# IS IT RATIONAL TO TREAT OR PREVENT AIDS WITH TOXIC ANTIRETROVIRAL DRUGS IN PREGNANT WOMEN, INFANTS, CHILDREN, AND ANYBODY ELSE ?

## THE ANSWER IS NEGATIVE

Roberto A. Giraldo[1], Michael Ellner[2], Celia Farber[3], Barnett J. Weiss[4], Francis R. Buianouckas[5], Tom DiFerdinando[6], Ray Vagg[7], and Edward A. Lieb[8].

Continuum (London) Summer 1999; 5(5): 38-52.

## Summary

The treatment and prevention of AIDS with antiretroviral medications is based on a singular set of well known beliefs: that AIDS is an infectious disease caused by a virulent virus called HIV; that HIV belongs to family of retroviruses; that AIDS can therefore be treated with antiretroviral drugs; that AIDS is a transmissible disease that is transmitted through body fluids including blood, genital secretions, and breast milk; that a positive result on the so-called "AIDS test" is indicative of infection with HIV; that once positive on the "AIDS test" the individual will develop AIDS; that a person who reacts positive on the "AIDS test" can prevent the development of AIDS by using several antiretroviral drugs; that the consumption of antiretroviral drugs will prevent the transmission of HIV from HIV positive pregnant women to their babies; that the use of antiretroviral drugs is safe and free of harmful effects; and that, therefore, it is rational to treat and to prevent AIDS with antiretroviral medications.

However, not a single one of the above beliefs can be scientifically substantiated. On the contrary, there are many scientific facts indicating that: the tests used for the diagnosis of HIV are extraordinarily inaccurate; that being HIV positive does not mean that the person is infected with HIV, the so-called "AIDS virus"; that there are more than 70 different non-HIV related reasons to have a positive result on the "AIDS test"; that the transmission and infectivity of AIDS is not real; that the risk of developing AIDS after being labeled "HIV positive" is unknown; that HIV is not the cause of AIDS;

[1] Physician, Specialist in Infectious and Tropical Diseases. Member of the Board of Directors of the Group for the Scientific Reappraisal of the HIV/AIDS Hypothesis and of Health Education AIDS Liaison, HEAL - New York.
[2] Medical Hypnotherapist. Ordained Minister. President of HEAL - New York.
[3] Journalist. Member of the Board of Directors of the Group for the Scientific Reappraissing of the HIV/AIDS Hypothesis. New York.
[4] CSW. Member of the Board of Directors of HEAL - New York.
[5] Ph.D. Professor of Mathematics. Member of the Board of Directors and Scientific Adviser of HEAL - New York.
[6] Alternative Physical Therapist. Executive Director of HEAL - New York.
[7] AIDS Activist. Member of the Board of Directors of HEAL - New York.
[8] Producer of Accent on Wellness. Owner of Planet Health. Member of HEAL - New York.

that HIV may not even exist as a virus; that what is called "AIDS" is a toxic and nutritional syndrome; that all antiretroviral drugs are highly toxic to humans; that the antiretroviral medications can by themselves cause AIDS; and that pregnant women, infants, and children are especially vulnerable to the toxic effects of antiretroviral medications.

The scientific data presented here will demonstrate that, once and for all, it is not only irrational but unethical to treat or prevent AIDS with toxic antiretroviral drugs, and that to do so is a violation of the Universal Declaration of Human Rights.

If, as claimed, there is a genuine interest in doing the best for our fellow human beings, the use of these "AIDS medications" should cease immediately. It is urgent that the entire infectious model of AIDS be reappraised.

# Introduction

The treatment and prevention of the Acquired Immunodeficiency Syndrome, AIDS, with antiretroviral drugs is singularly based on the following set of well known beliefs:

a) that AIDS is an infectious disease, that it is caused by a very virulent virus, and that this virulent virus, which belongs to the family of retroviruses, is named the human immunodeficiency virus, HIV, or simply "the AIDS virus" (1-7),
b) that AIDS can be successfully treated with antiretroviral medications (8-12),
c) that AIDS is a physically contagious disease that is transmissible through body fluids including blood, genital secretions, breast milk, etc. (13-16), and that as a consequence, is believed to be transmitted both homosexually and heterosexually, as well as from mother to child during pregnancy or after birth through breastfeeding (13-18).
d) that a positive reaction on the "AIDS test" is indicative of infection with the so-called "AIDS virus" or HIV (2,19-22),
e) that once reacting positive on the "AIDS test" the reactant person will very likely develop AIDS at some totally undetermined time in the future (3,5,7,23,24),
f) that a person infected with "the virus that causes AIDS" can delay the development of AIDS by the use of several antiretroviral drugs (10,11,25-27),
g) that the use of antiretroviral drugs may prevent the transmission of HIV from HIV-positive mothers to their fetuses (12,25,26), resulting in the worldwide promotion of their use for HIV-positive pregnant women (12,26-29),
h) that the antiretroviral drugs are safe, and that only in a few cases do they induce some light side effects (10,12,26,30), and so are promoted worldwide for children's use (31), and
i) that therefore it is absolutely rational and ethical to treat and to prevent AIDS with the use of antiretroviral drugs in pregnant women, children and anybody else (12,25,30-34).

However the above list of beliefs and assumptions, all of which collectively serve as the foundation for the infectious hypothesis of AIDS, have never been scientifically validated. On the contrary, there are many scientific facts indicating that

these beliefs are not only unfounded but that the entire model of AIDS as an infectious condition must be reappraised immediately. The following comprehensive list of facts support our statement:

## 1. The tests used for the diagnosis of "HIV infection" are highly inaccurate.

The following scientific facts support the assertion that "the tests used in the diagnosis of HIV infection are highly inaccurate":

**1.1.** 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 (35). This definition is accepted worldwide. 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" (1,4,6,23,36,37).

**1.2.** The tests that are used most frequently to diagnose HIV status are the ELISA "screening test", the Western blot "confirmatory test" and the PCR "Viral Load test" (19-22). In the United States the ELISA and Western blot tests, when done together, have become known as "the AIDS test". These tests supposedly detect antibodies against HIV. The "Viral Load" or PCR test is a genetic test that makes copies of small fragments of nucleic acids that, it is claimed, belong exclusively to HIV. These are the same tests that are used to check for HIV in mothers, infants, children, and in the population at large. The problem with all of these tests is that a positive HIV reaction does not guarantee that the person is really infected with HIV at all (38-47).

**1.3.** Currently, a positive result on "the AIDS test" - ELISA and Western blot antibody tests - is synonymous with HIV infection and the attendant risk of developing AIDS (19-22).

However, these antibody tests are neither standardized nor reproducible, with respect to HIV they are themselves meaningless because they mean different things in different individuals, they also mean different things in different laboratories and in different countries (38). They are interpreted differently in the United States, Russia, Canada, Australia, Africa, Europe and South America (48-53), 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 (54). The other problem is that the same sample of blood when tested in 19 different laboratories gets 19 different results on the Western blot test (55).

**1.4.** The Western blot antigens, proteins or bands - p120, p41, p32, p24/25, p17/18 - which are considered to be specific to HIV, may not be encoded by the HIV genome and may in fact represent human cellular proteins (38-40,46,56).

**1.5.** 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. Since HIV has never been isolated as an independent free and purified viral entity (57), it is not possible to properly define the sensitivity or the specificity of any of the tests for HIV (38). Currently, the sensitivity and the specificity of the tests for HIV are 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 (38). "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" (58). 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.

**1.6.** 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 (38-40,43,45,56). In other words, there are more than 70 scientifically acknowledged reasons for false positives when testing for HIV. This fact has been abundantly documented in the scientific literature.

**1.7.** Of course, it is shocking to find out that a diagnosis of HIV infection is based on tests that are not specific for HIV. However, the scientific evidence tells us that a person can react positive on the test for HIV even though he or she is not infected with HIV (38-40,43,47,56,59).

**1.8.** The pharmaceutical companies that make and commercialize the kits for these tests acknowledge the inaccuracy of them, and this is why the inserts that come with the kits typically state the following: "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" (58). 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" (60). The insert that comes with a popular kit to run 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" (61). The problem is that not only most AIDS researchers, journalists and lay people but health care workers themselves do not know these facts about the tests because they do not have access to them. There likewise appears to be little or no concern on the part of the knowing faculty of institutions to communicate these facts to physicians, let alone the general public.

**1.9.** Since the viral load results are given in copies per ml of plasma (61) AIDS researchers, health care professionals, and lay people may think that they represent copies or counts of the virus itself (38,62-67). However, the Viral load test only makes copies of fragments of nucleic acids. It does not count HIV itself. A positive viral load test cannot

be regarded as signifying the presence of the whole HIV genome, and therefore the test cannot be used to measure virus.

**1.10.** Results of the viral load test cannot be reproduced. This can be seen in the wide range of variability that is accepted in the quality controls set by the companies that make and commercialize the test kits. For example, Roche accepts low control having a variability between 880 and 7,900 copies per ml [Lot # 0034], and high control having a variability between 79,000 and 710,000 copies per ml [Lot # 0041] [Roche, Amplicor HIV-1 Monitor test Lot # 88618, expiration January 1999]. Most important of all, the problems with the lack of a gold standard for HIV infection also apply to the evaluation of the accuracy of the PCR or Viral load test (38,67,68). As a consequence, the specificity of the Viral load test for HIV has never been defined properly. Therefore, all viral load positive results are likewise potential false-positives for HIV.

**1.11.** People have the right to make informed choices (69-71). However, the right of informed choice implies a right to good information. There is no justification for the fact that most people have not been informed about the serious inaccuracy of the tests for HIV infection. Withholding or obscuring these facts is a serious breach of public trust, violating as it does a person's right to informed consent when making decisions about their health care. The legal implications of this situation has been noted (72).

## 2. Being "HIV-positive" does not mean that a person is infected with "HIV".

The following scientific facts support the assertion that "being HIV-positive does not mean that the person is infected with HIV":

**2.1.** There are a growing number of scientific publications explaining in detail that the tests for HIV infection are not specific for HIV (38-40,73). There are many reasons other than a past or present HIV infection to explain why an individual reacts positive on these tests. In other words these tests can react positive in the absence of HIV (38-40,43-45,56).

**2.2.** 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 gammaglobulins 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, to mention a few of them (38-40,43,44,56).

**2.3.** It is interesting to note that all of these conditions that cause the "HIV tests" to react positive in the absence of HIV, are conditions which are present with varied distribution and concentration in all of the conventionally recognized AIDS risk groups in the developed countries, as well as in the vast majority of inhabitants of the underdeveloped world. This means that in all probability many drug users [including 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 (38-40,56,74).

**2.4.** Further, it is well known that people with or at risk for AIDS have high levels of antibodies - immunoglobulins - as a consequence of having been exposed to significant quantities of a variety of foreign substances such as recreational drugs, semen, factor VIII, blood and blood components, sexually transmitted infections and other infections (38-40,75). All these substances are oxidizing agents that cause oxidative stress (73,76,77).

Recently one of us showed that all blood react positively on the ELISA test when run the test with neat or non-diluted serum (361). This can indicate that everybody has antibodies against what is supposed to be HIV. The ones that only react positively with straight or neat serum may have fewer amount of antibodies than the ones that continue reacting positively even when the serum is diluted 400 times (361), as it is usually run in the test (58). Since there is not scientific evidence that the ELISA test is specific for HIV antibodies, a reactive ELISA test at any concentration of the serum would mean presence of non-specific or polyspecific antibodies (361).

**2.5.** There is also a great deal of scientific data indicating the widespread presence of non-specific interactions between what are considered to be retroviral antigens and unrelated antibodies (38,78-80). It is then possible to conclude that the tests for HIV react positively in the presence of those antibodies; in other words, that a positive antibody test for HIV may be the result of previous antigenic over-stimulation, rather than a result of an HIV or any other retroviral infection (38-40).

**2.6.** Finally, it has been proposed that antibodies against HIV are surrogative markers for recreational drug use in the United States and in Europe (81,82).

**2.7.** Being "HIV-positive" - reacting positive on the tests for HIV - would then mean simply that the person has been exposed to many antigenic and toxic challenges, i.e., to many oxidizing agents (73). His or her immune system has been responding a lot to these immunogenic and immunotoxic challenges (77,83). The immune system of these "HIV-positive" individuals could eventually be debilitated - oxidized - after it has been over-stimulated, and therefore their risk for AIDS may be higher than those who are "HIV-negative" (75,77).

**2.8.** On the other hand, even if "the AIDS test" were able to detect antibodies to HIV, it would not be logical to say that the presence of those antibodies indicate an active infection. 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 (74,84). One can have antibodies against many germs without those germs being active, pathogenically active or even present at all (84,85). In most instances, antibodies against viruses indicate immunity. This is the very basis of vaccination against viral diseases (74,84,86). Even if the tests were specific for antibodies against HIV, the question would then be the following: Why is it that only in the case of AIDS the presence of antibodies indicates the presence of disease, rather than protection against it ?

**2.9.** There is no justification for the fact that both patients and the general public have had all of the preceding facts withheld from them. Without the merits and demerits of the tests for HIV, people cannot make informed decisions.

## 3. The transmission and infectivity of AIDS is not real.

The following scientific facts support the assertion that "the transmission and infectivity of AIDS is not real":

**3.1.** Today, there is an alarming worldwide increase in toxic agents environmentally, in the workplace and in the home. Many new diseases can be attributed to exposure to these agents (87). The same is true for people at risk for AIDS. Different AIDS risk groups develop the same AIDS related diseases simply because they are exposed to the same agents - the same toxins or stressors - and not because they get or transmit a new virulent germ.

**3.2.** Within the groups at risk for AIDS, the trends of AIDS incidence parallel the trends of immunological stressor agents (73,76,81,82,87,88).

For example, drug-addicted gay men who develop AIDS are usually exposed for long periods of time to alcohol, drugs, nitrite inhalants, sperm, STD's, other infections, anti-infective therapy, mental distress, and malnutrition (77,81,83,87,88), all immunological stressor agents that although sexually related, are not sexually transmitted. In hemophiliacs blood, factor VIII, infections, anti-infective therapy, and mental distress are present (77,83,87,89,90); all these are immunological stressor agents that are related to their basic illness and not only to the transfusions. In babies born to drug addicted mothers immunological stressors like alcohol and other street drugs, congenital infections, and anti-infective therapy are present and can alter the baby's immune and other systems (77,81-83,87). In the "third world", malnourished mothers transmit malnutrition to their babies with all its adverse immune and biological consequences (83,91).

All these immunological stressor agents are enough to weaken and even destroy the immune and other systems. One does not need HIV - or any other germ - to either perform or explain this destructive job (75,77,81-83,88,92).

**3.3.** The homosexual transmission of AIDS (93) is an assumption based on the high frequency of AIDS in a very specific group of drug-addicted gay men (94).

The belief in the heterosexual transmission of AIDS in Africa is also unsubstantiated (91,95-98). It is based on the fact that in Africa both men and women have the same possibility of developing "AIDS". However, the conditions of life in Africa are bad for both men and women and instead of getting better, every day these conditions are getting worse (83). Very often, the diagnosis of "AIDS" in Africa is indistinguishable from the rampant symptoms of common tropical diseases (91,93,99). In Africa, both men and women are exposed to the same immunological stressor agents. Therefore, no matter the sexual preference, everybody in Africa is at risk for AIDS. The same is true in places with similar conditions to Africa.

**3.4.** In the early 80's it was postulated that HIV was a highly contagious virus. However, now it is even accepted by mainstream researchers (94,100-102) that seroconversion depends upon as many as 1000 sexual contacts of vaginal intercourse, and from 100 to 500 contacts for anal intercourse (81,82).

Note also that seroconversion from HIV-negative to HIV-positive can occur in the absence of sexual transmission because of continued exposure to immunological stressor agents - oxidizing agents - during sexual activities, such as pharmaceutical aphrodisiacs (38-40,73,76,77,83).

