

Berkeley, June 22, 2000

From: Peter Duesberg

Re: The African AIDS Epidemic

To: The AIDS panel, particularly those who participated at the May 5 to 6 meeting in Pretoria

**THE AFRICAN AIDS EPIDEMIC:
NEW AND CONTAGIOUS
OR
OLD UNDER A NEW NAME?**

An infectious epidemic is typically diagnosed by scientists and non-scientists by a sudden increase in morbidity and mortality of a population. As a result the affected population declines significantly, and a relatively immune population emerges. The most readable modern description of such an epidemic is Albert Camus' "The plague".

Roy Anderson, professor of zoology at the Wellcome Trust Centre for Epidemiology of Infectious Diseases in Oxford, UK, provides a recent scientific description in a piece entitled "The spread of HIV and sexual mixing patterns" (Anderson, 1996). According to Anderson, "The historical and epidemiological literature abounds with accounts of infectious diseases invading human communities and of their impact on social organization and historical events. We typically think of a new epidemic in a "virgin" population as something that arises suddenly, sweeps through the population in a few months, and then wanes and disappears. Indeed, the classical epidemic curve for many respiratory or intestinal tract viral and bacterial infections is

bell-shaped, with an overall duration of a few months to a year or so. Figure 4-1 illustrates a well-documented example, the 1665 plague in London, believed to have killed about one-third of the population in a few months.” The seasonal poliomyelitis epidemics from the days prior to the polio vaccine, and the ever new, seasonal flu epidemics are specific modern examples of viral epidemics.

All of these viral and microbial epidemics have the following in common:

- (i) They rise exponentially and then decline within weeks or months as originally described by William Farr in the early 19th century (Bregman & Langmuir, 1990). The rise reflects the exponential spread of contagion and the fall reflects the resulting natural vaccination or immunity of survivors.
- (ii) The epidemics spread randomly (“heterosexually” in the words of AIDS researchers) in the population.
- (iii) The resulting infectious diseases are highly specific reflecting the limited genetic information of the causative microbe. As a consequence the viral diseases are typically more specific than those caused by the more complex bacteria or fungi. It is for this reason that the viruses and microbes are typically named for the specific disease they cause. For example influenza virus is called after the flu, polio virus after the poliomyelitis, and hepatitis virus after the liver disease it causes.
- (iii) The microbial and particularly the viral epidemics are self-limiting and thus typically seasonal, because they induce anti-microbial and viral immunity and select also for genetically resistant hosts.

By contrast, the following are characteristics of diseases caused by non-contagious, chemical or physical factors:

- (i) They follow no specific time course, but one that is determined by the dose and duration of exposure to the toxin;
- (ii) They spread according to consumption or exposure to toxic agents, but not exponentially;
- (iii) They spread either non-randomly with occupational or lifestyle factors, or randomly with environmental or nutritional factors;
- (iv) They range from relatively specific to unspecific depending on the nature of the toxin;
- (v) They are limited by discontinuation of intoxication, but not self-limiting because they do not generate immunity.

For example, the American pellagra epidemic of the rural South in the early decades of the 20th century lasted for decades and no immunity emerged, until a vitamin B rich diet proved to be the cure (see my posting of June 10). And it did not spread to the industrial North with vitamin B rich diets. Similarly the rather unspecific American epidemic of lung cancer-emphysema-heart disease-etc. rose steadily, not exponentially, in the 1950s and has lasted now for over 50 years without evidence for immunity. It did not spread randomly in the population but was restricted to smokers. And it is now slowly coming down as smoking slowly declines (Greenlee *et al.*, 2000). Likewise, the American and European AIDS epidemics rose steadily, not exponentially, and completely non-randomly biased 85% in favor of males, and have followed first the recreational and then also the anti-AIDS-viral drug epidemics of these countries exactly (Duesberg & Rasnick, 1998). American and European AIDS does not manifest in one or even just a few specific diseases typical of microbial epidemics and does not spread to the general

non-drug using population. Consistent with the heterogeneity of the causative toxins AIDS manifests in a bewildering spectrum of 30 non-specific, heterogeneous diseases. There is no evidence for AIDS-immunity in 18 years, but the American/European AIDS epidemics are now coming down slowly as fewer people use recreational drugs (Duesberg & Rasnick, 1998).

