

# Duesberg on AIDS

*"The important thing is to not  
stop questioning."*

Albert Einstein

Welcome to Peter Duesberg's HIV/AIDS research web site.

Peter H. Duesberg, Ph.D. is a professor of Molecular and Cell Biology at the University of California, Berkeley. [Biographical Sketch](#)

He isolated the first cancer gene through his work on retroviruses in 1970, and mapped the genetic structure of these viruses. This, and his subsequent work in the same field, resulted in his election to the National Academy of Sciences in 1986. He is also the recipient of a seven-year Outstanding Investigator Grant from the National Institutes of Health.

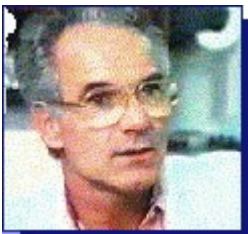
On the basis of his experience with retroviruses, Duesberg has challenged the virus-AIDS hypothesis in the pages of such journals as Cancer Research, Lancet, Proceedings of the National Academy of Sciences, Science, Nature, Journal of AIDS, AIDS Forschung, Biomedicine and Pharmacotherapeutics, New England Journal of Medicine and Research in Immunology. He has instead proposed the hypothesis that the various American/European AIDS diseases are brought on by the long-term consumption of recreational drugs and/or AZT itself, which is prescribed to prevent or treat AIDS. See [The AIDS Dilemma: Drug diseases blamed on a passenger virus](#).

For a detailed discussion of American/European AIDS as opposed to African AIDS, see [The African AIDS Epidemic: New and Contagious or Old Under a New Name](#).

This is Duesberg's official site, containing his written works on the subject, as well as other scientists that support his views such as Kary B. Mullis. Kary Mullis won the 1993 Nobel Prize in Chemistry for his invention of the polymerase chain reaction technique for detecting DNA. This is the technique used to search for fragments of HIV in AIDS patients.

Prof. Duesberg's findings have been a thorn in the side of the medical establishment and drug companies since 1987. Instead of engaging in scientific debate, however, the only response has been to cut-off funding to further test Professor's Duesberg's hypothesis.

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## **The AIDS Dilemma: drug diseases blamed on a passenger virus**

by Peter Duesberg & David Rasnick

Genetica 104: 85-132. 1998

For the complete article, see [The AIDS Dilemma](#), an Adobe Acrobat file (.pdf)

### **Abstract**

Almost two decades of unprecedented efforts in research costing US taxpayers over \$50 billion have failed to defeat Acquired Immune Deficiency Syndrome (AIDS) and have failed to explain the chronology and epidemiology of AIDS in America and Europe. The failure to cure AIDS is so complete that the largest American AIDS foundation is even exploiting it for fundraising: 'Latest AIDS statistics 0,000,000 cured. Support a cure, support AMFAR.' The scientific basis of all these unsuccessful efforts has been the hypothesis that AIDS is caused by a sexually transmitted virus, termed Human immunodeficiency virus (HIV), and that this viral immunodeficiency manifests in 30 previously known microbial and non-microbial AIDS diseases.

In order to develop a hypothesis that explains AIDS we have considered ten relevant facts that American and European AIDS patients have, and do not have, in common:

- (1) AIDS is not contagious. For example, not even one health care worker has contracted AIDS from over 800,000 AIDS patients in America and Europe.
- (2) AIDS is highly non-random with regard to sex (86% male); sexual persuasion (over 60% homosexual); and age (85% are 25-49 years old).
- (3) From its beginning in 1980, the AIDS epidemic progressed non-exponentially, just like lifestyle diseases.
- (4) The epidemic is fragmented into distinct subepidemics with exclusive AIDS-defining diseases. For example, only homosexual males have Kaposi's sarcoma.
- (5) Patients do not have any one of 30 AIDS-defining diseases, nor even immunodeficiency, in common. For example, Kaposi's sarcoma, dementia, and weight loss may occur without immunodeficiency. Thus, there is no AIDS-specific disease.
- (6) AIDS patients have antibody against HIV in common only by definition-not by natural coincidence. AIDS-defining diseases of HIV-free patients are called by their old names.
- (7) Recreational drug use is a common denominator for over 95% of all American and European AIDS patients, including male homosexuals.
- (8) Lifetime prescriptions of inevitably toxic anti-HIV drugs, such as the DNA chain-terminator AZT, are another common denominator of AIDS patients.
- (9) HIV proves to be an ideal surrogate marker for recreational and anti-HIV drug use. Since the virus is very rare (< 0.3%) in the US/European population and very hard to transmit sexually, only those who inject street drugs or, have over 1,000 typically drug-mediated sexual contacts are likely to become positive.
- (10) The huge AIDS literature cannot offer even one statistically significant group of drug-free AIDS patients from America and Europe.

