



Contents lists available at ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

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

Peter H. Duesberg^{a,*}, Joshua M. Nicholson^a, David Rasnick^b, Christian Fiala^c, Henry H. Bauer^d

^a Department of Molecular and Cell Biology, Donner Laboratory, UC Berkeley, Berkeley, CA 94720, USA

^b Private residence, Oakland, California, USA

^c Gynmed Ambulatorium, Mariahilferguelert 37, 1150 Vienna, Austria

^d Chemistry and Science Studies, Virginia Tech, Blacksburg, VA 24060-5623, USA

ARTICLE INFO

Article history:

Received 9 June 2009

Accepted 11 June 2009

Available online xxx

SUMMARY

A recent study by Chigwedere et al., “Estimating the lost benefits of antiretroviral drug use in South Africa”, claims that during the period from 2000 to 2005 about 330,000 South African AIDS-deaths were caused by the Human Immunodeficiency Virus (HIV) per year that could have been prevented by available anti-HIV drugs. The study blamed those who question the hypothesis that HIV is the cause of AIDS, particularly former South African President Thabo Mbeki and one of us, for not preventing these deaths by anti-HIV treatments such as the DNA chain-terminator AZT and the HIV DNA inhibitor Nevirapine. Here we ask, (1) What evidence exists for the huge losses of South African lives from HIV claimed by the Chigwedere study? (2) What evidence exists that South Africans would have benefited from anti-HIV drugs? We found that vital statistics from South Africa reported only 1 “HIV-death” per 1000 HIV antibody-positives per year (or 12,000 per 12 million HIV antibody-positives) between 2000 and 2005, whereas Chigwedere et al. estimated losses of around 330,000 lives from HIV per year. Moreover, the US Census Bureau and South Africa reported that the South African population had increased by 3 million during the period from 2000 to 2005 instead of suffering losses, growing from 44.5 to 47.5 million, even though 25% to 30% were positive for antibodies against HIV. A similar discrepancy was found between claims for a reportedly devastating HIV epidemic in Uganda and a simultaneous massive growth of the Ugandan population. Likewise, the total Sub-Saharan population doubled from 400 millions in 1980 to 800 millions in 2007 during the African HIV epidemics. We conclude that the claims that HIV has caused huge losses of African lives are unconfirmed and that HIV is not sufficient or even necessary to cause the previously known diseases, now called AIDS in the presence of antibody against HIV. Further we call into question the claim that HIV antibody-positives would benefit from anti-HIV drugs, because these drugs are inevitably toxic and because there is as yet no proof that HIV causes AIDS.

© 2009 Elsevier Ltd. All rights reserved.

Introduction

Based on the hypothesis that Human Immunodeficiency Virus (HIV) is the cause of a recent AIDS epidemic in South Africa, Chigwedere et al. estimated that 330,000 died unnecessarily from AIDS caused by HIV during the period from 2000 to 2005, “because a feasible and timely antiretroviral drug treatment program was not implemented in South Africa” [1]. The HIV-AIDS hypothesis postulates that HIV causes around 27 previously known diseases, but only 5 to 10 years after infection and induction of antiviral immunity [4,11]. Accordingly, Chigwedere et al. blamed all those who question the HIV-AIDS hypothesis for the failure to use anti-HIV drugs to prevent the estimated losses of lives, above all South African president Thabo Mbeki and even one of us. Moreover, they suggest that about 30,000 newborns could have been saved annually by preventing “mother-to-child trans-

mission” of HIV by brief treatments to all pregnant mothers with the inevitably toxic anti-HIV drugs AZT and Nevirapine (see below).

In view of our goal to solve the AIDS epidemic, and the specific accusations that those who question the HIV-AIDS hypothesis may be responsible for the loss of hundred thousands of lives we ask here, (1) What evidence exists for the huge losses of South African lives from HIV claimed by Chigwedere et al.? and (2) What evidence exists that South Africans would have benefited from anti-HIV drugs, such as AZT and Nevirapine?

A new perspective of South African AIDS

No evidence for huge losses of South African lives from HIV

Since 1984 a steady flow of publications has advanced the hypothesis that a new epidemic of HIV is decimating Africa and that high percentages of Africans are already infected by HIV [2–4]. In view of this and the recent study by Chigwedere et al.