AFRICAN AIDS IN NUMBERS

Now I will briefly analyze how African AIDS measures up with “the historical and epidemiological literature” described by Anderson and others (Fenner *et al.*, 1974). My analysis is based on statistical numbers from the World Health Organization (WHO) in Geneva, the United Nations and the U.S. Agency for International Development & the U.S. Census Bureau (USAID). According to the WHO’s Weekly Epidemiological Records the whole continent of Africa has generated between 1991 and 1999 a rather steady yield of 60,000 to 90,000 AIDS cases annually, on average about 75,000 (WHO’s Weekly Epidemiological Records since 1991). Based on the last available data from South Africa, 8,976 cases were reported there between 1994 and 1996 by the WHO, corresponding to about 4,500 cases per year (WHO’s Weekly Epidemiological Records 1998 and 1995). (I managed to show these data briefly to all participants of the May meeting with the permission of the Facilitator-in-Chief, Dr. Stephen Owen.) The WHO does not report how many of these cases are deaths, how many survive with, and how many recover from AIDS. However, it is evident from the WHO data that the African AIDS epidemic is not following the bell-shaped curve of an exponential rise and subsequent sharp drop with immunity, that are typical of infectious epidemics. Instead it

drags on like a nutritionally or environmentally caused disease (Seligmann *et al.*, 1984), that steadily affects, what appears to be only a very small percentage of the African population.

Given a current African population of 616 million (United Nations Environment Programme, June 15, 2000), and an average of 75,000 African AIDS cases per year, it follows that only 0.012% of the African population is annually suffering or dying from AIDS. Likewise only 0.01% of the South African population was suffering from AIDS between 1994 and 1996, based on the 4,500 annual cases and a population of approximately 44 million (US Agency for International Development, “HIV/AIDS in the developing World”, May 1999). This means that the new African AIDS epidemic only represents a very small fraction of normal African mortality.

Based on a current average life expectancy for Africa of about 50 years (US Agency for International Development, “HIV/AIDS in the developing World”, May 1999), the annual mortality of 616 million people is 12.3 million. Thus even if we assume that all AIDS cases reported by the WHO are deaths, the African AIDS epidemic represents only 75,000 out of 12,300,000 deaths per year, or 0.6% of all African mortality. Thus African AIDS is certainly not one of the historical microbial epidemics described by Camus and Anderson (see above). Since no immunity has emerged in over a decade the restriction of African AIDS to a relatively small fraction of the large reservoir of susceptible people indicates non-contagious risk factors that are limited to certain subsets of the African population.

In view of the very small share (0.6%) that the African AIDS epidemic seems to hold on Africa's total mortality, the question arises whether the mortality claimed for

AIDS is in fact new mortality, that can be distinguished from conventional mortality, or whether it is a minor fraction of conventional mortality under a new name. Similar questions have already been asked by our colleagues Harvey Bialy (USA), Christian Fiala (Austria), Charles Gesheker (USA), and David Rasnick (USA) (see their postings). To answer these questions we must try to distinguish African AIDS diseases from conventional African diseases (i) clinically as well as (ii) statistically.

