African population doubles during HIV-AIDS era, from 400 to 800 million

AIDS cui bono - conference
Vienna, July 16-17, 2010

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Why don’t we have the answers to these questions?

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AIDS - Knowledge and Dogma

Why has there been no AIDS epidemic in Europe or North America, despite repeated predictions over the last 25 years?

Where is the vaccine against HIV that’s been “just around the corner” since 1985?

What’s happened to the tens of billions of dollars invested in AIDS over the last 25 years?

How did Africa manage to double its population in the last two decades while we were told the continent was drowning in disaster?

How did Uganda become one of the fastest growing countries today, even though it’s been hit harder by HIV/AIDS than any other African nation? And how did it overcome the epidemic without AIDS drugs?

Why has the discoverer of HIV, Prof. Luc Montagnier, declared that “someone with a good immune system can get rid of HIV within a few weeks”?

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Have you ever asked yourself these or other questions? Do you wonder why there are so few critical comments about HIV/AIDS in the public discourse? Are you curious to know who’s profiting from the HIV/AIDS hysteria? Aids - Cui bono?

Do you suspect we might have been misled or fooled with HIV, just like we were fooled with ‘swine flu’, ‘bird flu’, ‘mad cow disease’ and other epidemics that failed to materialize?

If so, we invite you to an international conference in Vienna – a completely independent one from the official AIDS conference.
Because we have to “believe” AIDS is something that it is not

We believe – or better are taught to believe that AIDS is the newest one of the great microbial epidemics to which the plague, the Flus and choleras and polios belong.

This belief has generated numerous paradoxa – unanswerable questions - like those listed in the cui bono poster.

But there are no paradoxa in nature – only flawed hypotheses.

And the HIV-AIDS hypothesis is perhaps the biggest one of those in scientific history.
HIV-AIDS and the germ theory of disease

The hypothesis that the Human immunodeficiency Virus (HIV) causes AIDS is generally presented as a modern example of the classical germ theory of disease (Durban Declaration, 2000).

Since Koch and Pasteur in the 1880s, and before them Semmelweis in Vienna, the germ theory has explained and helped to prevent numerous fatal epidemics of pathogenic microbes and viruses.

Classical examples are the medieval plagues, the global Flu epidemic of 1918 and the polio epidemics of the 1950s and 1960s.

The “AIDS virus” was added to this list in 1984, just a day after the announcement of its discovery on a press conference.
Questions persist about viral AIDS

But, despite unprecedented propaganda for the virus-AIDS hypothesis since 1984, questions persist that cannot be answered in terms of the classic ‘germ theory’ of disease.

Several are pointed out on our “AIDS cui bono” poster.
Asking those questions is not popular in the AIDS establishment

Here I will try to focus on two of these questions:

- Why are huge AIDS epidemics reported in Africa, but not in the US, Europe and Asia?

- Why is it not published that the African population doubled in the presumably devastating HIV-AIDS era?

A first attempt to answer these questions with my colleagues Fiala, Bauer and two others in Medical Hypotheses was just censored, amid other sanctions, by the publisher, Elsevier.
The germ theory of disease

So here is what we should expect, if viral AIDS were a modern example of the germ theory of disease:

- A new pathogenic virus or microbe manifests itself by causing an epidemic of new microbe-specific illnesses and deaths (i.e. new per population).

- Such an epidemic spreads exponentially in a population within weeks to months, because each newly infected carrier infects dozens of others.

- Thus a germ epidemic runs like a biological chain reaction until all susceptible subjects have either acquired immunity or are killed, when the epidemic declines exponentially.

- The resulting bell-shaped epidemiological curves of illnesses and deaths were first described for a plague in London in 1665.
Bell-shaped curve of first recorded plague epidemic, London 1665

This plague was the first germ epidemic that was statistically recorded. Its exponential rise and subsequent fall over weeks form the classical bell-shaped curve, typical of all new germ epidemics (Anderson & May).
Classical bell-shaped Flu epidemic of 1918 in the US and Europe

Note: Abscissa in months. Mortality increased 6-fold.
Seasonal polio epidemic, US 1948
Biological basis of fast kinetics of germ epidemics

The fast kinetics of such biological chain reactions are the results of the short incubation periods between germ-infection and disease.

During these incubation periods a few infecting germs multiply at high rates to billions of progeny.

