

HIV TESTS CANNOT DIAGNOSE HIV INFECTION

A reply to several of the numerous fallacies contained in the document entitled "Errors in Celia Farber's March 2006 article in Harper's Magazine"
(Gallo et al 2006).

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1. Several false statements regarding HIV testing by Gallo, Geffen, Gonsalves, et al. (Gallo et al 2006).

On March 4, 2006, Robert Gallo, together with pro-antiretroviral HIV activists from the Treatment Action Campaign in South Africa, the Gay Men's Health Crisis in the USA, the Elizabeth Glaser Pediatric AIDS Foundation, also in the USA, and others (Gallo et al 2006), released an alleged rebuttal of an article by Celia Farber in the March 2006 issue of Harper's Magazine: "Out of control: AIDS and the corruption of medical science" (Farber 2006).

Regarding HIV testing, Gallo and his co-authors assert that (Gallo et al 2006):

"HIV tests were highly accurate from the time they were developed in 1984 and have become much more accurate over time as the underlying technology has evolved. HIV tests are amongst the most accurate available in medical science."

"A PCR test for the presence of the virus itself can accurately determine a child's HIV status."

"An AIDS diagnosis cannot be considered definitive without an HIV test."

"Farber's comment about hopping on a plane from Uganda to Australia to change HIV diagnosis is simply silly hyperbole."

"The risk of a false positive HIV test in Africa, as elsewhere, is very small if the correct protocol is followed. Some HIV antibody tests have been tested in Africa and found to be very accurate. These are the ones generally used. For example, the Abbott Determine rapid test

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used widely in South Africa has a specificity of at least 98% (and in some studies has achieved close to 100%). When this test is combined with a second rapid test or an ELISA test to determine HIV status, the risk of a false positive is negligible. The contribution of TB and malaria to false positives on today's tests is also negligible."

"A properly conducted HIV-test protocol (which involves at least two HIV tests) has very small chance of giving a false positive, irrespective of pregnancy status."

However, available scientific data do not validate these statements. Several established scientific facts supporting the contention that HIV tests cannot diagnose HIV infection are as follows:

2. Pharmaceutical companies acknowledge that HIV tests are not specific for HIV.

The primary tests for the diagnosis of HIV infection are two antibody tests, the ELISA and Western blot, and a genetic test, the PCR or "Viral Load" test. However, the ELISA and Western blot tests only detect antibodies against what are erroneously accepted to be HIV proteins or antigens. Similarly, the PCR or Viral Load test for HIV only detects copies of fragments of RNA that have arbitrarily been regarded as the nucleic acid of HIV. None of these tests detect the HIV virus itself, nor do they detect HIV particles.

The pharmaceutical corporations that manufacture and commercialize these test kits acknowledge the inaccuracy of the tests. This explains the seemingly surprising statement included in the kit inserts: "Elisa testing alone cannot be used to diagnose AIDS, even if the recommended investigation of reactive specimens suggests a high probability that the antibody to HIV-1 is present" (Abbott 1997).

The insert for one of the kits for administering the Western blot warns: "Do not use this kit as the sole basis of diagnosis of HIV-1 infection" (Epitope Organon Teknika).

In like manner, the insert that accompanies a very frequently used test for PCR Viral Load warns: "the Amplicor HIV-1 Monitor test is not intended to be used as a screening test for HIV or as a diagnostic test to confirm the presence of HIV infection" (Roche 2003).

Therefore, the pharmaceutical drug manufacturers acknowledge the fact that neither the ELISA, nor the Western blot, nor the Viral Load tests for HIV are specific to diagnosis HIV infection.

Interestingly, the only valid method of establishing the sensitivity and the specificity of a diagnostic test in clinical medicine is to compare the test in question with its gold standard. The only possible gold standard for the HIV tests is the human immunodeficiency virus itself, HIV. Since HIV has never been isolated as an independent, free and purified viral particle, it is not possible to properly define either the sensitivity or the specificity of any of these tests. Currently, the sensitivity and the specificity of the tests for HIV are arbitrarily defined, not by comparison to purified HIV itself, but by comparison of the tests in question with the clinical manifestations of AIDS, or with T4 cell counts. This explains why Abbott clearly states: "At present there is no recognized standard for establishing the presence and absence of HIV-1 antibody in human blood. Therefore sensitivity was computed based on the clinical diagnosis of AIDS and specificity based on random donors" (Abbott 1997). Since there is no gold standard for defining the specificity of the tests used for the diagnosis of HIV infection, all HIV-positive results for HIV infection must be considered false positives. Therefore no individual can validly be identified as either HIV-positive or HIV-negative.

The large majority of AIDS researchers, journalists, lay people, and health care workers themselves do not understand the limitations of these tests because they do not have access to

the relevant data. Additionally, little or no concern is expressed by medical faculties and research institutions with regard to communicating these facts to physicians, let alone to the general public.

3. HIV has never been either isolated or purified as a real virus.

Proper procedures for isolating and purifying retroviruses (formerly known as RNA tumor viruses) were established as early as 1964 (O'Connor et al 1964; De Harven 1965a,b, 1974).

The most common sources of material from which retroviruses can be isolated and purified are blood (viremia), other tissue homogenates, and supernatant fluids from infected cell cultures (de Harven 1965a,b).

The most frequently used technique for isolation and purification of retroviruses includes the following primary steps: (1) Concentration of the viral particles by centrifugation; (2) Electron microscopy monitoring of the concentrated viral particles; (3) Biochemical and genetic analysis of the purified viral particles; (4) Controlling the experiments to avoid misinterpreting endogenous retroviruses as exogenous infectious retroviruses; and (5) Biological tests to ascertain if the isolated retrovirus is indeed potentially pathogenic and virulent (O'Connor et al 1964; De Harven 1965a,b, 1974).

However, neither Montagnier, nor Gallo, nor Levy et al. had adhered to these techniques when they claimed to have isolated "the AIDS virus" in 1983 and 1984 (Barré-Sinoussi et al 1983; Papovic et al 1984; Gallo et al 1984; Levy et al 1984). The first two steps were omitted; they did not provide the electron microscope evidence that particles from the "infected" culture supernatant, sedimenting at 1.16 gm/ml of sucrose, were composed primarily of viral particles (concentrated viral particles). Instead, they provided electron microscope photographs of stimulated/activated cultured lymphocytes releasing particles similar to retroviruses. These same particles, however, can be released by "non infected" stimulated/activated lymphocyte cultures (Dourmashkin et al 1993). Unfortunately, the experiments were not properly controlled; where were the electron microscopy photographs of "infected" as well as "not infected" culture supernatants sedimenting at 1.16 gm/ml of sucrose, EM micrographs required to determine whether or not viral particles were concentrated at that gradient? Additionally, where were the electron microscopy pictures of "non infected" lymphocytes grown under identical culture conditions?

