BACK

The Perth Group

November 2016

Cellular oxidation and AIDS

THE CELLULAR OXIDATIVE THEORY OF AIDS

Although the HIV theory of AIDS is almost universally accepted it is neither the first nor the only theory to account for the clinical syndrome.[1-4] However with few exceptions, after the acceptance of HIV as the causative agent, the alternative theories were discarded by most scientists and physicians, including by some of those who proposed them. When approaching a problem scientists, being human, bring along their biases. A decade before the AIDS era both Robert Gallo and Luc Montagnier made many but ultimately unsuccessful attempts to prove a viral aetiology of cancer. And since one of the two defining clinical manifestations of AIDS was the malignancy Kaposis' sarcoma (KS), it was not in the least surprising that as the AIDS era dawned these two scientists (among others) turned from studying cancer and retroviruses to studying AIDS and retroviruses. At the same time our group was also attempting to solve the problem of cancer although from a different perspective.[5-10] During this process we became aware of the oxidative properties of the agents to which individuals in the AIDS risk groups were being exposed and it was this that led us to propose the non-infectious, redox theory of AIDS as early as 1983.

The theory postulates that the level of cellular redox and its oscillations, that is, the cyclic variation between oxidation and reduction, plays a key role in both normal and abnormal cellular structure and function. The cell is regarded as being similar to a three-dimensional Belousov–Zhabotinsky reaction with superimposed oscillations in which a charge transfer in a myosin/actin redox couple plays the pivotal role.[5-9, 11-13] Diseases such as cancer, cardiovascular disorders, clotting abnormalities and ageing are the result of perturbations of cellular redox level and its oscillations. In regard to AIDS, individuals in the risk groups are exposed to many agents (semen, nitrites, recreational drugs, Factor VIII concentrates, infectious agents and the drugs used to treat them) which share the common property of oxidising the sulphydryl groups of the acid-soluble proteins in particular myosin.

The redox theory postulates that these non-infectious agents, acting alone or in combinations, are sufficient to induce the biological and pathological effects that characterise AIDS. That is, both the immune deficiency and the clinical AID syndrome, as well as the phenomena claimed to constitute proof of HIV "isolation" and hence "infection" with HIV. More importantly, the theory predicted that administration of reducing agents, in particular substances containing sulphydryl groups which were readily available and relatively cheap, would prove beneficial in both the prevention and treatment of AIDS patients.[14-20] Unfortunately the theory was rejected by *Nature*. The July 10th 1986 resubmission letter to *Nature* ends with the following: "If my [EPE] 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 illness. In my opinion this alone would more than justify its publication". Further discussion HERE [15]

The AIDS risk groups are comprised of homosexual men and heterosexual women who practise passive anal intercourse, recreational drug users, haemophiliacs and Africans. The oxidising agents to which these groups are exposed are semen (one of the most potent biological oxidants, a property which is essential for fertilisation of the ovum), intravenous and non-intravenous drugs and factor VIII concentrates. In regard to sub-Saharan Africans,

immune deficiency and AIDS diseases such as KS and tuberculosis are long endemic in Africa and have been knowingly associated with poverty and malnutrition, poor sanitation, all of which may result in biological oxidation; as well as other factors including lack of access to health care.[21-23] Endemic cellular immunodeficiencies and diseases predate the AIDS era in Africa.[24-27]

Most people know that a theory is only as good as its predictions. The HIV theory made certain predictions but after more than 30 years there is still no scientific evidence to prove even its main prediction, that is, the spread of AIDS to the general population. To the contrary. It was predicted that HIV would spread from bisexual men to female prostitutes and thence into the general population. Yet despite the prevalence of sexual intercourse and problematic use of condoms, HIV remains in the original risk groups. In fact the HIV theory cannot even account for one of the original AIDS defining diseases KS, the principle reason the retrovirus theory was pursued in the first place. Since the early 1990s all HIV experts have accepted, as we argued,[19, 28] that KS is not caused directly (nor indirectly via immune deficiency) by HIV. Yet KS remains an AIDS defining disease.[19, 28, 29] At the beginning of the AIDS era Montagnier accepted the only way to prove HIV is the cause of AIDS is to have an animal model.[30] Although no effort has been spared, no model of a retrovirus causing AIDS has been forthcoming. Indeed the only animal model that bears any resemblance to human AIDS fully supports a non-infectious *modus operandi*".[30, 31] Instead, the HIV experts delegate simian immunodeficiency virus and simian AIDS, SIV/SAIDS, as the preferred model for AIDS. However, monkeys with monkey retrovirus infections cannot prove the causative role of a human retrovirus infecting humans. Contrary to the failed predictions of the HIV theory, to date the non-infectious, cellular oxidation theory has fulfilled all its predictions.[15]