Bacteria double in only 30 minutes.

Viruses multiply 100- to 1000-fold in 8 to 24 hours.

And the individual diseases coincide with maximal multiplication of the germ.

Thus there is no ‘slow virus’ or ‘latent virus’ causing fatal disease.
HIV-AIDS hypothesis predicts global epidemic since 1984

In awe of the popular germ theory –

The New York Times names a newly discovered retrovirus, “AIDS virus”, in 1984, just because anti-viral antibodies were found in 1 of 3 AIDS patients.

Without delay, the US National Academy of Sciences and the Institute of Medicine appoints a blue ribbon committee to confront the expected epidemic. The committee warns:

“There are 1 to 1.5 million Americans currently infected with HIV. Of these, 20 to 30 percent are expected to develop AIDS by 1991. AIDS cases ... acquired through heterosexual contact will increase from 1100 in 1986 to almost 7000 in 1991. ... Pediatric AIDS cases will increase almost 10-fold during the next 5 years to more than 3000 cumulative cases ...” (Confronting AIDS, 1986).
No bell-shaped AIDS epidemic anywhere since 1984 … except in Africa?

But, no “heterosexual” or “pediatric” epidemics occurred in the US and Europe or Asia and South America to this very day.

AIDS remained restricted to two major risk groups, intravenous drug users and male homosexuals using psychoactive aphrodisiac and anti-HIV drugs.

Except perhaps in Africa?
Harvard study claims HIV killed 1.8 million South Africans between 2000 and 2005

In 2008, at last, a Harvard study by Essex, Chigwedere et al. announced that HIV had killed 1.8 million South Africans between 2000 and 2005 (JAIDS, 2008).

Their “estimates” were based on statistics from the World Health Organization (WHO).

Moreover, Essex and Chigwedere complained that at least 330,000 of those 1.8 million “estimated” deaths could have been prevented by anti-HIV drugs, specifically the DNA chain terminator AZT and a new DNA inhibitor Nevirapine.
Biological paradox: How could HIV cause fatal epidemics only in Africa?

If correct, the Harvard discovery would have found the first general HIV-AIDS epidemic since the discovery of the “AIDS virus” in 1984.

However, this discovery also defines a biological paradox!

How could HIV kill 1.8 million South Africans, although it failed to cause such epidemics in the US, Europe and Asia?

Why was there no such AIDS epidemic in the US with 1 million HIV-positives since 1985, many of which “African-Americans”? 
To solve Essex’s African paradox, we asked two questions

1) What is the evidence for the huge losses of South African lives from HIV claimed by Essex, Chigwedere et al?

2) What is the evidence for the claims that South Africans would have benefited from anti-HIV drugs?
The WHO, Essex’s source of information, does not report losses of 1.8 million

Our first surprise in trying to solve the paradox of the new African HIV-AIDS epidemic was:

The WHO does not list any specific numbers for “Reported HIV cases” and “Reported AIDS cases” of South Africa in their epidemiological “Fact Sheet” for the period from 1996 until 2007 (see Figure 2, next).
Reported AIDS- and HIV cases from South Africa according to the WHO

### Reported AIDS cases

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Source:

### Reported HIV cases

A case of HIV infection is defined as an individual with HIV infection irrespective of clinical stage confirmed by laboratory criteria according to country definitions and requirements.

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Source:

Note: In some instances, the number in the total column is not the sum of the individual years due to differing reporting, estimation processes or available data.
Uncertainties about WHO statistics

Indeed, we were not the only ones to wonder about WHO statistics.


“AIDS programs developed by international agencies and faith based on organizations have been and continue to be more socially, politically, and morally correct than epidemiologically accurate.”

More recently the WHO has also been accused of manipulating epidemiological numbers of a new Flu epidemic.
In contrast to claimed losses, South Africa gained 3 million between 2000 and 2005.

Next, we searched South African and American statistics for evidence of 1.8 million AIDS deaths.

Surprisingly again – *Statistics South Africa* and the US Census show steady annual gains of 500,000 for the period from 2000 to 2005. Instead of declining from Essex’s HIV-epidemic, the SA population had increased by 3 million from 44.5 to 47.5 million (See Table 1).

