

## **Critical analysis of the “Nelson Mandela Foundation/HSRC Study of HIV/AIDS: South African National HIV Prevalence, Behavioral Risks and Mass Media. Household Survey 2002”**

By Roberto Giraldo, MD<sup>1</sup>

### **CONTENTS**

1. The possibility of false positive reactions on the “HIV test” was not considered.
2. Prevalence of HIV infection or prevalence of reactivity to an “HIV test” in saliva.
3. Poverty was not properly addressed.
4. Nutritional status was not included.
5. Alternative explanation for 5.6 % reactivity on saliva “HIV test” of children 2-14 years of age.
6. Exaggerated focusing on sexual behavior.
7. Male circumcision and “sexual transmission of HIV.”
8. Mother to child “transmission of HIV” (MTCT).
9. Breastfeeding “transmission of HIV.”
10. Promotion of HIV testing.
11. Promotion of antiretroviral medications.
12. Media and public HIV/AIDS beliefs.
13. Conclusions.
14. Recommendations.
15. References.
16. Appendix A – Tests for HIV are highly inaccurate.
17. Appendix B – The role of nutrition in improving the health of people living with HIV/AIDS.
18. Appendix C – Circumcision and AIDS in Africa.
19. Appendix D – The origin of the “transmission” of AIDS.
20. Appendix E – Breastfeeding and AIDS in Africa.
21. Appendix F – Antiretrovirals can cause AIDS.

The following are several key pints in this poorly designed and biased study:

1. *The possibility of false positive reactions on the “HIV test” was not considered.*

This study leads the reader to conclude that the detection of antibodies against what are supposed to be HIV antigens in saliva by an ELISA test is synonymous with HIV infection and that the only reason for reacting positively on the so-called tests for HIV is infection with HIV. In this regard the report states: “The HIV test used in this study, as with all ELISA HIV tests, detects the presence of HIV antibodies in the oral fluid of the participant. Over 99% of people over the age of two years and

---

<sup>1</sup> Physician, specialist in internal medicine, infectious and tropical diseases. New York. <RobGiraldo@aol.com> <www.robertogiraldo.com>

who are infected with HIV will test positive on the ELISA because almost all PLWHA produce HIV antibodies” (page 32).

The report states: “The sensitivity and specificity of the OraSure device when tested with the Vironostika EIA is 99% and also 99% according to the manufacturers (Gallo et al. 1997)” (page22). However, the report does not explain how the sensitivity and the specificity were determined.

The laboratory used in this study, at the University of Natal Durban, “recently did a comparison of sensitivity and specificity of HIV testing on saliva versus serum specimens (Perumal 1999). Paired saliva and serum specimens from 500 individuals were tested using ELISAs, and positive serum specimens were confirmed using Western Blot. The sensitivity and the specificity of the saliva tests were 100% and 99.3% respectively” (page 43). How could they be certain that the saliva test was 99.3% specific for a virus named HIV? The report did not explain this crucial matter.

In the “reliability of the HIV test results” section, the study explains in detail the different negative and positive internal as well as external controls used every time the test was run at the three laboratories chosen for it. Then the study states: “In conclusion, quality control clearly confirms the validity of HIV results obtained in this study” (page 45). However, it is a regular clinical laboratory practice to run controls with known results each time a test is run to be sure that the test is working. The fact that the test works does not mean that the test is valid or specific to detect infection with HIV.

Researchers and other designers of this study considered false positive tests only after they excluded children under two years of age because “the infant may not be actually infected with HIV him or herself but may, if the mother was HIV infected, still be carrying the mother’s antibodies. The child will therefore test positive on the ELISA test even though he/she is not infected (false positive test)” (page 32).

It is internationally known that “HIV antibody tests” are inaccurate, which is why, in countries such as the USA, the law requires that a person have 4 ELISA tests be positive and one Western blot test be positive before he or she is declared to be “HIV positive.” However, in the present survey only one ELISA test was run on saliva. The insert included with the kit used to conduct one of the more popular ELISA tests for “HIV” warns: “At present there is no recognized standard for establishing the presence or absence of antibodies to HIV-1 and HIV-2 in human blood” (Abbott 1996).

The scientific literature shows that there are more than 70 known human conditions that can produce false positive results on so-called tests for HIV. Most of these conditions are common to the African scenario. Some of them 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 autoantibodies against a variety of cells and tissues, vaccinations, and the administration of gammaglobulins or immunoglobulins; the presence of autoimmune 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 factors. This is only a partial list (Johnson 1996; Giraldo et al 1999). Unfortunately, researchers and institutions responsible for this survey ignored these facts.

The questionnaires designed for this survey did not record data regarding past medical history of the participants. This could have helped to determine if participants had current or past conditions that could cause “tests for HIV” to react positively, without the person being actually infected with HIV.

For a detailed analysis of the problems with “HIV tests” see my article: “Tests for HIV are highly inaccurate”, in APPENDIX A of this document.

## **2. Prevalence of HIV infection or prevalence of reactivity to an “HIV test” in saliva.**

One of the main goals of this study was to “determine HIV prevalence in a population of South Africa using linked anonymous HIV saliva tests” (page 9).

All ELISA and Western blot HIV tests, including the Vironostika test kit used in this study, are merely tests to detect the presence of what are supposed to be HIV antibodies. They do not detect infection with HIV. None of these tests can be used to make a diagnosis of HIV infection or AIDS. Therefore, neither of these tests can determine the prevalence of HIV infection.

The only conclusion to be drawn from this study is that a sampling of South Africans demonstrate antibodies against what is believed to be HIV antigens in saliva.

Therefore, this study cannot be interpreted as a “nationwide community-based survey of the prevalence of HIV in South Africa,” as stated by Dr. Orkin in the Preface.

After describing “HIV prevalence” for all races, ~~the~~ study states: “people living with HIV/AIDS are found in every race group in South Africa” (page 46). Here the study goes even further in leading readers to believe that people who react positively on this ELISA test are infected with HIV or have AIDS. However, it is internationally recognized that none of the so-called HIV tests can diagnose HIV infection or AIDS (see APPENDIX A).

Although this survey cannot be used as a measure of the prevalence of HIV infection in South Africa, it does include some interesting data. Examples include:

The overall prevalence of reacting positively on an ELISA test “for HIV” in saliva was found to be 11.4%, much lower than expected by researchers (20%) or has been estimated by International institutions like UNAIDS.

According to data reported in this study, the main risk factor for reacting positively on the saliva “HIV test” are as follows: being African (12.9%), being female (12.8%), living in urban areas, especially if these are informal (21.3%), being poor, and having had sexually transmitted infections (summary in page 57). It is important to note that a common denominator to all of these conditions is poverty and its consequences.

It will be crucial to investigate in the next survey all potential risk factors (immunological stressors) that cause Africans, women, people living in urban informal areas, persons who are poor, and those who have had sexually transmitted infections to react more positively than others on the ELISA saliva test (page 57).

The report states: “Although Africans have the highest estimated HIV prevalence, whites and coloureds also have high estimated prevalence. The estimated HIV prevalence among whites in South Africa is much higher than that observed in predominantly white societies, for example in the USA, Australia, France and UK, which have HIV prevalence less than 1% (UNAIDS 2002)” (page 59). This means that the very fact of living in South Africa is a risk factor for reacting positively on this ELISA test, even for white people.

“HIV prevalence derived from antenatal data: This study calculated the HIV prevalence among women who reported being pregnant in the 12 months before the study (n=244) and found that 24% (CI: 15.8-34.8%) were HIV positive, a finding similar to the Department of Health’s survey of clinics, which found 24.8%” (page 59). However, 24% ELISA reactivity in pregnant women doubles the national average of 11.4% found for all South Africans and that of 12.8% found for women in this study. The high percentage of reactivity on ELISA tests found in pregnant women by both this study and the Department of Health’s surveys can be explained due to the fact that pregnancy and multiparity are known risk factors for reacting positively on antibody tests “for HIV” (See appendix A).

Unfortunately, several important groups of people were excluded from this survey: infants less than 2 years, homeless people, soldiers, prisoners, and students living in boarding schools (page 31). Additionally, several groups of particular interest for the understanding of the AIDS epidemic were not captured in sufficient numbers in this survey: homosexual and bisexual men, drug abusers, and sex workers (page 31).

### 3. Poverty was not properly addressed.

It is not true that: “This is the first South African study that systematically investigates the socio-cultural, political, economic and structural context within which HIV-related behavior occurs” (page 30), since this study did not properly investigate the economic status of South Africans that participated in it. The report acknowledges this: “While this study cannot claim to have adequately measured poverty, a perceived rating of adequacy of household income was utilized as a measure” (page 62).

Researchers and institutions responsible for this study knew of the importance of poverty: “It is widely acknowledged that poverty plays a pivotal role in increasing vulnerability to HIV infection in sub-Saharan countries including South Africa” (page 4). However, in this study they did not address poverty properly.

Although the matter of poverty was not properly addressed, this study does include interesting data:

The report states: “Wealthy Africans have similar levels of risk as less wealthy Africans. However, in the other race groups, lower socio-economic status appears to be related to higher likelihood of HIV infection, even after multivariate adjustment. Further work is required to create an index of poverty comprising detailed employment categories such as work in informal sector, formal sector, part-time employment, occupation, and sources of income. This might shed more light on the relationship between HIV and poverty” (page 63).

Regarding socio-economic status, the report states: “there is a decrease in HIV prevalence from the poorer to richer homes when all participants are included. This means there is a negative correlation between HIV and socio-economic status. However, this trend disappears when only Africans are considered, as in this group there is no discernable trend” (page 54, Table 23). This means that people living in poverty conditions are more exposed to the factors that make this ELISA test react positively, and that being African, regardless if rich or poor, they are more exposed to the factors that make this ELISA test react positively. We need to recall that Africans, during Apartheid, were subjected to the worst of living conditions and that it will take time to correct these conditions. It is therefore possible to postulate that among the effects being detected in South Africa by the so-called “tests for HIV” are the consequences of Apartheid.

It is interesting that “the two provinces with highest HIV prevalence also have the highest proportion of persons living in urban informal areas. These are Gauteng (19.9%) and Free State (16.9%)” (page 62).

Regarding sexually transmitted infections, the report states: “Table 25 displays HIV prevalence by history of sexually transmitted infections and shows that despite the relatively low reporting levels, there is a strong association between HIV and STDs”,

and “there was a significantly higher prevalence of STIs among those living in informal areas” (page 55).

The report states: “Whether or not the STI epidemic is driving the HIV epidemic or whether it is piggybacking on the same risk factors cannot be established from this survey” (page 60). It is scientifically known that all infectious diseases, including sexually transmitted ones, deteriorate the immune system (Ware & Kline 1996). The finding reported in this section could be interpreted as follows: Poverty brings malnutrition and immunodeficiency which make individuals more prone to infections, including sexually transmitted ones, and this deteriorates and oxidizes the immune system, which can be reflected in reacting positively on the ELISA/saliva test used in this survey (Giraldo 1997a,b; Giraldo et al. 1999).

I completely agree with one of the conclusions of this study: “establishing poverty alleviation projects to address the root causes of HIV/AIDS, STIs and TB” (page 105).

#### **4. Nutritional status was not included.**

One of the largest gaps in this study is that it did not investigate the nutritional status of participants.

It is difficult to understand why the researchers and institutions responsible for this survey ignored the fact that, since the beginning of the AIDS epidemic, there has been a growing number of scientific indices supporting the possibility that AIDS can be effectively prevented, treated, and overcome by guaranteeing an optimal nutritional status to individuals that react positively on “tests for HIV” or who have the clinical manifestations of AIDS (Kiure, Msamanga & Fawzi 2002).

It would have been straightforward and useful to have included queries regarding nutrition on the questionnaires and to have evaluated, inexpensively, aspects of the nutritional status of the participants. There will be no excuse for not doing this on the next survey. I would be happy to assist the designers of the next survey in these matters.

For a detailed description of this issue see my article: “The role of nutrition in improving the health of people living with HIV/AIDS”, presented on November 28, 2002 in Johannesburg at a meeting on “Nutrition and HIV/AIDS” organized by the Health Unit of the Southern African Development Community (SADC) (see appendix B).

#### **5. Alternative explanation for 5.6 % reactivity on the saliva “HIV test” of children 2-14 years of age.**

“The observation that the estimated HIV prevalence among children aged 2-14 years is 5.6% was unexpected.” “It remains unclear as to how these children could

have been infected. An emerging theory that warrants further investigation is that there is unexplained HIV prevalence in children who have had no sexual exposure, or have parents with HIV negative mothers.” “Possible factors to be investigated include sexual abuse and unsterile needles” (page 63).

There is the possibility that these children react positively on the ELISA saliva “HIV test” used in this study because they have been exposed to multiple, variable, and chronic immunological stressor challenges. It is possible that, instead of being infected, they are simply intoxicated or oxidized due to malnutrition, infections, parasites and other consequences of poverty (Giraldo 1997a,b; Giraldo et al 1999). People from this age group deserve special attention on the next survey.

#### **6. Exaggerated focusing on sexual behavior.**

This study is highly influenced by the attitudes and beliefs toward HIV/AIDS of the researchers and institutions that designed the survey. This explains why the main focus was on sexual behavior.

The Conceptual Framework, described on page 10, seems to indicate that the only reason for reacting positively on so-called “tests for HIV” is getting “HIV” through sexual activity: “The conceptual framework that informs this study is the second-generation surveillance system, designed by the World Health Organization, UNAIDS and Family Health International. This framework is based on surveys of Knowledge-Attitudes-Beliefs and Practices in relation to sexual behaviors and HIV infection that have been carried out worldwide during the past 15 years” (page 10).

The report demonstrates that, contrary to what has been suggested by Western HIV/AIDS researchers, institutions, and the media, South Africans are not promiscuous at all: “With regard to polygamy, only 3.4% of those who were married (both male and female) reported that they were in a polygamus relationship” (page 77); “When asked if they practiced anal sex, only 2.2% of the 4280 participants who responded to the question answered affirmatively” (page 77); “the median age of sexual debut was 18 years of age for both sexes” (page 78); “Concerning levels of sexual activity, very low levels were reported among children in the 12-14 year age group, and relatively low levels (25%) amongst 15-17 year old youth” (page 78); “sexual frequency amongst sexually active youth is quite low, with the majority of youth (70%) having sex four or less times a month, and 29% having no sex at all” (page 79).

Furthermore, according to this study, South Africans do use condoms: “The levels of condom use of 57% and 46.1% amongst male and female youth respectively are encouraging” (page 79).

This survey demonstrates once again that the issue of sexual behavior has very little or nothing to do with AIDS. Africans need to stand up and demand of the

international institutions dealing with AIDS and the media that they cease placing blame on the sexual behavior of Africans.

**7. Male circumcision and “sexual transmission of HIV.”**

“Research into the influence of male circumcision on the spread of HIV infection has suggested that the practice protects people from HIV infection” (Page 4).

The hypothesis that circumcision can prevent the spread of AIDS has never been scientifically validated. It is merely an assumption based on racial, religious, and sexual prejudices. For a detailed description of this issue see my article: “Circumcision and AIDS in Africa” (appendix C).

The queries regarding circumcision in the questionnaires of this survey are completely unfocused and only serve to increment the stigma concerning AIDS.

I am convinced that Africans will continue questioning and rejecting ethnic fictions and racial slanders. They are already actively defending their integrity.

**8. Mother to child “transmission of HIV” (MTCT).**

“Children under two years were excluded by the protocol of this study” (page 32).

The hypothesis that mothers can transmit HIV or AIDS to their babies during pregnancy, at birth, or by breastfeeding their infants, has never been scientifically validated. For a detailed description of this issue see my article: “The origin of the ‘transmission’ of AIDS” (appendix D).

Therefore, the questions regarding what is known as MTCT in the questionnaires are completely unfocused, are misleading, and increment the stigma concerning AIDS.

**9. Breastfeeding and “transmission of HIV.”**

The questions regarding breastfeeding and the transmission of AIDS in the questionnaires of this survey are also completely unfocused, are misleading and increment the stigma concerning AIDS.

“Issues related to transmission of HIV via blood, PMTCT, and post-exposure prophylaxis were not closely assessed in this study. However, knowledge of HIV transmission via breastfeeding was poor and this should be emphasized in relation to PMTCT interventions” (page 105). Which means that, even though there is no scientific evidence that HIV or AIDS can be transmitted by breast milk, the researchers and institutions responsible for this survey continue to insist that this be included in future surveys.



For a better understanding of this crucial matter having to do with the survival of future generations of Africans, see my article: “Breastfeeding and AIDS in Africa” (appendix E).

***10. Promotion of HIV testing.***

46.4% of participants 15-24 years of age and 35.9% of those 25-49 responded affirmatively to the question “If you do not know your HIV status would you consider going for an HIV test?” (page 99).

This question is a way of promoting HIV testing and making people believe that these tests can really diagnose HIV infection and AIDS, when in reality they cannot.

Promoting HIV testing without explaining the inaccuracy of the tests will raise fears about AIDS and promote sexual stigmatization of the South African community.

How can the researchers and institutions responsible for this survey state: “Awareness of VCT [voluntary counseling and testing] services is relatively low”; when “one in five South Africans had had an HIV test” (page 104). This is typical of the language used by pharmaceutical companies when they want to test everyone for “HIV” and then advise them to take toxic antiretrovirals, alleging that they are thereby preventing the development of AIDS in those who react positively on “tests for HIV” (appendix A).

***11. Promotion of antiretroviral medications.***

96.5% of participants aged 15 and older responded affirmatively to the question: “Should government provide antiretroviral medications for preventing mother to child transmission of HIV/AIDS” (page 91).

95% of participants aged 15 and older responded affirmatively to the question: “Should government provide antiretroviral medications for those with HIV/AIDS-related illness” (page 91).

These types of questions, by their very nature, suggest certain answers and thus cannot provide objective information. In like manner, there is little objectivity in stating: “The fact that the overwhelming majority of people of all races believe that the government should provide ARVs to prevent transmission of HIV from mother to child and also to treat people living with HIV/AIDS, demonstrates the high level of awareness of South Africans on this issue” (page 92).

The types of queries posed concerning antiretroviral medications in the survey’s questionnaires, apart from lacking objectivity, are misleading to the public in that they lead participants to believe that there exists no other solution for preventing and treating AIDS, other than the toxic ARVs. For a detailed description of this issue see my article: “Antiretrovirals can cause AIDS” (appendix F).

Appendix B describes the role of nutritional therapy in HIV/AIDS, which is only one example of a nontoxic, effective, and inexpensive alternative for the prevention and treatment of AIDS.

### **12. Media and public HIV/AIDS beliefs.**

75.7% of adult (15 years+) participants in this study believe that “HIV causes AIDS”; 53.2% believe that “a baby can become HIV positive through breastfeeding”; 11.1% believe that “HIV can be transmitted by kissing”; 4.2% believe that “AIDS can be caused by witchcraft”; 5.1% believe that “HIV can be transmitted by touch”; and only 1.6% believe that “AIDS can be cured by sex with a virgin” (page 82).

These percentages lead to the interpretation that South Africans hold beliefs about AIDS similar to those in most of the countries on Earth and would seem to indicate that the media in South Africa is concentrating its reporting on the HIV/AIDS hypothesis. However, media professionalism requires objective coverage of all sides of a historical controversy, especially when human health is implicated.

### **13. Conclusions.**

- a) “It is vital to have accurate data and a comprehensive understanding of the [AIDS] epidemic” (page 101). Regrettably, I am afraid that this study does not provide this.
- b) It is true, as Nelson Mandela states in the foreword to this study, that: “We have to manage the disease, or the disease will manage us.” We need to understand all of the many facets of AIDS, excluding no possibility, not even those far from or even antagonistic to the widely accepted knowledge and beliefs concerning AIDS. Otherwise, indeed, “the disease will manage us.”
- c) It makes little sense for Nelson Mandela to be promoting HIV testing and antiretroviral medications, since he may well have been introduced to information regarding the inaccuracy of the tests for HIV and the high toxicity of antiretroviral medications, unless he is pursuing some hidden personal agenda. Upon studying the critical analysis provided in this paper, he may come to understand that the Nelson Mandela Foundation’s study does not provide “the data to tackle the epidemic [of AIDS] more vigorously.”
- d) “60 million people worldwide have lived with HIV/AIDS since the beginning of the epidemic and 20 million of these have died (UNAIDS 2002). HIV/AIDS now affects every country in the world. Despite advances in knowledge about HIV prevention, the disease continues to spread” (page 1). If this is true, and everything indicates that it is, then public health officials, epidemiologists

and researchers need to look at alternative explanations for the causation, prevention, and treatment of AIDS.

***14. Recommendations.***

- a) The study contains so many “leaps of faith” that it should be used as a negative example for the teaching of epidemiology.
- b) Since the “HSRC is committed to repeating this study at regular intervals” (preface), it is important that, before the next study is carried out, all available scientific literature on HIV/AIDS be carefully considered.

Before performing the next survey, the Human Sciences Research Council of South Africa, the Nelson Mandela Foundation, and all other institutions responsible for this study must become familiar in detail with the reasons why the so-called tests for HIV are in no way accurate in diagnosing HIV infection or AIDS; must understand that there are more than 70 well known reasons for reacting positively on these tests, many of which reasons are quite prevalent in South Africa; must appreciate that nutritional and antioxidant deficiencies have crucial roles in the pathogenesis of AIDS and in the reactivity to the so-called tests for HIV; and must realize that there are nontoxic, effective, and inexpensive alternatives for the treatment and prevention of AIDS.

- c) Include on the next survey an evaluation of the nutritional and antioxidant deficiencies of participants.
- d) Include in the next survey a serious evaluation of the income of the participants. According to the data reported in this study, the primary risk factor for reacting positively on the saliva “HIV test” are as follows; being African (12.9%), being female (12.8%), living in urban areas especially if these are informal (21.3%), being poor, and having had sexually transmitted infections (summary in page 57). It is important to notice that a common denominator in all of these conditions is poverty and its consequences.
- e) Include in the next survey an evaluation of all potential risk factors (immunological stressors) that make Africans, women, people living in urban informal areas, people who are poor, or people who had STIs, more likely than others to react positively on the ELISA (Giraldo 1997a,b; Giraldo et al. 1999).
- f) Stop promoting HIV tests for the diagnosis of HIV or AIDS, since they simply cannot do that.

