NOTE: At the 2006 International AIDS Conference Professor John Moore presented a session entitled "HIV Science and Responsible Journalism". In his presentation Professor Moore referred to Eleni Papadopulos, Valendar Turner and the Perth Group.

http://aidstruth.org/hiv-science-and-responsible-journalism.php

Soon after we sent Professor Moore this response and also entered into some brief correspondence via email. This is appended at the end of this file.

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September 23rd 2006

Dear Professor Moore,

Since in recent years including the 16th International AIDS Conference you have had so much to say about the Perth Group, we would like to put a few things straight.

Kind regards,

Eleni Papadopulos-Eleopulos

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1. Let us make it clear that we are not AIDS denialists. That is, we do not deny that in 1981 a syndrome involving a high frequency of KS and a number of opportunistic infections was identified in gay men and subsequently became known as AIDS. What we are doing and have been doing from the very beginning is to question the accepted cause of AIDS and to put forward an alternative theory for the cause of AIDS which has a number of well-defined predictions, most of which have been satisfied.[1]

2. You said: "**Any one, man or woman, who's persuaded that safe sex or using clean needles is not necessary and then becomes HIV infected and dies of AIDS, the person advising them inappropriately bears responsibility.**"

In our publications we have stressed that all the evidence shows passive anal intercourse plays a key role in the causation of AIDS. This being the case safe sex is extremely important in its prevention. However ten years ago the "HIV" experts claimed that "HIV" can be eliminated and that AIDS can be treated with HAART. The acceptance of this claim by some led to an increased frequency of unprotected sex.

In our publications we not only stressed the need for clean needle usage but according to our theory no recreational drugs should be used no matter how they are delivered be it either by needles clean or dirty, or orally.

3. You said: "**Anyone persuaded not to take antiretrovirals and use instead alternative medicines — lemon and garlic, potatoes and whatever — is also dying unnecessarily.**"

Since in our view at present no evidence exists that AIDS is caused by a retrovirus, we see no reason for AIDS patients to be treated with antiretroviral drugs. We did write a critical analysis on the use of AZT as an antiretroviral agent when we showed that, given its pharmacological properties, it is not possible for it to have an antiretroviral effect.[2] We have also presented evidence that AZT and nevirapine do not prevent mother-to-child transmission.[3, 4] However, we never advised that antiretroviral drugs should never be prescribed since up till now the possibility had not been excluded that they may have clinical benefits acting by means other than as antiretroviral agents. However, given the latest publication on HAART, this may not be the case.[5]

At the very beginning of the AIDS era we put forward alternative ways of preventing and treating AIDS.[6] However, nowhere in our publications have we even suggested that AIDS can be treated by "lemon and garlic, potatoes and whatever".

4. You said: "**Anyone persuaded not to be screened for HIV status and deprived of the chance of treatment or counselling dies unnecessarily.**"

The only test for screening for "HIV" status is the antibody test. In our publications we have never said that either blood used for transfusion or patients belonging to the AIDS risk groups should not be tested. However, we do claim that up to now, no evidence exists that a positive "HIV" antibody test proves "HIV" infection.[7] All the presently available evidence shows that a positive test may represent nothing more than a non-specific indicator of altered homeostasis connoting a propensity to develop particular diseases. Clinical medicine has an abundance of non-specific tests and their non-specificity does not preclude their utility.[4]

5. You said: "**And infants whose HIV infected mothers listen to AIDS denialists never got the chance to make their own decisions.**"

How can a 3-year old infant make his or her own decision?

6. You said: "**Now the AIDS denialists abuse the peer-reviewed literature. They abuse science. They cite only old, long refuted papers as if they still represented state of the art knowledge, which they don't. So they argue that TB, malaria, leprosy, pregnancy cause false positive tests in an HIV assay. Now this is simply not true of the modern tests, and it's questionable how significant it was with the early generation of assays.**"

Which "old, long refuted papers" are you referring to? In particular, which "old, long refuted papers" regarding "HIV" antibody test specificity have we cited to back our claim that the specificity of the "HIV" antibody test has not been determined?

In a book *Retroviral Testing and Quality Assurance, Essentials for Laboratory Diagnosis*[8] written in 2005 by three of the "HIV" experts in "HIV" testing, Niel Constantine (Professor of Pathology, Department of Pathology, University of Maryland School of Medicine & Director Clinical Immunology Laboratory, University of Maryland Medical Center & Laboratory of Viral Diagnostics, Institute of Human Virology, Baltimore, Maryland, USA), Rebecca Saville (Food and Drug Administration, FDA/CDER/OND/ODEIV/DSPIDP, Rockville, Maryland, USA), Elizabeth Dax (Director, National Serology Reference Laboratory, Australia. A World Health Organization Collaborating Centre on HIV/AIDS, Fitzroy, Victoria, Australia), on page 94 one reads "Among the medical conditions that are suspected or occasionally known to produce false-positive screening test results are as follows:

- Malaria
- Syphilis
- Pregnancy
- Hypergammaglobulinemia, renal failure, liver disease
- Some parasitic diseases and viral diseases (e.g., influenza)
- Autoantibodies (autoimmune diseases)
- HIV vaccination (becoming a major cause)
- Transfusions (usually multiple)"

NOTE:

1. the same conditions are cited on page 194 for causing "indeterminate" (false-positive) Western Blot tests.
2. No mention is made of Mycobacteria in general or TB in particular (see below).
3. In countries such as South Africa, a positive screening test is considered proof for "HIV" infection.

Regarding the Western blot, on page 197 the authors wrote: "Contrary to what most individuals believe, false-positive Western blot results do occur, although this is not common…This is because the original Western Blot criteria [in fact the criteria introduced in 1987 by some laboratories were not the first criteria] included the need for reactivity to each of the three gene products (gag, pol, and env), but

when these criteria were changed in 1993 to a less stringent criteria (to the CDC criteria that dropped the requirement for reactivity to p31) more false positives occurred. [Was the "HIV" p31 dropped because there is unambiguous proof that p31 is a cellular protein?[9, 10]]. This change was instituted in an attempt to decrease the number of indeterminate results…In a report in 1998, it was documented that false-positive Western blot results occur to a higher degree in low-risk populations. Of 421 blood donors who were positive for HIV-1 by Western blot and who lacked reactivity to p31 (polymerase antigen), 39 (9.3%) met the criteria of possibly being falsely positive."

On page 184 the authors wrote: "HIV serologic confirmatory tests should more correctly be called supplemental tests…The purpose of serologic confirmatory tests is to rule out false-positive results by screening tests, not to confirm that a person in unequivocally infected with HIV or to confirm that a person is negative for HIV."

Indeed, a positive Western blot cannot be considered as proof for "HIV" infection.[7] Especially when one considers that even today the criteria for a positive test varies from country to country, from laboratory to laboratory within the same country. Also the criteria for a positive test have changed over time in a totally arbitrary fashion. Initially, the presence of one reactive band either p24 or p41 was considered proof for "HIV" infection. When it was realised that most of us would test positive at one time or another more stringent criteria requiring more than one band were introduced. Then, as the above authors pointed out, when "these criteria were changed in 1993 to a less stringent criteria (to the CDC criteria that dropped the requirement for reactivity to p31) more false positives occurred. "This change was instituted in an attempt to decrease the number of indeterminate results". Given the consequences on being diagnosed "HIV" positive, it is quite bizarre that the criteria can be changed in a totally arbitrary fashion.

