them, including in some detail banding in density gradients. "HIV" is not even mentioned. On page 28 and 29 which de Harven includes, there is a piece entitled "Summary of Montagnier and Colleagues 1983 Science Paper". As the title suggests, this was not meant to contain our doubts about "HIV" or an analysis of that paper, but only a description of Montagnier's experimental evidence, to make it easier for readers to understand Montagnier's interview with Djamel Tahi, page 30-34, and our detailed critical analysis of the paper, and the interview, page 35-44, and "HIV" isolation in general which de Harven conveniently forgot. As we all know, Peter never had any doubts and the title of his book does not refer to questioning the existence of "HIV".

In 2008 something (we still cannot make out what) made de Harven and Andrew Maniotis think we were going to meet with Peter. They wrote to us and asked to convey to Peter their view that "HIV" is not an exogenous retrovirus, and that their interpretation of "HIV" meets all the observed data, namely that "HIV" is an endogenous retrovirus/endogenous retroviral sequences/retroid.

De Harven wrote: "I wish I would have a chance to be with you in Washington DC, when you will talk to Peter Duesberg! Yes, I do have ONE question, a critical question for Peter: I think that a definite answer to that question by Peter, could clarify a difficult ambiguity RA has been struggling for years...".

In our response on 18 July we wrote: "Unfortunately, although we would very much like to meet with Peter and have indeed requested a meeting to discuss our differences, no such meeting has eventuated or is presently planned.

In your emails you asked us to convey to Peter that HIV is an endogenous retrovirus/endogenous retroviral sequences/retroid. Do neither of you recall the lengthy paper we wrote in Continuum in 1996, before you became dissidents, in response to Peter's claim that HIV had been isolated? In this paper we presented all possible interpretations of the so called "HIV". DNA/RNA, including its being that of an exogenous retrovirus, and everything you suggest. This is discussed in detail under the subtitle 6.3. SPECULATIONS ON "HIV DNA". You will appreciate that in 1996 the evidence for any of these possibilities was not as definitive as it is at present. <http://www.the.perthgroup.com/CONTINUUM/pgvsvdusbergreward.html>

Andrew

In your email you refer to an "HIV" molecular signature. A "signature" as you know, is specific but we do not see anything specific about the so called "HIV" molecular

signature. In fact it's not just its molecular signature that is not specific but everything else about it, particles, proteins, RT and antibodies.

At present there is no evidence that proves the existence of endogenous retroviruses. This is at least one point of agreement between the Perth Group and Gallo. During the Parenzee trial, Gallo said a number of times, by definition, a particle can be considered to be a virus if, and only if, evidence exists that it is transmissible. Responding to a question put to him by Kevin Borick he stated: "...endogenous retroviruses aren't viruses as your first witness [E.P-E] properly said, they are particles, they have never been transmitted. A virus is something that infects, that you prove goes from person. A to B. Short of that they are particles. Where a virus at least has to be transmitted in vitro in the laboratory, it goes from one cell to another, it's never been demonstrated for endogenous retrovirus". (T1298)...".

De Harven did not respond. Maniotis did. The subject was discussed in a few email exchanges and included three references which he claimed proved the existence of HERVs. They were:

Marie Dewannieux, Francis Harper, Aurelien Richaud, Claire Letzelter, David Ribet, Gerard Pierron, and Thierry Heidmann. Identification of an infectious progenitor for the multiple-copy HERV-K human endogenous retroelements. *Genome Res.* Oct. 31, 2006 (The "Phoenix Virus").

Bannert N, Kurth R. *Proc Natl Acad Sci USA* 2004 Oct 5; 101 Retroelements and the human genome: new perspectives on an old relation. *Suppl 2*: 14572-9. Epub 2004 Aug 13.

McClure MA, Richardson HS, Clinton RA, Hepp CM, Crowther BA, Donaldson EF. *Genomics*. Automated characterisation of potentially active retroid agents in the human genome. *Apr* 85(4):512-23, 2005.

Below we reproduce the relevant posts of Maniotis's lengthy email of 7 August, in which he quotes from our last email on the subject and then responds (in capitals).

"You agree with us but then you contradict yourself by saying that the molecular signature is an HERV. In your July 25th email you said "A likely explanation of the origin of "HIV's" molecular signature comes not from racist notions of primate-human transmission or from Special Virus Program conspiracy theory, but from recent studies in genomic research that suggests that the so-called template for the protein molecular signatures of "HIV" derives from our own endogenous DNA sequences known collectively as HERVs (coming from cellular origin instead of viral origin-Human Endogenous Retroviruses). "HIV's" molecular signature represents a HERV (Human Endogenous Retrovirus) nucleic acid sequence, or, what is called a 'retroid' of one kind or another". However, in not one of the references you cited is their evidence that proves the existence of human endogenous retroviruses.

NO SELF-CONTRADICTION AS I SEE IT. IF A HERV IS A PIECE OF CELLULAR DNA OR RNA, THEN WHY DO YOU NEED TO THINK OF THEM AS VIRUSES AT ALL? YOU MIGHT AS WELL CALL THEM HUMAN ENDOGENOUS RETROELEMENTS (AS THE PHOENIX PAPER DID I

BELIEVE) AND IT MEANS THE SAME THING. BECAUSE ALL VIRUSES COME FROM CELLS (REAL VIRUSES AND IMAGINED VIRUSES) [For the “real” viruses to come out of the cells, they first must go inside the cells] IT IS SEMANTICS TO SPLIT HAIRS HERE BUT YOU ARE CORRECT, THE LANGUAGE SHOULD BE CHANGED TO SAY HERE’S (HUMAN ENDOGENOUS RETROELEMENTS) INSTEAD OF “VIRUSES.”.....

I’M PERFECTLY CONTENT CALLING THEM HERE’S (PRONOUNCED HARE’S-LIKE RABBITS) AND TO DO AWAY WITH HERV’S ALTOGETHER. IT’S JUST AN EASIER REACH FOR THE FOLEYS OF THIS WORLD, PERHAPS, TO ACCEPT THAT THEY WERE SNOOKERED BY HERV’S INSTEAD OF HERE’S.

In one of our previous emails we pointed out that in the Parenzee court case Gallo stated: “A virus is something that infects, that you prove goes from person A to B. Short of that they are particles. Where a virus at least has to be transmitted in vitro in the laboratory, it goes from one cell to another. It’s never been demonstrated for endogenous retrovirus...endogenous retroviruses aren’t viruses as your first witness properly said, they are particles, they have never been transmitted”. In one of our previous emails we asked if you agreed with Gallo: (a) there is no proof for the existence of HERVs (b) his definition of viruses. You did not respond to (b) but you disagreed with (a). This can only mean you disagree with both Gallo and us since “your first witness” (to whom Gallo was referring in the Parenzee case) is EPE.

As evidence for the existence of HERVs you gave a reference by Marie Dewannieux et al (Identification of an infectious progenitor for the multiple-copy HERV-K human endogenous retroelements) (which says that the Phoenix virus proves there are endogenous retroviruses). Unless you disagree with the definition of viruses as given by Gallo and with which we agree the evidence in this paper does not prove the existence of a virus.

I THINK WHAT I HAVE SAID ABOVE CLARIFIES THESE OBJECTIONS. AS A MATTER OF WHIMSICAL FACT, I DON’T REALLY KNOW WHAT GALLO THINKS A VIRUS IS.

In McClure et al (Automated characterisation of potentially active retroid agents in the human genome) the authors did not set out to produce evidence for the existence of HERVs. Instead “The Genome Parsing Suite (GPS), a generic multistep automated process, was developed to characterise all RT-like sequences in the human genome database”.

YES!!! BUT DID YOU LOOK AT SOME OF THE SEQUENCES THEY CAME UP WITH IN THEIR ANALYSIS? 60 COPIES OF HBV? 11 SEQUENCES OF “HIV” [not quite right]...

