

PERTH GROUP COMMENTARY ON THE RETHINKING AIDS RESPONSE TO THE GALLO *ET AL* CRITICISM OF CELIA FABER IN HARPERS

In the view of the "HIV" experts there are two groups of "dissidents": " Mullis, Giraldo, De Harven etc have all made statements that build upon the "logic" of Duesberg and the Perth Group. Nothing original". (N Bennett: Re:Re:Re: Analysis: the properness of the HIV hypothesis is a media hype, 21 March 2005 http://www.rethinking.org/bmj/response_101072.html). The "HIV" experts as well as the "dissidents" are fully aware there are significant differences between Peter Duesberg and our "logic". The aim of Celia Faber's article in Harpers seems two-fold: exposure of the unethical behaviour in some scientific circles and the promotion of the "dissidents" view. We were surprised to see that the only "dissident" view presented was that of Peter Duesberg. We were even more surprised to see that the 56 "items" meant to respond to the Robert Gallo *et al* criticism of Celia Faber's article are also based on Peter Duesberg's "logic". Nonetheless, we would like to make some comments in regard to some.

Items 1, 4, 5, 12, 13

The statements "False Positive HIV Tests" in pregnancy, "HIV Antibodies in Babies", "Problems with HIV Tests", "Are HIV Tests Widely based in Africa", and "Tropical Diseases and False Positive HIV Tests" assume the existence of a unique human retrovirus, "HIV" as well as antibodies specific to it in same group or individuals. No such evidence exists.¹⁻¹² In 1996 when Peter Duesberg claimed the *Continuum* prize, he also claimed that the existence of the "HIV infectious molecular clone" is the proof that HIV exists. However, he was never able to present any evidence for the existence of the "HIV" molecular clone. At the same time he also wrote "...particles and proteins could reflect non-viral material altogether". Now he claims some recently published electron micrograph of particles found in cultures, which have never been shown to be retroviral particles, are proof for the existence of HIV. <http://www.theperthgroup.com/REJECTED/StructureLetterPG.pdf>

In the BMJ on-line debate, Brian Foley, the "custodian" of the "HIV" genome at the Los Alamos Laboratories used Peter Duesberg's 1996 argument as evidence for the existence of HIV. Yet like Peter Duesberg he too was never able to present any evidence for the existence of the "HIV infectious molecular clone".

Item 11 - Does AIDS in Africa Differ from Malnutrition, Malaria, Parasitic Infections and other Common Tropical Illnesses?

A thorough and original analysis of item 11 can be found in our papers AIDS in Africa Distinguishing Fact and Fiction;¹³ Deconstructing AIDS, Reconstructing AIDS;¹³⁻¹⁵ a letter to the South African, Sunday Independent newspaper, <http://www.theperthgroup.com/SOUTHAFRICA/NEWSPSA1.pdf>; our Mother to Child Transmission Monograph¹⁰ and the BMJ on-line debate.¹¹

Item 18, Did HIVNET 006 Lower Viral Load?; Item 20, HIVNET 012 Protocol Changes; Item 21, HIVNET 012: Phase II or Phase III?; Item 22, HIVNET

012 Not Placebo Controlled; Item 28, Maternal Nevirapine Proven in 'Multiple Studies'; Item 29, Valendar Turner, Nevirapine and Placebo

The most thorough and original analysis of these items can be found in Part IV of our Mother to Child Transmission Monograph,¹⁰ our letter to Nature¹⁶ and PowerPoint presentation

<http://www.theperthgroup.com/PRESENTATIONS/nevpps1.ppt>

Item 30, Nevirapine Study with 561 People

Neither in the letter to *Nature* by one of us (Val Turner), nor in Gallo's critic, is there any mention of "Nevirapine study with 561 people". To the contrary. In the 1998 Rwanda study of 561 "people" where the transmission rate was reported to be 12%, no antiretroviral drugs were used.¹⁷

Item 31, Vitamin A and HIV Transmission Rates

An analysis of "Vitamin A and HIV Transmission Rates" is found in part V of our Mother to Child monograph.¹⁰

Item 38, How Does AZT Work?