The alleged existence of HIV was asserted from the study of proteins, reverse transcriptase activity (RT), and RNA fragments that were found in culture supernatants, not from the direct analysis of purified viral particles.

Surprisingly, the existence of HIV was then claimed indirectly, on the basis of the presence in complex cell cultures and/or "HIV-positive" individuals of (1) proteins/glycoproteins such as gp160/150, gp120, gp41/45/40, p34/32, p24, and p18/17, each claimed to belong to HIV; (2) enzymes such as reverse transcriptase that supposedly belongs to HIV; and (3) RNA or DNA fragments that supposedly belong to HIV (Papadopoulos-Eleopoulos et al 1993, 1996, 1997a, 1997b, 1997/8; Turner 1996, 1997/1998, 1998; Philpott 1997; Giraldo et al 1999; de Harven 1997/8, 1998, 2002a,b). However, none of these substances have been proven to belong to HIV. How could it be proven that the molecules found in those cultures actually belong to viral particles that have never been properly purified? How could it possibly be demonstrated that these substances are not simply cellular microvesicles or cell debris contained in the cultures and that happen to sediment at the same density as retroviruses? In order to prove that those molecules, allegedly regarded as "markers", are part of a retrovirus named HIV, it would have been absolutely necessary to purify the retroviral particles, separating the particles from everything else. This has never been done with HIV (Papadopoulos-Eleopoulos et al 1996; de Harven 1998; Giraldo et al 1999).

However, long before the appearance of the first cases of AIDS, researchers working on “RNA tumor viruses”, currently known as retroviruses, clearly knew that the first prerequisite for the study of virus subcomponents or molecules is to obtain highly purified virus preparations (de-Thé & O’Connor 1966). After purifying the “murine leukemia virus”, these authors were able to employ selected chemicals (i.e. tween-ether, ribonuclease, detergents) to disrupt the purified particles and release the internal components (de-Thé & O’Connor 1966). This was never done with HIV.

One of us has insisted that: “The specificity of viral markers depends on the success of virus isolation and purification. Without fully demonstrated success in virus isolation and purification, identification of *viral markers* is extremely hazardous and can lead to severe misinterpretation of clinical data. A dramatic illustration of this is to be found in current HIV research. In this case, the virus (HIV) has never been properly isolated, since sedimentation in sucrose gradient at the density of 1.16 g/mL was erroneously considered to yield *pure virus*, systematically ignoring that material sedimenting at that density contains large amounts of cell debris and cellular microvesicles (Gluschankof et al 1997; Bess et al 1997). Therefore, proteins and nucleic acids found in such *1.16 bands* are very likely to be of cellular origin and cannot be used as viral markers. Such a faulty methodology has had extremely serious consequences, i.e. the world-wide use of HIV-antibody tests, ELISA and Western blot, which dangerously lack specificity, as demonstrated in 1993 by Papadopoulos et al. (1993), in Australia” (de Harven 1999).

“More disturbing is the fact that some ‘markers’ are searched for in the 1.16 gradient sedimenting material which is the density where intact virions are expected to be found, but not their molecular fragments. If lysed retrovirus particles released molecular markers, the 1.16 samples should at least initially allow researchers to demonstrate virus particles by electron microscopy. However, after 15 years of most intensive HIV research, two independent groups finally decided to explore by electron microscopy the ultrastructural features of the material sedimenting at the 1.16 density. Working on ‘HIV-1 infected T-cell’ cultures supernatants, both groups found that it contains primarily cellular debris and cell membrane vesicles which could definitely not be identified with HIV particles and rare ‘virus-like’ particles (Gluschankof et al 1997; Bess et al 1997). Still this is the type of sample in which ‘viral markers’ are currently identified and used to measure the effects of anti-viral drugs in current clinical trials” (de Harven 1998).

The reverse transcriptase activity (RT) found in culture supernatants by researchers who claim to have isolated “the AIDS virus” (Barré-Sinoussi et al 1983; Papovic et al 1984; Gallo et al 1984; Levy et al 1984) could just as well have a cellular origin, since this enzyme is ubiquitous (Ross et al 1971; Beljanski 1972; Varmus 1987; Coffin et al 1997). RT is not a unique feature of retroviruses, as it was mistakenly thought to be by Montagnier, Gallo and Levy’s group.

HIV has never been either isolated or purified as intact viral particles. Therefore, there is no scientific data validating the contention that what is currently referred to as HIV is in fact a virus!

There does not exist a single test tube in any laboratory anywhere containing purified particles of HIV. Researchers working with what they believe to be HIV in laboratories all around the world are most likely not working with HIV particles at all. They are working with proteins, enzymes, or fragments of RNA that have been arbitrarily regarded as belonging to HIV.

The fact that after 25 years of intense research HIV has been neither isolated nor purified in terms of classical virology indicates to us that the infectious view of AIDS as a contagious viral disease is based on an apparently non-existent microbe!

4. So-called "HIV proteins" are not specific markers of HIV.

In the early 1980s, frustrated retroviral cancer researchers trying to prove that AIDS was a retroviral disease, arbitrarily defined what they erroneously called "the AIDS virus proteins," "the AIDS virus enzymes," and "the AIDS virus RNA," which were found in the supernatant of cultures, without having previously either isolated or purified the retroviral particles, i.e., separated them from cellular microvesicles and cell debris, as has been explained in the previous section.

Montagnier's group from the Pasteur institute in France, for example, determined what they call "viral antigens" through a series of immunoprecipitation experiments (Western blot) using cord blood lymphocytes mixed within very complex cell culture systems, with virus from patient 1 as a source of "viral antigens" and antiserum to HTLV-I P24 and serum from patient 1 and 2, and arbitrarily decided that: "three major proteins could be seen: the p25 protein and protein with molecular weights of 80,000 and 45,000. The 45K protein may be due to contamination of the virus by cellular actin which was present in immune precipitates of all the cell extracts" (Barré-Sinoussi et al 1983). Without having previously purified viral particles, they erroneously concluded that, "these results, together with the immuno precipitates, indicate that the retrovirus from patient 1 contains a major p25 protein, similar in size to that of HTLV-1 and different immunologically" (Barré-Sinoussi et al 1983).