* Corresponding author. Tel.: +1 510 642 6549; fax: +1 643 510 6455.

E-mail address: duesberg@berkeley.edu (P.H. Duesberg).

Table 1
Vital statistics of the South African population from 1980 to 2008.

| Year | Population $\times 10^{-6}$ | HIV+ (%) | Deaths $\times 10^{-3}$ | HIV-Deaths $\times 10^{-3}$ |
|------|-----------------------------|----------|-------------------------|-----------------------------|
| 1980 | 29.3 | | | |
| 1981 | 30.2 | | | |
| 1982 | 31.1 | | | |
| 1983 | 32.1 | | | |
| 1984 | 33.2 | | | |
| 1985 | 34.3 | | | |
| 1986 | 35.1 | | | |
| 1987 | 35.9 | | | |
| 1988 | 36.8 | | | |
| 1989 | 37.6 | | | |
| 1990 | 38.5 | 0.7 | | |
| 1991 | 39.3 | 1.7 | | |
| 1992 | 40.1 | 2.2 | | |
| 1993 | 40.9 | 4.0 | | |
| 1994 | 41.6 | 7.6 | | |
| 1995 | 42.2 | 10.4 | | |
| 1996 | 42.8 | 14.4 | | |
| 1997 | 43.3 | 17.0 | 317 | * |
| 1998 | 43.9 | 22.8 | 365 | * |
| 1999 | 44.5 | 22.4 | 381 | 10 |
| 2000 | 45.1 | 24.5 | 415 | 10.5 |
| 2001 | 45.6 | 24.8 | 453 | * |
| 2002 | 46.1 | 26.5 | 500 | * |
| 2003 | 46.6 | 27.9 | 554 | * |
| 2004 | 47.0 | 29.5 | 572 | 13 |
| 2005 | 47.5 | 30.2 | 591 | 14.5 |
| 2006 | 47.9 | 29.1 | 607 | 15 |
| 2007 | 48.4 | | | |
| 2008 | 48.8 | | | |

* Not reported because HIV-deaths were below 10th rank.

“estimating” about 330,000 preventable deaths from HIV per year, between 2000 and 2005, it comes as a surprise that South African statistics report only 1 “HIV-death” in 1000 HIV antibody-positive South Africans per year [5].

This number was obtained as follows: the average total South African population per year from 2000 to 2005 was obtained from consistent American and South African population statistics shown in Table 1 and Fig. 1A [5–7]. It was approximately 45 million. The HIV antibody-positive population was then calculated from the annual percentages of HIV antibody-positives of the total population, recorded in Fig. 1B and also in Table 1 [8]. It can be seen in Fig. 1 and Table 1 that the average number of HIV antibody-positive South Africans between 2000 and 2005 was about 12 million, or 25% to 30% of the average total of 45 million South Africans. The annual “HIV-death” rate per HIV antibody-positive South African was then calculated by dividing the total number of “HIV-deaths” per year by 12 million. It is shown in Table 1, that “HIV-deaths” made up only 2.5% of total registered mortality (10,471) in 2000; were below 10th rank and thus was not listed in 2001, 2002 and 2003; were 10th with 2.3% of cases (13,440) in 2004 and 10th with 2.5% of cases (14,532) in 2005 [5,9]. Thus South African statistics recorded an average of only about 12,000 “HIV-deaths” per 12 million HIV antibody-positives per year, or 1 per 1000, between 2000 and 2005. This is 25-fold less than the 300,000 HIV-deaths per year estimated by Chigwedere et al.

In other words, the HIV-attributable mortality of the approximately 12 million South Africans, which were HIV antibody-positive between 2000 and 2005 (Table 1; Fig. 1), was only 0.1%. Since all-cause mortality of South Africans was reported to be about 0.9 to 1.3% over the period from 1999 to 2006 (Table 1; Fig. 1) [5,9], the HIV-mortality of HIV antibody-positive South Africans represents less than 1/10 of the norm.