According to Peter Duesberg, AZT is triphosphorylated extracellularly. The triphosphorylated AZT enters the cells and attaches to the DNA and stops its synthesis - AZT is a DNA chain terminator and by doing so becomes toxic to the cells.¹⁸ However, the enzymes which triphosphorylate AZT are found only intracellularly which means that AZT cannot be triphosphorylated extracellularly. In addition, the cell membrane is impermeable to the phosphorylated nucleotides.¹⁹ Furthermore, all the presently available data show there is no significant triphosphorylation of AZT even intracellularly.²⁰ Since for triphosphorylation of AZT reducing equivalents are necessary, and since AZT is an oxidising agent by which property it induces its toxicity, this is not surprising.²⁰

Item 39, Does HIV Cause Any Diseases?

An answer to this question presumes the existence of HIV. No such evidence exists.²¹ (See previous).

Item 40, Does HIV Fulfil Koch's Postulates for AIDS?

If one accepts that "HIV" and "HIV" antibodies exist, then one has no choice but to also accept that Koch's postulates have been fulfilled which means that HIV is the cause of AIDS.

Item 42, Is HIV Active In The Bodies of AIDS Patients?; Item 43, Can HIV be Isolated Without 'Reactivating' Latent Copies?

These questions imply that "HIV" exists in the bodies of patients in an inactive form and that it can be isolated by "Reactivating Latent Copies". If "HIV" exists in an inactive form in AIDS patients and those at risk then, given that AIDS patients and those at risk are exposed to numerous agents which activate retroviruses, it would also be present in an active form as well. In fact "HIV" has never been isolated even *in vitro* without the use of a plethora of activating agents. By "isolation" the "HIV" experts including Montagnier mean detection of reverse transcriptase (RT) activity. Nowadays the non-specificity of RT is

known even to the general public in the form of magazine reports evaluating the investment potential of biotechnology stocks.²²

Item 44, Antibodies Mean Immunity...Except for HIV?

Only a minority of people (Peter Duesberg among them) believe that "Antibodies Mean Immunity". Many "HIV" experts including Gallo *et al* in their critique repeatedly presented evidence which disproves the claim that "Antibodies Mean Immunity". Yet, Peter Duesberg still insists that "Antibodies Mean Immunity". Antibodies do not mean immunity as was shown as long ago as 1935 by no less an authority than Albert Sabin.²³⁻²⁵

Item 45, Does 'Viral Load' Measure Live Virus?

By the nature of the test, viral load cannot measure either "Live" or not "Live" virus. It can only measure the "viral" RNA. Since there is no evidence that proves the existence of a unique human retrovirus, "HIV" either "Live" or not "Live" and thus "HIV" RNA, it is not possible to claim that the "viral load" measures any of them.

Item 47, Does HIV Spread Randomly? Should it?

Before one asks "Does HIV spread randomly", one must first have evidence which proves that:

- (i) "HIV" exists
- (ii) "HIV" spreads by any means.

No such evidence exists.^{10, 13-15, 21, 26}

Item 50, Does HIV Kill T-Cells?

A most comprehensive answer to the question "Does HIV kill T-cells?" can be found in our *Genetica* paper²⁷ and in the BMJ Online debate.²⁸

Item 52, Non-HIV Causes for AIDS

Before "HIV" was accepted as a cause of AIDS we were the first to propose a non-infectious theory of AIDS which encompassed all the AIDS risk groups (gay men, haemophiliacs, drug users) and included a mechanism of pathogenesis. In our non-infectious theory of AIDS, drugs both recreational and prescribed were considered to play a significant role. The discovery and acceptance of "HIV" as a causative agent of AIDS led us to critically analyse the evidence which was claimed to prove the existence of "HIV" as well as its role in AIDS. It was concluded that (a) neither Montagnier's *et al* nor Gallo's *et al* evidence proved the existence of a unique human retrovirus, LAV/HTLV-III (HIV) even *in vitro*, much less in AIDS patients and those at risk; (b) AIDS and the phenomena which were claimed to prove the existence of HIV were the result of the many oxidative agents such as recreational and prescribed drugs (including factor VIII infusions), to which the AIDS patients and those at risk were exposed. The conclusions were incorporated in a paper sent to *Nature* entitled: Reappraisal of AIDS: Is the oxidation induced by the risk factors the primary cause? The paper was rejected. The 10th of July 1986 letter of re-submission to *Nature* was accompanied by a response to the reasons given by the Journal for rejection. The response ended with the following: "If my paper does nothing