In view of this, we propose that the long-term consumption of recreational drugs (such as cocaine, heroin, nitrite inhalants, and amphetamines) and prescriptions of DNA chain-terminating and other anti-HIV drugs, cause all AIDS diseases in America and Europe that exceed their long-established, national backgrounds, i.e. >95%. Chemically distinct drugs cause distinct AIDS-defining diseases; for example, nitrite inhalants cause Kaposi's sarcoma, cocaine causes weight loss, and AZT causes immunodeficiency, lymphoma, muscle atrophy, and dementia. The drug hypothesis predicts that AIDS:

- (1) is non-contagious;
- (2) is non-random, because 85% of AIDS causing drugs are used by males, particularly sexually active homosexuals between 25 and 49 years of age, and
- (3) would follow the drug epidemics chronologically.

Indeed, AIDS has increased from negligible numbers in the early 1980s to about 80,000 annual cases in the early '90s and has since declined to about 50,000 cases (US figures). In the same period, recreational drug users have increased from negligible numbers to millions by the late 1980s, and have since decreased possibly twofold. However, AIDS has declined less because since 1987 increasing numbers of mostly healthy, HIV-positive people, currently about 200,000, use anti-HIV drugs that cause AIDS and other diseases. At least 64 scientific studies, government legislation, and non-scientific reports document that recreational drugs cause AIDS and other diseases. Likewise, the AIDS literature, the drug manufacturers, and non-scientific reports confirm that anti-HIV drugs cause AIDS and other diseases in humans and animals. In sum, the AIDS dilemma could be solved by banning anti-HIV drugs, and by pointing out that drugs cause AIDS –modeled on the successful anti-smoking campaign.

*An unflinching determination to take the whole evidence into account is the only method of preservation against the fluctuating extremes of fashionable opinion.*

Alfred North Whitehead (1861-1947)

(Whitehead, 1967).

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

**From Peter Duesberg to the AIDS panel, 6/22/00**

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
- (iv) 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. And it did not spread to the industrial North which had a diet rich in Vitamin B.

Similarly the rather unspecific American epidemic of lung cancer-empysema-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:**

- (i) rose steadily, not exponentially,
- (ii) were completely non-randomly biased 85% in favor of males,

(iii) have followed first the over-use of recreational drugs, and then the extensive use of anti-AIDS-viral drugs (Duesberg & Rasnick, 1998),

(iv) do not manifest in one or even just a few specific diseases typical of microbial epidemics,

(v) do not spread to the general non-drug using population.

AIDS manifests in a bewildering spectrum of 30 non-specific, heterogeneous diseases. This is consistent with the heterogeneity of the causative toxins. 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).

**The above summary indicates that American and European AIDS epidemics exhibit the characteristics of diseases caused by non-contagious, chemical or physical factors NOT viruses.**

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## AFRICAN AIDS

### 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).

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.

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, for example:**

- (i) "weight loss over 10%,
- (ii) chronic diarrhea for more than a month,
- (iii) fever for more than a month,
- (iv) persistent cough,
- (v) generalized pruritic dermatitis,
- (vi) recurrent herpes zoster (shingles),
- (vi) candidiasis oral and pharyngeal,
- (vii) chronic or persistent herpes,
- (viii) cryptococcal meningitis,
- (ix) 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 "estimates", 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 would be 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**

(i) clinically because of its total lack of a clinical identity, nor

(ii) 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 be warned 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. 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.