The problem is not that TB, malaria, leprosy, pregnancy and other conditions cause false positive tests in an "HIV" assay. The problem is there is still no evidence that a positive result in an antibody test in any individual, no matter how many reactive bands there are, proves "HIV" infection. The only way to determine the specificity of the antibody tests is to use a gold standard which for the "HIV" antibody tests is "HIV" itself. However, to date nobody has determined the specificity of the "HIV" antibody test using the gold standard and in fact two of the best known AIDS/"HIV" experts, Blattner and Mortimer accept that no such gold standard exists.[11, 12]

The "excuse" of "old, long refuted papers" is one of the most often used arguments by "HIV" experts in advising rejection of our papers by scientific journals.

In 1988 we submitted a paper to the *Medical Journal of Australia*.[13] This argued that HIV does not cause Kaposis' sarcoma and it was thrice rejected on the advice of "established experts". Among others, including the use of "old references", one of the reviewers stated, "The author tries to argue that Kaposis' sarcoma cannot be caused by HIV infection, and that therefore AIDS is not due to HIV infection. [In the paper we did not argue about what causes AIDS but only argued the cause of KS]. 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 later on, even a small fraction of this "epidemiological, immunological and cellular literature" led the "established experts" to conclude that "this fascinating manifestation of HIV infection", is not caused by HIV infection.

Another common outcome is that "HIV" experts advise rejection of our papers for no scientific reason. For example, in 2000 we submitted a paper on antibody testing to the International Journal of STD and AIDS. Please note that not a single scientific fact addressed by us in this paper is mentioned let alone discussed or refuted.

INTERNATIONAL JOURNAL OF STD & AIDS Referee's Report

Author: E Papadopulos-Eleopulos
Title: Are "HIV" antibodies caused by a retroviral infection?
Manuscript No: 04215

Please type comments for transmission to author on this sheet: DO NOT SIGN

Dr. Valendar F. Turner and several of the other authors are members of the "Perth group" of "HIV / AIDS dissidents",

The Perth Group argues (http:/ /www.theperthgroup.com):

- *That AIDS and all the phenomena inferred as "HIV" are induced by changes in cellular redox brought about by the oxidative nature of substances and exposures common to all the AIDS risk groups*
- *That the cessation of exposure to oxidants and/or use of anti-oxidants will improve the outcome of AIDS patients.*
- *That AIDS will not spread outside the original risk groups*
- *That the pharmacological data prove AZT cannot kill "HIV" and AZT is toxic to all cells and may cause some cases of AIDS.*

This paper discusses HIV antibody tests and the authors conclude that there is no scientific basis for the claim that HIV antibody detection is specific for infection with a retrovirus.

Essentially most of the arguments in this paper are published on their website and some of it has actually been published in various scientific journals.

These are extreme and unconventional views. The use of evidence is highly selective and I think misleading. I do not think that there is any merit in further recycling of this material in the International Journal of STD and AIDS.

7. You said: "**They highlight legitimate scientific uncertainties within AIDS research as evidence for incompetence or worse. So the fact that HIV pathogenesis knowledge evolves over time is twisted in a way that says, "Well, you were wrong, therefore you must always be wrong.**"

The problem is not scientific uncertainties but that there has never been any published proof that "HIV" causes AIDS irrespective of the mechanism. That is, it has never been proved that "HIV" induces immune deficiency (destroys the T4 cells) which in turn leads to the clinical syndrome. At the beginning of the AIDS era, evidence rapidly accumulated that some of the patients with AIDS or at risk of AIDS, had lower than normal numbers of T4 cells. The same patients were shown to have a higher than normal number of T8 cells. It was postulated then that the decrease in T4 cells was due to their killing by "HIV". Since then an army of researchers spared no effort trying to determine the mechanism of "HIV pathogenesis". This postulate is astonishing.

Let us remind ourselves (mainly for the benefit of others as you being an immunologist know this) of the history of the T4/T8 cells. In 1974, a group of researchers observed that when normal lymphocytes were cultured with T-cells from hypogammaglobulinaemic patients in the presence of PWM, the synthesis of immunoglobulin (antibodies) by the normal lymphocytes was depressed by 84% to 100%. They put forward the hypothesis "that patients with common variable hypogammaglobulinemia have circulating suppressor T lymphocytes that inhibit B-lymphocyte maturation and immunoglobulin synthesis".[14] By 1980 it was accepted that there are two subsets of T-lymphocytes, the T8 subset (T-suppressor cells) which "suppresses the proliferate response of other T-cells and B-cells immunoglobulin production and secretion" and the T4 cells (helper subsets) which produce "a variety of helper factors that induce B cells to secrete immunoglobulin and all lymphocyte subpopulations (T,B and null) to proliferate".[15]

By the beginning of the AIDS era, evidence existed that under certain conditions (which are satisfied in "HIV" cultures and AIDS patients) there is a phenotypic change of T4 cells to T8 cells, a fact known to both Montagnier and Gallo.[16] In 1984 Montagnier and his colleagues wrote: "this phenomenon [decrease in T4 cells] could not be related to the cytopathic effect" of HIV but is "probably due to either modulation of T4 molecules at the cell membrane or steric hindrance of antibody-binding sites".[17, 18] In 1983 Zagury (one of Gallo's collaborators) and his colleagues wrote: ""Testing functional properties we found that NK activity was mediated not only by T10+ cells but also, in some cases, by T4+ and T8+ cells. Moreover, TCGF production, which may reflect helper activity, was mediated not only by T4+ cells. Only the cytotoxic (CTL) activity seems to be confined to the T8 phenotype. Thus, it appears that T antigens, which seemed to be molecular markers of differentiation, are not markers for terminal differentiation and do not always reflect defined functional properties".[19] In 1988 Göran Möller (an immunologist from the University of Stockholm) wrote: "There are three good and several not so good reasons for questioning the existence of suppressor T cells as a separate T cell subpopulation".[20] Commenting on Möller's editorial, researchers from the Pasteur Institute wrote: "It follows that the difference between these two cell populations concerns their repertoires and, in consequence, their maturative or activation stages, possibly their differential mechanisms of activation... As discussed here,