In Bannert et al (Retroelements and the human genome: New perspectives on an old relation) the authors state “Meanwhile, the formation of infectious HERV particles and their potential for transmission remain controversial open question”. Further on “A clear proof for the existence of a HERV capable of productive replication remains

elusive...” In other words, at present there is no proof for the existence of human endogenous retro-VIRUSES”.

On 11 August Maniotis sent another email, copied to many dissidents, in which he wrote: “In deference to our ongoing discussion, I am editing all past writings to eliminate HERV’s when I have used the term (or explain it in the way Val and Eleni first explained them) and replace HERV’s with the modern term being used by the genomics people who find “HIVS” molecular signature in normal (uninfected) DNA (non-specific markers), which they call “retroid,” “retroids,” or the older term, retroelements”.

This means that ultimately Maniotis agreed with us that at present there is no evidence in the scientific literature, including the three references he gave us, which proves the existence of HERVs. Particles, even if they have all the morphological characteristics of a retrovirus, cannot be said to be HERVs for the single reason that today nobody has proven the existence of such entities.

We never interpreted HERVs (human endogenous retroviruses) or as HEREs (Human endogenous retroelements) or as human endogenous retrovirus sequences (HERSs). HERVs cannot be considered the same thing as HEREs or HERSs.

The main property of a retrovirus is that it is a particle. HEREs and HERVs are not particles, they are nucleic acid sequences present in everybody’s DNA.

To claim that the “HIV” molecules signature is a retroid, evidence must exist which proves that:

- (a) The “HIV” genome is identical with a HERE (retroid);
- (b) The “HIV” proteins are coded by the HERE, HERS, retroid, or whatever you wish to call them.

No such evidence exists. To the contrary. For many years we have been presenting evidence that “HIV” proteins are coded by cellular genes, including actin (p41, 120, 160) and HLA DR (p32). Yet, for some unknown reason(s) some dissidents make unsubstantiated claims and in the process muddle the dissident science.

De Harven did not comment although all the correspondence was copied to him.

THE VIENNA MEETING

At the beginning of this year several European dissidents started talking about a dissident meeting to be held somewhere in Europe, to coincide with the International AIDS Conference. In Martin Barnes’s and Georg von Wintzingerode’s view, for the meeting to be successful the “Existential Question” must be resolved beforehand. They said to put Duesberg and Eleni into a room and let them out only after they had resolved this important scientific question. Not only did this not happen, Eleni was not even invited to the conference.

Needless to say, in his response Crowe considered the idea of a conference “wonderful”, especially if Montagnier was also invited. But, “I don’t quite understand your position. You want the Perth Group and Duesberg to come to a

“unified position”. That’s consensus. But you’re opposed to science by consensus. When you say, “the current situation is intolerable, what do you think the current situation is and why do you think it’s intolerable?””.

Incredibly, the leader of a scientific group does not know, or does not want those whom he leads to know that, unlike politicians, scientists do not come to an agreement by consensus. They debate and the evidence which fits the facts prevails. Without such a process chaos will prevail and there will be no science. The fact is: to claim proof for the existence of “HIV” evidence must exist for its isolation/purification. Nowadays any information is at our fingertips and yet, even after 25 years, we still are divided. This can only mean something is not quite right in the dissident movement.

Bauer sent two emails. In one of them he wrote: “I share David Crowe’s views on this...what we ALL agree on, so far as media and public are concerned, is the single most critical issue: Whatever HIV tests detect, whatever HIV may or may not be, it isn’t the cause of AIDS...We don’t need to add further opportunities for media and HIV/AIDS vigilantes to throw up red herrings and confusions and distractions from the really ONLY important issue, which is the dogma that is inflicting wholesale physical and psychological damage. Media and mainstream would listen to us no better if we were to all agree that HIV is a harmless passenger virus, or if we were all to agree that HIV doesn’t exist. It’s an ACADEMIC argument and issue, in both meanings of “academic””.

Since Bauer is a latecomer to the dissident movement, let us remind him that “the dogma” is based on science. The only way to disprove a scientific dogma is through better science. What is Bauer going to give to the “media and mainstream” to disprove the dogma if not science?

If science, and if we all agree the critical issue is that “HIV” “isn’t the cause of AIDS”, which of his science pronouncements is Bauer going to use to prove this fact to the “media and mainstream”?:

(i) A positive antibody test proves HIV infection. And the antibodies are so good they neutralise the virus making HIV a harmless passenger virus;

or

(ii) “HIV Tests Are Not HIV Tests”;

or

(iii) The tests detect HIV antibodies, the problem is “the occurrence of false-positive HIV tests”.

Bauer claims:

(i) The existence question is only “academic”. (He either did not read, did not understand, or does not want to understand, our response to Fabio Franchi, or

- “The Final Act”, or the BMJ debate, in which it is explained in “LAY TERMS” why the “existence” question is not academic);
- (ii) Antibodies are the key in the dissident effort against the dogma;
 - (iii) “A great variety of reported observations that present puzzles under the HIV-causes-AIDS theory are accommodated by this hypothesis”, that is “his” hypothesis regarding the “HIV” antibody tests.

Let us quote from Bauer’s latest peer-reviewed publication and see how his hypothesis regarding the antibody test helps in disproving the “HIV-causes-AIDS” hypothesis:

“The unreliability of HIV/AIDS models is only one reason for questioning estimates of the prevalence of active HIV infection; another is the occurrence of false-positive HIV tests”.

What proof does he have for the existence of false-positive “HIV” tests?

- (a) “One possible reason for false positives is that few if any testing facilities, particularly perhaps in Africa, are able to engage in the elaborate interplay between clinical observation, medical history of patients, and laboratory work-up that are called for if HIV tests are to be used to diagnose actual infection (Weiss & Cowan, 2004)”.

So if you are gay and test positive you are infected, if you are heterosexual and test positive, you are not, the test is false positive. If you are sick you are infected, if you are a healthy blood donor you are not infected, the test is false positive. Does Bauer not know, after all we have written, that this was the antibody “science” which Weiss, Gallo and their colleagues used in 1985 to introduce the “HIV” antibody tests? Indeed, if it wasn’t for this antibody “science” there would never have been “HIV” antibody tests. Can’t he see that such tests may tell you something about:

- the state of stimulation of one’s immune system;
- being gay or heterosexual;

but will tell you nothing regarding infection with a unique retrovirus “HIV”?

Bauer’s argument against the “HIV” antibody test, especially lately, is based on Weiss and Cowan’s views. It appears he has never come across:

1. Papadopoulos-Eleopoulos E. Reappraisal of AIDS: Is the oxidation caused by the risk factors the primary cause? *Med Hypotheses* 1988;25:151-162. <http://www.theperthgroup.com/SCIPAPERS/reappraisalofaids.html>
2. Papadopoulos-Eleopoulos E, Turner VF, Papadimitriou JM, Bialy H. AIDS in Africa: Distinguishing fact and fiction. *World J Microbiol Biotechnol* 1995;11:135-143. <http://www.theperthgroup.com/SCIPAPERS/africafactandfiction.html>
3. Papadopoulos-Eleopoulos E, Turner VF, Papadimitriou JM, Causer D, Page BA. HIV antibody tests and viral load--more unanswered questions and a further plea for

clarification. *Curr Med Res Opinion* 1998;14:185-186.
<http://www.theperthgroup.com/SCIPAPERS/furtherplea.html>

4. Papadopoulos-Eleopoulos E, Turner VF, Papadimitriou JM, Stewart G, Causer D. HIV antibodies: further questions and a plea for clarification. *Curr Med Res Opinion* 1997;13:627-634. <http://www.theperthgroup.com/SCIPAPERS/epcurmedres97.html>

5. Papadopoulos-Eleopoulos E, Turner VF, Papadimitriou JM. Is a positive Western blot proof of HIV infection? *Biotechnology* 1993;11:696-707. <http://www.theperthgroup.com/SCIPAPERS/biotek8.html>

6. Turner VF. Emergency physicians roles in managing HIV seroconversion illness: Take stock or take HAART? *Emergency Medicine Australia* 1999;16:201-203. <http://www.theperthgroup.com/SCIPAPERS/EMAHIVLetterandReply.pdf>

7. Turner VF. Detection of acute HIV infections. *N Engl J Med* 2005;353:631-633; author reply 631-3. <http://www.theperthgroup.com/SCIPAPERS/VFTNEJM.pdf>

He just visits our homepage and when he sees: “Due to irreconcilable scientific and ethical differences we disassociate ourselves from the Rethinking AIDS Group” he is very surprised. Since Bauer considers Weiss’s work the bible for “HIV” antibody testing, what is he doing among the dissidents?

