DECONSTRUCTING DUESBERG

A Critique of

HIV-AIDS hypothesis out of touch with South African AIDS – A new perspective

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From an epidemiological point of view, the intrinsic logic of Peter Duesberg's passenger virus theory of HIV is the same as the logic of the pathogenic virus theory of HIV. Since both rely on the same epidemiological data and the premises on which those data are presented, Duesberg cannot argue his passenger virus theory without lending support to the HIV = AIDS theory, and conversely he cannot critique the HIV = AIDS theory without exposing the flaws in his own passenger virus theory. Duesberg's latest paper, a reply to Chigwedere et al. *Estimating the Lost Benefits of Antiretroviral Drug Use in South Africa*[1], is a perfect illustration of this self-imposed dilemma.

The stated aim of the Duesberg paper[2] is to (1) question the evidence for the huge losses of South African lives from HIV claimed by the Chigwedere study (2) question the evidence that South Africans would have benefited from anti-HIV drugs.

It is important to understand Chigwedere et al. are not contributing new facts. They merely use figures from UNAIDS and other official sources to calculate loss of life. A major weakness of the Chigwedere et al. paper is the sweeping assertions made to validate this approach. For instance:

Except among very few scientists, such as Peter Duesberg, the scientific community has accepted HIV as the cause of AIDS for more than 20 years. (6) HIV satisfies all 3 of Koch's postulates, the traditional standard of infectious disease causation, (7) and all of Sir Bradford Hill's epidemiological guidelines for assessing causality.(8)

The references given are:

6. Essex M, Mboup S. The etiology of AIDS. In: Essex M, Mboup S, Kanki PJ, et al, eds. AIDS in Africa. 2nd ed. New York, NY: Kluwer Academic/ Plenum Publishers; 2002:1–10.

7. Cohen J. Fulfilling Koch's postulates. Science. 1994;266:1647.

8. Aschengrau A, Seage GR. The epidemiologic approach to causation. In: Essentials of Epidemiology in Public Health. Sudbury, MA: Jones & Bartlett; 2003:375–401.

Duesberg et al. accept these basic references (one of them to a science journalist) without challenge, and in doing so concede all the ground on which Chigwedere et al. stand. Their strategy is narrowly focused on showing that epidemiological data show

1 www.aids.harvard.edu/Lost_Benefits.pdf

2 www.tig.org.za/Duesberg_2009.pdf

a disconnect between the number of HIV positives and the number of HIV deaths in South Africa.

Inexplicably, however, Duesberg et al. do not present the HIV prevalence rates Chigwedere et al. are relying on. Instead they premise their central argument on antenatal clinic statistics that inflate the estimated HIV prevalence in South Africa from 5.5 million to 12 million. The adjusted 5.5 million was the figure used by Chigwedere et al. when performing their calculations. The case can therefore not be made that Duesberg et al. merely accept the numbers used by Chigwedere et al. for the sake of argument.

Duesberg et al. use their inflated HIV statistics, abandoned even by the enthusiastic HIV promoters in UNAIDS, to arrive at their desired very low AIDS mortality rate relative to the high HIV prevalence. They then present this as evidence for Duesberg's Harmless Passenger Virus theory.

Duesberg et al. arrive at an even lower AIDS mortality rate by uncritically accepting the cause of death stated on South African death certificates. However, it has long been observed that AIDS is rarely given as the cause of death because of associated stigma, uncertainty about HIV status etc. It is therefore vital to address this argument, unless one merely wishes to put one set of numbers up against another. But Duesberg et al. fail to do so, thus laying themselves open to the charge that they are ignorant of the facts on the ground.