Further, the Chigwedere study from Harvard “estimates” that between 5% in 2000 to 55% in 2005 of 60,000 newborns were lost

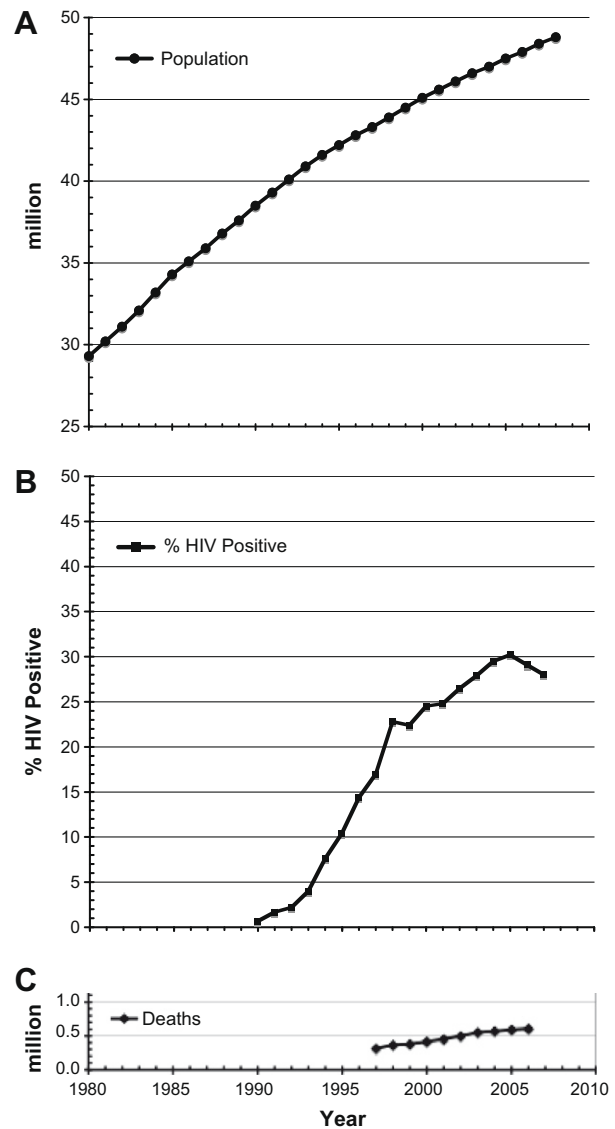


Fig. 1. The population growth curve (A), HIV-antibody incidence (B), and mortality (C) of the South African population between 1980 and 2008, as available from South African and American sources cited in the text.

from mother to child transmission of HIV because there were no anti-HIV drugs available to prevent infection. During this period the population increased on average by 0.5 million per year, and about 0.5 million died per year (Table 1; Fig. 1). It follows that there were annually about 1 million newborns in this period, of which the Harvard study estimates annual losses of 3000 to 30,000 to AIDS. 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.

We conclude that South African statistics provide no evidence for the huge losses of South African lives from HIV during 2000–2005, which estimated, namely 300,000 HIV-deaths above normal mortality and around 30,000 additional losses due to HIV-based infant mortality [1]. Since we could not confirm the huge numbers of

HIV-deaths claimed by Chigwedere et al., we did not analyze their estimates of how many such deaths could have been prevented by anti-HIV drugs.

Rapid population growths despite simultaneous HIV-epidemics

Further we asked, whether South Africa population statistics support the view that Africa is being devastated by a new HIV epidemic [1,4], which, according to HIV-AIDS researchers, began in 1984 [2,3].

As shown in Fig. 1B and Table 1, *The National HIV and Syphilis Prevalence Survey South Africa* first reported antibodies against HIV in 1990 in 0.7% of the South African population [12]. In the following 8 years, the percentage of South Africans with antibodies against HIV increased gradually to 23%. After 1998 the prevalence of HIV antibody-positives leveled off, oscillating between peak levels of 23% and 30% (Table 1; Fig. 1B).

But, instead of causing devastating losses of lives [1,4], the South African HIV-epidemic coincided with a steady, massive increase of the South African population (Fig. 1A). During the specific period from 2000 to 2005, the South African population gained 3 million, increasing from 44.5 to 47.5 million. And this happened, even though 25% to 30% or an average of 12 million South Africans were positive for antibodies against HIV during that time (compare Fig. 1A and B).

