

## PERTH GROUP RESPONSE TO FABIO FRANCHI

February 28<sup>th</sup> 2010

We were asked to comment on Montagnier's claim that a healthy immune system can rid the body of HIV infection. And so we did. However, we agree with you and a few other dissidents that Montagnier's claim will not be "very useful to support the dissidents' ideas". And certainly Montagnier's claim does not endorse Peter Duesberg's claim that the "HIV" antibodies "neutralise the virus". This is because firstly, a healthy immune system does not equal antibodies – there is a lot more to the immune system than antibodies. Secondly, there cannot be "HIV" antibodies without "HIV" proteins. According to all retrovirologists, including Peter, to have "HIV" proteins the "HIV" genome must first be integrated into the host, cellular DNA. They all tell us that the integration is very rapid, at most days, while the antibodies are not detected for weeks or months, even up to six months, after exposure. As far as we know, there is no mechanism by which antibodies can excise a given sequence of DNA from the human genome.

You wrote: "If I were an HIV-positive individual, I would be sorry to watch these too passionate discussions about "virus/non-virus", that are undoubtedly important and must be settled. I would be more in favour of discussions on how to improve my health and about concrete alternative, really effective proposals...Because I would expect good results from a good theory and vice versa".

We agree with you – good theories should lead to good clinical results.

At present there are three main theories in regard to the cause of AIDS. (In what follows, by AIDS we mean low T4 cells and the high frequency of Kaposi's sarcoma and opportunistic infections that were diagnosed in gay men, haemophiliacs and IV drug users at the beginning of 1980s). The three theories are:

1. HIV.
2. Drugs ( passenger virus)
3. Oxidative (oxidative agents, mainly drugs and semen). There is no evidence which proves the existence of HIV.

Since:

- (i) We all agree there are problems with the "HIV" theory, in what follows we will ignore it.
- (ii) We agree theories are important. It follows that the existence/non-existence question is not only of academic interest but also of vital importance to "an HIV-positive individual" and those at risk of developing AIDS. These individuals and physicians like yourself who look after them, will have several questions including the following:

## FIRST QUESTION

What does an "HIV positive" antibody test mean and is it in any way related to my well being?

The answers given to these questions by the two theories are significantly different.

(a) Passenger virus theory

A positive antibody test means you are infected with a retrovirus, HIV. The antibodies neutralise the virus and as a consequence the virus is rendered non-pathogenic. This is why it is a harmless passenger virus. Apart from a means of diagnosis a positive antibody test has no clinical significance. Antibody positive haemophiliacs or gay men have the same probability of having or developing AIDS as HIV negative haemophiliacs or gay men.

(b) Oxidative theory

Our theory was published in a paper entitled "Reappraisal of AIDS – Is the Oxidation Induced by the Risk Factors the Primary Cause?" [HERE](#) It was initially submitted to *Nature* at the beginning of 1986 and was eventually published in *Medical Hypotheses* in 1988. That is a long saga. In this paper the specificity of "HIV" antibodies is questioned. The substantial evidence in support of this claim included: "...the disulfide links of the antibody molecule play an essential role in the acquisition of immunological specificity and by virtue of their covalent nature, provide for the stabilisation of the particular structure underlying the specific activity of the molecule. Furthermore, the pattern of pairing of sulfhydryl groups to form disulfides is not an invariant property of the linear chain but depends on extrinsic factors including the redox. In other words protein synthesis and specificity in general and antibody synthesis and specificity in particular are redox dependent. If this is so, then any agents which will induce the same redox changes as a virus, could induce the synthesis of viral antibodies and antigens in the absence of the virus". It is concluded: "The only sensible conclusion is therefore that seropositivity does not mean virus positivity".

In 1991 we submitted a paper entitled "HIV antibody testing – autoreactivity and other associated problems" to *Research in Immunology*, a Pasteur Institute publication. The paper was first accepted for publication in the *Bulletin de l'Institut Pasteur*, and then rejected (see [correspondence](#)). A modified version of this paper was published in 1993 in *Bio/Technology* (now *Nature Biotechnology*) under the title "Is a positive Western Blot proof of HIV infection?" [HERE](#)

In the 1991 (original) paper submitted to *Research in Immunology* we wrote: "Currently the WB is accepted as nearly 100% specific for HIV infection but the test is not standardised and because there is no suitable gold standard for the presence of HIV infection it is not possible to calculate its sensitivity and specificity...In healthy individuals as well as both non-AIDS and AIDS patients a positive WB

does not indicate HIV infection but represents a non-specific marker for a variety of unrelated conditions. Consequently, the general belief that almost all individuals, healthy or otherwise, who are HIV antibody positive are infected with a lethal retrovirus, has not been scientifically substantiated".