even primary populations of lymphocytes may follow functional rules in vitro that depart substantially from those operating in vivo, and cells may look and function differently simply because they are either connected or isolated. In essence, and this is both more interesting and difficult to approach, it seems unavoidable that systems (such as the immune) are more than the sum of isolated clonal activities".[21] In a 1981 commentary in *JAMA* entitled: "OKT3, OKT4, and all that", one reads: "The T- and B-cell measurers-having run through the sick, the elderly, the young, the pregnant, the bereaved-had finally run out of diseases. Each condition was the subject of many reports; so that now, to give but one example, we can conclude with some assurance that T-cell numbers are up, down, or unchanged in old folks….And now it's starting all over again, this time with T-cell subsets. Think, dear reader, and grieve, dear editor, about how many investigators are at this very moment measuring T-cell subsets in systemic lupus erythematosus, in rheumatoid arthritis, in solid tumours (all different sorts - one article for each), in lymphomas, in pneumonia, after surgery, after burns, after trauma, in asthma, in cirrhosis, in Crohn's disease, in glomerulonephritis, in myositis, in familial Mediterranean fever, in leprosy, in Dengue fever, after cardiac transplants, and so on. Meanwhile others will be out measuring blacks, whites, Orientals, native Americans, men, women, children, babies, old folk, astronauts, and laboratory technicians. Cells will be garnered and measured from blood, from lungs, from kidneys, from liver, and from CSF and ascitic fluid…What can be done to stanch the anticipated outflow?…We might legitimately ask, why fight? Why not let us unimaginative immunologists publish to our heart's content? I will ignore the obvious economic arguments for fear that they might be taken seriously. My strongest argument is this: Measurement of T and B cells and their subsets in diseases has no clinical meaning…There is a feeling about that T- and B-cell numbers mean something, an immunologic equivalent of an SGOT level or creatinine clearance…Nonimmunologists have naturally assumed that any subject occupying so much journal space must be relevant in some way – a logical but incorrect assumption".[22] Experimental depletion of T4 cells in mice used as models for systemic lupus erythematosus in humans did not lead to increased frequencies of neoplasms, nor did mice "develop infectious complications, even though they were housed without special precautions". In fact mice with low T4 cell numbers had "prolonged life".[23] It is also of interest that despite the indispensable role attributed to T4 and T8 lymphocytes in antibody production (helper and suppressor respectively), AIDS patients in the presence of low numbers of T4 cells and high numbers of T8 cells, have increased levels of serum gammaglobulins, and are not hypogammaglobulinaemic as might be expected. Also, although human umbilical cord T-cells produce suppressor factors(s), the factor(s) is produced by T8- (T4+) not T8+ cells[24] According to the "HIV" theory of AIDS, the diseases which constitute the acquired immune deficiency syndrome, the S in AIDS, are the consequence of the low T4 cell number, (AID), induced by "HIV". However, according to the same "HIV" experts these diseases continue to appear even after HAART induces "immune restoration" but now the diseases are "Immune Restoration Disease (IRD)", not AIDS.[25] Thus, T4 and T8 cells do not seem to possess the generally accepted functions attributed to them.[16]

8. You said: "**Science evolves, but the denialists are stuck in a time warp. They cherry pick what suits them. Preferential citation is what it's known as in the technical language. They ignore the much greater weight of contradictory evidence and they wilfully or incompetently misrepresent the information reported in individual papers. The Perth group did this in Nature in a study on maternal, mother-to-child, transmission in Rwanda.**"

In our publications we have cited hundreds upon hundreds of papers published by "HIV" experts. The fact is that when you write a paper and more so when you write a letter, you have to limit the references to the most crucial regarding that subject. In the case of the use of nevirapine to inhibit mother-to-child transmission, the Rwanda study is considered to be the definitive study. The *Nature* correspondence is based on a large, detailed, critical analysis of mother-to-child transmission in which hundreds of references are cited.[4] We wrote a response to *Nature* in regard to a comment published in *Nature* that the letter had misrepresented the Rwanda data but *Nature* would not publish this response. It is posted at www.theperthgroup.com/LATEST/Geffen.html

9. You said: "**But the denialists don't publish any of their own work. They simply criticize, ignorantly, the work of scientists who do.**"

Our publications contain a lot of original ideas and work. Although it is not necessary for us to perform experiments based on our ideas, we would have preferred to do them. However, due to lack of funds we have been unable to perform our original experiments. Science has progressed on the basis of new ideas and theories being presented many times by either one person or a group of people and then experiments being carried out by either another person or group of people. In fact, some of the most important progressions in science were based on ideas of people who never performed the experiments themselves.

10. You said: "**Now what are their core beliefs? The core beliefs tend to be somewhat different because different sub-cliques of denialists differ in what they choose to emphasize. One of the more bizarre episodes was the Perth group claims that HIV simply does not exist; whereas Duesberg accepts that HIV exists but believes it's harmless. So when the Perth group put out a competition on their website with a cash prize for anyone who could prove that HIV exists Duesberg actually claimed the price. It gets that silly.**"

There are many bizarre episodes in "HIV"/AIDS research but our scientific disagreement with Peter Duesberg is not one of them. In our publications we have never claimed that "HIV simply does not exist". We have claimed that the presently available data does not prove its existence. We have never "put out a competition on" our website offering "a cash prize for anyone who could prove that HIV exists". In fact, at that time (1996) we did not even have a website. The prize was offered by *Continuum* magazine, not by the Perth Group. When Peter Duesberg claimed it, we challenged his claim. Peter claimed that the existence of the "HIV infectious

molecular clone" proves that "HIV" exists. However, he never gave any evidence for the existence of the "HIV infectious molecular clone". Peter's argument that the existence of the "HIV infectious molecular clone" proves that "HIV" exists was also used by Brian Foley in the *British Medical Journal* Online debate. Like Peter, Brian Foley ultimately was not able to present any evidence for the existence of the "HIV infectious molecular clone". Our repeated request to Brian Foley remains unanswered. So were many other repeated requests including providing references with evidence which demonstrates the specificity of the "HIV" antibody tests, sexual transmission of "HIV" and that the "HIV" proteins are coded by the "HIV" gag, pol and env genes. When it was seen that neither Brian Foley nor any other participant in the debate could provide such evidence, instead of coming to their rescue by providing such evidence, Wain-Hobson, Brian Foley and you attempted to stop the debate. Ultimately you succeeded.

Let us give you a few of the "bizarre episodes" in "HIV"/AIDS research:

(a) One of the most important morphological characteristics of retroviruses is the presence of spikes (knobs) on the particle's surface. There is agreement among all the proponents of the "HIV" theory of AIDS that the "HIV" particles' spikes (gp120) is absolutely necessary for infectivity. In 1997 you wrote: "HIV infection of CD4+ cells is initiated by an interaction between its surface glycoprotein gp120, and the cellular antigen CD4+".26 In Montagnier's book Virus27 (2000) one reads: "Particles of HIV are shaped like little spheres, each with roughly eighty rounded projections shaped like pegs. Each peg contains three or four molecules of a large protein, gp120, which has a strong affinity for the receptors (now called CD4) or T4 lymphocytes". In 1991, you wrote: "On the virus surface, mature gp120/gp41 heterodimers are grouped together into oligo-meric spikes that are clearly visible in electron micrographs".26 In the 2005 Constantine et al book, one reads: "The gp120 antigen, expressed from instruction from one of the env genes, is a major component of the 72 knobs or spikes of the external envelope of HIV-1…" To date, nobody has produced electron-micrographic (EM) evidence for the existence of such spikes on the "HIV" particles.www.theperthgroup.com/LATEST/ZhuNatureRejected.doc In a paper you published in 199228 you said the spikes are lost very rapidly after they are released, that immediately after release there are approximately 0.5 spikes/particle. But you also added: "It was possible that structures resembling knobs might be observed even when there was no gp120 [spikes] present, i.e. false positives".