The antenatal clinic statistics given by Duesberg et al show a relatively stable HIV prevalence of around 12 million for the period in question, 2000-2005. They interpret this as evidence in favour of Duesberg's hypothesis that HIV is a mainly perinatally transmitted virus, endemic to South Africa:

It is consistent with the passenger virus-hypothesis that HIV (i) is naturally transmitted most effectively from mother to child, much like all other retroviruses [10], (ii) is asymptomatic for up to 25 years (since it is known) in persons free of chemical AIDS risks [10] including HIV-positive persons from the US Army [21], (iii) has remained ***epidemiologically stable, at about 25% to 30% in South Africans (Fig. 1b)****, at about 5% in Uganda (Fig. 2C, and [16]), and at about 0.3% (1 million in 300 millions) in America since 1985.* (Our emphasis)

One does not have to be an epidemiologist to see that Fig. 1b shows anything but epidemiological stability. The curve rises sharply from near zero in 1990 to 30% in 2005, but Duesberg et al. choose a small segment of the curve and call it epidemiologically stable because there is "only" a 20% increase in prevalence, from 25% to 30%, between 2000-2005.

Duesberg et al. compare the South African data with Uganda, where, according to their figures, HIV prevalence increased from 5.8% in 1989 to 13% within the next year (!), then decreased to 5% by 2006. Duesberg et al. call Uganda's HIV rate epidemiologically stable as well.

Even if we accept a stable Ugandan HIV prevalence at 5% and a stable South African prevalence at 25-30%, the implied passenger virus argument is still as improbable as the HIV/AIDS hypothesis, and for the same reasons. No cogent explanation is offered

for the huge differences in the cited prevalence rates of the supposedly endemic, perinatally transmitted virus, ranging from 5% in Uganda to 30% in South Africa. This compels the reader to ask of Duesberg et al. the question previously asked of racist HIV promoters: What are those South Africans doing to each other that Ugandans are not?

If Duesberg et al. in retrospect try closing the gap a bit by adopting the adjusted 5.5 million South African HIV prevalence that every expert accepts, they still face the task of explaining why the prevalence rate is so much higher among pregnant women than the rest of the population. How this can be done without discrediting the tests, and thereby all the statistics Duesberg et al. rely on, is an open question.

Duesberg et al. suggest that South African HIV rates have been epidemiologically stable at 25-30% in South Africa not only between 2000-2005, but for decades, and that the soaring prevalence is an illusion created by an epidemic of HIV testing:

The passenger-HIV hypothesis also offers the simplest explanations for the discrepancies between the massive population growths and the presence of the new reportedly devastating HIV epidemics in South Africa (Figs. 1 and 2). This explanation holds that HIV is a long-established, non-pathogenic passenger virus, neutralized by antibody after asymptomatic, perinatal or non-perinatal infections (just like all other human and animal retroviruses) [10]. The perceived novelty of the HIV epidemics would then reflect a novel epidemic of HIV-testing.

The references for the claim that there has indeed been such an epidemic of HIV testing in South Africa are the Durban Declaration and co-author Henry Bauer's book, *The Origin, Persistence and Failings of the HIV Theory*. Bauer is not a virologist or epidemiologist, nor is he an authority on African studies. His book deals with the US HIV statistics rather than the South African ones, and he focuses more on the flawed computer models used to estimate HIV rates the world over than on epidemics of HIV testing in South Africa.

These flawed models, based on unadjusted antenatal clinical statistics, are the very same that Duesberg et al. rely on to arrive at their own steady prevalence rates. In other words, by accepting the raw antenatal clinic statistics, Duesberg's co-authors contradict their self-references.

Bauer argues in *The Origin, Persistence and Failings of the HIV Theory* that HIV has never been isolated, and that the US HIV statistics show conclusively that the HIV tests do not measure a sexually transmissible agent. Another co-author, Christian Fiala, has previously co-authored two papers with the Perth Group, explicitly supporting their view that HIV has not been isolated, that there is no gold standard for the HIV tests, and that all the proteins said to belong to HIV are of cellular origin. One wonders how Bauer and Fiala will explain their contradictory positions when debating mainstream HIV experts in public, or worse under cross-examination in a courtroom.