Acknowledgment: I thank Charles Geshekter, professor of history, Cal State University Chico, Chico, California for advice and critical statistics (see the Geshekter posts on this

panel).

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## Books

**Oncogenes, Aneuploidy, and Aids:** A Scientific Life and Times of Peter H. Duesberg  
by Harvey Bialy (North Atlantic Books, 2004)

**Inventing the AIDS Virus**

by Peter H. Duesberg  
Foreword by Nobel Prize winner Kary Mullis  
(Regnery USA, 1996)

**AIDS: Virus or Drug Induced?**

A collection of 27 articles by scientists, independent scholars, and investigative journalists from Australia, Europe and the US, (Kluwer Academics Publishers, 1996)

**Infectious AIDS: Have We Been Misled?**

A collection of 13 articles by Duesberg, published in scientific journals between 1987 and 1996, (North Atlantic Books, 1995)

**AIDS: The good news is...**

by Duesberg and John Yiamouyiannis, (Health Action Press, 1995)

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***Oncogenes, Aneuploidy and AIDS: A Scientific Life & Times of Peter H. Duesberg.***  
**By: Harvey Bialy**

Published by The Institute of Biotechnology, National University of México, and distributed by North Atlantic Books, 2004, Berkeley, CA, 328 pages, ISBN 1-55643-531-2. \$19.95

**Iconoclast to the Max**

Review by George L. Gabor Miklos, *Nature Biotechnology*, Vol. 22, pp. 815-816, July 2004

In this authoritative and elegantly written book, Harvey Bialy exposes a microcosm of today's medical science in a blistering analysis of the history of modern cancer and AIDS research. An almost unique hybrid of scientific biography and autobiography, *Oncogenes, Aneuploidy and AIDS* is replete with Nobel laureates, editors of prestigious journals like *Nature* and *Science*, presidents of the USA and South Africa and 'colorful' characters such as "Honest Dollar Bill", and the "OncoMouse". But the central figure of Bialy's book is Peter Duesberg, a classical, no-nonsense U.C. Berkeley professor who has for more than twenty years presented data and interpretations to cancer and AIDS scientists that call into question the fundamental notions of causality they espouse and which represent the dominant, mainstream positions— that specific genes via mutations cause cancer, and HIV causes AIDS. The sadly predictable result of questioning these two sacred cows of modern biomedicine was the almost complete destruction of a once lofty professional standing. Of late, however, Duesberg's name has begun to undergo some significant reconstruction as Bialy makes clear in telling the fascinating and instructive story of his banishment from the High Table and his recent partial return to favor.

To this reader, Duesberg's situation suggests parallels with that of another cell geneticist, the Nobel Prize winner Barbara McClintock. For decades her work was ignored by all except the very few who understood that the ideas and data were persuasive and worth serious consideration even though they did not fit the existing fashion -- yet how right she turned out to be. The inescapable conclusion; clean data and perceptive, unbiased analysis win every time. Near the end of a chapter entitled "Good Mourning America", Bialy uses an analogy to an old television police series where

the LA cop Joe Friday continually reiterates "Just the facts, ma'am", to emphatically make this point.

*Oncogenes, Aneuploidy and AIDS* has other global themes such as how science *should* be done, and the prominent role of metaphoric language in popular and professional scientific writing. Bialy's method is to examine the most important review articles and scientific papers in both cancer and AIDS that Duesberg published between 1983 and 2003, and the responses to them in the journals. He does this by weaving the hard science with historical and personal reflections to produce a contextual fabric that makes the book appealing and comprehensible to even a non-specialist reader. As far as this reviewer is concerned, Duesberg gets the Big Picture correct on both cancer and AIDS because he demands the highest standards of data interpretation, something that is a common casualty in the cancer and AIDS fields where fame, stock options, potential blockbuster drugs, appearances on Larry King Live and the front cover of *Time* or *Newsweek*, often appear to take precedence. As the founding scientific editor of *Nature Biotechnology* and an early Ph.D. from the first department of molecular biology in the country at U.C. Berkeley, Bialy has a privileged position which he uses to impeccably demonstrate that Duesberg represents a golden era of molecular biology when there was no room for the shoddy over-interpretations, and unimpressive correlations that pass for some of today's cancer and AIDS "breakthroughs".