THE LONG LIST OF AFRICAN AIDS DISEASES CAN NOT BE CLINICALLY DISTINGUISHED FROM THEIR CONVENTIONAL COUNTERPARTS

According to the WHO's Bangui definition of AIDS (Widy-Wirski *et al.*, 1988; Fiala, 1998) and the "Anonymous AIDS Notification" forms of the South African Department of Health, African AIDS is not a specific clinical disease, but a battery of previously known and thus totally unspecific diseases, ie. "weight loss over 10%, chronic diarrhea for more than a month, fever for more than a month, persistent cough, generalized pruritic dermatitis, recurrent herpes zoster (shingles), candidiasis oral and pharyngeal, chronic or persistent herpes, cryptococcal meningitis, Kaposi's sarcoma". Since these diseases include the most common diseases in Africa and in much of the rest of the world, it is impossible to distinguish clinically African AIDS diseases from previously known, and concurrently diagnosed, conventional African diseases. Thus African AIDS is clinically unspecific, unlike microbial diseases, but just like some nutritionally and chemically caused diseases (see above).

**AFRICAN AIDS IS TOO SMALL TO BE DETECTED STATISTICALLY
AGAINST THE BACKGROUND OF NORMAL AFRICAN MORBIDITY,
MORTALITY AND GROWTH RATES**

We have already pointed out that it is almost impossible to be certain about the existence of a new African AIDS epidemic that claims only 0.6% of African mortality, particularly since all AIDS defining diseases are profoundly conventional African diseases.

The same is true if we try to determine the effect of the presumably new African AIDS epidemic on the current growth rates of Africa. The annual population growth rates of Africa have been between 2.4 and 2.8% per year since 1960 based on the American Agency for International Development & the U.S. Census Bureau's "HIV/AIDS in the Developing World" (U.S. Agency for International Development & U.S. Census Bureau, Feb. & May 1999) and the United Nations' "African population Database Documentation" (United Nations Environment Programme, June 15, 2000). As a result of the high African growth rates, the population of the whole African continent has grown from 274 million in 1960, to 356 million in 1970, to 469 million in 1980, and to 616 million in 1990 (United Nations Environment Programme, June 15, 2000). By comparison the annual growth rate of the US is only 1% and that of Europe is only 0.5% (USAID, Feb. & May 1999).

Because of the numerical discrepancy between the relatively high African growth rates (2.4 to 2.8%) and the small annual deficits of these growth rates to be expected from AIDS mortality (0.6%), an African AIDS epidemic can not be identified or confirmed based on its effect on the high African growth rates. In view of this, and the complete overlap between the complex battery of diseases that define the AIDS epidemic and their

conventional counterparts, it appears that the presumably new AIDS epidemic can be neither distinguished epidemiologically nor clinically from conventional African diseases and mortality.

DECEPTIVE REPORTING OBSCURES ANALYSIS OF AFRICAN AIDS

To all of us who have been subjected to the American AIDS rhetoric, and indeed the rhetoric of our first meeting in Pretoria last May, about the “catastrophic dimensions” of African AIDS (Washington Post, April 30, 2000), the healthy African growth rates come as a big surprise. Take as an example of this rhetoric president Clinton’s recent designation of AIDS as a “threat to US national security ... spurred by US intelligence reports that looked at the pandemic’s broadest consequences, ... particularly Africa ... [and] projected that a quarter of southern Africa’s population is likely to die of AIDS ...” (Washington Post, April 30, 2000).

In view of this rhetoric it would appear that neither president Clinton nor the “U.S. intelligence” are aware of information available to the American Agency for International Development & the U.S. Census Bureau. Indeed the USAID & Census Bureau seem to have noticed the discrepancy between the facts and the rhetoric and are trying to hide it – the possible reason why “the largest demographic impact of AIDS” is cautiously described either as just a relatively small reduction in “life expectancy” or in expected population growth (not loss!): “Differences in population size between the AIDS-adjusted and the non-adjusted scenarios are often substantial ... By the year 2010 ... South Africa will have 5.6 million fewer people ...” than expected based on current growth rates (“HIV/AIDS in the Developing World”, U.S. Agency for International

Development & U.S. Census Bureau, May 1999). A “catastrophe” 10 years down the road – and a “threat to U.S. national Security” now!