Gallo's group from the National Cancer Institute performed Western blot using "lysates of HTLV-III producer cell clones" and serum diluted 1:500, and, also without having previously purified viral particles, arbitrarily decided that, "antigens newly expressed after viral infection and recognized by the human serum used included p65, p55, p41, p39 and p24. A large protein with a molecular weight of approximately 130,000 and a protein of 48,000 were also detected" (Schüpbach et al 1984). However, they also concluded that, "these results show clearly that the antigens detected after viral infection are either virus-coded proteins or cellular antigens specifically induced by the infection" (Schüpbach et al 1984). Additionally, they concluded that, "extensive accumulation of p24 and p41 occurred in the virus preparation which showed that these molecules are the major components of the virus preparation. Allegedly, P24 and p41 were, therefore, considered as viral structural proteins" (Schüpbach et al 1984).

Levy's group of researchers, from the University of California in San Francisco, performed standard indirect immune fluorescence procedures using HTLV-1, LAV and ARV "infected cells" and serum diluted 1:10. They found that antibodies against what was supposed to be ARV (AIDS Related virus) in 88% of AIDS with Kaposi's sarcoma, 100% in AIDS with opportunistic infections, in 93% of male sexual partners of AIDS patients, and in 57% of clinically healthy homosexual men (Levy et al 1984).

These three groups of researchers decided, arbitrarily, that the proteins they found in cell cultures apparently infected with "the AIDS virus" were "HIV proteins." These proteins had not been and have never been extracted directly from isolated, purified viral particles. They could, therefore, just as well have a human cellular origin.

On the other hand, in 1997, the Gluschankof group in France and Germany, as well as the Bess et al group in the United States demonstrated that when one follows the routine procedure to isolate retroviruses from cultures that are supposedly infected with HIV, it is not possible to either isolate or purify virus particles, separated from cellular microvesicles and cell debris, even in fractions sedimenting at the density, in sucrose gradients, where retroviruses are classically known to sediment (Gluschankof et al 1997; Bess et al 1997). They rightly warned that, "caution must therefore be exercised in terms of the presence of cellular vesicles when viral immunogens (*proteins*) are density gradient enriched" (Gluschankof et al 1997), because "human cellular antigens have been found associated with HIV-1 preparations" (Gluschankof et al 1997). Therefore, these 1997 papers from the Gluschankof and Bess groups provide an

objective demonstration that what are commonly called “HIV proteins” or “HIV antigens” or “HIV immunogens” are not specific markers of HIV and could very well originate from the cultured cells.

In this regard, our colleagues from Perth, Australia, have explained several times that the Western blot antigens, proteins, glycoproteins or bands - p120, p41, p32, p24/25, p17/18 - allegedly considered to be specific HIV proteins may not be encoded by the HIV genome but may in fact represent cellular proteins originating from the cultured human cells (Papadopoulos-Eleopoulos et al 1993, 1997a; Turner 1996, 1997/1998). The normal cell component actine probably corresponds to what is known as gp41, while gp120/160 probably represent gp41 oligomers (Papadopoulos-Eleopoulos et al 1993).

Therefore no one has, to date, presented evidence that the so-called HIV proteins or antigens [gp160/150, gp120, gp41/45/40, p34/32, p24, p18/17], are really constituents of HIV (Papadopoulos-Eleopoulos et al 1993, 1996; de Harven 1998, 2002a, 2003; Giraldo 2002a; Giraldo et al 1999).

The proteins and glycoproteins listed above (“HIV antigens”) are claimed to appear exclusively when one co-cultures supposedly infected blood with abnormal cells from leukemic patients, or from umbilical cord lymphocytes (Papadopoulos-Eleopoulos et al 1996; de Harven 1998). Quite probably, the same molecules could be obtained from similar cultures in the absence of “HIV” infection. However, very crucial control experiments were never performed (de Harven 1998, 2003, 2004) specially when researchers used cord blood lymphocytes. These cells of placenta provenance are very likely to be a source of endogenous, probably defective retroviruses (Panem 1979; de Harven 2002b).

Moreover, the cultures where the above substances have been found have been heavily stimulated with phytohemagglutinin, IL-2, antiserum to human interferon, and other agents (Papadopoulos-Eleopoulos et al 1996; de Harven 1998, 2003). These culture stimulants are oxidizing agents and could be expected to stimulate the expression of endogenous retroviruses (Papadopoulos-Eleopoulos et al 1996). Control experiments on these important points cannot be found in the literature. Interestingly, neither HIV itself nor any HIV markers can be found when the cultures are treated with antioxidants (Papadopoulos-Eleopoulos 1988, 1998/9; Papadopoulos-Eleopoulos et al 1992, 1993).

Unfortunately, these alleged “HIV proteins” or “HIV antigens” are used as antigens in the serologic tests for HIV, and this explains the complete lack of specificity of these tests.

5. So-called HIV-RNA is not a specific marker of HIV.

The HIV viral load test is an amplification genetic test that makes copies of fragments of RNA that arbitrarily have been regarded as parts of the HIV genome. These fragments of RNA are found in culture supernatants or in patient’s blood. They are never, however, extracted directly from purified viral particles. What is known as “HIV RNA” might just as well originate from cultured cells or be present in the blood of persons undergoing stress. It could also originate from endogenous, non-infectious retroviruses.

Moreover, it has been established that the human genome contains a sizable proportion of endogenous retrovirus-related sequences (Mager & Freeman 1987; Lieb-Mösch et al 1990).

In the decade prior to the appearance of AIDS, during President Richard Nixon’s “War Against Cancer”, in order to identify “viral proteins” and to extract “viral RNA” samples, researchers successfully used highly purified retrovirus specimens from “viremic” animals. The method applied to achieve this purification of a typical retrovirus was rapid, inexpensive and reproducible (de Harven 1965a,b). However, “most surprisingly, nobody has ever succeeded in

demonstrating HIV particles in the blood of any AIDS patient by this simple method, even though patients could have been selected for presenting a so-called high 'viral load' as determined by PCR methods" (de Harven 2003). PCR is a genetic technique that does not count viral particles at all (Mullis & Faloone 1987), as physicians and lay people may think. It merely makes copies of what is supposed to be HIV RNA (Roche 2003).

"It appears very likely that PCR methods amplify small RNA fragments, more frequently observed under conditions of stress and other chronic illnesses (Urnovitz et al 1999), and which include retroviral segments originating from human endogenous retroviruses. This is not surprising since about 2% of the human genome have marked homology with retroviral genome (Löwer et al 1996). Consequently, 'measuring' the 'viral load' by PCR methods is likely to have no relationship whatsoever with real quantification of a hypothetical exogenous HIV viremia. Kary Mullis himself, Nobel Prize laureate for his discovery of the PCR method, categorically rejects the use of 'his' method for quantitative measurements of a hypothetical HIV viremia (Mullis 1998)" (de Harven 2003).