(b) “Second: Antibody tests can only deliver information about the presence of antibodies, not about active infection. If one defines a genuine positive as indicative of active infection – which is the appropriate definition if one is interested in possible dangers of accidental infection during dissection – then one must take into account that seroconversion alone, the presence of antibodies does not necessarily indicate active infection. That presumably is why the tests have been approved only for screening purposes and why the diagnosis of actual infection calls for the painstakingly elaborate procedures outlined by Weiss & Cowan (2004)”.

Let’s ignore the fact that Bauer introduces his own definition of infection and see what Weiss has to say in the very reference Bauer is citing: “It is important to remember that many viruses, including HIV, are characterised by persistence; i.e. the presence of antibodies does not indicate resolution of infection. Exogenous retroviruses, such as HTLV and HIV, can integrate within the genome (DNA) of the host cell...In this vein, prospective epidemiologic serologic studies indicate that once an adult produces antibodies to HIV (“seroconversion” to a “seropositive” status), complete loss of antibodies (“seroreversion”) is rare (20); virus can usually be recovered from seropositive persons. The presence of anti-HIV antibodies is therefore generally interpreted as evidence of persistent infection, not resolution of past infection” (page 148).

“The EIA (ELISA) was the first type of test to be licensed in the U.S. to detect infection with HIV...In the U.S., the latest generation of licensed EIA screening tests typically has sensitivities of $\geq$ and specificities of $\geq$ 99.9% when using serum, plasma, or dried blood spot specimens” (page 51).

Can dissidents overcome “the dogma” by inventing a personal definition of infection and by misrepresenting the “HIV” experts?

- (c) “The manner in which HIV tests are calibrated, and the possibility of cross-reactions correlated with HLA type (Weiss and Cowan, 2004, p.151), suggest why HIV tests might be racially biased: HLA type is racially correlated (see for example, Creemers and Khan, 1998). Blood donors are typically used as low-risk “true negative” controls, yet among blood donors, African Americans tested positive 14 times more often than white Americans (Petersen and Doll, 1991; Ward, 1988). In South Africa, among regular (repeat) blood donors, blacks tested positive 23 times more often than whites or Indians (Manto, 2004). Therefore, if tests were to be calibrated separately in different racial categories with blood donors as controls, apparent HIV prevalence among Africans and people of recent African ancestry would be much lower than present estimates”.

Let us see what Weiss and Cowan say on page 151: “The first generation HIV EIA kits used purified disrupted whole virus, which included steps that attempted to remove the cellular contaminants during the manufacturing process. However, simply removing cellular debris from HIV preparations produced in human cell lines, such as H9/HTLV-IIIB, did not remove residual reactivity to high-titer HLA-antibody sera due to the physical association of HIV virions with HLA class II molecules...Methods to reduce HLA reactivity were by necessity subsequently developed and the second generation of HIV EIAs had improved specificity. Another opportunity for problems with specificity came about with the advent of viral components produced through recombinant methods in bacteria such as *Escherichia coli*”.

So, the introduction of the second generation ELISA and especially by the use of a few recombinant “HIV” antigens in the third generation ELISA resolved the problems raised by Bauer.

Let’s assume that Bauer is correct: if the ELISA used to test Africans is calibrated using African “blood donors” as controls, the specificity of the test will be improved, then according to Bauer’s data and logic South Africa will have about 3–3.5 million infected with “HIV” instead of 4–5 million.

Since in America infection is based on the WB, then Bauer has no choice but to accept that “HIV” infection in African Americans is much higher than among whites, that is, there is no racial discrimination in “HIV” testing in the USA. (Don’t forget that, according to protagonist South African scientist participating in the Presidential AIDS Advisory Panel meeting, the Western blot is used in South Africa).

In his other email regarding the conference Bauer wrote: “There is no reason to assume or to believe that the existential question is ripe to be settled. Disagreements over interpretation of evidence can continue and have persisted in science and in medicine, sometimes for decades. [Are two decades not sufficient to determine if a virus does or does not exist?] Working scientists use their own interpretations as guides to their own research. Duesberg continues to offer explanations based on his view, and the Perth Group offer explanations based on their view, and neither has proved compelling enough to be universally adopted.

As a non-virologist, non-molecular-biologist observer, I – like, I suppose, most Rethinkers – would love to get a full explanation in LAY TERMS of how the HIV genome was elucidated; what bits of RNA or DNA are amplified for PCR tests; how those bits were chosen; how the retroviral genes were connected to the proteins that are supposedly generated by those genes...But none of my questions will be answered by a private discussion between Duesberg and Perth. Nor do I see any reason why such a discussion should end in anything other than an agreement to disagree. If the manifest evidence were clear and compelling enough, both parties would already and anyway have reached the same conclusion”.

Firstly, if “Duesberg and Perth” do not discuss and come to an agreement regarding “how the HIV genome was elucidated, what bits of RNA or DNA are amplified [does he mean primers used?] for PCR tests; how these bits were chosen; how the retroviral genes were connected to the proteins that are supposedly generated by these genes”, what questions are they going to discuss and give answers to?

Secondly, if Bauer does not have answers to the above questions, then on what does he base his claim that he and de Harven have proven that “HIV” has not been isolated?

Thirdly, the answer to his questions, in “LAY TERMS”, can be found in many of our writings, including in great detail in “The isolation of HIV: has it really been achieved? The case against” (*Continuum*, 1996; 4:1s-24s) and the BMJ on-line debate. If he does not read our papers, then on what scientific basis did he reach his authoritative conclusion that we did not offer compelling explanations and answers to all his questions? (After all, even Brian Foley agrees with us how the “HIV” genome was elucidated). Is this ignorance or a deliberate attempt to cause confusion? For what purpose? Will the existence question be “ripe to be settled” only when Bauer is ready? How long will the dissidents have to wait for Bauer to think he understands and why? How does one know that he will do a better job with the genome than he did with the antibody tests? After all, so much has been written both in the scientific and popular press long before he became a dissident and in the most lay terms possible.

Responding to Bauer, Eugene Semon wrote: “Dear God Henry, why do you act like the wheel has to be reinvented? Have you ever heard of Lanka’s “HIV Reality or Artifact”? It’s been at virusmyth site for years. It does exactly that, explain in clear language to boot how the HIV genes were “elucidated” and omigod, the guy’s a virologist! The origin of the first DNA template by which the various clones were put together is simplicity itself, as I’ve been trying to get through for years. It’s the HTLV 70S isolated by Gallo from HUT 102 in 1981”.

Responding to Eugene we wrote: “We are sorry to disagree with you. The “HIV” genome is not the “HTLV 70S RNA isolated by Gallo from HUT 102 in 1981”. It is simply pieces of poly(A) RNA that happen to band at 1.16 g/ml. As Brian Foley ultimately agreed with us in the BMJ online debates. Please see also <http://thepertthgroup.com/REJECTED/GENOME1f.doc>”.