Despite being the past recipient of an Outstanding Investigator Grant from the U.S. National Institutes of Health, its most competitive and highly regarded award, and a member of the U.S. National Academy of Sciences since 1986, Duesberg became unfundable in parallel with his questioning of AIDS and cancer etiologies. But in testament to what makes America the epicenter of privately financed innovation, he succeeded in attracting support from a far sighted San Francisco philanthropist, and his ailing laboratory was rejuvenated. So real science continued, and together with Berkeley and University of Heidelberg collaborators, Duesberg produced a rigorous, quantitative genetic explanation of cancer that is based on massive chromosomal upheavals, a phenomenon called aneuploidy that for almost one hundred years has been known to be the most consistent genetic alteration associated with solid tumors. In its modern metamorphosis at the hands of Duesberg and his colleagues, this explanation of cancer has begun to receive serious and well deserved attention. The aneuploidy view is very different from the current mainstream one, in which cancers arise because of the stepwise accumulation of mutations in oncogenes and tumor-suppressor genes, sometimes assisted by mutator genes in a chromosomally normal human cell. The implications for drug development are also very different between the two modes of cancer genesis and herein lay the makings of an unavoidable clash that was almost as vicious as the more obvious one between viral and chemical causes of AIDS.

Unfortunately for the establishment position, it has so far proven experimentally impossible to produce cancer in normal, diploid cells by multiple mutational routes. To use a phrase repeated to devastating effect in the book ---- to what should be the shame of numerous and very public defenders of both oncogenes and HIV ---- while specific mutated genes have "told us many things that we did not know, they have thus far not provided the answer to the all important question of how". The mutation-cancer field is so befuddled, it will come as no surprise when the reader learns that the "guardian of the genome", the *capo de tutti capo* of anti-oncogenes, p53, was reclassified as a tumor -suppressor after being a *bona fide* dominant oncogene for over a decade. As Bialy writes in one of the must -read, "gracenote" annotations, mutated oncogenes and tumor -suppressors are about *processes* and have almost no individual value except in defined genetic backgrounds. They contribute to phenotypic endpoints that are only applicable in the context of a network perturbation. Thus, as is well known, but nevertheless conveniently ignored, a mutation in a tumor -suppressor gene may be associated with a high frequency of colorectal carcinomas in one genetic background, but the same mutation in a different genetic background yields a perfectly normal colon totally free of carcinomas.

For this reviewer, steeped in chromosomal mechanics, segmental aneuploidy and mutational profiles of eukaryotic genomes in different genetic backgrounds, the severe limitations of what

individual mutations can and cannot do, is based on rigorous and well tested experimental foundations. The mutational underpinnings of cancer, by contrast, as currently set out by mainstream cancer researchers, simply don't cut the mustard in either predictive value or clinical usefulness. Trying to prolong the lives of cancer patients based on research emanating from the academic and pharmaceutical sectors has made it clear that something is seriously amiss with current approaches. For example, after three decades of research, there has been no reduction whatsoever in the incidence of the major solid tumors of the breast, lung, prostate and colon. All we have to show for the effort is a massive clinical black hole into which hundreds of billions of dollars of public and pharmaceutical money continues to be poured. This situation has been extensively (and exquisitely) documented by a devastating recent article in *Fortune* magazine by Clifton Leaf, entitled, "Why we are losing the war on cancer". As Andy Grove, the Chairman of Intel pointed out to the magazine's editor, "It's like a Greek tragedy. Everybody plays his part, everybody does what's right by his own life, and the total just doesn't work". In this context, Duesberg's work on the importance of gross upheavals in the human genome in the etiology of cancers is of enormous significance, as the real clinical issue concerns the series of unstable network problems leading to metastasis. This metastatic dot is slowly assuming more prominence on the cancer radar screen, as the technology to examine the transcriptional outputs of single cancerous cells is being honed by pioneers such as Christoph Klein in Germany, and the methylation status of single cells is now being probed by Douglas Millar, one of the inventors of the basic methylation methodologies, in Australia.

