

## **AIDS in Africa: distinguishing fact and fiction**

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Review

E. Papadopulos-Eleopulos (1) Valendar F. Turner (2) John M. Papadimitriou (3) Harvey Bialy (4)

(1) Corresponding author, Department of Medical Physics, The Royal Perth Hospital, Perth 6000 Western Australia; (2) Department of Emergency Medicine, Royal Perth Hospital; (3) Department of Pathology, University of Western Australia; (4) Bio/Technology 65 Bleecker St. New York, NY 10012 USA.

**The data widely purporting to show the existence and heterosexual transmission in Africa of a new syndrome caused by a retrovirus which induces immune deficiency is critically evaluated. It is concluded that both acquired immune deficiency (AID) and the symptoms and diseases which constitute the clinical syndrome (S) are long standing in Africa, affect both sexes equally and are caused by factors other than HIV. The presence of positive HIV serology in Africans represents no more than cross-reactivity caused by an abundance of antibodies induced by the numerous infectious and parasitic diseases which are endemic in Africa, that is, a positive HIV antibody test does not prove HIV infection. Given the above, one would expect to find a high prevalence of "AIDS" and "HIV" antibodies in Africa. This is not proof of heterosexual transmission of either HIV or AIDS.**

### **Introduction**

Following the appearance in the West in the early 1980s of AIDS in gay men, many European and American researchers looked for AIDS in Africa. There were three reasons for this. One was Dr. Robert Gallo's hypothesis that AIDS is caused by a retrovirus HTLV-I, or a similar virus. (At the time it was known that Africans had a high prevalence of positive HTLV-I serology). The other reasons were the high prevalence of Kaposi's sarcoma (KS) in Africa, and the diagnosis of "AIDS" in a small number of patients of African origin who were living in Europe. Yet, there were so many problems with the HTLV-I theory of AIDS that by 1984 it had been abandoned, even by Gallo himself, and although KS was practically non-existent in gay men before the AIDS era, KS has been present in Africa since antiquity. Its characteristic clinical appearances are described in the Ebers papyrus which dates from 1600 B.C. As for the AIDS cases described by Belgian doctors in the patients of African descent, the doctors who reported these cases did not exclude the possibility that AIDS has always been present in Africa (Clumeck et al., 1984). Despite these facts, the claim that the cause of AIDS everywhere, including Africa is HIV, has been overwhelmingly accepted. In fact, AIDS in Africa became of such pivotal significance to the HIV/AIDS theory that in 1990 nearly 600 "AIDS-related" studies were conducted in Africa. Yet even up to 1994, "There have been few studies of the impact of HIV-1 infection on mortality in Africa, and none for a general rural population" (Mulder et al., 1994). In this widely publicized report, which appeared in the *Lancet*, Mulder and his colleagues tested blood samples from Ugandan rural subsistence farmers for "HIV -1 antibodies at the Uganda Virus Research Institute". Of 9389 individuals tested, 4.8% were found to be positive. "Deaths

were ascertained over 2 years" and 198 were recorded. Of these 109 were in seronegative individuals and 89 in seropositive individuals. Of the latter, 73 were adults. In a commentary accompanying publication of this study, researchers from the CDC wrote: "An ironic feature of this work is that it does not require a belief that HIV is the cause of AIDS. Rather, the study shows that the simple finding of antibodies against HIV in an individual's serum predicts a likelihood of death within the next several years far above that for a seronegative individual. Although most reasonable observers do accept that HIV causes AIDS, even sceptics cannot fail to acknowledge the high prevalence of antibody to HIV in Africa. If there are any left who will not even accept that antibody to HIV indicates infection with the virus, their explanation of how HIV seropositivity leads to early death must be curious indeed" (Dondero & Curran, 1994). In the following we present just such an alternative explanation of this, and other published studies on the "epidemic" of AIDS in sub-Saharan Africa. We leave it to the reader to judge exactly how curious it is.

### **Acquired Immune Deficiency (AID)**

AIDS researchers in Africa, including those from the CDC and WHO, admit that immune deficiency in Africa has existed for a considerable time and this has not been due to HIV. "Tuberculosis, protein calorie malnutrition, and various parasitic diseases can all be associated with depression of cellular immunity" (Piot et al., 1984). "A wide range of prevalent [in Africa] protozoal and helminthic infections have been reported to induce immunodeficiency" (Clumeck et al., 1984). "...among healthy Africans resident in a non-AIDS area, the numbers of helper and suppressor lymphocytes were the same in HTLV-III/LAV seropositive and seronegative subjects..." (Biggar, 1986). "Africans are frequently exposed, due to hygienic conditions and other factors, to a wide variety of viruses, including CMV, EBV, hepatitis B virus, and HSV, all of which are known to modulate the immune system...Furthermore, the Africans in the present study are at an additional risk for immunologic alterations since they are frequently afflicted with a wide variety of diseases, such as malaria, trypanosomiasis, and filariasis, that are also known to have a major effect on the immune system" [CMV=cytomegalovirus; EBV=Epstein-Barr virus; HSV=herpes simplex virus] (Quinn et al., 1987).