**3.5.** Mainstream researchers do not consider immunological stressor agents as risk factors for AIDS (83,92,94,103,104). They do not feel the need to do that. In fact, everybody seems to be hypnotized by HIV, using as they do an HIV diagnosis not to reveal, but to conceal these risks, the true etiologic, or causal factors for AIDS (105-111).

**3.6.** If AIDS were a physically contagious disease, an exponential growth of cases would be seen in the general population, at least during the early phase of the outbreak. But instead, AIDS remains confined to the same groups in which it was first observed (94,103).

**3.7.** There is still not a single case of AIDS acquired by health care professionals at their work sites (81,82,94,112). In all the alleged cases it is found that the health care worker who tested positive for HIV, or who developed AIDS, did so due to their exposure to immunological stressors other than HIV (77,81-85).

**3.8.** There is still no scientific proof of the wife of a hemophiliac with AIDS contracting the syndrome from her husband (89,94,113,114).

**3.9.** The perinatal transmission of HIV from HIV positive mothers to their babies is likewise theoretical. Even the HIV/AIDS supporters agree that vertical transmission of HIV is very inefficient (115).

**3.10.** UNAIDS, UNICEF and the WHO, all agencies of the United Nations, are currently promoting a worldwide campaign to stop HIV-positive mothers from engaging in the healthy practice of breastfeeding their babies with the pretext that in this way it is possible to prevent the transmission of HIV (116-118). However, after carefully studying 167 publications on this issue, a recent review states clearly that, "From the database analysis, we know that the relative role of breastfeeding in the epidemiology of AIDS is still uncertain" (119). The transmission of HIV through breast milk is a scientifically invalidated assumption (120-122).

It is both illogical and counter intuitive to discourage HIV positive mothers from breastfeeding their babies. To do so is a dangerous and erroneous policy based on an unproven assumption. This is a violation of both the right of women to breastfeed their babies, and the right of babies to be fed with the breast milk of their mothers (123).

The logical conclusion of the foregoing is that the transmission and infectivity of AIDS has never been scientifically validated. It is merely an assumption that has morphed into dogma.

## 4. The risk of developing AIDS after being labeled "HIV positive" is unknown.

The following scientific facts support the assertion that "the risk of developing AIDS after being labeled HIV positive is unknown":

**4.1.** It is believed internationally that once receiving a positive result on the "AIDS test", an individual will develop AIDS at some moment in the future, this despite the fact that the latency period has increased every year since 1985 (13,16). However, the equation "HIV = AIDS" has never been scientifically validated. Even the pharmaceutical company that makes and commercializes the most popular test to run the ELISA test for HIV warns in the test kit, "The risk of an asymptomatic person with a repeatable reactive serum sample developing AIDS or an AIDS-related condition is not known" (58,124).

**4.2.** According to the claims made at the 12$^{th}$ World AIDS Conference in Geneva, there are 31 million people throughout the world who are HIV positive (125). The immense majority of these people are absolutely healthy, and this is the reason why these persons are called "Long term survivors" or cases of "non-progressive HIV infection" (6,126).

**4.3.** Even mainstream researchers state that "5-10% of HIV-infected people live for 10 years - perhaps 20 or more years - without developing AIDS-related symptoms or having any laboratory evidence of progression to AIDS" (126,127).

**4.4.** Mainstream AIDS researchers are looking at the absence of the so-called "cofactors" in these "long term survivors" or "non-progressive HIV infections". These "cofactors" include "other sexually transmitted diseases, drug use, nutrition and stress", as well as genetic factors to try to explain this fact (126-130).

**4.5.** It is also important to remember that in the scientific literature there are more than 5,000 individuals that have AIDS and are HIV-negative (131-135). These patients are dying from AIDS-related diseases that are not called "AIDS" because they are HIV-negative. In reality, they are dying from conditions caused by the same agents as the HIV-positive cases diagnosed as AIDS (74,82,103). The supporters of the HIV-AIDS model arbitrarily decided to call these HIV-negative AIDS cases "Idiopathic CD4 T-lymphocytopenia" (131,133).

**4.6.** The mortality in AIDS has been related to the presence of other factors such as the use of street-drugs, anti-viral medications, and not to HIV by itself (136).

**4.7.** In both HIV-positive and HIV-negative AIDS cases, it is always possible to find a variety of "cofactors" to which the patients were exposed, generally for long periods of time and always prior to the development of clinical AIDS (81-83,92). Since all these "cofactors" are well known agents able to cause immunodeficiency (73,76,77,81,104,137), it is much more correct to call them immunological stressor agents (77,83,104,137).

**4.8.** It is also interesting to note that there are some examples of HIV-positive individuals seroconverting to a negative state, then remaining that way for years (138,139).

**4.9.** As was stated above, reacting positive on the tests for antibodies to HIV most likely means that the person has been exposed to many antigenic and toxic challenges, i.e., to many oxidizing agents (38,73,76,77,83,92,137). His or her immune system has been responding to too many immunogenic and immunotoxic challenges (75,77,83). It is in this way that the immune system of "HIV-positive" individuals may be more debilitated - oxidized - than those who are "HIV-negative". And it is in this way that the risk for AIDS in "HIV-positives" may be higher. In other words: it is the exposure to immunological stressor agents that cause an individual to react positive on "the AIDS test". And it is this exposure which, if not stopped, could eventually cause the "HIV-positive" individual to go on to develop AIDS (75,77,83).

# 5. HIV is not the cause of AIDS.

The following scientific facts support the assertion that "HIV is not the cause of AIDS":

**5.1.** Most people believe that AIDS scientists know the mechanisms by which HIV destroys the immune system and causes AIDS. However, after more than a decade of "HIV science", no one has the answer for this basic question. The ways in which HIV

supposedly destroys the immune system (1,3,140) are highly speculative (74,77,92,103,141-143).

In a recent survey done by the journal Science, it was found that the main question that bothers most of the mainstream AIDS researchers is how it is that HIV destroys the immune system and causes AIDS (144).

**5.2.** Within both lay and scientific circles it is virtually unknown that for more than a decade, there has been a scientific debate about the etiology or cause of AIDS. In the March 1987 issue of the journal Cancer Research, Peter Duesberg from the University of California wrote his first article questioning the infectious model of AIDS (141). Since then many scientific articles, documents, and books have been written in different countries by dissident researchers and AIDS activists, trying to get a reappraisal of the unproven viral hypothesis for AIDS (75,88,92,105,106,113,141,143,145-151).

**5.3.** There are many scientific facts which show that HIV fails to fulfill the epidemiological, biological, even the common sense requirements to be the cause of AIDS (73,75,113,143,148-153).

**5.4.** HIV is neither necessary nor sufficient to cause AIDS, nor does it always precede the development of the syndrome (94,104,154). This is demonstrated by thousands of AIDS cases that are HIV negative (131,133,155). It is also demonstrated by a host of HIV-positive people who are absolutely healthy and have never developed AIDS (74,104,113,146,154,156,157).

**5.5.** There are many individuals who first develop immunodeficiency and only later they become “HIV-positive” (158-162). It is however, a natural law that in all situations, including human diseases, the effect comes after cause.

**5.6.** The scientific data do not prove that HIV preferentially destroys T4 cells or has any cytopathic effect; they do not demonstrate that T 4 cells are preferentially destroyed in AIDS patients, they do not demonstrate that T 4 cell destruction is either necessary or sufficient as a prerequisite for the development of AIDS (92,103).

**5.7.** HIV like all retroviruses, has never been proven to be a pathogenic agent; therefore it cannot explain the immunological alterations, pathogenesis, natural history nor different clinical forms within the groups of people that develop AIDS (77,92,94,103,104,141,146).

**5.8.** Since it has never been proven that HIV can cause AIDS, the investigators that enthusiastically defend HIV as its cause have proposed a vast variety of agents as helpers or “cofactors” in the genesis of AIDS (1,140). However, these “cofactors” are by themselves causal agents of immunodeficiency and can generate AIDS with or without the presence of HIV (77,81,92,137,146). Again, it is for more accurate to call the “cofactors” primary immunological stressor agents (74,77,83,87,104,137,154). They are

the real risk factors for AIDS. They are the etiological or causal factors of AIDS. They are the cause of AIDS.

**5.9.** It simply goes against common sense to propose an infectious cause of AIDS. The new and real circumstances that surround all the groups of people that develop AIDS with the greatest frequency is their exaggerated exposure in the last decades to a variety of stressor agents that have a chemical, physical, biological, mental or nutritional origin (77,104,137). People who develop AIDS are exposed both voluntarily and involuntarily to immunological stressor agents that are unique to either their conditions of life, or to their style of life (81-83,94,163).

**5.10.** The toxic and non-infectious nature of AIDS has been suspected since 1981, when the very first publication on it announced the first 5 cases (164).

# 6. The so-called "AIDS virus", HIV, may not even exist.

Biophysicist Eleni Papadopulos-Eleopulos and her group of researchers at Royal Perth Hospital in Perth, Western Australia, were the very first scientists to mention the fact that HIV has never been isolated (38). For several years Papadopulos-Eleopulos and co-workers have been publishing papers where they have described in detail the scientific facts that support the assertion that "the so-called AIDS virus, HIV, may not even exist" (38-40,46,56,57,73,76,168,175,177,179):

**6.1.** The correct procedures (57) employed for over half a century to achieve isolation of a retrovirus are: a) to find in infected cell cultures particles with a diameter of 100-120 nM that contain the so-called condensed inner bodies or cores and that have surfaces studded with projections - spikes, knobs - b) In sucrose density gradients the particles band at a density of 1.16 gm/ml; c) At the density of 1.16 gm/ml there is nothing else but particles with the morphological characteristics of retroviral particles; d) The particles contain only RNA and not DNA, and the RNA consistently has the same length [number of bases] and composition no matter how many times the experiment is repeated; e) When the particles are introduced into secondary cultures they are taken up by the cells, the entire RNA is reverse transcribed into cDNA, the entire cDNA is inserted into the cellular DNA, and the DNA is transcribed back into RNA which is then translated into proteins; f) As a result of e the cells in the secondary cultures release particles into the culture medium; g) The particles released into the secondary culture medium have exactly the same characteristics as the original particles, that is, they must have identical morphology, band at 1.16 gm/ml and contain the same RNA and proteins (57).

None of these procedures have been achieved in the case of HIV (38,40,57,73).

**6.2.** None of the researchers who claim to have isolated HIV have shown the presence of particles with the morphological characteristics of retroviruses banding at 1.16 gm/ml (57).

Even the word "isolation" as used by the most noted researchers (165-167) is incorrect and misleading since neither Montagner, Gallo nor Levy isolated HIV particles,

particles of any other human retrovirus, or even virus-like-particles at all (38-40,56,57,73,142,168-174).

**6.3.** Since no "retroviral particles" [retroviruses] have ever been isolated from any culture (38-40,57,73,168-177), the existence of HIV has been established indirectly: by the presence in blood cultures of AIDS and "HIV-positive" individuals, proteins/glycoproteins such as gp 160/150, gp120, gp41/45/40, p34/32, p24, and p18/17, each claimed to belong to HIV; by the presence of enzymes such as reverse transcriptase that supposedly belongs to HIV; and by the presence of RNA or DNA fragments that supposedly belong to HIV (38-40,57,73,168-175).

However, none of these substances have been proven to belong to HIV at all (38-40,57,73,168-175). How can anybody prove that the substances found in those cultures belong to a viral particle that has never been found at 1.16 gm/ml? To prove that those substances are part of a retrovirus named HIV, it is absolutely necessary that the retroviral particles have been previously separated - isolated - from everything else. This has never been done with HIV (57).

**6.4.** It is interesting to note that the substances listed in 6.3. are claimed to appear exclusively when one co-cultures supposedly infected blood with abnormal cells from leukemic patients, or from umbilical cord lymphocytes (57). The problem is that the same substances can be obtained from the same cultures in the absence of the supposedly HIV-infected blood (57).

**6.5.** The cultures where the above substances have been found are cultures that have been heavily stimulated with substances such as phytohemagglutinin, IL-2, antiserum to human interferon, and other agents (57). These culture stimulants are oxidizing agents (57,73). The problem is that the same type of material can be observed in stimulated cultures of lymphocytes from healthy persons (57,178).

It is interesting to note than in the presence of antioxidants, no HIV phenomena can be observed in culture; nor can HIV substances be found (38,178,179).

**6.6.** The substances listed in 6.3. are not specific to HIV at all (57). For instance, it is currently known that reverse transcriptase can be found associated with entities other than retroviruses, including eukaryote cells, some animal and plant DNA viruses, and even some introns (180).

Gallo and co-workers have claimed that the cell-free supernatants from "infected" cultures have HIV-DNA (181,182). They forgot that by definition retroviruses are infectious particles which contain only RNA. When retroviruses enter a cell the RNA is reverse transcribed into DNA, which is then integrated into cellular DNA as a provirus, which means that "HIV DNA" will be present only in the cell and no where else (57).

There is also ample evidence that any RNA or DNA present in the supernatant of the cultures is there as an effect of stimulation by polycations and oxidizing agents, rather than as an effect of the presence of a retrovirus (57).

"HIV cloning" is likewise misleading. Without isolating a retroviral particle containing RNA inside its core, the cloning of that "specific HIV-RNA" is not possible (57).

**6.7.** To date nobody has presented evidence that the so-called HIV proteins or antigens [gp160/150, gp120, gp41/45/40, p34/32, p24, p18/17], are constituents of a retrovirus particle or even retrovirus-like particle let alone a unique retrovirus, HIV (57).

**6.8.** The proteins or antigens derived from stimulated cultures form the basis for the ELISA and Western blot HIV antibody tests (57,173). Fragments of RNA from stimulated cultures form the basis of the HIV viral load test (57,173). This is the main reason why the current tests used for the diagnosis of HIV are not specific for it (38-40,57,168,175).

**6.9.** In the January 1997 issue of the journal Virology, two independent groups of researchers published experiments claiming to isolate HIV. Now and for the first time in the history of HIV, the researchers followed the internationally accepted procedures to isolate retroviral particles. Not surprisingly, in the sedimented bands at 1.16 gm/ml of sucrose, where retroviruses are known to be located, nothing was found but cellular debris. At 1.16 gm/ml there was nothing that even looked like a retroviral particle (183,184). They could not have isolated HIV simply because HIV was not there to be isolated.

It has been proposed that all those substances that indicate the existence of HIV are nothing more than non-viral material altogether, induced by the agents to which the AIDS patients and cultures are exposed (57). When found in people, these substances would be seen as regular products of the stress response (185), secondary to exposure to chemical, physical, biological, mental, and nutritional stressor agents (74,77,83,87,104,137, 148,154).

**6.10.** It is therefore possible to conclude that the entire model of AIDS as an infectious and transmissible viral disease has its basis on a non-existing organism. The foundation stone for the HIV-AIDS model then, is a ghost.

## 7. “AIDS” is a toxic and nutritional syndrome.

The following scientific facts support the assertion that “AIDS is a toxic and nutritional syndrome”:

**7.1.** The toxic nature of AIDS was suspected since the announcement of the very first five cases of AIDS in Los Angeles: “All 5 reported using inhalant drugs” (164).

**7.2.** In the early 80’s, researchers proposed the possibility that drugs were the cause of the new disease first diagnosed in young drug-using gay males (186-189). Nitrite inhalants or “poppers” were the recreational drugs suspected of being the culprit (186,190).

This logical hypothesis was supported by studies demonstrating the immunotoxic and carcinogenic effects of nitrite inhalants (158,191-193). Also the first

epidemiologic studies both in Europe and the United States linked AIDS to inhaled nitrites and to other recreational drugs such as cocaine and amphetamines (194,195).