(b) In the early 1970s Gallo reported reverse transcription in normal uninfected but mitogenically stimulated lymphocytes.29 Barre-Sinoussi and Chermann, the principle and second authors of the 1983 paper in which the existence of "HIV" was claimed to be proven, were fully aware that reverse transcription is present in normal cells.30, 31 In 1975, an International Conference on Eukaryotic DNA polymerases defined DNA polymerase gamma as the cellular enzyme which "copies An.dT_{15} with high efficiency but does not copy DNA well".32 Yet in 1983 transcription of An.dT_{15} in a stimulated culture containing lymphocytes from a patient at risk of AIDS, was considered proof for "HIV" isolation! The detection of the same reverse transcriptase activity in a consecutive culture was considered proof for "HIV" transmission. At present most, if not all, molecular biologists are of the opinion that a significant part of the human genome was obtained by reverse transcription of RNA into DNA. Nowadays, the non-specificity of reverse

transcriptase is known even to the general public in the form of magazine reports evaluating the investment potential of biotechnology stocks (Pachacz M. No need to be phased, Shares Magazine, February 2001, p 28-32).
Yet in a completely "off the wall" manner, "HIV" experts are still using reverse transcription to prove "HIV" infection and even to quantify it as one can read in the 2005 Constantine *et al* book.

c) According to Montagnier, "analysis of the proteins of the virus demands mass production and purification. It is necessary to do that".33 In 1983 Montagnier and in 1984 Gallo claimed to have obtained "purified" "HIV" but did not publish proof for their claims. In his "purified virus", Montagnier found a p25 protein (now known as p24), which reacted with his patient's serum and claimed that this is specific "HIV" protein. He also found a p45 protein which also reacted with his patient's serum but said that this protein is cellular actin (the molecular weight of actin is 41,000). In 1984, in his "purified virus", Gallo found both of these proteins to react with AIDS patients' sera and claimed that p41 was the most specific "HIV" protein. In 1997 in an interview Montagnier gave to the French Journalist Djamel Tahi, he stated: "I repeat we did not purify". "Gallo?…I don't know if he really purified. I don't believe so". In fact Montagnier admitted that in what he and his colleagues called "purified virus", even after "Roman effort", they could not find any particles which even had morphological characteristics of retroviruses. "We saw some particles but they did not have the morphology typical of retroviruses. They were very different".33 This is as good a proof as anybody can get that Montagnier's p24 was not an "HIV" protein or a protein of any other retroviruses. Yet Montagnier's p24 is considered to be the most specific "HIV" protein. Reaction of antibodies directed against this protein with antigens in cell cultures is considered proof for "HIV" isolation! According to the 2005 Constantine et al book, "The best antigen preparations to detect established HIV infection are viral lysates…". In 1997 some of the best "HIV" experts acknowledged that the 'Virus' "used for biochemical and serological analysis or as an immunogen is frequently prepared by centrifugation through sucrose gradients" and that in no study "has the purity of the virus preparation been verified".10, 34 In 2003 we asked Gallo for evidence of "HIV purification. He responded "Montagnier subsequently published many EM pictures of purified HIV particles, as, of course, we did in our first papers. You have no need of worry. The evidence is obvious and overwhelming". Charles Dauguet, the Pasteur Institute electron-microscopist, was interviewed by Djamel Tahi in December 2005. He said that at no time did they have purified virus, all he could find in the "purified virus" was "cellular debris".

(d) According to many "HIV" experts, including yourself, "gp120 and gp41 [are] produced by cleavage of a common precursor gp160" which is an "HIV" protein present in the infected cells but not the viral particles. That is gp160 cannot be present in the purified virus. Despite this, when "purified virus" is tested against AIDS sera, gp160 bands are observed. According to researchers from New York this is because gp120 and gp160 are oligomers of gp41,35 and not distinct "HIV" proteins. They stressed: "….some clinical specimens may have been identified erroneously as seropositive, on the assumption that these bands reflected specific reactivity against two distinct viral components and fulfilled a criterion for true or probable positivity. The correct identification of these bands will affect

the standards to be established for Western Blot positivity; it may necessitate the reinterpretation of published results". Nobody took any notice of this warning. In fact according to the African criteria for a positive WB, two glycoprotein bands (two of gp41, gp120, gp160) are considered proof for infection. In other words in Africa anybody who has antibodies which react with actin is considered infected with "HIV". Neither has anybody taken notice of the work by researchers from the AIDS vaccine program, National Cancer Institute, who label all the proteins with molecular weights higher than 31,000 as cellular proteins,10, 36 including p41 as actin. Instead many "HIV" experts including yourself, dedicate a great deal of time to study the "HIV envelope" protein gp41, gp120 and gp160.

(e) In the early 1970s Gallo as well as other retrovirologists found that the RNA of retroviral particles contained "poly(A) regions and hypothesised "therefore that poly(A) might be a diagnostic property of tumour viruses" (retroviruses), despite the fact that ample evidence exists which shows that poly(A) is not specific to retrovirus, and Gallo was aware of it.37 Indeed, "poly(A) sequences were found in both messenger RNA (mRNA) and their nuclear precursors…poly(A) sequences provided that basis for a long-sought route for mRNA purification".38 Yet, from their "purified virus", Montagnier's and Gallo's groups selected a number of poly(A)-RNA fragments and claimed this RNA was the "HIV" genome. In the BMJ Online debate, Brian Foley admitted that the poly(A)-RNA is not specific to retroviruses and that the "HIV" genome was a poly(A)-RNA originating from the "purified virus" but was not able to produce evidence for purification. However, he insisted that this poly(A)-RNA was "HIV RNA" as proven by the existence of the "HIV infectious molecular clone". But he was unable to give us not even a single reference containing evidence for the existence of the "HIV infectious molecular clone". Our repeated request: "Would Brian Foley please give us a summary of the evidence (not just the title) of a study as well as the evidence from a few confirmatory studies where the existence of an "infectious molecular clone" (as defined by Brian Foley) of "HIV-1" has been proven. If Brian Foley fails to respond with his summaries and references then we must conclude his whole argument for the existence of "HIV-1", based upon the existence of the "HIV-1 infectious molecular clone", collapses." - remains unanswered.

(f) The main and absolutely necessary property of sexually transmitted agents is bi-directionality. That is, transmission from the passive (semen recipient) to the active (semen donor) partner and vice versa. In 1984, Gallo wrote: "Of eight different sex acts, seropositivity correlated only with receptive anal intercourse… and with manual stimulation of the subject's rectum (receptive "fisting")…and was inversely correlated with insertive anal intercourse".39 Two years later they confirmed their 1984 findings: "In this analysis, only receptive rectal intercourse, douching, rectal bleeding…were significant predictors ($p<.05$) of anti-HTLV-III positivity…We found no evidence that other forms of sexual activity contributed to the risk".40 In a 1994 review of all the major studies conducted in gay men the authors concluded:
"(1) unprotected anogenital receptive intercourse poses the highest risk for the sexual acquisition of HIV-1 infection; (2) anogenital insertive intercourse poses the highest risk for the sexual transmission of HIV-1 infection; (3) there is mounting epidemiologic evidence for a small risk attached to orogenital receptive sex,…(4) sexual practices involving the rectum and the presence of

(ulcerative) STD facilitate the acquisition of HIV-1; (5) no or no consistent risk for the acquisition of HIV-1 infection has been reported regarding other sexual practices such as anogenital insertive intercourse and oroanal sex…".[41] Since only the passive partner develops a positive "HIV" antibody test (acquires "HIV"), the following questions arise:

- How is it possible to claim that "HIV" is sexually transmitted?
- How is it possible to claim that tens of millions of people have been infected by heterosexual sex? Why the passive partner in a gay relationship cannot infect the active partner, but a woman can infect her heterosexual male partner?

Everyone will agree that pregnancy is a sexually acquired phenomenon and not a sexually transmitted phenomenon. That is, the active partner can make pregnant the passive partner, the passive partner can never make pregnant the active partner. So doesn't it follow that "HIV" (a positive antibody test) is also a sexually acquired phenomenon?