- g) **Stop promoting antiretroviral medications and instead promote nontoxic, effective, and inexpensive alternatives for the treatment and prevention of AIDS.**
- h) **Stop promoting stigmatization about the sexual behavior of Africans. AIDS in Africa cannot be explained by vertical or sexual transmission (Gisselquist et al. 2002).**

## **15. REFERENCES**

- **Abbott Laboratories. Human immunodeficiency virus types 1 and 2: (E. coli, B. megaterium, recombinant antigen) HIVAB HIV-1/HIV2 (rDNA) EIA. *Abbott Laboratories Diagnostics Division*, 68-0158/R12, December 1996.**
- **Gallo D et al. Evaluation of a system using oral mucosal transudate for HIV-1 antibody screening and confirmatory testing. OraSure HIV Clinical Trial Group. *JAMA* 1997; 277: 254-258.**
- **Giraldo RA. AIDS and stressors II: a proposal for the pathogenesis of AIDS. In: *AIDS and stressors*. Medellín: Impresos Begón; 1997a: 57-96.**
- **Giraldo RA. AIDS and stressors III: a proposal for the natural history of AIDS. In: *AIDS and stressors*. Medellín: Impresos Begón; 1997b: 97-131.**
- **Giraldo RA et al. Is it rational to treat or prevent AIDS with toxic antiretroviral drugs in pregnant women, infants, children, and anybody else? The answer is negative. *Continuum* (London) 1999; 5(6): 38-52.**
- **Gisselquist D et al. HIV infection in Sub-Saharan Africa not explained by sexual or vertical transmission. *International J of STD & AIDS* 2002; 13 (10): 657-666.**
- **Johnson C. What you don't know can make you "HIV-positive": Factors known to cause false-positive HIV antibody tests. *Zenger's* magazine, San Diego, California; September 1996: page 8.**
- **Kiure AK, Msamanga GI, Fawzi WW. Nutrition and HIV infection. In: Essex M et al. *AIDS in Africa*. Second edition. New York: Kluwer Academic/Plenum Publishers. 2002: 419-435.**
- **Perumal BG et al. An assessment of saliva and urine as alternate body fluids to blood for the diagnosis of HIV in pregnancy. *South Afr J Epidemiol & Inf* 1999; 14: 73-75.**

## **16. APPENDIX A**

### **Tests for HIV are highly inaccurate**

By Roberto Giraldo, June 2000

For the last 6 years I have been working at a laboratory of clinical immunology and molecular diagnosis in one of the most prestigious university hospitals in New York City. Here I have had the opportunity to personally run and get to know in detail the current tests used for the diagnosis of HIV status, namely, the ELISA, Western blot and Viral Load tests.

## **1. The ELISA, Western blot, and Viral Load tests, used for the diagnosis of "HIV infection" are not at all accurate**

There are many arguments against the accuracy of the tests used to diagnose infection by what is known as HIV. For those who want to research the issue more deeply I strongly recommend beginning with the study of a 1993 article in *Bio/Technology* by Eleni Papadopulos-Eleopoulos and her group of researchers from Perth, Western Australia (12).

Below are some of the facts supporting the proposition that a person who reacts positively on these tests is not necessarily infected with HIV:

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 (1). 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, following the lead of the United States' Institutes of Medicine, the National Academy of Sciences, and most AIDS researchers, now refer to "AIDS" as "HIV Disease" (2-7).

However, AIDS in Africa can be diagnosed without HIV tests or any other laboratory tests. This was decided by American public health officials at a conference in Bangui, in the Central African Republic, in October 1985 (*WHO's Weekly Epidemiological Record* 1986; 61:69-76 and *Science* magazine 21 November 1986). This permits health professionals to diagnose AIDS in Africa based solely upon the symptoms and signs that a patient manifests.

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" (8-11). 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 confirm 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 (12-21).

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 (8-11).

However, these antibody tests are neither standardized nor reproducible. With respect to HIV they are meaningless because they are interpreted differently for different individuals, and are interpreted variously in different laboratories and in different countries (12). Test interpretations vary across the United States, Russia, Canada, Australia, Africa, Europe, and South America (22-27), meaning, for instance, 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 (28). Additionally,

a given sample of blood, when tested in 19 different laboratories, will produce 19 different results on the Western blot test (29).

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 but may in fact represent human cellular proteins (12-14,20,30).

1.5. The only valid method for 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 (31), it is not possible to properly define the sensitivity or the specificity of any of the tests for HIV (12). Currently, the sensitivity and the specificity of the tests for HIV are defined not by comparison to purified HIV itself, but through a comparison of the tests in question with the clinical manifestations of AIDS, or with T4 cell counts (12). The Abbott corporation, a test kit manufacturer, states: "At present there is no recognized standard for establishing the presence or 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" (32). Since there is no gold standard for defining the specificity of the tests used for the diagnosis of HIV infection, all HIV-positive results must be considered false-positives.

1.6. There are numerous scientific publications documenting the more than 70 different conditions that can cause the antibody tests to react positively in the absence of any actual HIV infection (12-14,17,19,30). 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. It is, of course, shocking to find that a diagnosis of HIV infection can be based upon tests that are not specific for HIV. However, the scientific evidence tells us that a person can react positively on the tests for HIV even though he or she is not infected with HIV (12-14,17,21,30,33).

1.8. The pharmaceutical companies that make and commercialize the kits for these tests acknowledge the inaccuracy of them, and 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" (32). 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" (34). 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" (35). The difficulty is that most AIDS researchers, journalists, lay people, and health care workers themselves are unaware of these facts concerning the tests because they do not have access to the test kit inserts. Additionally, there appears to be little or no concern on the part of knowledgeable institute faculty to communicate these facts to physicians, let alone to the general public.

1.9. Since viral load results are given in copies per ml of plasma (35), AIDS researchers, health care professionals, and lay people believe that these numbers represent copies or counts of the virus itself (12,36-41). 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 entire HIV genome, and therefore the test cannot be used to detect the 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 a low control range having between 1,200 and 11,000 copies per ml [Lot # 0047], and a high control having a range between 99,000 and 750,000 copies per ml [Lot # A00246] [Roche, Amplicor HIV-1 Monitor test Lot # B00985, expiration August 2000]. Most importantly, the difficulties inherent in the lack of a gold standard for HIV infection also apply to the evaluation of the accuracy of the PCR or Viral load test (12,41,42). As a consequence, the specificity of the viral load test for HIV has never been defined properly. Therefore, all viral load positive results for HIV are false-positives.

1.11. The fact that those who propose HIV as the cause of AIDS were forced to appeal to what amounts to a genetic trick — the PCR test — is a strong argument *against* HIV as the cause of AIDS. To have to amplify infinitesimal amounts of genetic material in the blood of AIDS patients in order to identify HIV, instead of culturing the entire virus, isolating it, and purifying it, violates one of the central tenets of infectious disease: at the climax or maximum state of severity of any infectious disease is when the patient has the greatest number of microbes in his/her tissues. It is in those moments that it is easiest to isolate and purify the microbes that are actually causing a disease.

1.12. People have the right to make informed choices (43-45). However, the right to informed choice implies a right to solid 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 health care. The legal implications of this situation have been noted (46).

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

2.1. There exists a growing number of scientific publications detailing the fact that the tests for HIV infection are not specific for HIV (12-14,47). There are numerous reasons other than a past or present HIV infection to explain why an individual might react positively on these tests. In other words, even in the absence of HIV, these tests can react positively (12-14,17-19,30).

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, or a history of sexually transmitted diseases; the presence of polyspecific antibodies, hypergammaglobulinemias, the presence of auto-antibodies against a variety of cells and tissues, vaccinations, and the administration of gamma globulins or immunoglobulins; the

presence of auto-immune diseases like erythematous systemic lupus, sclerodermia, dermatomyositis and rheumatoid arthritis; the existence of pregnancy and multiparity; a history of rectal insemination; addiction to recreational drugs; several kidney diseases, renal failure and hemodialysis; a history of organ transplantation; the 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 but a few (12-14,17,18,30).

2.3. It is interesting to note that all of these conditions that cause the "HIV tests" to react positively 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 (12-14,30,48).

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 (12-14,49). All these substances are oxidizing agents that cause oxidative stress (47,50,51).

2.5. Recently I had the opportunity to carry out an experiment in which I was able to demonstrate that all blood reacts positively on the ELISA test when the test is run with "neat", or undiluted, serum. This could indicate that everybody has antibodies against what is supposed to be HIV. The individuals that react positively only with straight or neat serum would have a smaller amount of antibodies than the ones that continue reacting positively even when the serum is diluted 400 times (88). This possibility has been confirmed by Yugoslavian and Italian researchers (90).

2.6. 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 (12,52-54). It is therefore possible to conclude that the tests for HIV react positively in the presence of those antibodies; in other words, that a positive result on an 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 (12-14).

2.7. Finally, it has been proposed that antibodies against HIV are surrogate markers for recreational drug use in the United States and in Europe (55,56).

2.8. On the other hand, even if "the AIDS test" were able to detect antibodies to HIV, it would be illogical 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 (48,58). One can have antibodies against many germs without those germs being active, pathogenically



active or even present at all (58,59). In most instances, antibodies against viruses indicate immunity. This is the very basis of vaccination against viral diseases (48,58,60). 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 does the presence of antibodies indicate 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 knowledge of the merits and demerits of the tests for HIV, people cannot make informed decisions.

### 3. 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 document the fact that HIV has never been isolated (12). For several years Papadopulos-Eleopulos and coworkers have been publishing papers in which they have described in detail the scientific facts supporting the assertion that the so-called AIDS virus, HIV, may not even exist (12-14,20,30,31,47,50,61-64).

3.1. The correct procedures (31), employed for over half a century, to achieve isolation of a retrovirus are: a) 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 or 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 (31).

None of these procedures have been achieved in the case of HIV (12,14,31,47).

3.2. 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 (31).

Even the word "isolation," as used by the most noted researchers (65-67), is incorrect and misleading, since neither Montagnier, Gallo nor Levy isolated HIV particles, particles of any other human retrovirus, or any virus-like particles at all (12-14,30,31,47,61,68-74).

3.3. Since no "retroviral particles" (retroviruses) have ever been isolated from any culture (12-14,31,47,61-63,69-75), the existence of HIV has been established indirectly: by the presence, in the blood cultures from AIDS and "HIV-positive" individuals, of 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, supposedly unique to HIV; and by the presence of RNA or DNA fragments that supposedly belong to HIV (12-14,31,47,61-63,69-75).

However, none of these substances have been proven to belong to HIV (12-14,31,47,61-63,69-75). How can it be proven that the substances found in these 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 (31).

3.4. It is interesting to note that the substances listed in 3.3 are claimed to appear exclusively when one co-cultures supposedly infected blood with abnormal cells from leukemia patients, or with cells from umbilical cord lymphocytes (31). The difficulty is that identical substances can be obtained from these same cultures in the absence of the supposedly HIV-infected blood (31).

3.5. The cultures in which the above substances have been found are cultures that have been heavily stimulated with chemicals such as phytohemagglutinin, IL-2, antiserum to human interferon, and other agents (31). These culture stimulants are oxidizing agents (31,47). The difficulty is that identical types of material can be observed in the stimulated cultures of lymphocytes from healthy persons (31,76).

It is interesting to note that in the presence of antioxidants, no HIV phenomena is observed in culture, nor can HIV substances be found (12,64,76).

3.6. The substances listed in 3.3 are not specific to HIV (31). 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 (77).

Gallo and co-workers have claimed that the cell-free supernatants from "infected" cultures contain HIV-DNA (78,79). They forget that, by definition, retroviruses are infectious particles that 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 within the cell and nowhere else (31).

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 (31).

"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 (31).

3.7. To date no one 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 a retrovirus-like particle, let alone a unique retrovirus, HIV (31).

3.8. The proteins or antigens derived from stimulated cultures form the basis for the ELISA and Western blot HIV antibody tests (31,73). Fragments of RNA from stimulated cultures form the basis of the HIV viral load test (31,73). This is the primary reason why the current tests used for the diagnosis of HIV are, in fact, not specific for HIV (12-14,31,61,62).

3.9. In the January 1997 issue of the journal *Virology*, two independent groups of researchers published experiments claiming HIV isolation. Here, for the very first time in the history of HIV, researchers followed the internationally accepted procedures for the isolation of 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 resembled a retroviral particle (80-81). They were unable to isolate HIV simply because HIV was not there to be isolated.

It has been proposed that all of the substances that are said to indicate the existence of HIV are nothing more than non-viral material, the production of which is induced by the chemical agents to which the AIDS patients and cultures are exposed (31). When found in patients, these substances could be seen as the regular products of the stress response (82), secondary to exposure to chemical, physical, biological, mental, and nutritional stressor agents (48,51,57,83-87).

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

#### **4. The real meaning of being HIV-positive**

4.1. The above considerations allow one to propose that reactivity on the ELISA, Western blot, and PCR tests is caused by multiple, repeated, and chronic exposure to chemical, physical, biological, mental, and nutritional stressor agents. The degree of reactivity would be proportional to the level of exposures to immunological stressor or oxidizing agents (12-14,20,30,31,63,88,89).

Positive results on ELISA and Western blot tests, can also be understood as the consequence of the presence of high levels of polyspecific antibodies, due to a state of chronic polyantigenic stimulation (52-54). The reactivity on the three main tests for HIV — ELISA, Western blot, and PCR or viral load — would be simply the result of the stress response (82,88,89,91-94).

4.2. Being "HIV-positive" — reacting positively 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 (47,50,89). His or her immune system has been over-responding to these immunogenic and immunotoxic challenges (51,57,89). The immune system of these "HIV-positive" individuals would be debilitated — oxidized — after it had been over-stimulated and intoxicated. Therefore, in such individuals, the risk for AIDS would be higher than in those who are "HIV-negative" (12,13,49,51).

4.3. Without doubt, there exists an almost perfect correlation between the reactivity on the so-called "tests for HIV" and AIDS.

Exposure to immunological stressors causes the tests to react positively. In like manner, exposure to immunological stressors or oxidizing agents is the cause of the mild to moderate levels of immune suppression present in all non-symptomatic individuals who react positively on the "tests for HIV." If exposure to immunological stressors is not relieved, and if the individual is not disintoxicated, it is highly probable that the non-symptomatic "HIV-positive" individual will experience greater immune suppression and will develop the clinical manifestations of AIDS.

What is known as HIV has no causative role in AIDS. On the contrary, the HIV phenomenon is itself one of the effects of the stress response to multiple, repeated, and chronic exposures to chemical, physical, biological, mental, and nutritional stressor agents.

### **5. Possible trial to find out the real meaning of the tests for HIV**

Take blood from four groups of people and run the tests highly diluted, undiluted and at a wide spectrum of dilutions in between: a) The first group would be a group of healthy people from many age groups; b) the second group would be a group of people from the conventional AIDS risk groups; c) the third group would be a group of people with clinical conditions unrelated to AIDS; and d) the fourth group would be a group of patients with full manifestations of AIDS.

All groups would be subjected to both ELISA and Western blot tests. Additionally, all blood samples could be subjected to the viral load test for HIV.

The results of such an experiment could determine whether these test measurements bear any relationship to an individual's level of exposure to stressor or oxidizing agents. If so, the tests could be salvaged as a measure of an individual's level of intoxication.

### **REFERENCES**

1. 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.
2. FAUCI AS. Immunopathogenesis of HIV Infection. *J Acq Immunodeficiency Syndromes* 1993; 6:655-662.
3. STAPRANS SI and FEINBERG MB. Natural History and Immunopathogenesis of HIV-1 Disease. In: SANDE MA and VOLBERDING PA. *The Medical Management of AIDS*. 5<sup>th</sup> Edition. Philadelphia: W.B. Saunders Company, 1997: 29-56.
4. LEVY JA. Overall 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.
5. 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.
6. INSTITUTE OF MEDICINE & NATIONAL ACADEMY OF SCIENCES. *Confronting AIDS*. Washington DC: National Academy Press, 1986.

7. 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.
8. 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.
9. 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.
10. 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. 4<sup>th</sup> Edition. Philadelphia: Lippincott - Raven, 1997: 177-196.
11. 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.
12. PAPADOPULOS-ELEOPULOS E, TURNER V & PAPADIMITRIOU JM. Is a Positive Western Blot Proof of HIV Infection ? Bio/Technology 1993; 11:696-707.
13. 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.
14. PAPADOPULOS-ELEOPULOS E, TURNER V, PAPADIMITRIOU J, et al. Why No Whole Virus? Continuum (London) 1997; 4(5):27-30.
15. JOHNSON C. Playing Russian Roulette in the Lab: Can you Really Trust the AIDS Test? New York: The HEAL Bulletin, Special Edition, 1993.
16. JOHNSON C. Is Anyone Really Positive? Continuum (London); April/May 1995.
17. JOHNSON C. Factors Known to Cause False-Positive HIV Antibody Test Results; Zenger's San Diego, California, September 1996: 8-9.
18. JOHNSON C. Whose Antibodies Are They Anyway? Continuum (London), September/October 1996; 4(3):4-5.
19. 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.
20. TURNER VF. Do HIV Antibody Tests Prove HIV Infection? Continuum (London) 1996; 3:8-11.
21. BAUMGARTNER M and The International Forum for Accessible Science. Information Dossier: United Nations Commission on Human Rights, Geneva, Switzerland. April 1998: 64.
22. 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.

23. ZOLLA-PAZNER S, GORNY MK & HONNEN WJ. Reinterpretation of Human Immunodeficiency Virus Western Blot Patterns. *NEJM* 1989; 320:1280-1281.
24. BURKE DS. Laboratory Diagnosis of Human Immunodeficiency Virus Infection. *Clin Lab Med* 1989; 9:369-392.
25. DE COCK KM, SELIK RM, SORO B, et al. AIDS Surveillance in Africa: A Reappraisal of Case Definition. *BMJ* 1991; 303:1185-1189.
26. MASKILL WJ & GUST ID. HIV-1 Testing in Australia. *Australian Prescriber* 1992; 15:11-13.
27. VOEVODIN A. HIV Screening in Russia. *Lancet* 1992; 399:1548.
28. 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.
29. LUNDBERG GD. Serological Diagnosis of Human Immunodeficiency Virus Infection by Western Blot Testing. *JAMA* 1988; 260:674-679.
30. TURNER VF. Do Antibody Tests Prove HIV Infection?. Interview by Huw Christie editor of *Continuum*. *Continuum (London)* Winter 1997/8; 5(2):10-19.
31. 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.
32. ABBOTT LABORATORIES. Human Immunodeficiency Virus Type 1. HIVAB HIV-1 EIA. Abbott Laboratories, Diagnostics Division. January, 1997 (66-8805/R5), 5 pages.
33. BUIANOUCAS FR. HEAL's Alternative AIDS Test. A Practical Alternative to T-Cell and Antibody Tests. HEAL (Health Education AIDS Liaison) Comprehensive Packet 1993.
34. EPITOPE, ORGANON TEKNIKA. Human Immunodeficiency Virus Type 1 (HIV-1). HIV-1 Western Blot Kit. PN201-3039 Revision # 6, page 11.
35. ROCHE. Amplicor HIV-1 Monitor test. Roche Diagnostic Systems, 13-06-83088-001, 06/96.
36. PIATAK N, SAAG MS, YANG LC, et al. High Levels of HIV-1 in Plasma During All Stages of Infection Determined by Competitive PCR. *Science* 1993; 259:1749-1754.
37. 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.
38. 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.
39. 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.
40. 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.

41. 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.
42. PHILPOTT P & JOHNSON C. Viral Load of Crap. *Reappraising AIDS* 1996; 4(10):1-4.
43. 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.
44. O'MARA P. Life, Liberty, and Informed Consent. *Mothering* September/October 1998; (90): 6-9.
45. SILVERMAN WA. Informing and Consenting. In: *Where's The Evidence ? Controversies in Modern Medicine*. Oxford: Oxford University Press, 1998: 78-84.
46. CHRISTIE H. Wake the Law. Damaging, Non-Specific HIV Testing at the Hands of the Medical Industry Must Soon Prompt Large Financial Compensation for "the Diagnosed" It's Time to Sue! *Continuum* (London) 1998; 5(3):28-29
47. PAPADOPULOS-ELEOPULOS E. Reappraisal of AIDS - Is the Oxidation Induced by the Risk Factors the Primary Cause? *Medical Hypothesis* 1988; 25:151-162.
48. GIRALDO RA. AIDS and Stressors IV: The Real Meaning of HIV. In: *AIDS and Stressors*. Medellín, Colombia: Impresos Begón, 1997: 133-173.
49. SHALLENBERGER F. Selective Compartmental Dominance: An Explanation for a Noninfectious, 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.
50. TURNER VF. Reducing Agents and AIDS - Why Are We Waiting? *Med J Austr* 1990; 153:502.
51. GIRALDO RA. AIDS and Stressors II: A Proposal for the Pathogenesis of AIDS. In: *AIDS and Stressors*. Medellín: Impresos Begón, 1997: 57-96.
52. 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.
53. 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.
54. WING MG. The Molecular Basis for a Polyspecific Antibody. *Clin Exp Immunol* 1995; 99:313-315.
55. DUESBERG PH. AIDS Acquired by Drug Consumption and other Non Contagious Risk Factors. *Pharmac Ther* 1992; 55:201-277.
56. DUESBERG PH & RASNICK D. The Drug-AIDS Hypothesis. *Continuum* (London) 1997; 4(5):S1-S24.
57. GIRALDO RA. AIDS and Stressors III: A Proposal for the Natural History of AIDS. In: *AIDS and Stressors*. Medellín: Impresos Begón, 1997: 97-131.
58. ZINKERNAGEL RM. Immunity to Viruses. In: PAUL WE. *Fundamental Immunology*. Third Edition. New York: Raven Press, 1993: 1211-1250.