**7.3.** The hypothesis that street drugs could cause AIDS was then known as “the lifestyle hypothesis” (189).

In 1983, just a year after the first cases of AIDS were announced, researchers from the CDC abandoned the lifestyle hypothesis in favor of a transmissible agent (196). They even conducted research to try to prove that the lifestyle hypothesis was wrong (195,197).

**7.4.** Early during the HIV era, John Lauritsen and Frank Buianouckas were some of the first who began to warn about the possibility that recreational drugs were the real cause of AIDS (147,163,198).

Peter H. Duesberg, the retrovirologist from the University of California at Berkely named this possibility the “drug-AIDS hypothesis” and has used very elegant and detailed arguments to describe it (81,82,94,145,146,156,199,200).

Supporters of the HIV-AIDS hypothesis have recently published attempts to falsify the drug-AIDS hypothesis (201,202). However, neither of these efforts was able to show a single case of AIDS without previous exposure to recreational or antiretroviral drugs in the developed countries (201,202) The HIV believers have gone even further in their attempt to diminish the etiologic role of drugs with assertions such as “heroin is a blessedly untoxic drug” (203).

**7.5.** Almost 100% of the gay male AIDS cases occur within gay male drug users (82,153,204,205).

Many reports link AIDS and more specifically Kaposi’s sarcoma to the use of nitrite inhalants, otherwise known as “poppers” or the “gay drug” (152,163,192,193,201, 206,207).

Also, a large proportion of gay men are now using steroids cosmetically (208).

About 30 % of the AIDS cases in Europe and the United States occur in intravenous users of cocaine, heroine, and other drugs (82,209). Practically all of the female and heterosexual AIDS cases in the developed world are intravenous drug users (82,210).

Supporters of the HIV-AIDS hypothesis reject drug use as a risk factor for AIDS, going only so far as to accept it as a risk factor for “unsafe sex” (211,212).

**7.6.** About 1% of all AIDS patients in the developed countries are intoxicated babies born to drug addicted mothers (209,213-218).

Intoxicated babies “used drugs” because their mothers used drugs while carrying the fetus (219,220). It is interesting to note that T-cell levels of these babies went up to “normal” one or two years after birth, despite being HIV-positives. The ones who did not recover were the ones treated with AZT (219).

In Europe, most of the babies born to drug-addicted mothers are able to recover from bacterial infections, pneumonia, yeast and cryptosporidial infections, despite being HIV positive, and stay healthy at 6 years of age (213,221).

In the European study, 40% of the children died. Those who died were exactly the same ones who were treated with AZT (213,214).

**7.7.** The HIV/AIDS supporters accuse us of not being able to differentiate cause from confounding factors (222). We think that it is time for them to start watching their own epidemiological steps.

The epidemiological association between chemical, physical, biological, mental and nutritional immunological stressor agents and AIDS is easily demonstrable (74,77,83,87,104).

One can always find immunological stressors acting as etiologic factors in AIDS [predisposing, starting, and keeping risk factors]. They are always present in the groups of people developing the syndrome: drug abusing gay men, IV and non-IV drug users and alcoholics, prostitutes, babies born to drug-using or malnourished mothers, hemophiliacs, users of antiretroviral medications, AIDS-phobic people, Central African, Caribbean and similar people, African and Hispanic Americans, and in individuals exposed to immunological stressors in their work places (77,83).

**7.8.** Malnutrition is known as the world's first cause of immunodeficiency (223). Poverty is the main risk factor for malnutrition. Economical disparities have increased all over the world, but mainly in Africa, Asia, Latin America, and the Caribbean, as well as in the large impoverished strips of the developed cities. Never before has poverty been so prevalent and intense, nor has affluence been so big and concentrated in the hands of so few (87,224,225).

Recreational drugs also induce suppression of appetite and fatigue (81,226-229).

**7.9.** There is an enormous amount of scientific evidence showing the cytotoxic and immunotoxic properties of all recreational drugs (158,193,226,229-232). Recreational drugs can also act as carcinogens in animals and humans (233).

The immunosuppressive effects of recreational drugs are decreased after stopping their use. This immunological improvement has been recorded for both adults (234-237) and babies after birth (213,219,238).

The degenerative effects of immunological stressors on the immune system network have well known immunotoxic and immunogenic mechanisms (75,77,137). At a molecular level, these immunotoxic and immunogenic effects generate a state of oxidative stress (73,77,179,239), which is generally concentrated in the mitochondria (240,241). A causal relationship between immunological stressors and AIDS has been documented (77).

**7.10.** The metabolic disturbances, infections, opportunistic infections and tumors seen in AIDS patients are a consequence of the action of immunological stressor agents on the immune and other human systems (75,83Giraldo 1997c).

The varied clinical manifestations of AIDS, otherwise known as "AIDS-defining diseases", are different for each group of people developing the syndrome, simply because different stressors generate different diseases. Each group of people at risk for AIDS is exposed to group specific stressors (75,82,83,89,242-244).

**7.11.** AIDS is a new syndrome because in developed countries the recreational drug epidemic is new (87,217,218,245-247), and because never before have the people of Central African countries and similar countries in the under developed world been as poor and malnourished as they are now (83,87,224,225).

Street drugs and malnutrition are the main etiologic risk factors for AIDS in both developed and in under developed countries.

The recreational drug epidemic is reaching such a high level in the United States that almost 80% of all one dollar bills have detectable amounts of cocaine, since the bills have been rolled for drug inhalation (248).

**7.12.** In short, AIDS is a severe acquired immunodeficiency due to multiple, repeated, and chronic exposures to immunological stressor agents, with degenerative immunotoxic and/or immunogenic effects on immunocompetent cells and immunological chemical reactions. These progressive and continuous assaults on the immune system network bring the individual into a functional immunological deficit, with the subsequent appearance of infections, neoplasias, and metabolic conditions, all leading to a probable early death. Therefore AIDS rather than being an infectious syndrome, is a chronic degenerative toxic/nutritional one (83,104).

**7.13.** At the molecular level, AIDS is caused by an excess of reactive free radicals, specially oxidizing agents (73,77,179).

## 8. All antiretroviral drugs are highly toxic to humans.

The following scientific facts support the assertion that “all antiretroviral drugs are highly toxic to humans”:

**8.1.** After more than a decade of treating and trying to prevent AIDS with antiretroviral therapies, neither individual nor public health benefits have been achieved (200,249,250).

**8.2.** Zidovudine [AZT], the most popular of the AIDS medications, was originally developed for chemotherapy in cancer, but due to its toxicity it was never approved for human use (251). AZT is now licensed by the Food and Drug Administration [FDA] as an anti-HIV medication (81,252,253).

AZT is a potent cytotoxic DNA chain-terminator (81,82).

The toxicity of AZT, the drug now prescribed indefinitely to both healthy HIV-positive individuals and to AIDS patients, has been solidly documented (81,88,152,198,254-257).

AZT is highly toxic to human cells, including T4 lymphocytes, at the “antiretroviral” dosage recommended by the manufacturer (256).

The immunotoxicity of AZT, as well as its myelotoxicity [toxicity to the bone marrow], are very well recognized (258). Granulocytopenia [decrease of white blood

cells called granulocytes] is one of the most common effects seen in persons treated with AZT (259,260).

There are also very well documented investigations showing that AZT has carcinogenic properties with respect to fast growing human and animal immune and other cells (256). In humans, AZT increases the risk of lymphomas by 50 times (261). And AZT has been confirmed to be carcinogenic in mice (262-264). However, AZT is sold in the United States, where it is illegal to sell drugs that are carcinogenic (114,263,265).

AZT can also cause anemia, lymphocytopenia, hepatitis, pancreatitis, myositis, muscle atrophy, wasting disease, dementia, lactic acidosis, severe hepatomegalia with steatosis, vasculitis, and it prevents mitochondrial DNA synthesis (266-270).

The toxicity of AZT is so well documented that the pharmaceutical company that makes and commercializes it typically writes, “Retrovir (Zidovudine) may be associated with severe hematologic toxicity including granulocytopenia and severe anemia particularly in patients with advanced HIV disease” and they add that, “Myopathy and myositis with pathologic changes similar to that produced by HIV disease, have been associated with prolonged use of Retrovir” (253).

The use of AZT for pregnant women can induce abortion, congenital malformation such as cavities in the chest, abnormal indentations at the base of the spine, misplaced ears, triangular faces, heart defects, extra digits and albinism (271). This toxicity for embryos has also been documented in animals (272).

The American National Institute of Child Health and Human Development has warned about the toxicity of AZT for children (273). It is recognized that AZT impedes normal child growth and development (273).

AZT can also destroy non-growing cells, such as neurons and muscle cells (270), thus causing muscle atrophy (266,275-280), and dementia (269,281).

It is well known that a great deal of illegal actions were carried out to achieve the 1987 FDA marketing approval of AZT (282).

**8.3.** The toxicity of AZT can be potentialized by other DNA chain terminators such as gancyclovir and acyclovir, drugs that are frequently prescribed together with AZT in the treatment and prevention of opportunistic viral infections (283,284).

**8.4.** Currently, the HIV-AIDS supporters are prescribing hydroxyurea, an inexpensive drug used for chemotherapy of leukemia (285). This too is an inhibitor of DNA synthesis.

**8.5.** The toxicity of the new protease inhibitors, prescribed as part of the so-called AIDS treatment “cocktails”, is also well documented (286).

The “cocktails” contain a protease inhibitor in conjunction with two DNA chain-terminators (286).

Researchers have been documenting that persons on protease inhibitors are developing abnormal fat accumulations, termed “buffalo humps” and “crixbelly” (287-289).

The hepatotoxicity of protease inhibitors has also been documented (290). Dogs and rats treated with protease inhibitors develop hepatic cell necrosis 30 minutes after administration of the drug (291).

As time passes, more and more metabolic and endocrine disturbances are described in individuals placed on protease inhibitors. Recent studies report hypertrophy of the breasts; increase of blood sugar [diabetes], cholesterol, and triglycerides; abnormal subcutaneous and visceral fat accumulation; peripheral fat wasting and lipomatosis; pancreatitis and angina (287,288,292-294). Hypertriglyceridemia [high level of one of the fat components of blood] is being described in 79% of the individuals taking protease inhibitors (295).

It has even been documented that protease inhibitors can induce the development of AIDS-defining diseases such as mycobacterial infections (296).

All these effects of the protease inhibitors are taking the edge off cocktail euphoria (297).

The scientific evidence shows that antiretroviral drugs are highly toxic to both humans and animals.

## 9. Antiretroviral drugs can by themselves cause AIDS.

The following scientific facts support the assertion that the "antiretroviral drugs can by themselves cause AIDS":

**9.1.** Many healthy HIV-positive individuals along with AIDS patients, are being placed on lifetime prescriptions of nucleoside analogues that act as DNA chain-terminators, such as AZT, the analogue of the nucleoside thymidine (25,26).

Currently, protease inhibitors are being prescribed as anti-HIV medications for the lifetime of the individual (11,27).

All the drugs that are currently used as antiretroviral medications are drugs that act specifically on cells that are either metabolically active or in constant division (298). By definition, the immunocompetent cells, as well as the bone marrow cells, are cells that are dividing constantly. A very unique characteristic of the cells of the immune system is that they have to divide during the immune response (299,300). This makes the cells of the immune system much more vulnerable to the actions of these chemicals.

All the antiretroviral medications are known to be very toxic chemicals (81,114,143,152,301).

The toxic effects of AZT on people's immune systems have been documented (302). AZT was given to 14 healthy health care professionals who were exposed to AIDS blood through needle sticks and similar accidents. Fully half of the 14 health professionals had to quit the drug because of severe toxic effects. Neutropenia [low count of one type of white blood cells] developed in 36% of the 11 persons who completed at least 4 weeks of AZT treatment. 5 of the 14 individuals could not even make it to four weeks due to "severe subjective symptoms". One professional had to be stopped prematurely because his neutropenia was so severe that he developed a respiratory infection. These toxic effects developed in only weeks, while persons with an HIV-positive diagnosis often take AZT for years (302).

**9.2.** There is a great deal of scientific evidence showing that the antiretroviral drugs can induce the development of AIDS-defining diseases. The possibility that AZT may actually contribute to the pathogenesis of AIDS is real (77,81,88,137,152,256,257).

The British-French Concorde trial found that AZT was unable to prevent AIDS, and instead increased mortality by 25%, compared to the untreated controls (303).

Another British study found that AZT prophylaxis decreased survival and induced wasting syndrome, cryptosporidiosis, and cytomegalovirus infection (304).

The American MAC study shows that AZT increases the risk of pneumonia, one of the AIDS defining diseases (305).

Studies often show that individuals given AZT have a worse prognosis (82), but the main stream researchers prefer to blame HIV (306).

The lymphocyte counts decreased significantly in humans treated with AZT, but not in the non-treated controls (266,307). Interestingly, these are the experiments that the Food and Drug Administration Office evaluated before the licensing of AZT (81,82,88,143).

Another study similarly found that AZT users experienced more rapid CD4+ cell depletion (308).

Prophylactic AZT has also been shown to increase significantly the risk of AIDS in hemophiliacs when compared with the untreated controls (159).

Since AZT use has begun, the mortality of British HIV-positive hemophiliacs has increased 10-fold (309).

A similar finding has occurred with American hemophiliacs (310). However, most of the AIDS researchers insist on blaming HIV (309-312).

**9.3.** The immunological alterations secondary to antiretroviral therapy and described in section 8, can be reversed after individuals stop taking these medications. 10 out of 11 individuals recovered their cellular immunity after stopping AZT (313).

Even patients suffering from severe pancytopenia and bone marrow aplasia recover after discontinuing AZT (254).

Clinical manifestations of mycobacterial infection started 1-3 weeks after starting the protease inhibitor Indinavir. Symptoms disappeared after the patients stopped the medication (296).

Two babies born to mothers treated with AZT for 6 months and then treated themselves for an additional month and a half, developed *Pneumocystis carinii* pneumonia, one of the clinical manifestations of AIDS. Since the babies were HIV-negative, AZT was suspended and they completely recovered, remaining healthy beyond the one year period of observation (115,314).

**9.4.** Merck itself, the pharmaceutical company that produces and commercializes the protease inhibitor Crixivan warns, “It is not yet known whether taking Crixivan will extend your life or reduce your chances of getting other illnesses associated with HIV” (315).

**9.5.** In animals, there are several examples of immunotoxicity due to antiretroviral medications:

Rats and mice treated with AZT for 7 weeks developed anemia, neutropenia, lymphopenia, thrombocytopenia, bone marrow depletion and weight loss (316).

In a similar experiment, mice were also treated with AZT for 7 weeks and developed anemia, leukopenia, thrombocytopenia and myelodysplasia (317).

Hamsters treated with AZT for one or two weeks developed T-cell depletion and atrophy of the thymus (318).

Mice treated with the drug for 2 weeks developed anemia, nephrotoxicity, and lymphotoxicity (319).

AZT is also toxic to the liver (320).

The carcinogenic properties of AZT have been documented in animal experiments (318). AZT can stimulate leukemias (317).

**9.6.** Besides the antiretroviral drugs, healthy people who are "HIV-positive" are taking lots of prescribed antibiotics, anti-mycobacterials, antifungals, antivirals, antidepresants, as well as many over-the counter medications (321,322). All are potentially immunotoxic stressor agents (77), and all help in generating AIDS (83).

The HIV-AIDS supporters will always have the excuse that HIV is mutating and developing resistance to the current medications. However, there is no scientific substantiation for the assertion that "HIV is mutating" (323).

**9.7.** AIDS patients are also taking a polypharmacy of immunotoxic medications (82) that, rather than improving, very often debilitate the patient's immune and other systems, and therefore contribute to the eventual death of the individual. Medications such as metronydazol, pyrimethamine, daraprim, amphotericin B, clotrimaxole, dapsone, interferon, pentamidine, vincristine, fluocytosine, adriamycin, vinblastine, to mention some of the more frequently used, are potent immunotoxic, myelotoxic, lymphotoxic, nephrotoxic, hepatotoxic drugs (77,284).