(g) In a prospective study published in 2003, researchers of the Amsterdam Cohort study, analysed "CD4 and CD8 T cell activation marker expression in 102 individuals with known seroconversion data, before and after seroconversion. They concluded: "This study demonstrated for the first time that low preseroconversion numbers of CD4 T cells and increased levels of immune activation were associated with an increased risk to develop AIDS after seroconversion...In conclusion, our data show that chronic immune activation and the size of the CD4 T cell pool are critical factors in HIV-1 pathogenesis, even when measured before seroconversion".[42] The authors from the MultiCenter AIDS Cohort Study concluded "These data suggest that greater sexual activity following establishment of HIV-1 infection leads to exposure to promoters or co-factors that argument (or determine) the rate of progression to AIDS".[43] Since immune deficiency before infection is critical for the development of AIDS and after infection factors other than "HIV" augment (or determine) the rate of progression to AIDS, would you please tell us what role does "HIV" infection play in the causation of AIDS?

(h) By definition, "Virions [of the family *Retroviridae*] are spherical, enveloped and 80–100 nm in diameter".[44] According to Gelderblom "The Family of retroviruses are "enveloped viruses with a diameter of 100-120 nm budding at cellular membranes. Cell released virions contain condensed inner bodies (cores) and are studded with projections (spikes, knobs)".[45] Before the AIDS era, retrovirologists were fully aware that not all the particles which have the morphological characteristics of retroviruses are infectious, that is, they are viruses. In 1976 Gallo wrote: "virus-like particles morphologically and biochemically resembling type-C virus but apparently lacking the ability to replicate, have been frequently observed".[46] There are a few reports of "HIV" particles in AIDS patients, all from lymph nodes. The first was published in 1984 by researchers from Royal Perth Hospital.[47] They have repeatedly pointed out that what they have seen were "virus-like" particles. However, there are significant steps in showing these "virus-like" particles are indeed virus and that this virus is indeed "HIV". Nevertheless, this is still cited as the first paper to have proven the existence of "HIV" particles *in vivo*. In none of the few EM in vivo studies were controls used. In the only EM study, either *in vivo* or *in vitro* in which suitable controls were used and in

which extensive blind examination of controls and test material was performed, particles indistinguishable from “HIV” were found in 18/20 (90%) of AIDS as well as in 13/15 (87%) of non-AIDS related lymph node enlargements. This led the authors to conclude: “The presence of such particles do not, by themselves indicate infection with HIV”.[48]

Furthermore, although it is claimed that there are AIDS patients which have a million particles per ml of blood, to date nobody has published EM data to prove such claims. At present, most “HIV” experts consider “HIV” to be a Lentivirus. That is, particles of 100 nm to 120 nm, cone-shaped core, having lateral bodies and surface studded with spikes. There is not one single study, either *in vitro* or *in vivo*, with evidence of the existence of particles having all these morphological characteristics.

Many “HIV” researchers have found particles in cultures with diameters less than 100 nm or larger than 120 nm.[10, 49] The average diameter of the “HIV” particles reported in the Bess *et al* paper was 234nm. In 2003, Kuznetsov *et al* wrote: “Among the particles displayed…were some that were much smaller, on the order of 80 to 100 nm in diameter, and some that were much larger, on the order of 160 to 240 nm in diameter” having a myriad of core types including no core at all.[50] If the particles of 100 - 120 nm, found in the “HIV” infected cultures, are “HIV”, what is the origin of and what are the other particles?

In his 1983 paper, Montagnier stated that the “HIV” particles were a “typical type C RNA tumor virus”,[51] so did Gallo in 1984.[52] In his book *Virus* Montagnier said that by June 1983 he considered “HIV” to be a Lentivirus. In 1984 he said that “HIV” is a type D particle.[17] At present, most “HIV” experts consider “HIV” to be a Lentivirus. However, in 2003, Kuznetsov *et al* said that the “HIV” particles “are virtually indistinguishable from virions of MuLV…” – a type C particle. In their 2005 book, Constantine *et al* wrote: “The Lentivirinae (lentiviruses) are complex type D-type viruses that include the human pathogenic HIV viruses…”.[8]
Would you please tell us the precise taxonomical classification of the particle HIV experts claim to be the cause of AIDS?

According to Montagnier: “it is tuberculosis that constitutes the greatest public health problem today: 1.7 billion people have latent infections of *Mycobacterium tuberculosis* (the bacillus that causes tuberculosis), while eight million are actively infected.”[27] “Tuberculosis kills more people than any other single disease - almost 3 million in 1990”.[53]

According to researchers from the USA and the Indian Council of Medical Research, in India, a “community with pre-existing endemic diseases such as tuberculosis and diarrhoea disease makes the clinical diagnosis of AIDS difficult”.[54] Before the AIDS era it was known that: “In TB as well as in lepromatous leprosy, an immunosuppressive state will frequently develop in the host. This state is characterised by T lymphopenia with a decreased number of T helper cells and an inverted T-helper/T-suppressor cell ratio...Immunosuppression induced by the infection with *M.tuberculosis* can persist for life, even when the TB is not progressive”.[55] In 1994 Essex and his associates presented evidence

which shows that 64.9% of leprosy patients and 23.1% of their contacts tested positive by two different ELISAs. They also showed that 83.6% of patients and 64.9% of contacts had "indeterminate" WB. In fact the WB patterns satisfied all the criteria for a positive WB test including the Australian criteria which at that time was the most stringent. They were said to be "indeterminate" because they had only one glycoprotein band, gp41 and thus did not satisfy the WHO African criterion which is two of "three" glycoproteins (gp41, gp120, gp160). The presence or absence of gp120 and gp160 does not depend on the presence or absence of antibodies directed against them but on how the WB strips are made.[35] Furthermore, according to the Constantine *et al* book on page 197 one reads: "It may be noted also that persons with envelope reactivity only are rarely found to be infected with HIV in some populations."

Essex *et al* performed experiments in order to determine the reason(s) for the "indeterminate" results. They concluded: "Overall, this data suggests that LAM [lipoarabinomannan, which is also present in other *Mycobacteria* including *M. tuberculosis*.] or PGL-I [phenolic glycolipid I] antibodies can bind to HIV-1 proteins and cause false-positive reactivity…Our observation of cross-reactivity between LAM, and to a lesser extent PGL-I, with HIV-1 antigens suggest that HIV-1 ELISA and WB results should be interpreted with caution when screening individuals infected with *M. tuberculosis* or other mycobacterial species. ELISA and WB may not be sufficient for HIV diagnosis in AIDS-endemic areas of Central Africa where the prevalence of mycobacterial diseases is quite high."[56] Yet on the basis of these tests (or no test at all by the Bangui definition) we are all led to believe by the "HIV" experts that the developing world "bears more than 90% of the global burden of HIV infection" and that "Tuberculosis (TB) is the leading cause of death worldwide among people with HIV".(*Lancet* Editorial July 11, 1998, p122).

Even if such tests were to be performed, given the fact that neither ELISA nor WB are sufficient to diagnose "HIV" infection in TB patients, where is the proof that "AIDS" patients with TB, "the leading cause of death worldwide among people with HIV", are indeed infected with this retrovirus? Even if the antibody tests were 100% specific and all TB patients were tested and found positive, where is the proof that since the AIDS era the major precipitating cause of TB is "HIV" and not still drug abuse,[57] "crowding, poor sanitation, lack of proper hygiene"[55] or "malnutrition and general lack of medical services", which according to Essex, contribute to "diarrhoea, tuberculosis and other common African diseases that signify AIDS"?[58] Is it possible that the leading cause of death from AIDS worldwide is based on mistakenly identifying *M. tuberculosis* antibodies for HIV antibodies?