Since then we have published more evidence in support of our claims: (a) There is no proof that the antibody test proves HIV infection; (b) yet there is no denying that a positive antibody test, at least in the AIDS risk groups, increases the probability of the presence or the development of AIDS. The fact that the test does not prove "HIV" infection does not preclude its use as a non-specific test, along with many tests of the same nature employed in clinical practice. For example, the peripheral blood white cell count and erythrocyte sedimentation rate. Hence "HIV" antibodies may be nothing more than a non-specific indicator, serendipitously discovered in 1983/84, of altered homeostasis connoting a propensity to develop particular diseases.

In response to the question, does a positive antibody test prove "HIV" infection, (the question does not refer to false positive results, which are characteristic of all antibody tests), "the HIV-positive individual" must consider the following:

- (1) There is no evidence that "HIV" antibodies neutralise the virus;
- (2) The only way to claim that a positive antibody test is proof of "HIV" infection is to compare the test against "HIV", that is, to use "HIV" isolation/purification as a gold standard. To date this has not been done. Which means at present no evidence exists to prove a positive antibody test even in one person if caused by "HIV" infection;
- (3) If the antibodies are directed against the "HIV" proteins then all infected individuals would be expected to have at least similar if not identical WB patterns regardless of where they live. This is not the case.
- (4) All "HIV" experts admit the "purified" virus contains cellular proteins which have the same molecular weight as the "HIV" proteins, including p24 and p41. However, to date nobody has shown that the electrophoretic bands, for example the p41 band, in addition to containing the cellular protein actin also contains an "HIV" specific, p41 protein;
- (5) All the "HIV" experts admit that the p120 and p160 bands in the WB are polymers of p41. Nobody has evidence which proves these polymers are those of an "HIV" p41 protein and not that of the cellular protein actin, which has a molecular weight of 41,000;
- (6) All "HIV" experts accept that "HIV" positive individuals and those at risk have auto-antibodies. Auto-antibodies are a sign of incipient or actual illness;

- (7) Unlike patients with other infections, "HIV" positive individuals and those at risk have hypergammaglobulinemia. This is an immunological phenomenon not found in healthy people;
- (8) Recently evidence has been published showing that immunoglobulins present even in normal individuals exhibit auto-antibody binding reactivity. The appearance and disappearance of this property is redox dependent. This discovery has had such a profound effect it constitutes one of the main arguments "...for rethinking much of what we assume to be true in basic immunology, including [immunological] tolerance";
- (9) The epidemiological evidence of the last 25 years shows that an "HIV positive" test can be sexually acquired but cannot be sexually transmitted. This single fact proves that whatever the test may signify it cannot be infection with a sexually transmitted agent.

The "HIV positive individual" and the dissidents in particular must also consider the main prediction of the passenger virus protagonists, namely: If two groups of people, for example, HIV positive and HIV negative haemophiliacs are followed up for several years, the frequency of the "HIV related diseases" will be the same in each group. If this prediction is fulfilled it will prove the antibodies are "HIV" and the virus is harmless.

However, given the above evidence, and in particular (4-7), will one expect this to be proven or will its outcome be extremely detrimental to the dissident cause?

## **SECOND QUESTION**

What can be done to prevent becoming "HIV positive"?

Passenger virus theory:

There is no need to take any precautions. Being "HIV" positive has no bearing on an individual's well being or future health prospects.

Oxidative theory:

A positive antibody test signifies exposure to foreign antigens (factor VIII and the impurities in it; antigens in dirty needles; semen, often from many individuals; and oxidising agents (factor VIII, drugs, oral or IV, semen). All are detrimental to health especially if exposure is large and prolonged.

A positive antibody test can be avoided by limiting exposure to foreign antigens and oxidising agents or, wherever possible, eliminating such exposure.