"HIV cloning" is, likewise, very misleading. Without first isolating and purifying retroviral particles, the cloning of a "specific HIV-RNA" is not possible (Papadopoulos-Eleopoulos et al 1996; de Harven 1998; Giraldo et al 1999). Neither does the cloning of fragments of nucleic acid found in supernatants of supposedly "HIV-infected" cultures indicate HIV. The only way to properly achieve HIV cloning would be first to isolate and purify HIV particles and then to extract RNA from the core of the purified particles. This has never been done with HIV!

However, in 1985, researchers from the National Cancer Institute and from the Dana-Farber Cancer Institute of Harvard University claimed to have found the "complete nucleotide sequence of the AIDS virus, HTLV-III" (Ratner et al 1985). They arbitrarily stated that: "The complete nucleotide sequence of two human T-cell leukemia type III (HTLV-III) proviral-DNA each have four long open readings frames, the first two correspond to the *gag* and *pol* genes. The fourth open reading frame encodes two fractional polypeptides, a large precursor of the major envelope glycoprotein and a smaller protein derived from the 3'-terminus long open reading frame analogous to the long open reading frame (*lor*) product of HTLV-I and -II;" "the HTLV-III is 9,749 pairs (bp) long. The overall structure of the provirus resembles that of other retroviruses" (Ratner et al 1985). And, they continue, "sequences from different clones of HTLV-III allow an analysis of the level of sequence diversity of the virus. A comparison of clones BH8 and BH5 with BH10 demonstrates a 0.9% base pair polymorphism in the coding regions of the genome and a 1.8% base pair polymorphism in the non-coding regions. The heterogeneity among HTLV-III clones shown here could represent sequence divergence developing in culture in a given individual over a period of time, or polymorphic differences in viruses from different individuals. Diversity among different HTLV-III isolates seems to be greater than that between different HTLV-I isolates. Thus, it is likely that most of the divergence among the HTLV-III clones analyzed here represents differences in strains in different individuals" (Ratner et al 1985). However, this statement can only be valid for a fragment of DNA (HTLV-III clone) that the American researchers arbitrarily considered to be "T-cell leukemia type III (HTLV-III) proviral-DNA." Individuals reading this without a critical perspective might therefore be misled by the researchers from the NIH and Harvard University.

One of us described this chaotic situation during a debate on AIDS in Africa, held at the European Parliament in Brussels, as follows: "the 'Viral Load' of newspapers and magazines, all over the world is extremely high, meaning the number of pictures of HIV published almost daily in the world's press. These pictures are extremely attractive, and are frequently rich in artificial colors. They clearly exemplify the danger of misinforming the public with computer graphics. To publish such images brings to the attention of the general public, and of the medical profession as well, an apparently crystal-clear message: yes, HIV has been isolated since one can portray it under the electron microscope. All these images represent computerized rationalizations" (de Harven 2003), always derived from particles observed in

complex and probably contaminated cell cultures, but never derived directly from one single AIDS patient.

“HIV viral load” cannot, therefore, diagnose HIV infection.

6. False positive reactions on the HIV tests.

There are abundant scientific publications explaining that there are more than 70 different documented conditions that can cause the antibody tests to react positive without an HIV infection (Johnson 1993, 1995, 1996a,b; Hodgkinson 1996; Turner 1996, 1997/8; Shenton 1998; Papadopulos-Eleopulos et al 1993; Giraldo 1997d, 2000a; Giraldo et al 1999).

Some of the conditions that cause false positives on the so-called "AIDS test" are: past or present infection with a variety of bacteria, parasites, viruses, and fungi including tuberculosis, malaria, leishmaniasis, influenza, the common cold, leprosy and a history of sexually transmitted diseases; the presence of polyspecific antibodies, hypergammaglobulinemias, the presence of auto-antibodies against a variety of cells and tissues, vaccinations, and the administration of gamma globulins or immunoglobulins; the presence of auto-immune diseases like erythematous systemic lupus, sclerodermia, dermatomyositis and rheumatoid arthritis; the existence of pregnancy and multiparity; a history of rectal insemination; addiction to recreational drugs; several kidney diseases, renal failure and hemodialysis; a history of organ transplantation; presence of a variety of tumors and cancer chemotherapy; many liver diseases including alcoholic liver disease; hemophilia, blood transfusions and the administration of coagulation factor; and even the simple condition of aging and some vaccinations, to mention the most important (Johnson 1993, 1995, 1996a,b; Hodgkinson 1996; Turner 1996, 1997/8; Shenton 1998; Papadopulos-Eleopulos et al 1993; Giraldo 1997d, 2000a).

Christine Johnson, from California, has listed, from the scientific literature, the following conditions that cause false-positive reactions in the antibody tests for HIV (Johnson 1996a,b):

- Natural occurring polyspecific antibodies (Barbacid et al 1980; Healey & Bolton 1993).
- Anti-carbohydrate antibodies (Snyder & Fleissner 1980; Healey & Bolton 1993; Cordes & Ryan 1995).
- Antibodies with a high affinity for polystyrene used in the test kits (Arnold et al 1994; Pearlman & Ballas 1994; Yoshida et al 1987).
- HLA antibodies to class I and II leukocyte antigens (Blanton et al 1987; Bylund 1992; Cordes & Ryan 1995; Profitt & Yen-Lieberman 1993; Sayers et al 1986; Schlepner 1990; Schochetman & George 1992; Steckelberg & Cockerill 1988; Yu et al 1989).
- Passive immunization (receipt of gammaglobulin or immune globulin as prophylaxis against infection) (Ascher & Roberts 1993; Cordes & Ryan 1995; Gill et al 1991; Jackson et al 1988; Lai-Goldman et al 1987; Isaacman 1989; Profitt & Yen-Lieberman 1993; Piszkiwicz 1987; Yale et al 1994).
- Administration of human immunoglobulin preparations (Bylund et al 1992).
- Hypergammaglobulinemia (high levels of antibodies) (Moore et al 1986; Peterman et al 1986).
- Globulins produced during polyclonal gammopathies, very common in groups at risk for AIDS (Bylund et al 1992; Cordes & Ryan 1995; Schlepner 1990).
- Anti-lymphocyte antibodies (Mathe 1992; Ujhelyi et al 1989).
- Anti-collagen antibodies (found in gay men, hemophiliacs, Africans of both sexes and people with leprosy) (Mathe 1992).
- Multiple blood transfusions (Cordes & Ryan 1995; Ng 1991; Peterman et al 1986; Profitt & Yen-Lieberman 1993; Schochetman & George 1992; Yu et al 1989; Sayre 1996).
- Individuals with coagulation defects (Bylund et al 1992; Schochetman & George 1992).
- Hepatitis B vaccination (Jackson et al 1988; Lee et al 1992; Pearlman & Ballas 1994; Profitt & Yen-Lieberman 1993).

