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Rapid Responses to:

EDUCATION AND DEBATE:
Didier Fassin and Helen Schneider
The politics of AIDS in South Africa: beyond the controversies
BMJ 2003; 326: 495-497 [Full text]

... (Clipping:)

Analysis: the properness of the HIV hypothesis is a media hype

17 March 2005

Torsten Engelbrecht, journalist
20359 Hamburg/Germany

Send response to journal:
Re: Analysis: the properness of the HIV hypothesis is a media hype

dear sir, dear madam,

my name is torsten engelbrecht and i am journalist in hamburg (www.torstenengelbrecht.com). i am following the subject hiv/aids personally since many, many years. recently i addressed the subject more closely as journalist with my in-depth media analysis "sex, blood and death: 'the hi-virus causes aids.'. the universally held acceptance of this theory demonstrates how scientific journalism disregards significant inconsistencies to dispel any doubts about authenticity." this analysis has been published recently in germany's most recognised special interest media magazine "message" (www.message-online.com). the original version of this analysis you can find on my website under the following link: http://www.torstenengelbrecht.com/en/artikel_medien.html. the purpose of this research was to find out scientifically if all these very respectful scientists - like the nobel laureates kary mullis and walter gilbert, harry rubin, etienne de harven, "nature-biotechnology"-founder harvey bialy, eleni-papadopulos from the australian perth group, etc. - are right in saying that it has never been proven that (1) so-called hiv exists, that (2) so-called hiv causes aids, that (3) so-called hiv antibody tests detect hiv, that (4) haart prolong lives, and so on.

so i did an in-depth media analysis which took me several months to carry out. one of the basis was a worldwide survey asking the most important media outlets (e.g., "nature", "science", "washington post", "spiegel", "new york times", "die zeit", "time", "the new yorker", "newsweek") if they have any experimental proof/clear-cut study for all the claims the hiv-hypothesis is based on: that (1) so-called hiv exists, that (2) so-called hiv causes aids, that (3) so-called hiv antibody tests detect hiv, that (4) haart prolong lives, that (5) pcr- and cd4-tests are reliable in the context of aids-diagnosis, or that (6) the so called drug- hypothesis (aids = drugs like poppers or crystal meth, antiretroviral drugs, malnutrition and/or re-definition of well-known diseases) makes no sense. the result: not one media outlet could deliver one single proof for one of these claims. additionally, i wanted to know, to which drgree the media was dealing with the (obviously justified) criticism of the hiv- hypothesis. so i checked the coverage of the subject aids of several german speaking print media ("spiegel", "faz", "nzz", "berliner zeitung", "sz", "tagesspiegel", and "taz") during the last 10 years. and the result is unmistakable, as i write in my article "sex, blood and death": altogether we counted more than 20,000 texts on the subject. of the 60 reputable experts who criticised the hiv-hypothesis during that time, only nine were mentioned: among them peter duesberg in 20 articles related to aids and kary mullis in seven, which constitute only 0.135 percent of all reports about aids. yet even in those few articles, the authors avoided confrontation with the arguments of the critics, such as the criticism of conventional aids-testing. which confirms what the us-media critic michael tracey discovered once (see his analysis "mere smoke of opinion - aids and the making of the public mind": http://www.virusmyth.net/aids/continuum/article6.htm): "we decided to interrogate the majpap (major papers) file in the nexis database of 37 newspers, which includes the british broadsheets, "guardian", "times", "sunday times", "independent", "ft", and the "daily telegraph", as well as most major us papers. (for some reason the mass circulation british tabloids, the "sun" and the "star" are not included.) we searched for the number of stories in which the phrase "aids virus" was employed - a phrase which he correctly took as representing the notion of causality within the aids thesis. in 1984 - the year of the heckler-gallo-conference - there were just 31 mentions of the phrase, but by 1991 it was appearing in more than 3000 stories a year in these 37 papers. by 1993 there had in fact been 20,024 uses of the term. of countervailing theories there is barely a bat's squeak. then we had a look at how gallo had fared. he found that alongside the hundreds of references were attached phrases such as 'noted', 'superstar', 'famed', 'vindication', 'significant strides', 'the one scientific hero', 'brilliant, dynamic', 'pioneering researcher', 'who discovered [or co-discovered] the aids virus', 'gallo's virus', and so on."

so what we see here is a clear example of the shaping of public discourse, the construction of a way of seeing aids. and the media made it possible by sending out the

unproven messages that a virus named hiv causes aids or that a positive hiv test means getting aids (or better: one of the dozens of well-known diseases like kaposi's sarcoma) sooner or later. what kary mullis and all the other critics fo the hiv-hypothesis addresses should also be fundamental to the media: facts count first. but the media has been having problems with just these facts since the beginning of the aids-issue, when, in 1983, the "new york times" wrote about a "worldwide health problem". at that time, not taking the facts into account, for example "spiegel" and "bild der wissenschaft" were already quite sure that civilisation was going downhill and anticipated that the last german would die of aids in 1996 (see: "aids: die bombe ist gelegt", in: "der spiegel" 45/1984; "aids - eine neue krankheit erschüttert deutschland", in "bild der wissenschaft" 12/1985). and on what or whom did the media base this catastrophic scenario? mostly - so the result of my analysis - on the cassandra-like talk of fame hungry physicians who turned the subject into a headline from the very beginning. it must be a contagious disease, following the principle spread by louis pasteur - "one disease, one cause, one cure" (to quote sociologist steven epstein)

in 1959 pulitzer-prize winning microbiologist rené jules dubos wrote that "the search for THE (single) cause would remain a hopeless enterprise, since most disease-conditions are the indirect result of a constellation of life circumstances." Understanding this concept, one must ponder whether or not the matter is too complicated and at the same time too mundane, to be turned into an exciting media story.

additionally, the scientific journalists suffer from a fundamental problem: dealing with uncertainty. in striving to provide their audience with simple answers, the journalists reduced overly complex aids-issues to simple, mostly causal patterns of explanation. hence, they created the superstition that, with "safer sex" or "hiv testings", aids could be defeated.

thanks for your attention

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Competing interests: None declared

Re: Analysis: the properness of the HIV hypothesis is a media hype

18 March 2005

Nicholas Bennett,
Infectious Disease Postdoc/Clinician
Department of Pediatrics, University Hospital, Syracuse NY

Send response to journal:
Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

The "60 or so reputable experts" can effectively be boiled down to Duesberg, the Perth Group and their followers. Other commentators have chipped in with one or two specific points (Mullis, De Harven, Giraldo) but that's about it. Anyone who studies this sad field for more than a few months will quickly realise that.

If one takes the arguments of Duesberg and the Perth Group out of the dissident literature, there is very little else. Arguably without their underlying framework none of the others would have even said anything at all.

Interestingly, in searching the dissident websites there is almost no mention of any mainstream researchers except for Ho, Montagnier and Gallo. The work of Mellors which

neatly shows that HIV viral load predicts progression to AIDS is ignored. That of Pantaleo which states clearly that lymph nodes are the site to judge HIV infection, rather than peripheral blood is nowhere to be found. In fact none of the _thousands_ of reputable orthodox scientists are represented in their arguments or their results explained away according to the dissident logic. Rather they are simply ignored.

When one such as myself attempts to contribute to the dissident forums he is silenced, censored, and eventually banned from contributing. This even when pointing out non-HIV related science such as the link between cervical cancer and HPV infection (greater than 98% last I heard). It's as if the moderators prefer to keep their readers uneducated, because that's the only way the anti-HIV pseudoscience can persist. "Reputable" dissidents such as Duesberg and Bialy choose to ignore or abuse those who attempt to confront them (a recent week-long diatribe by the good Dr Bialy against myself on a public discussion board caused considerable embarrassment to the dissident cause recently for example). The Perth Group of course have been repeatedly shown on this forum to have misrepresented or ignored the literature, and have not accepted or conceded a single criticism of their "science".