other than draw attention to the oxidative nature of the risk factors and its biological importance, then it offers what is so far the only hope of treatment which will arrest and reverse the otherwise invariable fatal course of the disease. This alone would more than justify its publication". The paper was again rejected and was eventually published in *Medical Hypotheses* in 1988.² In the same year the paper was sent to Peter Duesberg.

In a paper²⁹ in which Peter Duesberg accepted that the "hallmark" of AIDS "is a defect in T-cells" and that the existence of "HIV" has been proven, he put forward a number of arguments against the "HIV" hypothesis of AIDS. He did not give an alternative explanation for AIDS. In 1989 paper Peter Duesberg wrote: "What Are the Causes of AIDS? I propose that AIDS is not a contagious syndrome caused by one conventional virus or microbe... Since AIDS is defined by new combinations of conventional diseases, it may be caused by new combinations of conventional pathogenic factors. The habitual administration of factor VIII or blood transfusions or of drugs, chronic promiscuous male homosexual activity that is associated with drugs, numerous acute parasitic infections, and chronic malnutrition, —each for an average of 8 years—are factors that appear to provide biochemically more tangible and plausible basis for AIDS than an idle retrovirus".³⁰

In a 1990 paper he wrote "The risk-AIDS hypothesis suggest that AIDS is caused primarily by non-infectious agents. These include psychoactive drugs, over-medication with antibiotics and above all AZT, a chain terminator of DNA synthesis administered to treat HIV infection since 1987".³¹ Subsequently Peter Duesberg published numerous papers and a book³² in which he argued against the "HIV" theory of AIDS and claimed that "HIV" is an "innocent", "passenger" virus and that "antibodies to HIV" are one of the "most specific markers" for AIDS.³⁰

Unlike Peter Duesberg, from the beginning of the "HIV" era we claimed there was no evidence which proves the existence of "HIV" proteins or antibodies. Our claims were vindicated in 1997.^{12, 33-35}

According to Montagnier, "analysis of the proteins of the virus demands mass production and purification" of the viral particles. In 1983 Montagnier and his colleagues claimed to have purified "HIV". In their "purified" virus they found three proteins, p80, p45 (now known as p41) and p24 that reacted with antibodies in their patient's serum. They made no comments regarding p80 and the reacting antibodies, claiming that p41 was the cellular protein actin, and p24 to be the "HIV" specific protein and the antibodies which reacted with "HIV" antibodies. In 1997 Montagnier admitted that in their "purified" virus they did not have even particles with "the morphology typical of retroviruses" much less a specific retrovirus, "HIV".^{35, 36}

The minimum absolutely necessary but not sufficient condition to claim that what are called "HIV" proteins are viral and not cellular is to show that the sucrose density fraction said to represent "purified" virus contains proteins which are not present in the same fraction obtained from non-infected cells, what Bess *et al* referred to a "mock" virus. In 1997 Bess *et al* showed this is not

the case. The only difference one can see in their SDS-polyacrylamide gel electrophoresis strips of "purified" virus and "mock" virus is quantitative, not qualitative. Bess *et al* left the strip from the "mock" virus unlabelled. The proteins of molecular weight 30,000 and above were labelled as cellular proteins. Although Bess *et al* did not determine the identity of the p24, p13, p6/7 proteins, they were labelled "HIV" proteins " when one of the reviewers asked for them".

Thus according to the presently available evidence the ultimate origin of all the "HIV" proteins including the most famous, p24, are cellular proteins.