### **The Syndrome (S)**

If AIDS in Africa is the same condition with the same cause as anywhere else in the world then AIDS in Africa and AIDS in the West should be identical. This is not the case and what is called AIDS in Africa is almost unrecognizably different from AIDS in the West, so much so that if African patients suddenly switched continents, very few Africans would remain AIDS cases. This is due to the existence of multiple AIDS definitions, one for Africa (the same for adults and children), one for adults in North America, Europe and Australia, one for children in these countries and one for Latin America. Unlike the AIDS definition in the West, the WHO Bangui definition for Africa does not require immunological (T4 lymphocyte cell or antibody) tests or a specific disease diagnosis but consists largely of symptoms such as weight loss, diarrhoea, cough and fever. For example, an African with diarrhoea, fever and persistent cough for longer than one month is, by definition, an AIDS case. However, the symptoms listed in the Bangui definition (WHO, 1986) are common and non-specific manifestations of many diseases which are endemic in Africa and were so long before the AIDS era. This is accepted by some of the best known AIDS researchers including

those from Belgium, the WHO and the CDC. According to Jonathan Mann, former director of the WHO Global AIDS program, and his colleagues, "...recognition of pediatric AIDS is particularly difficult in Kinshasha [Zaire], since many children have severe infant and childhood diseases with similar manifestations (eg, weight loss, chronic diarrhoea)" (Mann et al., 1986). Anthony Fauci, Director of the National Institute of Allergy and Infectious Diseases in the United States, discussing the AIDS definition in Africa states: "Well, of course it will be less reliable (than that used in non-Third-World countries). One typical example is what we call 'slim disease'. It's a wasting syndrome seen in Africa. Now that wouldn't fall under any categorization of AIDS by the standard empiric definition, but nevertheless, (slim disease) is being considered AIDS in Africa" (AIDS Alert, January 1987). According to Myron Essex on who's work speculations as to the African origin of HIV is mainly based, "malnutrition and general lack of medical services contributed to diarrhoea, tuberculosis, and other common African diseases that signify AIDS" (New Scientist, 18<sup>th</sup> February 1988). In summary, although, the best known researchers of African AIDS clearly accept that both AID and the AID syndrome (S) existed in Africa long before the AIDS era and that they were caused by agents other than HIV, the same researchers expect the world to accept that in Africa there is a new disease, AIDS, caused by a new virus, HIV.

### **Antibody Tests for HIV**

The evidence for the existence of HIV in Africa is based on the random testing of Africans for the presence of HIV antibodies. The HIV antibody tests rely on the presence or absence of reactions between antibodies present in patients' blood and certain proteins which are believed to be unique to HIV. Even if the proteins are proven to unique to HIV, which at present is not the case, a positive test cannot be considered proof of HIV infection. This is because non-HIV antibodies can (and do, see below) react with the "HIV proteins" producing positive tests in individuals who are not HIV infected. Because of this, before the test is used to diagnose HIV infection the test's specificity must be determined, one must determine how often false-positive tests occur. For this one must:

- 1. Test a large number of patients, both AIDS and non-AIDS patients.
- 2. Simultaneously perform tests for true HIV isolation.
- 3. Compare the antibody test results with the results of HIV isolation, that is, use HIV as a gold standard for the antibody test.

A thorough search of the HIV antibody test literature fails to show a single instance of the use of the above, the only scientifically valid method of determining the specificity of the HIV (or any) antibody tests. Indeed, comparisons between the published work on retrovirology and the presently worldwide data on HIV reveals that no researcher has yet met the requirements for an HIV gold standard. This is because the phenomena collectively inferred as HIV (reverse transcriptase, virus-like particles, "HIV antigens", and "HIV PCR"), are all non-specific (Papadopoulos-Eleopoulos et al., 1993a, 1993b). The lack of a gold standard has already been adduced by one of the best known HIV/AIDS researchers, William Blattner: "One difficulty in assessing the specificity and sensitivity of retrovirus assays is the absence of a final 'gold standard'. In the absence of gold standards for both HTLV-I and HIV-1, the true sensitivity and specificity for the detection of viral antibodies remain imprecise" (Blattner, 1989). For some unknown reason, HIV experts (such as Mulder) determine the specificity of the

HIV antibody tests by repeating an antibody test or a combination of antibody tests an arbitrary number of times and use another antibody test as a gold standard. This was the method used by Burke and his colleagues and many HIV/AIDS experts, including David Ascher, believe this data shows the false-positive rate of the HIV antibody tests to be 1/1,000,000 (Weiss & Thier, 1988; Ascher & Roberts, 1993). According to Mulder and colleagues, their "HIV testing algorithm had a sensitivity and specificity of close to 100%". Mulder's algorithm (Nunn et al., 1993) is a far less substantial version of Burke's algorithm and, like Burke's, uses the Western blot as a gold standard. For them, the true serostatus depends on repeating two different ELISAs until they are concordant, an outcome which could eventuate by making the same mistake twice. A positive ELISA followed by a positive WB is also regarded as proof of HIV infection. However, it is not possible to determine the specificity of an HIV antibody test by repeating the test, or using combinations of the same and other antibody tests as Burke and Mulder and many others have done. According to Philip Mortimer, director of the Virus Reference Laboratory of the Public Health Laboratory Service, London, UK: "Diagnosis of HIV infection is based almost entirely on detection of antibodies to HIV, but there can be misleading cross-reactions between HIV -1 antigens and antibodies formed against other antigens, and these may lead to false-positive reactions. Thus, it may be impossible to relate an antibody response specifically to HIV-1 infection. In the presence of clinical and/or epidemiological features of HIV -1 infection there is often little doubt, but anti-HIV-1 may still be due to infection with related retroviruses (e.g. HIV -2) which, though also associated with AIDS, are different viruses" [italics ours] (Mortimer, 1989). Although Mortimer et al. (1985) as well as Gallo and his colleagues (Weiss et al., 1985) used clinical and/or epidemiological features" to determine the specificity of the HIV antibody tests, this is scientifically invalid. The use of clinical and/or epidemiological features is not a gold standard for the presence or absence of a retrovirus and use of such a scheme creates many problems. For example, because the vast majority of positive tests occur in individuals who are asymptomatic, the vast majority of positive tests must be construed as false-positives. Mulder et al had 377 individuals with a positive test. Of these, 89 died within 2 years. Although not stated, we can assume that many of the remaining HIV positive cases were asymptomatic and thus, according to Mortimer, all these individuals had false-positive tests. Of the 73 adults who died, only 5 had "AIDS"! the other 68 died of unlisted causes and if asymptomatic for "AIDS" must all be regarded as false-positive results.