Over all, it can be seen in Table 1, column 2, and in Fig. 1A that from 1980 until 2008 the South African population increased from 29 million to 49 million at a high rate of about 1 million per year in the early 1980s and about 0.5 million per year since the 1990s [5–7]. The trajectories of the South African population growth curves and of the corresponding mortality curves [5] were continuous and were compatible between 1997 and 2006 (Fig. 1A and C).

Thus 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 and the annual mortality would have increased from an average of 500,000 to over 1 million during that time (Table 1). But this was not observed.

A similar discrepancy was found by one of us previously [13] between claims for a devastating AIDS epidemic in Uganda [3,14–16] and a simultaneous, unexpected growth of the Ugandan population [7]. It can be seen in Fig. 2 and Table 2, that the Ugandan population increased dramatically from 12 to 31 million during the period from 1980 to 2008 [7]. In 1989, the Minister of Health of Uganda first reported that 5.8% of the population was HIV antibody-positive [16]. This number reportedly increased by 1990 to about 13% and then slowly declined to 5% again by 2006 and 2007 (Fig. 2C) [17].

Moreover, the massive population increases of South Africa and Uganda during the AIDS-era are no exceptions among Sub-Saharan African countries. The total Sub-Saharan African population has indeed doubled from 400 millions in 1980 [10] to 800 million in 2007 [18].

We conclude that, contrary to the claims of Chigwedere et al., there was a massive increase of 3 million in the South African population between 2000 and 2005, which fits exactly into the continuous South African population growth trajectory that extends from 1980 until 2008 (Fig. 1A). In addition, there was a similar massive population growth in Uganda, although Uganda was also simultaneously subjected to an HIV-epidemic. Likewise there was a similar massive increase of the total Sub-Saharan population during the African HIV-epidemics. Thus the massive gain of 3 million South Africans during 2000 to 2005 and the absence of abnormal losses of 330,000 per year, or 1.8 million combined from 2000 to 2005, call the estimates of Chigwedere et al. into question.