**9.8.** It is unethical, to say the least, to treat or prevent AIDS with medications known to be highly toxic to the cells of the immune system, of the bone marrow, and to the cells of other tissues and systems. The mainstream AIDS researchers are simply trying to stop the fire with gasoline.

## 10. Pregnant women, infants, and children are much more vulnerable to the toxic effects of antiretroviral drugs.

The following scientific facts support the assertion that "pregnant women and children are much more vulnerable to the toxic effects of antiretroviral drugs":

**10.1.** For decades, medical science has known that growing cells are much more vulnerable to the toxic effects of many different agents (324.325). This has been the very basis for the effort to avoid exposing as much as possible, pregnant women and their fetuses to any potential toxic agent (326,327).

It is also important to keep in mind that the immune system of a child only attains its own maturity after the age of ten (299,300).

**10.2.** However, in the era of AIDS, AIDS researchers are changing all the rules. Currently, toxic medications are recommended and prescribed worldwide to pregnant women and children (328,329). As of 1993, even HIV-free babies are taking AZT: this is

because HIV-positive pregnant women are prescribed AZT for the last two trimesters in the hope of preventing HIV transmission from mothers to babies (115).

Babies who test “HIV-negative” but who are born to HIV-positive mothers, are prescribed AZT anyway for six weeks after birth (115,328,330).

**10.3.** Many HIV-positive healthy newborns, infants, and young children are placed on combinations of potentially immunotoxic medications such as antiretrovirals, antifungals, antivirals, and antibiotics. All are currently prescribed indefinitely as prophylactic drugs (31,331).

It is as if they have forgotten the vulnerability of newborns and young children to toxic substances (332).

**10.4.** The toxicity of antiretroviral drugs for embryos and fetuses has been documented both in humans and animals, as well as *In Vitro*:

AZT is a potent cytotoxic DNA chain-terminator (81,82) and “it has been well known for many years that the compounds which can alter DNA metabolism often exhibit pronounced prenatal toxicity” (298).

The use of AZT for pregnant women can induce abortion, congenital malformation such as cavities in the chest, abnormal indentations at the base of the spine, misplaced ears, triangular faces, heart defects, extra digits and albinism (271). In some instances intrauterine growth retardation has been documented (333). The hemoglobin at birth in infants exposed to AZT was found to be significantly lower than in the placebo group (115,334,335).

The American National Institute of Child Health and Human Development is well aware of the toxicity of AZT (273). AZT has been shown to impede normal child growth and development (273).

The toxicity of AZT in animal embryos has been recognized; if used before the implantation of the embryos, the effects seem to be even worse (272).

When administered to pregnant mice, AZT reduced the number of fetuses by 60%, altered the liver of newborns, and caused a significant reduction of hematocrit in the pregnant animals (336). A similar experiment with pregnant mice also showed a significant reduction in the number of fetuses (337). These effects are worse if mice embryos are preimplanted (338).

There are also *In Vitro* data documenting the toxicity of AZT: it induces reduction in the number of thymocytes in cultured thymic lobes from rat fetuses (339). It inhibited the erytroid colony formation of liver cells from mouse fetuses (320). Also, exposure of two-cell mice embryos to zidovudine was consistently associated with significant inhibition of blastocyst formation (340).

**10.5.** A recent comprehensive review of this issue concluded: “Sufficient data regarding the safety of zidovudine in human pregnancy are not available” (298).

In spite of the scientific evidence about the toxicity of AZT for pregnant women, a review of the issue by the National Center for Toxicological Research of the Food and Drug Administration [FDA] states that, “Initial human studies suggest that maternal use of AZT during pregnancy is very well tolerated by both mother and child and provides a promising degree of protection from vertical HIV transmission to the

infant". And that, "Although in vitro and in vivo laboratory animal studies suggest the potential for toxicity with preimplantation exposure, the risk for teratogenic events after postimplantational exposures appears to be low at therapeutically effective concentrations of these dideoxynucleosides" (341).

It is unethical, to say the least, to insist on prescribing AZT and other antiretrovirals to prevent AIDS in healthy HIV-positive pregnant women, in infants, and in children. The potential cytotoxic, mutagenic, theratogenic, immunotoxic, carcinogenic properties of these chemicals have been scientifically documented (82,137,329,342).

Before the AIDS epidemic, antimicrobials were only prescribed prophylactically for the prevention of a relapse of rheumatic fever (343). There were no other exceptions. Besides, antimicrobial, especially antibiotics were only prescribed for short periods of time, like a few days for the treatment of an infectious disease. Why are they changing the rules now? Where is the scientific justification that researchers have for changing the rules now?

## Conclusions and recommendations.

**1.** There are no scientific facts to support the following beliefs: that AIDS is an infectious disease caused by a retrovirus named HIV; that AIDS is a physically contagious illness transmitted through body fluids including blood, genital secretions and breast milk; that a positive result in the so-called "AIDS test" is indicative of infection with HIV; that once positive on the "AIDS test" the individual will develop AIDS; that a person who has a positive reaction to the "AIDS test" can prevent the development of AIDS by using several antiretroviral drugs; that the use of antiretroviral drugs can prevent the transmission of HIV from HIV-positive pregnant women to their babies; that AIDS can be treated with antiretroviral drugs; that the use of antiretroviral drugs is safe and free of harmful effects; and that therefore, it is rational to treat and prevent AIDS with antiretroviral medications. These are just unvalidated assumptions.

**2.** On the contrary, there are many scientific facts indicating that: the tests used for the diagnosis of HIV are extraordinarily inaccurate; that being HIV positive does not mean that the person is infected with HIV, the so-called "AIDS virus"; that there are more than 70 different non-HIV related reasons to have a positive result on the "AIDS test"; that the transmission and infectivity of AIDS is not real; that the risk of developing AIDS after being labeled "HIV positive" is unknown; that HIV is not the cause of AIDS; that HIV may not even exist as a virus; that what is called "AIDS" is a toxic and nutritional syndrome; that all antiretroviral drugs are highly toxic to humans; that the antiretroviral medications can by themselves cause AIDS; and that pregnant women, infants, and children are especially vulnerable to the toxic effects of antiretroviral medications.

**3.** The scientific data presented here demonstrate that it is not only irrational but indeed unethical to treat or prevent AIDS with toxic antiretroviral drugs in anybody (344-346). Besides that, it is contrary to common sense to treat or prevent a highly toxicological syndrome with even more toxicity.

**4.** To treat or prevent AIDS with toxic antiretroviral medications is also a violation of the Universal Declaration of Human Rights.

Article 5 of the Universal Declaration of Human Rights states: “No one shall be subjected to torture or to cruel, inhuman or degrading treatment or punishment” (347). Therefore, no one has the right to “subject persons with HIV or AIDS to inhuman and degrading treatment even if purportedly in the community’s interests” (347).

**5.** The use of antiretroviral medications to treat or prevent AIDS should therefore be stopped immediately.

At the very least, there are serious legal implications with respect to the damage caused by these irrational treatments, as well as possibilities for legal suits and monetary compensation (72,348-350).

**6.** It is likewise urgent that the entire conception of AIDS as an infectious and transmissible viral disease caused by HIV be reappraised immediately.

**7.** People have the right to know both sides of a story, especially when they have to make decisions regarding their own health care. Not informing people of all the facts - as mentioned in this article - is a serious violation of the person’s right to make informed consent medical decisions (69,71).

“Self-determination and autonomy have been recognized, in fact, as a fundamental moral value in US law and are routinely applied to a medical context. In the 1914 Schloendorff case, Justice Nenjamin Cordozo opined: Every Human Being of adult years and sound mind has a right to determine what shall be done with his own body” (70).

“The requirements for informed consent are as follows: 1) The practitioner must disclose all information, including risks and benefits that a reasonable person would need to know in order to make a decision. 2) The one consenting must be competent and must understand the information provided. 3) The consent must be given voluntarily and without coercion” (70).

> Is it really rational or even ethical to use toxic antiretroviral drugs in the treatment and prevention of AIDS in pregnant women, infants, children or anybody else?

It is the hope of the authors of this paper that the scientific arguments containing in this document can be used to alert people to the other side of the story.

# DEDICATION

With this investigation, we want to honor the memory of Dr. Eduardo A. Verzini, MD, from La Plata, Argentina. Dr. Verzini died in May, 1998 of stress-related heart conditions after facing several years of court trials against him. He was accused of causing the HIV-positivity of some of his patients at “El Centro de Dialysis”, a hospital for the treatment

of kidney patients. Dr. Verzini was the Director of this hospital until it was closed by the Argentinean Health authorities, who argued that patients were infected with the "AIDS virus" there.

There is a huge amount of scientific documentation which shows that patients with renal insufficiency or in chronic dialysis programs - like Dr. Verzini's patients - react positive in the tests for HIV due to their deteriorated health, rather than due to infection with HIV (351-360).

Dr. Verzini's name is now added to the long list of people killed by the horrendous consequences of the belief that AIDS is an infectious and contagious condition.

## ACKNOWLEDGMENTS

We thank Tanya Milosevic, Srdjan Milosevic and Lucia Madrid for their generous help in checking and correcting the manuscript of this article.

This investigation was not financially supported by any grant. It was possible thanks to the effort of its authors.

## REFERENCES

1. FAUCI AS. Immunopathogenesis of HIV Infection. J Acq Immunodeficiency Syndromes 1993: 6:655-662.
2. SAAH A. The Epidemiology of HIV and AIDS in Women. In: FADEN RR and KASS NE. HIV, AIDS and Childbearing. Public Policy, Private Lives. New York: Oxford University Press, 1996: 3-14.
3. PANTALEO G, COHEN O, GRAZIOCI C, et al. Immunopathogenesis of Human Immunodeficiency Virus Infection. In: DEVITA VT, CURRAN J, HELLMAN S, et al. AIDS: Etiology, Diagnosis, Treatment and Prevention. 4th Edition. Philadelphia: Lippincott - Raven, 1997: 75-88.
4. STAPRANS SI and FEINBERG MB. Natural History and Immunopathogenesis of HIV-1 Disease. In: SANDE MA and VOLBERDING PA. The Medical Management of AIDS. 5th Edition. Philadelphia: W.B. Saunders Company, 1997: 29-56.
5. CLARK R. Clinical Manifestations and the Natural History of Human Immunodeficiency Virus Infection in Women. In: COTTON D and WATTS DH. The Medical Management of AIDS in Women. New York: John Wiley & Sons, 1997: 115-124.
6. LEVY JA. Overal Features of HIV Pathogenesis: Prognosis for Long-Term Survival. In: HIV and the Pathogenesis of AIDS. Second Edition. Washington DC: ASM Press, 1998: 311-338.
7. GENDELMAN HE and STEVENSON M. Pathogenesis of HIV Infection. In: WORMSER GP. AIDS and Other Manifestations of HIV Infection. 3rd Edition. New York: Lippincott - Raven, 1998: 123-150.
8. WORTH LA and VOLBERGING PA. Clinical Applications of Antiretroviral therapy. In: COHEN PT, SANDE MA and VOLBERDING PA. The AIDS Knowledge Base. Boston: Little, Brown and Company, 1994: 4.5.

9. VOLBERDING PA. Antiretroviral Therapy. In: SANDE MA and VOLBERDING PA. The Medical Management of AIDS. Philadelphia: W.B. Saunders Company, 1997: 113-124.
10. HIRSCH MS, D'AGUILA RT and KAPLAN JC. Antiretroviral Therapy. In: DEVITA VT, CURRAN J, HELLMAN S, et al. AIDS: Etiology, Diagnosis, Treatment and Prevention. 4th Edidion. Philadelphia: Lippincott - Raven, 1997: 495-508.
11. LEVY JA. Antiviral Therapies. In: HIV and the Pathogenesis of AIDS. Second Edition. Washington DC: ASM Press, 1998: 339-364.
12. SCHOOLEY RT. Antiretroviral Chemotherapy. In: WORMSER GP. AIDS and Other Manifestations of HIV Infection. New York: Lippincott - Raven, 1998: 717-732.
13. OSMOND DH. Epidemiology and Transmission. In: COHEN PT, SANDE MA & VOLBERDING PA. The AIDS Knowledge Base. Second Edition. Boston: Little, Brown and Co., 1994: 1.1-1.15.
14. HEYMANN J and BRUBAKER J. Breast Feeding and HIV Infection. In: COTTON D and WATTS DH. The Medical Management of AIDS in Women. New York: John Wiley & Sons, 1997: 55-66.
15. LUZURIAGA K & SULLIVAN JL. Transmission of Human Immunodeficiency Virus From Mother to the Fetus and Infant. In: DEVITA VT, CURRAN J, HELLMAN S, et al. AIDS: Etiology, Diagnosis, Treatment and Prevention. 4th Edition. Philadelphia: Lippincott - Raven, 1997: 167-176.
16. LEVY JA. Features of HIV Transmission. In: HIV and the Pathogenesis of AIDS. Second Edition. Washington DC: ASM Press, 1998: 15-42.
17. HUTTON N. Health Prospects for Children Born to HIV-Positive Women. In: FADEN RR and KASS NE. HIV, AIDS and Childbearing. Public Policy, Private Lives. New York: Oxford University Press, 1996: 63-77.
18. SPERLING R. Perinatal Transmission of HIV. In: COTTON D and WATTS DH. The Medical Management of AIDS in Women. New York: John Willey & Sons, 1997: 45-54.
19. FEINBERG MA and VOLBERDING PA. Testing for Human Immunodeficiency Virus. In: COHEN PT, SANDE MA and VOLBERDING PA. The Aids Knowledge Base. Boston: Little, Brown and Company, 1994: Section 2.
20. PINS MR, TERUYA J and STOWELL CP. Human Immunodeficiency Virus Testing and Case Detection: Pragmatic and Technical Issues. In: COTTON D and WATTS DH. The Medical Management of AIDS in Women. New York: John Wiley & Sons, 1997: 163-176.
21. METCALF JA, DAVEY RT and LANE HC. Acquired Immunodeficiency Syndrome: Serologic and Virologic Tests. In: DEVITA VT, CURRAN J, HELLMAN S, et al. AIDS: Etiology, Diagnosis, Treatment and Prevention. 4th Edition. Philadelphia: Lippincott - Raven, 1997: 177-196.
22. WEISS SH. Laboratory Detection of Human Retroviral Infection. In: WORMSER GP. AIDS and Other Manifestations of HIV Infection. New York: Lippincott- Raven, 1998: 175-200.
23. VOLBERDING PA and COHEN PT. Natural History, Clinical Spectrum, and General Management of HIV Disease. In: COHEN PT, SANDE MA and

VOLBERDING PA. The AIDS Knowledge Base. Boston: Little, Brown and Company, 1994: Section 4.