Epidemiological data show that AIDS patients in general and Africans including healthy Africans have high levels of antibodies. For example, United States data indicates that serum IgG levels are higher in HIV+ American Blacks (mean 2234 ± 930 mg/dl) than in HIV+ Caucasians (mean 1601 ± 520 mg/dl). Serum IgG levels are also higher in Black blood donors (mean 1356 ± 220 mg/dl) than in Caucasians (mean 1072 ± 243 mg/dl) (Lucey et al., 1991). Thus, in these individuals with high level of antibodies one must expect cross-reactions with HIV antigens to be the rule rather than the exception. That this is the case is amply demonstrated by the African evidence and in fact is accepted by the best known expert on African HIV/AIDS. In 1986, Quinn, Mann, Curran and Piot wrote, in Africa "...serodiagnosis is complicated by the need for confirmatory testing because of the presence of possible cross-reacting antibodies" (Quinn et al., 1986). One year earlier Biggar stated that "...reactivity in both ELISA and Western blot analysis may be non-specific in Africans...the cause of the non-specificity needs to be clarified in order to determine how they might affect the seroepidemiology of retroviruses in areas other than Africa, such as the Caribbean and Japan...Serological studies from Africa

would need to be re-evaluated with a more specific test before conclusions can be drawn" (Biggar et al., 1985). Other eminent HIV/AIDS researchers including Weiss accepted that African sera "may give a false-positive result on direct binding assay systems, or on western blots" (Serwadda et al., 1985). Not only are positive HIV antibody tests in Africa considered proof of HIV infection, without any re-evaluation the criteria used for a positive test are far less stringent than those used in the West. However, this year no less a person than Myron Essex and his colleagues presented unambiguous evidence that both ELISA and WB may not be specific in Africa. Essex and his colleagues reported that "...leprosy patients and their contacts show an unexpectedly high rate of false-positive reactivity of HIV-1 proteins on both WB and ELISA". The cross-reactivity was found to be caused by antibodies directed against two major carbohydrate-containing *M. leprae* antigens--phenolic glycolipid I and especially lipoarabinomannan, an arabinose-containing lipopolysaccharide which is also present in *M. tuberculosis* and other mycobacteria. They warned, "ELISA and WB may not be sufficient for HIV diagnosis in AIDS-endemic areas of Central Africa where the prevalence of mycobacterial diseases is quite high" (Kashala et al., 1994). Cross-reactivity of antibodies to mannans with "HIV proteins" was also reported by Muller and colleagues who found, "Polyclonal antibodies to mannan from yeast also recognize the carbohydrate structure of gp120 of the AIDS virus" (Muller et al., 1990). Others have "shown that normal human serum contains antibodies capable of recognizing the carbohydrate moiety of the HIV envelope glycoproteins", gp41, gp120 and gp160 (Tomiyama et al., 1991). In 1986, Mann and colleagues reported that in a tuberculosis sanatorium in Kinshasa, Zaire, half of the suspected pulmonary cases, one third of the confirmed cases and two thirds of the confirmed extra-pulmonary cases had a positive HIV Western blot antibody test (Nzilambi et al., 1986). Tuberculosis (TB), the cause of which is a mycobacterium, is endemic in Africa. Of the 661 million people in sub-Saharan Africa, 2-3 million have active TB with an annual mortality of 790,000. Despite this and the fact that in adults, "HIV infection" usually follows TB infection, TB has now become an AIDS defining illness, indeed 30-50% of African "AIDS" deaths are from TB. It is of great significance that although neither the Mulder paper nor the commentary on it elaborated on the causes of death in the 5 "AIDS" cases, the authors of the latter wrote, "More information is needed to clarify how many of the excess deaths could have been delayed through optimum medical prevention and therapy of such HIV-associated illnesses as tuberculosis, other pneumonias, and diarrhoeal disease". However, since:

- 1. Tuberculosis has existed in Africa for many generations;
- 2. According to Essex, in patients with tuberculosis, positive HIV antibody tests may be false-positives; the most one can conclude from the African antibody tests is that the finding of a positive test indicates an underlying abnormal propensity to develop a number of illnesses, some of which may prove fatal. A positive "HIV antibody" test is no more than a non-specific marker for this proclivity.

Thus, those "who will not even accept that antibody to HIV indicates infection with the virus" have no need to postulate a "curious" or even a novel explanation for the relationship between "a positive HIV antibody test" and AIDS, or between positive HIV serology and mortality. In fact, Mulder's data does nothing more than prove their predictions (Papadopoulos-Eleopoulos et al., 1993a). Indeed, non-specific antigen/antibody reactions are frequently exploited in clinical practice. For example:

- 1. Antibodies to an extract of ox heart (cardiolipin) predict the development of syphilis, including death from syphilis, but such patients are not infected with ox heart and ox heart is not the cause of syphilis.
- 2. Patients with infectious mononucleosis develop antibodies that react with sheep and horse red blood cells. However, neither sheep nor horse red blood cells are not present in these patients and they are not the cause of the disorder.