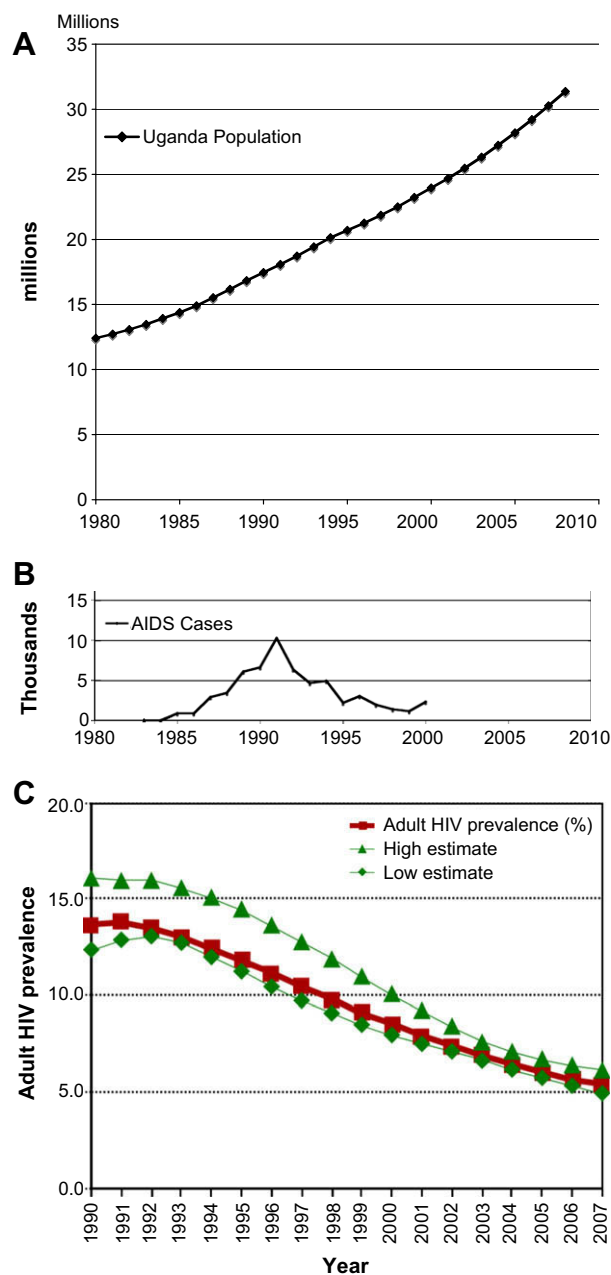


Fig. 2. The population growth curve (A), the AIDS-incidence (B), and the HIV-antibody incidence (C) of the Ugandan population between 1980 and 2008, as available from the Ugandan and American sources and the World Health Organization cited in the text.

Since the African HIV-epidemics coincided with steady and massive growths of the affected populations, we conclude that HIV-epidemics are not likely causes of AIDS epidemics. In view of this, we ask next whether HIV is a passenger virus.

Is HIV a passenger virus?

A passenger virus can be defined as one that is not sufficient and not necessary to cause a disease. Indeed the Centers of Disease Control's (CDC) definition of AIDS, which is any one of 27 previously known diseases in the presence of antibody against HIV, practically defines HIV as passenger virus [11]. It acknowledges that all AIDS-defining diseases have existed and continue to exist independent of HIV, e.g. tuberculosis and pneumonia. Thus HIV is not necessary for these diseases. At the same time the CDC and

Table 2
Vital statistics of the Ugandan population from 1980 to 2008.

| Year | Population* $\times 10^{-6}$ | AIDS cases** $\times 10^{-2}$ |
|---------------------------------|------------------------------|-------------------------------|
| <i>Uganda health statistics</i> | | |
| 1980 | 12.4 | |
| 1981 | 12.7 | |
| 1982 | 13.0 | |
| 1983 | 13.5 | 0.2 |
| 1984 | 13.9 | 0.1 |
| 1985 | 14.4 | 8.8*** |
| 1986 | 14.9 | 8.8*** |
| 1987 | 15.5 | 29.1 |
| 1988 | 16.2 | 34.3 |
| 1989 | 16.8 | 60.9 |
| 1990 | 17.5 | 66.2 |
| 1991 | 18.1 | 102.4 |
| 1992 | 18.7 | 63.5 |
| 1993 | 19.4 | 46.4 |
| 1994 | 20.1 | 49.3 |
| 1995 | 20.7 | 21.9 |
| 1996 | 21.2 | 30.3 |
| 1997 | 21.9 | 19.6 |
| 1998 | 22.5 | 14.1 |
| 1999 | 23.2 | 11.5 |
| 2000 | 24.0 | 23.0 |
| 2001 | 24.7 | |
| 2002 | 25.5 | |
| 2003 | 26.3 | |
| 2004 | 27.2 | |
| 2005 | 28.2 | |
| 2006 | 29.2 | |
| 2007 | 30.3 | |
| 2008 | 31.4 | |

* Population from US Census Bureau.

** AIDS cases from Ministry of Health Kampala, Uganda.

*** About 8.8×10^{-2} is representative for the years 1985 and 1986 combined.

other proponents of the HIV-AIDS hypothesis acknowledge the existence of millions of HIV antibody-positives, who are healthy [4], just as the millions of HIV antibody-positive Africans described here. It follows that HIV is not sufficient for AIDS.

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, inspired by the HIV-AIDS hypothesis [4,19]. The passenger virus-hypothesis also explains the failures to find a mechanism for the hypothesis that HIV causes AIDS by killing immune cells, despite over 25 years of research [20].

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], (ii) 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 [10,19]. By contrast, pathogenic viruses spread exponentially and then decline exponentially within a few months due to antiviral immunity, forming classical bell-shaped curves as described by Farr's law [22,23]. Take, for example, the typical time course of several months of a seasonal flu epidemic [22].

In sum, we conclude that HIV is a passenger virus. This would explain the low percentage of 0.1% "HIV-deaths" among 12 million HIV antibody-positive South Africans, recorded between 2000 and 2005 (see above) [5]. This explanation holds that most of the roughly 12,000 annual South African "HIV-deaths" are conven-

tional tuberculosis and pneumonias attributed to HIV, because the patients happened to be infected by the passenger virus HIV. This is all-the-more-likely, since tuberculosis and pneumonia are the primary causes of death and also the predominant AIDS-defining diseases in South Africa [5,9].