All HIV prevalence and incidence estimates are extrapolations from sample material. Duesberg et al. offer no explanation in their paper for why more testing would necessarily mean increasingly higher extrapolated numbers. Neither do they explain

why the 2000-2005 estimates would be more reliable than, say, the 1993 -1998 estimates.

It stands to reason that increases in testing and improvements of models over time would produce more accurate results. But if that is the implicit argument, one wonders why Uganda was chosen as a parallel. The reader is expected to accept without explanation that the early models inflated Uganda's stable 5% HIV prevalence to 13%, while in South Africa the opposite was the case, in which HIV prevalence rose from near zero to 30%.

Were radically different methods and models used for these two countries? Duesberg et al. do not tell us.

Assuming for argument that the passenger virus conclusion is valid, what have Duesberg et al. achieved at this point? They have demonstrated that Chigwedere et al. are relying on flawed computer models that underestimate early South African HIV prevalence. Because they have failed to anticipate the obvious counter-arguments regarding unreliability of death certificates, Duesberg et al. have simply confirmed the validity of the tests and the validity of the current computer models. They have even explained why HIV death rates have not had a clear and increasing impact on population growth between 2000-2005 (which is one of the Duesberg paper's core arguments – see below), since the virus is long established, according to them. This is hardly a devastating blow to the HIV/AIDS construct.

Duesberg et al. choose to zoom out further and argue that the population growth in South Africa has been a steady 500,000 per year since the 1990s, before which it was 1 million, so the impact of an increasing HIV prevalence is not evident. For some reason they do not think it important to account for how an increasing South African population can suddenly experience a 50% decrease in population growth, followed by a steady growth rate. Nor do they seem to be aware that population growth is not calculated by counting every head once a year. The models that estimate population growth are typically adjusted for various factors, including presumed impact of HIV/AIDS.

These things taken into consideration, it is far from clear how they can argue that:

there is no statistical evidence for the loss above normal mortality of 300,000 lives per year or 1.8 million total lives from 2000 to 2005, as the Harvard study claims. ***The steady growth trajectory would have dropped from 3 million to 1.2 million during that time*** (Our emphasis)

If the models calculating population growth are already adjusted for HIV/AIDS impact, one can hardly use them to disprove this impact. But even if the figures were not adjusted, one wonders why Duesberg et al. have chosen to build their argument almost exclusively on the steady 500,000 population growth rate, when a juxtaposition with other sources makes a much stronger case for them. For example, in a publication dated 2004-2005, the US Census Bureau's "With AIDS" estimates show *negative* population growth for South Africa after 2003, at -0.075 for 2004 and -0.235 for 2005.

In response to the excess HIV-related child mortality calculated by Chigwedere et al., Duesberg et al. write:

But estimated losses of 3000 to 30,000 among 1 million newborns (.3% to 3%) are difficult to detect statistically, and are even more difficult to attribute to HIV, because all AIDS-defining diseases are previously known, HIV-independent diseases called AIDS only in the presence of antibody against HIV [10,11]. In view of this one wonders whether the Harvard study was aware of the South African vital statistics, and whether it took into consideration the difficulties of telling HIV-positive from negative AIDS-defining diseases.

The cited .3% to 3% out of 1 million newborns may not sound like much, but 3% amounts to more than half the estimated annual infant mortality (5%) in South Africa. Fifty percent of a given whole is usually statistically detectable, so one is, at best, left wondering what the point is here. The last part of the argument is equally mysterious: Why does it matter if it is difficult to tell HIV-positive from HIV-negative deaths? Unless Duesberg et al. want to concede that HIV can cause AIDS in some cases, the argument is meaningless. The basic premise of Chigwedere's calculation is that infant mortality *ceteris paribus* has increased in the HIV era. This premise is not addressed, presumably because Duesberg et al. think it has already been answered by the steady population growth argument.