11. You said: "**Duesberg has argued and many people in his clique have accepted his views on this that AIDS is caused by poppers, by drug use, over stimulated immune systems, poverty — anything but HIV. Space aliens will no doubt be a cause soon.**"

We have put forward these (apart from "space aliens") and additional factors as causative agents of AIDS even before Peter Duesberg expressed this view. In fact we have given a mechanism by which these factors induce their pathogenic effects, a theory which leads to predictions regarding AIDS prevention and treatment. Interestingly, Luc Montagnier, "the discoverer of HIV", little by little, has become an apologist of our oxidative theory of AIDS.[59, 60]

12. You said: "**One of the views is that Africa is different because Africa has to be different because the denialists otherwise can't explain why HIV has killed so many people there. It's held that diagnostic assays simply don't work, which of course isn't true. They hold that PCR-based viral load assays don't measure HIV, which of course isn't true. The details get more and more bizarre, and they're often mutually contradictory.**"

The antibody tests do not prove "HIV" infection anywhere in the world, not just Africa. Describing AIDS in Africa, in his book *Virus*, Montagnier tells how a team of researchers led by Peter Piot in 1983 diagnosed AIDS in Zaire using "primitive" means: "About thirty cases had been diagnosed with the means available, which were primitive. There were as many female patients as male, which proved for the first time the disease's heterosexual transmissibility. Piot had very carefully kept the serums of these patients. In late 1983, with a reliable LAV ["HIV"] antibody test (RIPA) already at our disposal for a number of months, I suggested to him that we blindly look for the presence of LAV antibodies. (His serums had code numbers.) Piot enthusiastically agreed. I gave him the results by telephone: all the patients whose AIDS diagnosis had been based on clinical findings and on the decrease in blood lymphocytes tested positive for the *gag* protein of LAV. Piot later told me that it was the biggest thrill of his career as a researcher." Note "Ten patients had 'Chronic mucocutaneous HSV [herpes simplex virus] infection', 14 bilateral interstitial pneumonia 'with severe dyspnoea, unresponsive to antibiotics or tuberculostatics', 31 oral and/or oesophageal candidiasis and six had disseminated KS…Since KS has long been endemic in Zaire, only patients with fulminant KS were included."[61] The sera were tested by RIPA (radioimmunoprecipitation assay, similar to the Western blot). The test was considered positive if a p24 band was present. The p41 band and also a 84-kD band were not considered diagnostic because "The 43-kD [p41] band and the 84-kD band are cellular contaminants that are immunoprecipitated in all the tested sera", from both patients and controls. Thirty two (88%) patients were positive. So were six out of 26 (23%) controls.[62] Like Montagnier, Gallo and his associates also tested Africans for "HIV" antibodies.[63] Of 53 patients with "AIDS", including the first 26 patients reported from Rwanda, "46 (87%) tested positive...67 (80%) of 84 prostitutes [without any clinical symptoms] and five (12.5%) of 40 and eight (15.5%) of 51 healthy controls and blood donors, respectively", also tested positive. "All blood donors were of good socioeconomic status". Sera which had one positive ELISA were considered as proof for HIV infection. [Today, using an

ELISA type test, which cannot be used in Australia even as a screening test, one single positive result was deemed to be sufficient to estimate that 4.5 million South Africans are infected with "HIV"[64]. Sera which had a borderline ELISA were further tested with the WB. In the WB, "serum samples possessing reactivity to HTLV-III ["HIV"] p41 and/or p24 were scored positive".

In 1985 Gallo conducted tests in Uganda. "The Ugandan serum tested was primarily from clinically healthy donors randomly selected as controls for Burkitt's lymphoma patients on the basis of age, sex, and community. All samples were collected between August 1972 and July 1973." All the samples were tested by ELISA and WB. "Of the 75 samples, 50 of 55 that exceeded the cutoff of 2 standard deviations recognized specific viral bands with an overall positive rate of 66 per cent. The most prominent reactions were with antigens having molecular weights of 76K, 55K, 41K, and 24K. Less frequently recognized antigens had molecular weights of 64K, 59K, 32K, and 18K. These values coincide with the previously described molecular weights of HTLV-III antigens recognized by serum from AIDS patients or individuals at risk for AIDS." Gallo was surprised by the high level of "infectivity" and the apparent lack of AIDS in Africa. "If, as we suspect, the antibody reactivities found represent widespread exposure or infection by HTLV-III [HIV], then it must be asked why the incidence of AIDS in the Ugandan population (and neighbouring Zaire) has gone unnoticed for so long…It is possible that AIDS existed in African populations without being recognized as a separate disease entity" or "the virus detected may have been a predecessor of HTLV-III or is HTLV-III itself but existing in a population acclimated to its presence. It further suggests an African origin of HTLV-III".65

This problem of a very high level of "infectivity" and lack of AIDS in Africa was "solved" by introducing a unique definition for AIDS in Africa, the Bangui AIDS definition and of unique criteria for a positive WB. Unlike the AIDS definition in the West, the WHO Bangui definition for Africa does not require immunological (T4 lymphocyte count) or antibody tests or a specific disease diagnosis but consists largely of symptoms such as weight loss, diarrhoea, cough and fever. For example, an African with diarrhoea, fever and persistent cough for longer than one month is, by definition, an AIDS case. Any WB in which two of the "three" glycoproteins (gp41, gp120, gp160) are reactive is considered proof for "HIV" infection. This is despite the fact that as far back as 1981 Gallo accepted that antibodies which react with retroviral glycoproteins are directed "against the carbohydrate moieties on the molecule that are introduced by the host cell as a post-transcriptional event, and which are therefore cell-specific and not virus-specific".66

Regarding the use of the PCR to prove "HIV" infection:

(a) According to the CDC "In adults, adolescents, and children infected by other than perinatal exposure, plasma viral RNA nucleic acid tests should NOT be used in lieu of licensed HIV screening tests (e.g., repeatedly reactive enzyme immunoassay" (emphasis in original).67 In other words, the CDC acknowledges that the PCR test is not as good as the antibody test.

(b) According to Montagnier: "PCR is also not reliable because, paradoxically, it is too precise [?sensitive]. Indeed, PCR gene amplification is so sensitive that it may cause a false-positive result in the blood sample, which may contain some of the mother's infected cells, erroneously indicating infection in the child."27

(c) One group of “HIV” experts states “Plasma viral [RNA] load tests were neither developed nor evaluated for the diagnosis of HIV infection”.68
(d) Roche, the company that manufactures the AMPLICOR HIV-1 RNA MONITOR test, includes the following statement in the test kit packet insert: “The COBAS AMPLICOR HIV-1 MONITOR Test v1.5 is not intended to be used as a screening test for blood or blood products for the presence of HIV-1 or as a diagnostic test to confirm the presence of HIV-1 infection”.