It cannot be overemphasized that cancer is not really a disease of uncontrolled growth. Cancer cells often divide more slowly than their progenitors and metastatic cells often become arrested at ectopic sites in the body. Henry Harris, a distinguished early pioneer of the tumor suppression field at the University of Oxford, made this same point when he wrote in the pages of *Nature* recently, "It would reduce confusion considerably, if it could be agreed that cancer, in the first instance, is not a disease of cell multiplication, but a disease of differentiation" (as quoted in the book). The triumvirate of Bialy, Leaf and Harris is a lethal cocktail for conventional theories of cancer. Either you are blasted from a cocooned world, or one of the great paradigm shifts in medicine has just passed you by.

There comes a time when throwing money at a problem is counterproductive and what is required is more cortical horsepower. It is a corollary of what the Nobel laureate Sydney Brenner has been saying for decades and which he put in prose in a favorite essay, "Sillycon Valley Fever". Brenner's point is brutal in its simplicity. How can you perform academic or commercially relevant biology if you don't think deeply? If you don't have a coherent theory and if you are dependent upon sophisticated technologies and bioinformatic protocols that you don't understand, then your data interpretations are in the realm of voodoo science. It is painfully obvious by now that this is where many cancer and AIDS researchers have located themselves --- a conclusion attested to by the mortality rates of breast, lung, prostate and colorectal cancers and the mountain of contradictions in the scientific literature concerning presumed HIV pathogenesis, AIDS morbidity, mortality, epidemiology and demography. Having got it so wrong, they can't buy their way out of their self generated cul-de-sacs. The almost pathological obsession with gene-based solutions (cellular or viral), neat gene-based circuit diagrams, mutator genes and "Molecular Portraits" of cancers, has led to a medical science that has wasted a massive amount of resources and spawned a plethora of failed drugs.

*Oncogenes, Aneuploidy and AIDS* should be compulsory reading for those concerned with what the U.S. (and other Western) governments are buying when they spend public money on cancer and AIDS research. It should also be compulsory for pharmaceutical and biotech executives, since most of their potential targets for solid tumors are irrelevant entities that continue to clog drug development pipelines. Finally, it should be read by anyone who is interested in the way scientific theories develop and are shaped by historical circumstances.

In his detailing of the academic trials, tribulations and recent emerging triumphs of professor

Duesberg, Bialy provides a number of salient lessons. One of them is that something precious has been lost in our love affair with the technological marvels that permeate today's biomedical science. It is, after all, the human cortex that sets the standards of excellence. If those standards are compromised, we are on the inexorably downward slope of shallow thought and mindless turning of the millstone. The proposition is indeed a stark one, and it is a measure of Bialy's skill and artistry that he makes it thinkable.

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## **Inventing the AIDS Virus** by Peter H. Duesberg

Regnery USA 1996, 720 pages, ISBN 0-89526-470-6.

Also available in Italian "AIDS - Il Virus Inventato"  
(Baldini & Castoldi, 1998, ISBN 88-8089-743-8)

### **HIV does not cause AIDS... AIDS is not sexually transmitted... AZT makes AIDS worse, not better...**

So argues Dr. Peter Duesberg, one of the world's leading microbiologists, a pioneer in the discovery of the HIV family of viruses, and a member of the National Academy of Sciences.

Duesberg's evidence - revealed in top scientific journals but kept out of the mainstream press - raises questions the AIDS research establishment has so far declined to answer:

If HIV causes AIDS, why have thousands of AIDS victims *never* had HIV?

Why have hundreds of thousands who have had HIV - for many years - remained perfectly healthy?

Why does the discoverer of the HIV virus now claim it can not be the sole cause of AIDS?

Why has more than ten years of AIDS research - costing tens of billions of dollars - failed to show how (or even if) HIV causes AIDS or attacks the immune system?

With annual federal funding at more than \$7 billion, AIDS research is better funded than any other disease - including cancer. Yet it has also produced the least results. Why? Duesberg explains how the lure of money and prestige, combined with powerful political pressures, have tempted otherwise responsible scientists to overlook - even suppress - major flaws in current AIDS theory.