A year after the publication of our theory German researchers proved one of its main predictions, that is, the tissues of AIDS patients and those at risk will be oxidised. In fact we were very surprised that these researchers measured the acid-soluble SH because at the time and even today this method is not used to prove "oxidative stress". As we predicted they found "Blood plasma samples from HIV-1 infected persons contain…relatively low concentrations of acid-soluble thiol (i.e. decreased cysteine concentrations). The intracellular glutathione concentrations in peripheral blood-mononuclear cells (PBMC) and monocytes from HIV antibody-positive persons are also significantly decreased".[32]

In 1991 one of the authors of this paper, the late Wulf Dröge, a well known immunologist from the Institute for Immunodeficiency and Genetics at Heidelberg wrote "…we agree with Papadopulos-Eleopulos and colleagues on the basic interpretation that a distorted balance of oxidation and antioxidants may play a key part in the immunopathology of HIV/SIV infection".[33] In the same year we sent a letter to Anthony Fauci, commenting on his publication "Suppression of human immunodeficiency virus expression in chronically infected monocytic cells by glutathione, glutathione ester, and N-acetylcysteine".[34] We wrote:

"Although your findings were no surprise to us, we were suitably impressed by your well executed experimental work. You and your colleagues are apparently unaware of work published by us in which, in addition to predicting your experimental results, we advocate the use of antioxidants in the prevention and treatment of AIDS and also, based on well known facts, hypothesise:-

(a) the mechanism which produces a striking decrease in the level of a major cellular antioxidant in AIDS patients and those at risk of developing it,
(b) the mechanism of retroviral induction.

Please find enclosed our published work and a letter, which my colleagues, Dr.V .F. Turner, Prof. J.M . Papadimitriou and I submitted for publication in *Lancet*, February 1990 which was rejected. In it we give the causes and a mechanism for the decrease in cellular sulphydryl groups, GSH and total glutathione in AIDS patients and those at risk of AIDS".

In response Fauci wrote: "I have read with interest your manuscript on the role of oxidation in the pathogenesis of HIV infection. I certainly agree with your general hypothesis that oxidation and anti-oxidation may be critical factors in the control of virus expression as well as in determining certain systemic dysfunctions associated with HIV infection".[35]

The first and only study which supports the prediction of our theory – that AIDS can be prevented and treated by stopping exposure to the oxidising risk factors and by using "currently available therapeutic [antioxidants in general and SH-containing, in particular] substances" was conducted by Leonore and Leonard Herzenberg and their colleagues from Stanford University. First they sought data to show a relationship between SH concentration (reduced glutathione, GSH) and survival in AIDS patients. They showed "that GSH levels are lower in subjects with CD4 T cell counts below 200/µl (CD4<200) than in subjects at earlier stages of HIV disease: that among subjects with CD4<200, lower levels of GSB [glutathione-*S*-bimane fluorescence = a measure of the intracellular GSH concentration] predict decreased survival; and that the probability of surviving 2-3 years increases dramatically as GSB levels approach normal range".[36]

Second they conducted a randomised, double-blind placebo-controlled trial to determine if the GSH can be replenished by oral administration of N-acetylcysteine (NAC) for 8 weeks. They showed that NAC increased whole blood GSH levels and the effect was more effective in the subjects who had the lowest levels at the start of the trial. After 8 weeks all patients were offered open-label NAC for up to 8 months. Although the study was not specifically designed to discover the effect of NAC on survival they reported "To our surprise, given the relatively short time (8-32 weeks) that NAC was administered, we found that NAC ingestion was associated with substantially longer survival…The association of prolonged survival in the oral administration of NAC in this study is very dramatic…[our study] established GSH as a key determinant of survival in HIV disease".[37]