59. 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.
60. EVANS AS. Viral Infections of Humans, Epidemiology and Control. New York: Plenum Publishing Corporation, 1989.
61. PAPADOPULOS-ELEOPULOS E. Is HIV the Cause of AIDS. Interview by Christine Johnson. Continuum (London) 1997; 5(1):8-19.
62. 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.
63. TURNER VF. Where Have We Gone Wrong? Continuum (London) 1998; 5(3):38-44.
64. PAPADOPULOS-ELEOPULOS E, TURNER V & PAPADIMITRIOU J. Oxidative Stress, HIV and AIDS. Res Immunol 1992; 143:145-148.
65. BARRE-SINOUSSE 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.
66. PAPOVIC M, SARNGADHARAN MG, READ E, et al. Detection, Isolation, and Continuous Production of Cytopathic Retroviruses (HTLV-III) from Patients with AIDS and Pre-AIDS. Science 1984; 224:497-500.
67. LEVY J, HOFFMAN AD, KRAMER SM, et al. Isolation of Lymphocytopathic Retroviruses from San Francisco Patients with AIDS. Science 1984; 225:840-842.
68. BUIANOUCAS FR. HIV an Illusion. Nature 1995; 375:197.
69. LANKA S. HIV: Reality or Artefact? Continuum (London) 1995.
70. LANKA S. Collective Fallacy. Rethinking HIV. Continuum (London) 1996; 4(3):19-20.
71. LANKA S. No Viral Identification: No Cloning as Proof of Isolation. Continuum (London) 1997; 4(5):31-33.
72. DE HARVEN E. Pioneer Deplores "HIV" "Maintaining Errors is Evil" Continuum (London) 1997/8; 5(2):24.
73. DE HARVEN E. Remarks on Methods for Retroviral Isolation. Continuum (London) 1998; 5(3):20-21.
74. 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.
75. HODGKINSON N. Origin of the Specious. Continuum (London) 1996c; 4(3):17-18.
76. KLATZMANN D & MONTAGNIER L. Approaches to AIDS Therapy. Nature 1986; 319:10-11.
77. DOOLITTLE RF, FENG DF, JOHNSON MS, et al. Origins and Evolutionary Relationships of Retroviruses. Quart Rev Biol 1989; 64:1-30.



78. 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.
79. 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.
80. 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.
81. 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.
82. KOVAL TM. *Stress-Inducible Processes in Higher Eukaryotic Cells*. New York: Plenum Press, 1997: 256.
83. GIRALDO RA. AIDS and Stressors I: Worldwide Rise of Immunological Stressors. In: *AIDS and Stressors*. Medellín: Impresos Begón, 1997: 23-56.
84. GIRALDO RA. Polemica Científica Internacional Acerca de la Causa del SIDA. *Investigacion y Educacion en Enfermeria (University of Antioquia, Colombia)* 1996; 14(2):55-74.
85. GIRALDO RA. Papel de Estresantes Inmunologicos en Inmunodeficiencia. *IATREIA (University of Antioquia, School of Medicine, Colombia)* 1997; 10:62-76.
86. 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.
87. GIRALDO RA. AIDS in Neither an Infectious Disease nor is Sexually Transmitted. In: *AIDS and Stressors*. Medellín: Impresos Begón, 1997: 175-187.
88. GIRALDO RA. Everybody Reacts Positive on the ELISA Test for HIV. *Continuum (London)* 1999; 5(5):8-10.
89. GIRALDO RA, ELLNER M, FARBER C, et al. Is it Rational to Treat or Prevent AIDS with Toxic Antiretroviral Drugs in Pregnant Women, Infants, Children, and Anybody Else? The Answer is Negative. *Continuum (London)* 1999; 5(6): 38-52.
90. METLAS R, et al. Human Immunodeficiency Virus V3 Peptide-Reactive Antibodies are Present in Normal HIV-Negative Sera. *AIDS Research and Human Retroviruses* 1999; 15: 671-677.
91. MORIMOTO R, TISSIERES A, GEORGOPOULOS C. *Stress Proteins in Biology and Medicine*. Cold Spring Harbor Laboratory Press 1990: 450.
92. SCHLESINGER MJ, SANTORO MG, GARACI E. *Stress Proteins: Induction and Function*. Berlin: Springer-Verlag 1990: 123.
93. VAN EDEN W, YOUNG DB. *Stress Proteins in Medicine*. New York: Marcel Dekker, Inc. 1996: 578.
94. LATCHMAN DS. *Stress Proteins*. Springer, 1999: 422

**17. APPENDIX B****THE ROLE OF NUTRITION IN IMPROVING THE HEALTH OF PEOPLE  
LIVING WITH HIV/AIDS****Southern African Development Community (SADC) Health Meeting,  
Johannesburg, South Africa, November 28 and 29, 2002****By Roberto Giraldo\*****CONTENTS**

- 1. Nutritional immunology.**
- 2. Nutritional deficiencies and HIV/AIDS.**
- 3. Nutritional deficiencies and the progression of HIV-positive individuals to AIDS.**
- 4. Nutritional deficiencies and the “transmission” of HIV/AIDS.**
- 5. Oxidative stress and HIV/AIDS.**
- 6. Nutritional and antioxidant deficiencies in the pathogenesis of AIDS.**
- 7. Nutritional and antioxidant therapy for the prevention and treatment of AIDS.**
- 8. Conclusions.**
- 9. References.**

**1. NUTRITIONAL IMMUNOLOGY.**

The effects of malnutrition on lymphoid organs were first described during the middle of the 19<sup>th</sup> century (1). Lymphoid tissues are particularly vulnerable to the damaging effects of malnutrition and lymphoid atrophy is a prominent feature in nutritional deprivation (2-5). Cell division is a very singular characteristic of the functioning of immunocompetent cells. All types of immune cells and their products, such as interleukins, interferons, and complement, are known to depend on metabolic pathways that employ various nutrients as critical co-factors for their actions and activities (5,6). Most of the host defense mechanisms are altered in protein caloric malnutrition (PCM), as well as during deficiencies of trace elements and vitamins (2,4,7,8).

Patients with PCM have impaired delayed cutaneous hypersensitivity, poor lymphocyte proliferation response to mitogens, lower synthesis of lymphocyte DNA, reduced numbers of rosetting T lymphocytes, impaired maturation of lymphocytes

---

\* Physician, specialist in internal medicine, infectious and tropical diseases. New York. E-mail: RobGiraldo@aol.com Website: www.robortgiraldo.com

seen through an increased deoxynucleotidyl transferase activity, decreased serum thymic factor, fewer CD4<sup>+</sup> cells, decreased CD4<sup>+</sup>/CD8<sup>+</sup> ratio, impaired production of interferon gamma and interleukin 2, altered complement activity (especially reduction of C3, C5, factor B and total hemolytic activity), poor secondary antibody response to certain antigens, reduced antibody affinity, impaired secretory immunoglobulin A response, decreased antibody affinity, and phagocyte dysfunction (2-7).

Human malnutrition is usually a composite syndrome of multiple nutrient deficiencies. However, isolated micronutrient deficiencies do happen. Vitamin A deficiency results in reduction in the weight of the thymus, decreased lymphocyte proliferation, impaired natural killer cell and macrophage activities, and increased bacterial adherence to epithelial cells (8-11). Vitamin B6 deficiency produces failure of several components of both cell-mediated and humoral immune responses (2,4,7). Vitamin C deficiency impairs phagocytosis and cell-mediated immune reactions (12). Vitamin E deficiency also alters immune responsiveness (2,4,7). Zinc deficiency generates lymphoid atrophy, reduces lymphocyte responses and skin delayed hypersensitivity (2,4,7). Copper and selenium deficiencies impair T and B lymphocyte functions (2,4,7). Dietary deficiencies of selected amino acids such as glutamine and arginine also alter immunity (2,4,7).

Intrauterine malnutrition causes prolonged, even permanent, depression of immunity in offspring (13-14).

Considerable data implicate excess lipid intake in the impairment of immune responses (15). The potential for free radical damage is dependent in large part on the level of potentially oxidizable fatty acids, mainly polyunsaturated fatty acids (PUFAs) in the diet (15). High levels of dietary PUFAs have been shown to be immunodepressive. Dietary fats may undergo free radical-mediated oxidation prior to ingestion, as can occur when foods are fried (15). Animals fed oxidized lipids show marked atrophy of the thymus and T lymphocyte dysfunctions (15).

At the molecular level, the damage to immunocompetent cells by several nutritional deficiencies (PCM, Vitamin A, Vitamin C, Vitamin E, zinc, copper, selenium deficiencies) is caused by increased free radicals through oxidative stress (8-11,15,16).

## **2. NUTRITIONAL DEFICIENCIES AND HIV/AIDS.**

Since the beginning of the AIDS epidemic, researchers have provided scientific evidence that supports the possibility that AIDS can be effectively prevented, treated, and overcome by guaranteeing an optimal nutritional status to the individual or the patient (17,18). However, it seems that propaganda spread by pharmaceutical companies to commercialize antiretroviral medications has prevented these ideas from being widely accepted, despite the toxicity of these medications.

Early in the AIDS era, well recognized researchers in the field of nutrition and immunology, such as Dr. Ranjit Kumar Chandra, noticed that: “There is an uncanny similarity between the immunological findings in nutritional deficiencies and those seen in acquired immunodeficiency syndrome, AIDS” (17).

“There is a similarity between the immune deficiency, multiple infections, and severe weight loss seen in AIDS patients, and the association of protein caloric malnutrition (PCM) with reduced resistance to infection observed in malnourished children, particularly in the Third World.” “It is also possible that nutritional deficiency may play a significant role in the clinical course of the immunodeficient state.” “These similarities between AIDS and PCM suggest that nutrition may contribute to the immunodeficient state. The immunodeficiency in children with PCM can be reversed by nutritional rehabilitation, which suggests that restoration of nutritional state may be a useful adjunct to therapy for AIDS patients” (19).

As described above, the immunological alterations found in PCM are practically identical to those of AIDS: impaired delayed cutaneous hypersensitivity, lymphocyte proliferation response to mitogens, complement activity and secondary response to antigens. There is also a reduced number of rosetting T lymphocytes, increased deoxynucleotidyl transferase activity, decreased serum thymic factor, fewer helper T cells, impaired production of interferon gamma and interleukins 1 and 2, reduced antibody affinity, impaired secretory immunoglobulin A (IgA) antibody response and phagocyte dysfunction. The proportion of helper/inducer T lymphocytes recognized by the presence of CD4 positive antigen on the cell surface is markedly decreased. The ratio CD4/CD8 is significantly decreased. Lymphoid atrophy is a prominent feature of nutritional deprivation. Serum antibody responses are generally intact in PCM. Most complement components are decreased, especially C3, C5, factor B and total hemolytic activity (20-26).

“Nutritional problems have been a part of the clinical aspects of AIDS from its earliest recognition as a new disease” (20,24). “In fact, in many AIDS patients, death seems to be determined more by the individual’s nutritional status than by any particular opportunistic infection. This is, when wasting of lean body mass approaches 55% of normal for age, sex, and height, death is imminent regardless of the forces resulting in such profound malnutrition” (20-24). Moreover, the severity of the clinical manifestations of AIDS is proportional to the degree of the nutritional deficiencies (27-30).

In addition to supporting optimal function of the immune system, nutrition is especially critical in children, as it provides the best opportunity for normal growth and development (31,32).

“All persons with HIV infection should be screened for nutritional problems and concerns at the time of their first contact with a health care professional, and routine monitoring should be performed on an ongoing basis” (31).

Scientific evidence strongly suggests that nutritional and antioxidant deficiencies are a prior requisite to both reacting positively on the tests for HIV (ELISA, Western blot, Viral Load) (33-36) and progressing to AIDS (37,38).

### **3 NUTRITIONAL DEFICIENCIES AND THE PROGRESSION OF HIV-POSITIVE INDIVIDUALS TO AIDS.**

An optimal nutritional status as well as adequate vitamin levels are known to be, by themselves, enough to prevent the development of AIDS in people who react positively on the tests for HIV (39-46).

For example, regarding vitamins in HIV disease progression and vertical transmission, researchers from the Harvard School of Public Health state: “The higher rates of HIV progression and vertical transmission in developing countries coincide with similarly higher rates of malnutrition and vitamin deficiencies, indicating that HIV infection, may be modified by nutritional status.” “Numerous observational studies report inverse association between vitamin status, measured bio-chemically or as levels of dietary intake, and the risk of disease progression or vertical transmission.” “Adequate vitamin status may also reduce vertical transmission through the intra-partum and breastfeeding routes by reducing HIV viral load in lower genital secretions and breast milk” and “vitamin supplements may be one of the few potential treatments that are inexpensive enough to be made available to HIV-infected persons in developing countries” (47).

Growing numbers of scientific trials implicate low serum vitamin A levels as a risk factor for HIV-positive individuals to progress to the clinical manifestations of AIDS (48-60).

“The risk of death among HIV-infected subjects with adequate serum vitamin A levels was 78% less, when compared with Vitamin A-deficient subjects” (47,52).

“In a study carried out among HIV-positive homosexual men, development of Vitamin A deficiency over an 18-month period was associated with a decline in CD4 cell count, widely used as a marker of HIV immune impairment. Normalization of vitamin A was associated with higher CD4 cell counts” (37, 47).

“Lower serum levels of vitamin A were associated with a faster rate of progression among men who participated in the Multicenter AIDS Cohort Study (MACS)” (42, 47).

On the other hand, “among well nourished HIV seropositive men who participated in the San Francisco Men’s Health Study, high energy-adjusted vitamin A intake at baseline was associated with higher CD4 cell counts at baseline, as well as with lower risk of developing AIDS during the 6 year period follow up” (44, 47).

“In a placebo-controlled trial in South Africa among children born to HIV-positive women, Vitamin A supplements resulted in approximately 50% reduction in diarrheal morbidity among HIV-infected children” (47,51).

Besides vitamin A, a growing number of studies show that “HIV-positive” individuals are at higher risk of deficiency of vitamins B1, B2, B6, B12, C, D, and E (47,61-68). Furthermore, deficiencies of B-complex vitamins, vitamin C, vitamin E and vitamin D increment the risk of progression of “HIV-positive” individuals to AIDS (47,61-68).

#### **4. NUTRITIONAL DEFICIENCIES AND THE “TRANSMISSION” OF HIV/AIDS.**

Several studies show that vitamin A deficiency is more prevalent among HIV-positive persons compared with HIV-negative individuals (28,38,40,50,57).

Low levels of vitamin A and carotenoid were found to be a risk factor for reacting positively on HIV tests in Pune, India (69), for seroconversion among Kenyan men with genital ulcers (70), and for seroconversion among Rwandan women (71).

There are several trials investigating the role of vitamin A and carotenoid deficiencies in mother to child transmission of HIV/AIDS (MTCT) during pregnancy, delivery, and breastfeeding (72-89).

In Tanzania, for example: “Multivitamin supplementation is a low-cost way of substantially decreasing adverse pregnancy outcomes and increasing T-cell counts in HIV-1 infected women” (72,73).

“A growing body of data suggests that low serum levels of vitamin A among HIV-infected pregnant women is associated with higher risk of vertical transmission of HIV” (47).

“Mean vitamin A concentration in 74 mothers who transmitted HIV to their infants was lower than that in 264 mothers who did not transmit HIV to their infants” (77).

“In Malawi, higher serum retinol of HIV-infected pregnant women was associated with a reduced risk of vertical transmission” (47,77).

“Women who had increasing serum retinol levels over time, however, were at a lower risk, whereas women who had declining serum retinol were at a higher risk of transmitting the virus” (47,89).

“Vitamin A supplementation to a population of HIV-infected pregnant women, many of whom had low vitamin A levels, was associated with a decreased number of preterm births and with reduced mother-to-child transmission of HIV in preterm babies, but was not associated with a reduction in HIV transmission overall. Vitamin A decreased HIV transmission in the preterm babies by 47%” (80).

“Detection of vaginal HIV-1 DNA was associated with abnormal vaginal discharge, lower absolute CD4 cell count, and severe vitamin A deficiency” (85).

**“Women with CD4 cell depletion, especially those with vitamin A deficiency, may be at increased risk of transmitting HIV-1 to their infants through breast milk” (88).**

**“Increased risk of maternal-infant transmission was associated with severe vitamin A deficiency among non-breastfeeding women” in the United States (76).**

Scientific studies, therefore, support the contention that the use of vitamins by themselves, especially vitamin A, could be enough to avoid what is known as transmission of HIV (47,69-89). If this is the case, as many clinical trials and scientific papers contend, supplementation with vitamin A and carotenoids would constitute an effective, inexpensive and non-toxic practice for African countries.

## **5. OXIDATIVE STRESS AND HIV/AIDS.**

Moreover, since the beginning of the AIDS epidemic, free radicals and, specifically, oxidizing agents, have been implicated in the pathogenesis of this new syndrome (90,91). There have been international meetings on the role of oxygen free radicals in HIV/AIDS (92,93).

There are currently increasing numbers of scientific publications demonstrating that oxidizing stress is an absolute requisite for both testing positive on the tests for HIV (94-100) and developing the clinical manifestations of AIDS (101-123).

Free radical reactions of special significance to immunological phenomena include, for example, the many oxidizing agents that can abstract a hydrogen atom from thiol groups to form thiol radicals (124-126). Thiol groups are important for enzyme activities, receptor functions, disulphite links in immunoglobulins, and T cell activation and proliferation. The super oxide anion radical can react with nitric oxide, resulting in a loss of endothelium-derived relaxing factor activity, which is important in the inflammation/disinflammation process. Methionine oxidation can cause protein damage with subsequent changes in immunogenicity. Proteolysis can be increased by free radical damage. The per oxidation of lipids by reactive free radicals produces many biological modulators, such as, for example, the 4-hydroxy-alkelans, which produces strong chemotactic activity for phagocytes, alters the adenyl cyclasa system, increases capillary permeability, and alters lymphocyte activation. Lipid hydroperoxides, also from per oxidation of lipids, alter lymphocyte activation. Conditions favoring lipid per oxidation may result in chemo taxis of leukocytes, protein modification, immune complex injury, and cell death (124-126).

Free radicals are produced throughout the regular immune system network. Despite the beneficial effects of the inflammation responses, it can also aggravate existing tissue damage by releasing free radicals. When uncontrolled, initiated by an abnormal stimulus, or occurring for prolonged periods of time, inflammation may become a disease process (124-126). It is critical for optimal immune responses that there be a balance between free radical generation and antioxidant protection. During phagocytosis by polymorphonuclear leukocytes, for example, super oxide anion radicals are released. These oxygen free radicals can oxidize thiol groups to thiol radicals and can stimulate lipid per oxidation with the formation of  $H_2O_2$ ,

which is highly significant in the mechanisms of cell injury. Oxygen free radicals produced during phagocytosis of immune complexes are associated with injury due to immune complexes (124-126).

It has been proposed many times that free radicals and, specifically, oxidizing species, play important roles in the pathogenesis of AIDS (91-93,127-129).

The above are the scientific fundamentals for the use of antioxidants such as vitamin A and carotenoids, vitamin C, vitamin E, selenium, n-acetyl cysteine, l-gluthamin, zinc, cooper, manganese, alphasipoic acid, coenzyme Q10, and flavonoids or vitamin P, as supplementation for the prevention and treatment of AIDS (90-129).

## **6. NUTRITIONAL AND ANTIOXIDANT DEFICIENCIES AND THE PATHOGENESIS OF HIV/AIDS.**

African countries have a high incidence of malnutrition, vitamin deficiencies, anemia, bacterial, viral, fungal, and parasitic infections and infestations.

For any infectious or parasitic disease to begin, it is always requisite that the host suffers immunodeficiency (130). At the same time, infectious and parasitic diseases cause, by themselves, more immune suppression and more malnutrition (131,132). This immunesuppression is secondary to the accumulation of free radicals, especially oxidizing species, that occur during and after infectious and parasitic diseases (125,133).

Therefore, in African countries, a persistent cycle occurs: poverty, malnutrition, immunesuppression, infectious and parasitic diseases, more immunesuppression, and more malnutrition (134,135).

On the other hand, there is growing scientific data showing that many chronic diseases of adulthood have their origin at “*in utero* programming” (136-139). This includes illnesses such as coronary heart disease and stroke, hypertension, type II diabetes and other endocrine alterations (136-141), as well as several immunological disturbances (142-152). Therefore, it appears that whatever happens during embryonic and fetal times is remembered by cells, tissues, organs, and systems throughout the lifetime.

“Research in Gambia associated season of birth with infectious disease mortality after the age of 15 years, suggesting an association between prenatal undernutrition, immune function, and adult vulnerability to infectious disease” (148,152). Prenatal undernutrition has been found to impair antibody responses to vaccination with *Salmonella thyfi* that last at least up to adolescent times (146). The findings of these researchers “suggest a role for fetal and early infant experience in programming the immune system” which may accompany the individual during its entire life (145,146).

It has been scientifically demonstrated that prenatal undernutrition alters several aspects of cell-mediated immunity, causes involution of lymphoid tissues such as the



thymus, and suppression of antibody responses to vaccination. These deficits persist for weeks or, in some cases, even years (142-152).

In addition, “murine models have documented impairments in immunity after maternal undernutrition that last through adulthood and into the next generation, despite ad libitum feeding of both F1 and F2 generations” (153). Also in mice, deprivation of zinc during pregnancy causes immunodeficiency that can last at least for three generations (154).

It is therefore very probable that in Africa the consequences of poverty and malnutrition are being transmitted from generation to generation with a cumulative effect and that AIDS in Africa may be the topmost consequence of these cumulative effects of poverty.

In this light, the crucial role of maternal undernutrition in the pathogenesis of pediatric AIDS must seriously be considered a reality in developing countries (155,156). This reasoning indicates that malnutrition constitutes the main risk factor for AIDS in adults in developing countries (155,156). Scientifically speaking, there is no rationale for indicting sexual promiscuity as the cause of AIDS in Africa, while underestimating the role of poverty, malnutrition, infections and parasites.

## **7. NUTRITIONAL AND ANTIOXIDANT THERAPY FOR THE PREVENTION AND TREATMENT OF AIDS.**

“It is not surprising, therefore, that dietary therapy for AIDS has been proposed, debated, and, more importantly, surreptitiously or overtly used from the early days of the epidemic” (24).

Twenty years later, researchers insist: “Because nutrient deficiencies may play an important role in the pathogenesis of HIV disease, medical nutrition therapy and counseling are critical aspects of treatment” (31). Nutritional (157-179) and antioxidant (180-198) therapy is therefore requisite in preventing and treating AIDS.

Clinical trials have identified in detail the vitamin and mineral needs of HIV-positive persons and those with AIDS. These studies suggest the need for increasing the intake of the following micronutrients as supplementation for the prevention and treatment of AIDS: vitamin A and carotenoids, vitamin C, vitamin E, selenium, n-acetyl cysteine, l-gluthamin, zinc, cooper, manganese, alphasalipoic acid, coenzyme Q10, flavonoids or vitamin P, and B-complex vitamins (17-38,90-129,157-198).

When providing Vitamin A as a supplement its potential teratogenic property should be kept in mind (199). In this regard, the World Health Organization recommends that pregnant women should not take more than 10,000 IU of Vitamin A per day (47).

If we really want to prevent and treat AIDS in Africa, it is absolutely requisite to provide at least the minimum food needs to HIV-positive individuals, to AIDS patients, as well as to all African communities.