The alarming tone of WHO’s joint United Nations Programme on HIV/AIDS, “AIDS epidemic update: December 1999” (UNAIDS December 1999), announcing that Africa had gained 23 million “living with HIV/AIDS”, because they are “estimated” carriers of antibodies against HIV, since the “early 80s” (WHO, Weekly Epidemiological Record 73, 373-380, 1998) is equally surprising in view of information available to the agency. Neither the WHO nor the United Nations point out that Africa had gained 147 million people during the same time in which the continent was said to suffer from a new AIDS epidemic. Likewise, South Africa has grown from 17 million to 37 million in 1990 (United Nations Environment Programme, June 15, 2000), and to 44 million now (“HIV/AIDS in the Developing World”, U.S. Agency for International Development & U.S. Census Bureau, May 1999). In the last decade South Africa has also gained 4 million HIV-positive people (A. Kinghorn & M. Steinberg, South African Department of Health, undated document probably from 1998, provided at the Pretoria meeting). Thus South Africa has gained 4 million HIV-positives during the same decade in which it grew by 7 million people.

Moreover, although the 23 million “estimated” HIV-antibody positives are said to be “living with HIV/AIDS” by the WHO, the agency does not offer any evidence for morbidity or mortality exceeding the modest numbers, ie. about 75,000 cases annually, reported by the it’s Weekly Epidemiological Records (see above). The agency’s estimates of HIV-positives are indeed just that, because according to the 1985-Bangui definition of African AIDS as well as to the current “Anonymous AIDS Notification”

forms of the South African Department of Health no HIV tests are required for an AIDS diagnosis (Widy-Wirski *et al.*, 1988; Fiala, 1998).

In addition the WHO promotes the impression of a microbial AIDS epidemic, by reporting African AIDS cases cumulatively rather than annually (WHO's Weekly Epidemiological Records since the beginning of the epidemic). This practice creates the deceptive impression of an ever growing, almost exponential epidemic, even if the annual incidence declines (Fiala, 1998).

It would follow that the estimated increases in African HIV antibody (!)-positives do not correlate with decreases in any African population. On the contrary, they correlate with unprecedented simultaneous increases in the country's populations – hardly the “catastrophe” imagined by the Washington Post and propagated by the WHO and the American AIDS establishment. But this deceptive AIDS propaganda biases a scientific analysis of African AIDS by all those who are not aware of the facts.

CONCLUSIONS

(1) The African AIDS epidemic fails all criteria of a microbial or viral epidemic:

(i) It is steady, i.e. about 75,000 cases per year since the early 1990s, instead of growing exponentially into the large reservoir of 617 million susceptible people, as is typical of a new viral or microbial epidemic;

(ii) It is not self-limiting via immunity within weeks or months, as is typical of a microbial and particularly of a viral disease. Instead it appears to maintain for years a rather steady share of African morbidity and mortality.

(iii) It is clinically exceedingly heterogeneous totally lacking any specificity of its own, unlike all conventional viral and even bacterial diseases.

In conclusion, the African AIDS epidemic does not have even one of the specific characters of a viral or microbial epidemic.

(2) Since the suspected African AIDS epidemic of an average of 75,000 annual cases can neither be identified as a new epidemic clinically because of its total lack of a clinical identity, nor numerically because of its small share of the total African morbidity and because of undetectable effects on the rapid growth of the African population, the primary scientific task of our AIDS panel will now be to determine whether there is in fact a new epidemic of AIDS defining diseases in Africa, or whether a fraction normal morbidity and mortality has been renamed AIDS. The answer to this question would be the first order of business for all AIDS prevention and treatment programs considered by president Mbeki. To find this answer, I second the proposal from an African AIDS researcher published 13 years ago, "Clinical epidemiology, not [HIV] seroepidemiology, is the answer to Africa's AIDS problem" (Konotey-Ahulu, 1987).