24. ELDRED L and CHAISSON R. The Clinical Course of HIV Infection in Women. In: FADEN RR and KASS NE. HIV, AIDS and Childbearing. Public Policy, Private Lives. New York: Oxford University Press, 1996: 15-30.
25. CONNOLLY L and JENKINS M. Strategies for Antiviral Therapy Based on the Retroviral Life Cycle. In: COHEN PT, SANDE MA and VOLBERDING PA. The AIDS Knowledge BASE. Boston: Little Brown and Company, 1994: 3.5.
26. FADEN RR, KASS NE, ACUFF KL, et al. HIV Infection and Childbearing: A Proposal for Public Policy and Clinical Practice. In: FADEN RR and KASS NE. HIV, AIDS and Childbearing. Public Policy, Private Lives. New York: Oxford University Press, 1996: 447-462.
27. HITTI J and WATTS DH. Antiviral Therapy During Pregnancy. In: COTTON D and WATTS DH. The Medical Management of AIDS in Women. New York: John Wiley & Sons, 1997: 213-220.
28. STINE GJ. Preventing the Transmission of HIV. In: Acquired Immunodeficiency Syndrome. Biological, Medical, Social, and Legal Issues. Englewood Cliffs, NJ: Prentice Hall, 1996: 224-267.
29. WATTS DH. Care of HIV-Positive Pregnant Women. In: COTTON D and WATTS DH. New York: John Wiley & Sons, 1997: 193-204.
30. LEE BL. Drug Interactions and Toxicities in Patients with AIDS. In: SANDE MA and VOLBERDING PA. The Medical Management of AIDS. Philadelphia: W.B. Saunders Company, 1997: 125-142.
31. LUZURIAGA K, BRYSON Y, KROGSTAND P. et al. Combination Treatment with Zidovidine, Diadenosine, and Nevirapine in Infants with Human Immunodeficiency Virus Type 1 Infection. NEJM 1997; 336:1343-1349.
32. MANN JM. Acquired Immunodeficiency Syndrome and Human Rights. In: DEVITA VT, CURRAN J, HELLMAN S, et al. AIDS: Etiology, Diagnosis, Treatment and Prevention. 4th Edition. Philadelphia: Lippincott - Raven, 1997: 627-632.
33. GRADY C. Human Immunodeficiency Disease: Ethical Considerations for Clinicians. In: DEVITA VT, CURRAN J, HELLMAN S, et al. AIDS: Etiology, Diagnosis, Treatment and Prevention. 4th Edition. Philadelphia: Lippincott - Raven, 1997: 633-644.
34. BAYER R. AIDS and the Ethics of Prevention, Research, and Care. In: WORMSER GP. AIDS and Other Manifestations of HIV Infection. 3rd Edition. New York: Lippincott - Raven, 1998: 799-808.
35. CDC. Centers for Disease Control and Prevention. 1993 Revised Classification System for HIV Infection & Expanded Surveillance Case Definition for AIDS Among Adolescents & Adults. MMWR 1992; 41: 1-19.
36. INSTITUTE OF MEDICINE & NATIONAL ACADEMY OF SCIENCES. Confronting AIDS. Washington DC: National Academy Press, 1986.
37. WORTLEY PM, CHU SY and BERKELMAN RL. Epidemiology of HIV/AIDS in Women and Impact of the Expanded 1993 CDC Surveillance Definition of AIDS. In: COTTON D and WATTS DH. The Medical Management of AIDS in Women. New York: John Wiley & Sons, 1997: 3-14.

38. PAPADOPULOS-ELEOPULOS E, TURNER V & PAPADIMITRIOU JM. Is a Positive Western Blot Proof of HIV Infection ? Bio/Technology 1993; 11:696-707.
39. PAPADOPULOS-ELEOPULOS E, TURNER V, PAPADIMITRIOU J & CAUSER D. HIV Antibodies: Further Questions and a Plea for Clarification. Curr Med Res Opin 1997; 13:627-634.
40. PAPADOPULOS-ELEOPULOS E, TURNER V, PAPADIMITRIOU J, et al. Why No Whole Virus? Continuum (London) 1997; 4(5):27-30.
41. JOHNSON C. Playing Russian Roulete in the Lab: Can you Really Trust the AIDS Test? New York: The HEAL Bulletin, Special Edition, 1993.
42. JOHNSON C. Is Anyone Really Positive? Continuum (London); April/May 1995.
43. JOHNSON C. Factors Known to Cause False-Positive HIV Antibody Test Results; Zenger's San Diego, California, September 1996: 8-9.
44. JOHNSON C. Whose Antibodies Are They Anyway? Continuum (London), September/October 1996; 4(3):4-5.
45. HODGKINSON N. Science Fails the "AIDS Test". In: AIDS: The Failure of Contemporary Science. How a Virus that Never Was Deceived the World. London: Fourth Estate, 1996: 232-262.
46. TURNER VF. Do HIV Antibody Tests Prove HIV Infection? Continuum (London) 1996; 3:8-11.
47. BAUMGARTNER M and The International Forum for Accessible Science. Information Dosier: United Nations Commission on Human Rights, Geneva, Switzerland. April 1998: 64.
48. CDC. Centers for Disease Control and Prevention. Interpretation and Use of the Western Blot Assay For Serodiagnosis of Human Immunodeficiency Virus Type 1 Infections. MMWR 1989; 38 :S1-S7.
49. ZOLLA-PAZNER S, GORNY MK & HONNEN WJ. Reinterpretation of Human Immunodeficiency Virus Western Blot Patterns. NEJM 1989; 320:1280-1281.
50. BURKE DS. Laboratory Diagnosis of Human Immunodeficiency Virus Infection. Clin Lab Med 1989; 9:369-392.
51. DE COCK KM, SELIK RM, SORO B, et al. AIDS Surveillance in Africa: A Reappraisal of Case Definition. BMJ 1991; 303:1185-1189.
52. MASKILL WJ & GUST ID. HIV-1 Testing in Australia. Australian Prescriber 1992; 15:11-13.
53. VOEVODIN A. HIV Screening in Russia. Lancet 1992; 399:1548.
54. CONTINUUM. HIV Positive ? - It Depends Where You Live. Take a Look at the Criteria that Determine a Positive HIV Test Result. Continuum (London) 1995; 3(4):20.
55. LUNDBERG GD. Serological Diagnosis of Human Immunodeficiency Virus Infection by Western Blot Testing. JAMA 1988; 260:674-679.
56. TURNER VF. Do Antibody Tests Prove HIV Infection?. Interview by Huw Christie editor of Continuum. Continuum (London) Winter 1997/8; 5(2):10-19.
57. PAPADOPULOS-ELEOPULOS E, TURNER VF, PAPADIMITRIOU JM & CAUSER D. The Isolation of HIV: Has it Really Been Achieved? The Case Against. Continuum (London) 1996; 4(3): S1-S24.

58. ABBOTT LABORATORIES. Human Immunodeficiency Virus Type 1. HIVAB HIV-1 EIA. Abbott Laboratories, Diagnostics Division. January, 1997 (66-8805/R5), 5 pages.
59. BUIANOUCKAS FR. HEAL's Alternative AIDS Test. A Practical Alternative to T-Cell and Antibody Tests. HEAL (Health Education AIDS Liaison) Comprehensive Packet 1993.
60. EPITOPE, ORGANON TEKNIKA. Human Immunodeficiency Virus Type 1 (HIV-1). HIV-1 Western Blot Kit. PN201-3039 Revision # 6, page 11.
61. ROCHE. Amplicor HIV-1 Monitor test. Roche Diagnostic Systems, 13-06-83088-001, 06/96.
62. PIATAK N, SAAG MS, YANG LC, et al. High Levels of HIV-1 in Plasma During All Stages of Infection Determined by Competitice PCR. Science 1993; 259:1749-1754.
63. VAN GEMEN B, KIEVITS T, SCHUKKINK R, et al. Quantification of HIV-1 RNA in Plasma Using NASBA During HIV-1 Primary Infection. J Virol Meth 1993; 43:177-188.
64. KWOK S & SPINSKY JJ. PCR Detection of Human Immunodeficiency Virus Type 1 Proviral DNA Sequences. In: PERSING DH, SMITH TF, SMITH FC, et al. (eds.) Diagnostic Molecular Biology: Principles and Applications. Washington DC:ASM Press, 1993.
65. MULDER J, MCKINNEY N, CRISTOPHERSON C, et al. Rapid and Simple PCR Assay for Quantitation of Human Immunodeficiency Virus Type 1 RNA in Plasma: Application to Acute Retroviral Infection. J Clin Microbiol 1994; 32:292-300.
66. DEWAR RL, HIGBARGER HC, SARMIENTO MB, et al. Application of Branched DNA Signal Amplification to Monitor Human Immunodeficiency Virus Type 1 Burden in Human Plasma. J Inf Dis 1994; 170:1172-1179.
67. JOHNSON C. The PCR to Prove HIV Infection. Viral Load and Why They Can't Be Used. Continuum (London) 1996; 4:33-37 and 39.
68. PHILPOTT P & JOHNSON C. Viral Load of Crap. Reappraising AIDS 1996; 4(10):1-4.
69. KENT G, DELANY L, HOPE T and GRANT V. Teaching Analysis. Informed Consent: A Case for Multidisciplinary Teaching. Health Care Analysis 1996; 4(1):65-79.
70. O'MARA P. Life, Liberty, and Informed Consent. Mothering September/October 1998; (90): 6-9.
71. SILVERMAN WA. Informing and Consenting. In: Where's The Evidence ? Controversies in Modern Medicine.Oxford: Oxford University Press, 1998: 78-84.
72. CHRISTIE H. Wake the Law. Damaging, Non-Specific HIV Testing at the Hands of the Medical Industry Must Soon Prompt Large Finantial Compensation for "the Diagnosed" It's Time to Sue! Continuum (London) Spring 1998; 5(3):28-29
73. PAPADOPULOS-ELEOPULOS E. Reappraisal of AIDS - Is the Oxidation Induced by the Risk Factors the Primary Cause? Medical Hypothesis 1988; 25:151-162.
74. GIRALDO RA. AIDS and Stressors IV: The Real Meaning of HIV. In: AIDS and Stressors. Medellín, Colombia: Impresos Begón, 1997: 133-173.
75. SHALLENBERGER F. Selective Compartmental Dominance: An Explanation for a Nonifectious, Multifactorial Etiology for Acquired Immune Deficiency Syndrome

(AIDS), and a Rationale for Ozone Therapy and other Immune Modulating Therapies. Med Hypothesis 1998; 50:67-80.
76. TURNER VF. Reducing Agents and AIDS - Why Are We Waiting? Med J Austr 1990; 153:502.
77. GIRALDO RA. AIDS and Stressors II: A Proposal for the Pathogenesis of AIDS. In: AIDS and Stressors. Medellín: Impresos Begón, 1997: 57-96.
78. SNYDER HW and FLEISSNER E. Specificity of Human Antibodies to Oncovirus Glycoproteins: Recognition of Antigen by Natural Antibodies Directed Against Carbohydrate Structures. Proc Nat Acad Sci USA 1980; 77:1622-1626.
79. BARBACID M, BOLAGNESI D & AARONSON SA. Humans Have Antibodies Capable of Recognizing Oncoviral Glycoproteins: Demonstration that these Antibodies are Formed in Response to Cellular Modification of Glycoproteins Rather than as Consequence of Exposure to Virus. Proc Nat Acad Sci USA 1980; 77:1627-1621.
80. WING MG. The Molecular Basis for a Polyspecific Antibody. Clin Exp Immunol 1995; 99:313-315.
81. DUESBERG PH. AIDS Acquired by Drug Consumption and other Non Contagious Risk Factors. Pharmac Ther 1992; 55:201-277.
82. DUESBERG PH & RASNICK D. The Drug-AIDS Hypothesis. Continuum (London) 1997; 4(5):S1-S24.
83. GIRALDO RA. AIDS and Stressors III: A Proposal for the Natural History of AIDS. In: AIDS and Stressors. Medellín: Impresos Begón, 1997: 97-131.
84. ZINKERNAGEL RM. Immunity to Viruses. In: PAUL WE. Fundamental Immunology. Third Edition. New York: Raven Press, 1993: 1211-1250.
85. MIMS CA, DIMMOCK NJ, NASH A & STEPHEN J. The Immune Response to Infections. In: Mims' Pathogenesis of Infectious Diseases. Chapter 6. London: Academic Press, 1995: 136-167.
86. EVANS AS. Viral Infections of Humans, Epidemiology and Control. New York: Plenum Publishing Corporation, 1989.
87. GIRALDO RA. AIDS and Stressors I: Worldwide Rise of Immunological Stressors. In: AIDS and Stressors. Medellín: Impresos Begón, 1997: 23-56.
88. LAURITSEN J. Poison by Prescription: The AZT Story. New York: Asklepios, 1990.
89. DUESBERG PH. Foreign-Protein-Mediated Immunodeficiency in Hemophiliacs with & without HIV. Genetica 1995; 95: 51-70.
90. PAPADOPULOS-ELEOPULOS E, TURNER V, PAPADIMITRIOU J, et al. Factor VIII, HIV and AIDS in Hemophiliacs: An Analysis of their Relationship. Genetica 1995; 95:25-50.
91. GESHEKTER CL. Rethinking AIDS in Africa. Reappraising AIDS 1995; 3(2):1-4.
92. PAPADOPULOS-ELEOPULOS E, TURNER V, PAPADIMITRIOU J, et al. A Critical Analysis of the HIV-T4-Cell-AIDS Hypothesis. Genetica 1995; 95:5-24.
93. CDC Editorial note. In: FANNIN S, GOTTLIEB MS, ROGOLSKI E, et al. Cluster of Kaposi's Sarcoma and *Pneumocystis carini* Pneumonia Among Homesexual Males Residents of Los Angeles and Orange Counties, California. MMWR 1982; 31:305-307.

94. DUESBERG PH. AIDS Epidemiology: Inconsistencies with Human Immunodeficiency Virus and with Infectious Disease. Proc. Natl Acad Sci USA 1991; 88:1575-1579.
95. CHARIMUUTA RC & ROSALIND J. AIDS, Africa, and Racism. London: Free Association Books, 1989.
96. PAPADOPULOS-ELEOPULOS E & TURNER V. AIDS in Africa? Reappraising AIDS 1995; 3(1)1-2.
97. GESHEKTER CL. AIDS, Under Development, and Racial Stereotypes: Rethinking AIDS in Africa. Reappraising AIDS 1997, 5(7):1-5.
98. MWEBE W. Wganda on Mind. Continuum (London) 1998; 5(3):30-31.
99. FARBER C. Out of Africa. Spin March 1993.
100. PETERMAN TA, STONEBURNER RL, ALLEN JR, rt al. Risk of Human Immunodeficiency Virus Transmission From Heterosexual Adults with Transfussion-Associated Infection. JAMA 1988; 259:55-58.
101. JACQUEZ JA, KOOPMAN JS SIMON CP, et al. Role of the primary Infection in Epidemics of HIV Infection in Gay Chorts. J. Acquired Immune Deficiency Syndromes 1994; 7: 1169-1184.
102. PADIAN NS, SHIBOSKI SC, GLASS SO, et al. Heterosexual Transmission of Human Immunodeficiency Virus (HIV) in Northern California: Results from a Ten-Year Study. Am J Epidemiol 1997; 146:350-357.
103. KOLIADIN VL. Critical Analysis of the Current Views on the Nature of AIDS. Genetica 1995; 95:71-90.
104. GIRALDO RA. Polemica Cientifica Internacional Acerca de la Causa del SIDA. Investigacion y Educacion en Enfermeria (University of Antioquia, Colombia) 1996; 14(2):55-74.
105. ELLNER M & DIFERDINANDO T. Deconstructing AIDS. The Advanced Iatrogenic Disease Syndrome. HEAL (Health Education AIDS Liaison) Comprehensive Information Packet 1993.
106. ELLNER M & DIFERDINANDO T. Unmasking HIV. AIDS, Hope and the Psychospiritual Healing Crisis. HEAL (Health Education AIDS Liaison) Comprehensive Information Packet 1993.
107. DIFERDINANDO T. Dirty Words. The AIDS Fraud in Context. HEAL (Health Education AIDS Liaison) Comprehensive Information Packet 1993.
108. DIFERDINANDO T. Geneva, AIDS and the Real Gold Standard. Continuum (London) 1998; 5(4):37-39.
109. ELLNER M. “Cultural Hypnosis” and Questioning the HI-Virus. HEAL (Health Education AIDS Liaison) Comprehensive Information Packet 1991.
110. ELLNER M. Protective Stupidity. The Role of Hysteria and Mass Hypnosis in the AIDS Belief System. Continuum (London) 1997/8; 5(2):49.
111. EDWARDS N. In Prison, HIV-Paranoia Simmers. Continuum (London) 1998; 5(3):55-56.
112. GIRALDO RA. AIDS Spread: Scientific Proof Missing. Advance for Medical Laboratory Professionals 1994; 6(32):4.
113. DUESBERG PH. Inventing The AIDS Virus. Washington DC: Regnery Publishing Inc. 1996: 722.