A diet that provides adequate sources of vitamins, minerals, and antioxidants might have quantities of fruits, especially papaya, mango, kiwi, pineapple, avocado, bananas, and dry fruits, and vegetables, legumes, and alga. Use few animal products. Prefer fatty white fish, sheep and goat meat. Prefer sea salt. Use 60-80% fresh, whole, raw organic food. Use garlic, onions, asparagus, beets, cabbage, broccoli, cauliflower, Brussels sprouts, carrots, yeast, wheat, pollen, as well as sprouts, legumes, and cereals. Prefer cool press oils (below 40 degrees Celsius) since this process preserves essential and polyunsaturated fatty acids needed in anti-inflammatory and regenerative processes. Carcama, sunflower, and olive oils, in this order, are good sources of vitamin E or linoleic acid. Lino oil is a good source of alpha linoleic acid. Eat whole cereals in any preparation (rice, barley, wheat, oat). Decrease sugar and candies. Prefer raw organic vegetables and legumes. Drink lots of liquids: water (at least 1.5 liters per day), juices from fresh fruits and vegetables, especially carrots, vegetable broths, and green juices as a source of chlorophyll (for example, blend water, lettuce, spinach, celery, mint, parsley, coriander, and similar ingredients, and take without draining). It is also very convenient to use bifidogenic foods, for example yogurt and kumis better, if made with sheep or goat's milk, tofu, or miso. Coconut oil is a good source of lauric and caprylic acids which are anti-candida (164,169,173-179,200).

Immune stimulating and/or antioxidant herbs include: Aloe (*Aloe vera*), astragalus (*Astragalus membranaceus*), Siberian ginseng (*Eleutherococcus senticosus*), Fo-ti (*Polygonum multiflorum*), turmeric (*Curcuma longa*), echinacea (*Echinacea angustifolia* y *E. purpurea*), garlic (*Allium sativum*), licorice or liquorice (*Glycyrrhiza glabra*), golden seal (*Hydrastis Canadensis*), cat's claw (*Uncaria tomentosa*), ginkgo (*Ginkgo biloba*), grape fruit seeds (*Vitis vinifera*), zarzaparrilla or smilax (*Smilax officinalis* y *S. aspera*), Southerlandia (African herb); sedative and relaxing herbs include peace flower (*Passiflora incarnata*), valerian (*Valeriana officinalis*), chamomile (*Matricaria chamomilla*), mint (*Menta sativa*), lavender (*Lavanda officinalis*), and Siberian ginseng (*Eleuterococcus senticosus*) (182,186,187,200-203).

A large number of scientific papers and books have been published reviewing the issue of nutritional and antioxidant therapy for the prevention and treatment of AIDS (157-206).

## 8. CONCLUSIONS

A) Nutritional and antioxidant deficiencies have been documented in all patients with AIDS.

B) Nutritional and antioxidant deficiencies are a requisite prior to reacting positively on "HIV tests."

C) Nutritional and antioxidant deficiencies are also a requisite of "HIV-positive" individuals prior to the development of the clinical manifestations of AIDS.

**D) Nutritional and antioxidant deficiencies play a major role in the pathogenesis of AIDS.**

**E) Nutritional and antioxidant supplements are being used successfully in preventing and treating AIDS. An optimal nutritional and antioxidant status can guarantee success in preventing and treating AIDS.**

**F) Some of the nutritional and antioxidant supplements that have been used in the treatment and prevention of AIDS are: vitamin A and carotenoids, vitamin C, vitamin E, selenium, n-acetyl cysteine, l-gluthamin, zinc, cooper, manganese, alphalipoic acid, coenzyme Q10, B-complex vitamins, and flavonoids or vitamin P.**

**G) To prevent and treat AIDS in Africa, an absolute requisite is the provision of at least the minimum food needs to HIV-positive individuals, to AIDS patients, and to all African communities. Moreover, diets rich in fresh and organic fruits, vegetables, and cereals, as well as diets rich in bifidogenic foods (yogurt, kumis) are known to be immune stimulants.**

## **REFERENCES**

### **1. NUTRITIONAL IMMUNOLOGY.**

1. Simon J. *A physiological essay on the thymus gland*. London: Ranshaw; 1845: 100.
2. Beisel WR. Single nutrients and immunity. *Am J Clin Nutr* 1982; 35: 417-468.
3. Beisel WR. The history of nutritional immunology. *J Nutr Immunol* 1991; 1: 62-78.
4. Chandra RK. Micronutrients and immune functions, an overview. *Ann NY Acad Sci* 1990; 587: 9-16.
5. Chandra RK. Nutrition and Immunity. In: Lachmann PJ et al. *Clinical aspects of immunology*. Boston: Scientific Publications; 1993: 1325-1338.
6. Chandra RK. Reduced secretory antibody response to live attenuated measles and poliovirus vaccines in malnourished children. *BMJ* 1975; 2: 583-585.
7. Bendich A, Chandra RK. Micronutrients and immune function. New York: New York Academy of Sciences; 1990.
8. Prabhala RH et al. Immunomodulation in humans caused by beta-carotene and vitamin A. *Nutr Res* 1990; 10: 1473.
9. Semba RD. Vitamin A, immunity, and infection. *Clin Inf Dis* 1994; 19: 489-499.
10. Semba RD. The role of vitamin A and related retinoids in immune function. *Nutr Rev* 1998; 56: S38-S48.
11. Chandra RK, Au B. Single nutrient deficiency and cell-mediated immune responses. III. Vitamin A. *Nutr Res* 1981; 1: 181-185.

12. Anderson R, et al. Vitamin C and cellular immune functions. In: Bendich A, Chandra RK. *Micronutrients and immune function*. New York: New York Academy of Sciences 1990: 34-48.
13. Chandra RK. Fetal malnutrition and postnatal immunocompetence. *Am J Dis Chil* 1975; 125: 450-455.
14. Chandra RK. Interactions between early nutrition and the immune system. In: Barker DJL, Whelan J. *The childhood environment and adult disease*. Siba Foundation Symposium # 156. London: Wiley; 1991: 77-88.
15. Gurr MI. The role of lipids in the regulation of the immune system. *Prog Lip Res* 1983; 22: 257-287.
16. Jacob RA, et al. Immunocompetence and oxidant defense during ascarbate depletion of healthy men. *Am J Clin Nutr* 1991; 54: 1302s-1309s.

## 2. NUTRITIONAL DEFICIENCIES AND HIV/AIDS.

17. Jain VK, Chandra RD. Does nutritional deficiency predispose to acquired immunodeficiency syndrome? *Nutr Res* 1984; 4: 537.
18. Beach RS, Laura PF. Nutrition and the acquired immunodeficiency syndrome. *Ann Intern Med* 1983; 99: 565-566.
19. Gray RH. Similarities between AIDS and PCM (Protein Caloric Malnutrition). *Amer J Publ Health* 1983; 73: 1332.
20. Keusch GT, Farthing MJG. Nutritional aspects of AIDS. *Annu Rev Nutr* 1990; 10: 475-501.
21. Coodley GO. Nutritional deficiency and AIDS. *Ann Intern Med* 1990; 113: 809.
22. Bristol-Myers. *Malnutrition and the immune response: Focus on cancer and AIDS*. Princeton, NJ; 1994; 26.
23. Raiten DJ. *Nutrition and HIV infection: A review and evaluation of the extant knowledge of the relationship between nutrition and HIV infection*. FDA Contract No. 223-88-2124; 1990.
24. Keusch GT, Thea DM. Malnutrition in AIDS. *Med Clin NA* 1993; 77(4): 795-814.
25. Beisel WR. AIDS. In: Gershwin ME, German JB, Keen CL. *Nutrition and immunology: Principles and practice*. Totowa, NJ: Human Press; 2000; 389-402.
26. Watson RR. *Nutrition and AIDS*. 2<sup>nd</sup> Ed. Boca Raton: CRC Press; 2001: 231.
27. Chelluri L, Jastremski MS. Incidence of malnutrition in patients with acquired immunodeficiency syndrome. *Nutr Clin Pract* 1989; 4: 16-18.
28. Coodley GO *et al*. Micronutrient concentrations in the HIV wasting syndrome. *AIDS* 1993a; 7: 1595-1600.

29. Kotler DP et al. Body composition studies in patients with the acquired immunodeficiency syndrome. *Am J Clin Nutr* 1985; 42: 1255-1265.
30. Chlebowski RT. Significance of altered nutritional status in acquired immune deficiency syndrome (AIDS). *Nutr Cancer* 1985; 7: 85-91.
31. Mahan LK, Escott -Stump S. Medical nutritional therapy for human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS). In: *Food, nutrition, and diet therapy*. Philadelphia: W.B. Saunders Company. 2000; 889-911.
32. Heller L. Nutrition support for children with HIV/AIDS. *J Am Diet Assoc* 1997; 97: 473.
33. Melchior JC et al. Resting energy expenditure is increased in stable malnourished HIV-infected patients. *Am J Clin Nutr* 1991; 53: 437-441.
34. Ehrenpreis Ed et al. Malabsorption and deficiency of vitamin B12 in HIV-infected patients with chronic diarrhea. *Dig Dis sci* 1994; 39: 2159-2162.
35. Ward BJ et al. Vitamin A status in HIV infection. *Nutr Res* 1993; 13: 157-166.
36. Semba RD et al. Increased mortality associated with vitamin A deficiency during human immunodeficiency virus type 1 infection. *Arch Intern Med* 1993; 153: 2149-2154.
37. Semba RD et al. Vitamin A deficiency and wasting as predictors of mortality in human immunodeficiency virus-infected injection drug users. *JID* 1995; 171: 1196-1202.
38. Karter DL et al. Vitamin A deficiency in non-vitamin-supplemented patients with AIDS: a cross-sectional study. *J AIDS Hum Retrovirol* 1995; 8: 199-203.

### **3. NUTRITIONAL DEFICIENCIES AND THE PROGRESSION OF HIV-POSITIVE INDIVIDUALS TO AIDS.**

39. Baum MK et al. Micronutrients and HIV-1 disease progression. *AIDS* 1995; 9: 1051-1056.
40. Beach RS et al. Specific nutrient abnormalities in asymptomatic HIV-1 infection. *AIDS* 1992; 6: 701-708.
41. Moseson M et al. The potential role of nutritional factors in the induction of immunologic abnormalities in HIV-positive homosexual men. *JAIDS* 1989; 2: 235-247.
42. Tang AM et al. Dietary micronutrient intake and risk progression to acquired immunodeficiency syndrome (AIDS) in human immunodeficiency virus type 1 (HIV-1)-infected homosexual men. *Am J Epidemiol* 1993; 138: 1-15.
43. Bogden JD et al. Micronutrients status and human immunodeficiency virus (HIV) infection. *Ann NY Acad Sci* 1990; 547: 189-195.
44. Abrams B et al. A prospective study of dietary intake and AIDS in HIV-seropositive homosexual men. *JAIDS* 1993; 6: 949-958.

45. Skurnick JH, et al. Micronutrients profiles in HIV-1-infected heterosexual adults. *J Acq Immundef Syndr Hum Retrov* 1996; 12: 75-83.
46. Periquet BA et al. Micronutrient level in HIV-1 infected children. *AIDS* 1995; 9: 887-893.
47. Fawzi WW, Hunter DJ. Vitamins in HIV disease progression and vertical transmission. *Epidemiology* 1998; 9: 457-466.
48. Fawzi WW et al. A randomized trial of vitamin A supplements in relation to mortality among human immunodeficiency virus-infected and uninfected children in Tanzania. *Pediatr Infect Dis J* 1999; 18: 127-133.
49. Tang AM et al. Association between serum vitamin A and E levels and HIV-1 disease progression. *AIDS* 1997; 11: 613-620.
50. Ullrich R et al. Serum carotene deficiency in HIV-infected patients. *AIDS* 1994; 8: 661-665.
51. Coutsooudis A et al. The effects of vitamin A supplementation on the morbidity of children born to HIV-infected women. *Am J Public Health* 1995; 85: 1076-1081.
52. Semba RD et al. Vitamin A deficiency and wasting as predictors of mortality in human immunodeficiency virus-infected injection drug users. *JIF* 1994; 171: 1196-1202.
53. Semba RD. Vitamin A and human immunodeficiency virus infection. *Proc Nutr Soc* 1997; 56: 1-11.
54. Landesman S. Vitamin A relationships to mortality in HIV disease and effects on HIV infection: recent and late breaking studies. Presented at forum, Lawton Chiles International House, *National Institutes of Health*, Bethesda, MD, May 16, 1996.
55. Tang AM et al. Association between serum vitamin A and E levels and HIV-1 disease progression. *AIDS* 1997; 11: 613-620.
56. Garewal HS et al. A preliminary trial of beta-carotene in subjects infected with the human immunodeficiency virus. *J Nutr* 1992; 122: 728-732.
57. Ullrich R et al. Serum carotene deficiency in HIV-1 infected patients. *AIDS* 1994; 8: 661-665.
58. Loya S et al. The carotenoid halocynthiaxanthin: a novel inhibitor of the reverse transcriptase of human immunodeficiency viruses type 1 and type 2. *Arch Biochem Biophys* 1992; 293: 208-212.
59. Watson RR et al. Enhanced survival by vitamin A supplementation during retrovirus infection causing murine AIDS. *Life Sci* 1988; 43: 13-18.
60. Yang Y et al. Retinoic acid inhibition of *ex vivo* human immunodeficiency virus-associated apoptosis of peripheral blood cells. *Proc Natl Acad Sci USA* 1995; 92: 3051-3055.
61. Harakeh S, Jariwalla RJ, Pauling L. Suppression of human immunodeficiency virus replication by ascarbate in chronically and acutely infected cell. *Proc Natl Acad Sci U.S.A.* 1990; 87: 7245-7249.
62. Harakeh S et al. Mechanistic aspects of ascarbate inhibition of human immunodeficiency virus. *Chemico-biological Interactions* 1994; 91: 207-215.

63. Tang AM et al. Association between serum vitamin A and E levels and HIV-1 disease progression. *AIDS* 1997; 11: 613-620.
64. Wang Y et al. Nutritional status and immune responses in mice with murine AIDS are normalized by vitamin E supplementation. *J Nutr* 1994; 124: 2024-2032.
65. Wang Y et al. Modulation of immune function and cytokine production by various levels of vitamin E supplementation during murine AIDS. *Immunopharmacol* 1995; 29: 225-233.
66. Tang AM et al. Low serum vitamin B12 concentrations are associated with faster human immunodeficiency virus type 1 (HIV-1) disease progression. *J Nutr* 1997; 127: 345-351.
67. Baum MK et al. Association of vitamin B6 status with parameters of immune function in early HIV-1 infection. *JAIDS* 1991; 4: 1122-1132.
68. Haug C et al. Subnormal serum concentration of 1,25 -vitamin D in HIV infection: Correlation with degree of immune deficiency and survival. *JID* 1994; 169: 889-893.

#### 4. NUTRITIONAL DEFICIENCIES AND THE "TRANSMISSION" OF HIV/AIDS.

69. Mehendale SM et al. Low carotenoid concentration and the risk of HIV seroconversion in Pune, India. *JAIDS* 2001; 26: 352-359.
70. McDonald KS et al. Vitamin A and risk of HIV-1 seroconversion among Kenyan men with genital ulcers. *AIDS* 2001; 15: 635-639.
71. Moore PS et al. Role of nutritional status and weight loss in HIV seroconversion among Rwandan women. *JAIDS* 1993; 6: 611-616.
72. Fawzi WW et al. Randomized trial of vitamin supplements in relation to vertical transmission of HIV-1 in Tanzania. *JAIDS* 2000; 23: 246-254.
73. Fawzi WW et al. Randomized trial of effects of vitamin supplements on pregnancy outcomes and T cell counts in HIV-1-infected women in Tanzania. *Lancet* 1998; 351: 1477-1482.
74. Landers DV. *1995 Nutrition and immune function II: Maternal factors influencing transmission. Nutrition in pediatric HIV infection: Setting the research agenda*, Bethesda, MD, September 1995.
75. Stiehm RE. Newborn factors in maternal-infant transmission of pediatric HIV infection. In: *Nutrition in pediatric HIV infection: Setting the Research Agenda*. Bethesda, MD: NIH, September 1995.
76. Greenberg BL et al. Vitamin A deficiency and maternal-infant transmission of HIV in two metropolitan areas in the United States. *AIDS* 1997; 11: 325-332.
77. Semba RD et al. Maternal vitamin A deficiency and mother-to-child transmission of HIV-1. *Lancet* 1994; 343: 1593-1597.

78. Phuapradit W et al. Serum vitamin A and betha-carotene levels in pregnant women infected with human immunodeficiency virus-1. *Ostet Gynecol* 1996; 87: 564-567.
79. Semba RD et al. Infant mortality and maternal vitamin A deficiency during human immunodeficiency virus infection. *Clin Inf Dis* 1995; 21: 966-972.
80. Coutsooudis A et al. Ramdomized trial testing the effect of vitamin A supplementation on pregnancy outcomes and early mother-to-child HIV-1 transmission in Durban, South Africa. *AIDS* 1999; 13: 1517-1524.
81. Lan Y et al. Carotenoid status of pregnant women with and without HIV infection in Malawi. *East Afr Med J* 1999; 76: 133-137.
82. Semba RD *et al.* Maternal vitamin A deficiency and mother-to-child transmission of HIV-1. *Lancet* 1994a; 343: 1593-1597.
83. Semba RD et al. Maternal vitamin A deficiency and child growth failure during human immunodeficiency virus infection. *JAIDS* 1997; 14: 219-222.
84. Coutsooudis A et al. Effect of vitamin A supplementation on viral load in HIV-1 infected pregnant women. *JAIDS* 1997; 15: 86-87.
85. John GC et al. Genital shedding of human immunodeficiency virus type 1 DNA during pregnancy: association with immunosuppression, abnormal cervical or vaginal discharge, and severe vitamin A deficiency. *JID* 1997; 175: 57-62.
86. Mostand SB et al. Hormonal concentration, vitamin A deficiency, and other risk factors for shedding of HIV-1 infected cells from cervix and vagina. *Lancet* 1997; 350: 922-927.
87. Shah RS *et al.* Liver stores of vitamin A in human fetuses in relation to gestational age, fetal size and maternal nutritional status. *Br J Nutr* 1987; 58: 181-189.
88. Nduati RW et al. Human immunodeficiency virus type-1 infected cells in breast milk: association with immunosuppression and vitamin A deficiency. *JID* 1995; 172: 1461-1468.
89. Landesman S. *Vitamin A relationships to mortality in HIV disease and effects on HIV infection: recent and late breaking studies.* Presented at forum, Lawtom Chiles International House, National Institutes of Health, Bethesda, MD, May 16, 1996.

##### 5. OXIDATIVE STRESS AND HIV/AIDS.

90. Dworkin BM, Rosenthal W, Wormser G, Weiss L. Selenium deficiency in the acquired immuno-deficiency syndrome. *J Parenteral Enteral Nutr* 1986; 10: 405.
91. Papadopulos-Eleopulos E. Reappraisal of AIDS – Is the oxidation induced by the risk factor the primary cause? *Medical Hypothesis* 1988; 25: 151-162.
92. Favier A. The place of oxygen free radicals in HIV infection. A collection of papers presented at a conference on “The place of oxygen free radicals in HIV infection”, Les Deux Alpes, France, January 1993. *Chemico-Biological Interactions* 1994; 91: 77-224.



93. Piette et al. Molecular mechanisms of virus activation by free radicals. Collection of 5 articles presented at a conference on "The place of oxygen free radicals in HIV infection" Les Deux Alpes, France, January 1993. *Chemico-Biological Interactions* 1994; 91: 79-132.
94. Fuchs J et al. Oxidative imbalance in HIV infected patients. *Med Hypothesis* 1991; 36: 60-64.
95. Shallenberger F. Selective compartmental dominance: An explanation for a noninfectious, 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.
96. Jarstrand C, Akerlund B. Oxygen radical release by neutrophils of HIV-infected patients. *Chemico-Biological Interactions* 1994; 91: 141-146.
97. Salvain B, Mark AW. The role of oxidative stress in disease progression in individuals infected by the human immunodeficiency virus. *J Leukocyte Biol* 1992; 52: 111.
98. Baruchel S, Wainberg MA. The role of oxidative stress in disease progression in individuals infected by the human immunodeficiency virus. *J Leukocyte Biol* 1992; 51: 111-114.
99. Polkyov VM et al. Superoxide anion production and enzymatic disbalance in peripheral blood cells isolated from HIV-infected children. *Free Radic Biol Med* 1994; 16: 15-21.
100. Hommes MJT et al. Resting energy expenditure and substrate oxidation in human immunodeficiency virus (HIV)-infected asymptomatic men: HIV affects host metabolism in the early asymptomatic stage. *Am J Clin Nutr* 1991; 54: 311-315.
101. Passi S. Progressive increase of oxidative stress in advanced human immunodeficiency. *Continuum* (London) 1998; 5(4): 20-26.
102. Favier A et al. Antioxidant status and lipid peroxidation in patients infected with HIV. *Chemico-Biological Interactions* 1994; 91: 165-180.
103. Fuchs J et al. Oxidative imbalance in HIV infected patients. *Med Hypothesis* 1991; 36: 60-64.
104. Lacey CJ et al. Antioxidant-micronutrients and HIV infection. *International J STD & AIDS* 1996; 7: 485-489.
105. Javier JJ et al. Antioxidant micronutrients and immune function in HIV-1 infection. *FASEB Proc* 1990; 4A: 940-945.
106. Allard VP et al. Oxidative stress and plasma antioxidant micronutrients in humans with HIV infection. *Am J Clin Nutr* 1998; 67: 143-147.
107. Revillard JP, Favier AE, Zittoun M. Lipid peroxidation in human immunodeficiency virus infection. *J AIDS* 1992; 5: 637-638.
108. Constants J et al. Fatty acids and plasma antioxidants in HIV-positive patients: correlation with nutritional and immunological status. *Clinical Biochemistry* 1995; 28: 421-426.