The question is what are the antibodies present in the patient sera which react with these proteins? Many researchers, including Montagnier, have shown that AIDS patients and those at risk have high levels of auto-antibodies such as anti-cardiolipin, anti-nuclear factor, anti-cellular, anti-platelet, anti-red cell, anti-actin, anti-DNA, anti-tubulin, anti-albumin, anti-myosin and anti-thymosin antibodies.^{6, 37, 38} Anti-lymphocyte auto-antibodies have been found in 87% of HIV seropositive patients and their levels correlate with clinical status.^{39, 40} It is also known that serum IgG levels are higher in African-American blood donors than in Caucasians.⁴¹ Furthermore, AIDS patients and those at risk are exposed to many infectious agents, whose antibodies may cross-react with the "HIV" proteins.

The presently available data shows that among the risk groups in North America, Europe and Australia a positive test confers upon an individual a propensity to develop and die of diseases defined as AIDS. This should be expected. Healthy individuals do not develop high levels of antibodies which persist for a long time. The fact that individuals belonging to the high risk groups have high levels of antibodies means that something is amiss in the body but it does not reveal exactly what is the problem.

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 is simply the rate at which red blood cells fall to the bottom of a test tube of blood. It was discovered by John Hunter in the late 1700s as an "indicator of inflammatory conditions"⁴² and rediscovered in 1918 by Fahraeus while seeking an early test for pregnancy. It is a commonly used but non-specific test which, when elevated, "is a measure of the presence and intensity of morbid processes within the body".⁴³ 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". Even asymptomatic, non-anaemic HIV positive individuals may have an increased ESR⁴⁴ and the test may be predictive for disease progression.⁴⁵ In HIV positive children a correlation exists between seropositivity, hypergammaglobulinaemia and an elevated ESR.⁴⁶ As far back as 1988 researchers from the Institut 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".⁴⁷ In other words the ESR is a superior predictive marker for the development of the clinical AID syndrome than is a decrease in the CD4 cell count, although the latter is said to be the cause of the syndrome. One important factor which affects the ESR is the size of the red cells, especially rouleaux formation where the red blood cells clump together. Rouleaux formation may result from changes in the negative charge of red cells, caused by "the dielectric effect of proteins in the surrounding plasma", especially by "fibrinogen, immunoglobulins, and other acute-phase reaction proteins",⁴³ and their increased levels in some disease states. Diseases such as tuberculosis and AIDS are not caused by red blood cell clumping induced by "the dielectric effect of proteins" but the fact this can be demonstrated and measured *in vitro* is of great diagnostic and prognostic utility in clinical practice.

Given that the evidence that "HIV" proteins are existing 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, like the ESR, represent nothing more than a non-specific indicator, serendipitously discovered in 1983/84, of altered homeostasis connoting a propensity to develop particular diseases. Whether or not a positive test is predictive of such diseases outside the risk groups, remains to be proven. As long as the present interpretation of a positive test is accepted this may never be ascertained because knowledge of seropositivity by both patient and physician attracts multiple confounding factors virtually impossible to eliminate.¹⁰

The fact that "HIV" antibodies do predict illness from AIDS indicator diseases without proof of their being caused by a retrovirus appears to be a stumbling block for some scientists. It should not be. Every mother of every baby knows that if her baby appears unwell she can measure his temperature. If this measurement shows he has a fever she knows something is amiss. She will expect other symptoms and signs to develop over the ensuing days. When they do she may gauge resolution of the illness by noting the fever abates. However, she does not imagine for a moment that her baby's temperature is diagnostic of one of the several dozen common diseases of childhood. Mothers would have no problem understanding our argument in regard to "HIV" antibodies and the propensity to illness.