However, raw estimates of the total population do not tell us much about what happens in the various demographics. It would therefore have been a better strategy for Duesberg et al. to have availed themselves of some of the brilliant work already done by other dissidents to point out that:

Assumptions made about the effectiveness of chemotherapy drugs, sexual behaviour change and other communication programs and of condoms in preventing transmission of a fatal disease among children cannot be based on the statistical evidence reported in the HSRC surveys.

And:

There is no statistical evidence in the HSRC surveys of any correlation between the HIV measurement and later mortality among children.[3]

Concerning "normal mortality", the annual South African death rate increased from 317,000 in 1997 to 605,000 in 2006, according to official figures. It is notoriously difficult to determine precisely which factors are at play behind such statistics. The impact of population growth, migration, death registrations, changing living conditions, changing computer models, changing demographics and a plethora of other factors go into these figures. "Bird's-eye view" statistics of the kind Duesberg et al. are relying on are meaningless without further analysis. That is why one wonders why they have not addressed issues such as these:

(…) *data show that between 1997 and 2004, the death rate among men aged 30-39 more than doubled, while that among women aged 25-34 more than quadrupled. The changes are even more pronounced when deaths from natural causes only are*

3 Chris Rawlins; "Analysis of HIV Prevalence 2008 Survey" www.tig.org.za/HSRC_stats_Rawlins.pdf

examined. Over the same period there was relatively little change in the death rates among people aged over 55 and those aged 15-20. (Avert.org/Statistics South Africa)

If Duesberg et al. were aware of these challenges, they chose to ignore them. They also seem ignorant of Rodney Richards's work[4], detailing how many of these issues can be explained by factors such as improvements in death registration. Dr. Richards also demonstrates the non-correlation between HIV prevalence and AIDS deaths in individual South African provinces. It is hard to imagine that Duesberg et al. would have overlooked these well-documented discrepancies in official statistics if they were not so strongly committed to arguing the Passenger Virus theory.

In order to refute the second leg of the Chigwedere et al argument, that the benefits of ARVs outweigh the risks, Duesberg et al. start with an argument relating to the lacking evidence that HIV synthesizes DNA *in vivo*, because, they say, it is "suppressed" by antibodies and is inactive.

This is a powerful argument; it is also an old one, originally based on the understanding of HIV in the late 1980s. Since then it has been claimed that HIV infection is never completely suppressed, that HIV "hides in secret places" in the body, and that tests such as "viral load" are indirect proof of this. Inexplicably co-authors Duesberg and Rasnick, who possess qualifications in relevant fields, do not take the opportunity to examine these arguments.

Instead they call AZT a DNA chain terminator, thereby disregarding the work of the Perth Group, notwithstanding that co-author David Rasnick has personally been made aware of it[5]. They even promote AZT from a "failed cancer drug" to a cancer drug in current use:

AZT is used against cancer, since cancer cells typically make more DNA than normal cells and are thus more susceptible to DNA chain-termination than most normal cells.

Duesberg and Rasnick thus needlessly give the HIV/AIDS mainstream what it does not have: the virus, the HIV tests and the efficacy of their trademark AIDS drug. Moreover they once again choose to rely on bird's eye-view statistics rather than biology and chemistry as their punch-line:

Aware of some of these life threatening toxicities of anti-HIV drugs, the Harvard study maintains that the ''benefits" of these drugs ''outweigh" their inevitable toxicity [1]. But, contrary to these claims hundreds of American and British researchers jointly published a collaborative analysis in The Lancet *in 2006*[6] *concluding that treatment of AIDS patients with anti-viral drugs has ''not translated into a decrease in mortality"*

4 Rodney Richards; "Increases in reported death in South Africa from 1997 to 2002: evidence for increasing mortality or improving death registration?" & "Response to Bradshaw et al." http://rethinkingaids.com

5 Anthony Brink; "Is AZT a DNA Chain Terminator? The phenomenal stupidity of David Rasnick" www.tig.org.za/Is_AZT_a_DNA_chain_terminator.htm

6 www.ncbi.nlm.nih.gov/pubmed/16890831

This piece of quote-mining is a misrepresentation of what the study and its authors say. The quotation in context says that *in the era of HAART*, the improvement in virological response has not translated into a decrease in *first-year mortality*. The authors do not conclude that HAART is not far better than no HAART or AZT monotherapy. They have also made various attempts at rationalising their results in the paper, which Duesberg et al ignore.