By design, the reverse transcriptase and protease inhibitors do not inhibit transcription of proviral DNA into RNA. Rather they prevent new rounds of “infection” of uninfected cells by “HIV”. Since “infected” cells die within a few days, and there are no “new infections” taking place, these drugs should result in a decrease the “HIV” DNA. Which means the decrease in “viral load” is indirect, that is, is via the decrease in “HIV” DNA. This means that any decrease in viral load should be preceded or at least accompanied by a decrease in “HIV” DNA. However, Italian researchers observed a “dichotomy” with HAART: “A dramatic drop in the levels of cell-free virus in plasma [“viral load”] and PBMC intracellular transcripts was observed in all but one patient, whereas a significant increase in PBMC proviral DNA …occurred in the majority of cases”. In fact, no patient had a decrease in viral DNA.[69] Similar results were reported by some of the best Australian “HIV”/AIDS experts.[70] The purpose in treating with HAART is to decrease the “HIV” DNA, yet in reality the opposite is found.

In the 2005 Constantine *et al* book, on page 293 one reads: “In addition to their utility for monitoring HIV infection, viral load measurements can be used to estimate the time until development of AIDS and to estimated the time until death…It has been clearly shown that RNA levels are predictive of risk for progression to AIDS, CD4 decline, and death.” On page 296 one reads: “The maintenance of low levels of viral RNA in patients during the course of antiretroviral therapy results in a decreased risk of progression to AIDS.” Indeed, this should be the case if “HIV” is the cause of AIDS, the viral load tests “measure HIV”, and HAART results in a decreased risk of progression to AIDS. However, in a 2006 *Lancet* paper involving over 22,000 patients, the authors report a “paradoxical” finding. They reported that there was a “discrepancy between the clear improvement…recorded for virological response and the apparently worsening rates of clinical progression”.[5] This means either the risk/benefit associated with HAART treatment is very high or “HIV” is not the cause of AIDS.

Let us remind you what Montagnier said at the European parliament in 2003 regarding AIDS pathogenesis in Africa: Montagnier said that the cause of the “clinical phase of opportunist infections and cancers which result in death [AIDS] “ is principally due to a decline in the numbers of T4 cells. The decline in T4 cells is due to their “propensity to die from apoptosis”. In turn apoptosis is due to “potent oxidative stress”. Significantly, with the exception of African patients, Montagnier did not address the cause of the oxidation in the AIDS risk groups. In regard to African patients he said that the oxidative stress “exists even in the non-infected individuals because of malnutrition” (our translation from French).[71] That is, the cause of AIDS is oxidation, not an infectious retrovirus, and the cause of oxidation in African is malnutrition. Thus what “killed so many people there” is poverty not “HIV”. This is what we have been advocating from the beginning of the AIDS era. Since Montagnier agrees with our oxidative theory of AIDS, do you consider him also a dissident?

13. You said: "**HIVNET 012, a trial of single dose nevirapine to prevent mother-to-child transmission in Uganda. Paperwork discrepancies arose in this trial because of administrative problems at rural African sites. ... The conclusions of the trial are scientifically valid and they were endorsed by the Institute of Medicine in an independent evaluation. But Celia Farber et al. twists the facts to make is appear as if this important trial equates to Tuskegee style abuse, criticizing, amongst other things, the lack of a placebo arm, which is nowadays an ethical necessity not to have a placebo. Farber's version of events becomes accepted wisdom in the Boston Globe, the New York Observer over the past few months have simply parroted her views as if they had merit.**"

There are many scientific and methodological problems with the HIVNET 012 trial, paperwork discrepancies is only a minor problem. The powerpoint presentation is at www.theperthgroup.com/PRESENTATIONS/nevppsn1.ppt

Regarding the absolute necessity of having a placebo, suffice it to quote Brooks Jackson, the senior author of the HIVNET 012 trial.

"No researcher can assess a drug's effectiveness with scientific certainty without testing it against a placebo. That's the only way we can know for sure if a short course of AZT or nevirapine is better than nothing."
www.hopkinsmedicine.org/hmn/S01/feature.html

14. You wrote: "**Nancy Padian's paper: Nancy Padian of UCSF publishes a classic study on heterosexual HIV transmission in 1997. ... AIDS denialists though conclude that the Padian paper proves that HIV is not heterosexually transmitted and contradicts the author's own conclusions and to the social science literature. ... Nancy Padian is here today, or said she was going to be here today, and she can speak to this — she's here — and she can speak to this, how her own paper is being abused and twisted.**"

This year, the following correspondence was conducted with Professor Padian:

"*As far as I can judge, your data does not prove that HIV is heterosexually transmitted. Am I wrong in my interpretation? If so, would you please give me some details why I am wrong.*"

Professor Padian's response was: "*Yes you are wrong. Read the papers. The discussion in very thorough in each.*"

The follow up correspondence was:

"*In your publications, you repeatedly pointed out that the data from cross-sectional studies are not reliable. In your 1997 prospective study you "observed no seroconversions...". In your discussion, you also pointed out that "No transmission occurred among the 25 percent of couples who did not use condoms consistently at their last follow-up nor among the 47 couples who intermittently practiced unsafe*

sex during the entire duration of follow-up." This is the information which led me to come to the conclusion which you have stated is wrong. I would be grateful if you would tell me what information I am missing."

Professor Padian did not respond.

This sequence of events is typical of "HIV" experts. When they are asked initial questions regarding their research, they respond in a patronising manner. Naturally, their response leads to more questions where we "dig deeper". Faced with such questions they refuse to respond.

It is interesting to note that Professor Padian did not talk on "how her own paper is being abused and twisted". She said that "*scientists need to be trained as to their responsibility to journalists and their responsibility to make their views known through the public venues as well as scientific venues. …we're working in an anti-science era, and we have our role to play*."

Surely, scientists must be aware of their responsibility to journalists but above all as Howard Temin pointed out ''when an experiment is challenged no matter who it is challenged by, it's your responsibility to check. That is an ironclad rule of science, that when you publish something you are responsible for it. . .even the most senior professor, if challenged by the lowliest technician or graduate student, is required to treat them seriously and consider their criticisms. It is one of the most *fundamental aspects* of science'' (emphasis in original).[72]

15. You said: "**They misrepresent their academic credentials to create an illusion of competence. …The Perth group, Papadopulos-Eleopulos and Turner, claimed to have academic appointments at the University of Western Australia. That's not the case, and they're now being disowned by the university.**"

It is out of our control in regard to how people label us. In neither our publications nor on our website is there a claim that "Papadopulos-Eleopulos and Turner…have academic appointments at the University of Western Australia".

The value of a theory or any other work cannot be judged on the basis of whatever academic credentials or lack of them the person has. Last year's Nobel Prize for medicine was given to two individuals from Royal Perth Hospital, the oldest and largest teaching hospital in Western Australia, a gastroenterologist and a pathologist working in the same department with a member of our group. Neither of them had academic credentials when they performed their work; one of them still has no academic credentials.

16. You said: "**Some AIDS denialists work in bona fide universities. Some even teach students. If this happens in your neighborhood ask the university authorities why they allow this and then write about it. There's a case in Chicago I know about. Science and health journalists should talk to the editorial desk and letters editors and vice versa to ensure that AIDS denialist letters are spotted on arrival and spiked, not published.**"

In response we can do no better than to quote Dr Richard Smith, past editor of the *British Medical Journal*: "We should never forget Galileo being put before the inquisition. It would be even worse if we allowed scientific orthodoxy to become the inquisition." Here is the full text of Dr Smith's response to your attempts to silence us and stop the online BMJ debate:

"Sir:

Your News story "Medical journal under attack as dissenters seize AIDS platform" (Nature 426, 215; 2003) was a fair report of researchers' objections to rapid responses being posted on the website of the *British Medical Journal (BMJ)* by people who are sceptical about a link between AIDS and HIV. As editor of the *BMJ*, however, I find it disturbing to see scientists arguing for restrictions on free speech. Surely open communication and argument is a fundamental value of science?