- Tetanus vaccination (Pearlman & Ballas 1994).
- False positive in other serologic tests, including RPR for syphilis (Bylund et al 1992; Fleming et al 1987; Moore et al 1986; Schleupner 1990; Schocherman & George 1992).
- Healthy individuals as a result of poorly-understood cross-reactions (Bylund et al 1992).
- Anti-hepatitis A IgM antibody (Schleupner 1990).
- High levels of circulating immune complexes (Biggar et al 1985; Moore et al 1986).
- Presence of normal human ribonucleoproteins (Cordes & Ryan 1995; Schleupner 1990).
- Malaria (Biggar et al 1985; Charmot & Simon 1990).
- Visceral Leishmaniasis (Ribiero et al 1993).
- Leprosy (Andrade et al 1991; Kashala et al 1994).
- Tuberculosis (Kashala et al 1994).
- Mycobacterium avium (Kashala et al 1994).
- Autoimmune diseases: systemic lupus erythematosus, scleroderma, connective tissue disease, dermatomyositis (Bylund et al 1992; Leo-Amador et al 1990; Pearlman & Ballas 1994; Proffitt & Yen-Lieberman 1993; Ranki et al 1992; Schochetman & George 1992).
- Systemic Lupus erythematosus (Esteva et al 1992; Jindal et al 1993).
- Rheumatoid arthritis (Ng 1991).
- Serum-positive for rheumatoid factor, antinuclear antibodies, and other autoantibodies (Dock et al 1988; Steckelberg & Cockerill 1988; Yoshida et al 1987).
- Anti-smooth muscle antibody (Schleupner 1990).
- Anti-mitochondrial antibodies (Cordes & Ryan 1995; Schleupner 1990).
- Anti-microsomal antibodies (Mortimer et al 1985).
- Other antinuclear antibodies (Cordes & Ryan 1995; Schleupner 1990; Steckelberg & Cockerill 1988).
- Anti-T-cell antigen antibodies (Cordes & Ryan 1995; Schleupner 1990).
- Renal failure (Cordes & Ryan 1995; Jindal et al 1993; Schleupner 1990).
- Hemodialysis (Bylund et al 1992; Fassbinder et al 1986; Peterman et al 1986; Schochetman & George 1992; Ujhelyi et al 1989).
- Alpha interferon therapy in hemodialysis patients (Sungar et al 1994).
- Renal transplantation (Burkhardt et al 1987; Cordes & Ryan 1995; Neale et al 1985; Schleupner 1990; Ujhelyi et al 1989).
- Organ transplantation (Agbalika et al 1992; Ng 1991).
- Upper respiratory tract infection (cold or flu) (Challakere & Rapaport 1993).
- Acute viral infections, DNA viral infections (Cordes & Ryan 1995; Pearlman & Ballas 1994; Profitt & Yen-Lieberman 1993; Schleupner 1990; Steckelberg & Cockerill 1988; Voevodin 1992).
- Flu (Ng 1991).
- Flu vaccination (Arnold et al 1994; Challakere & rapaport 1993; Cordes Y Ryan 1995; Hsia 1993; MacKenzie et al 1992; Profit & Yen-Lieberman 1993; Simonsen et al 1995).
- Herpes simplex I (Langedijk et al 1992).
- Herpes simplex II (Challakere & rapaport 1993).
- Epstein-Barr virus (Ozanne & Fauvel 1988).
- Recent viral infection or exposure to viral vaccines (Challakere & Rapaport 1993).
- Pregnancy in multiparous women (Cordes & Ryan 1995; Ng 1991; Profitt & Yen-Lieberman 1993; Steckelberg & Cockerill 1988; Ujhelyi et al 1989; Abbott 1997).
- Cancers (Pearlman & Ballas 1994).
- Multiple myeloma (Bylund et al 1992; Profitt & Yen-Lieberman 1993; Steckelberg & Cockerill 1988).
- Hematologic malignant disorders and lymphomas (Burkhardt et al 1987; Cordes & Ryan 1995; Profitt & Yen-Lieberman 1993; Schleupner 1990; Steckelberg & Cockerill 1988).
- Q fever with associated hepatitis (Yale et al 1994).
- Hepatitis (Sungar 1994).

- Alcoholic liver disease (Bylund et al 1992; Cordes & Ryan 1995; Mendenhall et al 1986; Pearlman & Ballas 1994; Schlepner 1990; Schochetman & George 1992; Steckelberg & Cockerill 1988).
- Primary sclerosing cholangitis (Schochetman & George 1992; Steckelberg & Cockerill 1988).
- Primary biliary cirrhosis (Cordes & Ryan 1995; Profitt & Yen-Lieberman 1993; Schlepner 1990; Steckelberg & Cockerill 1988).
- Stevens-Johnson syndrome (Burkhardt et al 1987; Cordes & Ryan 1995; Profitt & Yen-Lieberman 1993).
- Sticky blood in Africans (Mortimer et al 1985; Papadopoulos-Eleopoulos 1988; Pearlman & Ballas 1994).
- Heat-treated specimens (Jungkind et al 1986; Schlepner 1990; Schochetman & George 1992; Smith et al 1987; Van Beers et al 1985).
- Lipemic serum (Schochetman & George 1992).
- Hemolyzed serum (Schochetman & George 1992).
- Hyperbilirubinemia (Bylund et al 1992; Cordes & Ryan 1995).
- Proteins in the equipment used for these tests (Cordes & Ryan 1995).
- Other retroviruses (Blomberg et al 1990; Cordes & Ryan 1995; Dock et al 1988; Schlepner 1990; Tribe et al 1988).

Therefore, there is a growing number of conditions known to cause the tests for HIV to react positively in the absence of HIV, i.e. false positives.

Interestingly, all of the conditions causing "HIV tests" to react positive in the absence of HIV are conditions which are present, with varied distribution and concentration, in many recognized "AIDS risk groups" in the developed countries, as well as in a large percentage of Africans and people from other parts of the developing world. This means that in all probability many drug users [including some mothers], certain gay males, and some hemophiliacs in the developed countries, as well as the vast majority of inhabitants in most countries of Africa, Asia, South America and the Caribbean, who have positive reactions to the tests for HIV, may very well do so due to conditions other than being infected with HIV (Johnson 1993, 1995, 1996a,b; Hodgkinson 1996; Turner 1996, 1997/8; Shenton 1998; Papadopoulos-Eleopoulos et al 1993, 1997; Giraldo 1997c, 2000a).