According to these researchers their study "established GSH deficiency as a key determinant of survival in HIV disease". They further commented "Therefore, our findings basically argue for the initiation of a placebo-controlled trial designed specifically to determine the therapeutic value of NAC in AIDS and/or that of other GSH-replenishing drugs. Since NAC is nontoxic and could be used where medical services are limited, our findings indicate that such a trial should be initiated as soon as possible. Other pharmaceuticals that replenish GSH should also be tried for the same purpose. In any event, the poor survival that we have demonstrated in GSH-depleted subjects with AIDS underscores the importance of finding ways to replenish GSH in these individual and ways to prevent GSH depletion in the disease…However, HIV-infected individuals would be better served if we could identify the mechanisms that underlie the GSH depletion and intervene, if possible, to prevent its occurrence. If ways could be found to do this on a long-term basis, HIV disease progression might be controlled in a way that would prevent the worst aspects of the disease…At a more immediate level certain rather simple precautions might help to slow the progress of HIV disease. Since studies presented here associate GSH depletion oxidative stress with poor survival in AIDS, we believe that HIV-infected individuals should avoid excessive exposure to sun and UV irradiations and excessive use of drugs such as acetaminophen (Tylenol) [paracetamol] that are known to deplete GSH. Physicians treating HIV-infected individuals should similarly consider exercising caution in prescribing formulations or recommending over-the-counter preparations containing such GSH-depleting drugs. These conservative measures could eliminate some of the more accessible causes

of GSH depletion and thus could potentially prevent decrease of GSH to the level that predicts death within the following 2 years".[37]

Although there are numerous publications on "oxidative stress" and HIV and AIDS, and billions of dollars spent to deploy a large number and variety of drugs to treat HIV/AIDS patients (over 30 are approved at present), no HIV expert has thought it worthwhile to initiate a trial "as rapidly as possible" of the readily available, relatively non-toxic and inexpensive substances such as NAC or other SH containing compounds which the Herzenbergs urged in 1989 and we advocated at the beginning of the AIDS era.

At the time of writing a *Medline* search using the keywords [HIV and redox] resulted in 654 publications. Our hypothesis, *Reappraisal of AIDS--is the oxidation induced by the risk factors the primary cause?* 25th March 1988 is listed 654, that is, the first such publication on record at the *National Library of Medicine*. Although we were the first to propose a non-infectious redox theory of AIDS and identified the cause and "the mechanism that underlie[s] the GSH depletion" and thus to point the way "to prevent its occurrence" and its treatment, for reasons upon which we can only speculate, with one exception,[38] no one refers to our numerous publications on this subject. This applies even to Dröge and the Herzenbergs although both were made aware of our work. It is also astonishing to read the scope of the Herzenbergs' preventative measures: avoid UV exposure and paracetamol, as if these constituted the lion's share of GSH depleting agents to which the AIDS risk groups are exposed.

Addendum: The oxidative theory, the Perth Group and Luc Montagnier

In July 1991 a full-page advertisement appeared in *Nature* in which Pasteur Mérieux Sérums & Vaccins offered a Post-Doctoral Fellowship "designed to encourage the research on examining relationships between oxidative stress and HIV infection".[39] We expected any scientist interested in this subject, including Montagnier, to be aware of our contribution to this topic.[14] Nonetheless, following the advertisement we decided to make him personally aware of our theory,[40] sending copies of the 1988 paper and two others, one of which was a recently completed draft on the pathogenesis of Kaposi's sarcoma (later published in *Medical Hypotheses*[19]) Montagnier acknowledged receipt of our material in a letter dated 25th October 1991[41] stating "I will certainly return to you after reading them" but failed to do so.

After all this, in his book *Virus*[42] Montagnier wrote: "Aside from a small number of pioneers – Dröge in Germany and Leonard and Lena Herzenberg in the United States – very few researchers have shown interest in this phenomenon ["oxidative stress"], despite its fundamental role in the illness…The phenomenon is massive, and occurs at an early stage". As to its cause, "In addition to the virus, [HIV], all opportunistic agents can likewise contribute to oxidative stress, and from the very onset of infection, so can mycoplasmas".

Not only did Montagnier forget or ignore our group and our explicit evidence that cellular oxidation plays a "fundamental role in illness", but, like the Herzenbergs, he forgot or ignored our list of oxidising agents to which the AIDS-risk patients are exposed. And that such exposures occur long before the patients develop HIV and opportunistic infections. Montagnier also seems unaware that it is oxidation which leads to the opportunistic infections. After all, it is his compatriot Pasteur who taught us "Bernard was right; the pathogen is nothing; the terrain is everything".