Peter Duesberg, claims that the "Antibody to HIV is a Marker for American AIDS Risk" but "HIV is a harmless passenger". He claims that it is easy to prove that HIV is only a passenger virus because if a study is done one will discover that "HIV" positive and "HIV" negative individuals from the risk groups will develop AIDS and die from it with the same frequencies. (See the Duesberg Phenomenon.⁴⁸ Duesberg and Critics Agree: Haemophilia Is the Best Test.⁴⁹ Although we as well as others have pointed out the ample evidence to the contrary, he still insists this is the case. Some of the best evidence is published by the MultiCenter AIDS Cohort and similar studies.^{50, 51} In Celia Farber's article one reads: "Given that the evidence for HIV is coincidental, a number of research avenues suggest themselves, yet orthodox AIDS researchers have failed to demonstrate, using large-scale controlled studies, that the incidence of

AIDS-defining diseases is higher among individuals infected with HIV than among the general uninfected population. Consequently, it could very well be the case that HIV is a harmless passenger virus...no controlled studies have been carried out to prove that hemophiliacs infected with HIV die sooner than those who are not infected".

Gallo *et al* presented well documented evidence that a positive test signifies an increased probability of having or developing AIDS. It is amazing that this response by Gallo was not addressed in the 56 items considered for response to Gallo *et al*. Since this item relates to the "crucial" experiments proposed by Peter Duesberg, does it mean that Peter Duesberg is in agreement with the "HIV" experts? Or would he agree with us that a positive antibody test in the AIDS risk groups predicates an increased probability of having or developing AIDS but is not proof for the presence in humans of a unique retrovirus "HIV"? If the former is the case he may find himself in disagreement with the discoverer of "HIV", Luc Montagnier who appears to have become an apologist of our oxidative theory of AIDS.

In 1997, that is more than a decade after we proposed an oxidative theory of AIDS and "HIV" and fully aware of it, Montagnier and his associates wrote: "in AIDS pathogenesis, oxidative stress is *proposed* as a metabolic alteration that favours disease progression by inducing both viral replication and apoptotic death".⁵² A speech which Montagnier gave in 2003 to the European parliament can be summarised as follows:

- (i) the cause of the clinical syndrome, that is of the diseases and thus death of the patients is acquired immune deficiency (AID) whose main determinant is T4 cell decrease.
- (ii) T4 Cell death is due to apoptosis.
- (iii) The cause of apoptosis is oxidation.
- (iv) The cause of oxidation is African is malnutrition.

Thus, according to the discoverer of "HIV" the cause of AIDS is OXIDATION.

REFERENCES

1. Papadopulos-Eleopulos E. A Mitotic Theory. *J Theor Biol* 1982; 96:741-758
<http://www.theperthgroup.com/EPE/MitoticTheory.pdf>
2. Papadopulos-Eleopulos 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>.
3. Papadopulos-Eleopulos E. Looking back on the oxidative stress theory of AIDS. *Continuum* 1998; 5:30-35
<http://www.healtoronto.com/oxstress.html>.
4. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Oxidative stress, HIV and AIDS. *Res Immunol* 1992; 143:145-8
<http://www.theperthgroup.com/SCIPAPERS/oxstresshiv aids.html>.
5. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Has Gallo proven the role of HIV in AIDS? *Emerg Med [Australia]* 1993; 5:113-123
<http://www.theperthgroup.com/SCIPAPERS/emedhivgallo.html>.
6. Papadopulos-Eleopulos 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>.
7. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Virus Challenge. *Continuum* 1996; 4:24-27
<http://www.altheal.org/continuum/Vol4no1.pdf>.
8. Papadopulos-Eleopulos 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; 14:185-6
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=9787984.
9. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Stewart G, Causer D. HIV antibodies: further questions and a plea for clarification. *Curr Med Res Opinion* 1997; 13:627-34
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=9327197.
10. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, et al. Mother to Child Transmission of HIV and its Prevention with ATZ and Nevirapine. Perth: The Perth Group, 2001:204
<http://www.theperthgroup.com/MONOGRAPH/MTCTMay2005.pdf>.
11. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Alfonso H, Causer D. The Last Debate. *Reappraising AIDS* 1992
<http://www.virusmyth.net/aids/data/epdebate.htm>.
12. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, et al. A critique of the Montagnier evidence for the HIV/AIDS hypothesis. *Med Hypotheses* 2004; 63:597-601
<http://www.theperthgroup.com/SCIPAPERS/MHMONT.pdf>
- 10.1016/j.mehy.2004.03.025.
13. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Bialy H. AIDS in Africa: Distinguishing fact and fiction. *World J Microbiol Biotechnol* 1995; 11:135-143
14. Papadopulos-Eleopulos E, Turner VF. Deconstructing AIDS in Africa. *The Independent Monthly* 1994:50-51
15. Papadopulos-Eleopulos E, Turner VF. Reconstructing AIDS in Africa-Reply to Kaldor and Ashton. *The Independent Monthly* 1995; February:23-24
16. Turner VF. HIV drug remains unproven without placebo trial. *Nature* 2005; 434:137
<http://www.theperthgroup.com/SCIPAPERS/NatureVFTNVP.pdf>.
17. Ladner J, Leroy V, Hoffman P, et al. Chorioamnionitis and pregnancy outcome in HIV-infected African women. *Pregnancy and HIV Study Group. Journal of the Acquired Immune Deficiency Syndrome and Human Retrovirology* 1998; 18:293-8
18. Lauritsen J. *Poison by prescription--The AZT story*. New York: Asklepois Press, 1990.