It is shocking to realize that a diagnosis of HIV infection is so frequently based on tests that are not specific for HIV, and even more so when one realizes that these non specific tests lead to the prescription of highly toxic anti-retroviral drugs.

7. The real meaning of being "HIV-positive" or "seropositive"

The definition of AIDS, as developed by the United States Federal Government's Centers for Disease Control and Prevention, requires a positive result on the antibody test for HIV (CDC 1992). The importance of HIV in this definition is so strong that, currently, many AIDS researchers, health care professionals and lay people, in following the lead of the United States Institute of Medicine, the National Academy of Sciences and most AIDS researchers, now refer to "AIDS" as "HIV Disease" (Institute of Medicine 1986; Volberding & Cohen 1994; Fauci 1993; Staprans & Feimberg 1997; Lewis & Ho 2003; Wormser 2004).

However, AIDS in many countries of Africa can be diagnosed without an HIV test or any other laboratory test. This was decided by American public health officials and the World Health Organization at a conference in Bangui, in the Central African Republic, in October 1985 (Quinn et al 1986). This allows health professionals to diagnosis AIDS in Africa based only on routine clinical symptoms and signs presented by the patient. However, the most prevalent diseases in Africa are a direct consequence of chronic poverty and are usually manifested by symptoms and signs that are included in the Bangui definition of AIDS, such as weight loss, chronic

diarrhea, prolonged fever, persistent cough, generalized pruritus. Even worse: "the presence of generalized Kaposi's sarcoma, cryptococcal meningitis are sufficient, by themselves, for the diagnosis of AIDS" in Africa (Quinn et al 1986).

In the United States, a positive result on "the AIDS test" - ELISA and Western blot antibody tests - is indicative of HIV infection and predictive of AIDS (Feimberg & Volberding & Cohen 1994; Pins et al 1997; Metcalf et al 1997; Weiss 1998; Holodny & Busch 2003). Also in the United States a diagnosis of HIV-positivity can be done only after the same blood of a person has reacted positive four times in the ELISA test on two consecutive days and one time in the Western blot test. If AIDS is an infectious disease, it would be the very first infectious disease that requires the repetition of the same antibody test four times in order to know if those antibodies are present or not. If the ELISA test was as specific for HIV as claimed, why is it that this test needs to be repeated four times on the same blood specimen before declaring a positive HIV result? This does not happen with any other well-known infectious disease!

The antibody tests are neither standardized nor reproducible, with respect to HIV. They are, by themselves, meaningless because they mean different things in different individuals, in different laboratories and in different countries (Papadopoulos-Eleopoulos et al 1993). They are interpreted differently in the United States, Russia, Canada, Australia, Africa, Europe and South America (CDC 1989; Zolla-Pazner et al 1989; De Cock et al 1991; Voevodin 1992; Maskill & Gutz 1992), which means that a person who is positive in Africa can be negative when tested in Australia; or a person who is negative in Canada can become positive when tested in Africa (Continuum 1995). More embarrassingly, when the same sample of blood was tested on the Western blot test in 19 different laboratories, 19 different results were obtained (Lundberg 1988).

Nor are results from the "HIV Viral Load test" reproducible. This can be seen in the wide range of variability that is accepted in the quality controls set by the companies making and commercializing the test. For example, Roche accepts low control having a range between 630 and 10,000 copies per ml [Lot # G05467], and high control having a range between 80,000 and 720,000 copies per ml [Lot # G05466] [Roche, Amplicor HIV-1 Monitor test Lot # G13330, expiration October 2006]. Most important of all, the problems with the lack of a gold standard for "HIV infection" also apply to the evaluation of the specificity of the PCR or Viral Load test (Papadopoulos-Eleopoulos et al 1993; Johnson 1996c; Philpott & Johnson 1996; Giraldo 2000a). As a consequence, the specificity of the Viral Load test for HIV has never been defined properly and, therefore, "Viral Load" positive results are likely to be false-positives for HIV.

The fact that the defenders of the "HIV is the cause of AIDS" hypothesis had to use genetic amplification - the PCR test - is a strong argument against HIV as the cause of AIDS. To have to amplify tiny amounts of genetic material in the blood of AIDS patients in order to identify HIV, instead of culturing the entire virus and isolating it, violates one of the central rules of infectious diseases, because in the severity climax of any real infectious disease the patient has the highest amount of microbes in his/her tissues. It is at that time, therefore, that it should have been easy to isolate the microbes without having to use PCR genetic amplification.

Interestingly, there are many HIV researchers who are now questioning the identical issues that we (AIDS dissidents) have been critiquing for more than two decades: Where is the scientific proof that AIDS can be sexually transmitted and that it can also be transmitted from mothers to babies during pregnancy, delivery and breastfeeding? (Gisselquist et al 2002; Brewer et al 2003; Gisselquist & Potter 2004).

On the other hand, all of the medical conditions listed in the previous section and that cause false-positive results on "tests for HIV" are characterized by states of inflammation with the subsequent chronic stimulation/activation of the immune system. They are also characterized

by having high levels of immunoglobulins (antibodies) in the blood, as well as high levels of oxidative stress.

Similarly, individuals “at risk for AIDS” and who react positively on “HIV tests” are also characterized as having high levels of antibodies, chronic stimulation/activation of their immune systems (Papadopulos-Eleopulos et al 1993, 1997a,b; Shallenberger 1998; Giraldo et al 1999; Giraldo 1997b, 2000a), as well as high levels of free radicals, specially oxidizing species (Dworkin et al 1986; Fabris et al 1988; Papadopulos-Eleopulos 1988; Turner 1990; Giraldo 1997a,b,c, 2000a; Shallenberger 1998; Giraldo et al 1999).

Moreover, a prerequisite for a person to turn his/her “HIV-negative” status into “HIV-positive” is to have low levels of antioxidants in the blood, such as vitamins A, C and E, zinc and selenium (Moore et al 1993; Mehendale et al 2001; McDonald et al 2001; Giraldo 2003b). Also, antioxidant vitamins have been found to avoid the progression of “HIV-positive” individuals into the clinical manifestations of AIDS (Fawzi & Hunter 1998; Fawzi et al 2004; McNeil 2004). Moreover, “HIV-positive” mothers who have a normal blood level of vitamin A and zinc seem to deliver “HIV-negative” babies (Fawzi & Hunter 1998; Fawzi et al 2004).