HIV causes a single disease, AIDS. How that manifests will naturally depend on what other infectious agents are currently likely to infect the particular individual. It's no different from any other immune deficiency syndrome. Is that really so hard to accept?

Nick Bennett njb35@cantab.net

Competing interests: None declared

Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

21 March 2005

Torsten Engelbrecht,
journalist
20359 Hamburg

Send response to journal:
Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

"when an experiment is challenged no matter who it is challenged by, it's your responsiblity to check. that is an ironclad rule of science, that when you publish something you are responsible for it." howard temin, nobel laureate

"these guys [from aids orthodoxy] just don't have it [the experimental proof that hiv causes aids]" kary mullis, nobel laureate

dear mr. bennett,

many thanks for your answer. please allow me to explain why you are mistaken in every single point of your rapid response message:

1. there much more than 60 (there are hundreds) of reputable experts that state that the hiv hypothesis has not been proven yet - and my survey confirms again that nobody can deliver any proof (study) for the hiv hypothesis.

2. even if there was only one expert or person who says that the hiv hypothesis is unproven, that's enough (keeping also in mind that nobody is able to deliver any clear-cut proof!). we should never forget: it's not the majority that counts in order to establish scientific facts, it's "experimental proof", as nobel laureate kary mullis commemorates. and isn't it said that this has to be commemorated? nobel laureate howard temin: "when

an experiment is challenged NO MATTER WHO IT IS CHALLENGED BY, it's your responsiblity to check. THAT IS AN IRONCLAD RULE OF SCIENCE, THAT WHEN YOU PUBLISH SOMETHING [e.g., an article based on the assumption that the hiv hypothesis is true] YOU ARE RESPONSIBLE FOR IT."

3. so you are responsible for delivering the clear-cut proofs for the hiv- hypothesis: so where is the clear-cut proof (in form of a single study) that (1) hiv exists, that (2) hiv causes aids, that (3) hiv/aids is sexually transmitted, or that (4) anti-retroviral drugs prolong life? do you mind send me these kind of studies? again, it's you (saying that the hiv hypothesis explains aids) who has the burden of proof!

4. you write that other scientists (than the ones who follow duesberg and the perth group) like mullis, de harven or giraldo "just chipped in with one or two specific points". this is definitely also not true. kary mullis, for example, has not only chipped in with something, he confronted the aids establishment with the most fundamental criticism: that there's no proof that hiv causes aids. also, he said (e.g., in an interview in the year 2000 with the german newspaper "sueddeutsche zeitung") that aids must be a lifestyle-disease (so he agrees witht duesberg and many others that the drug hypothesis explains aids). also etienne de harven: he has not only chipped in with something, either, because he confronted the aids establishment with the other most fundamental criticism: that there's no proof that hiv exists - which, by the way, caves in the whole hiy=aids-bulding. and he also said: (1) stop all use of antiretroviral drugs until the isolation of hiv and its pathogenicity are scientifically established; (2) stop using highly crossreacting serological tests, the hiv specificity of which is far from demonstrated; (3) provide african people with means do combat malnutrition, clean drinking water, proper housing and sanitation, and efficient health-care infrastructures (see his speech at a 2003 eu-conference "problems with isolating hiv: http://www.altheal.org/texts/isolhiv.htm). and also robert giraldo is not just chipping in "with one or two specific points", either! please just have a look on his paper "IS IT RATIONAL TO TREAT OR PREVENT AIDS WITH TOXIC ANTIRETROVIRAL DRUGS IN PREGNANT WOMEN, INFANTS, CHILDREN, AND ANYBODY ELSE?" (see http://www.robertogiraldo.com/eng/papers/IsItRational.html). then you will see that giraldo shows up the absurdity of every single thesis of the hiv hypothesis!

5. you quote studies that you think are proving the usefulness of pcr-tests. but first, if hiv has not been proven (which is obviously the case because nobody is able to deliver a clear-cut proof) all these studies are just useless because they don't have a proven base. moreover, there are several studies showing that pcr-tests are useless (even if you assume that the hiv-hypothesis is correct); for example: Josiah D. Rich et al., "Misdiagnosis of HIV infection by HIV-1 plasma viral load testing: a case series" (1999) Annals of Internal Medicine 130: 37-39. and last but not least kary mullis himself - the inventor of the pcr - says that pcr-tests are useless for aids-diagnosis. they are much too sensivite. so no one, not even montagnier or gallo, could plausibly explain why so few of the helper cells so important for the immune defence are infected even in terminal aids-patients. hence, they couldn’t explain the breakdown of the immune system with just the hiv-theory. "proceedings of the national academy of sciences" already called attention to this paradox in 1985 (Pahwa et al., 1985, p. 8198-8202). that same year, montagnier admitted in "annals of internal medicine" that the virus alone could not be responsible for aids but that co-factors were necessary. this is the standpoint still taken today. however, orthodox science as well as the media aren't interested in this point of view. they have already committed themselves to the hiv/aids-hypothesis without having any proof for it...

6. you say you are silenced by dissidents. i don't know if this is true, but i am sure that you won't be silenced if you deliver what everybody is asking for since many, many years: the clear-cut proofs for the hiv hypothesis. why don't you just write them down here in this forum, and all the "dissident-spook" that obviously gets on your nerves is over?

7. again, you are wrong in saying that "hiv causes a single disease, aids". i mean, how can you claim that? the opposite is true! the cdc- definition of aids says that you are an aids-patient if you have been tested positively and (1) has one of dozens(!) well-known(!!!) diseases (many of them actually not being an infectious disease!!!) like kaposi's sarcoma, herpes zoster, etc. and/or (2) less than a specific amount of cd4 cells. and the bangui-deifintion of aids applied in third world countries says that you are an aids-patient just if you have a specific (again well- known) diseases (plural!!!) like tuberculosis, cough, weight-loss, etc. whatever aids is caused by, aids is not a single disease, that's for sure!

8. malnutrition, drugs like poppers or crystal meth, and anti-retroviral drugs are immune suppressive. practically all aids-patients in rich countries are heavy drug users - so what makes you so sure that drugs don't play an important role in the development of aids syndroms? even gallo admitted that hiv cannot be the primary cause of aids-kaposi's-sarcome, but poppers is probably the primary cause." and what makes you so sure that in poor countries (in africa, for example one third of the population is affected by malnutrition) malnutrition does not contribute to the people's illnesses called aids? and what makes you so sure that all theses illnesses called aids are not just a re-definition of well-known diseases like tuberculosis?

you write: "dissidents prefer to keep their readers uneducated, because that's the only way the anti-hiv-pseudoscience can persist." but actually it's exactly the other way around: as long as you are not able to deliver clear-cut proofs for the assumptions of the hiv-hypothesis - especially for the claims that (1) hiv exists, that (2) hiv causes aids, that (3) hiv/aids is sexually transmitted, or that (4) anti-retroviral drugs prolong life - hiv-science is pseudoscince in pure culture because you do science that is based on pure believes, and not on experimental proof.

thanks for your attention and best wishes

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Competing interests: None declared

Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

21 March 2005

Mark Bartlett,
CD Investigator
Canada

Send response to journal:
Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

Dr. Bennett wrote:

"The "60 or so reputable experts" can effectively be boiled down to Duesberg, the Perth Group and their followers. Other commentators have chipped in with one or two specific points (Mullis, De Harven, Giraldo) but that's about it. Anyone who studies this sad field for more than a few months will quickly realise that."

Considering the quality of some of your other contributions, I find this statement unfortunate. Science is not governed by a show of hands. Consensus proves nothing. Most of the truly great "eureka" moments were from lone voices who had incredible insight. Often the truth of their contributions remained misunderstood and/or ridiculed for decades by the "experts of the day.

One could just as easily argue that all who supported Gallo and Montagnier were no more than "followers" except that they seemed to have the support of the scientific establishment (CDC) despite how premature that support was in the context of the state of Gallo's research in 1983.