(3) The African statistics of AIDS and HIV antibody-positives confirm Mbeki's suspicion about discrepancies between the African and American AIDS epidemics (Mbeki's letter to U.S. president Clinton, Washington Post, April 19, 2000): In Africa 23 million HIV-positives generate per year 75,000 AIDS patients, ie. 1 AIDS case per 300 HIV-positives. But in the US, 0.9 million HIV-positives (WHO, Weekly Epidemiological Record 73, 373-380, 1998) now generate per year about 45,000 AIDS

cases (Centers for Disease Control, 1999), ie. 1 AIDS case per 20 HIV-positives. Thus the AIDS risk of an American HIV-positive is about 15-times higher than that of an African! Since over 150,000 healthy (!) HIV-positive Americans are currently treated with DNA chain-terminating and other anti-HIV drugs (Duesberg & Rasnick, 1998), and since American HIV-positives have a 15-fold higher AIDS risk than African HIV-positives, president Mbeki must warn about American advice on “treatments” of HIV-positives.

(4) The discrepancies between African AIDS and infectious disease, and the discrepancies between the high AIDS risk of American compared to African HIV-positives can both be readily explained by the hypothesis that AIDS is caused by non-contagious risk factors and that HIV is a harmless passenger virus (Duesberg, 1996; Duesberg & Rasnick, 1998). According to this hypothesis the African AIDS diseases are generated by their conventional, widespread causes, malnutrition, parasitic infections and poor sanitation as originally proposed by leading AIDS researchers including Fauci, Seligmann et al. (Seligmann *et al.*, 1984).

This hypothesis also offers a simple explanation for the “heterosexual” distribution of AIDS in the African people, a question also asked by Mbeki in his letter to president Clinton (see above). Malnutrition, parasitic infections and poor sanitation do not discriminate between sexes. By contrast, American AIDS would be caused by recreational drugs consumed by millions and anti-HIV drugs prescribed to about 200,000 including 150,000 still healthy HIV-positives (Duesberg & Rasnick, 1998). The non-random, 85%-male epidemiology of American AIDS reflects the male prerogative on

hard recreational drugs (heroin, cocaine) and the wide-spread use of drugs as male homosexual stimulants (Haverkos & Dougherty, 1988; Duesberg & Rasnick, 1998).

In the light of this hypothesis the new epidemic of HIV-antibodies would simply reflect a new epidemic of HIV-antibody testing, introduced and inspired by new American biotechnology. This technology was developed during the last 20 years for basic research to detect the equivalents of biological needles in a haystack, but not to “detect” the massive invasions of viruses that are necessary to cause ALL conventional viral diseases (Duesberg, 1992; Duesberg & Schwartz, 1992; Duesberg, 1996; Mullis, 1996; Duesberg & Rasnick, 1998; Mullis, 1998). But this technology is now faithfully but inappropriately used by thousands of AIDS virus researchers and activists to detect latent, ie. biochemically and biologically inactive HIV or even just antibodies against it (Duesberg & Bialy, 1996)! The same technology also provides job security for other virologists and doctors searching for latent, and thus biologically inactive, viruses as their preferred causes of Kaposi’s sarcoma, cervical cancer, leukemia, liver cancer, and rare neurological diseases – without ever producing any public health benefits (Duesberg & Schwartz, 1992).

(5) President Mbeki must also be warned about Dr. Joe Sonnabend’s answer to the president’s question about the epidemiological discrepancy between the “heterosexual” AIDS epidemic in Africa and the non-random, 85%- male epidemic in the U.S. (Mbeki’s letter to U.S. president Clinton, Washington Post, April 19, 2000). According to Sonnabend’s hypothesis, Africans acquire HIV heterosexually, because they simultaneously suffer from a long list of diseases, including “tuberculosis, malaria, other

protozoal infections, bacterial diarrheal infections, pneumonia, plasmodium, Leishmania” etc. (see Sonnabend’s posts and my own responses to Sonnabend on this website).

However, the very low AIDS risk of an African HIV-positive, compared to an American, calls this hypothesis into question. If the Sonnabend-hypothesis were correct, African HIV-positives should develop AIDS much more readily than their American counterparts. But the opposite is true. In fact according to Sonnabend most Africans should already have AIDS by the time they pick up HIV “heterosexually”.