109. Dworkin BM. Selenium deficiency in HIV infection and the acquired immunodeficiency syndrome (AIDS). *Chemico-Biological Interactions* 1994; 91: 181-186.
110. Cirelli A et al. Serum selenium concentration and disease progress in patients with HIV infection. *Clin Biochem* 1991; 24: 211-214.
111. Schrauzer GN, Sacher J. Selenium in the maintenance and therapy of HIV-infected patients. *Chem Biol Inter* 1994; 91: 199.
112. Simon G et al. Effects of glutathione precursors on human immunodeficiency virus replication. *Chemico-Biological Interactions* 1994; 91: 217-224.
113. Staal FJT et al. Intracellular glutathione levels in T-cells subsets decrease in the blood plasma of HIV-1 infected patients. *Biol Chem Hoppe Seyler* 1989; 370: 101-108.
114. Buhl R et al. Systemic glutathione deficiency in symptom-free HIV seropositive individuals. *Lancet* 1989; ii: 1294-1298.
115. Dorge W, Eck HL, Mihm S. HIV-induced cysteine deficiency and T-cell dysfunction: a rationale for treatment with n-acetyl-cysteine. *Immunol Today* 1992; 13: 211.
116. Passi S et al. Study on plasma polyunsaturated fatty acids and vitamin E, and on erythrocyte glutathione peroxidase in highrisk HIV infection categories and AIDS patients. *Clin Chem Enzym Comms* 1993; 5: 169-177.
117. Quey B et al. Glutathione depletion in HIV-infected patients: role of cysteine deficiency and effect of oral n-acetyl-cysteine. *AIDS* 1992; 5: 814.
118. Kalevic T et al. Suppression of human immunodeficiency virus expression in chronically infected monocytic cells by glutathione, glutathione ester and N-acetyl-cysteine. *Proc Natl Acad Sci U.S.A.* 1991; 88: 986
119. Fabris N et al. AIDS, zinc deficiency and thymic hormone failure. *JAMA* 1988; 259: 839.
120. Walter R et al. Zinc status in human immunodeficiency virus infection. *Life Sci* 1990; 47: 1579.
121. Falutz J et al. Zinc as a cofactor in HIV-induced immunosuppression. *JAMA* 1988; 259: 2850-2851.
122. Graham N et al. Relationship of serum copper and zinc levels to HIV-1 seropositivity and progression to AIDS. *JAIDS* 1991; 4: 976-980.
123. Staal FJT et al. Antioxidants inhibit stimulation of HIV transcription. *AIDS Res Hum Retroviruses* 1993; 9: 299-306.
124. Kehrer JP. Free radicals as mediators of tissue injury and disease. *Crit Rev Toxicol* 1993; 23: 21-48.
125. Slater TF. Free radicals: formation, detection, reactivity, and cytotoxicity. In: Lachman PJ, Peters SK, Rosen FS, Walport MJ. *Clinical aspects of immunology*. Boston: Blackwell Scientific Publications; 1993: 377-393.
126. Reid L. Oxidative stress and antioxidants. A nutritional perspective. *Continuum (London)* 1998; 5(3): 52-54.

127. Papadopulos-Eleopulos E. Looking back on the oxidative stress theory of AIDS. *Continuum* ( London ) 1998/1999; 5(5): 30-35.
128. Papadopulos-Eleopulos E, et al. Oxidative stress, HIV and AIDS. *Res Immunol* 1992; 143: 145-148.
129. Giraldo RA. Role of free radicals in immunodeficiency. In: *Aids and Stressors*. Medellín: Impresos Begón; 1997: 72-75.

## 6. NUTRITIONAL AND ANTIOXIDANT DEFICIENCIES AND THE PATHOGENESIS OF AIDS.

130. Peterson PK. Host defense abnormalities predisposing the patient to infection. *Amer J Med* 1984; 72: 2.
131. Playfair JHL. Overview: parasitism and immunology. In: Llachmann PJ et al. *Clinical aspects of immunology*. Boston: Blackwell Scientific Publications; 1993: 1439-1454.
132. Ware RE, Kline MW. Immunodeficiency secondary to infectious disease. In: Rich RR et al. *Clinical immunology: principles & practice*. St. Louis: Mosby; 1996: 808-826.
133. Slater TF. Free radicals mechanisms in tissue injury. *Biochem J* 1984; 222: 1-15.
134. Chandra RK. Nutrition, immunity and infection: present knowledge and future directions. *Lancet* 1983; i: 688-691.
135. Giraldo RA. Papel de las enfermedades tropicales en el debilitamiento del sistema inmunológico y en la fisiopatogénesis del sida. In: *Sida y agentes estresantes*. Medellín: Editorial Universidad de Antioquia; 2002: 37-46.
136. Barker DJ. In utero programming of chronic disease. *Clin Sci* (Colch) 1998a; 95: 115-128.
137. Barker DJ. *Fetal and infant origins of adult diseases*. London: BMJ Publishing Group; 1992; 343.
138. Barker DJ. *Mothers, babies and diseases in later life*. London: BMJ Publishing Group; 1994.
139. Barker DJ. *Mothers, babies & health in later life*. 2<sup>nd</sup> ed. Church Press 1998b; 217.
140. Naeye RL et al. Relation of poverty and race to birth weight and organ and cell structure in the newborn. *Pediat Res* 1971; 5: 17 -22.

141. Leon DA. Fetal growth and adult disease. *Eur J Clin Nutr* 1998; 52(suppl ): S72-S82.
142. Chandra RK. Fetal malnutrition and postnatal immunocompetence. *Am J Dis Child* 1975b; 129: 450-454.
143. Chandra RK. Serum thymic hormone activity and cell-mediated immunity in healthy neonates, preterm infants, and small-for-gestational age infants. *Pediatrics* 1981; 67: 407-411.
144. Ferguson AC. Prolonged impairment of cellular immunity in children with intrauterine growth retardation. *J Pediatr* 1978; 93: 52-56.
145. McDade TW *et al.* Prenatal undernutrition is associated with reduced immune function in adolescence. *FASEB* 2000; 14: A792 (abs.).
146. McDade TW *et al.* Prenatal undernutrition, postnatal environments, and antibody response to vaccination in adolescence. *Am J Clin Nutr* 2001a; 74: 543-548.
147. McDade TW *et al.* Prenatal undernutrition and postnatal growth are associated with adolescent thymic function. *J Nutr* 2001b; 131: 1225-1231.
148. Moore SE *et al.* Prenatal or early postnatal events predict infectious deaths in young adulthood in rural Africa. *Int J Epidemiol* 1999; 28: 1088-1095.
149. Lewis D, Wilson C. Developmental immunology and the role of host defenses in neonatal susceptibility. In: Remington J, Klein J. *Infectious diseases of the fetus and newborn infant*. 4<sup>th</sup> ed. Philadelphia: W.B. Saunders; 1995; 108-139.
150. Moscatelli P *et al.* Defective immunocompetence in foetal undernutrition. *Helv Paediatr Act* 1976; 31: 241-247.
151. Hasselbachh H *et al.* Thymus size in infants from birth until 24 months of age evaluated by ultrasound. *Acta Radiol* 1999; 40: 41-44.
152. Moore SE *et al.* Season of birth predicts mortality in rural Gambia. *Nature* 1997; 338: 434.
153. Chandra RK. Antibody formation in first and second generation offspring of nutritional deprived rats. *Science* 1975c; 190: 289-290.
154. Beach RS *et al.* Gestational zinc deprivation in mice: persistence of immunodeficiency for three generations. *Science* 1982; 281: 469-471.
155. Giraldo RA. AIDS and Stressors II: A proposal for the pathogenesis of AIDS. In: *AIDS and Stressors*. Medellín, Colombia : Impresos Begón ; 1997; 57-96.

156. Giraldo RA. AIDS and Stressors III: A proposal for the natural history of AIDS. In: *AIDS and Stressors*. Medellín , Colombia : Impresos Begón ; 1997; 97-131.

**7. NUTRITIONAL AND ANTIOXIDANT THERAPY FOR THE PREVENTION AND TREATMENT OF AIDS.**

157. Steinbrook R. Battling HIV on many fronts. *NEJM* 1997; 337: 779.
158. Goldberg B. AIDS. In: *Alternative Medicine. The definitive guide*. Fife, Washington: Future Medicine Publishing Inc.; 1994: 494-509.
159. Abrams DI. Alternative therapies. In: Sande MA, Volberding PA. *The medical management of AIDS*. 6a ed. Filadelfia: W.B. Saunders Company; 1999: 601-612.
160. Null G. Alternative treatments. In: *AIDS: A second opinion*. New York: Seven Stories Press; 2002: 487-581.
161. Badgley L. *Healing AIDS naturally: natural therapies for the immune system*. Foster City, California: Human energy Press; 1990: 410.
162. Byrnes S. *Overcoming AIDS with natural medicine: A program for recovery*. Revised, 2<sup>nd</sup> edition. Honolulu, Hawaii: Ecclesia Life Mana; 2001: 183.
163. Chaitow L. *The natural way: HIV & AIDS. Your guide to complementary therapies, alternative techniques and conventional treatments*. Shaftesbury, UK: Element Books Limited; 1999: 150.
164. Passi S, De Luca C. Dietetic advice for immunodeficiency. *Continuum* (London) 1998-1999; 5(5): 43-52.
165. Ferguson A, Griffin GE. Nutrition and the immune system. In: Garrow JS, James WPT, Ralph A. *Human nutrition and dietetics*. Edinburgh: Churchill Livingstone; 2000: 747-766.
166. Embid A. Inmunidad aumentada por incremento de micronutrients. *Medicinas Complementarias* 1994; No. 35: 172.
167. Embid A. Inmunoterapia a dosis infinitesimales. *Medicinas Complementarias* 1995; No. 38: 170.
168. Gerrior J, Wanke C. Nutrition and Immunodeficiency Syndromes. In: Coulston AM, Rock CL, Monsen ER. *Nutrition in the prevention and treatment of disease*. San Diego: Academic Press; 2001; 741-750.
169. Life Sciences Research Office, FASEB. Nutritional therapy and nutrition education in the care and management of AIDS patients. Tentative report, Task Order 7. Washington, DC: Center for Food Safety and Nutrition, FDA, DHHS, 1990.
170. Tang AM et al. Effects of micronutrients intake on survival in human immunodeficiency virus type 1 infection. *Am J Epidemiol* 1996; 143: 1244-1256.

171. Romeyn M. *Nutrition and HIV: A new model for treatment*. San Francisco : Jossey -Bass Publishers; 1995: 353.
172. Young J. HIV and medical nutrition therapy. *J Amer Diet Assoc* 1997; 97: S161.
173. Collins CL. Nutrition care in AIDS. *Diet Curr* 1988; 15: 1.
174. Fenton M, Silverman E. Medical nutritional therapy for human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS). In: Mahan K, Escott-Stump S. *Krause's Food, nutrition and diet therapy*. Philadelphia: W.B. Saunders Company; 2000: 889-911.
175. Hickson JF. Diet and nutrition for optimal immune function. In: Bahl SM, Hickson JF. *Nutritional care for HIV-positive persons: a manual for individuals and their caregivers*. Boca Raton: CRC Press; 1995: 1-36.
176. Fenton M, Silverman E. Medical nutrition therapy for human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS). In: Mahan LK, Escott-Stump S. *Food, nutrition, & diet therapy*. Philadelphia: W.B. Saunders Company; 2000: 889-911.
177. American Dietetic Association (ADA). Position of the ADA and the Canadian Dietetic Association: Nutrition intervention in the care of persons with HIV infection. *J Am Diet Assoc* 1994; 94: 1042.
178. American Dietetic Association (ADA), HIV/AIDS medical nutrition therapy protocol. Medical nutrition therapy across the continuum of care, 2<sup>nd</sup> ed. Chicago: ADA, 1998.
179. Bahl SM, Hickson JF. *Nutritional care for HIV-positive persons: A manual for individuals and their caregivers*. Boca Raton: CRC Press; 1995: 190.
180. Bendich A. Role of antioxidants in the maintenance of immune function. In: Frei. *Natural antioxidants in human health and disease*. (Chapter IV, Immunity and Infection). San Diego: Academic Press; 1994: 447-467.
181. Bendich A. Antioxidant micronutrients and immunity. In: Roitt IM, Delves PJ. *Encyclopedia of immunology*. San Diego: Academic Press; 1992: 151-153.
182. Fishman RHB. Antioxidants and phytotherapy. *Lancet* 1994; 344: 1356.
183. Turner VF. Reducing agents and AIDS - Why are we waiting? *Med J Austr* 1990; 153:502.
184. Halliwell B, Cross C. Reactive oxygen species, antioxidants and acquired immunodeficiency syndrome. *Arch Intern Med* 1991; 151: 29-31.
185. Adam ES. Antioxidant supplementation in HIV/AIDS. *Nurse Practit* 1995; 20: 8.
186. Greenspan HC. The role of oxidative oxygen species, antioxidants and phytopharmaceuticals in human immunodeficiency virus activity. *Med Hypothesis* 1993; 40: 85.

187. Greenspan HC, Arouma O. Oxidative stress and apoptosis in HIV infection: a role for plant-derived metabolites with synergistic antioxidant activity. *Immunol Today* 1994; 15: 209.
188. Greenspan HC, Arouma O. Could oxidative stress initiate programmed cell death in HIV infection? A role for plant derived metabolites having synergistic antioxidant activity. *Chemico-Biological Interactions* 1994; 91: 187-197.
189. Tang AM et al. Improved antioxidant status among HIV-infected injecting drug users on potent antiretroviral therapy. *JAIDS* 2000; 23: 321-326.
190. Byrnes SC. Staying on top of oxidative stress. *Healthy and Natural Journal, Millenium Wellness Guide* 1999. sbyrnes@chaminade.edu. Available in <http://www.powerhealth.net>.
191. Zhang Z, Inserra PF, Watson RR. Antioxidants and AIDS. In: Garewal HS. *Antioxidants and disease prevention*. Boca Raton: CRC Press; 1997; 45-66.
192. Garewal HS. *Antioxidants and disease prevention*. Boca Raton: CRC Press; 1997: 186.
193. Sies H. *Oxidative stress: oxidants and antioxidants*. London: Academic Press; 1991: 507.
194. Frei B. *Natural antioxidants in human health and disease*. San Diego: Academic Press; 1994: 588.
195. CoodleyY GO. Beta-carotene therapy in human immunodeficiency virus infection (Abstract). *Clin Res* 1991; 29: 634A.
196. Coodley GO et al. Beta-carotene in HIV infection. *JAIDS* 1993; 272-276.
197. Coodley et al. Beta-carotene in HIV infection: an extended evaluation. *AIDS* 1996; 10: 967-973.
198. Cathart R. Vitamin C in the treatment of acquired immune deficiency syndrome (AIDS). *Med Hypothesis* 1984; 14: 423.
199. Rothman KJ et al. Teratogenicity of high vitamin A intake. *NEJM* 1995; 333: 1369-1373.
200. Rogers SA. *Man does not live by bread alone: enzymes, juicing, cleansing, flushing and brushing*. In: *Wellness against all odds*. Syracuse, NY: Prestige Publishing; 1994: 110-144.
201. Schultz V, Hansel R, Tyler VE. Agents that increase resistance to disease: adaptogens; immune stimulants; botanical antioxidants. In: *Rational phytotherapy. A physician guide to herbal medicine*. Springer; 1998: 269-273, 273-281, 282-286.
202. Yuan-kun L et al. Modulation of immune responses. In: *Handbook of probiotics*. New York: John Wiley & Sons, Inc.; 1999: 161-177.
203. Embid A. El yogur estimula la inmunidad. *Medicinas Complementarias* 1994; No. 35: 171.
204. Giraldo RA. An effective alternative for the prevention of AIDS. Internet Discussion during the South African Presidential AIDS Advisory

- Panel, 2000  
 <<http://www.robertogiraldo.com/eng/papers/AnEffectivePreventionForAIDS.html>>
205. Giraldo RA. An effective alternative for the treatment of AIDS. Internet Discussion during the South African Presidential AIDS Advisory Panel, 2000.  
 <<http://www.robertogiraldo.com/eng/papers/AnEffectiveTreatmentForAIDS.html>>
206. Giraldo RA, Ródenas P, Flores JJ, Embid A. Tratamiento y prevención del sida: guía de principios básicos para una alternativa no tóxica, efectiva y barata. November 2002  
[http://www.robertogiraldo.com/esp/articulos/Tratamiento\\_y\\_Prevenccion\\_2002.html](http://www.robertogiraldo.com/esp/articulos/Tratamiento_y_Prevenccion_2002.html)

## 18. APPENDIX C

### Circumcision and AIDS in Africa

By Roberto Giraldo, June 2000

The idea that HIV, the virus that supposedly causes AIDS, is "heterosexually transmitted" in African countries and mostly "homosexually transmitted" in Western countries cannot be explained by known epidemiological rules.

This is why, since the beginning of AIDS in Africa, when reports showed that the syndrome was equally distributed in men and women, researchers have been speculating about explanations for what they call "An Epidemiologic Paradigm" (1).

The difficulty is that the belief that HIV is the cause of AIDS prevents health care professionals, researchers, journalists, and lay people from perceiving genuine explanations for the ways in which the AIDS epidemic is manifesting itself within different communities, countries, and continents. HIV is an obstacle to discovering the objective causes of AIDS. Nor does the HIV theory permit proper measures to be taken to stop the spread of the epidemic. This is the true danger of HIV!

The following are some of the reasons that researchers who believe that HIV is the cause of AIDS have given to explain why, in Africa, AIDS affects both sexes equally: late age at marriage; sexual cravings and excesses; gross heterosexual promiscuity; high levels of polygyny; the rubbing of monkey's blood into cuts as an aphrodisiac; truck drivers who get HIV from prostitutes and then infect their wives; duration of postpartum abstinence; women being allowed to participate in commerce and maintain separate budgets from husbands; high levels of sterility caused by widespread sexually transmitted diseases; unusual sexual practices that facilitate transmission; the practice of female circumcision; the lack of male circumcision; etc. (2-6).

Western health experts and journalists accuse Africans of gross heterosexual promiscuity. Do they have proof for it? Recently, Nobel Prize winner Nadine Gordimer wrote in the



New York Times that African promiscuity "is difficult to condemn when sex is the cheapest or only available satisfaction for people society leaves to live on the street" (7).

Regarding male circumcision, the following are among the arguments that defenders of HIV as the cause of AIDS provide to promote male genital mutilation in Africa (2,8-12):

"A joint Canadian-Kenyan medical research team working in Kenyatta Medical School in Nairobi, where the epidemic is intense, had reported a year earlier that AIDS rates were higher among Luo migrants from western Kenya than among the Kikuyu from central Kenya." Later the authors "surmised that the Luo were at greater risk because, unlike the Kikuyu, they were not circumcised" (2,10).

"An American team led by John Bongaarts of the Population Council published a paper showing that the regions across sub-Saharan Africa with high levels of HIV infection among local peoples corresponded well with the areas where men were typically uncircumcised" (9).

"Most of the ideas we investigated failed to explain the extraordinarily high rate of infection in the AIDS belt. One factor did stand out, however: lack of male circumcision. In the area where men are typically uncircumcised, HIV rates are among the highest in the AIDS belt" (2).

"We noted that the areas of Africa with large numbers of uncircumcised men were almost exactly the same as the regions suffering from the severe AIDS epidemic," and "The link between lack of circumcision and elevated levels of HIV infection appears robust" (2).

"For uncircumcised men, thorough cleaning of the genitals can be particularly challenging" (2).

"Outside the AIDS belt, in the city of Abidjan, the capital of Ivory Coast, levels of HIV infection are as high as they are in some cities of the AIDS belt; we believe the epidemic in Abidjan is very likely sustained by immigrants who come from a surrounding area where the majority of men are uncircumcised" (2).

"Thus, we concluded that in the AIDS belt, lack of male circumcision in combination with risky sexual behavior, such as having multiple sex partners, engaging in sex with prostitutes and leaving chancroid untreated, has led to rampant HIV transmission. Unsafe sexual practices have certainly contributed to the spread of AIDS across Africa and indeed around the world" (2).

HIV researchers have gone further: "In sub-Saharan Africa, circumcision could be offered as a reinforcement of other protective measures" (2).

"These men are appearing at hospitals in sharply increasing numbers, requesting circumcision for themselves and often for their sons. Clinics that offer adult male circumcision as a protection against AIDS now advertise in Tanzanian newspapers" (2).

However, HIV researchers knew in advance that such measures would not be sufficient: "Although the epidemic in sub-Saharan Africa may subside somewhat, because of greater

use of condoms and probably increased incidence of circumcision, Africans in the AIDS belt remain at extremely high risk of HIV infection" (2). These researchers are opening doors to pharmaceutical companies to bring to Africa the expensive "help" of the World Bank, to medicate with immunotoxic antiretrovirals HIV-positive Africans and those who are merely presumed to be positive (13).

The words of a professor of African History speak for themselves: "Racist assumptions about African sexuality merit scrutiny. Generalizations about African sexual practices are analytically useless but perpetuate racist stereotypes about insatiable sexual appetites and carnal exotica. Media misinterpretations of African sexuality and its alleged link to AIDS have spawned inordinate anxieties and pervasive despair in regions already afflicted with extreme poverty, ravaged by war, and deprived of primary health care delivery systems" and, he continues, "the political economy of underdevelopment and environmentally caused endemic sickness, not extraordinary sexual behavior or a sexually transmitted virus, are what's killing Africans. The so-called AIDS epidemic has become the medicalization of poverty to justify Western medical intervention in the form of vaccine trials, drug testing, and evangelistic demands for behavior modification. AIDS scientists and public health planners must reconsider the role of malnutrition, poor sanitation, anemia, and parasitic and endemic infections for producing the clinical AIDS symptoms that are manifestations of non-HIV insults" (5).

Belief in HIV prevents the understanding that AIDS in Africa is occurring now because never before has poverty been so prevalent and intense as it is now in the African areas where AIDS is epidemic. The only rational way to stop the spread of the AIDS epidemic in the African continent is by finding solutions for the economic disparities that are rampant in Africa (14,15).

AIDS in Africa is not an epidemiologic paradigm. There exists a serious crisis in the scientific methodology; currently, the problem is that epidemiologic ignorance is pandemic. Let us go back to the teaching of epidemiology to find a solution to AIDS in Africa and elsewhere (16-41).

President Thabo Mbeki is absolutely correct when he demands a scientific answer to the question: "Why is HIV/AIDS in sub-Saharan Africa heterosexually transmitted while in the Western world it is said to be largely homosexually transmitted?"

I am certain that Africans will continue questioning and rejecting the ethnic fictions and racial slanders described here. They are already standing up to defend their integrity.

## **REFERENCES**

1. Quinn TC, Mann JM, Curran JW, Piot P. AIDS in Africa: An Epidemiologic Paradigm. *Science* 1986; 234: 956-963.
2. Caldwell JC, Caldwell P. The African AIDS Epidemic. In parts of sub-Saharan Africa, nearly 25 percent of the population is HIV-positive as a result of heterosexual transmission of the virus. Could lack of circumcision make men in this region particularly susceptible? *Scientific American* 1996; 274(3): 62-68.