For 50 years we have been fed another line of garbage about the connection between cholesterol and heart disease. Yet, when that association has been decontructed, it gets little press. Much of the "research" that supported that connection has been discredited -- nevertheless the nonsense continues. Recently, the pharmaceutical companies have won their way again and had the "acceptable" level of cholesterol lowered -- I wonder how many millions more people that added to the ranks of the "high risk" and coincidentally candidates for their drugs.

But that is a discussion for another day and another thread. However it does bring me to my point. When there are huge amounts of money to be made from treatments vs cures, I am VERY skeptical of the truth behind the science. And so should you be and everyone else.

Competing interests: None declared

Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

21 March 2005

Nicholas Bennett,
Infectious Disease Postdoc/Clinician
Department of Pediatrics, University Hospital, Syracuse NY

Send response to journal:
Re: Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

Mark Bartlett's points are correct, but my point was more that the dissenting camp is extremely picky in what they choose to deconstruct - or attempt to at least.

I _am_ skeptical about the science, as all scientists are, but only when such the claims make no sense and have no supporting evidence.

In my experience in dealing with AIDS dissent and denial, it is the latter word that is the most important. It is not so much that various people see the same science from a different, if valid point of view (although that too is true) but that most often thay simply choose to ignore other scientific facts - and in fact HAVE to if their logic stands any chance of consistency. If my post lacked some of the professionalism of previous contributions it is due to the frustration of over six years trying to educate those who chose not to be educated.

For example I recently brought one dissident to task for quoting a CDC report that he claimed proved that condoms could not possibly protect against HIV - his statements had carefully deleted the CDC's reference to condoms being responsible for around 85% reduction in HIV transmission.

It is impossible to teach someone about CD4 downregulation, nef- mediated apoptosis, synctia formation, anergic responses and accelerated T cell death if primarily they refuse to accept that the methods used to isolate HIV were valid.

That is a personal choice. It is not science.

Nick Bennett njb35@cantab.net

Competing interests: None declared

Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

21 March 2005

Nicholas Bennett,
Infectious Disease Postdoc/Clinician
Department of Pediatrics, University Hospital, Syracuse NY

Send response to journal:
Re: Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

In reply to Herr Engelbrecht's points:

1. Has he tried writing to journals asking if they can provide proof that the Moon isn't made of cheese? All joking aside, I do not think this is a survey many will take seriously at all.

2. One dissenting view is far from enough! In science one must back it up with solid evidence. NONE of the anti-HIV scientists have performed A SINGLE EXPERIMENT designed to support their views that HIV is harmless or non-existent. The only two experiments I know of are Duesberg showing that AZT is toxic to certain cells in culture (which we already knew) and Giraldo performing HIV tests without following the directions in the kit.

3. The clear-cut proofs have been presented and argued here over and over. The problem is not whether they exist or not, but whether one chooses to accept them.

4. Mullis, Giraldo, De Harven etc have all made statements that build upon the "logic" of Duesberg and the Perth Group. Nothing original.

5. With no intent to cause disrespect, If you want a clear-cut proof I suggest that you re-read the literature supporting HIV causing AIDS with rather more than the education provided by reading the denialist websites. The case series are published because they are _rare_ and _important_. PCR for research is very different from PCR-based viral load tests (which these days aren't even done using PCR technology!). Mullis' concern was NOT that PCR was used to detect HIV, but that PCR was used to _quantitate_ HIV. The meaning has been twisted over the years by those not able to make the distinction. Montagnier made that statement based on some of his research that suggested a mycoplasma may be involved. His results were later shown to be due to the cell line he used. Either way, whether co- factors are involved or not is irrelevant, since the epidemiology proves that infection with HIV is the primary risk factor.

6. The science has been presented and debated here ad nauseum. [1]

7. The confusion, whether intentionally created or otherwise, regarding AIDS as a single or disparate collection of diseases is common. The fact is that AIDS is a specific loss of cellular immunity, due to a loss of CD4 T helper cell number and function. In the same way as other immune deficiencies will predispose the patient to many different diseases, so too with AIDS. It highlights how important our immune systems are, and how ubiquitous pathogens can be in our environment.

8. AIDS-KS is of course due to a co-infection with HHV8, so Gallo was right. Ascher et al actually showed that not only was drug use NOT predictive of AIDS (when you removed the effect of HIV serostatus!) but that drug users actually had higher CD4 counts than their controls. Other situations, malnutrition especially, may result in similar loss of health status BUT do NOT cause the immune dysfunction seen in AIDS. This is what

distinguishes AIDS from other immune suppression conditions. This is why it is important to realise why AIDS is a single disease, because once you start to think that the disease is only the symptoms, then of course you will find similar conditions! Under this criteria there is no distinction between rhinovirus, coronavirus, adenovirus infection etc.

HIV exists because you can quite simply see it under EM, and we can grow it and make more of the stuff. The genome has been cloned (which even Duesberg thinks is sufficient to consider it to exist). The mechanism by which HIV causes AIDS has been laid out by myself, as current understanding permits, on this forum [2].

If HIV is not sexually transmitted, it seems odd that condom use protects against it [3]. It is also odd that the use of anti-retrovirals DOES prolong life [4]. I really do wonder where you're getting your "facts" from.

I once gave a list of reasons why HSV-2 could not cause genital herpes, employing the same "logic" enjoyed by those who deny that HIV is harmfull. I think I made a rather good case.

" Can the Perth Group tell us all the standard by which HSV-2, an important STD, is judged? Is it any less or more than that of HIV? HIV serology has been confirmed by culture, PCR, lymph node biopsy and EM. As far as I can tell, the Gold Standard for HSV is western blot. HSV tests "may have extensive cross-reactivity" [5]. HSV co-exists with many other kinds of STD including bacterial infections - who is to say that it isn't these that cause the vesicles? Aciclovir is an entirely non-specific antiviral that will readily chain-terminate non-viral DNA, and the infection is supposedly never cleared despite the disappearance of symptoms. Seroconversion may occur after the appearance of lesions, with a time of 25-47 days. How can the virus possibly be the cause of the vesicular lesions? One of the side effects of aciclovir is itching: how can this be distinguished from the itching of "herpes" infection?"

It's rather easy to construct a false argument using pseudoscience - far harder for scientists to counter it to those already "tainted" by mis- education.

Nick Bennett njb35@cantab.net

1. http://bmj.bmjjournals.com/cgi/eletters/326/7387/495

2. http://bmj.bmjjournals.com/cgi/eletters/326/7387/495#76614

3. de Vincenzi N Engl J Med. 1994 Aug 11;331(6):341-6. "A longitudinal study of human immunodeficiency virus transmission by heterosexual partners. European Study Group on Heterosexual Transmission of HIV."

4. Egger et al. BMJ. 1997 Nov 8;315(7117):1194-9. "Impact of new antiretroviral combination therapies in HIV infected patients in Switzerland: prospective multicentre study. Swiss HIV Cohort Study."

5. Field et al Pathology. 1993 Apr;25(2):175-9. The reliability of serological tests for the diagnosis of genital herpes: a critique.

Competing interests: None declared

A Summary of Our Debate with Nicholas Bennett 22 March 2005

Eleni Papadopulos-Eleopulos,
Biophysicist
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Valendar F Turner, John Papadimitriou, Barry Page, David Causer, Helman Alfonso, Sam Mhlongo, Todd Miller, Christian Fiala

Send response to journal:

Re: A Summary of Our Debate with Nicholas Bennett

A SUMMARY OF OUR DEBATE WITH NICHOLAS BENNETT

Nicholas Bennett is a self-confessed "Expert" in "HIV" and an ardent protagonist of the "HIV" theory of AIDS. So let us summarise his contribution to this debate regarding the existence of "HIV" and two of its main properties, namely killing of the T4 cells and sexual transmission.