114.DUESBERG PH. Infectious AIDS; Have We Been Misled? Berkeley, CA: North Athantic Books, 1996: 582.
115.CONNOR EM, SPERLING RS, GELBER R, et al. Reduction of Maternal-Infant Transmission of Human Immunodeficiency Virus Type 1 With Zidovudine Tretment. NEJM 1994; 331: 1173-1180.
116.ALTMAN LK. AIDS Brings Shift on Breast-Feeding. To Combat AIDS, the U.N. Now Cautions on Breast-Feeding. The New York Times July 26, 1998: A1 and A6.
117.SPECTER M. Breast-Feeding and HIV: Weighing Health Risks and Treatment Costs. The New York Times. August 19, 1998: A14.
118.RIORDAN J. Viruses in Human Milk. Human Immunodeficiency Virus. In: RIORDAN J & AUERBACH K (eds). Breastfeeding and Human Lactation. Second Edition. Sudbury, Massachussetts: Jones and Barlett Publishers, 1999: 221-238.
119.KENNEDY SB IV. Human Immunodeficiency Virus in Breast Milk. Medical Hypothesis 1998; 51:75-77.
120.FARBER C. HIV and Breastfeeding: The Fears. The Misconceptions. The Facts. Mothering 1998 Sept/Oct No.90: 66-71.
121.GIRALDO RA. Milking the Market. Will Mothers Dish Out the W.H.O. Formula? Continuum (London) 1998; 5(4):8-10.
122.PHILPOTT P. HIV Hysteria Leads UN to Launch Anti-Breastfeeding Campain. Reappraising AIDS 1998; 6(9):1-3.
123.KENT G. HIV/AIDS and the Nutrition Rights of Infants. 1998; 38 pages. <http://www2.hawaii.edu/~kent>
124.TAYLOR JM, SCHWARTZ K & DETELS R. The Time from Infection with Human Immunodeficiency Virus (HIV) to the Onset of AIDS. J Inf Dis 1986; 154:694-697.
125.CONLAN MG. What Really Happened in Geneva. Physician Roberto Giraldo and Journalist John Lauritsen at 1998 AIDS Conference in Geneva. Reappraising AIDS 1998; 6(8):1-6.
126.ALTMAN LK. Long Term Survivors May Hold Key Clues to Puzzle of AIDS. The New York Times, Science Times January 24, 1995: C1 & C11.
127.BALTIMORE D. Lessons from People with Nonprogressive HIV Infection. NEJM 1995; 332:259-260.
128.CAO Y, QIN L, ZHANG L, et al. Virologic and Immunologic Characterization of Long-Term Survivors of Human Immunodeficiency Virus Type 1 Infection. NEJM 1995; 332:201-208.
129.PANTALEO G, MENZO S, VACCAREZZA M, et al. Studies in Subjects with Long-Term Nonprogressive Human Immunodeficiency Virus Infection. NEJM 1995; 332: 209-216.
130.KIRCHHOFF F, GREENNOUGH TC, BRETTLER DB, et al. Brief Report:Absence of Intact nef Sequences in a Long-Term Survivor with Nonprgressive HIV-1 Infection. NEJM 1995; 332:228-232.
131.FAUCI AS. CD4+ T-Lymphocytopenia Without HIV Infection. - No Lights, No Camera, Just Facts. NEJM 1993; 328:429-431.
132.SMITH DK, NEAL JJ, HOLMBERG SD, and the CDC Idiopathic CD4 T-Lymphocytopenia Task Force. Unexplained Opportunistic Infections and CD4 T-Lymphocytopenia. NEJM 1993; 328:373-379.

133.HO DD, CAO Y. AHU T, et al. Idiopathic CD4+ T-Lymphocytopenia-Immunodeficiency Without Evidence of HIV Infection. NEJM 1993; 328:380-385.
134.SPIRA TJ, JONES BM, NICHOLSON JKA, et al. Idiopathic CD4+ Lymphocytopenia - An Analysis of Five Patients With Unexplained Opportunistic Infections. NEJM 1993; 328:386-392.
135.DUNCAN RA, VON REYN F, ALLIEGRO GM, et al. Idiopathic CD4+ T-Lymphocytopenia - Four Patients with Opportunistic Infections and no Evidence of HIV Infection. NEJM 1993; 328:393-398.
136.WELLS J. We Have to Question so Called “Facts”. Capital Gay August 20, 1993: 14.
137.GIRALDO RA. Papel de Estresantes Inmunologicos en Inmunodeficiencia. IATREIA (University of Antioquia, School of Medicine, Colombia) 1997; 10:62-76.
138.BRYSON YJ, PANG S, WEI LS, et al. Clearance of HIV Infection in a Perinatally Infected Infant. NEJM 1995; 332:833-838.
139.MCINTOSH K and BURCHETT SK. Clearance of HIV - Lessons from Newborns. NEJM 1995; 332:883-884.
140.LEVY JA. Pathogenesis of Human Immunodeficiency Virus Infection. Microbiological Reviews 1993; 57:183-298.
141.DUESBERG PH. Retroviruses as Carcinogens and Pathogens: Expectations and Reallity. Cancer Research 1987; 47:1199-1220.
142.BUIANOUCKAS FR. HIV an Illusion. Nature 1995; 375:197.
143.HODGKINSON N. AIDS: The failure of Contemporary Scince. London, UK: Fourth Estate, 1996: 420.
144.JASNY B. AIDS: The Unanswered Questions (Science Editorial). Science 1993; 260:1219.
145.DUESBERG PH. With Therapies Like This, Who Needs Disease? In: Inventing the AIDS Virus. Washington DC: Regnery Publishing, 1996: 299-359.
146.DUESBERG PH. How Much Longer Can We Afford the AIDS Virus Monopoly? In: AIDS: Virus - or Drug Induced? Dordrecht: Kluwer Academic Publishers, 1996: 241-270.
147.BUIANOUCKAS FR. Genious for Corruption. The Great AIDS Caper. Copyright February 1992. <http://www.healaids.com>
148.GIRALDO RA. AIDS and Stressors: AIDS in Neither an Infectious Disease nor is Sexually Transmitted. It is a Toxic-Nutritional Syndrome Caused by the Alarming Worldwide Increment of Immunological Stressor Agents. Medellín, Colombia: Impresos Begón, 1997: 205.
149.SHENTON J. AZT: Cause for Concern. In: Positively False. Exposing the Myths Around HIV and AIDS. London: I.B. Tauris, 1998: 110-139.
150.SHENTON J. Positively False: Wrong Tests and long-term Survivors. In: Positively False: Exposing the Myths Around HIV and AIDS. London: I.B. Tauris, 1998: 238-239.
151.MAGGIORE C. What if Everything you Thought you Knew about AIDS was Wrong? Third Printing. Los Angeles: HEAL (Health Education AIDS Liaison), 1997: 55.

152.LAURITSEN J. AZT: Iatrogenic Genocide. In: The AIDS War: Propaganda, Profiteering and Genocide from the Medical-Industrial Complex. New York: Asklepios, 1993: 71-86.
153.LAURITSEN J & YOUNG I. Psychological and Toxicological Causes of AIDS. In: The AIDS Cult: Essays on the Gay Health Crisis. Provincetown: Asklepios, 1997: 75-100.
154.GIRALDO RA. AIDS in Neither an Infectious Disease nor is Sexually Transmitted. In: AIDS and Stressors. Medellín: Impresos Begón, 1997: 175-187.
155.ELLISON BJ, DOWNEY AB & DUESBERG PH. HIV as a Surrogate Marker for Drug Use: a Re-analysis of The San Francisco Men's Health Study. In: Virus- or Drug Induced? Dordrecht, The Netherlands: Kiuwer Academic Publishers, 1996: 97-104.
156.DUESBERG PH. Can Epidemiology Determine Whether Drugs or HIV Cause AIDS? AIDS-Forschung 1993; 12: 627-635.
157.ALFONSO HS. El Porque del Fiasco. In: El Gran Fiasco: El SIDA no es Causado por el VIH. Barranquilla: Prestigio Editorial Colombiana, 1996: 149-163.
158.GOEDERT JJ, NEULAND CY, WALLEN WC, et al. Amyl Nitrite May Alter T Lymphocytes in Homosexual Men. Lancet 1982; i : 412-416.
159.GOEDERT JJ, COHEN AR, KESSLER CM, et al. Risks of Immunodeficiency, AIDS, & Death Related to Purity of Factor VIII Concentrate. Lancet 1994; 344: 791-792.
160.NICOLOSI A, MUSICO M, SARACCO A, et al. Incidence and Risk Factors of HIV Infection: A Prospective Study of Seronegative Drug Users From Milan and North Italy. 1987-1989. Epidemiology 1990; 1:453-459.
161.PHAIR J, JACOBSON L, DETELS R, et al. AIDS Occurring Within 5 Years of Infection with Human Immunodeficiency Virus Type 1: The Multicenter AIDS Cohort Study. J Acq Immunodef Syndromes 1992; 5:490-496.
162.DES JARLAIS DC, FRIEDMAN SR, MARMOR R, et al. CD4 Lymphocytopenia among Injecting Drug Users in New York City. J AIDS 1993; 6:820-822.
163.LAURITSEN J & WILSON H. Death Rush, Poppers and AIDS. New York: Pagan Press, 1986.
164.GOTTLIEB MS, SCHANKER HM, FAN PT, et al. Pneumocystic Pneumonia-Los Angeles. MMWR 1981; 30:250-252.
165.BARRE-SINOUSSI F, CHERMANN JC, REY F et al. Isolation of a T-Lymphotropic Retrovirus from a Patient at Risk for Acquired Immune Deficiency Syndrome (AIDS) Science 1983; 220:868-871.
166.PAPOVIC M, SARNGADHARAN MG, READ E, et al. Detection, Isolation, and Continious Production of Cytopathic Retroviruses (HTLV-III) from Patients with AIDS and Pre-AIDS. Science 1984; 224:497-500.
167.LEVY J, HOFFMAN AD, KRAMER SM, et al. Isolation of Lymphocytopathic Retroviruses from San Francisco Patients with AIDS. Science 1984; 225:840-842.
168.PAPADOPULOS-ELEOPULOS E. Is HIV the Cause of AIDS. Interview by Christine Johnson. Continuum (London) 1997; 5(1):8-19.
169.LANKA S. HIV: Reality or Artefact? Continuum (London) 1995.
170.LANKA S. Collective Fallacy. Rethinking HIV. Continuum (London) 1996; 4(3):19-20.

171.LANKA S. No Viral Identification: No Cloning as Proof of Isolation. Continuum (London) 1997; 4(5):31-33.
172.DE HARVEN E. Pioneer Deplores “HIV” “Maintaining Errors is Evil” Continuum (London) 1997/8; 5(2):24.
173.DE HARVEN E. Remarks on Methods for Retroviral Isolation. Continuum (London) 1998; 5(3):20-21.
174.PHILPOTT P. The Isolation Question. Does HIV Exist? Do HIV Tests Indicate HIV Infection? Here’s Why Some Scientists Say No. How an Australian Biophysicist and her Simple Observations Have Taken Center Stage Among AIDS Reappraisers. Reappraising AIDS 1997; 5(6):1-12.
175.PAPADOPULOS-ELEOPULOS E, TURNER V, PAPADIMITRIOU J, et al. Between the Lines. A Critical Analysis of Luc Montagnier’s Interview Answers to Djamel Tahi. Continuum (London) 1997/8; 5(2):35-45.
176.HODGKINSON N.Origin of the Specious. Continuum (London) 1996c; 4(3):17-18.
177.TURNER VF. Where Have We Gone Wrong? Continuum (London) 1998; 5(3):38-44.
178.KLATZMANN D & MONTAGNIER L. Approaches to AIDS Therapy. Nature 1986; 319:10-11.
179.PAPADOPULOS-ELEOPULOS E, TURNER V & PAPADIMITRIOU J. Oxidative Stress, HIV and AIDS. Res Immunol 1992; 143:145-148.
180.DOOLITTLE RF, FENG DF, JOHNSON MS, et al. Origins and Evolutionary Relationships of Retroviruses. Quart Rev Biol 1989; 64:1-30.
181.LORI D, DI MARZO VERONESE F, DE VICO AL, et al. Viral DNA Carried by Human Immunodeficiency Virus Type 1 Virions. J Virol 1992; 66:5067-5074.
182.ZHANG H, ZHANG Y, SPICER TS, et al. Reverse Transcription Takes Place Within Extracellular HIV-1 Virions: Potential Biological Significance. AIDS Res Hum Retrovirus 1993; 9:1287-1296.
183.GLUSCHANKOF P, MONDOR I, GELDERBLOM HR, et al. Cell Membrane Vesicles are a Major Contaminant of Gradient-Enriched Human Immunodeficiency Virus Type-1 Preparations. Virology 1997; 230:125-133.
184.BESS JW, GORELICK RJ, BOSCHE WJ, et al. Microvesicles are a Source of Contaminating Cellular Proteins Found in Purified HIV-1 Preparations. Virology 1997; 230:134-144.
185.KOVAL TM. Stress-Inducible Processes in Higher Eukaryotic Cells. New York: Plenum Press, 1997: 256.
186.DURACK DT. Opportunistic Infections & Kaposi’s Sarcoma in Homosexual Men. NEJM 1981; 305: 1465-1467.
187.RAPPOPORT J. AIDS Inc.: Scandal of the Century. San Bruno, CA: Human Energy Press, 1988: 275.
188.ADAMS J. AIDS: The HIV Myth. New York: St. Martin’s Press, 1989.
189.OPPENHEIMER GM. Causes, Cases, and Cohorts: The Role of Epidemiology in the Historical Construction of AIDS. In: FEE R & FOX DM. AIDS: The Making of a Chronic Disease. Berkeley, CA: University of California Press, 1992: 49-83.
190.FETTNER AG & CHECK WA. The Truth About AIDS: Evolution of an Epidemic. New York: Holt, Rinheart & Wilson, 1985.

