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Review

AIDS in Africa: distinguishing fact and fiction

E. Papadopulos-Eleopulos,* V.F. Turner, J.M. Papadimitriou and H. Bialy

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 are critically evaluated. It is concluded that both acquired immune deficiency (AID) and the symptoms and diseases which constitute the clinical syndrome (S) are of long standing in Africa, affect both sexes equally and are caused directly and indirectly by factors other than human immunodeficiency virus (HIV). Seropositivity to HIV 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. The apparently high prevalence of 'AIDS' and 'HIV' seropositives is therefore not surprising and is not proof of heterosexual transmission of either HIV or AIDS.

Key words: Acquired immunodeficiency syndrome, Africa, heterosexual transmission, human immunodeficiency virus, seropositivity.

Following the appearance in the early 1980s of acquired immunodeficiency syndrome (AIDS) in heterosexual men, many European and American researchers looked for AIDS in Africa. There were three reasons for this. One was Robert Gallo's hypothesis that AIDS is caused by a retrovirus designated HTLV-I (human T cell lymphotropic virus 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 homosexual men before the AIDS era, it had been present in Africa since antiquity. Its characteristic clinical appearances are described in the Ebers papyrus which dates from 1600 BC. The authors of the report of AIDS cases in patients of African descent, did not exclude the possibility that AIDS has always been present in Africa (Clumeck *et al.* 1984). Despite these facts, the claim that AIDS is a new disease of viral aetiology everywhere, including Africa, has been overwhelmingly accepted. In fact, AIDS in Africa became of such pivotal significance to the human immunodeficiency virus (HIV)/AIDS theory that, in 1990, nearly 600 'AIDS-related' studies were conducted there. 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 a widely publicized study, which was reported in *The Lancet*, Mulder *et al.* (1994) 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 sero-negative individuals and 89 in seropositive individuals. Of the latter, 73 were adults. In a commentary accompanying publication of this study, researchers from the US Centers for Disease Control (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 sero-negative individual. Although most reasonable observers

E. Papadopulos-Eleopulos is with the Department of Medical Physics, The Royal Perth Hospital, Perth 6000, Western Australia, Australia; fax: 619 224 2511. V.F. Turner is with the Department of Emergency Medicine, The Royal Perth Hospital, Perth 6000, Western Australia, Australia. J.M. Papadimitriou is with the Department of Pathology, University of Western Australia, Perth, Western Australia. H. Bialy is with Bio/Technology, 65 Becker St, New York, NY 10012, USA. *Corresponding author.

do accept that HIV causes AIDS, even skeptics 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 sero-positivity leads to early death must be curious indeed" (Dondero & Curran 1994). Below, 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) and ". . . among healthy Africans resident in a non-AIDS area, the numbers of helper and suppressor lymphocytes were the same in HTLV-III/LAV sero-positive and sero-negative subjects . . ." (Biggar 1986). "Africans are frequently exposed, due to hygienic conditions and other factors, to a wide variety of viruses, including cytomegalo virus (CMV), Epstein Barr virus (EBV), hepatitis B virus, and herpes simplex virus (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" (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 unrecognizable as AIDS in the West—so much so that if African patients suddenly switched continents, very few would remain AIDS cases. This is due to the existence of two completely different AIDS definitions, one applying to Africa and the other to the rest of the World (except Asia and Latin America). Unlike the AIDS definition in the West, the 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 World Health Organization's (WHO) Bangui definition (Anon. 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 paediatric AIDS is particularly difficult in Kinshasha [Zaire], since many children have severe infant and childhood diseases with similar manifestations (e.g. weight loss, chronic diarrhoea)" (Mann *et al.* 1986). Anthony Fauci, Director of the National Institute of Allergy and Infectious Diseases in the USA, 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 empirical definition, but nevertheless, (slim disease) is being considered AIDS in Africa" (*AIDS Alert*, January 1987). According to Myron Essex, on whose 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 February 1988).

In summary, although, the best known researchers of African AIDS clearly accept that both AID and the AID syndrome 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 antibodies to HIV. 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 unique to HIV, a positive test cannot be considered proof of HIV infection. This is because non-HIV antibodies can and do 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, i.e. one must know how often false-positive tests occur. For this it is mandatory to:

(1) test a large number of subjects, 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 literature on HIV antibody tests has failed to show a single instance of the use of the above parameters, the only scientifically valid method of determining the specificity of the HIV antibody tests. Indeed, comparisons between the published work on retrovirology and the present World-wide 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 (Papadopulos-Eleopulos *et al.* 1993a, b). 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 unknown reasons, HIV experts (such as D. 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 method was used by Burke and his colleagues and apparently many HIV/AIDS experts, including David Ascher, believe that the false-positive rate of the HIV antibody tests is 1 in 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 (WB) as a 'gold standard'. For them, the true sero-status depends on repeating two different ELISA until they are concordant, an outcome which could result 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 by 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 clinicial 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' (Mortimer 1989). Although Mortimer *et al.* (1985) as well as Gallo and his colleagues (Weiss *et al.* 1985) used the '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.* (1994) recorded 377 individuals with a positive test. Of these, 89 died within 2 years. Although not stated, we can assume that many of the remaining apparently HIV-positive cases were asymptomatic and thus, according to Mortimer, all these individuals had false-positive tests. Of the 73 adults who died, only five had 'AIDS'! The other 68 died of unlisted causes and, if asymptomatic for 'AIDS', must all be regarded as false-positives.