The existence of "HIV"

Nicholas Bennett entered the debate with the following sentence: "Further to previous replies concerning published EMs of [purified] HIV, the best example I have seen personally have been those in the paper: Richieri et al Vaccine, 1998 Jan-Feb;16(2-3)119-29". ("Re: HIV EM Purification", 28 June 2004). When we pointed out to him that the EM does not show purified retroviral particles he still insisted that it showed "wall-to-wall virions" ("One AIDS patient will suffice", 6 July 2004).

Nicholas Bennett claimed that our request for a EM showing HIV particles in the bloodstream is "a straw-man argument". We pointed out that according to Hans Gelderblom, it is possible to see particles in the bloodstream, at least in individuals which have particle concentrations of one million/cm^3, and that nobody, not even Hans Gelderblom published such EMs. ("Notes from Hans Gelderblom", 7 July 2004; "Where is the virus", 9 July 2004).

Subsequently Nicholas Bennett claimed that in order to prove the existence of the "HIV" genome and proteins and thus "HIV" it is not necessary to purify the "HIV" particles. "In my mind, I actually have to side with Duesberg on the concept that the purist forms of any virus is the molecular clone, and sequencing of viral RNA or proviral DNA (with subsequent analysis) is a perfect acceptable method of proving [the] existence" of "HIV". However, to date nobody, including Nicholas Bennett and the other participants in this debate, could provide one single reference with evidence for the existence of the "HIV-1 infectious molecular clone".

Several rapid responses are in regard to the "HIV" specificity of the reverse transcriptase activity which Montagnier detected in 1983 ("Re: One AIDS patient will suffice", 6 July; "Re: Where is the virus", 10 July; "Re: Where are the proper controls in HIV" research", 20 July; "Montagnier's reverse transcriptase activity", 23 July; "Re: "HIV", HHV-8 and KS", 21 August; "Nicholas Bennett and Montagnier's RT activity", 8 September; "Re: Nicholas Bennett and Montagnier's RT activity", 9 September; "Various responses to the Perth Group", 9 September; "More on Nicholas Bennett and Montagnier's RT activity, 28 September; "Relative values", 29 September; "Repeat the origin of the CK and RT activity cannot be determined by their "RELATIVE" amounts", 11 October; "Re: Repeat the origin of the CK and RT activity", 11 October; "Long irrelevant yarns are actually hard science", 26 October, 2004).

To begin with, Nicholas Bennett claimed that in his 1983 paper Montagnier proved the specificity for "HIV" of the reverse transcriptase activity as follows: "Montagnier's RT prefers Mg^{2+} at 5mM and pH 7.8…The keyword is PREFERS. DNA pol gamma will work with $Mg2^{+}$ at pH 7.4…Montagnier also used further confirmatory methods, such as inhibition with Actinomycin D, which does not affect RT [retroviral RT activity] but does inhibit cellular DNA polymerases and RNA polymerases".

We provided the reference to a study by Montagnier himself in support of our claim. We pointed out to Nicholas Bennett that in 1984 Montagnier himself showed that his RT activity was inhibited by 50% with actinomycin and that "DNA pol gamma [and b] will work" with $Mg2^{+}$ at 5mM and pH 7.8". Hence, since Actinomycin D "does not affect RT [retroviral RT activity]" the RT activity reported by Montagnier cannot be retroviral. Subsequently, Nicholas Bennett did not even mention if he read Montagnier's reference but claimed that the "HIV" RT activity can be distinguished from the RT activity from other sources by its "RELATIVE" amount, but the "Perth Group seem unable to recognise the principle of RELATIVE amounts". We have pointed out that the origin of an enzymatic activity, especially non-specific activities such as RT and creatine phosphokinase (CK) cannot be determined by their "RELATIVE amounts". Nicholas Bennett again claimed that we are wrong and gave a reference which he claimed shows that the origin of CK can be determined by its "RELATIVE" amount. However, the reference he gave us showed the exact opposite.

In our rapid response, "Repeat, the origin of the CK and RT activity cannot be determined by their "RELATIVE amounts", again we gave a reference where Montagnier showed that non "HIV" infected cells, using the same condition as in 1983 have high levels of RT activity. We wrote: "If we are as wrong and dangerous as Nicholas Bennett claims, then he must write to Medical Hypothesis and refute our latest critique of Montagnier's work".

We repeat: (i) The enzyme reverse transcriptase is not specific to retroviruses or even viruses, a fact accepted by such experts in retrovirology as Varmus and Weiss; (ii) the human genome contains endogenous retroviruses (proviruses) and thus reverse transcriptase genes which, given the right conditions, can be activated; (iii) reverse transcription can be induced not only by the enzyme reverse transcriptase but also by the cellular DNA polymerases.

The ex cathedra statement that beyond a certain level RT activity is caused by the reverse transcriptase enzyme of a particular retrovirus is ludicrous. Is Nicholas Bennett suggesting that regardless of variable and unknown concentrations of "viral" and cellular enzymes and their individual activities according to culture conditions, a certain "level" always means "HIV" RT and nothing else? Even if this were remotely possible what is his proof and what is that "level"? In the Montagnier et al paper HERE, to which we have repeatedly drawn Nicholas Bennett's attention and which he has ignored, the authors presented evidence which shows: (i) contrary to what Nicholas Bennett claims, actinomycin D significantly inhibited (50%) the reverse transcriptase activity which Montagnier claimed to be "HIV" RT; (ii) in figure 2 of the Rey and Montagnier paper, in uninfected cells DNA polymerase alpha, beta and gamma had reverse transcriptase activity of approximately 5, 42 and 17 enzyme units respectively. In "infected" cells the level of "HIV" RT was 57 units and the RT activity of DNA polymerase alpha, beta and gamma were 12, 50 and 14 enzyme units respectively. If one totals the uninfected cell enzyme units it comes to 64 which is greater than the "HIV" activity. At what "level" does Nicholas Bennett suggest the reaction becomes "HIV" specific?

In his reply ("Re: Repeat the origin of the CK and RT activity", 11 October) Nicholas Bennett wrote: "Since the Perth Group have issued the invitation to refute them in formal print, I would be happy to do so. Not least because Montagnier's work contributes only to the molecular biology of HIV and not whether or not HIV causes AIDS, his "contribution" to that question ought to be relatively easy to refute. I found half a dozen glaring errors in the abstract alone. The rest of the paper appears to have been repeated ad nauseum on the Rapid Responses, and therefore the material to refute it is also here. Saves me a lot of time." We are still waiting.

Amazingly, after all this, recently but elsewhere Nicholas Bennett wrote: "I called the Perth Group in public on their lie that Montagnier didn't distinguish viral RT from cellular activity. I'm still waiting for a recognition of the fact 6 months later, despite several reminders to do so. They have not even tried to defend their conclusion, just ignored any comments, even though it underpins most of what they argue about HIV being endogenous cellular phenomenon"! (Dean's World; 1.21.2005 4:05pm).

In his rapid response: "Re: Where are the proper controls in "HIV" research", 20 July, Nicholas Bennett wrote: "As to how I know that the majority of scientists agree that HIV RNA and proteins exist, I have to stop myself laughing loud…The Perth Group clearly failed to comprehend the basis of my arguments for the infectious molecular clone….EM is not necessary, because the molecular evidence is so strong…I provided extensive evidence for the existence of a molecular clone already". In another response, Nicholas Bennett claimed that "there are a plethora of infectious molecular clones of HIV-1 in circulation" (Re: Where is the HIV-1 infectious molecular clone?", 14 July).

We asked, ("More on Nicholas Bennett's "HIV" infectious molecular clone", 28 September 2004): "…would Nicholas Bennett please provide evidence for the existence of an "HIV-1 infectious molecular clone" as defined by Brian Foley (not us). "The clone must produce virus particles that are identical by serology, morphology, protein sequences, RFLP, Southern blotting, etc., to the parental virus, and the particles must also be infectious. If a cloned viral genome does not meet these criteria, it is not an INFECTIOUS molecular clone of the virus, be it HIV-1 or any other virus" (Foley's emphasis).