Among the many things Maniotis said in his email regarding the Vienna meeting were the following:

“After all the fuss, screaming, bitching, criticism, what if I told you that from a perfectly Occam’s razor point of view, that both Perth and Duesbergian points of view are not in any conflict at all nor have they ever been? [One wonders why in all this time neither Peter nor we did not realise that there are no scientific differences between us.] ...A “passenger virus” to a retrovirological audience can mean a sequence that is 60 million years old or even as Varmus put it, a billion years old. This sequence would be a retroid...or HERV. In this context, exogenous viruses aren’t needed, which is why perhaps, Peter entitled his famous book, “Inventing the AIDS virus” instead of “Attributing human diseases to passenger sequences, “or something like that.”

Let us repeat:

- (i) Maniotis told us and many other dissidents that he agrees: no HERVs exist. In fact on the Gary Null show he implied that he and the Perth Group have shown this to be the case;
- (ii) A “passenger virus” is a virus by definition, retroids are not;
- (iii) A “passenger virus”, by definition, is an exogenous retrovirus;
- (iv) It is incredible, and disrespectful, for Maniotis and de Harven to tell Peter what the title of his book means. In his book Peter never said that HIV is a HERV or a retroid. He insisted, and still does, that “HIV” is an exogenous retrovirus and the “HIV” experts “invented” its AIDS causing properties.

Maniotis concludes: “Perth is correct as well of course when they say no viral entity has even been isolated without contaminating debris (proteins, nucleic acids, lipids) in cells, in conclusion, there really is little discrepancy between Perth and Duesberg’s points of view – only a huge PR problem covering the issues involved”.

We have never said “no viral entity has ever been isolated without contaminating debris (proteins, nucleic acids, lipids)”.

It is a pity that Maniotis joins de Harven in his effort to distort what we are saying and in the process talks nonsensically. Isolation, by definition means to obtain something separate from everything else. This in turn means that we have either said that “HIV” has not been isolated, or it has been isolated.

Addressing Claus Jensen, Maniotis wrote: “And Claus, please stop criticising my efforts. I like to give people a complete info. It is what I do. You do your thing, OK? Go teach folks how to kill or something. It all depends on who your audience is, which should determine, what point of view is best advanced at a particular time. Parenzee was CONFUSING I say, for multi-factorial reasons, as Liam likes to point out about AIDS itself”.

It is true that for some reason, known only to him, Maniotis gives a glut of information. The problem is that when it comes to the topic at hand, he only makes unsubstantiated claims, and no matter how many times one proves him wrong he continues to repeat them (depending on who his audience is).

It is true that “Parenzee was CONFUSING”, and Maniotis told us who caused the confusion. It was he and the likes of, above all, “dear Crowe” who when they saw

how well we were doing became fearful the Perth Group would get rid of “HIV” by themselves and intervened.

De Harven wrote to Martin Barnes:

“Hi Martin, In a way, I agree with you that the “current situation is intolerable”, but probably not for the same reasons!!

I am referring to all the recent mails on “The Existential question”...

That question is still presented as a conflict between Perth/Duesberg, i.e. HIV does not exist/HIV is a harmless passenger.

It is as if the Oakland conference had never taken place!

I cannot imagine that you forgot that...somebody presented a paper in Oakland on “Questioning the existence of HIV”?

My alternative analysis, as presented in Oakland, is consistent with all the published scientific data.

Have you heard any scientific rebuttal of what I said there?

If not, why is it strictly ignored in all the current mailings on reactivating a debate only between Perth and Duesberg?

That’s where I share your words “the current situation is intolerable”....

Most surprisingly, our friends “Rethinkers” continue to ignore the role of HERVs...The scientific literature on HERVs is enormous.

Still, I bet not a single one of our “rethinking” friends has read any of the key HERV papers?

Neither Perth nor Duesberg “positions” make any scientific sense.

For us (RA) to insist on an agreement/consensus between two positions that are scientifically highly questionable would be embarrassing, and possibly damaging for RA’s credibility!

Moreover, the “existential question” is NOT a point of limited “academic” importance, as said by Henry”.

The reason de Harven did not have “any scientific rebuttal of what [he] said there” in Oakland is because his evidence was refuted long before he gave his talk. See, for example: (i) the above mentioned correspondence between him and Anita Allen regarding his analysis of the Montagnier paper; (ii) the above mentioned email exchange regarding his and Maniotis’ request to transmit to Peter their discovery that “HIV” is a HERV; (iii) the email sent to many dissident including de Harven “Rethinking AIDS and the Perth Group irreconcilable differences”.

DE HARVEN’S SCIENCE

In his talk at the Rethinking AIDS meeting in Oakland, de Harven stated: “AIDS Rethinkers [we are not members of the RA group and we do not rethink but reappraise] have different scientific arguments on the issue of the very existence of HIV. Two radically distinct positions;

- HIV “exists” but is a “harmless passenger virus”
- HIV does not exist...

Neither of these two positions is fully compatible with the available scientific evidence. An alternative analysis is therefore suggested”.

He claimed that “HIV” is an endogenous retrovirus.

In support of his claim that “Human Endogenous Retroviruses (HERVs)...May offer an alternative explanation of the published data”, de Harven cited “TWO significant papers on HERVs:

1. “The viruses in all of us: characteristics and biological significance of human endogenous retrovirus sequences”, Löwer R et al; Proc Natl Acad Sci, USA 1996.
2. “Demystified...Human endogenous retroviruses”, Nelson, P.N. et al, Mol Pathol, 2003”.

In the introduction to the Nelson et al paper one reads: “Human endogenous retroviruses (HERVs) represent footprints of previous retroviral infection...Over 20 HERV families have been identified during the past two decades. (1-3) Although many are defective through the accumulation of mutations, deletions, and termination signals within coding sequences, a limited number of HERVs have the potential to produce viral products and, indeed, to produce viral-like particles”.

In other words, HERVs are not viruses and when one sometimes sees particles (always in cultures) the particles are virus-like, not viruses. The rest of the paper is about molecular biology and HERVs and the “possible involvement of HERVs” in disease, including malignancy.

As the title of the Löwer’s and Kurth paper suggest, in their view HERVs are just sequences in the human genome.

Regarding the main experimental approach for their discovery they wrote: “Screening human genomic libraries under low-stringency conditions with probes derived from animal retroviruses has allowed the isolation and characterisation of multiple, albeit defective, proviruses, representing different families [e.g. HERV-E, HERV-R, HTDV/HERV-K]”.

It follows that HERVs are not viruses but a name given to some particular DNA sequences in the human genome. Furthermore, since they have been obtained by “low-stringency conditions”, and since, according to the authors, hardly any parts of the “defective proviruses” have been shown to lead to “protein expression”, the possibility cannot be excluded that the “human endogenous retrovirus sequences” as well as the particles sometimes seen in cultures may have nothing to do with retroviruses.

The evidence de Harven gave in support of his claims that “HIV” is a HERV can be divided in two parts.

1. “The method used for determination of the alleged “HIV viral load” does not include the isolation of retroviral particles and the analysed pellets have never been controlled by electron microscopy to verify the hypothetical presence of such particles”.
2. “Problems with isolating HIV”.

The method used for determining the viral load

At the July 2000 Presidential AIDS Advisory Panel meeting in Johannesburg, four experiments were jointly approved by the “HIV” experts and the dissidents. Two were epidemiological and were proposed by Peter and Harvey at the first (held in Pretoria) meeting. The others were our isolation and pre-absorption experiments. De Harven did everything possible to be part of our experiments. We told him we intended these experiments to be done on behalf of all the dissidents and everybody, including him, was welcome to participate.

Not long after the meeting ended, a few other dissidents, including de Harven and Gordon Stewart, proposed their own experiments.