191.NEWELL GR, MANSELL PWA, WILSON MB, et al. Risk Factor Analysis Among Men Referred for Possible Acquired Immunodeficiency Syndrome. Preventive Med 1985; 14:81-91.
192.HAVERKOS HW & DOUGHERTY JA. Helth Hazards of Nitrite Inhalants. Am. J. Med. 1988; 84: 479-482.
193.HAVERKOS HW & DOUGHERTY JA (EDS). Health Hazards of Nitrite Inhalants. Washington DC: US Dept. Health & Human Services, 1988: 83.
194.MCMANUS TJ, STARRETT LA & HARRIS RW. Amyl Nitrite Use by Homosexuals. Lancet 1982; I:503.
195.JAFFE HW, CHOI K, THOMAS PA, et al. National Case-Control Study of Kaposi's sarcoma & *Pneumocystis Carini* Pneumonia in Homosexual Men: Part 1, Epidemiologic Results. Ann. Intern. Med. 1983; 99: 145-151.
196.FRANCIS DP, CURRAN JW & ESSEX M. Epidemic Acquired Immune Deficiency Syndrome: Epidemiologic Evidence for a Transmissible Agent. J Natl Cancer Inst 1983; 71:1-4.
197.CDC. Centers for Disease Control and Prevention. An Evaluation of The Immunotoxic Potential of Isobutyl Nitrite. MMWR 1983; 32: 457-458, 464.
198.BUIANOUCKAS FR. Towards Health: A New Paradigm for AIDS. HEAL (Health Eduacation AIDS Liaison) Comprehensive Packet 1992.
199.DUESBERG PH. HIV, AIDS & Zidovudine. Lancet 1992; 339: 805-806.
200.DUESBERG PH. Alternative AIDS Hypotheses. Preface. Genetica 1995; 95:3.
201.ASCHER MS, SHEPPARD HW, WINKELSTEIN W, et al. Does Drug Use Cause AIDS ? Nature (London) 1993; 362:103-104.
202.SCHECHTER MT, CRAIB KJP, GELMON KA, et al. HIV-1 and the Aetiology of AIDS. Lancet 1993; 341:658-659.
203.COHEN J. The Duesberg Phenomenon. Science 1994; 266:1642-1649.
204.VEUGELERS PL, PAGE KA, TINDALL B, et al. Determinants of HIV Disease Progression Among Homosexual Men Registered in the Tricontinental Seroconverter Study. Am J Epidemiol 1994; 140:747-758.
205.GIBBONS J. Drugs & Us. Gay Times (London) 1996; September: p17-37.
206.HAVERKOS HW, DROTMAN DP & HANSON D. Surveillance for AIDS-Related Kaposi's sarcoma (KS): Update. Rockvile, MD/Atlanta, GA: NIDA/CDC, 1994: May.
207.LAURITSEN J. NIH Reconsiders Nitrites' Link to AIDS. Biotechnology 1994; 12:762-763.
208.SIGNORILE M. The Incredible Bulk: It Looks Like the Whole Gay World is on Steroids, Maybe That's Because It Is. Out, May 1997: P70,72,73 & 124.
209.CDC. Centers for Disease Control and Prevention. U.S. HIV & AIDS Cases Reported Through December 1996. HIV/AIDS Surveillance Report 1996; 8: 1-39.
210.MOSS AR. AIDS and Intravenous Drug Use: The Real Heterosexual Epidemic. Br Med J 1987; 294:389-390.
211.OSTROW DG, BELTRAN ED, JOSEPH JG, et al. Recreational Drugs and Sexual Behavior in the Chicago MACS/CCS Cohort of Homosexual Active Men. J Subst Abuse 1993; 5:311-325.
212.SIGNORILE M. Life Outside: The Signorile Report on Gay Men: Sex, Drugs, Muscles, and the Passages of Life. New York: Harper Collins Publishers, 1997.

213.EUROPEAN COLLABORATIVE STUDY. Children Born to Women with HIV-1 Infection: Natural History & Risk of Transmission. Lancet 1991; 337: 253-260.
214.EUROPEAN COLLABORATIVE STUDY. Natural History of Vertically Acquired Human Immunodeficiency Virus-1 Infection. Pediatrics 1994; 94:815-819.
215.CDC. Centers for Disease Control and Prevention. National HIV Serosurveillance Summary; Update 1993. United States Department of Health and Human Services 1995.
216.WHO, World Health Organization. The Current Global Situation of the HIV/AIDS Pandemic. WHO, Geneva, January 1995.
217.WHO, World Health Organization. WHO Report No. 45: AIDS Surveillance in Europe. AIDS Forschung 1995; 10:603-613.
218.WHITE HOUSE Office of National Drug Control Policy. Fact Sheet: Drug Data Summary. DHSS, The White House, Washington DC, 1998.
219.BLANCHE S, ROUZIOUX C,MOSCATO MLG, et al. A Prospective Study of Infants Born to Women Seropositive for Human Immunodeficiency Virus Type 1. NEJM 1989; 320:1643-1648.
220.SCHAMA KF, HOWELL LL & BYRD LD. Prenatal Exposure to Cocaine. In: HIGGINS ST & KATZ JL. Cocaine Abuse. Behavior, Pharmacology and Clinical Applications. San Diego: Academic Press, 1998: 159-179.
221.MOK JQ, DE ROSSI A, ADES AE, et al. Infants Born to Mothers Seropositive for Human Immunodeficiency Virus. Lancet 1987; I:1164-1168.
222.ASCHER MS, SHEPPARD HW & WINKELSTEIN W. AIDS-Associated Kaposi's Sarcoma (letter). Science 1995; 267: 1080.
223.SELIGMAN M, CHESS L, FAHEY JL. AIDS - An Immunologic Reevaluation. NEJM 1984; 311:1286-1292.
224.SANCTON TA. TIME Planet of the Year: What on Earth are We Doing? TIME 1989; 133(1):26-30.
225.WORLD HEALTH. Reaching Out to the Poorest. World Health, Magazine of the World Health Organization. 1994; 47(6):1-31.
226.LAYON J, IDRIS A, WASZYNSKI M, et al. Altered T-Lymphocyte Subsets in Hospitalized Intravenous Drug Abusers. Arch Intern Med 1984; 144:1376-1380.
227.LERNER WD. Cocaine Abuse and Acquired Immunodeficiency Syndrome: Tale of Two Epidemics. Am J Med 1989; 87:661-663.
228.LARRAT PE & ZIERLER S. Entangled Epidemics: Cocaine Use and HIV Disease. J Psychoactive Drugs 1993; 25:207-221.
229.MIENJES GH, MIEDEMA F, VAN AMEIJDEN EJ, et al. Frequent Injecting Impairs Lymphocyte Reactivity in HIV-Positive and HIV-Negatine Drug Users. AIDS 1991; 5:35-41.
230.NIEMAN RB, FLEMING J, COKER RJ, et al. The Effect of Cigarette Smoking on the Development of AIDS in HIV-1-Seropositive Individuals. AIDS 1993; 7:705-710.
231.RATAJCZAK HV, THOMAS PT, HOUSE RV, et al. Local Versus Systemic Immunotoxicity of Isobutyl Nitrite Following Subchronic Inhalation Exposure of Female B6C3F1 Mice. Fund Appl Toxicol 1995; 27:177-184.
232.HOLSAPPLE MP, KAMINSKI NE and PRUETT SB. T Lymphocyte Subpopulations and Immunotoxicology of Drug of Abuse. In: KIMBER I and

SELGRADE MK. T Lymphocyte Subpopulations in Immunotoxicology. Chichester: John Wiley & Sons, 1998: 73-102.
233.NATIONAL RESEARCH COUNCIL. Diet, Nutrition, and Cancer. Washington DC: National Academic Press, 1982.
234.DES JARLAIS DC, FRIEDMAN SR, MARMOR M, et al. Development of AIDS, HIV Seroconversion, & Pontential Cofactors for T4 Cell Loss in a Cohort of Intravenous Drug Users. AIDS 1987; 1: 105-111.
235.WEBER R, LEDERGERBER W, OPRAVIL M, et al. Progression of HIV Infection in Misusers of Injected Drugs Who Stop Injecting or Follow a Programme of Maintenance Treatment with Methadone. Br Med J 1990; 301:1362-1365.
236.KLIMAS NG, BLANEY NT, MORGAN RO, et al. Immune Function & Anti-HTLV-I/II Status in Anti-HIV-1-Negative Intravenous Drug Users Receiving Methadone. Amer J Med 1991; 90: 163-170.
237.HUGHES MD, STEIN DS, GUNDACKER HM, et al. Within-Subject Variation in CD4 Lymphocyte Count in Asymptomatic Human Immunodeficiency Virus Infection: Implications for Patient Monitoring. J Infect Dis 1994; 169: 28-36.
238.BLANCHE S, MAYAUX MJ, ROUZIOUX C, et al. Relation of the Course of HIV Infection in Childern to The Severity of The Disease in Their Mothers at Delivery. NEJM 1994; 330:308-312.
239.KRIEGER JA, HEO Y & LAWRENCE DA. Oxidative Stress and Heavy Metal Modification of T Lymphocyte Subsets. In: KIMBER I & SELGRADE MK. T Lymphocyte Subpopulations in Immunotoxicology. Chichester: John Wiley & Sons, 1998: 103-120.
240.RADI R, CASTRO L, RODRIGUEZ M, et al. Free Radical Damage to Mitochondria. In: BEAL MF, HOWELL N & BODIS-WOLLNER I. Mitochondria and Free Radicals in Neurodegenerative Diseases. New York: John Wiley & Sons, 1997: 57-90.
241.HASSIG A, KREMER H, LANKA S, LIANG W-X & STAMPFLI K. 15 Years of AIDS. The Continuous Failure in the Prevention and Treatment of AIDS is Rooted in the Misinterpretation of an Inflammatory Autoimmune Process as a Lethal, Viral Venereal Disease. Continuum (London) 1998; 5(3): 32-37.
242.MARMOR M, FRIEDMAN-KIEN AE, LAUBENSTEIN L, et al. Risk Factors for Kaposi's Sarcoma in Homosexual Men. Lancet 1982; i:1083-1087.
243.NEWELL GR, MANSELl PWA, SPITZ MR, et al. Volatil Nitrites: Use and Adverse Effects Related to the Current Epidemic of the Acquired Immunodeficiency Syndrome. Am J Med 1985; 78:811-816.
244.DUESBERG PH and YIAMOUYIANNIS J. HIV Does Not Cause AIDS, But… In: AIDS: The Good News is HIV Doesn't Cause it. The Bad News is "Recreational Drugs" and Medical Treatments like AZT Do. Delaware, Ohio: Health Action Press, 1995: 46-47.
245.WHO, World Health Organization. WHO Report No. 48. Weekly Epidemiological Record (WER) 1996; 48:361-368.
246.CLINTON W & THE WHITE HOUSE. The National Drug Control Strategy: 1996. Washington DC: The White House, 1996.
247.ASSOCIATED PRESS. $53.7 Billion Spent on Illegal Drugs in '95. San Francisco Chronicle 1997; November 10: p A6.

248.NEW YORK TIMES. Most $ 1 Bills Teinted by Cocaine, Lab Finds. The New York Times September 23, 1997.
249.FIELDS BN. AIDS: Time to Turn to Basic Science. Nature 1994; 369:95-96.
250.HAND T. Why Antiviral Drugs Cannot Resolve AIDS. Reappraising AIDS 1996; 4(9):1-4.
251.HORWITZ JP, CHUA J & NOEL M. Nucleosides. V. The Monomesylates of 1-(2'-Deoxy-Beta-D-Lyxofuranosyl)Thymidine. J Org Chem 1964; 29:2076.
252.KOLATA G. Imminent Marketing of AZT Raises Problems; Marrow Suppression Hampers AZT Victims. Science 1987; 235: 1462-1463.
253.GLAXO WELLCOME. Retrovir (Zidovudine). In: Physician's Desck Reference. Montvale, NJ: Medical Economic Co., 1998: 1167-1175.
254.GILL PS, RARICK M, BYRNES RK, et al. Azidothymidine Associated with Bone Marrow Failure in the Acquired Immunodeficiency Syndrome (AIDS). Ann Intern Med 1987; 107:502-505.
255.SMOTHERS K. Acquired Immunodeficiency Syndrome: Pharmacology and Toxicology. In: GOLDFRANK LR, FLOMENBAUM NE, LEWIN NA, et al. Goldfrank's Toxicologic Emergencies. Fith Edition. Norwalk, Connecticut: Appleton & Lange, 1994: 455-472.
256.CHIU DT and DUESBERG PH. Toxicity of Azidothymidine (AZT) on Human and Animal Cells in Culture at Concentrations Used for Antiviral Therapy. Genetica 1995; 95:103-109.
257.ZARETSKY MD. AZT Toxicity and AIDS Prophylaxis: Is AZT Benefitial for HIV + Asymptomatic Persons with 500 or More T4 Cell per Cubic Milimeter ? Genetica 1995; 95:91-101.
258.ROSENTHAL GJ and KOWOLENKO M. Immunotoxicologic Manifestations of AIDS Therapeutics. In DEAN JH, LUSTER MI, MUNSON AE and KIMBER I. Immunotoxicology and Immunopharmacology. Second Edition. New York: Raven Press, 1994: 249-265.
259.YARCHOAN R, MITSUYA H, MYERS CE, et al. Clinical Pharmacology of 3'-azido-1', 3'-dideoxythymidine (Zidovudine) and Related Dideoxynucleosides. NEJM 1989; 321:726-739.
260.KHOO SH & WILKINS EGL. Review: Controversies in Anti-Retroviral Therapy of Adults. J Antimicrob Chemother 1995; 35:245-262.
261.PLUDA JM, YARCHOAN R, JAFFE ES, et al. Development of Non-Hodgkin Lymphoma in a Cohort of Patients with Severe Human Immunodeficiency Virus (HIV) Infection on Long-Term Antiretroviral Therapy. Ann Intern Med 1990; 113:276-282.
262.COHEN SS. Antiretroviral Therapy for AIDS. NEJM 1987; 317:629.
263.YARCHOAN R & BRODER S. Antiretroviral Therapy for AIDS. NEJM 1987; 317:630.
264.COHEN J. The Media's Love Affair with AIDS Research: Hope vs. Hype. Science 1997; 275:298-299.
265.NUSSBAUM B. Good Intentions: How Big Business, Politics, and Medicine are Corrupting the Fight Against AIDS. New York: Atlantic Montly Press, 1990: 352.

266.RICHMAN DD, FISCHL MA, GRIECO MH, et al., and The AZT Collaborative Working Group. The Toxicity of Azidothymidine (AZT) in the Treatment of Patients with AIDS and AIDS-Related Complex. NEJM 1987; 317:192-197.
267.MCLEOD GX & HAMMER SM. Zidovudine: Five Years Later. Ann Intern Med 1992; 117:487-501.
268.FREIMEN JP, HELFERT KE, HAMRELL MR, et al. Hepatomegaly with Severe Steatosis in HIV-Seropositive Patients. AIDS 1993; 7: 379-385.
269.BACELLAR H, MUNOZ A, MILLER EN, et al. Temporal Trends in the Incidence of HIV-1-related Neurologic Diseases: Multicenter AIDS Cohort Study, 1985-1992. Neurology 1994; 44: 1892-1900.
270.PARKER WB & CHENG YC. Mitochondrial Toxicity of antiviral Nucleoside Analogs. The J of NIH Research 1994; 6:57-61.
271.KUMAR RM, HUGHES PF & KHURRANNA A. Zidovudine Use in Pregnancy: A Report of 104 Cases and the Occurrence of Birth Defects. J Acquire Immunodeficiency Syndromes 1994; 7:1034-1039.
272.BOX JL, FEARON ER, HAMILTON SR, et al.Prevalance of Ras Gene Mutations in Human Colorectal Cancers. Nature (London) 1987; 327: 293-297.
273.MOYE J, RICH KC, KALISH LA, et al. Natural History of Somatic Growth in Infants Born to Women Infected by Human Immunodeficiency Virus. J Pediatrics 1996; 128:58-67.
274.BESSEN LJ, GREENE JB, LOUIE E, et al. Severe Polymyositis-like Syndrome Associated with Zidovudine Therapy of AIDS and ARC. NEJM 1988; 318: 708.
275.GORARD DA & GUILODD RJ. Necrotizing Myopathy and Zidovudine. Lancet 1988; i:1050.
276.HELBERT M, FLETCHER T, PEDDLE B, et al. Zidovudine-Associated Myopathy. Lancet 1988; ii:689-690.
277.DALAKAS MC, ILLA I, PEZESHKPOUR GH, et al. Mitochondrial Myopathy Caused by Long-Term Zidovudine Therapy. NEJM 1990; 322:1098-1105.
278.TILL M & MACDONNELL KB. Myopathy with Human Immunodeficiency Virus Type 1 (HIV-1) Infection: HIV-1 or Zidovudine? Ann Intern Med 1990; 113:492-494.
279.HITCHCOCK MJM. Review: Antiviral Portrait Series, Number 1; 2',3'-didehydro-2',3'-dideoxythymidine (D4T), an anti -HIV Agent. Anitiviral Chem Chemother 1991; 2:125-132.
280.YARCHOAN R, PLUDA JM, PERNO CF, et al. Anti-Retroviral Therapy of Human Immunodeficiency Virus Infection: Current Strategies and Challenges for the Future. Blood 1991; 78:859-884.
281.SMOTHERS K. Pharmacology and Toxicology of AIDS Therapies. The AIDS Reader 1991; 1:29-35.
282.LAURITSEN J. FDA Documents Show Fraud in AZT Trials. New York Native, March 30, 1992.
283.JACOBSON MA, DE MIRANDA P, GORDON SM, et al. Prolongued Pancytopenia Due to Combined Ganciclovir and Zidovudine Therapy. J Inf Dis 1988; 158:489-490.
284.FOGELMAN I, LIM L, BASSETT R, et al. Prevalence and Patterns of Use of Concomitant Medications Among Participants in Three Multicenter Human