Evidence that HIV-positive Africans benefit from anti-HIV drugs called into question

The Harvard study proposes that inhibitors of HIV such as AZT and Nevirapine "benefit" South African AIDS patients as prophylactic against and ameliorative treatments for AIDS and to prevent newborns from becoming infected by HIV [1]. AZT and Nevirapine are thought to inhibit HIV because they inhibit HIV DNA synthesis. There are, however, three unsolved problems with this view:

- (1) HIV DNA synthesis has never been detected in HIV antibody-positive people, because replication of HIV is suppressed in the presence of antibody against HIV [10]. Thus inhibitors of DNA synthesis are unlikely to help against a virus that is latent and not making DNA, like HIV in antibody-positive persons.
- (2) AZT was developed 45 years ago to kill human cancer cells by terminating DNA synthesis [24]. Although termination of DNA synthesis is inevitably cytotoxic, 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 [10]. This advantage, however, does not apply when AZT is used against a target like latent HIV, which makes no new viral DNA. What remains under these conditions are only the inevitable DNA-toxicity, immuno-toxicity and aneuploidy, which are induced by AZT [10,25] and, which are euphemistically called "side effects" by the Harvard study [1]. These include life threatening, but not AIDS-defining liver-, kidney- and heart diseases were described recently [10,26,27]. The inhibitor of HIV DNA synthesis, Nevirapine, for example, induces life threatening "liver failure and severe skin reactions" in addition to "rash, headaches, diarrhea, fever, abdominal pain and myalgia" [28], (see also [26,27]). The NIH Treatment Guidelines acknowledge that "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; the risk for these events increases progressively as the CD4 T-cell count decreases from 350 to 200 cells/mm³" [29].
- (3) Over 50% of babies born to HIV antibody-positive mothers do not acquire maternal HIV [10] and thus would be treated unnecessarily with inevitably toxic anti-HIV drugs, if the Harvard study prevails. For example, the perinatal treatment of HIV-positive mothers and their babies with anti-HIV drugs, which the Harvard study recommends, has been shown to cause various forms of genetic damage in newborns, including "long-term mitochondrial toxicity" [31], "persistent mitochondrial dysfunction" due to defective or lost mitochondrial DNA [32], and "chromosome loss and duplication, somatic recombination, and gene conversion", which "justify their surveillance for long-term genotoxic consequences" [33]. These genetic defects are treatment-dependent and HIV-independent, because the same defects were found in HIV-negative children of HIV-positive mothers treated to prevent HIV transmission [10,31,34]. Moreover, Olivero et al. at the National Cancer Institute have shown genotoxicity and tumorigenicity in mice and monkeys born to AZT-treated mothers [35]. By contrast, no such genetic defects have been diagnosed in the estimated 34 million mostly untreated, asymptomatic HIV antibody-positives [4].

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 concluding that treatment of AIDS patients with anti-viral drugs has “not translated into a decrease in mortality” [30].

Conclusions

We have found no statistical evidence for the claim of the Harvard study that hundreds of thousands of South African lives were lost in the period from 2000 to 2005 due to an HIV-AIDS epidemic. Instead, South African statistics have recorded only about 1 “HIV-death” per 1000 HIV-positives per year (or 12,000 “HIV-deaths” among 12 million HIV antibody-positives) from 2000 to 2005. In contrast to the huge losses of lives claimed by the Harvard study of Chigwedere et al. the vital statistics of South Africa show that the population has increased from 2000 to 2005 by 3 million, from 44.5 to 47.5 million, continuing a steady trend since 1980, even though an average of 25% to 30% were positive for antibodies against HIV since 1998.

Therefore, we call into question the hypothesis that HIV causes AIDS and the proposal of Chigwedere et al. that huge hypothetical losses of lives from HIV can be prevented by treatments designed to inhibit HIV with inhibitors of DNA synthesis, not only because there is no evidence for lost lives and thus for a pathogenic HIV, but also because these drugs are inevitably toxic.