In an email to us on 22 July 2000, de Harven wrote: “I am convinced that we, i.e. both of you, myself, Roberto Giraldo, Gordon Stewart, Sam Mhlongo, Christian Fiala, plus any other who wishes to help, could, without delay, agree on a minimum, specific research plan (by minimum, I mean that the plan should at least include the co-cultures experiments Eleni described and the proposal #2 and #3 which I had posted in our website on June 19 and 20). Other proposals could of course be added, provided they can reasonably be terminated by the end of the year, as President Mbeki has requested. Our plan should then be submitted to the SA authorities for final approval and to decide who is going to do what and where. This will be my approach. I deeply believe that this is still feasible (provided we stay away from H.B.!), the self-appointed leader of the “etiology” group”.

De Harven’s experiment 3 was meant to determine if there was a correlation between “viral load” and “HIV” particles in blood (“viraemia”) as determined with the EM. Even before he posted his proposal we told him that, as we have shown, no evidence exists which proved the existence of such particles in the blood, so we could not see what he aimed to achieve with such an experiment. One cannot correlate “viral load” with something which has not been proven to exist, i.e. viral particles in the blood.

De Harven continued: “The total lack of EM correlation for high PCR viral load: I don’t understand why you bring your classic 1988 Med. Hypothesis paper at this point since “viral load” allegedly measured by PCR came up in the middle 1990. I should take the time to read over again your 1988 paper, but I presume that you were referring in that paper about the lack of EM evidence for retroviral particles in tissues, or lymph node biopsies from AIDS patients? I can hardly believe that in 1988 you were reviewing data concerning viraemia in AIDS patients, since that concept came up only a few years later? [Was not Gallo talking about the existence of “HIV” in the blood; viraemia, from day one?] That HIV particles were never found by EM in tissues from AIDS patients was indeed a well recognised fact in the 1980’s. (I myself was serving as consultant at Sloan Kettering in New York in the mid 1980’s, to review a massive amount of EM pictures coming from tissues of AIDS patients which was entirely negative for HIV particles!).”

On 11 August 2000 de Harven wrote further: “...it is true that I received a telephone call from Montagnier, early in July. He is interested in the research proposals I had submitted on the website (together with Gordon) on June 19 and 20. He seems particularly interested in our “Proposal #3” which you should have no difficulty to retrieve on the website. He gave me the impression that he would be willing to carry

it in his lab at Pasteur, with me! I didn't answer him directly at that time, because I wanted to have a chance to think about it a little more before any commitment on my part. In particular, I wanted to know how Sam Mhlongo would feel about this. Sam is currently in London and I just wrote to him.

Personally, I feel that a critical experiment done at Pasteur with Montagnier and myself could carry a lot of weight, don't you? Gordon is enthusiastic about the idea, although not directly involved.

In addition, I wonder who amplified the story by speaking about "attempts to complete the isolation of HIV"???!!! I sure never said anything like that.

What we speak about in our "Proposal #3 relates to a very specific point of PCR technology. A small point, but terribly important, however!!".

Responding to him we wrote: "...Believe us, we have been extremely busy trying to sort out problems related to the experiments. We do agree with you that there must be a consensus among all of us "on a minimum specific research plan". For this to be possible, it is absolutely necessary for everybody to keep each other informed.

Let us review the developments regarding the experiments based on President Mbeki's initiative as we see them:

Following the Pretoria meeting, Peter and Harvey proposed some epidemiological experiments. In our view, these experiments would have resulted in a totally misleading conclusion favouring the "HIV" theory. We expressed this view in one of our Internet postings and in repeated emails to Harvey to no avail. (In fact this view was expressed seven years before the AIDS Advisory Panel meetings by Val when he visited Harvey in New York City in November 1993). However, following lengthy discussion with Harvey in Johannesburg, he ended up saying repeatedly (we are paraphrasing): "I must be honest with you, Eleni, that we did not want you here. But the African land makes miracles and brought you here. I was running into the lion's den without realising and you stopped me. You are my salvation".

He then agreed to include the experiments we had proposed, that is, the pre-absorption and isolation experiments. He also said that we should write the protocols for these experiments and we should have a draft of the pre-absorption experiments by the end of August. We emailed him:

"In Johannesburg we had the impression all of us including you and Gordon agreed that the only way to answer the role of HIV in AIDS is to perform the isolation experiments and experiments designed to prove the specificity of the antibody tests. A definite answer to the latter experiments can only be obtained by using HIV isolation as a gold standard. An indication can be obtained by performing pre-absorption experiments and in our view, unfortunately, not by your proposed experiments #1 and #4. [2 and 3].

....Anita sent us your letter to Sam on 27th July and asked us to comment to you directly.

From your letter, do we understand correctly that you believe that the HIV isolation can be solved by performing experiments #2 and #3 proposed by you and Gordon? We cannot see how experiment #2 can achieve such an aim. In fact, to be honest with

you, we are at a loss to understand what you aim to achieve with this experiment. Neither can experiment #3 answer the question of isolation.

As far as experiment #3 is concerned, we agree with your description of it in your email to us on 1st August: “What we speak about in our “Proposal #3” relates to a very specific point of PCR technology. A small point, but terribly important, however!!”

This is why we agree with you that this experiment may be a useful auxiliary experiment to the basic experiment of isolation.

Concerning Montagnier’s eagerness to participate in the execution of experiment #3, do you think that this may be due to Montagnier being aware that:

- he has no proof for the existence of HIV,
- by performing experiment #3, he will avoid the isolation experiments.

Etienne, we may be wrong with our interpretation of the events so far and would very appreciate your comments. Look forward to hear from and collaborate with you”.

By 2001 de Harven transformed his correlation experiments (particles in blood versus viral load) into “HIV” isolation experiments. His, not our experiments, will solve the isolation question. In fact we never had anything to do with the isolation experiments. In November, 2001 de Harven wrote to Sam: “As I told you several times, we should not exclude the possibility (for the sake of speed) that the sampling from “High viral count” patients, the viral isolation/purification procedures, and the preparation of EM samples (plastic embedded blocks) be done in SA under my direct supervision, while I could organise the final EM observation of these plastic blocks in an EM lab either in Europe or in New York, providing this would be financially supported by the SA government”.

De Harven had a few problems, including the following:

- (1) He knew, and so did all the “HIV” experts familiar with EM, that if one is not able to purify “HIV” from the blood, it does not mean that “HIV” is not present in the blood. In fact even if one cannot see one particle it does not mean that “HIV” is not there. In 1965 de Harven went one step further: “It is fully realised that negative results in electron microscope virology do not mean that human leukaemia is not associated with or induced by viruses. Our remarks were presented mainly to stimulate a discussion on problems of interpretation in electron microscope virology”.

In other words, even if not one “HIV” particle is found in the blood of AIDS patients (not to mention that it is not possible to purify “HIV” from fresh blood), it does not mean that “HIV” is not there and is not the cause of AIDS. So where is de Harven’s crucial isolation experiment going to lead the dissidents?

- (2) De Harven’s second problem is so shocking that even now we cannot believe it. When he and Maniotis imagined that we were going to see Peter and wanted to teach him and us a few things, de Harven wrote:

“If we agree on the three following propositions:

- 1) That measurements of the so-called “viral load” are never made on isolated retroviral particles (isolated from the blood of “HIV+” individuals);
- 2) That, instead, these measurements are always made on extracts from circulating leukocyte nuclei;
- 3) That the human genome contains at least 6% of retroviral-analogous sequences”.

Incredibly, de Harven did not know what “viral load” is and since, according to him, “viral load”=“HIV” viraemia, he did not know what “HIV” viraemia is either. Nor did he know how “viral load” is measured.

Since:

- (i) this was so shocking;
- (ii) we heard that de Harven had been in a car accident and we did not know if he had fully recovered;
- (iii) we did not want to engage him in scientific argument unless fully recovered;

we made some enquiries with his good friend, Anthony Brink. Anthony told us that he had recently seen de Harven in France and that he was quite well.