Immunodeficiency Virus Type 1 Clinical Trials. J of Acquired Immunodeficienmcy Syndr 1994; 7:1057-1063.
285.MAUGH II TH. Researchers on Path to First Inexpensive AIDS Medication. San Francisco Chronicle, February 6, 1998: A6.
286.STOLBERG SG. Despite New AIDS Drugs, Many Still Lose the Battle. The New York Times, August 22 1997: A1, A6.
287.LIPSKY JJ. Abnormal Fat Accumulation in Patients with HIV-1 Infection. Lancet 1998; 351:847-848.
288.MILLER KD, JAMES E, YANOVSKI J, et al. Visceral Abdominal-Fat Accumulation Associated with Use of Indinavir. Lancet 1998; 351:871-875.
289.LO JC, MULLIGAN K, TAI VW, et al. “Buffalo Hump” in Men with HIV-1 Infection. Lancet 1998; 351:867-870.
290.BRAEU N, LEAF HL, WIECZORCK RL, et al. Severe Hepatitis in Three AIDS Patients Treated with Indinavir. Lancet 1997; 349: 924-925.
291.GROSSMAN SJ, REINFOED N, EYDELLOTH RS, et al. Hepatotoxicity of an HIV Protease Inhibitor in Dogs & Rats. Toxicol. Appl. Pharmacol. 1997;146: 40-52.
292.HENGEL RC, WARTTS NB & LENNOX JL. Benign Symmetric Lipomatosis Associatted with Protease Inhibitors. Lancet 1997; 350:1596.
293.HERRY I, BERNARD L, DE TRUCHIS P, et al. Hypertrophy of the Breasts in a Patient Treated With Indinavir. Clin Inf Dis 1997; 25:937-938.
294.JAMES JS. Protease Inhibitors Metabolic Side Effects: Cholesterol, Triglycerides, Blood Sugar, and “Crixbelly”. In: AIDS Treatment News 1997, Issue No. 273, August 15, 1997.
295.DI PERVI G, DEL BRAVO P & CONCIA E. HIV - Protease Inhibitors. NEJM 1998; 339:773-774.
296.RACE EM, ADELSON-MITTY J, KRIEGEL GR, et al. Focal Mycobacterial Lymphadenitis Following Initiation of Protease-inhibitor Therapy in Patients with Advanced HIV-1Disease. Lancet 1998; 351:252-255.
297.ISMACH J. AIDS’98 Protease Impediments: Lipodystrophy and Drug Resistance Take the Edge Off Cocktail Euphoria. News From the Geneva 12th World AIDS Conference. Physician Weekly 1998; 15(33):1.
298.STAHLMANN R & KLUG S. Antiviral Agents: Nucleoside and Non-Nucleoside Analogues. In KAVLOCK RJ & DASTON GP. Drug Toxicity in Embryonic Development. Advances in Understanding Mechanisms of Birth Defects: Mechanisting Understanding of Human Developmental Toxicants. Berlin: Springer, 1997: 231-264.
299.PAUL WE. The Immune System: An Introduction. In: Fundamental Immunology. Third Edition. New York: Raven Press, 1993: 1-20.
300.MALE D, COOK A, WOEN M, et al. The Immune System. In: Advanced Immunology. London: Mosby, 1996: 1.1.
301.FARBER C. AZT on Trial. The Treatment for AIDS Accused of Being Deadlier than the Disease itself. Spin July 1996.
302.SCHMITZ SH, SCHEDING S, VOLIOTIS D, et al. Side Effects of AZT Prophylaxis After Occupational Exposure to HIV-Infected Blood. Ann Hematol 1994; 69:135-138.

303.SELIGMAN M, WARRELL DA, ABOULKER JP, et al. Concorde: MRC/ANRS Randomized Double-Blind Controlled Trial of Immediate and Deferred Zidovudine in Symptom-free HIV Infection. Lancet 1994; 343:872-881.
304.POZNANSKY MC, COKER R, SKINNER C, et al. HIV Positive Patients First Presenting with an AIDS-Defining Illness: Characteristics and Survival. Br. Med J 1995; 311:156-158.
305.SAAH AJ, HOOVER DR, PENG Y, et al., and the Multicenter AIDS Cohort Study. Predictors For Failure of *Pneumocystis carinii* Pneumonia Prophylaxis. JAMA 1995; 273:1197-1202.
306.SABIN CA, PHILLIPS AN & LEE CA. Response: Arguments Contradict the "Foreign Protein-Zidovudine" Hypothesis. Br Med J 1996; 312:211-212.
307.FISCHL MA, RICHMAN DD, GRIECO MH, et al. The Efficacy of Azidothymidine (AZT) in The Treatment of Patients with AIDS & AIDS-Related Complex. NEJM 1987; 317: 185-191.
308.ALCABES P, SCHOENBAUM EE & KLEIN RS. Correlates of the Rate of Decline of CD4+ Lymphocytes Among Infection Drug Users Infected with the Human Immunodeficiency Virus. Amm J Epidemiol 1993; 137:989-1000.
309.DARBY SC, EWART DW, GIANGRANDE LF, et al. Mortality Before & After HIV Infection in Complete UK Population of Haemophiliacs (letter). Nature (London) 1995; 377:79-82.
310.CHORBA TL, HOLMAN RC, STRINE TW, et al. Changes in Longevity & Causes of Death Among Persons with Hemophilia A. Am. J. Hematol 1994; 45: 112-121.
311.HORTON R. Will Duesberg Now Concede Defeat? The Lancet 1995; 346: 656.
312.MADDOX J. More Conviction on HIV and AIDS. Nature 1995; 377:1.
313.SCOLARO M, DURHAM R & PIECZENIK G. Potential Molecular Competitor for HIV. Lancet 1991; 337:731-732.
314.HERESI GP, CACERES E, ATKINSJT, et al. *Pneumocystis carinii* Pneumonia in Infants Who Were Exposed to Human Immunodeficiency Virus But Were not Infected: An Exception to the AIDS Sirveillance Case Definition. Clin Infect Dis 1997; 25:739-740.
315.MERCK and Company. Crixivan (Indinavir Sulfate): Patient Information About Crixivan. SPIN June, 1997: 111-114.
316.MATSUOKA A, OZAKI M, TAKESHITA K, et al. Aneuploid Induction by Benzo[a]Pyrene and Polyploid Induction by 7,12-Dimetylbenz[a]Antracene in Chinese Hamster Cells V79-MZ and V79. Mutagenesis 1997; 12:365-372.
317.LEWIS R. Genetic Imprecision. BioScience 1991; 41:288-293.
318.BENEDICT WF. Early Changes in Number and Structure After Treatment of Fetal Hamster Cultures with Transforming Doses of Polycyclic Hydrocarbons. J Natl Cancer Inst 1972; 49: 585-590.
319.SANDLER L & HECHT F. Genetics Effects of Aneuploidy. Amer J Hum Genet 1973; 25:332-339.
320.GOGU SR, BECKMAN BS & AGRAWAL KC. Anti HIV-Drugs: Comparative Toxicities in Murine Fetal Liver and Bone Marrow Erythroid Progenitor Cells. Life Sci 1989; 45: iii-vii.
321.GREENBLAT RM, HOLLANDER H, MCMASTER JR, et al. Polypharmacy Among Patients Attending an AIDS Clinic: Utilization of Prescribed, unorthodox, &

Investigational Treatments. Journal of Acquried Immune Deficiency Syndromes 1991; 4: 136-143.
322.HAYES T, ALTMAN R, AKILI-OBIKA A, et al. HIV-Releted Deaths from Selected Infectious Disease Among Persons Without AIDS in New Jersey. Journal of Acquired Immune Deficiency Syndromes 1994; 7: 1074-1078.
323.MARTINEZ M. The Existence of Human Immunodeficiency Virus Resistance to Nucleoside-Analog Drugs Has not Been Shown. Med Hypothesis 1997; 49:235-240.
324.ROGERS J & KAVLOCK R. Developmental Toxicology. In: KLAASSEN CD, AMDUR MO & DOULL J. Casarett and Doull's Toxicology. The Basic Science of Poisons. Fith Edition. New York: McGraw-Hill, 1996: 301-332.
325.DESESSO JM & HARRIS SB. Principles Underlying Developmental Toxicology. In: FAN AM & CHANG LW. Toxicology and Risk Assessment. Principles, Methods, and Applications. New York: Marcel Dekker, 1996: 37-56.
326.MILLER RK, KELLOG CK & SALTZMAN RA. Reproductive and Perinatal Toxicology. In: HALEY TJ & BERNDT WO. Hadbook of Toxicology. Cambridge: Hemisphere Publishing Corporation, 1987: 195-309.
327.NEEDLEMAN HL & BELLINGER D. Prenatal Exposure to Toxicants: Developmental Consequences. Baltimore: Johns Hopkins University Press, 1994: 321.
328.LANCET. Zidovudine for Mother, Fetus, and Child: Hope or Poison? Lancet 1994; 344:207-209.
329.PHILPOTT P. AZT for Pregnant HIV+ Women and Their Newborns. Reappraising AIDS 1997; 5(2):1-2.
330.COTTON P. Trial Halted After Drug Cuts Maternal HIV Transmission Rate By Two Thirds JAMA 1994; 271: 807.
331.GRUBMAN S, GROSS E, LERNER-WEISS N, et al. Older Childern & Adolescents with Perinatally Acquried Human Immunodeficiency Virus Infection. Pediatrics 1995; 95: 657-663.
332.KACEW S & REASOR MJ. Toxicology and the Newborn. Amsterdam: Elsevier, 1984: 291.
333.SPERLING RS, STRATTON P, O'SULLIVAN MJ, et al. A Survey of Zidovudine Use in Pregnant Women With Human Immunodeficiency Virus Infection. NEJM 1992; 326:857-861.
334.ROGERS M & JAFFE HW. Reducing the Risk of Maternal-Infant Transmission of HIV: A Door is Opened. NEJM 1994; 331:1222-1223.
335.BAYER R. Ethical Challenges Posed by Zidovudine Treatment to Reduce Vertical Transmission of HIV. NEJM 1994; 331: 1223-1225.
336.GOGU SR, BECKMAN BS & AGRAWAL KC. Amelioration of Zidovudine-Induced Fetal Toxicity in Pregnant Mice. Antimicrob Agents Chemother 1992; 36:2370-2374.
337.TOLTZIS P, MARX CM, KLEINMAN N, et al. Ziduvudine-Associated Embryonic Toxicity in Mice. J Infect Dis 1991; 163:1212-1218.
338.TOLTZIS P, MOURTON T & MAGNUSON T. Effect of Zidovudine on Preimplantation Murine Embryos. Antimicrob Agents Chemother 1993; 37:1610-1613.

339.FOERSTER M, MERKER H-J, STAHLMANN R, et al. In vitro Effect of Acyclovir on Lymphopoiesis in Fetal Rat Thymus. Toxicol In Vitro 1992; 6:207-217.
340.TOLTZIS P. MOURTON T & MAGNUSON T. Comparative Embryonic Cytotoxicity of Antiretroviral Nucleosides. J Infect Dis 1994; 169:1100-1102.
341.SANDBERG JA & SLIKKER JR W. Developmental Pharmacology and Toxicology of Anti-HIV Therapeutic Agents: Dideoxynucleosides. FASEB J 1995; 9:1157-1163.
342.FARBER C. AZT Roulette: The Impossible Choices Facing HIV-Positive Women. Mothering 1998 Sept/Oct No. 90: 53-65.
343.GRAY BM. Streptococcal Infections. In: EVANS AS & BRACHMAN PS. Bacterial Infections of Humans. Epidemiology and Control. 3rd Edition. New York: Plenum Medical Book Co., 1998: 673-711.
344.GILLON R. Philosophical Medical Ethics. Chichester: John Wiley, 1985.
345.BEAUCHAMP T and CHILDRESS J. Principles of Biomedical Ethics, 3rd edition. New York: Oxford University Press, 1989.
346.SEEDHOUSE D. Ethics is the Key. In: Ethics: The Heart of Health Care. Second Edition. Chichester: John Wiley & Sons, 1998: 35-46.
347.GOSTIN LO and LAZZARINI Z. International Human Rights Law in the AIDS Pandemic. In: Human Rights and Public Health in the AIDS Pandemic. New York: Oxford University Press, 1997: 1- 42.
348.LOVELL BANKS T. Legal Challenges: State Intervention, Reproduction, and HIV-Infected Women. In: FADEN RR and KASS NE. HIV, AIDS and Childbearing. Public Policy, Private Lives. New York: Oxford University Press, 1996: 143-177.
349.MCGOVERN TM. Legal Issues Affecting Women with HIV. In: COTTON D and WATTS D H. The Medical Management of AIDS in Women. New York: John Wiley & Sons, 1997: 389-412.
350.STINE GL. Legal Aspects of HIV/AIDS: A Review of Legislation and Court Decisions in the United States. In: Acquired Immune Deficiency Syndrome. Biological, Medical, Social, and Legal Issues. Third Edition. Upper Saddle River, New Jersey: Prentice Hall, 1998: 544.
351.FASSBINDER W, KUHNI P, NEUMAYER H, et al. Prevalence of Antibody Against LAV/HTLV-III 9HIV) in Patients with Terminal Renal Insuficiency with Hemodialysis and Following Renal Transplantation. Deutsche Medizinische Wechenschriff 1986; 111:1087.
352.PETERMAN T, LANG G, MIKOS N, et al. Hemodialysis/Renal Failure. JAMA 1986; 255:2324.
353.MONOS DS, SENIOR TS, GADSON M, et al. Delineation of False-Positive HIV Antibody Response in Patients with Renal Failure and History of Multiple Transfusions. Transfusion 1989; 29:119-123.
354.UJHELYI E, FUST G, ILLEI G, et al. Different Types of False Positive Anti-HIV Reactions in Patients on Hemodialysis. Immunol Let 1989; 22:35-40.
355.SCHLEUPNER CJ. Detection of HIV-1 Infection. In: MANDELL GI, DOUGLAS RG & BENNETT JE. Priciples and Practice of Infectious Disease. Third Edition. New York: Churchill Livingstone, 1990: 1092.
356.BYLUND D, ZIEGNER U & HOOPER D. Review of Testing for Human Immunodeficiency Virus. Clin Lab Med 1992; 12:305-333.

357.JINDAL R, SOLOMON M & BORROWS L. False Positive Tests for HIV in a Women with Lupus and Renal Failure. NEJM 1993; 328:1281-1282.
358.GEORGE JR & SCHOCHETMAN G. Detection of HIV Infection Using Serologic Techniques. In: SCHOCHETMAN G & GEORGE JR. AIDS Testing: A Comprehensive Guide to Technical, Medical, Social, Legal, and Management Issues. Second Edition. New York: Springer-Verlag, 1994: 62-102.
359.CORDES R & RYAN M. Pitfalls in HIV Testing. Postgraduate Medicine 1995; 98:177.
360.VINCENT F, VINCENT N, RAYNAL A, et al. False-Positive p24 Antigenaemia Detected in Renal Allograft Recipients. Transplantation Proceeding 1995; 27:1738.
361.GIRALDO RA. Everybody Reacts Positive on the ELISA Test for HIV. Continuum (London), Midwinter 1998/9; 5(5): 8-10.

**********