The answer? Not more funding for more flawed research. Instead, start with an open airing of all the facts and failures, then determine the real cause of the disease.

This book does both. For Duesberg's solution to the AIDS mystery is as convincing as his critique of the HIV theory - and could save hundreds of thousands of lives at risk today

*Peter Duesberg is professor of molecular and cell biology at the University of California at Berkeley, a pioneer in retrovirus research, the first scientist to isolate a cancer gene, and recipient of the Outstanding Investigator Grant from the National Institutes of Health. His articles challenging the HIV/AIDS hypothesis have appeared in scientific journals including The New England Journal of Medicine, Science, Nature, The Lancet, British Medical Journal, Proceedings of the National Academy of Sciences, and Cancer Research.*

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## Praise for INVENTING THE AIDS VIRUS:

*"At last! this is the book every AIDS-watcher has been awaiting, in which the most prominent and persistent critic of HIV as the cause of AIDS states his case most exhaustively and popularly. Duesberg massively documents and cogently argues... [his] positions... [t]his book is a milestone..."*  
Booklist, American Library Association

*"We know that to err is human, but the HIV/AIDS hypothesis is one hell of a mistake. I say this rather strongly as a warning. Duesberg has been saying it for a long time. Read his book."*  
Kary B. Mullis, Nobel Prize in Chemistry, 1993

*"[Duesberg] is absolutely correct in saying that no one has proven that AIDS is caused by the AIDS virus. And he is absolutely correct that the virus cultured in the laboratory may not be the cause of AIDS."*

Dr. Walter Gilbert, Nobel Prize in Chemistry, 1980, Hippocrates

*"Peter Duesberg has fought with courage and tenacity to drag a reluctant scientific community back to AIDS sanity since detecting falsehoods in the HIV story more than a decade ago. His efforts have earned him much personal abuse, but may ultimately save countless lives."*

Neville Hodgkinson, former editor of The London Sunday Times

*"Dr. Peter Duesberg's courage and his book **Inventing the AIDS Virus** will save millions of lives. He is not only one of the few people in the world qualified enough to see through the scientific 'AIDS scam,' but he is the only one who has sacrificed his career to save the world from it."*

Tony Brown, host of PBS' Tony Brown's Journal

*"Duesberg, whatever else you may think of him, is unquestionably a fierce and highly disciplined scientist of the old school. He does not accept incomplete hypotheses or 'guilt by association' as normal aspects of modern virology. His one-man, ten-year crusade against sloppy science in the battle against AIDS is completely told in this highly readable book."*

Richard Strohmman, Professor Emeritus, Cell & Molecular Biology, UC Berkeley

*"Our 13 years of direct experience gravely contradict the conventional dogma that HIV causes AIDS. We agree with Dr. Duesberg... Reading this book is a matter of life and unnecessary death!"*

Rev. Dr. Michael Ellner, President, Health Education AIDS Liaison

*"A well-credentialed scientist's hard-driving attack on the accepted view that AIDS is an infectious disease caused by HIV... The serious charges [Duesberg] makes deserve serious answers."*

Kirkus Reviews

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[Preface](#) by the publisher

[Associate Press](#) early announcement

[Professors review](#) of "Inventing the AIDS Virus" for California Monthly

Other Reviews of "Inventing the AIDS Virus"

[New York Times](#) with Duesberg's [reply](#)

[Orlando Sentinel](#)

[Boston Herald](#)

[Washington Times](#)

[Laissez Faire Books](#)

[New York Review of Books](#)

[Daily Telegraph](#) (London)

[The Record](#)

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## **AIDS: Virus or Drug Induced?**

**Peter H.Duesberg (Editor)**

Contemporary Issues in Genetics and Evolution vol. 5, Monograph, Kluwer Academics Publishers 1996, 365 pages ISBN 0-7923-3961-4.

Despite enormous efforts, over 100.000 papers and over \$22 billion spent by the US taxpayers alone, the HIV-AIDS hypothesis has failed to produce any public health benefits, no vaccine, no effective drug, no prevention, no cure, not a single life saved. Is the science system to be blamed? Has science failed to reveal the truth about AIDS?