So we responded: “You say “That measurements of the so-called “viral load” are never made on isolated retroviral particles (isolated from the blood of “HIV+” individuals)”. If by this you mean they should measure the number of RNA molecules in particles isolated from blood then it makes no sense. Why count molecules to estimate how many particles you have when you can count the particles? [When you have already counted the particles?]. You say “That, instead, these measurements are always made on extracts from circulating leukocyte nuclei”. The viral load is never done using “circulating leukocyte nuclei” or even cells, where most if not all the “HIV RNA” is in the cytoplasm. Are you confusing “viral load” (RNA in plasma) with “viral burden” (DNA, “provirus”)?”.

De Harven did not reply. His claims betray his lack of understanding of some of the most basic retrovirological concepts, not just viral load.

Looking back on some old emails we realised that this basic scientific mistake in the famous de Harven experiment posted on his website (19/20June 2000) and intended to be performed in Johannesburg or at the Pasteur Institute in Paris with Montagnier, was present from the very beginning. He wrote to Anita (15/12/2002): “PCR and “Viral Load”. The methodology currently used is based on the amplification of short nucleic acid sequences found in **white blood cell nuclei**. Not on any attempt to concentrate hypothetical retroviral particles” (emphasis ours).

From the moment de Harven became a dissident he claimed he will prove the error of the “HIV” theory of AIDS by performing an experiment. His experiment would

prove there is no correlation between “viraemia” as determined by “viral load” and “viraemia” as determined by counting the number of “HIV” particles visualised on EM. This was explained in plain language in an RA copyrighted article dated 19 June 2008 entitled “PCR FOR THE SO-CALLED MEASUREMENT OF HIV VIRAL LOAD” <http://www.rethinkingaids.com/tabid/126/Default.aspx> (quoted in Appendix 2 annexed).

Again this article shows that de Harven has no idea what “viral load” means and how it is determined. The continued presence of this article on the RA website is an indictment both of de Harven and of RA. RA would be well advised to remove it.

Following our email one will have thought that he would have corrected himself by the time of the Oakland meeting. He may have tried, but instead of an improvement, his talk again demonstrated his ignorance of the most basic concepts in retrovirology, and not just “viral load”.

One of his slides reads: “A sizeable percentage of the human genome, perhaps as much as 8% shows strong analogies to the retroviral genome. Therefore, pellets centrifuged from human plasma, with variable amounts of circulating DNA, inevitably contain retroviral-like sequences. Identified and amplified by PCR methodologies, these sequences are possibly misinterpreted as HIV markers and used for the alleged quantification of the hypothetical HIV “viral load””.

Firstly, viral load is determined by using “HIV” RNA circulating in the blood, not DNA;

Secondly, in another slide he said: ““viral load” means the presence of virus particles in the peripheral blood, i.e. “viraemia””.

Since the retroviral particles contain only RNA and not DNA, then no matter how high the number of viral particles (“HIV” or endogenous) in the blood, if one “identified and amplified by PCR methodology” the “circulating DNA”, the “viral load” according to de Harven, will always be zero.

“Problems with isolating HIV”

De Harven let the audience know that “difficulties to isolate and purify a so-called HIV have been initially stressed by Eleni Papadopulos et al [anybody who tries to find our work by doing a search on this name will get nothing], as early as 1993, in the classical Biotechnology paper. Such difficulties are best explained by recognising the fact that a so-called exogenous “HIV” does not exist as stated in 1994 by Stefan Lanka”. So here we have the whole truth. Duesberg is wrong. The Perth Group has never said “HIV” has not been isolated/purified, that is proven to exist, they only said there were “difficulties to isolate and purify”. Stefan Lanka only stated that “HIV” does not exist, but in science it is not sufficient to make statements, you must have proof and anyhow, scientifically it is not possible to prove that something does not exist. In other words de Harven is the only person who has done an “original” analysis and come with the proof that “HIV” has not been proven to exist.

His analysis consisted of the following:

1. In the Löwer et al and Nelson et al paper there is proof for the existence of endogenous retroviruses;
2. “Exogenous and endogenous retroviruses look alike under the electron microscope”;
3. “A historic paper was published in Science in 1983 by F. Barre-Sinoussi, Luc Montagnier and their collaborators at the Pasteur Institute in Paris”;
4. “The picture, unquestionably, illustrates the assembly (“budding”) of retroviruses on the cell surface of lymphocytes which had been added to the complex cell cultures studied at the Pasteur Institute”. The picture, that is, “Fig. 2, in this 1983 paper shows TYPICAL retroviruses budding on the surface of a lymphocyte. There is absolutely no doubt about that. Anybody with an “EM eye” will agree with me on that. These particles ARE RETROVIRUSES”;
5. “Such electron microscopy evidence is similar to the classic images of “budding” retroviruses, which I have published many years ago in studies of the Murine Friend Leukaemia Virus”;
6. “...the retrovirus producing cells in the French publication are cord blood, placenta derived lymphocytes...”;
7.
 - “Human placenta is loaded with exogenous retroviruses (HERVs);
 - Pasteur Group added HERVs of placental origin to their cell cultures;
 - Retroviral particle formation could be shown under these conditions;
 - This was not possible when using peripheral blood lymphocytes instead of cord blood placenta derived HERVs were essential for observation of retroviruses;
7. “So-called “HIV” has never been either isolated nor purified directly from an AIDS patient”.

Thus de Harven is the first person to have proven that “HIV” is not “HIV” but an endogenous retrovirus.

COMMENTS

- (a) Infectious agents, including viruses, are isolated from cultures (a Koch postulate) not directly from patients.
- (b) Nowhere in the 1983 *Science* paper is there any mention of EM studies of the culture containing BRU’s lymphocytes (the patient) or the co-culture from BRU cells with lymphocytes from the healthy blood donor, that is, that the pictures originated from a “complex cell culture”. (We have corrected this mistake of de Harven’s more than once: the culture from which the EM originated was not a “complex”, “mixture” but a pure umbilical cord lymphocyte culture).

- (c) In our email “Rethinking AIDS and the Perth Group – irreconcilable differences” sent to dissidents before the Oakland meeting, with de Harven as one of the recipients, we wrote:

“It will be interesting and revealing to see if de Harven will:

- (1) Continue to claim he was the only person to conduct a proper analysis of the Montagnier et al 1983 paper;
- (2) Produce evidence which will prove that the particles Montagnier saw in his umbilical cord culture are indeed retroviral particles (exogenous or endogenous);
- (3) Explain what were the particles (if not “HIV”) subsequently seen by Gallo, Levy and Weiss, whose cultures did not contain cord lymphocytes; ...
- (5) Explain, if Montagnier’s particles are endogenous placenta retroviruses, what are the antibodies in gay men which react with Montagnier’s “retrovirus”;
- (6) Produce evidence to prove his claim that the idea that “HIV does not exist...was that of Stefan Lanka, in 1994(?). And the PG swiftly appropriated it!!!”.

In Oakland de Harven again implied, without presenting any evidence, that we “swiftly appropriated” Stefan’s idea. However, since he did not respond to any of the other requests, including (3) above and since at no time did he analyse the Gallo, Levy and Weiss work, what is the scientific basis for his conclusions that the “so-called “HIV” has never been either isolated nor purified?

- (d) De Harven stated that Montagnier did not have a control culture. He said the same thing at the Europe meeting in 2003. “What was not done [by Montagnier] were the essential verification experiments that could have clarified the endogenous (**) origin of these viruses”.

In the BMJ on-line debate, de Harven again said that Montagnier did not have a control and that he was going to present such evidence. We responded that this was true, but the control experiments have been done by others. Instead of posting his answer, de Harven sent us an email:

“I wish to answer your reply concerning my Rapid Responses under reference, and your interpretation of the corresponding literature...