## **HIV and AIDS**

In 1984, in the first ever published paper describing HIV antibody testing of Africans, Montagnier and his colleagues wrote, "The prediction that a single infectious agent is at the origin of AIDS implies that all those with proven AIDS show signs of infection" (Brun-Vezinet et al., 1984). The presence of HIV in all AIDS patients is a necessary condition but is not sufficient proof that the virus is the cause of AIDS. Correlation is not proof of causation (Duesberg, 1989). Ninety eight percent of haemophiliacs with AIDS test positive for the presence of hepatitis B virus (Brenner et al., 1991), in fact hepatitis B virus (HBV) seropositivity is a predictor for HIV seropositivity, but no one claims that HBV is the cause of AIDS. No such degree of correlation exists between AIDS and HIV seropositivity in Africa. In one study, 83% of patients with suspected AIDS were HIV positive, but so were 44% with malaria, 97% with herpes zoster, 43% with pneumonia, 67% with amoebic dysentery and 41% with carcinoma. In the other study, 42% of women with recurrent abortions, 67% with vaginal ulcerations and 33% with haemorrhoids had a positive HIV antibody test. While the Bangui AIDS definition had a positive predictive value for HIV seropositivity of 62% in one of the studies and 83% in the other, in the same studies the positive predictive value of amenorrhoea was 42% and 89% respectively (Widy-Wirski et al., 1988; Strecker et al., 1993).

One of the principal major signs of the Bangui definition is loss of body weight. However, in a study of Rwandan women over a 24 months period beginning in 1988, it was reported that nutritional status assessed by loss of body weight "was a significant predictor of eventual HIV seroconversion. Subsequent seroconvertors lost an average of 1.5 kg during the six months of the study compared with 1.0-Kg gain ( $p=0.001$ ) for nonconvertors. Nine of 27 (33%) seroconvertors, compared with one of 44 (2%) controls, lost at least 5Kg in the 6-month period beginning 1 year before their seroconversion...In addition to those findings for measured weight loss during follow-up, reported weight loss before enrolment was also a risk factor for subsequent seroconversion" (Moore et al., 1993). In other words, this study found that weight loss preceded HIV seroconversion by many months or even years. According to Myron Essex, "The more medical scientists research the AIDS epidemic in Africa, the more confusing the picture becomes...Among 37 people in Ivory Coast, West Africa, with symptoms of AIDS, as defined by the World Health Organization, 13 [35%] did not appear to have antibodies to HIV-1 or HIV-2, the second AIDS virus discovered more recently. A similar study in Sengal uncovered 16 of 44 [36%] patients with AIDS, who again showed no sign of infection with either virus" (New Scientist 18/2/88 page 27). Thus the HIV hypothesis of AIDS does not satisfy eventhe most fundamental criterion for proof of an aetiologic agent. More extensive, and thoroughly referenced critiques of its numerous deficiencies can be found in (Duesberg, 1992; Papadopulos-Eleopulos et al., 1992a, 1992b, 1993a, 1993b, 1994).

## **Heterosexual Transmission**

"To evaluate acquired immunodeficiency syndrome (AIDS) in central Africa a prospective study was done in Kigali, Rwanda, where Kaposi's sarcoma (KS) is endemic". The study was conducted by researchers from Belgium, the Netherlands and Rwanda (Van De Perre et al., 1984). In 1983, these workers "sent a questionnaire to all clinicians at the Centre Hospitalier de Kigali asking them to make a special note over a 4 week period of new patients who had clinical evidence of opportunistic infection (OI) and/or generalised or multifocal Kaposi's sarcoma (KS) or who had the AIDS prodrome. The prodrome [patients with the prodrome were ultimately classified as AIDS patients] was defined by at least two of the following: loss of more than 10% body weight, diarrhoea for at least 2 months with no pathogen isolated, chronic fever of unknown origin lasting for at least 2 months, and generalised lymphadenopathy consisting of palpable lymph nodes larger than 1 cm at two or more extrainguinal sites for more than 3 months...Subsequently, immunological evaluations were done in Kigali, after which we retained as having AIDS or probable AIDS patients presenting with the above clinical features provided they also had a decreased ratio of helper/inducer to suppressor/cytotoxic T cells", that is a decreased T4/T8 ratio. They found 26 such patients (17 males and 9 females), two of which were children. "The 24 adult patients denied bisexuality or homosexuality or intravenous drug use". Discussing their findings the authors wrote "The study confirms that AIDS exists in Rwanda, a central African country east of Zaire. The detection of 26 AIDS patients in a short period supports that AIDS may be a public health problem in central Africa...Characteristically, African AIDS affects women as well as men, a pattern very different from the sex ratio (15:1) described in the chronic form of KS that has for many years been seen in central Africa...The low sex ratio suggests that heterosexual contact in the most frequent mode of transmission in central Africa".

In the same year and month, researchers from Belgium, Zaire, and the USA including the CDC, searched for AIDS in Zaire. The authors stated that "Because of limited diagnostic facilities we used a case definition which included clinical features of AIDS and the immunological characteristics of low T helper cell counts and low helper to suppressor ratios which have been hallmarks of AIDS. We believe that this combination strengthens the case definition in an area where severe infectious diseases abound, often going undiagnosed". During a three week period they identified 38 such patients. Ten patients had "Chronic mucocutaneous HSV [herpes simplex virus] infection", 14 bilateral interstitial pneumonia "with severe dyspnoea, unresponsive to antibiotics or tuberculostatics", 31 oral and/or oesophageal candidiasis and 6 had disseminated KS. Regarding the latter they wrote "Since KS has long been endemic in Zaire, only patients with fulminant KS were included". Discussing their findings they wrote: "Two important differences between AIDS in Zaire and the disease in patients of European or American origin merit discussion-- namely, the sex distribution and apparent lack of risk factors among patients in Zaire...The essentially equal proportions of males and females would require that transmission occurs both male to female and female to male, since one-direction transmission would soon result in an imbalance in the ratio" (Piot et al., 1984).