Montagnier seems to have also forgotten that as far back as January 1986 he wrote "replication and cytopathic effect of LAV [HIV] can only be observed in activated T4 cells. Indeed, LAV infection of resting T4 cells does not lead to viral replication or to expression of viral antigen on the cell surface [that is, infection], while stimulation by lectins [mitogens] or

antigens of the same cells results in the production of viral particles, antigenic expression and the cytopathic effect".[43] And that in our 1992 "Oxidative Stress, HIV and AIDS" paper[18] which he must have also read, we presented evidence that cellular activation is an oxidative process and that oxidation is a prerequisite to detect the phenomena claimed to prove HIV infection. In other words, no activation, namely no oxidation = no infection.

We have also directly challenged Montagnier to defend "HIV" and the HIV theory and refute our theory in two scientific publications in 2004[44] and 2006.[45] Although this matter was now in the public domain again Montagnier did not respond. Remarkably nowadays Montagnier accepts that in Africa the cause of AIDS is oxidation and advocates treating AIDS patients with antioxidants.[46, 47] If Montagnier accepts oxidation as the cause of AIDS in Africa then why not in the other risk groups where individuals are exposed to strong oxidants?[14] Nonetheless, since he accepts that in Africans (a) AIDS is due to oxidation; (b) oxidation is due to malnutrition (poverty[48]); it follows that the cause of AIDS in the vast majority of people is not HIV. This being the case, why not in the remaining minority?

References and Notes

1. Sonnabend J. The Etiology of AIDS. AIDS Res 1983 1:1-12.
2. Lauritsen J, Wilson H. Death Rush: Poppers & AIDS. Pagan Press New York, 1986.
3. Root-Bernstein RS. Rethinking AIDS-The tragic cost of premature consensus. New York, Macmillan, Inc., 1993.
4. Root-Bernstein RS. Five myths about AIDS that have miscredited research and treatment. Genetica 1995 95:111-132.
5. Papadopulos E, Stanford R. Mitosis and Hyperthermia A Hypothesis. Australas Radiol 1977 21:5-20.
6. Papadopulos-Eleopulos E. The Role of myosin and actin in carcinogenesis: an hypothesis. Speculations in Science and Technology 1981 4:39-44. http://leederville.net/links/SpecSciTech1981.pdf
7. Papadopulos-Eleopulos E. A Mitotic Theory. J Theor Biol 1982 96:741-758. http://www.theperthgroup.com/EPE/MitoticTheory.pdf http://goo.gl/uwp7CU
8. Papadopulos-Eleopulos E. Carcinogenesis. Med J Aust 1984 140:180-181.
9. Papadopulos-Eleopulos E, Page BA, Causer D, Turner VF, Papadimitriou JM. Cancer and epigenetic reversion--the fundamental role of redox. Am J Pathol 2007 171:1726-1727. http://theperthgroup.com/EPE/AMJLetterNov07.pdf
10. Turner VF. User friendly explanation of the redox theory of cellular function. 2005. http://theperthgroup.com/EPE/RedoxTheory.pdf
11. Papadopulos-Eleopulos E, Fox RA. Effects of dimethyl sulfoxide on cerebral arteries. Stroke 1987 18:812.
12. Papadopulos-Eleopulos E, Knuckey N, Dufty A, Fox RA. Evidence that the redox state has a role in muscular contraction and relaxation. Physiol Chem Phys Med NMR 1985 17:407-412. http://theperthgroup.com/EPE/MuscleRedox.pdf
13. Papadopulos-Eleopulos E, Knuckey N, Dufty A, Fox RA. Importance of the redox state in vasoconstriction induced by adrenaline and serotonin. Cardiovasc Res 1989 23:662-665. http://theperthgroup.com/EPE/Serotonin.pdf
14. 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/EPEMedHyp1988.pdf
15. Papadopulos-Eleopulos E. Looking back on the oxidative stress theory of AIDS. Continuum 1998 5:30-35. http://theperthgroup.com/CONTINUUM/LookingBackContinuum.pdf
16. Papadopulos-Eleopulos E, Hedland-Thomas B, Causer DA, Dufty AP. An alternative explanation for the radiosensitization of AIDS patients. Int J Radiat Oncol Biol Phys 1989 17:695-697.
17. Papadopulos-Eleopulos E, Hedland-Thomas B, Causer DA, Turner VF, Papadimitriou JM. Changes in thiols and glutamate as consequence of simian immunodeficiency virus infection. Lancet 1991 338:1013-1014. http://theperthgroup.com/SCIPAPERS/SIVMac.pdf
18. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Oxidative stress, HIV and AIDS. Res Immunol 1992 143:145-148. http://www.theperthgroup.com/SCIPAPERS/EPEOxstressHIVAIDS.pdf
19. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Kaposi's sarcoma and HIV. Med Hypotheses 1992 39:22-29. http://www.theperthgroup.com/SCIPAPERS/EPEMedHyp1992.pdf
20. Turner VF. Reducing agents and AIDS--Why are we waiting? Med J Aust 1990 153:502. http://www.theperthgroup.com/SCIPAPERS/RedAgent.pdf
21. Chandra RK. Nutrition and immunology: from the clinic to cellular biology and back again. Proc Nutr Soc 1999 58:681-683.
22. Chandra RK, Gupta S, Singh H. Inducer and suppressor T cell subsets in protein-energy malnutrition: Analysis by monoclonal antibodies. Nutrition Research 1982 2:21-26.

23. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Alfonso H, Page BAP, Causer D, Mhlongo S, Fiala C, Miller T, Brink A, Hodgkinson N. Mother to Child Transmission of HIV and its Prevention with ATZ and Nevirapine. Perth, The Perth Group, 2001, pp. 204.
24. Konotey-Ahulu. AIDS in Africa: Misinformation and Disinformation. Lancet 1987 ii:206-207.
25. Papadopulos-Eleopulos E, Turner VF. Deconstructing AIDS in Africa. The Independent Monthly 1994 50-51.
http://theperthgroup.com/POPPAPERS/IndependentMonthly.pdf
26. Papadopulos-Eleopulos E, Turner VF. Reconstructing AIDS in Africa-Reply to Kaldor and Ashton. The Independent Monthly 1995 February:23-24.
http://theperthgroup.com/POPPAPERS/IndependentMonthly.pdf
27. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Bialy H. AIDS in Africa: Distinguishing fact and fiction. World Journal of Microbiology and Biotechnology 1995 11:135-143.
http://www.theperthgroup.com/SCIPAPERS/EPEWorldJMicro&BiotechOCR1995.pdf
28. As far as AIDS is concerned, KS is almost exclusively a disease of homosexual men. This is inexplicable given that infectious agents are not known to distinguish between humans on the basis of sexual orientation. In 1990 published data showed that KS occurs in homosexual men who are neither immune suppressed nor HIV positive. This means HIV cannot account for one of the two principal AIDS diseases for which the theory was proposed. Yet KS is still listed as an AIDS indicator disease.
In 1988, the Perth Group paper arguing that HIV does not cause Kaposi's sarcoma was thrice rejected by the *Medical Journal of Australia* on the advice of an "established expert". The reviewer stated, "The author tries to argue that Kaposi's' sarcoma cannot be caused by HIV infection, and that therefore AIDS is not due to HIV infection. The arguments put forward by the author are quite unsatisfactory, and are not supported by even a desultory reading of the literature quoted. In addition, the author fails to examine the body of epidemiological, immunological and cellular literature concerning the pathology, pathogenesis and clinical associations of this fascinating manifestation of HIV infection". Yet this is the very "epidemiological, immunological and cellular literature" which eventually led the "established experts" to accept that "this fascinating manifestation of HIV infection", is not caused by HIV infection.
See:
Friedman-Kien AE, Saltzman BR, Cao YZ, Nestor MS, Mirabile M, Li JJ, Peterman TA. Kaposi's sarcoma in HIV-negative homosexual men. Lancet 335: 168-9, 1990.
Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Kaposi's sarcoma and HIV. Medical Hypotheses 39: 22-9, 1992.