Nevertheless, the National Department of Health South Africa also reported that 25 to 30% South Africans had antibodies against HIV between 2000 and 2005 (Table 1).
Statistics South Africa & the US Census report gains of 3 million from 2000 to 2005

<table>
<thead>
<tr>
<th>Year</th>
<th>Population (a) x 10^-3</th>
<th>HIV+ (b) %</th>
<th>HIV-Death (c) x 10^-3</th>
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(a) Statistics South Africa and US Census Bureau [37, 38, 39].
(b) National Department of Health South Africa [42].
(c) Statistics South Africa [38, 46].

*not reported because HIV-deaths were below 10th rank.
Harvard study “overestimates” HIV-mortality at least 30-fold

Ever trying to accommodate Harvard (!), we asked whether SA statistics confirmed 300,000 annual “AIDS-deaths”.

It is theoretically possible, that SA gained 500,000 per year and also lost 300,000 per year from “AIDS-deaths”.

But surprisingly again, the mortalities attributed to HIV by *Statistics South Africa* were consistently low.

Between 2000 and 2005 they averaged 10,000 per year.

So the Harvard study “over-estimated” AIDS-deaths 30-fold! (See Table 1, column 4).
Net discrepancy between Harvard study and Statistics SA & US census

Discrepancies between SA population statistics and Harvard “AIDS epidemic”, 2000-2005

<table>
<thead>
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<th>Harvard study</th>
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<td>SA Statistics, US Census</td>
<td>+ 3,000,000</td>
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Discrepancy = 4.8 million
Essex’s AIDS epidemic also incompatible with germ theory

The Harvard study claims annual losses of 300,000 over 6 years, 2000-2005, as evidence for a new viral HIV-AIDS epidemic.

In contrast, the germ predicts that new infectious pathogens cause bell-shaped morbidity and mortality curves that rise and fall exponentially during a few months (see 1918 Flu).

A steady mortality over years is, instead ony compatible with life style associated non-microbial (smoking) and opportunistic endemic microbial diseases (TB).

We conclude that Essex’s SA AIDS-epidemic is numerically flawed and incompatible with the germ theory.
We propose that HIV is not pathogenic in S.A.

Since the South African population increased by 500,000 per year from 2000 to 2005, although it was 25 to 30% HIV-positive, we propose that HIV is a non-pathogenic passenger.

To test this proposal we have compared the population growth curve of South Africa since 1980 with the curve showing annual HIV-prevalence.
Population growth independent of HIV-prevalence

Our proposal predicts that South African growth curve is independent of HIV-prevalence.

By contrast, Essex et al. predict that growth declines and mortality increases in proportion with HIV-prevalence.

To decide between these two predictions we have plotted the South African growth curve since 1980 vs. HIV-prevalence [Figure 3, next].

As can be seen South African growth formed a monotonic trajectory since 1980 — independent of HIV-prevalence. So HIV is a passenger.

The growth trajectory was so steady that we could have predicted the population growth of 3 million between 2000 & 2005 before it happened.
Statistics South Africa & US report steady and positive growth trajectory, despite HIV
Ugandan population also doubles during HIV-AIDS era

To determine whether the steady population growth of South Africa, despite a concurrent, presumably new HIV epidemic, is exceptional or typical of other African countries, we have next investigated the effect of HIV on the population of Uganda.

As shown in Figure 4A, the Ugandan population also doubled during the HIV-AUIDS era, although the New Engl. J. Med described it as the epicenter of the African AIDS epidemic in 1990.

This also happened despite a 5–15% prevalence of HIV-antibodies (Figure 4B)
We conclude that Ugandan growth is also independent of HIV, much as it is in South Africa.
Sub-Saharan Africa doubles from 400 to 800 million during HIV-AIDS era

In an effort to raise our investigation above variations among AIDS epidemics of different African countries, we asked next whether the population of Sub-Saharan Africa as a whole was decreasing or increasing – in the face of the widespread prevalence of antibodies against HIV.

Again, we found in the statistics of the World Bank that the population of Sub-Saharan Africa as a whole had doubled during the HIV-AIDS era, from 400 million in 1980 to 800 million in 2007.

This confirms and extends our conclusion that HIV is non-pathogenic.
Indeed, one could argue that HIV has doubled the population of Africa!
Can HIV-positive South Africans “benefit” from anti-HIV drugs, as Essex et al. claim?