Incidentally, and this is why statistical/epidemiological arguments should never be used as a punch-line, follow-up results from the *Lancet* study have long been out, and they show decreasing mortality into the second year on HAART.[7] In a 2008 paper by the same study group, the interpretation of the results was changed to:

Life expectancy in HIV-infected patients treated with combination antiretroviral therapy increased between 1996 and 2005, although there is considerable variability between subgroups of patients. The average number of years remaining to be lived at age 20 years was about two-thirds of that in the general population in these countries.[8]

The Duesberg et al paper is from 2009. Did they check up on the later results and conclusions? Did they consider that the stagnant mortality in the era of HAART they are relying on is confined to a time period between 1996-2000? To base their argument on a limited period a decade or more ago, while ignoring possible confounders, is a high-stakes gamble.

Even more troubling, when an Italian colleague recently presented Duesberg with evidence seeming to contradict the hypothesis that anti-retrovirals cause AIDS –

(...) *we see a decrease of mortality among Italian people with AIDS (PWAIDS), notwithstanding a strong prevalence of people treated with "antiretrovirals"* (Fabio Franchi)

– Duesberg accepted without argument that there *had* been a decrease in mortality - the very thing he uses the *Lancet* 2006 quote to deny:

There are at least two factors that may explain such discrepancies: 1) In the US more and more healthy HIV-positives are diagnosed as "AIDS patients" since 1993,

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First-Year Mortality
1995/96: total n=1232 / #deaths=27 (2.2%)
1997: 4785 / 98 (2.1%)
1998: 4583 / 85 (1.9%)
1999: 3699 / 67 (1.8%)
2000: 3203 / 63 (2.0%)
2001: 2783 / 49 (1.8%)
2002/3: 1932 / 25 (1.3%)

Second-Year Mortality (cumulative)
1995/96: 1232 / 53 (4.3%)
1997: 4785 / 151 (3.2%)
1998: 4583 / 144 (3.1%)
1999: 3699 / 109 (3.0%)
2000: 3203 / 99 (3.1%)
2001: 2783 / 69 (2.5%)

8 www.thelancet.com/journals/lancet/article/PIIS0140673608611137/abstract

because they have antibodies against HIV (see D-K-R paper). Such "patients" survive toxic drugs much longer than the clinically ill patients of the early days of the epidemic. 2) The doses of the anti-HIV drugs have been lowered and their compositions are constantly changed to reduce toxicity. This also reduced mortality. (Duesberg)[9]

As is the case with Duesberg's co-authors, who argue for and against the Passenger Virus theory as circumstances dictate, it raises concerns about overall strategic coherence when a scientist accepts with such ease two mutually exclusive results and offers two mutually exclusive explanations for them.

CONCLUSION

Duesberg has famously said that "Epidemiology is like a bikini: what is revealed is interesting; what is concealed is crucial." It follows that raw epidemiological data can almost always be rationalised to suit one's favorite hypothesis. That is why epidemiological correlation should not be used to settle an argument about causation. This was Duesberg's initial insight in his seminal paper from 1987, *Retroviruses as Carcinogens and Pathogens: Expectations and Reality*. However, in this latest paper he repeatedly shows us a bikini of his own choosing and pretends it conceals nothing further. Because of this, his attacks on the HIV/AIDS construct become self-contradictory and end up lending support to many of the weakest points in its foundations.

9 Duesberg's answer to "Statistical correlation among recreational drugs, antiretroviral therapy and AIDS: Apparent incongruities of the drug-AIDS hypothesis of Peter Duesberg and al." www.dissensomedico.it/Bib_doc/Duesberg/Review,%20Franchi-D-K-R%20confutation_col.pdf