29. Beral V, Peterman TA, Berkelman RL, Jaffe HW. Kaposi's sarcoma among persons with AIDS: a sexually transmitted infection? Lancet 1990 335:123-128.
30. Vilmer E, Rouzioux C, Vezinet Brun F, Fischer A, Chermann JC, Barré-Sinoussi F, Gazengel C, Dauguet C, Manigne P, Griscelli C, Montagnier L. Isolation of new lymphotropic retrovirus from two siblings with Haemophilia B, one with AIDS. Lancet 1984 I:753-757.
31. Ter-Grigorov VS, Krifuks O, Liubashevsky E, Nyska A, Trainin Z, Toder V. A new transmissible AIDS-like disease in mice induced by alloimmune stimuli. Nat Med 1997 3:37-41.
32. Eck HP, Gmunder H, Hartmann M, Petzoldt D, Daniel V, Droge W. Low concentrations of acid-soluble thiol (cysteine) in the blood plasma of HIV-1-infected patients. Biol Chem Hoppe Seyler 1989 370:101-108.
33. Droge W. Changes in thiols and glutamate as consequence of simian immunodeficiency virus infection. Lancet 1991 338:1013-1014.
34. Kalebic T, Kinter A, Poli G, Anderson ME, Meister A, Fauci AS. Suppression of human immunodeficieny virus expression in chronically infected monocytic cells by

glutathione, glutathione ester, and N-acetylcysteine. Proc Natl Acad Sci U S A 1991 88:986-990.
35. Correspondence between EPE and Anthony Fauci. 1991. http://leederville.net/links/PasteurLetters/EPE&Fauci-letter.pdf
36. Herzenberg LA, De Rosa SC, Dubs JG, Roederer M, Anderson MT, Ela SW, Deresinski SC, Herzenberg LA. Glutathione deficiency is associated with impaired survival in HIV disease. Proc Natl Acad Sci U S A 1997 94:1967-1972.
37. Herzenberg L, De Rosa S, Herzenberg L. Low Glutathione Levels in CD4 T Cells Predict Poor Survival in AIDS: N-Acetylcysteine May Improve Survival. In Oxidative stress in cancer, AIDS, and neurodegenerative diseases. Edited by Marcel Dekker, 1997, p. pp. 379-388.
38. Sharma B. Oxidative stress in HIV patients receiving antiretroviral therapy. Curr HIV Res 2014 12:13-21.
39. Pasteur Mérieux Sérums & Vaccins - advertisement offering post-doctral Fellowship in "Oxidative Stress and HIV Infection". Nature 1991 252:Classified Section page 14. http://leederville.net/links/PasteurLetters/PasteurAd18July1991.pdf
40. E Papadopulos-Eleopulos' letter to Luc Montagnier. 1991 http://leederville.net/links/PasteurLetters/1EPE-LucM7Oct1991.pdf
41. Luc Montagnier's letter to E. Papadopulos-Eleopulos. 1991. http://leederville.net/links/PasteurLetters/2LucM-EPE25Oct1991.pdf
42. Montagnier L. Virus. New York, WW Norton & Company Inc, 2000,
43. Klatzmann D, Montagnier L. Approaches to AIDS therapy. Nature 1986 319:10-11.
44. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Page BA, Causer D, Alfonso H, Mhlongo S, Miller T, Maniotis A, Fiala C. A critique of the Montagnier evidence for the HIV/AIDS hypothesis. Med Hypotheses 2004 63:597-601. http://www.theperthgroup.com/SCIPAPERS/MHMONT.pdf
45. Papadopulos-Eleopulos E, Page BA, Causer D, Turner VF, Papadimitriou JM, Alfonso H. Would Montagnier please clarify whether HIV or oxidation by the risk factors is the primary cause of AIDS? Med Hypotheses 2006 67:666-668. http://theperthgroup.com/SCIPAPERS/PGMontOSMH2006.pdf
46. Montagnier L. Nobel Lecture. 2008. http://www.nobelprize.org/nobel_prizes/medicine/laureates/2008/montagnier_lecture.pdf
47. Passwater RA. Antioxidant Nutrients and AIDS: Exploring the Possibilities--An interview with Dr. Luc Montagnier, the discoverer of HIV. 1995. http://www.healthy.net/scr/interview.aspx?Id=187
48. http://apps.who.int/iris/bitstream/10665/43256/1/WHO_HTM_TB_2005.352.pdf http://www.who.int/whr/1995/media_centre/executive_summary1/en/