Finally, we call into question Essex et al’s claim that anti-viral drugs “benefit” HIV-positives without and with AIDS. There are three reasons for our reservations:

1) The germ theory of viral disease.
2) The inherent toxicities of anti-HIV drugs.
3) The evidence that HIV is not pathogenic.
Germ theory predicts inevitable toxicity of anti-viral drugs

The genetic complexity of all viruses is much too low to synthesize their nucleic acids and to make their proteins on their own (Crick & Watson, 1956).

Therefore, all viruses are obligatory parasites, depending on the host cell for the synthesis of their DNAs, RNAs and proteins.

It is for this reason that all inhibitors of virus replication must be inhibitors of cell replication – and thus inevitably toxic.

Even nature has never evolved any anti-viral drugs – except anti-viral immunity.
Does anti-HIV toxicity justify inevitable cell toxicity?

Although anti-viral drugs are inevitably toxic, it could be argued that they are less toxic to the cell than to the AIDS virus – much like the argument that is made for cancer chemotherapy.

But even this argument cannot justify treatments of HIV-antibody-positives with inhibitors of DNA synthesis, like AZT and Nevirapine that Essex et al. propose.

This is because (a) HIV-specific DNA, RNA and protein synthesis are already inhibited in antibody-positive people and thus no targets for inhibition, and (b) because there is no proof that HIV is pathogenic.

We conclude that there is no theoretical basis for the claims that HIV-positives “benefit” from anti-HIV drugs. Practical tests confirm the theory (next).
Controlled trials confirm the toxicity of anti-HIV drugs

The first, placebo-controlled trial of AZT, carried out in the US in 1987, showed life-threatening anemia in 24% and neutropenia in 16% within several weeks after treatment of AIDS patients.

In addition, “serious adverse reactions, particularly bone marrow suppression, were observed. Nausea, myalgia, insomnia, and severe headaches were reported more frequently by recipients of AZT; macrocytosis developed within weeks in most of the AZT group”.

Thirty of 149 AZT recipients could only be kept alive by blood transfusions.

By 21 months 42% of the AZT group and 35% of the control group, who possibly also died because they received AZT on a “compassionate basis”.

7/17/10
AZT does not prevent AIDS and increases mortality 25%

In 1994 the largest, placebo-controlled study of its kind, the British-French Concorde study, found that AZT is unable to prevent AIDS and increases the mortality of recipients by 25%.

In view of this the authors concluded, “The results of Concorde do not encourage the early use of zidovudine (AZT) in symptom-free HIV-infected adults.”
Anti-HIV drugs induce “non-AIDS-defining” diseases

Recent studies also demonstrate that about half of all HIV-positive American AIDS patients treated with anti-viral drug cocktails including AZT die from non-AIDS-defining diseases.

For example Reisler et al. observed in 2003,

“The most common grade 4 events [“serious or life-threatenining events that are not AIDS defining”] were liver related (148 patients, 2.6 per 100 person-years). Cardiovascular events were associated with the greatest risk of death (hazard ratio = 8.64; 95% CI: 5.1 to 14.5). The first grade 4 event and the first AIDS event were associated with similar risks of death, 5.68 and 6.95, respectively.”

El-Sadr et al. state in 2006, “an important secondary end point was major cardiovascular, renal, or hepatic disease.”
Nevirapine to prevent mother-child transmission of HIV

Chigwedere et al. recommend Neviparine as prophylaxis of mother-to-child transmission of HIV.

But, the NIH (National Institutes of Health) Treatment Guidelines advise,

“... the risk of several non-AIDS-defining conditions, including cardiovascular diseases, liver-related events, renal disease, and certain non-AIDS malignancies is greater than the risk for AIDS in persons with CD4 T-cell counts >200 cells/mm3".
Final recommendation

In view of -

1. The inevitable toxicity of anti-HIV drugs;

2. The evidence that HIV is not pathogenic and not biochemically active in antibody-positive people and thus not even susceptible to inhibition by drugs;

We conclude that anti-viral drugs are not beneficial for the prevention and treatment HIV-AIDS.

Limited applications might provide, however unspecific, serendipitous benefits against some AIDS-defining and non-defining diseases and cancers - much like mercury and arsenic against syphilis.

Our colleague Koehnlein has described this yesterday.

To advance this important experience to science, we propose to test anti-HIV drugs in animals, rather than humans, with and without AIDS-defining diseases.