In *AIDS: Virus or Drug Induced?* Two dozen scientists, scholars and journalists have investigated the status quo of AIDS research. Most of them have questioned the HIV-AIDS hypothesis before, but have since been censored, and sociologically excluded from AIDS research, politics and journalism. Here they are united for the first time to put on trial the HIV-AIDS hypothesis.

There are those who acquit HIV entirely. Others who make a case for HIV as a necessary, but not a sufficient cause of AIDS. And one medical scientist who, together with the huge AIDS literature, defends the hypothesis that HIV is sufficient to cause AIDS.

The book convincingly reveals that the scientific method could very well find a solution to AIDS, but only if ideas can be exchanged freely and if the HIV monopoly can be broken.

*AIDS: Virus or Drug Induced?* Illustrates that the solution to AIDS could be as close as one of several, very testable and very affordable alternatives to the unproductive HIV-AIDS hypothesis.

**CONTENTS AND CONTRIBUTORS:** E. Papadopulos-Eleopulos et al., ['A critical analysis of the HIV-T4-cell-AIDS hypothesis'](#) (*Genetica* 1995, vol.95 pp.5-24.)

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M.D. Zaretsky, 'AZT toxicity and AIDS prophylaxis: is AZT beneficial for HIV+ asymptomatic persons with 500 or more T4 cells per cubic millimeter?' (*Genetica* 1995, vol.95 pp.91-101.)

D.T. Chiu, P.H. Duesberg, 'The toxicity of azidothymidine (AZT) on human and animal cells in culture at concentrations used for antiviral therapy' (*Genetica* 1995, vol.95 pp.103-109.)

R.S. Root-Bernstein, 'Five myths about AIDS that have misdirected research and treatment' (*Genetica* 1995, vol.95 pp.111-132.)

R.S. Root-Bernstein, S. Hobbs de Witt, 'Semen alloantigens and lymphocytotoxic antibodies in AIDS and ICL' (*Genetica* 1995, vol.95 pp.133-156.)

H.W. Haverkos, D.P. Drotman, 'Measuring inhalant nitrite exposure in gay men: implications for elucidating the etiology of AIDS-related Kaposi's sarcoma' (*Genetica* 1995, vol.95 pp.157-164.)

B.J. Ellison, A.B. Downey, P.H. Duesberg, 'HIV as a surrogate marker for drug use: a re-analysis of the San Francisco Men's Health Study' (*Genetica* 1995, vol.95 pp.165-171.)

M. Craddock, 'A critical appraisal of the Vancouver men's study'

G.T. Stewart, 'The epidemiology and transmission of AIDS: a hypothesis linking behavioural and biological determinants to time, person and place' (*Genetica* 1995, vol.95 pp.173-193.)

K.B. Mullis, 'A hypothetical disease of the immune system that may bear some relation to the Acquired Immune Deficiency Syndrome' (*Genetica* 1995, vol.95 pp. 195-197.)

P.H. Duesberg, 'How much longer can we afford the AIDS virus monopoly?'

F. Harris, 'AIDS and good theory-making'

S. Lang, 'HIV and AIDS: Questions of scientific and journalistic responsibility'

N. Hodgkinson, 'Cry, beloved country; How Africa became the victim of a non-existent epidemic of HIV/AIDS'

P. Johnson, ['The thinking problem in HIV science'](#)

J. Lauritsen, ['HIV symposium at AAAS conference'](#)

T. Bethell, 'AIDS and poppers'

J. Lauritsen, ['NIDA meeting calls for research into the poppers-Kaposi's sarcoma connection'](#)

J. Lauritsen, 'The incidence quagmire'

C. Farber, 'The HIV test'

S. Lang, 'To fund or not to fund, that is the question: proposed experiments on the drug-AIDS hypothesis'

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**AIDS: The good news is HIV doesn't cause it; the bad news is recreational drugs and medical treatments like AZT do**  
**by Peter H. Duesberg and John Yiamouyiannis**

Health Action Press 1995

Also available in German "AIDS" (Michaels-Vertr., Peit., 1998, ISBN 3895392847)