NOTE: One of the most significant differences between the proponents of the passenger virus theory and our theory is exposure to semen. The proponents of the passenger virus theory claim that sex (semen) plays no role in AIDS. While some dissidents proclaim this message loud and clear (Rethinking

AIDS and its leader continue to do so) the evidence shows the opposite. Indeed the evidence for a role of sex in AIDS was obvious from the first study conducted in 1984 to examine the role of sex in AIDS. By the 1990s this would have been obvious to anybody who cared to take even a superficial look at the evidence.

Not surprisingly the dissidents have alienated the scientific community and, as a result, the editors of scientific journals have closed the doors to our many attempts at publication. One cannot ignore or disdain scientific evidence that has a bearing on public health. And in this regard the dissidents have been labelled "dangerous" and rightly so. In fact it is surprising that, following the publication of *The AIDS Trap* brochure, dissidents have not been taken to court. Maybe the "HIV" experts are fearful of a defence similar to the defence in the Parenzee case, this time conducted by a lawyer cognisant with the scientific arguments we have put forward and in the absence of an additional "defence team" stabbing the official team in the back.

From the beginning of the AIDS era we have presented basic scientific, epidemiological and experimental evidence that semen can have detrimental effects. We also presented evidence that, as with all toxins, it is not semen *per se* but, to paraphrase Peter Duesberg, it is the dose and duration of exposure that "kills". In other words, it is not sexual orientation or even passive anal intercourse ("anal intercourse may be practised by a much larger absolute population of heterosexuals than of homosexuals"), which is of critical importance.

For AIDS to appear a very high frequency of receptive anal intercourse over a long period is necessary. As we wrote in our response to *The AIDS Trap* brochure, AIDS is more like anal and cervical cancer. The effect is not the result of the act itself but the high frequency and duration of exposure to semen. But, as with cervical and anal cancer, other factors may promote or militate against the development of AIDS.

Last year, when UN declared the end of heterosexual AIDS, Michael Ellner was worried that sooner or later there will be a backlash against the gay community. If his occurs then, according to our theory, it will have no scientific basis.

### **THIRD QUESTION**

What can an "HIV positive individual" do to prevent (or at least minimise the probability) of the development of AIDS?

Passenger virus theory:

A "HIV positive individual" has the same probability of developing AIDS as a negative one. He can avoid AIDS by not taking drugs, including antiretroviral drugs.

Oxidative theory:

The probability of a seropositive individual developing AIDS is higher than that of a seronegative individual. The development of AIDS can be avoided or at

least diminished by avoiding or reducing exposure to foreign antigens and oxidising agents. Suffice to mention one example. By 1992 researchers from the MACS study found that following the acquisition of a positive "HIV" antibody test, "factors" associated with passive anal intercourse "augment" or "determine", the development of AIDS. Given its cytotoxic effects, semen must be one such factor, if not the only factor, especially in face of the epidemiological evidence showing that a positive antibody test and AIDS is significantly associated with trauma (bleeding) to the rectum during sexual contact.

#### **FOURTH QUESTION**

What should one do to treat AIDS?

Passenger virus theory:

According to the proponents of this theory AIDS should not be treated with ARVs or certain antibiotics (Septrim). No alternative treatment is offered.

Oxidative theory:

In 1986, when "Reappraisal of AIDS: Is the oxidation caused by the risk factors the primary cause", was resubmitted to *Nature*, the letter of re-submission ended with: "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 disease...by using currently available therapeutic substances". That is, antioxidants in general and –SH (sulphydryl) containing compounds in particular. In 1989 we published what the reviewer called a protocol to treat KS with –SH containing compounds and hyperthermia.

In our view, when a patient presents with an AIDS indicator disease, the disease (TB, PCP for example), should be treated the same way as in a non-AIDS patient. In addition, our theory demands that the underlying cause, cellular oxidation, must also be addressed. Both AIDS patients and HIV positive individuals should be treated with –SH containing compounds under the supervision of a doctor who has access to laboratory facilities to measure redox and at least until the redox is normalised. The treatment may include adjunct measures such as diet and stress reduction management. At the same time, exposure to oxidising substances should be minimised, or if possible, eliminated.