Briefly:

You quote Dourmaskins’s, 1992, Florence abstract. Unfortunately, you did not quote Dourmaskin’s paper published in 1993 in Journal of Medical Virology (1), and which is the development of the abstract you quoted. The abstract, most likely, didn’t contain any EM pictures, while the Journal Med. Virol. Paper did. If you (or John Papadimitriou) had seen these pictures you would have never taken Dourmaskin’s observations as a demonstration of “Budding retrovirus-like particles have been reported in non-HIV infected cord blood lymphocytes as well as many other cells used for HIV isolation” for the simple reason that the

particles published by Dourmashkin are about half the size of retroviruses and are not, therefore, “retrovirus-like”.

More importantly, if you read again the paper by Montagnier, Chermann, Barre Sinoussi et al in the 1984 Cold Spring Harbor Symposium (2) you will find, on page 377, that “virus production (LAV) by these lymphocytes REQUIRES STIMULATION AND THE CONTINUOUS PRESENCE OF TCGF”. As soon as I read again that sentence, I asked Dourmashkin, last month, if he had ever specifically used TCGF in his experiments on cord blood lymphocytes. His answer was negative. He used PHA, but not TCGF.

Consequently, and on the basis of these two experiments (1,2), it seems clear that the statement you made “Such an experiment has already been carried out” is not correct, because it has never been done, and certainly not by Dourmashkin”.

In his 1993 *Journal of Medical Virology* paper Dourmashkin defines as “HIV” the particles which had diameters of 130-200nm. However, according to Gallo and Gelderblom, retroviruses have diameters of 100-120nm. Many, including the CDC claimed that the diameter of “HIV” is 80-120 nm.

The cell-free particles which Dourmashkin saw have diameters of 70-80 nm. The very few cell-free particles in Montagnier’s paper (ref. 2) have a diameter of approximately 100nm, not double that of Dourmashkin’s particles. Both Dourmaskin and Montagnier published electron micrographs showing buds on the cell surface. Dourmashkin also pointed out that “different methods of preparation for EM” may lead to differences in size. The claim that Dourmashkin’s evidence cannot be considered a control for Montagnier’s findings because, unlike Montagnier who used TCGF, Dourmaskin used PHA (Montagnier used both), is so ridiculous it does not merit comment.

Obviously de Harven did not know we had done our homework and had already corresponded with Dourmashkin. De Harven wrote: “Searching for more consolidation of my analysis, I retrieved Sandra Panem, 1979, paper (3) on ³C-Type virus expression in the placenta². The EM evidence here is undisputable”.

De Harven wants us to believe that the particles released by Montagnier’s cord blood lymphocytes are identical to the particles released by Panem’s placental cells but not with those released by Dourmashkin’s cord lymphocytes.

He ended up by giving us an ultimatum: “I think that it is urgent to send to BMJ responses a short note correcting the statement you made that the control experiment, which I recommend had already been done years ago. This is obviously not the case. Personally, I would much prefer if we would sign such a little note together. It would carry a much better weight! (And our common HIV/AIDS struggle being hard enough, we cannot afford to look like having discordant views!). Could you, please, draft that little note and send it to me? Let us say that I shall wait 8-10 days for your reply, and I promise not to send anything by myself before June 16?”.

We responded: “Thank you for your email re the missing control experiment. We believe you should publish your concerns at the BMJ Online”. He never did.

For anyone to claim that Montagnier's particles were human endogenous retrovirus particles originating from the cord lymphocytes (placenta) evidence must exist which proves:

1. The existence of human endogenous retroviruses.

In not one of the references given to us by de Harven and Maniotis in support of their claim does such evidence exist. To the contrary. The authors specify that such viruses do not exist.

In 1994, writing in *Harrisons Internal Medicine* textbook, Gallo and Fauci stated "There are no known endogenous human retroviruses". In court in 2006 Gallo said "endogenous retroviruses aren't viruses as your first witness [Eleni] properly said, they are particles, they have never been transmitted. A virus is something that infects, that you prove goes from person A to B. Short of that they are particles. Where a virus at least has to be transmitted in vitro in the laboratory, it goes from one cell to another, it's never been demonstrated for endogenous retrovirus".

2. That Montagnier's (Panem) particles were infectious. Nowhere in Montagnier's paper is there evidence that the particles were transmitted "from one cell to another". In regard to Panem's particles, in one of the "two significant papers on HERVs" de Harven cited in his talk (Löwer et al) one reads: "The first indication that retroviruses had not spared the human species came from electron microscopic surveys of human placentas. **Retrovirus-like particles** were observed budding at the basal membrane of syncytiotrophoblasts" (emphasis ours).

In other words, the most one can say about Montagnier and Panem is that they have seen some retrovirus-like particles. However, in 1976 Gallo wrote, "Release of virus-like particles morphologically and biochemically [=containing RT activity] resembling type-C virus but apparently lacking the ability to replicate have been frequently observed from leukaemic tissue" (Some evidence for infectious type-C virus in humans. (1976). p. 385-405 In: *Animal Virology* Baltimore D, Huang AS, Fox CF, eds, Academic Press Inc., New York). Surely de Harven knows this. In 1965, following a talk he gave, "Remarks on viruses, leukemia and electron microscopy" (Wistar Institute Symposium Monograph Volume 4, 1965), one of the participants made the following comment: "In the enthusiasm to find a human leukemogenic virus, there is certainly danger of an unconscious willingness to think of virus-like particles as virus particles, or even oncogenic virus particles".

In his 1965 talk de Harven stated: "Identification of viruses under the electron microscope relies primarily on the observation of large populations of particles, the size of which should correlate with filtration data". However, de Harven is contradicting himself. Montagnier had only a few particles in the culture, none of which had all the morphological characteristics of retroviruses, and there were none in the 1.16 g/ml band. Hence, de Harven is contradicting himself a second time because there was

no correlation between EM in the culture and EM of the material banding at 1.16 g/ml. (Instead of filtration, Montagnier used banding in density gradients which de Harven admits is a better method than filtration for retroviral purification).

In an email, 11 April 2000, de Harven wrote: “Virus-like particles can be found everywhere and do not have any interest”.

3. The particles contain RNA (not DNA). The particles’ proteins are coded by this RNA and the RNA is a transcript of endogenous retroviral sequences. No such evidence exists in Montagnier’s paper. It is agreed that for such evidence to be obtained one must have purified the particles. As we all know in his “purified” virus Montagnier had only cellular debris. De Harven is telling us all that to prove the existence of a retrovirus one must purify the virus. His “original” analysis of the Montagnier paper shows that Montagnier did not isolate/purify “HIV”, that is, prove the existence of “HIV”. But, at the same time and despite the fact that “Monty” (Montagnier) did not isolate/purify his particles, he did prove the existence of a retrovirus, an endogenous retrovirus.

De Harven stated that Montagnier’s “...electron microscopy evidence is similar to the classic images of “budding” retrovirus which I have published many years ago in studies of the Murine Friend Leukaemia Virus”. We agree. And the reason is simple: neither Montagnier nor de Harven had any proof that their “images” are those of a retrovirus. Is de Harven protecting Montagnier’s “virus” or is he protecting his “Friend leukaemia virus”? In his email regarding the BMJ debate on the missing control, de Harven wrote: “Finally, you mentioned in your BMJ reply “cellular protrusions resulting from localised contraction of the actin-myosin system”, quoting your 1996 Continuum paper on “The isolation of HIV-Has it been achieved?” I read it again, with special attention to section 5.7 on “Budding”, and I am intrigued by the following question. If you are still “tempted to speculate” that “HIV” particles and proteins are nothing more than non-viral material altogether, induced by the agents to which the AIDS patients and cultures are exposed? you should also be “tempted to speculate” that the same reasoning applied to budding Rous sarcoma virus, budding Mouse mammary tumor virus, or Friend leukaemia virus, since they all “bud” exactly in the same manner, and likely with the same assistance from contractile cellular proteins? You would then reach some agreement with S. Lanka who was claiming 10 years ago, that the entire retrovirology is an artefact? As far as I can see, this is nothing more than your “tempting speculation”, and I don’t think that Peyton Rous should turn around in his grave!”.