Nicholas Bennett has not given us even one reference with evidence which proves the existence of an "HIV-1 infectious molecular clone" even by Brian Foley's definition ("Re: "HIV", HHV-8 and KS", 21 August, 2004; "Re: Cell death and oxidation", 1 September, 2004; "Re: Nicholas Bennett and the "HIV-1 infectious molecular clone", 8 September, 2004; "Give us the "HIV" "structure" please", 11 October).

Nicholas Bennett claimed that: "…the presumed HIV RNA actually encodes the presumed HIV proteins". ("Re: Retrovirologist, retroviruses and purification", 2 July, 2004). He made similar claims in subsequent rapid responses. ("Re: Where is the "HIV-1 infectious molecular clone?", 14 July, 2004).

We asked Nicholas Bennett "where is the evidence that the "presumed HIV RNA" that is the poly-A RNA which in sucrose density gradients bands at the density of 1.16 g/ml (the "HIV" genome) "actually encodes" the "HIV" proteins which band at the same density". ("Where are the proper controls in "HIV" research", 19 July, 2004). Nicholas Bennett gave us a long yarn but not one single reference with evidence that the "HIV" RNA encodes the "HIV" proteins ("Re: Where are the proper controls in "HIV" research?", 20 July, 2004).

From the beginning of the "HIV" era we have claimed there is no evidence that the "HIV" antibody tests prove "HIV" infection. The antibody tests have been extensively discussed in this debate and although we repeatedly asked for references which prove that a positive test means "HIV" infection, to date nobody (including Nicholas Bennett) has given us even one reference. We drew Nicholas Bennett's attention to this fact and provided many references in support of our claim. ("Mechanism by which HIV causes AIDS", 29 September, 2004; "Diagnosing "HIV" infection in neonates", 30 September, 2004). Nicholas Bennett gave us long yarns on the "HIV" antibody tests, but not one single reference where their specificity has been determined. (""HIV" mechanisms revisited", 2 October).

Incredibly, in his rapid response: "Re: Diagnosing "HIV" infection in neonates", 10 October 2004, Nicholas Bennett wrote: "The Perth Group do an admirable job of summarising the reasons why serology is a superior diagnostic tool in ADULTS, but of course we were talking about neonates". This statement is not only misleading but very wrong. How is it possible for us, on the one hand to present well documented evidence that at present there is no evidence that a

positive test even in one adult proves "HIV" infection and on the other to "do an admirable job of summarising the reasons why serology is a superior diagnostic tool in ADULTS…."? ("HIV" mechanisms revisited", 2 October).

Regarding the antibody tests in neonates in our rapid response: "Re: Diagnosing "HIV" infection in neonates", 1 October 2004, we wrote: "Nicholas Bennett wrote "Negative infants are followed up until their antibodies disappear, usually at 9 months".

In regard to maternal antibodies disappearing by 9 months would Nicholas Bennett please study the findings of the European Collaborative Study and the Ariel Project, summarised in our monograph "Mother to Child Transmission of HIV and its Prevention with AZT and Nevirapine" - pages 44-45 (Reference 5). In brief the arguments is as follows: The European Collaborative Group Study, is the only study providing a detailed analysis of the post partum loss of infant "HIV" seropositivity. The authors reported approximately 23% of children became seronegative between birth and 9 months. However, 59% became seronegative between 9 and 22 months. Since the latter cannot be due to loss of maternal antibodies, (the maternal antibodies have already been lost by 9 months) the only explanation is that either: (i) the antibody test is non-specific or (ii) the children managed to clear "HIV" infection without treatment. If 23% of children test positive because of maternal antibodies, and in 59% the test is non-specific, how can one be certain that the remaining 18% of children will not also serorevert after 22 months? If the test is non-specific in 59% of children one must also question whether such a test can be "extraordinarily accurate" when applied to the diagnosis of HIV infection of mothers, as well as to fathers and adults in the general population.

Again Nicholas Bennett responded with a long yarn but did not discuss the implication of these data in regard to the specificity of the antibody tests in general, not just in neonates. ("Re: Diagnosing "HIV" infection in neonates", 1 October 2004). Indeed, Nicholas Bennett's question "why the dogmatic hand-up with the magical 9 month value" appears to indicate he does not comprehend the implications of such data.

In our rapid response "The "HIV" antibody tests are not diagnostic", 14 October 2004, once again we drew Nicholas Bennett's attention of the facts about the neonatal antibodies and their implications and gave him a reference by researchers from the CDC where the authors reported "a rapid decay" of maternal "HIV" antibody" with a decline to background levels by 6 months (T½ = 28-30 days)". Nicholas Bennett responded with yet another long yarn ("Re: The "HIV" antibody tests are not diagnostic", 15 October 2004, but without giving even a single reference in which the specificity of the "HIV" antibodies has been proven for neonates or adults.

In our rapid response: "Repeat, the "HIV" antibody tests are not diagnostic", 20 October 2004, we reminded Nicholas Bennett once again that since the mother's antibodies disappear by 9 months, the seroreversion after 9 months means either the children eliminated "HIV" or the antibody tests are not specific. Although in one of his earlier rapid responses ("Neonatal HIV", 28 September 2004) Nicholas Bennett wrote: "Infants are followed up till their [mother's] antibodies disappear, usually at 9 months", in his rapid response "Re: Repeat the "HIV" antibody tests are not diagnostic in infants, but are fine in adults", 20 October 2004, he wrote: "why the dogmatic hand-up with the magical 9 month value". The answer is, because this is what the evidence shows and most importantly because it has profound implications regarding the specificity of the "HIV" antibody test in both infants and adults and thus for "HIV" infection (existence) and the claimed causal role of "HIV" in AIDS.

When at the European Parliament Meeting, "AIDS in Africa : What are the priorities as for medical assistance", 8 December 2003, Montagnier was asked if he isolated "HIV" in 1983, he replied: "Bien sûr on avait un virus [virus-like particles?] qui avait une densité de 1.16 en gradient avec une activité transcriptase inverse, et des protéines spécifiques reconnues par les anticorps du patient". (Web translation service at http://world.altavista.com/tr).

However, at present it is well known that:

(i) At the density of 1.16 even after "Roman" effort, Montagnier could not find any particles with the morphological characteristics of retroviruses, much less of a unique retrovirus "HIV";

In 1983 Montagnier, Barré-Sinoussi and Chermann were aware that reverse transcriptase activity is not specific to retroviruses. In 1984 they themselves showed that the template-primer used in 1983, under exactly the same conditions they used in that year, can be transcribed by the cellular polymerases beta and gamma of non infected cells.[1] We are not the only ones to have questioned the specificity of the RT activity detected by Montagnier. On page 81 in John Crewdson's book "Science Fictions: A Scientific Mystery, a Massive Cover-Up, and the Dark Legacy of Robert Gallo", one reads: "Gallo was questioning the reality of the reverse transcriptase activity" detected by Montagnier.[2]

(ii) Most importantly, since at the 1.16 band they had RT activity but not retrovirus particles it proves that the detected activity was not retroviral;

(iii) Montagnier reacted the proteins of the 1.16 band with serum from his patient BRU. Of the many proteins present in the 1.16 band, 3 (p25, p45, p80) were found to react with the patient serum. They said that p45 (p41) was actin, made no comment regarding p80,and claimed that p25 (p24) was an "HIV" protein and the antibodies which reacted with it "HIV" antibodies.

However, because the antibody cross-react from an antibody-antigen reaction it is not possible to determine the origin of one reactant even when the origin of the other is known, much less the origin of both.