High levels of antibodies, present in “HIV-positive” individuals, are regarded as resulting from exposure to significant quantities of recreational drugs, semen, sexual lubricants, factor VIII, blood and blood components, sexually transmitted infections, other infections, mental distress, as well as to parasites, malnutrition, lack of clean water, and other unsanitary conditions (Papadopulos-Eleopulos et al 1993, 1997a,b; Shallenberger 1998; Giraldo et al 1997b,c). All these cause oxidative stress (Papadopulos-Eleopulos 1988; Turner 1990; Papadopulos-Eleopulos et al 1993, 1997a,b; Giraldo 1997b,c; Shallenberger 1998; Giraldo 2000b; Giraldo et al 1999). Some defenders of the HIV dogma call these oxidizing agents “cofactors”. However, multiple and chronic exposures to a variety of these factors represent, by themselves, potential causes of AIDS (Giraldo 1997b, 2000a,b). As a result of chronic exposures to these factors, immune systems are chronically stimulated, with the subsequent production of polyspecific antibodies readily detected, non-specifically, on the ELISA and Western blots tests.

Biochemically speaking, the body responds, non-specifically, to exposures to cocaine, sex lubricants, malnutrition, electromagnetic fields or to unclean water and parasites. The non specificity of these “stresses” was discovered by Hans Selye in the middle of the last century (Selye 1936, 1946; 1982).

The serologic tests for HIV (ELISA and Western blot) may react positively in the presence of poly-specific antibodies. Positive result on antibody tests for HIV could, therefore, result from chronic antigenic stimulation, rather than from a hypothetical infection with an exogenous retrovirus such as HIV (Giraldo 1997a-e, 2000b; Giraldo et al 1999). Chronic antigenic stimulation of the immune system may be the consequence of multiple, repeated, and chronic exposures to immunological stressor agents (Snyder & Fleissner 1980; Barbacid et al 1980; Wing 1995). Similarly, positive results on PCR tests for HIV can result from the presence of fragments of genetic material in the blood of individuals exposed to a variety of stressor agents (Urnovitz et al 1999; Giraldo et al 1999). Therefore, the reactivity on the three main tests for HIV (ELISA, Western blot, and PCR or Viral Load) could simply result from multiple responses to a variety of chemical, physical, biological, mental and nutritional stress (Giraldo 1997a-e; Giraldo 2000b, 2002). Additionally, the degree of reactivity on “HIV tests” may be proportional to the level of exposures to immunological stressor or oxidizing agents.

In this regard, the HIV phenomenon has been plausibly explained as a response of cells to different types of stress: “Human Immunodeficiency Virus type-1 (HIV-1) replication and proviral gene expression are exquisitely responsive to factors that induce cellular stress” (Bate et al 2000).

Interestingly, Giraldo had the opportunity to demonstrate that all human blood samples react positively on the ELISA test when the tests are run with non-diluted serum. This indicates that all individuals have antibodies against what is supposed to be HIV. The individuals that react positively with straight serum would have a smaller quantity of antibodies than those reacting positively when the serum is diluted 400 times (Giraldo 1998/9). This observation has been confirmed by Yugoslavian and Italian researchers (Metlas et al 1999).

Along the same lines, no one is HIV viral load negative. All samples of human blood, tested by PCR Viral Load, always demonstrate the presence of copies of "HIV RNA." The standard protocol for HIV Viral Load declares a blood sample negative if less than 400 copies of HIV RNA are found. Similarly, the ultrasensitive protocol for HIV viral load declares a blood sample negative if less than 50 copies of HIV RNA are found (Roche 2003). No single human being is, therefore, entirely free of copies of "HIV RNA" in his/her blood. We all are "HIV Viral Load" positive to some degree. Whether this is due to minimal expression of endogenous retroviruses or to universal exposures to stressor agents remains to be analyzed.

In addition, exposure to immunological stressors or oxidizing agents is the cause of mild to moderate levels of immune suppression present in many non-symptomatic individuals who react positively on the "tests for HIV." If exposure to immunological stressors is not stopped, or if the individual is not detoxified, the health status of these individuals will frequently worsen, their immune systems eventually collapsing with the subsequent development of the clinical manifestations of AIDS. The immune system has three main functions: (a) defense against intruders, (b) surveillance of the growth of some tumors, and (c) homeostasis or balance of all body organs and systems. With the collapse of these three functions, opportunistic infections, opportunistic tumors, and opportunistic metabolic diseases may develop. As a matter of fact, this is AIDS. AIDS, rather than being an infectious/viral disease, appears to be a toxic and nutritional syndrome (Giraldo 1997a-e; 2002).

The successful nutritional and antioxidant therapies in the prevention and treatment of AIDS (Giraldo 2003a,b) can now be better understood.

On the other hand, if "the AIDS test" (ELISA and Western blot) were in fact detecting antibodies to HIV, it would not be logical to conclude that these antibodies indicate an active infectious process. The presence of antibodies to any virus simply means humoral immune response to that virus, and not necessarily that the virus is still active and pathogenic (Evans 1989; Zinkernagel 1993; Mims et al 1995). In most instances, antibodies against viruses indicate immunity. This is the very basis of vaccination against viral diseases. Even if ELISA and Western blot tests were specific for antibodies against HIV, the question would remain to find out why, in the case of AIDS, the presence of antibodies indicates disease, rather than protection against the incriminated microorganism?

There is no justification for the fact that patients as well as the general public have never been made aware of all of the preceding facts, a scientific dereliction resulting from widespread censorship. Without a clear awareness of the considerable uncertainties concerning the so-called tests for HIV, people cannot make informed decisions. Individuals should have the capacity to make informed choices (Ken et al 1996; O'Mara 1998; Silverman 1998). However, the possibility to express informed choice implies easy access to verifiable information. There is no justification for the fact that most people have not been informed about the serious inaccuracy of the tests for HIV. Withholding or obscuring these facts is a serious breach of public trust, violating as it does people's capacity to express valid informed consents that are essential in all decision making concerning their health care.

Fortunately, Celia Farber's article in the March 2006 issue of Harper's magazine (Farber 2006) is an example of a high level of professional journalism that gives us the hope that the era of inexcusable censorship on all matters related to HIV/AIDS is finally over.

8. Experiments proposed during the South African Presidential AIDS Advisory Panel.

In year 2000, during the South African Presidential AIDS Advisory Panel meetings (first in Pretoria, then in Johannesburg), the so-called "AIDS dissidents" proposed nine experiments. The goal of some of them was to determine, once and for all, whether HIV could be isolated and purified according to classical virological methods, and what is the real significance of testing positively on "tests for HIV." However, due to the strong censorship and pressure from the HIV establishment, these experiments have not been carried out yet.