To date, the best supporting evidence for our theory has been published by researchers from Stanford University who performed an investigation into the relationship between reduced glutathione levels in T4 cells and AIDS, including a double blind, placebo controlled study. They found:

- (i) a "**dramatically** better survival of individuals with higher GSB [glutathione SH] levels";
- (ii) "NAC [N-acetylcysteine an -SH compound] administration was shown to be associated with a **significantly decreased** rate of CD4 T cell loss during the trial period";

- (iii) "...among subjects with CD4 < 200, lower levels of GSB [a measure of glutathione-SH in CD4 cells]...predict decreased survival; and that the probability of surviving 2–3 years **increases dramatically** as GSB level approach normal range" following the administration of NAC (emphasis added).

In a PubMed search on [AIDS AND oxidation] the chronological order of publications shows the first paper to appear was [ours](#). And over the past 20 years many papers have been published which confirm our predictions. Seropositive individuals and AIDS patients are oxidised and the administration of anti-oxidant compounds results in beneficial effects including effects on T4 cells and their immune function. Yet despite our published papers, our names are never mentioned by these researchers including Montagnier, who was personally made aware of our work in 1991. Nonetheless, in 1997 Montagnier proposed "his" oxidative theory which he presented using virtually the same words as those our [paper](#) published in 1992 in *Research in Immunology*, a Pasteur Institute publication.

Incredibly, all this time Rethinking AIDS and its leader used easily refutable arguments against the HIV theory of AIDS but never our arguments. Montagnier's comments in the *House of Numbers* gave Crowe the opportunity to make the oxidative theory, "his", Montagnier's, "idea". Recently Bauer listed our oxidative theory alongside many other "theories" (in fact none is a theory with the possible exception of Rebecca Culshaw's which we have not read). Bauer seems to be most impressed with a "theory" which claims that "T-cell loss" in AIDS is due to a low concentration of "NAD<sup>+</sup>", that is, a low concentration of an oxidised enzyme ("+" means loss of electrons = oxidation). Presumably Bauer thinks AIDS can be ameliorated by raising the concentration of this oxidised enzyme, making already oxidised patients more oxidised. This means this "theory" is totally opposite ours.

Crowe and the RA Board of Directors did not ignore only us but everybody else, including journalists (Neville Hodgkinson and Djamel Tahj), who did not subscribe to the passenger virus theory. By doing so Crowe and the Board of Rethinking AIDS harmed not only the Perth Group but also dissidents in general and HIV seropositive individuals, and especially seropositive dissident individuals. As we testified in our cross-examination at the Parenzee hearing, in 1991 we approached Professor Martyn French, the immunologist in charge of AIDS patients at the Royal Perth Hospital, with a view to conducting a trial of -SH compounds in the treatment of AIDS patients. (During the Parenzee hearing the prosecution took great pains to deny any such collaboration but we have incontrovertible documentary evidence to the contrary). We began measuring the oxidation status of HIV positive patients provided by Professor French but no further progress was possible because of lack of money. (In 1991, Professor Peter McDonald, also an HIV expert witness at the Parenzee hearing, who was involved in AIDS research grant allocations in the 1990s, told us we had zero chance of obtaining funding).

We believe that if we could have obtained funding our results would have been even more "dramatic" because the Herzenbergs:

1. Measured the glutathione levels in T4 cells.
2. The NAC dose they used was arbitrary and was not adjusted for each patient.
3. The only advice they gave their patients in regard to the avoidance of oxidising substances was the pitifully inadequate “it may be prudent for these individuals to avoid excessive exposure to UV irradiation and unnecessary use of drugs that can deplete GSH—e.g., alcohol and prescription or over-the-counter formulations containing acetaminophen [paracetamol (Panadol)]”.

It appears the Herzenbergs were not aware of a number of significant factors:

1. The glutathione –SH are not the –SH which determine cellular functioning.
2. There are laboratory methods to measure cellular redox which should be used to adjust the dose of –SH compounds for individual patients;
3. AIDS patients and those at risk are exposed to many oxidising agents other than alcohol and paracetamol.

Unlike the Herzenbergs we consider diet and stress reduction management an integral part of therapy.

At present, in regard to conducting a clinical trial we have many problems apart from lack of funding. These include:

1. The care of AIDS patients in Australia is the exclusive domain of clinical immunologists. In fact one of us [EPE] has been officially instructed not to come into any contact with any AIDS patient, including any who may approach us.
2. Conducting trials with agents apart from different combinations of antiretroviral drugs is considered unethical.

However, we are ready as ever to design such a trial and collaborate with anyone who is interested and capable of conducting it.

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