Yet incredibly Montagnier claimed that the reaction between the p25 (p24) protein in the 1.16 band and antibodies in the patient serum proved that the p24 was "HIV" specific protein despite that fact that no retrovirus particles were present in the 1.16 band and the antibodies which reacted with it were "HIV" antibodies despite the fact that their patient had antibodies directed against a plethora of other antigens. This is academic fantasy.

Given the above indisputable facts, would Nicholas Bennett please tell us in 1983 did Montagnier prove the existence of "HIV"

Yes or no.

If yes, what is his scientific basis?

If no, when was "HIV" discovered and by whom?

"HIV" and the T4 cells

The T4 cell killing by "HIV" and their role in the development of AIDS has been discussed in many rapid responses ("Re: "HIV" and KS", 29 September 2004; "OKT3, OKT4 and all that", 2 September 2004; "CD3, CD4 and all the more modern names". 3 September 2004; "Causes of low CD4 counts in AIDS", 14 September 2004; "Mechanism by which HIV causes AIDS", 29 September 2004; "HIV mechanism revisited", 2 October 2004; ""HIV" and AIDS", 16 October 2004; "Re: "HIV" and AIDS", 18 October 2004; "Re" No scientific evidence - no scientific debate", 17 December 2004. Suffice to point out that in the early part of his debate Nicholas Bennett was adamant that the cause of the decrease in T4 cells in humans (in vivo) was "HIV", and at least in part due to their killing by "HIV". However, in his more recent posting he expressed a different and at times contradictory view.

In his rapid response: "Re: No scientific evidence - no scientific debate", Nicholas Bennett simply stated: "HIV" drops CD4 counts and causes the immune-suppression that allows a commensal organism to cause pneumonia (PCP)", without giving any evidence.

In his rapid response: "Re: "HIV" and AIDS", Nicholas Bennett wrote: "HIV induces a state of chronic, abnormal, immune activation with concomitant cytokine skewing that prevents normal T cell replacement from the thymus. Immune deficiency is a result of cell death due to (in part) HIV killing but probably more so apoptosis due to activation".

However:

(a) "activated $CD8^+$ T-cell count, CRP concentration, and intensity of marijuana use were all associated with increased oxidative damage";[3]

(b) as far back as 1994 Gallo stated: ""Oh, the role of HIV is likely to be in increasing these inflammatory cytokines" But we have learned - this should be of interest to everybody that isn't completely married to HIV - that the inflammatory cytokines are reportedly increased in gay men even without HIV infection. Inflammatory cytokines are usually promoted by immune activation, not by immune suppression. So here was a paradox…So the inflammatory cytokines may be increased by HIV, but I wish I knew what else was increasing them before a gay man was ever infected with HIV…The nitrites [oxidation] *could* be the primary factor" (Gallo's emphasis). See http://www.virusmyth.net/aids/data/jlpoppers.htm

That is, the cause of "activation" is oxidation not "HIV" as Nicholas Bennett claims.

In his rapid response: "HIV mechanism revisited", 2 October 2004, Nicholas Bennett wrote: "HIV infection ---> immediate CD4 cell loss through:

Trafficking to Lymph nodes [14] (activated T cells traffic).

Cell death [15, 16]".

In reference 14, "$CCR5^+$ CD4 T lymphocytes were studied in subjects with primary HIV-1 infection (PHI)". The authors do not claim to have shown that the loss of CD4 cells is due to "Trafficking to Lymph nodes", but that "The only decline of CD4 lymphocytes during PHI resulted from depletion of $CCR5^-$ CD4 T lymphocytes. After antiretroviral therapy, Ki-67 $CCR5^-$ CD4T cell counts rapidly increased in the circulation, which suggests that the initial decrease was due to an alteration in trafficking and/or sequestration" (emphasis ours).

Reference 15 is one of the 4 papers published in Science in 1984 by Gallo's team. There is no evidence that "HIV" causes T4 cells death. It is only stated: "That the viruses we have named HTLV-III belong to the HTLV family is indicated by their…cytopathic effects on T lymphocytes (3)…". Reference 3 is the first paper in the series of 4

published in Science, with Popovic as the principle author. Popovic et al did not present evidence that HTLV-III ("HIV") causes T4 cell death. To the contrary, "Some of the clones permanently grow and continuously produce large amounts of virus after infection…" (emphasis ours). Again, in reference 16 there is no evidence that "HIV" causes death of the T4 cells. Thus Nicholas Bennett once again gave us references which do not contain any evidence to support the claims he makes.

We have repeatedly pointed out that at the beginning of the AIDS era:

1. Gallo and Montagnier were aware that a decrease in the number of T4 cells does not necessarily mean cell death or "Trafficking to Lymph nodes", but SIMPLY A PHENOTYPIC change of T4 cells to other T lymphocyte subsets, such as T8 and can be caused by many agents including PHA.

2. The sum (T4 + T8) remains constant.

3. In 1986 Gallo showed that:

   "HIV" + PHA ---> decline in T4 cells

   "HIV" ---> no significant effect

   PHA ---> decline in T4 cells

4. T4 and T8 have similar functions.

5. In 1991 Montagnier showed that

   "HIV" + PHA ---> apoptosis

   PHA ---> apoptosis

In other words, for a decrease in T4 cells:

1. T4 cell death is not necessary.
2. "HIV" does not cause decrease in T4 cells by any means.
3. T4 cell decrease can be caused by many agents to which AIDS patients are subjected.
4. The T4 and T8 cells do not have unique immunological functions.

For references see http://www.theperthgroup.com/SCIPAPERS/ept4cells.html

One wonders then why this simple explanation has not been accepted and hundreds if not thousands of papers have been published trying to prove the unprovable. That is, that "HIV" decreases the T4 cells.

In a few of our rapid responses we have drawn attention to the fact that in 1986 Gallo and in 1991 Montagnier showed that HIV does not cause T4 cell death by any means including apoptosis.[4 5]

In one such rapid response we asked "Would Nicholas Bennett please tell us, does the Montagnier and Gallo data show that:

(i) "HIV" does not kill or decrease the T4 cells by any means.

Yes or no.

(ii) since the AIDS patients are exposed to repeated stimulation ("infections with various micro-organisms", "allogenic cells such as semen or blood", drugs (including factor VIII), the cause of the T4 decrease in these patients is the stimulation and not "HIV".

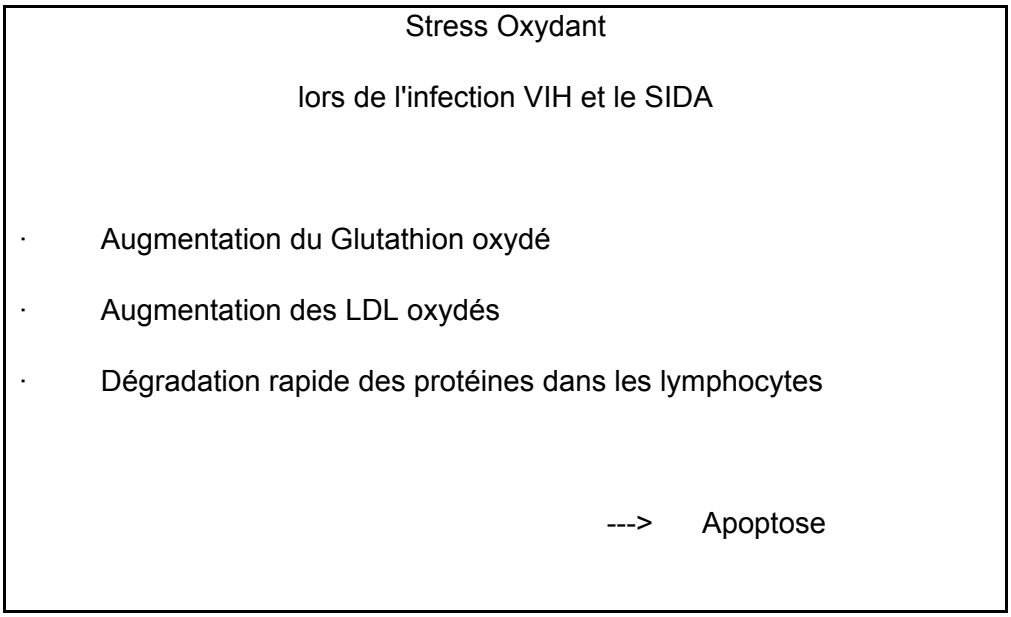
Yes or no.