In 1984, sera from 37 out of the 38 patients who were diagnosed in Kinshasha in October 1984 were tested for HIV antibodies by Montagnier and 19 of his associates including researchers from the CDC. The sera were tested by ELISA and followed by a RIPA (radioimmunoprecipitation assay, similar to the Western blot). The latter was considered positive if a p24 band was present. The p41 band and also a 84-kD band

were not considered diagnostic because "The 43-kD [p41] band and the 84-kD band are cellular contaminants that are immunoprecipitated in all the tested sera", from both patients and controls. (Yet today, in Africa, the p41 band on its own is considered to represent a positive WB and thus proof of HIV infection). Thirty two (88%) patients were positive by both tests. So were six out of 26 (23%) controls. (Brun-Vezinet et al., 1984). However: (a) with the exception of a few other reports from Africa (see below) no such correlation between ELISA and WB has ever been reported. For example, Burke and his colleagues tested 1.2 million healthy American military recruits and found that of 6000 individuals with two consecutive positive ELISAs, only 2000 subsequently had a positive Western blot (Burke et al., 1988). In Russia, in 1991, of 30,000 positive screening ELISAs, only 66 were Western blot positive (Voevodin, 1992); (b) since 1987, nobody in the world with the possible exception of Montagnier, considers the p24 band proof of HIV infection, not even in Africa.

In July 1984, the research groups who reported the first 38 cases of AIDS from Kinshasa started a new study in the same city. During an 8 month period they had "565 suspected AIDS cases", that is, they had 565 cases which satisfied "At least one of the following three clinical criteria: (a) A syndrome with profound weight loss (> 10% of normal body weight) plus either chronic diarrhoea (lasting at least 2 mo) or chronic fever and asthenia (lasting 1 + mo); (b) An opportunistic infection included in the Centers for Disease Control definition of AIDS (restricted resources limited recognized opportunistic infections to candidal esophagitis, cryptococcal meningitis and chronic ulcerated herpes infection; and/or (c) Disseminated Kaposi's sarcoma, with histopathologic evidence of visceral invasion". Of the 565 patients, 332 (58.8%) were found to have a positive HIV antibody test, and because of this were considered to be confirmed AIDS cases. "A specimen was considered positive for antibody to HTLV-III if it was repeatedly reactive on two separate ELISA assays ...The male-female ratio was 1:1.1. Men with AIDS were significantly older than women... Nearly half of all patients (145) were not married... Women with AIDS more likely than men with AIDS to be unmarried". Commenting on their results the authors stated:

"Several epidemiologic features of AIDS in Kinshasa should be noted. A nearly equal sex distribution of cases has now been demonstrated in this large series. This age distribution by sex, including a lower mean age for female patients, is typical of sexually transmitted diseases. However, interpreting surveillance data on possible means of exposure to AIDS is difficult. For example, the finding that 61% of women with AIDS are unmarried has been cited to support theories of heterosexual transmission. However, 61% of nearly 933 women working at Mama Yemo Hospital are also unmarried" (Mann et al., 1986).

Like Montagnier and the CDC, Gallo and his associates also tested Africans for HIV antibodies. Of 53 patients with AIDS, including the first 26 patients reported from Rwanda, "46 (87%) tested positive...67 (80%) of 84 prostitutes [without any clinical symptoms] and five (12.5%) of 40 and eight (15.5%) of 51 healthy controls and blood donors, respectively", also tested positive. "All blood donors were of good socioeconomic status". Sera which had one positive ELISA were considered as proof for HIV infection. Sera which had a borderline ELISA were further tested with the WB. In the WB, "serum samples possessing reactivity to HTLV-III p41 and/or p24 were scored positive. Gallo and his associates concluded, "In Central Africa, as previously noted, the occurrence of the syndrome in young to middle-aged men and women

suggests that heterosexual contact is probably the predominant mode of transmission of the AIDS agent. Furthermore, among the 24 adults with AIDS that we saw in Rwanda, 12 of the 17 men had contact with prostitutes, and three of seven women were prostitutes" (Clumeck et al., 1985). The claims in the above studies that: (a) Africans have AIDS; (b) In Africa "Homosexuality, intravenous drug use and blood transfusions did not appear to be risk factors"; (c) an approximately equal number of male and females have AIDS, as well as a positive HIV antibody test; are interpreted as proof that in Africa, HIV and AIDS is heterosexually transmitted. Indeed, the perceived heterosexual spread of AIDS in Africa underlies the belief that HIV and AIDS will eventually overtake the West. But, "The mere absence of data to the contrary does not by itself make the opposite assertion true; if it did, science would be a much simpler thing. While it is true that in Africa the incidence of AIDS and infection with [HIV] is nearly equal among men and women, we ought not automatically assume that heterosexual transmission of the AIDS virus is likely here...parasitic disease has been found repeatedly to be a risk factor for seropositivity to the AIDS virus or AIDS itself in Africa and Venezuela" (Pearce, 1986).