In view of this it is likely that South Africa’s “failure to accept the use of available ARVs [anti-HIV drugs]” [1], which the Harvard study criticizes, may have saved hundreds of thousands of lives by avoiding exposure to life threatening inhibitors of DNA synthesis. Thus it is the HIV-AIDS hypothesis that is not only out of touch with, but also potentially dangerous for South Africa. It seems premature therefore, indeed unwarranted for the Harvard study to blame former South African president Thabo Mbeki and others, including one of us, for the presumed losses of lives in South Africa.

Conflict of interest statement

I and my co-authors have no commercial or other non-scientific conflicts of interest with our AIDS paper for *Med. Hypotheses*.

Acknowledgments

We thank Colonel Frank Anders, Lieutenant Colonel Clinton Murray and Major Jason Okulicz for providing preliminary results on HIV-Elite Controllers from the US Military cohort [21] and for other valuable information, and Bill Redfearn, Berkeley, for critical comments. We are very grateful for support from the Abraham J. and Phyllis Katz Foundation (Newnan, GA), the Foundation for Advancement of Cancer Therapy (New York), Stefan Ebeling (philanthropist, Duesseldorf, Germany), Robert Leppo (philanthropist, San Francisco), Peter Rozsa (philanthropist, Los Angeles) and other private sources. A precursor of this paper was rejected by the *Journal of AIDS*, which published the Chigwedere et al. article, with political and ad hominem arguments but without offering even one reference for an incorrect number or statement of our paper (available on request).

References

- [1] Chigwedere P, Seage 3rd GR, Gruskin S, Lee TH, Essex M. Estimating the lost benefits of antiretroviral drug use in South Africa. *J Acquir Immune Defic Syndr* 2008;49:410–5.