The only reply we had from Nicholas Bennett was the following: "I note again the obsession with the two initial protagonists in the HIV/AIDS story: researchers who have been one very minor part of the puzzle of HIV". (Re: "HIV" and AIDS", 18 October 2004). Maybe we should draw Nicholas Bennett's attention to the view presently held by one of the "two initial protagonists in the HIV/AIDS story", Luc Montagnier.

In his speech to the European Parliament Meeting, "AIDS in Africa: What are the priorities as for medical assistance", 8 September 2003, Montagnier said: "Enfin un des problèmes majeurs non totalement résolu de la pathogenèse du Sida, reste l'explication de la **mort massive des lymphocytes T4.** Contrairement à ce que l'on croyait il y a quelques années, cette disparition, qui existe dès la période asymptomatique, n'est pas due à l'infection directe des cellules par la souche virale, qui est alors peu cytopathogène, mais à des mécanismes indirects touchant les cellules CD4+ non infectées; celles-ci ont une propension à mourir **d'apoptose**,…."

Regarding the cause of the T4 cell death by apoptosis Montagnier said "Un des médiateurs de cette apoptose est l'existence d'un fort **stress oxydant** caractérisé par une prévalence de molécules oxydantes (radicaux libres) sur les défenses antioxydantes de l'organisme: ainsi le taux de glutathion oxydé est-il très élevé, de même que celui des LDL (Low density lipoproteins) oxydées (figure 5).

Fig. 5



Ces anomalies ni disparaissent pas totalement après traitement antirétroviral, suggérant qu'elles méritent d'être corrigées par la prise d'antioxydants appropriés. Des études préliminaires indiquent que ce stress oxydant est plus fort chez les patients africains et existe même chez les individus non infectés, du fait d'une malnutrition".

Montagnier also said: "Concomitamment, commence un déclin du système immunitaire portant principalement mais non exclusivement sur l'immunité cellulaire dépendant des lymphocytes T CD4+, qui aboutit finalement à une phase clinique d'infections opportunistes et de cancers qui entraînent la mort".

In summary, according to Montagnier:

(i) the cause of the clinical syndrome and thus death of the patients is T4 cell death;

(ii) T4 cell death is due to apoptosis;

(iii) The cause of apoptosis is oxidation.

In other words the cause of AIDS is OXIDATION.

If the principal cause of the syndrome is T4 deaths and T4 deaths is due to apoptosis which is caused by oxidation, would Nicholas Bennett please tell us what is the role of "HIV" in AIDS?

We would be grateful if Nicholas Bennett would please tell us how Montagnier's present day view concerning the role of oxidation in AIDS can be reconciled with Montagnier's present day view that "HIV" causes AIDS? (Note that: (i) Neither Montagnier nor anybody else has provided scientific evidence that "HIV" causes oxidation; (ii) In 1992 was Montagnier made aware of our oxidative theory of AIDS (including the causes of oxidation in each risk group) proposed at the beginning of the AIDS era[6] but has never acknowledged it).

If Nicholas Bennett is to be consistent he should now call Montagnier's comments on AIDS and oxidative stress as yet more "drivel" and "holes in the head". And as an HIV/AIDS expert scientist he should make it his immediate business to personally acquaint Montagnier with his views.

In his rapid response "HIV mechanism revisited", Nicholas Bennett wrote: "What is important is that HIV can induce a decline in CD4 counts, not that all low CD4 counts are due to HIV…HIV is cytotoxic to T4 cells, in vitro at least - in vivo data less convincing…It's not so much that all individuals with low CD4 counts should get AIDS OIs, or that all people with AIDS-OIs will have low CD4 counts".

With this, Nicholas Bennett himself has demolished the cornerstone of the "HIV" theory of AIDS.

Sexual transmission of "HIV"

In his rapid response, "Re: Oxidation - the primary cause for AIDS and "HIV"", 22 January 2005, Nicholas Bennett wrote: "HIV" serology should, obviously, appear to be transmissible and associated with the individuals and risk groups associated with AIDS".

In our rapid response, "More on oxidation - the primary cause for AIDS and "HIV"", 1 February 2005, we have cited several references in which the epidemiological evidence shows that unlike the other sexually transmitted disease which are bi-directional, "HIV" serology", like pregnancy is sexually acquired but is not sexually transmitted.

Incredibly, in his rapid response, "Re: More on oxidation - the primary cause for AIDS and HIV"", 2 February 2005, Nicholas Bennett does not make any comments in regard to the epidemiological evidence we gave him on the unidirectionally acquisition of "HIV serology", and gives a reference for the un-equal transmission of mice retroviruses. Is this the best he can do?

In conclusion Nicholas Bennett entered the debate apparently with the following beliefs:

(a) There is evidence for the existence of "HIV", namely,

(i) There is EM evidence for the purification of the "HIV" particles;

(ii) The specificity of RT for "HIV" has been proven;

(iii) The existence of the "HIV" infectious molecular clone has been proven;

(iv) The "HIV" RNA codes for the "HIV proteins;

(v) The specificity of the "HIV" antibodies has been proven

(b) The "HIV" decreases the T4 cells by trafficking to lymph nodes and by killing them and in its turn leads to the clinical syndrome.

(c) The "HIV serology", that is, "HIV" is transmissible by sexual intercourse.

During the course of this debate, Nicholas Bennett was asked to give references to substantiate his beliefs. The references that Nicholas Bennett gave us contained no evidence to support his beliefs and in many cases went against his beliefs. Clearly it is about time that Nicholas Bennett either comes up with the scientific evidence and not yarns (as he chronically has in the past) or starts to question his own beliefs (which even the best scientists do from time to time).

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The Predictions of the "HIV" and Oxidative Theories of AIDS

22 March 2005

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Send response to journal:
Re: The Predictions of the "HIV" and Oxidative Theories of AIDS

THE PREDICTIONS OF THE "HIV" AND OXIDATIVE THEORIES OF AIDS

It is accepted that a theory is as good as its predictions.

The predictions of the "HIV" theory of AIDS

When we have asked Nicholas Bennett to tell us which were the main predictions of the "HIV" theory of AIDS he replied (Re: Oxidation - the primary cause of AIDS and "HIV"", 22 January:

1. "HIV serology should precede AIDS". If "HIV" is the cause of AIDS then the first prediction should be: "HIV should precede AIDS". This can be paraphrased as "HIV serology should precede AIDS" only if evidence exists that the antibody tests are "HIV" specific.

We have always maintained that a positive antibody test predicts the presence of future development of pathology including the diseases which are said to indicate AIDS. The question is "Do these antibody tests prove "HIV" infection?" Neither Nicholas Bennett nor Peter Flegg nor Brian Foley nor anybody else has given any evidence to support "HIV" infection.

In his latest effort to justify the "HIV" Western Blot test, ("HIV tests", 26 January 2005) all Nicholas Bennett could give answering Rob McGregor (Re: Re: Oxidation – the primary cause for AIDS and "HIV"", 25 January 2005) was: "The differences in Western Blot criteria again boil down to differences in the test kits used. Each kit is developed differently - some using recombinant proteins, some using virus preps, some using peptides. It makes sense that depending on the specific antigen being presented and the mode of presentation, sensitivity and specificity for individual bands will vary. This is why each kit includes appropriate interpretation instructions, again based upon comparisons with standardised panels."