Nancy Padian and her colleagues who to date have most thoroughly investigated heterosexual transmission of HIV/AIDS wrote: "We question whether the ratio of male-to-female cases in Africa necessarily supports the hypothesis that AIDS is primarily spread in Africa by bidirectional heterosexual transmission" (Padian & Pickering, 1986). The fact that equal numbers of men and women have AIDS or antibodies to HIV does not prove that AIDS is heterosexually spread. Many diseases such as influenza, pneumonia, tuberculosis and appendicitis have an equal sex distribution but this is not construed as proof of heterosexual transmission. To prove that AIDS is spread by sexual activity one must study a large number of index cases, isolate HIV, prove it is the cause of AIDS, trace the sexual contacts of these cases and then isolate the same agent. To date, no reliable data of this type has ever been presented either in Africa, or anywhere else. In fact, according to Dr. Harry Haverkos from the US National Institute on Drug Abuse, "Sexual contact tracing, the standard practice in public health to combat such sexually transmitted diseases as gonorrhoea and syphilis, has been avoided for tracing of HIV-infected persons. Health department personnel are concerned about possible discrimination associated with AIDS, plus the fact that there is no cure for the disease" (Haverkos & Edelman, 1988). As far as Africa is concerned, one must note that "AIDS patients reported to the CDC are classified as HT [heterosexual] if they (1) report heterosexual contact with a person with HIV infection or at increased risk for HIV infection (US-born) or (2) were born in countries where HT is a major route of transmission (non-US born)" (Chamberland et al., 1988). This means that a man/woman born in Africa can be said to have acquired AIDS by heterosexual contact even if his/her partner were not proven to have "HIV infection", or even if he/she never had sexual intercourse. Given the fact that the best known HIV/AIDS experts on African AIDS admit that (a) what is known as AIDS in Africa has been present for centuries and was equally common in men and women; (b) a positive HIV antibody test may not be due to HIV antibodies but to the presence of antibodies formed in response to malaria, tuberculosis, leprosy and many parasitic diseases; one would predict that in Africa an equal number of men and women will have "AIDS" and positive antibody tests. To explain these observations one has no need to invoke the activity of a virus called HIV. In fact, the theory that AIDS in Africa is transmitted heterosexually creates more problems than it solves.

In 1986, Gallo and his colleagues wrote, "We found no evidence that other [than receptive anal intercourse] forms of sexual activity, contribute to the risk" of HIV seroconversion (Stevens et al., 1986). In the West, the largest (thousands of cases) and most judiciously conducted prospective epidemiological studies such as the Multicenter AIDS Cohort Study (Kingsley et al., 1987) have proven beyond all reasonable doubt that in gay men the only significant sexual act related to becoming HIV antibody positive and progressing to AIDS is receptive anal intercourse. A minority of the studies also report cases which suggest transmission by passive orogenital sexual activity (Caceres & van Griensven, 1994). Similarly, the largest and best conducted studies in heterosexuals including the European Study Group (1989) have also shown that for women, the only practice leading to an increased risk of becoming HIV antibody positive is anal intercourse. Therefore, in non-African countries the only risk factor for the acquisition of HIV antibodies is anal intercourse in the passive partner (male or female), and if the only cause for the development of HIV antibodies is HIV infection then one must conclude that in non-African countries HIV is unidirectionally sexually transmitted. Thus, at least in non-African countries "HIV", like pregnancy, can only be acquired by the passive sexual partner and cannot be transmitted to the active partner. The unidirectional transmission of "HIV" observed in the West is further supported by Nancy Padian's prospective study of heterosexual couples where, from a cohort recruited from 1985 to March 1991 involving 72 male partners of HIV infected women, there was "one probable instance" of female-to-male transmission (Padian et al., 1991). In the whole history of Medicine there has never been an example of a sexually transmitted disease which is spread unidirectionally, and certainly not one that is spread unidirectionally in one country and bidirectionally in another. Indeed, given this and the other differences between AIDS in the West and Africa it is necessary to postulate that HIV must indeed possess features even more unique than those already attributed to it. Since the only sexual behaviour risk factor for a gay man is receptive anal intercourse, an exclusively active male partner is at no risk of infection by his passive male partner. Yet if this same person travelled to Africa and changed his sexual orientation, he would now be at risk of infection by his passive female partner. Thus, HIV must be able to distinguish an individual's sexual preference, gender and country of residence.

More rationally, one might choose to agree with those African physicians and scientists including Richard and Rosalind Chirimuuta (Chirimuuta & Chirimuuta, 1987) who believe that immunosuppression and certain symptoms and diseases which constitute African AIDS have existed in Africa since time immemorial. According to Professor P.A.K. Addy, Head of Clinical Microbiology at the University of Science and Technology in Kumasi, Ghana "Europeans and Americans came to Africa with prejudiced minds, so they are seeing what they wanted to see...I've known for a long time that Aids is not a crisis in Africa as the world is being made to understand. But in Africa it is very difficult to stick your neck out and say certain things. The West came out with those frightening statistics on Aids in Africa because it was unaware of certain social and clinical conditions. In most of Africa, infectious diseases, particularly parasitic infections, are common. And there are other conditions that can easily compromise or affect one's immune system" (Hodgkinson, 1994). In the words of Dr. Konotey-Ahulu from the Cromwell Hospital in London, "Today, because of AIDS, it seems that Africans are not allowed to die from these conditions [from which they used to die before the AIDS era] any longer. ...Why do the world's media appear to have conspired with some scientists to become so gratuitously extravagant with the untruth?" (Konotey-Ahulu, 1987)

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## **References**

Ascher, D.P. & Roberts, C. 1993 Determination of the etiology of seroreversals in HIV testing by antibody fingerprinting. *Journal of Acquired Immune Deficiency Syndromes* 6, 241-244.

Biggar, R.J. 1986 The AIDS problem in Africa. *Lancet* I, 79-83.

Biggar, R.J. Gigase, P.L. Melbye, M. Kestens, L. Sarin, P.S. Demedts, P. Delacollette, C. Bodner, A.J. Paluku, L. Stevens, W.J. & Battner, W.A. 1985 Elisa HTLV retrovirus antibody reactivity associated with malaria and immune complexes in healthy Africans. *Lancet* II, 520-523.

Blattner, W.A. 1989 Retroviruses. in *Viral infections of humans*, 3<sup>rd</sup> Edition, ed Evans, A.S. pp. 545-592. Plenum Medical Book Company: New York.

Brenner, B. Schwartz, S. Ben-Porath, E. Tatarsky, I. Varon, D. & Martinowitz, U. 1991 The prevalence and interaction of human immunodeficiency virus and hepatitis B infections in Israeli hemophiliacs. *Israel Journal of Medical Sciences* 27, 557-561.  
Brun-Vezinet, F. Rouzioux, C. Montagnier, L. Chamaret, S.