19. Leibman KC, Heidelberger C. The metabolism of P³² labelled ribonucleotides in tissue slices and cell suspensions. *J Biol Chem* 1955; 216:823-830
20. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Causer D, Alphonso H, Miller T. A critical analysis of the pharmacology of AZT and its use in AIDS. *Curr Med Res Opinion* 1999; 15:1s-45s
<http://www.thepertgroup.com/SCIPAPERS/cmroazt.html>.
21. Johnson C. Is HIV the cause of AIDS? Interview with Eleni Papadopulos-Eleopulos. *Continuum* 1997; 5:8-19
22. Pachacz M. No need to be phased. *Shares*. Vol. 6, 2001:28-32.
23. Sabin AB. The mechanism of immunity to filterable viruses. II. Fate of the virus in a system consisting of susceptible tissue; immune serum and virus, and the role of the tissue in the mechanism of immunity. *British Journal of Experimental Pathology* 1935; 16:84-101
24. Sabin AB. The mechanism of immunity to filterable viruses. I. Does the virus combine with the protective substance in immune serum in the absence of tissue? *British Journal of Experimental Pathology* 1935; 16:70-84
25. Sabin AB. The mechanism of immunity to filterable viruses. IV. The nature of the varying protective capacity of antiviral serum in different tissues of the same species and in the same tissues of different species. *British Journal of Experimental Pathology* 1935; 16:169-181
26. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, et al. Global voices on HIV/AIDS. Heterosexual transmission of HIV in Africa is no higher than anywhere else. *Brit Med J* 2002; 324:1035.
<http://bmj.com/cgi/content/full/324/7344/1034#resp3>.
27. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Hedland-Thomas B, Causer D, Page B. A critical analysis of the HIV-T4-cell-AIDS hypothesis. *Genetica* 1995; 95:5-24
<http://www.thepertgroup.com/SCIPAPERS/ept4cells.html>.
28. Perth Group T. Rapid responses for Fassin and Schneider 2003-2005. *British Medical Journal Online* 2003
<http://bmj.bmjournals.com/cgi/eletters/326/7387/495#43617>.
29. Duesberg PH. Retroviruses as carcinogens and pathogens: Expectations and reality. *Cancer Res* 1987; 47:1199-1220
30. Duesberg PH. Human immunodeficiency virus and acquired immunodeficiency syndrome: correlation but not causation. *Proc Natl Acad Sci U S A* 1989; 86:755-764
31. Duesberg PH. AIDS: non-infectious deficiencies acquired by drug consumption and other risk factors. *Res Immunol* 1990; 141:5-11.
32. Duesberg PH. *Inventing the AIDS Virus*. Washington, USA: Regnery Publishing, Inc., 1996.
33. Bess JW, Gorelick RJ, Bosche WJ, Henderson LE, Arthur LO. Microvesicles are a source of contaminating cellular proteins found in purified HIV-1 preparations. *Virology* 1997; 230:134-144
<http://leederville.net/links/Bess.pdf>.
34. Gluschankof P, Mondor I, Gelderblom HR, Sattentau QJ. Cell membrane vesicles are a major contaminant of gradient-enriched human immunodeficiency virus type-1 preparations. *Virology* 1997; 230:125-133
35. Tahi D. Did Luc Montagnier discover HIV? Text of video interview with Professor Luc Montagnier at the Pasteur Institute July 18th 1997. *Continuum* 1998; 5:30-34
<http://www.virusmyth.net/aids/data/dtinterviewlm.htm>.
36. Papadopulos-Eleopulos E. Between the lines-a critical analysis of Luc Montagnier's interview answers to Djamel Tahi. *Continuum* 1997; 5:35-45
<http://www.altheal.org/continuum/Vol5no2.pdf>.
37. Calabrese LH. Autoimmune manifestations of human immunodeficiency virus (HIV) infection. *Clin Lab Med* 1988; 8:269-279