3. Geshekter CL. Rethinking AIDS in Africa. *Reappraising AIDS* 1995; 3(2): 1-4
4. Geshekter CL. Outbreak? AIDS, Africa, and the Medicalization of Poverty. Is Africa facing a lethal pandemic? *Transition* 1995; 5(3): 5-14.
5. Geshekter CL. AIDS, Underdevelopment, and Sexual Stereotypes: Rethinking AIDS in Africa. 39<sup>th</sup> Annual Meeting of the African Studies Association. San Francisco, California. November 23-26, 1996.
6. Bethell T. Mbeki takes on the AIDS Industry. South African President queries epidemic, *AZT*. *Reappraising AIDS* 2000; 8(3): 1-4.
7. Gordimer N. Africa's Plague, and Everyone's" *The New York Times*. April 11, 2000.
8. Ankomah B. Are 26 millions Africans Dying of AIDS? "The biggest lie of the century" under fire. *New African* 1998; No. 369: 34-42.
9. Bongaarts J, Reining P, Way P, Conant F. The Relationship Between Male Circumcision and HIV Infection in African Populations. *AIDS* 1989; 3(6): 373-377.
10. Moses S et al. Geographical Patterns of Male Circumcision Practices in Africa: Association with HIV Seroprevalence. *Internat J Epidemiol* 1990; 19(3): 693-697.
11. Orubuloye IO, Caldwell JC, Caldwell P, Santow G Editors. *Sexual Networking and AIDS in Sub-Saharan Africa: Behavioural Research and the Social Context*. Australian National University. 1994.
12. Forum: The East African AIDS Epidemic and the Absence of Male Circumcision: What is the Link? *Health Transition Review* 1995; 5(1): 97-117.
13. World Bank. *Confronting AIDS: Public Priorities in a Global Epidemic*. A World Bank Policy Research Report. New York: Oxford University Press; 1999: 365.
14. Giraldo R. *AIDS and Stressors: AIDS is not an infectious disease nor is sexually transmitted. It is a toxic-nutritional syndrome caused by the alarming worldwide increment of immunological stressor agents*. Medellin, Colombia: Impresos Begon, 1997: 205.
15. Giraldo R et al. Is it rational to treat or prevent AIDS with toxic antiretroviral drugs in pregnant women, infants, children, and anybody else? The answer is negative. *Continuum (London)* 1999; 5(6): 38-52.
16. Abramson JH. *Making Sense of Associations. Factors and Risk Markers. Causes and Effects*. In: *Making Sense of Data; A Self- Instruction Manual on the Interpretation of Epidemiological DATA*. New York: Oxford University Press, 1988: 193-264, 219-228 and 265-316.

17. Buck C, Llopis A, Najera E, et al. Etiologic Investigations. Studies in Epidemics. In: The Challenge of Epidemiology, Issues and Selected Readings. Pan American Health Organization, Scientific Publication No. 505. PAHO, Pan American Sanitary Bureau, Regional Office of the WHO. Washington DC, 1988: 147-166 and 415-482.
18. Elwood JM. The Diagnosis of Causation. In: Causal Relationships in Medicine. A Practical System for Critical Appraisal. New York: Oxford University Press, 1988: 163-182.
19. Elwood JM. The Importance of Causal Relationships in Medicine and Health Care. What is Causation? A Direct Test of Causation. In: Critical Appraisal of Epidemiological Studies and Clinical Trials. Oxford: Oxford University Press, 1998: 3-13.
20. Fletcher RH, Fletcher SW, Wagner EH. Risk. Cause. In: Clinical Epidemiology: the Essentials. Baltimore: Williams and Wilkins, 1996: 94-110 and 228-248.
21. Friedman GD. Making Sense out of Statistical Associations. In: Primer of Epidemiology. New York: McGraw-Hill, Inc., 1994: 194-224.
22. Garb JL. Understanding Medical Research. A Practitioner's Guide. Boston: Little, Brown and Company, 1996: 256.
23. Gordis L. Estimating Risk: Is There an Association? From Association to Causation: Deriving Inferences From Epidemiologic Studies. More on Causal Inferences: Bias, Confounding, and Interactions. In: Epidemiology. Philadelphia: W.B. Saunders Company, 1996: 141-154, 167-182 and 183-195.
24. Hutt MSR, Burkitt DP. Environment and the causes of disease. In: The Geography of Non-Infectious Disease. Oxford: Oxford University Press, 1986: 1-6.
25. Jekel JF, Elmore JG, Katz DL. The Study of Causation in Epidemiologic Investigations and Research. Assessment of Risk in Epidemiologic Studies. In: Epidemiology, Biostatistics and Preventive Medicine. Philadelphia: W.B. Saunders Company, 1996: 54-64 and 74-84.
26. MacMahon B, Trichopoulos D. Concepts of Cause. In: Epidemiology Principles and Methods. Boston: Little Brown and Company, 1996: 19-30.
27. Malenka DJ, Baron JA, Johnson S, et al. The Framing Effect of Relative and Absolute Risk. J Gen Intern Med 1993; 8:543-548.
28. McMaster University Health Services Centre, Department of Clinical Epidemiology and Biostatistics. How to Read Clinical Journals IV: To Determine Etiology or Causation. Can Med Assoc J 1981; 124:985-990.

29. Rothman KJ. Causal Inference in Epidemiology. Multiple Analysis. Interactions Between Causes. Analysis with Multiple Levels of Exposure. In: Modern Epidemiology. Boston: Little Brown, 1986: 7-22, 285-310, 311-326 and 327-350.
30. Rothman KJ, Greenland S. Causation and Causal Inference. In: Detels R et al. Oxford Textbook of Public Health. Third Edition. Volume 2; The Methods of Public Health. New York: Oxford University Press, 1997: 617-630.
31. Rothman KJ, Greenland S. Causation and Causal Inference. In: Modern Epidemiology. Lippincott – Raven, 1998: 7-28.
32. Schlesselman JJ. "Proof" of Cause and effect in Epidemiologic Studies: Criteria for Judgments. Prev Med 1987; 16:195-210.
33. Sheldon H. Causes of Disease. In: Boyd's Introduction to the Study of Disease. Philadelphia: Lea & Febiger, 1992: 49-82.
34. Soskolne CL, MacFarlane DK. Scientific Misconduct in Epidemiologic Research. In: Coughlin SS, Beauchamp TL. Ethics in Epidemiology. New York: Oxford University Press, 1996: 274-289.
35. Stolley Pd, Lasky T. Epidemics and Science. In: Investigating Disease Patterns. The Science of Epidemiology. New York: Scientific American Library, 1995: 1-22.
36. Streiner DL, Norman GR. Assessing Causation. In: PDQ Epidemiology. St. Louis: Mosby, 1996: 121-134.
37. Susser M. Causal Thinking in the Health Sciences: Concepts and Strategies of Epidemiology. Oxford: Oxford University Pres, 1973: 181.
38. Susser M. What is a Cause and How Do We Know One? A Grammar for Pragmatic Epidemiology. Amer J Epidemiol 1991; 133:635-648.
39. Torrence ME. Causality. In: Understanding Epidemiology. St. Louis: Mosby, 1997: 133-151.
40. Weed DL. On the Logic of Causal Inference. Am J Epidemiol 1986; 123: 965-979.
41. Weiss NS. Natural History of Illness. In: Clinical Epidemiology: The Study of the Outcome of Illness.

## **19. APPENDIX D**

# **The Origin of the "Transmission" of AIDS**

By Roberto Giraldo, June 2000

The transmission of AIDS from person to person is a myth. The homosexual transmission of AIDS in Western countries, as well as the heterosexual transmission of AIDS in Africa and in other underdeveloped countries, is an assumption made without any scientific validation.

Nor is there any logical scientific explanation for AIDS being "transmitted" primarily through homosexual sex in the West and "heterosexually" in poor countries. The sexual transmission of AIDS is an assumption based upon the high frequency with which AIDS occurs in drug addicted gay males in the developed world, and the similar frequency of the syndrome in both sexes within the underdeveloped countries.

Whenever there is an outbreak of a new disease, the first question to answer is: What are the new circumstances surrounding the individuals contracting the new illness.

In the report of the first 5 cases of AIDS to the CDC by Michael Gottlieb in June, 1981, he informed the CDC that "four had serologic evidence of past hepatitis B infection," "two of the five reported having frequent homosexual contacts with various partners," "all 5 patients had laboratory-confirmed CMV disease or virus shedding within 5 months of the diagnosis of *Pneumocystis pneumonia*," and "all five reported using inhalant drugs, and one reported parental drug abuse" (Gottlieb et al MMWR 1981; 30:250-252).

There is nothing in this report that could suggest a sexually transmissible germ as the cause for the new condition. Homosexuality has existed forever! The new circumstances around the people who were experiencing the collapse of their immune systems was the use of drugs by some members of the gay community in the USA and Europe, use that began in the late sixties and early seventies. The toxic nature of AIDS has been evident since the very first report of the new syndrome. There was no need to posit a microbe as the cause of this new toxic condition.

However, the Centers for Disease Control (CDC) decided, in an editorial note commenting upon Gottlieb's report, that "the fact that these patients were all homosexuals suggests an association between some aspect of homosexual lifestyle or disease acquired through sexual contact and *Pneumocystis pneumonia* in this population" (MMWR 1981; 30:250-252).

Even long before HIV was discovered and proposed as the cause of AIDS, the US Public Health Service decided upon the contagious nature of the new syndrome. On November 5, 1982 the CDC published "AIDS: Precautions for Clinical and Laboratory Staffs" (MMWR 1982; 31:577-580). Four months later the CDC, together with the Food and Drug Administration and the National Institutes of Health, recommended the prevention of AIDS as if it were an infectious and transmissible disease (MMWR 1983; 32:101-104). It was decided that AIDS was infectious and sexually transmitted despite the absence of any scientific proof.

In his recent book Luc Montagnier confesses: "It was in 1982 that AIDS began to capture the attention of researchers. By that time we knew, by the number of reported cases among homosexuals, that we were dealing with a communicable disease." (Montagnier

L. Virus: The Co-Discoverer of HIV Tracks Its Rampage and Charts the Future; New York: WW Norton & Co., 2000: page 42).

Robert Gallo, writing about "The AIDS Virus" in 1987, stated: "AIDS is probably the result of a new infection of human beings that began in Central Africa, perhaps as recently as the 1950's," and "it appears that the virus has had more time to spread in Africa than it has had in any other part of the world." Regarding the so-called T-lymphotropic virus III (STLV-III) of African green monkeys, he said: "A plausible hypothesis is that STLV-III somehow entered human beings, initiating a series of mutations that yielded the intermediate viruses before terminating in the fierce pathology of HTLV-III" (Gallo R. Scientific America 1987; 256:47-56).

The above is the supposedly "scientific" basis for the infectious and contagious view of AIDS.

The researchers and the institutions searching for the cause of AIDS in the early 1980's, groups that today continue making worldwide AIDS policies, seem to ignore that there exist epidemics of toxic diseases, epidemics of nutritional deficiencies, epidemics of high blood pressure, epidemics of cancer, epidemics of mental diseases, epidemics of allergies, etc. They consider only epidemics of infectious diseases. In addition, it would seem that these groups ignored the diseases that regularly affect gay people.

These researchers and their institutions are impregnated with a microbiologic prejudice, by which all diseases must be caused by germs. Gallo and Montagnier have both spent most of their lives searching for the virus that causes cancer.

It is unnecessary to emphasize the ethnic and sexual prejudices inherent in the above statements.

The world, conditioned throughout a century to panic toward microbes and other prejudices, committed a mistake concerning the etiology of AIDS. There was no way to avoid it. Similar errors had been committed with pellagra, beriberi, and scurvy, to mention only a few examples. Tragically, this time the consequences of the mistake are far more severe.

All individuals partake in sexual activity. Similarly, all people eat and sleep. The epidemiological correlation of AIDS with sexual life, as well as with eating and sleeping, is perfect. Therefore, just as it is said that AIDS is sexually transmitted, it could be said that it is transmitted through eating and sleeping.

During recent decades the novel circumstances surrounding gay males who developed AIDS in the West included exposure through their life style to drugs and other immunological stressors. In the West AIDS is mostly confined to male homosexuals because they are more frequently exposed to immunological stressor agents, not because of their sexual preferences. Homosexuality has always existed. However, in the late sixties and early seventies, gay males in the United States and Europe introduced drugs and aphrodisiacs to their life styles.

On the other hand, the novel circumstances surrounding individuals of both sexes in the poorest countries of Africa, Asia, and the Caribbean, are their involuntary exposure to the never before seen high levels of poverty, malnutrition, unsanitary conditions, infections, and parasites. Here both sexes are equally exposed to immunological stressor agents. Therefore, in these countries the risk for AIDS is equal for both genders.

The perinatal transmission of AIDS from mother to child during pregnancy and delivery, as well as the postnatal transmission through breast milk, are also myths without any scientific validation. Both mothers and infants who react positively on the tests for HIV, or who develop AIDS, do so due to exposures to immunological stressor agents.

Currently, humans, animals, and plants around the world are suffering from some level of immune suppression due to multiple, repeated, and chronic exposure to the alarming worldwide increment in immunological stressor agents, which can have chemical, physical, biological, mental, and nutritional origins. Immunodeficiency is pandemic.

Individuals can be exposed to immunological stressors involuntarily through their conditions of life and voluntarily through their life styles.

AIDS is the worst possible immunological human condition; if the course of AIDS is not arrested, it will eventually cause the death of the individual. Additionally, AIDS is the tip of an iceberg; beneath AIDS there are many other mild to moderate immunodeficiencies with or without clinical manifestations.

AIDS began in the second half of the last century, at a moment when the immune systems of humans were already saturated and could not tolerate further challenges and aggravations. AIDS is an alarm bell for an endangered species. However, belief in HIV prevents the real danger from being seen and precludes the taking of proper measures. The increasing epidemic of AIDS in underdeveloped countries of Africa and Asia demands that strong measures be taken before the populations on these continents vanish.

In the seventies a new medical science was born, IMMUNOTOXICOLOGY, which studies the effects of toxicants that can poison the immune system. About thirty years ago immunologists began to be preoccupied by the increasing amount of new immunosuppressive conditions that animals and humans were suffering due to voluntary or involuntary exposures to a great variety of substances and materials.

Similarly, during the last few decades, and due to the alarming worldwide increment in stressor agents affecting the human ecosystem, new medical sciences have had to be created, including dermatotoxicology, genotoxicology, neurotoxicology, endocrinotoxicology, cardiotoxicology, and hepatotoxicology. We must devote our attention to this and take the necessary actions to guarantee the future of our species. We ought to cease panicking about germs. Currently, the genuine problems are toxicants, poverty, and malnutrition.

Selected references concerning immunotoxicology:

1973 CUSHMAN P & GRIECO M. Hyperimmunoglobulinemia Associated with Narcotic Addiction. *Amer J Med* 54:320.



- 1976 ANDERSON RE & WARNER NL. Ionizing Radiation and the Immune Response. *Adv Immunol* 24:215.
- 1977 VOS JG. Immune Suppression as Related to Toxicology. *CRC Crit Rev Toxicol* 5:67.
- 1978 BEKESI JG, HOLLAND JF, et al. Lymphocyte Function of Michigan Dairy Farmers Exposed to Polybrominated Biphenyls. *Science* 199:1207.
- 1979 MOORE JA. The Immunotoxicology Phenomenon. *Drug Chem Toxicol* 2:1.
- 1982 DALLY S, WAUTIER J, THOMAS G. Grande Frequence des Complexes Immunes Circulants Chez les Toxicomanes. *Nouv Presse Med* 11:1011.
- 1983 GIBSON GG, HUBBARD R & PARKR DV. Immunotoxicology: Proceedings of the First International Symposium on Immunotoxicology, Held at the University of Surrey, England, 13-17 September 1982. London, New York: Academic Press. 505 p.
- 1986 HOLSAPPE MP & MUNSON AE. Immunotoxicology of Abused Drugs. In: DEAN JH et al. *Immunotoxicology and Immunopharmacology*. New York: Raven Press. P. 381.
- 1987 SHARMA RP & REDDY RV. Toxic Effects of Chemicals on the Immune System. In: HALEY TJ & BERNDT WO. *Handbook of Toxicology*. Washington: Hemisphere Publishing Corporation. P. 555.
- 1987 BERLIN A, DEAN J, FRAPER MH, et al. Immunotoxicology. Proceedings of the International Seminar on the Immunological System as a Target for Toxic Damage. Dordrecht: Martinus Nijhoff.
- 1987 LUSTER MI, BLANK JA & DEAN JH. Molecular and Cellular Basis of Chemical Induced Immunotoxicity. *Ann Rev Pharmacol Toxicol* 27:23.
- 1988 FIDELIOS RK. The Generation of Oxygen Radicals: A Positive Signal for Lymphocyte Activation. *Cell Immunol* 113:175-182.
- 1988 WOLFF SP & DEAN RT. Fragmentation of Proteins by Free Radicals and its Effects on Their Susceptibility to Enzymatic Hydrolysis. *Biochem J* 234:399-403.
- 1988 OLDHAM KT, GUICE KS, WARD PA & JOHNSON JK. The Role of Oxygen Radicals in Immune Complex Injury. *Free Radical Biol Med* 4:387-397.
- 1988 BELLAVITE P. The Superoxide-Forming Enzymatic System of Phagocytes. *Free Radical Biol Med* 4:225-261.
- 1988 DESCOTES JACQUES. *Immunotoxicology of Drugs and Chemicals*. Second Edition. Elsevier: Amsterdam. 450 p.
- 1988 AHLBOM A. A Review of the Epidemiologic Literature on Magnetic Fields and Cancer. *Scand J Work Environ Health* 14:337-343.

- 1990 DEAN JH, CORNACOFF JB & LUSTER MI. Toxicity to the Immune System: A Review. In: HADDEN JW & SZENTIVANYI A. Immunopharmacology Reviews, Vol 1. New York: Plenum Press. P. 377.
- 1991 PRYOR WA & GODBER SS. Oxidative Stress Status: An Introduction. Free Radical Biol Med 10:173.
- 1991 TYRRELL RM. UVA (320-380nm) Radiation as an Oxidative Stress. In: SIES H. Oxidative Stress: Oxidants and Antioxidants. London: Academic Press. P. 57-83.
- 1992 FISCHBEIN A & TARCHER AB. Disorders of the Immune System. In: TARCHER AB. Principles and Practice of Environmental Medicine. Part V. Disorders Associated with Exposure to Environmental Chemicals and Physical Agents. New York: Plenum Medical Book Company. P: 389-411.
- 1992 ANDERSON RE. Effects of Low Dose Radiation on the Immune Response. In: CALABRESE EJ. Biological Effects of Low Level Exposures to Chemicals and Radiation. Boca Raton: Lewis.
- 1992 MILLER K, TURK JL & NICKLIN S. Principles and Practice of Immunotoxicology. Boston: Blackwell Scientific. 379 p.
- 1993 LAKE JV, BOCK GR & ACKRILL K. Environmental Change and Human Health. Ciba Foundation Symposium 175. Chichester, New York: John Wiley & Sons. 270 p.
- 1993 SAHL JD, KELSH MA, GREENLAND S. Cohort and Nested Case Control Studies of Hematopoietic Cancers and Brain Cancer Among Electric Utility Workers. Epidemiology 4:104.
- 1993 SLATER TF. Free Radicals: Formation, Detection, Reactivity and Cytotoxicity. In: LACHMAN PJ et al. Clinical Aspects of Immunology. Fifth Edition, Volume 1. Boston: Blackwell Scientific Publications. P. 377-393.
- 1993 KEHRER JP. Free Radicals as Mediators of Tissue Injury and Disease. Crit Rev Toxicol 23:21-48.
- 1994 PRYOR WA. Free Radicals and Lipid Peroxidation: What They are and How They Got That Way. In: FREI B. Natural Antioxidants in Human Health and Disease. Chapter 1. Oxidants and Antioxidants. San Diego: Academic Press. P. 1-23.
- 1994 BENDICH A. Role of Antioxidants in the Maintenance of Immune Function. In: FREI B. Natural Antioxidants in Human Health and Disease. Chapter IV Immunity and Infection. San Diego: Academic Press. P. 447-467.
- 1994 FRIEDMAN H, SCHIVERS SC, KLEIN TW. Drugs of Abuse and the Immune System. In: DEAN JH, et al. Immunotoxicology and Immunopharmacology. New York: Raven Press. P. 303-322.

- 1994 WESTER PW, VETHAAK AD & VAN MUISWINKEL WB. Fish as Biomarker in Immunotoxicology. *Toxicology* 86(3):213-232.
- 1994 LUSTER MI, PORTIER C, PAIT DG & GERMOLEC DR. Use of Animal Studies in Risk Assessment for Immunotoxicology. *Toxicology* 92(1-3):229-243.
- 1994 FUCHS BA & SANDERS VM. The Role of Brain-Immune Interactions in Immunotoxicology. *Crit Rev Toxicol.* 24(2):151-176.
- 1994 SCHUURMAN HJ, KUPER CF & VOS JG. Histopathology of the Immune System as a Toll to Assess Immunotoxicity. *Toxicology* 86(3):187-212.
- 1994 TRYPHONAS H. Immunotoxicity of Polychlorinated Bephenyls: Present Status and Future Considerations. *Exp Clin Immunogenet* 11(2-3):149-162.
- 1994 DALE M, FOREMAN JC & FAN TPD. *Textbook of Immunopharmacology*. Boston: Blackwell Scientific Publications. 370 p.
- 1994 DEAN JH, LUSTER MI, MUNSON AE & KIMBER I. *Immunotoxicology and Immunopharmacology*. Second Edition. New York: Raven Press. 761 p.
- 1995 HORTON WF & GOLBERG S. *Power Frequency, Magnetic Fields and Public Health*. Boca Raton: CRC Press. 276 p.
- 1995 SZENTIVANYI A, ALI K, ABARCA C, PROCKOP L & BROOKS SM. Environmental Immunotoxicology. In; BROOKS SM et al. *Environmental Medicine*. St. Louis: Mosby. P. 139-155.
- 1995 MACCOA CA, SZENTINANYI A, ALI K, et al. Disorders of the Immune System. In: BROOKS SM et al. *Environmental Medicine*. St. Louis: Mosby. P. 326-350.
- 1996 RODGERS K. Biology of the Immune System and Immunotoxicity. In: FAN AM, CHANG LW. *Toxicology and Risk Assessment: Principles, Methods, and Applications*. New York: Marcel Dekker. P. 71-80.
- 1996 BURNS LA, MEADE BJ & MUNSON AE. Toxic Responses of the Immune System. In: KLAASEN CD, AMDUR MO, DOULL J. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. Fifth Edition. Unit 4.12. New York: McGraw Hill. P. 355-401.
- 1996 LUSTER MI. Immunotoxicology: Clinical Consequences. *Toxicol Ind Health* 12(3-4):533-535.
- 1996 DE SWART RL, ROSS PS, VOS JG & OSTERHOUS AD. Impaired Immunity in Harbour Seals (*Phoca vitulina*) Exposed to Bioaccumulated Environmental Contaminants: Review of a Long-Term Feeding Study. *Environ Health Perspect* 104 Suppl 4:823-828.