Gruest, J. Barre-Sinoussi, F. Geroldi, D. Chermann, J.C. McCormick, J. Mitchell, S. Piot, P. Taelman, H. Mirlangu, K.B. Wobin, O. Mbendi, N. Mazebo, P. Kalambayi, K. Bridts, C. Desmyter, J. Feinsod, F.M. & Quinn, T.C. 1984 Prevalence of antibodies to lymphadenopathy-associated retrovirus in African patients with AIDS. *Science* 226, 453-456.

Burke, D.S. Brundage, J.F. Redfield, R.R. Damato, J.J. Schable, C.A. Putman, P. Visintine, R. & Kim, H.J. 1988 Measurement of the false positive rate in a screening program for human immunodeficiency virus infections. *New England Journal of Medicine* 319, 961-964.

Caceres, C.F. & van Griensven, G.J.P. 1994 Male homosexual transmission of HIV-1. *AIDS* 8, 1051-1061.

Chamberland, M. Conley, L. & Dondero, T. 1988 Epidemiology and evolution of heterosexually acquired AIDS--United States. in *Abstracts IVth International Conference on AIDS*, No 4017 p264: Stockholm.

Chirimuuta, R.C. & Chirimuuta, R.J. 1987 *Aids, Africa and Racism*. R. Chirimuuta: Bretby House Stanhope Bretby, Burton-on-Trent, United Kingdom.

Clumeck, N. Robert-Guroff, M. Van De Perre, P. Jennings, A. Sibomana, J. Demol, P. Cran, S. & Gallo, R.C. 1985 Seroepidemiological studies of HTVL-III antibody prevalence among selected groups of heterosexual Africans. *Journal of the American Medical Association* 254, 2599-2602. Clumeck, N. Sonnet, J. Taelman, H. Mascart-Lemone, F. de Bruyere, D. Vandeperre, P. Dasnoy, J. Marcelis, L. Lamy, M. Jonas, C. Eyckmans, L. Noel, H. Vanhaeverbeek, M. & Butzler, J.P. 1984 Acquired immunodeficiency syndrome in African patients. *New England Journal of Medicine* 310, 492-497. Dondero, T.J. & Curran, J.W. 1994 Excess deaths in Africa from HIV: confirmed and quantified. *Lancet* 343, 989. Duesberg, P.H. 1989 Human immunodeficiency virus and acquired immunodeficiency syndrome: correlation but not causation. *Proceeding of the National Academy of Sciences of the United States of America* 86, 755-764.

Duesberg, P.H. 1992 AIDS acquired by drug consumption and other noncontagious risk factors. *Pharmacology and Therapeutics* 55, 201-277.

European Study Group. 1989 Risk factors for male to female transmission of HIV. *British Medical Journal* 298, 411-414.

Haverkos, H.W. & Edelman, R. 1988 The epidemiology of acquired immunodeficiency syndrome among heterosexuals. *Journal of the American Medical Association* 260, 1922-1929.

Hodgkinson, N. 1994. Research disputes epidemic of Aids. *Sunday Times*. London, May 22<sup>nd</sup>, p24.

Kashala, O. Marlink, R. Ilunga, M. Diese, M. Gormus, B. Xu, K. Mukeba, P. Kasongo, K. & Essex, M. 1994 Infection with human immunodeficiency virus type 1 (HIV-1) and human T cell lymphotropic viruses among leprosy patients and contacts: correlation between HIV-1 cross-reactivity and antibodies to lipoarabinomannan. *Journal of Infectious Diseases* 169, 296-304.

Kingsley, L.A. Kaslow, R. Rinaldo, C.R. Detre, K. Odaka, N. VanRaden, M. Detels, R. Polk, B.F. Chmiel, J. Kelsey, S.F. Ostrow, D. & Visscher, B. 1987 Risk factors for seroconversion to human immunodeficiency virus among male homosexuals. *Lancet* I, 345-348.

Konotey-Ahulu 1987 AIDS in Africa: Misinformation and Disinformation. *Lancet* II, 206-207.

Lucey, D. Hendrix, C. & Andrzejewski, C. 1991 Racial differences in anti-p24 antibody titers and total serum IGG levels in North American persons with HIV-1 infection. in *Abstracts VII International AIDS Conference* I, p.362: Florence.

Mann, J.M. Francis, H. Quinn, T. Asila, P.K. Bosenge, N. Nzilambi, N. Bila, K. Tamfum, M. Ruti, K. Piot, P. McCormick, J. & Curran, J.W. 1986 Surveillance for AIDS in a central African city. *Journal of the American Medical Association* 255, 3255-3259.