38. Matsiota P, Chamaret S, Montagnier L. Detection of Natural Autoantibodies in the serum of Anti-HIV Positive-Individuals. *Ann Inst Pasteur Immunol* 1987; 138:223-233
39. Bonara P, Maggioni L, Colombo G. Anti-lymphocyte antibodies and progression of disease in HIV infected patients, VII International AIDS Conference, Florence, 1991. Vol. II.
40. Tumietto F, Costigliola P, Ricchi E. Anti-lymphocyte autoantibodies: evaluation and correlation with different stages of HIV infection, VII International AIDS Conference, Florence, 1991. Vol. II.
41. Lucey D, Hendrix C, Andrzejewski C. Racial differences in anti-p24 antibody titers and total serum IGG levels in North American persons with HIV-1 infection, VII International AIDS Conference, Florence, 1991. Vol. I.
42. Madrenas J, Pottter P, Cairns E. Giving credit where credit is due: John Hunter and the discovery of the erythrocyte sedimentation rate. *Lancet* 2005; 366:2140-2141
43. Wintrobe WM, Richard Lee G, Boggs DR, et al. *Clinical Hematology*. Philadelphia: Lea & Febiger, 1981:2021.
44. Monsuez JJ, Dufaux J, Vittecoq D, Flaud P, Vicaut E. Hemorheology in asymptomatic HIV-infected patients. *Clin Hemorheol Microcirc* 2000; 23:59-66
45. Morfeldt-Manson L, Bottiger B, Nilsson B, von Stedingk LV. Clinical signs and laboratory markers in predicting progression to AIDS in HIV-1 infected patients. *Scand J Infect Dis* 1991; 23:443-9
46. Arango CA, Midani S, Alvarez A, Kubilis PS, Rathore MH. Usefulness of acute phase reactants in the diagnosis of acute infections in HIV-infected children. *Southern Med J* 1999; 92:209-13.
47. Lefrere JJ, Salmon D, Doinel C, et al. Sedimentation rate as a predictive marker in HIV infection. *AIDS* 1988; 2:63-4.
48. Cohen J. The Duesberg Phenomenon. *Science* 1994; 266:1642-1649
49. Cohen J. Duesberg and critics agree: hemophilia is the best test. *Science* 1994; 266:1645-1646
50. Detels R, English P, Visscher BR, et al. Seroconversion, sexual activity, and condom use among 2915 HIV seronegative men followed for up to 2 years. *J Acquir Immune Defic Syndr* 1989; 2:77-83
51. Moss AR, Osmond D, Bacchetti P, Chermann JC, Barre-Sinoussi F, Carlson J. Risk factors for AIDS and HIV seropositivity in homosexual men. *Am J Epidemiol* 1987; 125:1035-47
52. Montagnier L, Olivier R, Pasquier C. Oxidative stress in cancer, AIDS and neurogenerative diseases. In: Packer LP, Cadenas E, eds. *Oxidative Stress and Disease*. New York: Marcel Dekker Inc, 1998:558.