De Harven proposed to attempt isolation of HIV, following classical techniques for isolating and purifying retroviruses (O'Connor et al 1964; de Harven 1965a,b,1974). For that purpose he proposed to take blood from AIDS patients with very high results in the "HIV Viral Load test" and who accordingly should have large numbers of circulating HIV particles (viremia). (www.polity.org.za/govdocs/reports/aids/aidspanel).

Giraldo proposed studying the uncertain significance of "positive" HIV tests, comparing 6 different groups of people and performing ELISA, Western blot, Viral Load, together with complete hematological and chemical profiles as a means to evaluate their general health, as well as evaluating their immunological, nutritional, and oxidative status. Groups to be studied were: (a) A group of healthy individuals from different ages; (b) A group of patients with chronic clinical conditions unrelated to AIDS; (c) A group of non-symptomatic individuals from the conventional AIDS risk groups who react negatively on "HIV tests;" (d) A group of non-symptomatic individuals from the conventional AIDS risk groups who react positively on "HIV tests;" (e) A group of patients with clinical manifestations of AIDS who react positively on "HIV tests;" (f) A group of patients with clinical manifestations of AIDS who react negatively on "HIV tests" (www.polity.org.za/govdocs/reports/aids/aidspanel).

The result of such an experiment could determine whether the so-called HIV tests bear any relationship to an individual's level of exposure to stressor or oxidizing agents. If so, the tests could possibly be used as a measure of an individual's level of oxidative stress.

9. Conclusions and Recommendations

9.1. Particles closely resembling retroviruses demonstrated by electron microscopy in the classical paper concerning "HIV isolation" (Barre-Sinoussi et al 1983; Papovic et al 1984; Levy et al 1984) were not demonstrated as originating from "pre-AIDS" nor from AIDS patients. They could, most likely, originate from lymphocytes that were mixed in these complex cell cultures, i.e. cord blood lymphocytes.

9.2. "HIV reverse transcriptase" described in classical papers on "HIV isolation" (Barre-Sinoussi et al 1983; Papovic et al 1984; Levy et al 1984) is not a specific marker of HIV, since that enzyme is present in all living cells and could, therefore originate from the cell debris contaminating the alleged viral samples.

9.3. The specificity of the retroviral origin of so-called "HIV-proteins" described in the classical paper (Barre-Sinoussi et al 1983; Papovic et al 1984; Levy et al 1984) could have been demonstrated only after successful purification of HIV. As acknowledged by Luc Montagnier, HIV has not been purified (Papadopulos-Eleopulos et al 1997/98) and the "HIV-proteins" cannot, therefore, be used as reliable markers of HIV.

9.4. "Sequencing of HIV-nucleic acid" is not a specific marker of HIV either, for the same reason, i.e., the lack of any successful purification of the virus.

9.5. In 1997, Glushankoff's group in Europe, and Bess's group in the United States (Glushankoff et al 1997; Bess et al 1997), were not able to isolate nor to purify HIV from cell cultures regarded as active producers.

The word "isolation" as used by the most noted researchers (Barre-Sinoussi et al 1983; Gallo et al 1984; Levy et al 1984) can be very misleading, as has been pointed out many times (Papadopoulos-Eleopoulos 1988; Papadopoulos-Eleopoulos et al 1993, 1996, 1997a,b; Turner 1996, 1997/1998, 1998; de Harven 1998, 2003; Giraldo 2000a; Giraldo et al 1999).

9.6. Retroviral particles have never been either isolated or purified directly from any individual AIDS patient. Claims of successful isolation have always been made from the analysis of highly complex (and frequently contaminated) cell cultures.

9.7. Therefore, since no retrovirus has ever been clearly demonstrated to be associated with AIDS patients, the HIV/AIDS hypothesis has to be fundamentally reappraised.

9.8. If AIDS were indeed caused by a retrovirus, how can we explain that more than 25 years of considerable research efforts, based exclusively on that single hypothesis, have failed to isolate the responsible exogenous retrovirus? How can we explain that after more than twenty-five years we still have no curative treatment, no vaccine, and no verifiable epidemiological predictions? Obviously, time is pressing us to courageously ask the essential question, i.e., is the HIV=AIDS hypothesis correct? We must realize that it is possible to view AIDS differently, entirely outside the fields of infectious diseases and retrovirology.

Rather than being viral and infectious, AIDS could more likely be a toxic and nutritional disease caused by multiple, chronic and repeated exposures to immunological stressor agents, which can have a chemical, physical, biological, mental, or nutritional origin (Giraldo 1997a-d, 2000b, 2002).

Note: For further scientific facts demonstrating that "HIV tests cannot diagnose HIV infection" we recommend the careful study of the publications at the following websites:

www.rethinkaids.com
www.robertogiraldo.com
www.theperthgroup.com
www.virusmyth.net

Most unfortunately, the type of information provided in this article cannot be found in the "peer-reviewed" medical journals, due to the strong censorship exercised by the HIV orthodoxy. However, this should surprise no one, since it only reflects the profound crisis currently affecting the peer-review system (Horrobin 1990, 1996, 2001). "Peer-review is one of the sacred pillars of the scientific edifice" (Goodstein 2000). However, all indications are that: "Far from filtering out junk science, peer-review may be blocking the flow of innovation and corrupting public support of science...Those who disagree are almost always dismissed in pejorative terms such as 'maverick,' 'failure,' and 'driven by bitterness'...The peer-review processes in both academia and industry have destroyed rather than promoted innovation" (Horrobin 2001).

Furthermore: "Peer-review is also the process that controls access to funding, and here the situation becomes much more serious: Failure to pass the peer-review process might well mean that a project is never funded" (Horrobin 2001). Two decades of AIDS dissident efforts provide many examples of the systematic rejection of funding for non-HIV related AIDS research.

Interestingly, the scientific establishment, its journals, and its grant-giving bodies "consistently refuses open scrutiny" (Horrobin 2001). Rothwell and his group "have provided solid evidence

of something truly rotten at the core of science" (Rothwell et al 2000). They report: "it is not surprising that the public is increasingly skeptical about the agenda and the conclusions of science...Public support can only erode further if science does not put its house in order and begin a real attempt to develop validated processes for the distribution of publication rights, credit for completed work, and funds for new work...If science is to have any credibility — and also if it is to be successful — the peer-review process must be put on a much sounder and properly validated basis or scrapped altogether" (Rothwell et al 2000).

Let us unite with love and compassion to defend humankind from "AIDS and the corruption of medical science."

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