- Moore, P.S. Allen, S. Sowell, A.L. Van De Perre, P. Huff, D.L. Serufulira, A. Nsengumuremyi, F. & Hulley, S.B. 1993 Role of nutritional status and weight loss in HIV seroconversion among Rwandan women. *Journal of Acquired Immune Deficiency Syndromes* 6, 611-616.
- Mortimer, P.P. 1989 The AIDS virus and the AIDS test. *Medicine Internationale* 56, 2334-2339.
- Mortimer, P.P. Parry, J.V. & Mortimer, J.Y. 1985 Which anti-HTLV-III/LAV assays for screening and confirmatory testing? *Lancet* II, 873-877.
- Mulder, D.W. Nunn, A.J. Kamali, A. Naklylengi, J. Wagner, H.U. & Kengeya-Kayondo, J.F. 1994 Two-year HIV-1-associated mortality in a Ugandan rural population. *Lancet* 343, 1021-1023.
- Muller, W.E.G. Schroder, H.C. Reuter, P. Maidhof, A. Uhlenbruck, G. & Winkler, I. 1990 Polyclonal antibodies to mannan from yeast also recognize the carbohydrate structure of gp120 of the AIDS virus: an approach to raise neutralizing antibodies to HIV-1 infection in vitro. *AIDS* 4, 159-162.
- Nunn, A.J. Biryahwaho, B. Downing, R.G. van der Groen, G. Ojwiya, A. & Mulder, D.W. 1993 Algorithms for detecting antibodies to HIV-1: results from a rural Ugandan cohort. *AIDS* 7, 1057-1061.
- Nzilambi, N. Mann, J.M. & Francis, H. 1986 Seroprevalence among tuberculosis patients in Zaire. in Abstracts II International AIDS conference, No. 105:S17b: Paris.
- Padian, N. & Pickering, J. 1986 Female-to-male transmission of AIDS: a reexamination of the African sex ratio of cases. *Journal of the American Medical Association* 256, 590.
- Padian, N.S. Shiboski, S.C. & Jewell, N.P. 1991 Female-to-male transmission of human immunodeficiency virus. *Journal of the American Medical Association* 266, 1664-1667.
- Papadopulos-Eleopulos, E. Turner, V.F. & Papdimitriou, J.M. 1992a Kaposi's Sarcoma and HIV. *Medical Hypotheses* 39, 22-29.
- Papadopulos-Eleopulos, E. Turner, V.F. & Papdimitriou, J.M. 1992b Oxidative Stress, HIV and AIDS. *Research in Immunology* 143, 145-148.
- Papadopulos-Eleopulos, E. Turner, V.F. & Papdimitriou, J.M. 1993a Is a Positive Western Blot Proof of HIV Infection? *Bio/Technology* 11, 696-707.
- Papadopulos-Eleopulos, E. Turner, V.F. & Papadimitriou, J.M. 1993b Has Gallo proven the role of HIV in AIDS? *Emergency Medicine [Australia]* 5, 113-123.
- Papadopulos-Eleopulos, E. Turner, V.F. Papadimitriou, J.M. Hedland-Thomas, B. Causer, D. & Page, B. 1994 A critical analysis of the HIV-T4-cell-AIDS hypothesis. *Genetica* 95 No. 1-3, 2-24.

- Pearce, R.B. 1986 Heterosexual transmission of AIDS. *Journal of the American Medical Association* 256, 590-591. Piot, P. Taelman, H. Minlangu, K.B. Wobin, O. Mbendi, N.
- Ndangi, K. Mazebo, P. Kalambayi, K. Bridts, C. Feinsod, F.M. Stevens, W. Quinn, T.C. Mitchell, S. & McCormick, J.B. 1984 Acquired immunodeficiency syndrome in a heterosexual population in Zaire. *Lancet* II, 65-69.
- Quinn, T.C. Mann, J.M. Curran, J.W. & Piot, P. 1986 AIDS in Africa: An epidemiologic paradigm. *Science* 234, 955-963. Quinn, T.C. Piot, P. McCormick, J.B. Feinsod, F.M. Taelman, H. Kapita, B. Stevens, W. & Fauci, A.S. 1987 Serologic and immunologic studies in patients with AIDS in North America and Africa. *Journal of the American Medical Association* 257, 2617-2621.
- Serwadda, D. Sewankambo, N.K. Carswell, J.W. Bayley, A.C. Tedder, R.S. Weiss, R.A. Mugerwa, R.D. Lwegaba, A. Kirya, G.B. Downing, R.G. Clayden, S.A. & Dagleish, A.G. 1985 Slim disease: A new disease in Uganda and its association with HTLV-III infection. *Lancet* II, 849-852.
- Stevens, C. E. Taylor, P. E. Zang, E. A. Morrison, J. M. Harley, E. J. de Cordoba, S. R. Bacino, C., Y T. R. C. Bodner, A. J. Sarngadharan, M. G. Gallo, R. C. & Rubinstein, P. 1986 Human T-cell lymphotropic virus type III infection in a cohort of homosexual men in New York City. *Journal of the American Medical Association* 255, 2167-2172.
- Strecker, W. Gurtler, L. Binibangili, M. & Strecker, K. 1993 Clinical manifestations of HIV infection in Northern Zaire. *AIDS* 7, 597-598.
- Tomiyaama, T. Lake, D. Masuho, Y. & Hersh, E.M. 1991 Recognition of human immunodeficiency virus glycoproteins by natural anti-carbohydrate antibodies in human serum. *Biochemical and Biophysical Research Communications* 177, 279-285.
- Van De Perre, P. Lepage, P. Kestelyn, P. Hekker, A.C. Rouvroy, D. Bogaerts, J. Kayihigi, J. Butzler, J.P. & Clumeck, N. 1984 Acquired Immunodeficiency Syndrome in Rwanda. *Lancet* II, 62-65.
- Voevodin, A. 1992 HIV screening in Russia. *Lancet* 339, 1548. Weiss, R. & Thier, S.O. 1988 HIV testing is the answer--what's the question? *New England Journal of Medicine* 319, 1010-1012. Weiss, S.H. Goedert, J.J. Sarngadharan, M.G. Bodner, A.J. Gallo, R.C. & Blattner, W.A. 1985 Screening test for HTLV-III (AIDS agent) antibodies. *Journal of the American Medical Association* 253, 221-225.
- WHO 1986 Acquired Immunodeficiency Syndrome (AIDS) WHO/CDC case definition for AIDS. *Weekly Epidemiology Record* 61, 69-76.
- Widy-Wirski, R. Berkley, S. Dowing, R. Okware, S. Recine, U. Mugerwa, R. Lwegaba, A. & Sempala, S. 1988 Evaluation of the WHO clinical case definition for AIDS in Uganda. *Journal of the American Medical Association* 260, 3286-3289