- [2] Piot P, Quinn TC, Taelman H, Feinsod FM, Minlangu KB, Wobin O, et al. Acquired immunodeficiency syndrome in a heterosexual population in Zaire. *Lancet* 1984;2:65–9.
- [3] Quinn TC, Mann JM, Curran JW, Piot P. AIDS in Africa: an epidemiologic paradigm. *Science* 1986;234:955–63.
- [4] Durban Declaration, Over 5000 authors. The Durban declaration. *Nature* 2000;406:15–6.
- [5] Statistics South Africa. Mid-year population estimates, 2007. Department of Home Affairs, South Africa; 2007.
- [6] Statistics South Africa. Stats in brief 2000. Statistics South Africa; 2000.
- [7] US Census Bureau, International data base. Population South Africa. US Department of Commerce; 2008.
- [8] National Department of Health South Africa. The national HIV and syphilis prevalence survey South Africa; 2007.
- [9] Statistics South Africa. Mortality and causes of death in South Africa, 2006: Findings from death notification. Statistics South Africa; 2008.
- [10] Duesberg P, Koehnlein C, Rasnick D. The chemical bases of the various AIDS epidemics: recreational drugs, anti-viral chemotherapy and malnutrition. *J Biosci* 2003;28:383–412.
- [11] Centers for Disease Control. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. *Morb Mort Weekly Rep* 1992;41(No. RR17):1–19.
- [12] National Department of Health South Africa 2008. The national HIV and syphilis prevalence survey South Africa; 2008.
- [13] Fiala C. AIDS in Africa: a call for sense, not hysteria (Rapid Responses for Sidley, 327 (7408) 184). *bmjcom*; 2009.
- [14] Mulder DW, Nunn AJ, Kamali A, Nakiyingi J, Wagner HU, Kengeya-Kayondo JF. Two-year HIV-1-associated mortality in a Ugandan rural population. *Lancet* 1994;343:1021–3.
- [15] Mulder DW, Nunn AJ, Wagner HU, Kamali A, Kengeya-Kayondo JF. HIV-1 incidence and HIV-1-associated mortality in a rural Ugandan population cohort. *AIDS* 1994;8:87–92.
- [16] Goodgame RW. AIDS in Uganda—clinical and social features. *N Engl J Med* 1990;323:383–9.
- [17] World Health Organization, UNAIDS, UNICEF. Epidemiological fact sheet on HIV and AIDS; Core data on epidemiology and response Uganda; 2008.
- [18] World Bank Group. Population of Sub-Saharan Africa; 2008.
- [19] Bauer HH. The origin, persistence and failings of the HIV/AIDS theory. Jefferson, NC and London, UK: McFarland & Co.; 2007.
- [20] Henry WK, Tebas P, Lane HC. Explaining, predicting, and treating HIV-associated CD4 cell loss: after 25 years still a puzzle. *JAMA* 2006;296:1523–5.
- [21] Okulicz J, Marconi V, Dolan M. Characteristics of elite controllers, viremic controllers, and long-term nonprogressors in the US Military HIV cohort. Keystone symposium HIV pathogenesis. Banff, Alberta, Canada; 2008.
- [22] Mims C, White DO. Viral pathogenesis and immunology. Oxford: Blackwell Scientific Publications; 1984.
- [23] Bregman DJ, Langmuir AD. Farr’s law applied to AIDS projections. *JAMA* 1990;263:50–7.
- [24] Horwitz JP, Chua J, Noel M. Nucleosides. V. The monomesylates of 1-(2′-deoxy-beta-D-lyxofuranosyl)thymidine. *J Org Chem* 1964;29:2076.
- [25] Borojerdi JP, Ming J, Cooch C, Ward Y, Semino-Mora C, Yu M, et al. Centrosomal amplification and aneuploidy induced by the antiretroviral drug AZT in hamster and human cells. *Mutat Res* 2009;665:67–74.
- [26] Reisler RB, Han C, Burman WJ, Tedaldi EM, Neaton JD. Grade 4 events are as important as AIDS events in the era of HAART. *J Acquir Immune Defic Syndr* 2003;34:379–86.
- [27] El-Sadr WM, Lundgren JD, Neaton JD, Gordin F, Abrams D, Arduino RC, et al. CD4+ count-guided interruption of antiretroviral treatment. *N Engl J Med* 2006;355:2283–96.
- [28] Medicine.Net.com. Nevirapine index; 2008 [accessed].
- [29] Department of Health and Human Services, Panel on antiretroviral guidelines for adults and adolescents. Guidelines for the use of antiretroviral agents in HIV-1-infected adults and adolescents; January 29, 2008. p. 13. Available from: <<http://www.aidsinfo.nih.gov/ContentFiles/AdultandAdolescentGL.pdf>>.
- [30] May MT, Sterne JA, Costagliola D, Sabin CA, Phillips AN, Justice AC, et al. HIV treatment response and prognosis in Europe and North America in the first decade of highly active antiretroviral therapy: a collaborative analysis. *Lancet* 2006;368:451–8.
- [31] Poirier MC, Divi RL, Al-Harthi L, Olivero OA, Nguyen V, Walker B, et al. Long-term mitochondrial toxicity in HIV-uninfected infants born to HIV-infected mothers. *J Acquir Immune Defic Syndr* 2003;33:175–83.
- [32] Barret B, Tardieu M, Rustin P, Lacroix C, Chabrol B, Desguerre I, et al. Persistent mitochondrial dysfunction in HIV-1-exposed but uninfected infants: clinical screening in a large prospective cohort. *AIDS* 2003;17:1769–85.
- [33] Escobar PA, Olivero OA, Wade NA, Abrams EJ, Nesel CJ, Ness RB, et al. Genotoxicity assessed by the comet and GPA assays following in vitro exposure of human lymphoblastoid cells (H9) or perinatal exposure of mother-child pairs to AZT or AZT-3TC. *Environ Mol Mutagen* 2007;48:330–43.
- [34] Brogly SB, Ylitalo N, Mofenson LM, Oleske J, Van Dyke R, Crain MJ, et al. In utero nucleoside reverse transcriptase inhibitor exposure and signs of possible mitochondrial dysfunction in HIV-uninfected children. *AIDS* 2007;21:929–38.
- [35] Olivero OA, Anderson LM, Diwan BA, Haines DC, Harbaugh SW, Moskal TJ, et al. Transplacental effects of 3′-azido-2′, 3′-dideoxythymidine (AZT): tumorigenicity in mice and genotoxicity in mice and monkeys. *J Natl Cancer Inst* 1997;89:1602–8.