Moreover, the Sonnabend-hypothesis does not resolve the discrepancy between relatively high share of children from 0–14 years in African AIDS, ie. 7%, compared to the 1% share of AIDS by their American counterparts (WHO, Weekly Epidemiological Record, vol. 49, pp381-384, 4 December 1998). According to the WHO, “AIDS in children is an important phenomenon in many African countries, whereas it is relatively rare in industrialized countries.” Again AIDS in children is not compatible with “heterosexual transmission of HIV” while suffering from Sonnabend’s bewildering list of diseases. But AIDS in children is very compatible with malnutrition, parasitic infection and poor sanitation. Therefore, president Mbeki must be warned against treatment of these children with DNA chain-terminators and other anti-HIV drugs as suggested by Sonnabend’s hypothesis.

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References

- Anderson, R. M., (1996): The spread of HIV and sexual mixing patterns. In: AIDS in the World II, pp. 71, J. E. Mann & D. J. M. Tarantola (eds.). Oxford Univ. press. Oxford.
- Bregman, D. J. & A. D. Langmuir, (1990): Farr's law applied to AIDS projections. J. Am. Med. Assoc. 263: 50-57.
- Centers for Disease Control, (1999): U.S. HIV and AIDS cases reported through June 1999. HIV AIDS Surveillance Report 11: (1) 1-37.
- Duesberg, P. H., (1992): AIDS acquired by drug consumption and other noncontagious risk factors. Pharmacology & Therapeutics 55: 201-277.
- Duesberg, P. H., (1996): Inventing the AIDS Virus. Regnery Publishing Inc., Washington.
- Duesberg, P. H. & H. Bialy, (1996): Duesberg and the right of reply according to Maddox-Nature. In: AIDS: virus- or drug induced?, 5, pp. 111-125, P. H. Duesberg (eds.). Kluwer Academic Publishers. Dordrecht/Boston/London.
- Duesberg, P. H. & D. Rasnick, (1998): The AIDS dilemma: drug diseases blamed on a passenger virus. Genetica 104: 85-132.
- Duesberg, P. H. & J. R. Schwartz, (1992): Latent viruses and mutated oncogenes: no evidence for pathogenicity. Prog Nucleic Acid Res Mol Biol 43: 135-204.
- Fenner, F., B. R. McAuslan, C. A. Mims, J. Sambrook & D. O. White, (1974): The Biology of Animal Viruses. Academic Press, Inc., New York.
- Fiala, C., 1998. AIDS in Africa: dirty tricks. *New African*, p36-38.

Greenlee, R. T., T. Murray, S. Boldon & P. A. Wingo, (2000): *Cancer Statistics, 2000*.

CA cancer J Clin 50: 7-33.

Haverkos, H. W. & J. A. Dougherty (eds), 1988. *Health Hazards of Nitrite Inhalants*.

83, US. Dept. Health & Human Services, Washington, DC.

Konotey-Ahulu, F. I. D., (1987): Clinical epidemiology, not seroepidemiology, is the

answer to Africa's AIDS problem. *BMJ* 294: 1593-1594.

Mullis, K., (1996): Foreword. In: *Inventing the AIDS Virus*, pp. XI-XIV, P. Duesberg

(eds.). Regnery Publishing, Inc. Washington DC.

Mullis, K., (1998): *Dancing naked in the mind field*. Pantheon Books, New York.

Seligmann, M., L. Chess, J. L. Fahey *et al.*, (1984): AIDS—an immunologic

reevaluation. *N. Engl. J. Med.* 311: 1286-1292.

Widy-Wirski, R., S. Berkley, R. Downing, S. Okware, U. Recine, R. Mugerwas, A.

Lwegaba & S. Sempala, (1988): Evaluation of the WHO clinical case definition for
AIDS in Uganda. *JAMA* 260: (22) 3286-3289.