Epidemiological data show that AIDS patients in general and Africans, including healthy Africans, have high titres of many different antibodies. For example, United States data indicate that (mean $\pm$ standard deviation) serum IgG concentrations are higher in HIV-positive American Blacks (2234 $\pm$ 930 mg/dl) than in HIV-positive Caucasians (1601 $\pm$ 520 mg/dl). Serum IgG levels are also higher in Black blood donors (1356 $\pm$ 220 mg/dl) than in Caucasians (1072 $\pm$ 243 mg/dl) (Lucey *et al.* 1991). Thus, in those individuals with high concentrations 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 experts on African HIV/AIDS. In 1986, Quinn *et al.* (1986) wrote, in Africa ". . . serodiagnosis is complicated by the need for confirmatory testing because of the presence of possible cross-reacting antibodies". One year earlier, Biggar *et al.* (1985) 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 sero-epidemiology 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." 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, in 1994 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 WB and ELISA". The

cross-reactivity was found to be caused by antibodies directed against two major carbohydrate-containing *Mycobacterium leprae* antigens—phenolic glycolipid I and especially lipoarabinomannan, an arabinose-containing lipopolysaccharide which is also present in *Mycobacterium 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), caused by *M. tuberculosis*, is endemic in Africa. Of the 661 million people in sub-Saharan Africa, 2 to 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% to 50% of African so-called '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 five '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 tuberculosis has existed in Africa for many generations and, according to Essex, positive HIV antibody tests in patients with tuberculosis 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 do nothing more than prove their predictions (Papadopulos-Eleopulos *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, of course, is not the cause of syphilis.
(2) patients with infectious mononucleosis develop antibodies that react with red blood cells from sheep and horse. However, neither sheep nor horse red blood cells are 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 per cent of haemophiliacs with AIDS give a positive test 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 (Widy-Wirski *et al.* 1988), 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 another study (Strecker *et al.* 1993), 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 the former study and 83% in the latter, the positive predictive values of amenorrhoea were 42% and 89%, respectively.

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-month 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 non-convertors. Nine of 27 (33%) seroconvertors, compared with one of 44 (2%) controls, lost at least 5 kg 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.... A similar study in Senegal uncovered 16 of 44 [36%] patients said to have AIDS, who again showed no sign of infection with either virus" (*New Scientist*, 18 February 1988). Thus the HIV hypothesis of AIDS does not satisfy even the most fundamental criterion for proof of an aetiologic agent. More extensive, and thoroughly referenced critiques of its numerous other deficiencies can be found in Duesberg (1993) and Papadopulos-Eleopulos *et al.* (1992a, b; 1993a, b; 1994).

Heterosexual Transmission

In an attempt 'to evaluate acquired immunodeficiency syndrome (AIDS) in central Africa' a prospective study was done in Kigali, Rwanda, where Kaposi's sarcoma (KS) is endemic (Van De Perre *et al.* 1984). This study was conducted by researchers from Belgium, the Netherlands and Rwanda. In 1983, a questionnaire was sent "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 generalized 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 generalized lymphadenopathy consisting of palpable lymph nodes larger than 1 cm at two or more extra-inguinal 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" (i.e. a decreased T4/T8 ratio). Van De Perre *et al.* (1984) found 26 such patients (17 males and nine females), two of whom 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 is 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 (Piot *et al.* 1984). They 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 3-week period, Piot *et al.* (1984) 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 six had disseminated KS. Regarding the latter, Piot *et al.* (1984) wrote "Since KS has long been endemic in Zaire, only patients with fulminant KS were included". Discussing their findings, the authors 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 (Brun-Vezinet *et al.* 1984). The sera were tested by ELISA and then by a RIPA (radioimmunoprecipitation assay) similar to a Western blot. The latter was considered positive if a p24 band was present. The p41 band and also an 84-kDa band were not considered diagnostic because 'The 43-kD [p41] band and the 84-kDa 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 patients (88%) were positive by both tests. So were six out of 26 (23%) controls. However, with the exception of a few other reports from Africa (see below), no other such correlation between ELISA and WB has ever been reported. For example, in 1988, Burke *et al.* (1988) tested 1.2 million healthy American military recruits and found that, of 6000 individuals with two consecutive positive ELISA, only 2000 subsequently had a positive WB. In Russia, in 1991, of 30,000 positive screening ELISA, only 66 were WB positive (Voevodin 1992). Since 1987, nobody in the World, with the possible exception of Montagnier, considers that the p24 band is 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 (Mann *et al.* 1986). 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 were 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 (Clumeck *et al.* 1985). 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 these tests, '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 Africans have AIDS, that "Homosexuality, intravenous drug use and blood transfusions did not appear to be risk factors" in Africa and that 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 are 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, have written: "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 have ever been presented, either in Africa or anywhere else. In fact, according to 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)". This means that a man or 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 had never had sexual intercourse (Chamberland *et al.* 1988). Given the fact that the best known HIV/AIDS experts on African AIDS admit

that: (1) what is known as AIDS in Africa has been present for centuries and was equally common in men and women; and (2) 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 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 homosexual 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 reported 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 (Anon. 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 major 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. 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 unusual 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 long existed in Africa. 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." (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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