Really? As we have repeatedly pointed out, in 1911 Rous did not consider his experimental evidence as proof the cause of the chicken sarcoma was a virus: “The first tendency will be to regard the self-perpetuating agent active in this sarcoma of the fowl as a minute parasitic organism...But an agency of another sort is not out of the question. It is conceivable that a chemical stimulant, elaborated by the neoplastic cells, might cause the tumour in another host and bring about in consequence a further production of the same

stimulant”. In his Nobel lecture Rous said “Tumors destroy man in a unique and appalling way, as flesh of his own flesh which has somehow been rendered proliferative, rampant, predatory and ungovernable. They are the most concrete and formidable of human maladies, yet despite more than 70 years of experimental study they remain the least understood. This is the more remarkable because they can be evoked at will for scrutiny by any one of a myriad chemical and physical means which are left behind as the tumors grow”. The passing of 24 years did not alter that opinion. In 1935 he wrote: “Some authorities believe that virus phenomena should be interpreted in terms of what is known of bacteria, while at the other extreme are those who hold that certain viruses at least are the inanimate products of disordered cells”.

In 1999, two medical historians writing about Duran Reynals, “most likely the first Catalan to become a research scientist of world renown”, and his close collaboration with Peyton Rous, wrote “...even though the Nobel Committee recognised the “agent” as a virus when it awarded Rous the Nobel Prize for Medicine, he still refused to recognise it as such”.

Yet, de Harven wants us to accept (a) that Rous discovered the first retrovirus; (b) the existence of endogenous retroviruses and their multiple pathogenicities despite the fact that all retrovirologists, including Gallo, deny their existence. Have we not all had quite enough of the retroviral/oncogenic theory of cancer?

CONCLUSION

There is one thing we and de Harven agree on: the dissident science which he and a few others promote is “scientifically highly questionable”, “embarrassing” and “damaging for the RA’s credibility”. The problem is that RA claims to represent all dissidents and their science as being THE dissident science.

Appendix 1

-----Original Message-----

From: Page, Barry [mailto:Barry.Page@health.wa.gov.au]
Sent: Thursday, September 01, 2005 3:13 AM
To: Goswami, Prabhat
Subject: Redox Regulation of the G1 to S Phase Transition Evidence

Dear Professor Goswami

I am a physicist currently working in cancer radiation therapy.

I read your 2003 paper "Redox Regulation of the G1 to S Phase Transition in the Mouse Embryo Fibroblast Cell Cycle" [Cancer Research 63, 2109-2117, May 1 2003] which I found extremely interesting.

I would be grateful if you would tell me how you came up with such an interesting hypothesis. Are there any papers you can refer me to? Have you done any further work in regards to this hypothesis? How can your findings be used in clinical practice?

Thanking you in anticipation.

Regards

Barry Page

-----Original Message-----

From: Goswami, Prabhat [mailto:prabhat-goswami@uiowa.edu]
Sent: Wednesday, 7 September 2005 4:18 AM
To: Page, Barry
Subject: RE: Redox Regulation of the G1 to S Phase Transition Evidence

Hi Dr. Page,

Thanks a lot for your interest in our work. We have ongoing interest in this field of research. I have attached few of our published papers and a book chapter. Also, I have included couple of papers from a colleague of mine, which you might find interesting.

(1) My supervisor (who died in the year 1991) and I discussed about this while I was working in his laboratory, but never had a chance and resources to do this work. Finally, I managed to secure some funding and graduate students to jump-start the project. It is going on well so-far--

(2) The redox-regulation of the cell cycle will have significant impact in treating cancer and other pathophysiological conditions of aberrant cellular proliferation. You might find the paper that we published in the journal of Antioxidant & Redox-Signaling interesting. I hope our

paper published in the Journal of Biological Chemistry will have significant impact in the Aging and Cancer research field.

Hope to meet you sometime in one of the scientific avenues.

With regards

Prabhat Goswami

-----Original Message-----

From: Page, Barry

Sent: Tuesday, 15 November 2005 14:58

To: 'Goswami, Prabhat'

Subject: RE: Redox Regulation of the G1 to S Phase Transition Evidence

Dear Professor Goswami

Thank you for your email and the attached papers which I read with great interest. I gave your papers to a colleague to obtain her opinion on them as she has been working on cell function for some time. She claims that she predicted all of your findings and gave a detailed mechanism a quarter of a century ago. Are these claims true? Attached is one of her papers.

I would very much like to have your opinion on this.

Thanking you in anticipation.

Regards

Barry Page

Professor Goswami did not respond.

Appendix 2

Etienne de Harven, 19 June 2008

PCR FOR THE SO-CALLED MEASUREMENT OF HIV VIRAL LOAD

The idea of looking for retroviruses in the blood of AIDS patients was in a way logical, because it is indeed in the blood of mice and chickens that zillions of retroviruses (RNA tumor viruses) were readily isolated and purified 40 years ago...

BUT:

In all the old work (1960s) on experimental animals, the INITIAL step was the isolation of RETROVIRAL PARTICLES, either by sucrose density centrifugation, or (as in my work on the Friend virus) by two steps of Millipore ultrafiltration. This was leading, by a final, high speed centrifugation to a minuscule pellet that could readily be prepared for electron microscopy (transmission EM, plastic embedding, and thin sectioning), pellets in which thousands of packed retroviral particles could be easily demonstrated (see my 1997-78, vol 5 n°2, paper in Continuum, page 24), and pellets that could be then used for biological experiments (transmission of the disease to receptive experimental animals), and/or biochemical analysis (characterization of proteins and nucleic acids). The retroviral origin of these proteins and nucleic acids was unquestionable, because of the extremely high level of purity, demonstrated by EM, of the viral pellets. In all such experiments, all erythrocytes and leucocytes were first completely eliminated, by low speed centrifugation.

In today's so-called "viral load" studies this logical approach to retroviral isolation is completely ignored.

Simply because *NOTHING IS DONE* to first isolate retroviral particles!

Instead: the PCR "Viral load" method starts of by first collecting LEUCOCYTES!
Not viral particles!

Leucocytes are indeed collected, their nuclei extracted, their nuclear envelopes dissolved with detergent, and their CHROMATIN prepared for nucleic acid amplification by PCR !!! In any chromatin samples there is little surprise to find nucleic acid!

BUT: 6% or more of the human genome has striking homology with retroviral genome, a fact that is well documented for more than a decade. So, PCR has no difficulty to recognize short retroviral-like sequences in these human chromatin samples (never twice the same, but never mind: it keeps mutating !!), and to amplify it 1000 or million times! Bingo: this is HIV !!!!! NO: it is the amplification of endogenous retroviral sequences that are present in ALL OF US! It has NOTHING to do with the hypothetical presence of circulating retroviral particles! It has nothing to do with any "measurement" of the "viral load". By that method, WE ALL have some

level of ... "viral load"!!! Really? Yes, but to save the establishment from too much embarrassment NO CONTROL, on you and me, was ever made nor published! Do you know the reference of one single paper in which a large group of "normal" individuals would have been studied for HIV "viral load" by PCR measurement? I don't.

Add to this:

1. That at Mbeki's conference, Pretoria May 2000, I formally stated that not one single retroviral particle has ever been visualized by electron microscopy in the blood of any patient with a so-called high viral load, and that that statement has never been refuted;
2. That at the European Parliament debate, Brussels Dec 2003, I directly asked Luc Montagnier to give us his definition of the "viral load" and received an extremely ambiguous answer (page 196 of the proceedings), a fact that Prof. Gordon Stewart, who participated in that debate in Brussels, can most probably confirm.

In conclusion: the so-called measurements of HIV "viral load" by PCR methodology are completely missing any scientific relevance.

— *Etienne de Harven, June 19, 2008*