1997 SAINT-REMY JM. Epitope Mapping: A New Method for Biological Evaluation and Immunotoxicology. *Toxicology* 119(1):77-81.

1997 HOUSE RV, THOMAS PT & BHARGAVA HN. Immunotoxicology of Opioids, Inhalants and Other Drugs of Abuse. *NIDA Res Monog # 173* p175-200.

1997 NEAUNE PH & LECOEUR S. Immunotoxicology of the Liver: Adverse Reactions to Drugs. *J Hepatol* 26 Suppl 2:37-42.

1998 VANDEBRIEL RJ, VAN LOVEREN H & MEREDITH C. Altered Cytokine (Receptor) mRNA Expression as a Tool in Immunotoxicology. *Toxicology* 130 (1):43-67.

1998 THOMAS PT. Immunotoxicology: Hazard Identification and Risk Assessment. *Nutr Rev* 56(1 Pt 2) pS131-134.

1998 BIAGINI RE. Epidemiology Studies in Immunotoxicity Evaluations. *Toxicology* 129(1):37-54.

1998 ZELIKOFF JT. Biomarkers of Immunotoxicity in Fish and Other Non-Mammalian Sentinel Species: Predictive Value for Mammals? *Toxicology* 129(1):63-71.

1998 KIMBER I & SELGRADE MJK. T Lymphocyte Subpopulations in Immunotoxicology. Chichester: John Wiley & Sons. 302 p.

1998 VAN LOVEREN H, GERMOLEC D, KOREN HS, LUSTER MI, NOLAN C, REPETTO R, SMITH E, VOS JG & VOGT RF. Report of the Bilthoven Symposium: Advancement of Epidemiological Studies in Assessing the Human Health Effects of Immunotoxic Agents in the Environment and the Workplace. International Program on Chemical Safety (UNEP-ILO-WHO), the World Resources Institute (Washington, DC), the European Science Foundation, the National Institute of Environmental Health Sciences (USA), the Environmental Protection Agency (USA), the National Institute for Occupational Safety and Health (USA), CDC (USA), and the American Crop Protection Association (Washington DC). November 12-14, 1997, Bilthoven, The Netherlands. RIVM, National Institute of Public Health and the Environment. Bilthoven, The Netherlands. RIVM, P.O. Box 1, 3720 BA Bilthoven. Tel 31 30 2749111. Fax 31 30 2742971.

## **20. APPENDIX E**

### **Breastfeeding and AIDS in Africa**

By Roberto Giraldo, June 2000

For more than a decade publications have been addressing the possibility that AIDS can be transmitted through breastfeeding. The United Nations' agencies UNAIDS, UNICEF,

and WHO have suggested that HIV-positive mothers stop breastfeeding to avoid the transmission of HIV/AIDS from mother to child.

In the USA several states have gone further and have regulated the matter, making HIV testing mandatory for all pregnant women and their babies. Mothers and babies who react positively on tests for antibodies to HIV are medicated with antiretrovirals, and mothers are forced not to breastfeed their babies. Cesarean sections and the cleansing of the birth canal with antiseptic solutions are also suggested (1,2). Additionally, effective on June 1<sup>st</sup> 2000, the State of New York passed a new law requiring that every person who reacts positively on the "tests for HIV" must be reported to health authorities (name and address is required) (3).

Regarding the underdeveloped countries, UN agencies have engaged in a great deal of speculation upon these matters (4,5).

However, there is no objective evidence for the hypothesis that HIV/AIDS can be transmitted from mother to child through breast milk. This is an assumption without any scientific validation. Careful analysis of the entire body of research on HIV/AIDS reveals a great deal of bias. The trials conducted to test for HIV transmission through breastfeeding are no exception; they contain serious bias as well.

Permit me to analyze briefly several reports concerning frequently referenced experiments:

*1. Bobat R, Moodley D, Coutsooudis A, Coovadia H. Breastfeeding by HIV-1-infected Women and Outcome in Their Infants: A Cohort Study From Durban, South Africa. AIDS 1997; 11: 1627-1633.*

In this study the authors were able to follow 133 infants that were born HIV-negative to HIV-positive mothers. 21 infants (16%) were fed exclusively on formula, 36 infants (27%) exclusively breastfed, and 76 (57%) received both breast and formula feeds.

The South African researchers concluded: "it was found that infants who were exclusively formula-fed had a lower transmission rate (24%) than those who received either mixed feeding (32%) or were exclusively breastfed (39%); the relative risk for infection in the exclusively breastfed versus those on formula only, was 1.63 (CI, 0.71-3.76;  $P = 0.24$ )." And "there was a stepwise increase in the transmission rate with duration of exclusive breastfeeding of 1, 2, and 3 months (45%, 64%, and 75%, respectively)."

The researchers also concluded: "Deaths occurred only in the HIV-infected infants. Of the 17 infected infants who died, seven were exclusively breastfed and 10 had mixed feeding. No deaths occurred in the exclusively formula-fed group during the study period, compared to a mortality of seven out of 36 (19%) in the exclusively breastfed infants, and of 10 out of 76 (13%) in the infants receiving mixed feeding." And "we found mortality to be highest in the exclusively breastfed infants; seven out of 14 (50%), compared to 10 out of 24 (42%) in the infants receiving mixed feeding and 0 out of 5 (0%) in those infants receiving formula only."

"Among the infected infants, seven out of 14 (50%) of those exclusively breastfed, 13 out of 23 (54.1%) on mixed feeding, and none out of four (0%) on formula only, developed AIDS during the study period."

However, following are two of the more evident biases present in this report:

a) This study is strongly influenced by the researchers' beliefs. The authors believe that AIDS is an infectious disease caused by HIV, that AIDS is a transmissible disease, that a positive result in tests for antibodies to HIV is indicative of infection with HIV, and that once positive on these tests the individual will develop AIDS, to mention just some of the most evident assumptions, easily seen on reading the paper.

The authors craft definitions according to their beliefs. In this manner they declare: "Infants were regarded as infected if they were antibody positive at 15 months or had an HIV-related death." And: "They were classified as non-infected if the antibody test was negative from 9 months of age, or if death was non HIV-related"

The authors tested for antibodies to HIV in both maternal and infant blood by ELISA and immunofluorescent assays. "Samples were considered positive if a second ELISA or the IFA was positive."

The authors defined "transmission of HIV" from HIV-positive mother to infant through breastfeeding as an infant reacting positively on tests for antibodies to HIV after having reacted negatively at birth on the same tests.

However, if one defines "intoxicated" individuals — in this case infants — as those that react positively on the tests for HIV, and "non-intoxicated" those that react negatively, and if one assumes that the only source of intoxication is breast milk, the conclusion would be that what is being "transmitted" from mothers to infants are toxins, not HIV. But this conclusion would also be wrong since it negates the possibility of becoming intoxicated from exposure to external agents while being breastfed. The source of intoxication could be environmental toxins that have nothing to do with breastfeeding. Breastfeeding would then constitute a practice that occurs at the same time that infants are being intoxicated. The longer the time of breastfeeding, the longer the exposure to toxins, and so the greater the possibility of becoming intoxicated and testing positive on the so-called tests for HIV. The intoxication would occur independently of breastfeeding, formula feeding, or mixed feeding.

Also, since it was assumed that breastfeeding could be a source of transmission of the virus that supposedly causes AIDS, the South African researchers did not search for exposure to chemical, physical, biological, or nutritional immunological stressors as risk factors for reacting positively on the tests for HIV and for developing AIDS. They did not feel the need to search for other risk factors. For them, "HIV antibodies" explain everything. It would seem that these researchers are unaware of the immunotoxic properties of hundreds of stressor agents that South African families are often exposed to from the very moment of their birth (6,7).

Neither do the South African researchers describe in their article the financial position of the families involved in this study. They do not consider the possibility that mothers who

fed their babies only with formula enjoyed better financial conditions (they were able to afford formula) and therefore would have less exposure to immunological stressor agents and therefore the risk that their babies would react positively or would develop AIDS was lower.

The researchers also “found that infants who were exclusively formula fed had a lower transmission rate (24%).” However, researchers did not offer any explanation as to how these 5 infants were infected with HIV. Since the researchers assumed that infants were infected with HIV through their feedings, this could be interpreted to mean that these infants were infected with HIV from the formula itself or from the bottles in which the formula was placed.

Bobat and coworkers do not consider the possibility that babies who became positive on the tests for HIV months after birth, and who developed AIDS, did so probably due to having been exposed, like their mothers, to more immunological stressor agents than the ones that did not (6,8), and that this is unrelated to breastfeeding.

b) The researchers did not use controls. They state: “As the benefits of breastfeeding were well established, we did not include a control group of HIV-negative pregnant women and their offspring.” And “the women were not randomly allocated to breastfeeding versus non-breastfeeding groups; they self-selected their feeding method. It has been argued, among key research scientists, that randomized studies in poor countries will be unethical.”

It is extraordinary that the South African researchers did not consider it unethical to come to conclusions on breastfeeding based upon an uncontrolled study.

In light of these biases one cannot accept the conclusions from this study as being scientifically valid.

2. *Becquart P, Garin B, Sepou A, et al. Early Postnatal Mother-to-Child Transmission of HIV-1 in Bangui, Central African Republic. Abstract 242/Session 33. 5<sup>th</sup> Retrovir Oppor Inf. 1998 February 1-5; 124 (Abstract No. AIDS/98929169). Viomed <[http://130.14.32.44/cgi-bin/version\\_B/IGT-client?16132+detail+16](http://130.14.32.44/cgi-bin/version_B/IGT-client?16132+detail+16)>*

In this study, reported at the 5<sup>th</sup> Conference on Retroviruses and Opportunistic Infections, the authors concluded that “21 of 43 [48%] children were not infected at 6 months, and were therefore at risk for late postnatal HIV transmission. 14 [32%] children were infected perinatally, and 8 [19%] children postnatally.” The authors conclude: “These results underline that about 20% of children born from HIV-1-infected mothers are becoming HIV-1-infected by breastfeeding before 6 months. Stopping breastfeeding after 6 months, as previously proposed, could not reduce early postnatal HIV transmission; bottle-feeding or stopping breastfeeding earlier than 6 months should be more convenient.”

This study was carried out by African researchers, together with researchers from the laboratory on retroviruses at the Pasteur Institute in Paris, including Dr. Barre-Sinoussi, the principal author of the paper that in 1983 reported what was supposedly the first “isolation” of the virus currently known as HIV (9).

This research, concerning “Early Postnatal Mother-to-child Transmission of HIV-1 in Bangui, Central African Republic,” is also replete with bias. It is strongly influenced by the researchers beliefs. They state: “Breastfed children born to HIV-positive mothers are known to be at substantial risk of late postnatal HIV transmission.” However, the researchers provide no scientific evidence for presuming that infants “are known to be at substantial risk of late postnatal transmission.” They ignore the dictum that science is built on facts, not on “known” beliefs.

The African and French researchers employ definitions in accordance with what is “known” or believed about HIV causing AIDS: “HIV-1 infection was assessed by a positive PCR”; “HIV transmission was defined by a positive HIV-1 PCR at birth or 1 month”; “it was further confirmed by genetic relatedness between viral strains from PBMC’s child and those from breast milk.”

The African and French researchers ignored all scientific publications documenting that PCR is not specific for HIV infection (10,11). They are unaware that the reactivity of the PCR test for HIV can also be explained as part of the response of cells to exposure to a variety of stressors or oxidizing agents, rather than due to an infection with a virus named HIV (11). The authors also ignore the immunotoxic properties of malnutrition, infections, parasites, and other consequences of poverty from which many African communities suffer. They prefer to place the blame on HIV. They cannot see the genuine cause of AIDS in Africa. HIV does not permit them to see it.

*3. Lewis P, Nduati r, Kreiss JK, et al. Cell-Free Human Immunodeficiency Virus Type 1 in Breast Milk. J Inf Dis 1998; 177: 34-39.*

In this study, carried out by researchers at the University of Washington, Seattle, and the University of Nairobi, Kenya, 75 samples of breast milk from “HIV-1-seropositive women” were analyzed by quantitative competitive reverse transcription — polymerase chain reaction — and “HIV-1 RNA was detected in 29 (39%).” Additionally, they found that “the prevalence of cell-free HIV-1 was higher in mature milk (47%) than in colostrum (27%)”; and “Because mature milk is consumed in large quantities, these data suggest that cell-free HIV-1 in breast milk may contribute to vertical transmission of HIV-1.”

Again, this study is biased: no controls were used. Doctor Lewis and his colleagues did not match their breast milk specimens with breast milk from HIV-1-seronegative women. They do not consider possibilities other than HIV infection in explaining the PCR positive reactions to breast milk. It seems that they do not know that the PCR test can react positively in the absence of HIV (12,13).

Dr. Lewis and his group believe that the only reason for reacting positively on HIV-1 PCR is infection with HIV-1. It seems that they do not know that both antibody tests and amplification tests (PCR) for HIV can react positively to more than 70 different common conditions (8,10,11,14,15), all related to oxidative processes (14,16,17). Neither did they consider the possibility that the reactivity for HIV-1-QC-RT-PCR was higher in mature milk than in colostrum simply because mature milk may contain a higher amount of free



radicals — oxidizing agents — than colostrum, as happens in most human processes (18-21).

4. *Dunn DT, Newell ML, Ades AE, Peckham CS. Risk of Human Immunodeficiency Virus Type 1 Transmission Through Breastfeeding. Lancet 1992; 340: 585-588.*

In this review article from the Unit of Epidemiology and Biostatistics, Institute of Child, London, the authors came to the conclusion that “based on four studies in which mothers acquired HIV-1 postnatally, the estimated risk of transmission is 29%.” And this analysis of five studies showed that “when the mother was infected prenatally, the additional risk of transmission through breastfeeding, over and above transmission in uterus or during delivery, is 14%.”

It is extraordinary that these authors, who ought to be familiar with epidemiology, did not realize that all of the studies that they analyzed are biased by the belief that reactivity to the tests for HIV is due only and exclusively to an active infection with HIV. None of the articles that Dunn, Newell, Ades, and Peckhman analyzed consider the possibility that mothers and infants can react positively on the tests for HIV due to the exposure to stressor or oxidizing agents not related to HIV (6,8,11). They did not consider “human immunodeficiency virus type 1 transmission through breastfeeding” to be a strong epidemiological confounding factor.

In this review article it is easily seen that the authors were strongly influenced by the mainstream belief that HIV is the cause of AIDS, that it is transmitted through body fluids, and that testing positively on the tests for HIV means active infection with HIV. HIV did not permit the authors to consider other possibilities. HIV is by itself a source of bias.

In one of the articles analyzed in the above review study, one which is frequently cited as proof for of the transmission of HIV through breastfeeding, the authors consider the presence of “HIV antibodies” so specific to HIV infection that they propose the following definition: “in an infant or child with HIV-1 seroconversion after earlier negative PCR result, postnatal HIV-1 infection was considered possible if seroconversion occurred in the first three months of life and proved if seroconversion occurred after that time” (22). With this definition, the Rwandan, French, and Belgian researchers were able to come to the conclusion that “HIV-1 infection can be transmitted from mothers to infants during the postnatal period. Colostrum and breast milk may be efficient routes for the transmission of HIV-1 from recently infected mothers to their infants” (22). They do not consider the possibility that exposure to external stressor agents could cause the tests to react positively in both mothers and infants. Again, breastfeeding could perfectly well be an epidemiological confounding factor for “HIV transmission.”

The above studies on AIDS and breastfeeding provide excellent examples of the profound crisis in the scientific method that surrounds the entire field of AIDS research.

#### **A possible trial to determine if breastfeeding is a genuine risk factor for AIDS**

The only objective way to confirm the hypothesis of the transmission of HIV/AIDS through breast milk is by searching not only for HIV but also for all other potential risk

factors for testing positively on the tests for HIV and for immunodeficiency, in at least four different groups of people:

a) One group of HIV-positive mothers and their infants living in a variety of African conditions; b) one group of HIV-positive mothers and their infants living in a variety of developed conditions; c) one group of HIV-negative mothers and their infants living in a variety of African conditions; d) one group of HIV-negative mothers and their infants living in a variety of developed conditions.

In each group there must be a significant number of mothers that breastfeed, formulafeed and mixedfeed their babies.

Retrospective trial: Each mother will respond to a questionnaire with questions looking for past voluntary and involuntary exposure to immunological stressor agents.

Prospective trial: All groups should be followed up for several years to attempt to determine if seroconversion to HIV-positive or the development of AIDS is secondary to exposure to immunological stressors. Both mothers and children should be subjected to periodic clinical and laboratory evaluations of their health status.

All conclusions on breastfeeding and AIDS originating from uncontrolled surveys are simply subjective speculations and have nothing to do with science.

Until objectively proven to the contrary, even during the AIDS era breastfeeding is still the best choice!

## References

1. State of New York, Department of Health Memorandum. Maternal-Pediatric HIV Prevention and Care Program: HIV counseling and voluntary testing of pregnant women; routine HIV testing of newborns. AI 99-01. Effective on August 1, 1999.
2. State of Connecticut, Governor John Rowland. Law Public Act No. 99-2. Hospitals' administering tests for HIV infection and/or other HIV related tests to pregnant women and newborn babies. Effective on October 1, 1999.
3. State of New York Department of Health. Public Health Law, Article 21, Title III, Section 2139. HIV/AIDS Testing, Reporting and Confidentiality of HIV-Related Information. Effective June 1<sup>st</sup> 2000.
4. Giraldo RA. Milking the Market. Will mothers, dish out the W.H.O. formula? *Continuum* (London) 1998; 5(4): 8-10.
5. Farber C. HIV and Breastfeeding. The fears. The misconceptions. The Facts. *Mothering Magazine* 1998; No. 90: 66-71.
6. Giraldo RA. AIDS and Stressors: AIDS is neither an Infectious Disease nor is Sexually Transmitted. It is a Toxic-Nutritional Syndrome Caused by the Alarming Worldwide Increment of Immunological Stressor Agents. Medellin, Colombia: Impresos Begon, 1997a: 205.

7. Giraldo RA. Papel de Estresantes Inmunologicos en Inmunodeficiencia. IATREIA (University of Antioquia, School of Medicine, Colombia) 1997b; 10: 62-76.
8. Giraldo RA, et al. Is It Rational To Treat or Prevent AIDS With Toxic Antiretroviral Drugs in Pregnant Women, Infants, Children, and Anybody Else? The Answer is Negative. *Continuum* (London) 1999; 5(6): 38-52.
9. Barre-Sinoussi F, et al Isolation of a T-Lymphotropic Retrovirus from a Patient at Risk for AIDS. *Science* 1983; 220: 868-871.
10. Johnson C. The PCR to Prove HIV Infection. Viral Load and Why They Can't Be Used. *Continuum* (London) 1996b; 4: 33-37 & 39.
11. Papadopulos-Eleopulos E. et al. The Isolation of HIV: Has It Really Been Achieved? The Case Against. *Continuum* (London) 1996; 4(3): S1-S24.
12. Boriskin YS et al. HIV Primers Can Amplify Sequences of Human Satellite DNA. *AIDS* 1994; 8: 709-711.
13. Defer C et al. Multicentre Quality Control of Polymerase Chain Reaction for Detection of HIV DNA. *AIDS* 1992; 6: 659-663.
14. Papadopulos-Eleopulos E. et al. Is a Positive Western blot Proof of HIV Infection? *Bio/Technology* 1993; 11: 696-707.
15. Johnson C. Whose Antibodies Are They Anyway? *Continuum* (London) 1996a; 4(3): 4-5.
16. Papadopulos-Eleopulos E. Reappraisal of AIDS. Is the Oxidation Induced by the Risk Factors the Primare Cause? *Medical Hypothesis* 1988; 25: 151-162.
17. Papadopulos\_Eleopulos E. Looking Back on the Oxidative Stress Theory of AIDS. *Continuum* (London) 1998/9 5(5): 30-35.
18. Frei B. *Natural Antioxidants in Human Health and Disease*. San Diego: Academic Press; 1994: 588.
19. Pryor WA, Godber SS. Oxidative Stress Status: An Introduction. *Free Radicals Bio Med* 1991; 10: 173.
20. Sies H. *Oxidative Stress: Oxidants and Antioxidants*. London: Avademic Press; 1991: 507.
21. Slater TF. Free Radicals: Formation, Detection, Reactivity and ytotoxicity. In: Lachman PJ et al. *Clinical Aspects of Immunology*. Fifth Edition. Boston: Blackwell Scientific Publications. 1993: 377-393.
22. Van de Perre P, et al. Postnatal Transmission of the Human Immunodeficiency Virus Type 1 From Mother to Infant: A Prospective Cohort Study in Kigali, Rwanda. *NEJM* 1991; 325: 593-598.

## **21. APPENDIX F**

# **Antiretrovirals Can Cause AIDS**

By Roberto Giraldo, June 2000

### **1. All antiretroviral drugs are highly toxic to humans.**

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

**1.1.** After more than a decade of treating and trying to prevent AIDS with antiretroviral therapies, neither individual nor public health benefits have been achieved (1,2).

**2.2.** 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 (3). AZT is now licensed by the Food and Drug Administration (FDA) As an anti-HIV medication (1,4,5).

AZT is a potent cytotoxic DNA chain-terminator (1,6,7).

The toxicity of AZT, the drug now prescribed indefinitely to both healthy “HIV-positive” individuals and to AIDS patients, has been solidly documented (1,8-13).

AZT is highly toxic to human cells, including T4 lymphocytes, at the “antiretroviral” dosage recommended by the manufacturer (12).

The immunotoxicity of AZT, as well as its myelotoxicity, are well recognized (14). Granulocytopenia is one of the most common effects seen in persons treated with AZT (15,16)

There are also well documented investigations showing that AZT has carcinogenic properties with respect to rapidly growing human and animal immune and other cells (12). In humans, AZT magnifies the risk of lymphomas by 50 (17). AZT has also been confirmed to be carcinogenic in mice (18-20). Nevertheless, AZT is sold in the United States, where it is illegal to market drugs that are carcinogenic (19,21).

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 (22-26).

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.” They add that: “Myopathy and myositis with pathologic changes similar to that produced by HIV disease, have been associated with prolonged use of Retrovir” (5).