John Milton put the argument better than anybody in 1643, in his pamphlet *Areopagitica*. "Give me," he wrote, "the liberty to know, to utter, and to argue freely according to conscience, above all liberties. ... [W]ho ever knew Truth put to the worse, in a free and open encounter? ...Yet is it not impossible that she [truth] may have more shapes than one ... [I]f it come to prohibiting, there is not aught more likely to be prohibited than truth itself; whose first appearance to our eyes, bleared and dimmed with prejudice and custom, is more unsightly and unplausible than many errors ... Where there is much desire to learn there of necessity will be much arguing, much writing, many opinions; for opinion in good men is but knowledge in the making."

We should never forget Galileo being put before the inquisition. It would be even worse if we allowed scientific orthodoxy to become the inquisition.

I'm not arguing that those who doubt the link between HIV and AIDS are right, but I want to keep our threshold for posting rapid responses as low as possible.

How, I'm legitimately asked, does this fit with an editorial code I have drafted saying: "Editors should take all reasonable steps to ensure the accuracy of the material they publish." My first reaction is that perhaps "accuracy" is the wrong word to use. As editors we receive thousands of manuscripts containing millions of assertions. We can't possibly check every "fact", and distinguishing fact from opinion is not as straightforward as it sounds.

The answer, I think, lies in transparency. Our rapid responses are clearly unfettered debate full of crazy ideas, false logic, and unreadable, mis-spelt prose as well as some literary and scientific gems. What you see is what you get. In contrast, original articles have been as rigorously peer-reviewed as we can manage, with the recognition that peer review itself is a deeply flawed process.

Your News story states: “The dispute crystallizes the conflict in the Internet era between a journal’s desire to experiment with open electronic debate, and its fundamental obligation to its readers to provide them with authentic information.” I don’t agree that there is a conflict. The beauty of the electronic world is that we can have no-holds-barred debate alongside greater selectivity. On our website you can do a search that includes or excludes rapid responses. I suggest that those who want to see the world as it is — rather than how they would like it to be — include rapid responses in their search.”[73]

Dear Professor Moore, for a long time we have been proposing a way by which our views can be scientifically refuted by our critics. Let us propose it to you personally:

Let us have a scientific debate under the auspices of a scientific/medical journal or university of your choice between us and ten “HIV” experts again of your choice. The outcome to be decided by a panel of independent scientists, preferably Nobel Prize winners.

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EMAIL CORRESPONDENCE WITH PROFESSOR MOORE

Dear Professor Moore,

We attach a response to your recent presentation at the International AIDS conference which also addresses a few other matters.

Kind regards,

E Eleopulos

V Turner

WE RECEIVED THE FOLLOWING REPLY FROM PROFESSOR MOORE.

Turner,

You have gone through 21 drafts and a considerable amount of effort to say absolutely nothing that is of any conceivable interest to me. I'm glad you wasted your time though, as communicating with me (or trying to) is harmless, compared to the damage you AIDS denialists do to innocent people you attempt to confuse and thereby cause top be harmed. So, continue to knock yourself out, so to speak. All you will receive from me is my continued contempt, and derision.

John Moore

IN RESPONSE WE EMAILED BACK

Dear Professor Moore,

Thank you for your speedy reply.

1. You wrote: **"You have gone through 21 drafts…"**.

Once again you are wrong. The response we wrote to you was all one draft. It started out at Professor Moore1.doc and then Dear Professor Moore2.doc when we added the second half. MS Outlook appended '1' to the second file because an incoming email of the second file bore the same file name as the one residing on the receiving computer.

2. When we discuss AIDS topics, we thoroughly investigate them. So we don't give "off the cuff" answers. The only reason that we did not spend as much time responding to your talk as you think is because we are very familiar with the data.

3. You wrote: **"…to say absolutely nothing that is of any conceivable interest to me."**

What happened? It is astonishing!! From your talk at the AIDS conference it was obvious you were very interested in what we are saying. Why the sudden change of heart and why is what we say of "no conceivable interest"?

4. You wrote: **"…as communicating with me (or trying to) is harmless,…"**

What do you mean by "trying to"? Do you mean that you did not read our response?

5. You wrote: **"…compared to the damage you AIDS denialists do innocent people you attempt to confuse and thereby cause to be harmed."** Why are you still calling us "AIDS denialists"?

Let us repeat the first point we made in our original response to you:

"Let us make it clear that we are not AIDS denialists. That is, we do not deny that in 1981 a syndrome involving a high frequency of KS and a number of opportunistic infections was identified in gay men and subsequently became known as AIDS. What we are doing and have been doing from the very beginning is to question the accepted cause of AIDS and to put forward an alternative theory for the cause of AIDS which has a number of well-defined predictions, most of which have been satisfied.1

Furthermore, what is the "damage" we do"? How do we cause "innocent people…to be harmed"?

6. You wrote: **“All you will receive from me is my continued contempt and derision.”**

As an academic surely you must pursue scientific discussions and don’t your students learn by your example? Don’t you teach your students that science progresses through scientific debate?

Sincerely,

Eleni Papadopulos Eleopulos

1. Papadopulos-Eleopulos E. Looking back on the oxidative stress theory of AIDS. Continuum 1998; 5:30-35

http://www.healtoronto.com/oxstress.html.

PROFESSOR MOORE REPLIED

You are confusing me for someone who is interested in what you have to say, and you are confusing yourself for someone who merits a more detailed response. Kindly correct yourself of those delusional tendencies. I despise you and your fellow AIDS denialists, and I regard your level of “scientific analysis” as pitiful and laughable.

John Moore

WE REPLIED

Dear Professor Moore,

Thank you for your reply.

We have been pleading in our scientific publications for someone to produce scientific evidence which proves that our views are wrong including direct requests to Luc Montagnier.(“Would Montagnier Please Clarify Whether HIV or Oxidation By The Risk Factors Is The Primary Cause Of AIDS?” Medical Hypotheses (2006) 67, 666-668)

http://www.theperthgroup.com/SCIPAPERS/PGMontOSMH2006.pdf

You wrote: **"…I regard your level of "scientific analysis" as pitiful and laughable."**

Why?

You wrote: **"I despise you and your fellow AIDS denialists…"**

Why are you continuing to distort our position regarding AIDS? Let us repeat the first point we made in our original response to you:

"Let us make it clear that we are not AIDS denialists. That is, we do not deny that in 1981 a syndrome involving a high frequency of KS and a number of opportunistic infections was identified in gay men and subsequently became known as AIDS. What we are doing and have been doing from the very beginning is to question the accepted cause of AIDS and to put forward an alternative theory for the cause of AIDS which has a number of well-defined predictions, most of which have been satisfied. (Papadopulos-Eleopulos, E "Looking back on the oxidative stress theory of AIDS" Continuum (1998) 5(5), 30-35) http://www.theperthgroup.com/CONTINUUM/lookingback.html

Kind regards,

Eleni Papadopulos-Eleopulos

PROFESSOR MOORE REPLIED

Plead away, but I'll simply ignore your pleas, as will any bona fide scientist.

John Moore

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