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 (27). This toxicity for embryos has also been documented in animals (28).

The American National Institute of Child Health and Human Development has warned about the toxicity of AZT for children (29). It is recognized that AZT impedes normal child growth and development (29).

AZT can also destroy non-growing cells, such as neurons and muscle cells (26), thus causing muscle atrophy (22,30-34) and dementia (11,25).

It is well known that many illegal acts were committed in pursuit of the 1987 FDA marketing approval of AZT (35).

**1.3.** 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 (36,37).

**1.4.** Currently, the HIV-AIDS supporters are prescribing hydroxyurea, an inexpensive drug used for chemotherapy of leukemia (38). This too is an inhibitor of DNA synthesis.

**1.5.** The toxicity of the new protease inhibitors, prescribed as part of the so-called AIDS treatment “cocktails,” is also well documented (39).

These “cocktails” contain a protease inhibitor in conjunction with two DNA chain-terminators (39).

Researchers are documenting the fact that persons on protease inhibitors can develop abnormal fat accumulations, termed “buffalo humps” and “crixbelly” (40-42).

The hepatotoxicity of protease inhibitors has also been documented (43). Dogs and rats treated with protease inhibitors develop hepatic cell necrosis 30 minutes after administration of the drug (44).

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, cholesterol, and triglycerides; abnormal subcutaneous and visceral fat accumulation; peripheral fat wasting and lipomatosis; pancreatitis and angina (40,41,45-47). Hypertriglyceridemia is being described in 79% of the individuals taking protease inhibitors (48).

It has even been documented that protease inhibitors can induce the development of AIDS-defining diseases such as mycobacterial infections (49).

These protease inhibitor “side effects” are having a chilling effect on “cocktail euphoria” (50).

Thus, scientific evidence demonstrates that antiretroviral drugs are highly toxic to both humans and animals.

## **2. Antiretroviral drugs can themselves cause AIDS.**

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

**2.1.** 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 (51,52).

Currently, protease inhibitors are being prescribed as anti-HIV medications for the lifetime of the individual (53,54).

All of 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 (55). By definition, the immunocompetent cells, as well as the bone marrow cells, are cells that are dividing constantly. A unique characteristic of the cells of the immune system is that they must divide during the immune response (56). 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 (1,52).

The toxic effects of AZT on people's immune systems have been documented (57). 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 developed in 36% of the 11 people 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 (57).

**2.2.** 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 (1,9,10,12,13,58).

The British-French Concorde trial found that AZT was unable to prevent AIDS, and instead increased mortality by 25%, compared to the untreated controls (59).

Another British study found that AZT prophylaxis decreased survival and induced wasting syndrome, cryptosporidiosis, and cytomegalovirus infection (60).

The American MAC study shows that AZT increases the risk of pneumonia, one of the AIDS defining diseases (61).

Studies often show that individuals given AZT have a worse prognosis (6,7), but mainstream researchers prefer to implicate HIV (62).

Lymphocyte counts decreased significantly in humans treated with AZT, but not in the non-treated controls (22,63). Interestingly, these are the very experiments that the Food and Drug Administration evaluated prior to the licensing of AZT (1,6,7,9).

Another study similarly found that AZT users experienced more rapid CD4+ cell depletion (64).

Prophylactic AZT has also been shown to increase significantly the risk of AIDS in hemophiliacs when compared with untreated controls (65).

Since AZT use began, the mortality of British "HIV-positive" hemophiliacs has increased 10-fold (66).

A similar finding has occurred in American hemophiliacs (67). However, most AIDS researchers insist on implicating HIV (66-68).

**2.3.** The immunological alterations secondary to antiretroviral therapy and described in section 1 can be reversed after individuals cease taking these medications. 10 out of 11 individuals recovered their cellular immunity after stopping AZT (69).

Even patients suffering from severe pancytopenia and bone marrow aplasia recover after discontinuing AZT (8).

Clinical manifestations of mycobacterial infection started 1-3 weeks after starting the protease inhibitor Indinavir. Symptoms disappeared after the patients stopped the medication (49).

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 (70,71).

**2.4.** 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" (72).

**2.5.** 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 (73).

In a similar experiment, mice were also treated with AZT for 7 weeks and developed anemia, leukopenia, thrombocytopenia and myelodysplasia (74).

Hamsters treated with AZT for one or two weeks developed T-cell depletion and atrophy of the thymus (75).

Mice treated with the drug for 2 weeks developed anemia, nephrotoxicity, and lymphotoxicity (76).

AZT is also toxic to the liver (77).

The carcinogenic properties of AZT have been documented in animal experiments (75). AZT can stimulate leukemias (74).

**2.6.** In addition to the antiretroviral drugs, healthy people who are "HIV-positive" are taking many prescribed antibiotics, anti-mycobacterials, antifungals, antivirals, and antidepressants, as well as many over-the-counter medications (78,79). All are potentially immunotoxic stressor agents (80), and all contribute to generating AIDS (81).

Proponents of the HIV-AIDS theory constantly propose that HIV is mutating and developing resistance to the current medications. However, there is no scientific substantiation for the assertion that "HIV is mutating" (82).

**2.7.** AIDS patients are also taking a polypharmacy of immunotoxic medications (6,7) 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 metronidazole, pyrimethamine, daraprim, amphotericin B, clotrimazole, dapsone, interferon, pentamidine, vincristine, flucytosine, adriamycin, vinblastine, to mention some of the more frequently used, are potent immunotoxic, myelotoxic, lymphotoxic, nephrotoxic, and hepatotoxic drugs (37,80).

**2.8.** It is unethical, to say the least, to treat or attempt to prevent AIDS with medications known to be highly toxic to the cells of the immune system and the bone marrow, as well as to cells of other tissues and systems. It is the equivalent of mainstream AIDS researchers attempting to fight fire with gasoline.

### **3. 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 far more vulnerable to the toxic effects of antiretroviral drugs":

**3.1.** For decades, medical science has known that growing cells are far more vulnerable to the toxic effects of many different agents (83,84). 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 (85,86).

It is also important to keep in mind that the immune system of a child attains its own maturity only after the age of ten (56).

**3.2.** However, in the era of AIDS, mainstream AIDS researchers are changing all the rules. Currently, toxic medications are recommended and prescribed worldwide to pregnant women and children (87,88). 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 (70).

Babies who test "HIV-negative" but who are born to HIV-positive mothers are nevertheless prescribed AZT for six weeks after birth (70,87,89).



**3.3.** 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 (90,91).

It is as if the vulnerability of newborns and young children to toxic substances has been forgotten (92).

**3.4.** The toxicity of antiretroviral drugs for embryos and fetuses has been documented in humans and animals, as well as *in vitro*.

AZT is a potent cytotoxic DNA chain-terminator (1,6,7) and "it has been well known for many years that the compounds which can alter DNA metabolism often exhibit pronounced prenatal toxicity" (55).

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 (27). In some instances intrauterine growth retardation has been documented (93). The hemoglobin at birth in infants exposed to AZT was found to be significantly lower than in a placebo group (70,94,95).

The American National Institute of Child Health and Human Development is well aware of the toxicity of AZT (29). AZT has been shown to impede normal child growth and development (29).

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 (28).

When administered to pregnant mice, AZT reduced the number of fetuses by 60%, altered the livers of newborns, and caused a significant reduction of hematocrit in the pregnant animals (96). A similar experiment with pregnant mice showed a significant reduction in the number of fetuses (97). These effects are enhanced if mice embryos are preimplanted (98).

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 (99). It inhibited the erythroid colony formation of liver cells from mouse fetuses (77). Additionally, exposure of two-cell mice embryos to zidovudine was consistently associated with significant inhibition of blastocyst formation (100).

**3.5.** A recent comprehensive review of this issue concluded: "Sufficient data regarding the safety of zidovudine in human pregnancy are not available" (55).

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: "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:

“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” (101).

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, in children, or in anyone. The potential cytotoxic, mutagenic, theratogenic, immunotoxic, and carcinogenic properties of these chemicals have been scientifically documented (6,7,88,102-104).

Before the AIDS epidemic, antimicrobials were only prescribed prophylactically for the prevention of a relapse of rheumatic fever. There were no other exceptions. Additionally, antimicrobials, especially antibiotics, were only prescribed for short periods of time, such as a few days for the treatment of an infectious disease. Why are the rules being changed now? Where is the scientific justification that researchers have for changing the rules now?

## 4. Conclusions.

**4.1.** Scientific data presented here demonstrate that it is irrational to treat or prevent AIDS with toxic antiretroviral drugs in any patient. It is contrary to common sense to treat or prevent a highly toxicological syndrome with even more toxicity.

**4.2.** The use of antiretroviral medications to treat or prevent AIDS in pregnant women, infants, children, and anyone else should therefore be stopped immediately.

## REFERENCES

1. DUESBERG PH. AIDS Acquired by Drug Consumption and other Non Contagious Risk Factors. *Pharmac Ther* 1992; 55:201-277.
2. HAND T. Why Antiviral Drugs Cannot Resolve AIDS. *Reappraising AIDS* 1996; 4(9):1-4.
3. HORWITZ JP, CHUA J & NOEL M. Nucleosides. V. The Monomesylates of 1-(2'-Deoxy-Beta-D-Lyxofuranosyl) Thymidine. *J Org Chem* 1964; 29:2076.
4. KOLATA G. Imminent Marketing of AZT Raises Problems; Marrow Suppression Hampers AZT Victims. *Science* 1987; 235: 1462-1463.
5. GLAXO WELLCOME. *Retrovir (Zidovudine)*. In: *Physician's Desk Reference*. Montvale, NJ: Medical Economic Co., 1998: 1167-1175.
6. DUESBERG PH & RASNICK D. The Drug-AIDS Hypothesis. *Continuum (London)* 1997; 4(5): S1-S24.
7. DUESBERG PH & RASNICK D. The AIDS Dilema. *Drug Diseases Blamed on a Passenger Virus*. *Genetica* 1998; 104: 85-132.

8. 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.
9. LAURITSEN J. *Poison by Prescription: The AZT Story*. New York: Asklepios, 1990.
10. LAURITSEN J. AZT: Iatrogenic Genocide. In: *The AIDS War: Propaganda, Profiteering and Genocide from the Medical-Industrial Complex*. New York: Asklepios, 1993: 71-86.
11. SMOTHERS K. Acquired Immunodeficiency Syndrome: Pharmacology and Toxicology. In: GOLDFRANK LR, FLOMENBAUM NE, LEWIN NA, et al. *Goldfrank's Toxicologic Emergencies*. Fifth Edition. Norwalk, Connecticut: Appleton & Lange, 1994: 455-472.
12. 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.
13. ZARETSKY MD. AZT Toxicity and AIDS Prophylaxis: Is AZT Beneficial for HIV + Asymptomatic Persons with 500 or More T4 Cell per Cubic Milimeter ? *Genetica* 1995; 95:91-101.
14. 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.
15. YARCHOAN R, MITSUYA H, MYERS CE, et al. Clinical Pharmacology of 3'-azido-2', 3'-dideoxythymidine (Zidovudine) and Related Dideoxynucleosides. *NEJM* 1989; 321:726-739.
16. KHOO SH & WILKINS EGL. Review: Controversies in Anti-Retroviral Therapy of Adults. *J Antimicrob Chemother* 1995; 35:245-262.
17. 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.
18. COHEN SS. Antiretroviral Therapy for AIDS. *NEJM* 1987; 317:629.
19. YARCHOAN R & BRODER S. Antiretroviral Therapy for AIDS. *NEJM* 1987; 317:630.
20. COHEN J. The Media's Love Affair with AIDS Research: Hope vs. Hype. *Science* 1997; 275:298-299.
21. DUESBERG PH. *Infectious AIDS; Have We Been Misled?* Berkeley, CA: North Athantic Books, 1996: 582.

22. 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.
23. MCLEOD GX & HAMMER SM. Zidovudine: Five Years Later. *Ann Intern Med* 1992; 117:487-501.
24. FREIMEN JP, HELFERT KE, HAMRELL MR, et al. Hepatomegaly with Severe Steatosis in HIV-Seropositive Patients. *AIDS* 1993; 7: 379-385.
25. 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.
26. PARKER WB & CHENG YC. Mitochondrial Toxicity of antiviral Nucleoside Analogs. *The J of NIH Research* 1994; 6:57-61.
27. 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.
28. BOX JL, FEARON ER, HAMILTON SR, et al. Prevalance of Ras Gene Mutations in Human Colorectal Cancers. *Nature (London)* 1987; 327: 293-297.
29. 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.
30. BESSEN LJ, GREENE JB, LOUIE E, et al. Severe Polymyositis-like Syndrome Associated with Zidovudine Therapy of AIDS and ARC. *NEJM* 1988; 318: 708.
31. GORARD DA & GUILODD RJ. Necrotizing Myopathy and Zidovudine. *Lancet* 1988; i:1050.
32. HELBERT M, FLETCHER T, PEDDLE B, et al. Zidovudine-Associated Myopathy. *Lancet* 1988; ii:689-690.
33. DALAKAS MC, ILLA I, PEZESHKPOUR GH, et al. Mitochondrial Myopathy Caused by Long-Term Zidovudine Therapy. *NEJM* 1990; 322:1098-1105.
34. 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.
35. LAURITSEN J. FDA Documents Show Fraud in AZT Trials. *New York Native*, March 30, 1992.
36. 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.

37. 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 Immunodeficiency Syndr* 1994; 7:1057-1063.
38. MAUGH II TH. Researchers on Path to First Inexpensive AIDS Medication. *San Francisco Chronicle*, February 6, 1998: A6.
39. STOLBERG SG. Despite New AIDS Drugs, Many Still Lose the Battle. *The New York Times*, August 22 1997: A1, A6.
40. LIPSKY JJ. Abnormal Fat Accumulation in Patients with HIV-1 Infection. *Lancet* 1998; 351:847-848.
41. MILLER KD, JAMES E, YANOVSKI J, et al. Visceral Abdominal-Fat Accumulation Associated with Use of Indinavir. *Lancet* 1998; 351:871-875.
42. LO JC, MULLIGAN K, TAI VW, et al. "Buffalo Hump" in Men with HIV-1 Infection. *Lancet* 1998; 351:867-870.
43. BRAEU N, LEAF HL, WIECZORCK RL, et al. Severe Hepatitis in Three AIDS Patients Treated with Indinavir. *Lancet* 1997; 349: 924-925.
44. GROSSMAN SJ, REINFOED N, EYDELLOTH RS, et al. Hepatotoxicity of an HIV Protease Inhibitor in Dogs & Rats. *Toxicol. Appl. Pharmacol.* 1997;146: 40-52.
45. HENGEL RC, WARTTS NB & LENNOX JL. Benign Symmetric Lipomatosis Associated with Protease Inhibitors. *Lancet* 1997; 350:1596.
46. 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.
47. JAMES JS. Protease Inhibitors Metabolic Side Effects: Cholesterol, Triglycerides, Blood Sugar, and "Crixbelly". In: *AIDS Treatment News* 1997, Issue No. 273, August 15, 1997.
48. DI PERVI G, DEL BRAVO P & CONCIA E. HIV - Protease Inhibitors. *NEJM* 1998; 339:773-774.
49. RACE EM, ADELSON-MITTY J, KRIEGEL GR, et al. Focal Mycobacterial Lymphadenitis Following Initiation of Protease-inhibitor Therapy in Patients with Advanced HIV-1 Disease. *Lancet* 1998; 351:252-255.
50. ISMACH J. AIDS'98 Protease Impediments: Lipodystrophy and Drug Resistance Take the Edge Off Cocktail Euphoria. *News From the Geneva 12<sup>th</sup> World AIDS Conference. Physician Weekly* 1998; 15(33):1.
51. 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.

52. 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.
53. 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.
54. LEVY JA. Antiviral Therapies. In: HIV and the Pathogenesis of AIDS. Second Edition. Washington DC: ASM Press, 1998b: 339-364.
55. 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: Mechanistic Understanding of Human Developmental Toxicants. Berlin: Springer, 1997: 231-264.
56. MALE D, COOK A, WOEN M, et al. The Immune System. In: Advanced Immunology. London: Mosby, 1996: 1.1.
57. 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.
58. GIRALDO RA, et al. Is It Rational To Treat or Prevent AIDS With Toxic Antiretroviral Drugs in Pregnant Women, Infants, Children, and Anybody Else? The Answer is Negative. *Continuum (London)* 1999; 5(6): 38-52.
59. 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.
60. 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.
61. 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.
62. SABIN CA, PHILLIPS AN & LEE CA. Response: Arguments Contradict the "Foreign Protein-Zidovudine" Hypothesis. *Br Med J* 1996; 312:211-212.
63. 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.

64. 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.
65. 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.
66. 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.
67. 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.
68. MADDOX J. More Conviction on HIV and AIDS. *Nature* 1995; 377:1.
69. SCOLARO M, DURHAM R & PIECZENIK G. Potential Molecular Competitor for HIV. *Lancet* 1991; 337:731-732.
70. CONNOR EM, SPERLING RS, GELBER R, et al. Reduction of Maternal-Infant Transmission of Human Immunodeficiency Virus Type 1 With Zidovudine Treatment. *NEJM* 1994; 331: 1173-1180.
71. 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 Surveillance Case Definition. *Clin Infect Dis* 1997; 25:739-740.
72. MERCK and Company. Crixivan (Indinavir Sulfate): Patient Information About Crixivan. SPIN June, 1997: 111-114.
73. MATSUOKA A, OZAKI M, TAKESHITA K, et al. Aneuploid Induction by Benzo[a]Pyrene and Polyploid Induction by 7,12-Dimethylbenz[a]Antracene in Chinese Hamster Cells V79-MZ and V79. *Mutagenesis* 1997; 12:365-372.
74. LEWIS R. Genetic Imprecision. *BioScience* 1991; 41:288-293.
75. 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.
76. SANDLER L & HECHT F. Genetics Effects of Aneuploidy. *Amer J Hum Genet* 1973; 25:332-339.
77. 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.

78. GREENBLAT RM, HOLLANDER H, Mc MASTER JR, et al. Polypharmacy Among Patients Attending an AIDS Clinic: Utilization of Prescribed, unorthodox, & Investigational Treatments. *Journal of Acquired Immune Deficiency Syndromes* 1991; 4: 136-143.
79. HAYES T, ALTMAN R, AKILI-OBIKA A, et al. HIV-Related Deaths from Selected Infectious Disease Among Persons Without AIDS in New Jersey. *Journal of Acquired Immune Deficiency Syndromes* 1994; 7: 1074-1078.
80. GIRALDO RA. AIDS and Stressors II: A Proposal for the Pathogenesis of AIDS. In: *AIDS and Stressors*. Medellín: Impresos Begón, 1997: 57-96.
81. GIRALDO RA. AIDS and Stressors III: A Proposal for the Natural History of AIDS. In: *AIDS and Stressors*. Medellín: Impresos Begón, 1997: 97-131.
82. MARTINEZ M. The Existence of Human Immunodeficiency Virus Resistance to Nucleoside-Analog Drugs Has not Been Shown. *Med Hypothesis* 1997; 49:235-240.
83. ROGERS J & KAVLOCK R. Developmental Toxicology. In: KLAASSEN CD, AMDUR MO & DOULL J. Casarett and Doull's Toxicology. The Basic Science of Poisons. Fifth Edition. New York: McGraw-Hill, 1996: 301-332.
84. 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.
85. MILLER RK, KELLOG CK & SALTZMAN RA. Reproductive and Perinatal Toxicology. In: HALEY TJ & BERNDT WO. Handbook of Toxicology. Cambridge: Hemisphere Publishing Corporation, 1987: 195-309.
86. NEEDLEMAN HL & BELLINGER D. Prenatal Exposure to Toxicants: Developmental Consequences. Baltimore: Johns Hopkins University Press, 1994: 321.
87. LANCET. Zidovudine for Mother, Fetus, and Child: Hope or Poison? *Lancet* 1994; 344:207-209.
88. PHILPOTT P. AZT for Pregnant HIV+ Women and Their Newborns. *Reappraising AIDS* 1997b; 5(2):1-2.
89. COTTON P. Trial Halted After Drug Cuts Maternal HIV Transmission Rate By Two Thirds *JAMA* 1994; 271: 807.
90. GRUBMAN S, GROSS E, LERNER-WEISS N, et al. Older Children & Adolescents with Perinatally Acquired Human Immunodeficiency Virus Infection. *Pediatrics* 1995; 95: 657-663.
91. LUZURIAGA K, BRYSON Y, KROGSTAND P. et al. Combination Treatment with Zidovudine, Didanosine, and Nevirapine in Infants with Human Immunodeficiency Virus Type 1 Infection. *NEJM* 1997; 336:1343-1349.



92. KACEW S & REASOR MJ. Toxicology and the Newborn. Amsterdam: Elsevier, 1984: 291.
93. 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.
94. ROGERS M & JAFFE HW. Reducing the Risk of Maternal-Infant Transmission of HIV: A Door is Opened. NEJM 1994; 331:1222-1223.
95. BAYER R. Ethical Challenges Posed by Zidovudine Treatment to Reduce Vertical Transmission of HIV. NEJM 1994; 331: 1223-1225.
96. GOGU SR, BECKMAN BS & AGRAWAL KC. Amelioration of Zidovudine-Induced Fetal Toxicity in Pregnant Mice. Antimicrob Agents Chemother 1992; 36:2370-2374.
97. TOLTZIS P, MARX CM, KLEINMAN N, et al. Zidovudine-Associated Embryonic Toxicity in Mice. J Infect Dis 1991; 163:1212-1218.
98. TOLTZIS P, MOURTON T & MAGNUSON T. Effect of Zidovudine on Preimplantation Murine Embryos. Antimicrob Agents Chemother 1993; 37:1610-1613.
99. 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.
100. TOLTZIS P, MOURTON T & MAGNUSON T. Comparative Embryonic Cytotoxicity of Antiretroviral Nucleosides. J Infect Dis 1994; 169:1100-1102.
101. SANDBERG JA & SLIKKER JR W. Developmental Pharmacology and Toxicology of Anti-HIV Therapeutic Agents: Dideoxynucleosides. FASEB J 1995; 9:1157-1163.
102. FARBER C. AZT on Trial. The Treatment for AIDS Accused of Being Deadlier than the Disease itself. Spin July 1996.
103. FARBER C. AZT Roulette: The Impossible Choices Facing HIV-Positive Women. Mothering 1998b Sept/Oct No. 90: 53-65.
104. DUESBERG PH. HIV, AIDS & Zidovudine. Lancet 1992b; 339: 805-806.

\*\*\*\*\*