It is true that the test kits may contain different antigens. However, it is also true, and surely Nicholas Bennett is aware of this, that one and the same kit is used in different countries and different laboratories with different criteria of interpretation of the results based upon different "standardised panels". Furthermore, these "standardised panels" and the criteria for a positive test have been introduced arbitrarily. Surely Nicholas Bennett is aware that the specificity of the "HIV" antibody tests can be determined only by comparing the results of the antibody tests with the presence or absence of "HIV". (The presence or absence of "HIV" must be determined by a method which does not involve antibody-antigen reactions, namely "HIV" isolation).

To date nobody has presented such evidence. [2] There are many such statements in the HIV literature some of which have been documented by UK investigative journalist Neville Hodgkinson in his article " Why an HIV test may not provide proof positive at all. The critical flaws in the methods we use to detect the killer virus" published in *The Business* May 2004 HERE (search the page for "Thomas Zuck").

We have repeatedly mentioned in this debate that our claim that the specificity of the "HIV" antibody test has not been determined (no gold standard exists) has been accepted by some of the best experts in HIV testing, including Mortimer and Blattner, as well as antibody test kit manufacturers.

2. "HIV serology should predict HIV detection via other means (e.g. culture, PCR, antigen testing)"

Repeat: "In the antibody tests, the "HIV" proteins are given and are reacted with sera containing antibodies. When a reaction takes place, the reacting antibodies are said to be "HIV".

In "culture" and "antigen testing", the "HIV" antibodies are given and are reacted with either the culture contents or with antigens. If a reaction takes place, then it is claimed proof for "HIV isolation" or detecting of "HIV" proteins. (See the very often cited papers by David Ho or Brooks Jackson).

Since it is the same reaction involved in the three tests, it is likely that some correlation is found between them.

We have repeatedly asked Nicholas Bennett to give us a few references in which the specificity of the PCR for "HIV" has been proven". Nicholas Bennett has never responded to our request.

In fact, no correlation exists between "serology", "culture", "PCR" and "antigen testing". [3 4]

See also "The isolation of HIV - Has it really been achieved?"
http://www.theperthgroup.com/CONTINUUM/pgvsduesbergreward.html

Furthermore and most importantly, even if a correlation between all these tests did exist, since there is no evidence that even one of them is specific to "HIV", it is difficult to see how Nicholas Bennett's second predictions will add credit to the "HIV" theory of AIDS.

3. "Pharmacological intervention against the virus should inhibit the viral replications in vitro and in vivo, and result in restriction of the immune dysfunction seen in AIDS and pre-AIDS complex patients".

The most often used method to determine "viral replication", is "viral load". We asked Nicholas Bennett "to give us a few references where it has been shown that "viral load" means "HIV" infection and that a decreased "viral load" means inhibition of "HIV" replication". No such references have been given to us. In fact all the "HIV" experts (including the CDC's) as well as "viral load" test kit manufacturers concur that "viral load" cannot be used to prove "HIV" infection.

Again, given the fact that no evidence exists that the "viral load" test means "HIV" RNA, it is difficult to see how it's inhibition by "pharmacological intervention", that is Nicholas Bennett's third prediction, will lend weight to the "HIV" theory of AIDS.

4. "HIV" serology should, obviously, appear to be transmissible and associate with the individuals and risk groups with AIDS".

Let us repeat. The main mode of "HIV" transmission is said to be sexual and the main risk groups in the developed countries to be gay men. Let us once again remind Nicholas Bennett that bi-directional transmission is required for an agent to be sexually transmitted. In a previous rapid response we drew attention to the following facts. In 1984, Robert Gallo and his colleagues wrote "Of eight different sex acts, seropositivity correlated only with receptive anal intercourse….and with manual stimulation of the subject's rectum (receptive "fisting")….and was inversely correlated with insertive anal intercourse." Two years later they confirmed their 1984 findings: "In this analysis, only receptive rectal intercourse, douching, rectal bleeding…were significant predictors ($p<.05$) of anti-HTLV-III positivity…We found no evidence that other forms of sexual activity contributed to the risk." In a 1994 review of all the major studies conducted in gay men including the longest, largest, best-designed and executed published study of gay men anywhere in the world, the MultiCenter AIDS Cohort Study, the authors concluded: "(1) unprotected anogenital receptive intercourse poses the highest risk for the sexual acquisition of HIV-1 infection; (2) anogenital insertive intercourse poses the highest risk for the sexual transmission of HIV-1 infection; (3) there is mounting epidemiologic evidence for a small risk attached to orogenital receptive sex,…(4) sexual practices involving the rectum and the presence of (ulcerative) STD facilitate the acquisition of HIV-1; (5) no or no consistent risk for the acquisition of HIV-1 infection has been reported regarding other sexual practices such as anogenital insertive intercourse and oroanal sex…".

Since the main and absolutely necessary property of sexually transmitted agents is bidirectionality, that is, transmission from the passive to the active partner and vice versa, this means "HIV" cannot be sexually transmitted. In other words, "HIV serology" like pregnancy, is sexually acquired but is not sexually transmitted.

For more than 20 years the "HIV"/AIDS experts have been claiming that humans are infected with a bi-directionally sexually transmitted virus, "HIV", and that by now more than 50 million individuals have been infected by this mode. Yet, incredibly the only reference Nicholas Bennett could provide for a bi-directional transmission of "HIV serology" is a paper on murine (mice) retroviruses.

For some unknown reason Nicholas Bennett failed to mention three of the main predictions of the "HIV theory of AIDS. Namely: (a) "HIV" and thus AIDS would spread throughout the heterosexual population in the USA, Europe and Australia. This has not happened. The prediction that a vaccine would be developed by 1986 has not been fulfilled. Even today no such vaccine exists or is likely to in the foreseeable future.

In 1984 Montagnier said that the only way to prove HIV is the cause of AIDS is to have an animal model. According to Dr. Joseph L. Bryant, Director of the Animal Models Division at Gallo's Institute of Human Virology, Baltimore, "To my knowledge, almost all major scientific successes on unraveling and conquering human diseases has been with the use of animal models. AIDS and AIDS-associated diseases will be no exception." http://www.ihv.org/research/animal.html

Although no effort has been spared no model of an "HIV" AIDS animal model has been forthcoming. In fact the only AIDS animal model to date supports the oxidative theory of AIDS. [5]

In conclusion, at present no evidence exists to support the predictions of the "HIV" theory of AIDS. In fact the "HIV" theory cannot even account for the two main phenomena for which it was put forward, KS and T4 cell decrease and thus the opportunistic infections. At present it is accepted that the cause of KS is not "HIV". The "discoverers" of "HIV", Montagnier and Gallo, have shown that "HIV" does not kill the T4 cells. [6-8]

In fact, according to Montagnier the decrease in T4 cells precedes, does not follow, "HIV" infection [10]

In conclusion, at present no evidence exists to support the predictions of the "HIV" theory of AIDS.

The cornerstone of the "HIV" theory of AIDS is:
(i) "HIV" kills the T4 cells (AID).
(ii) The decrease in T4 cells causes the clinical syndrome (S).

But, according to Nicholas Bennett ("HIV mechanism revisited", 2 October 2005):

(i) "What is important is that HIV can induce a decline in CD4 counts, not that all low CD4 counts are due to HIV…HIV is cytotoxic to T cells, in vitro at least - in vivo data less convincing"
(ii) "It's not so much that all individuals with low CD4 counts should get AIDS OIs, or that all people with AIDS-OIs will have a low CD4 count."

Here Nicholas Bennett himself has demolished the cornerstone of the "HIV" theory of AIDS.

The predictions of the oxidative theory of AIDS

1. AIDS would remain restricted to the risk groups.

2. The only sexual act leading to AIDS and a positive antibody test is a very high frequency of receptive anal intercourse in either sex.

3. Both antibody positive and antibody negative drug users will develop AIDS (with a higher frequency in the former) and that not only individuals who use dirty needles but also those who use clean needles or even non-parenteral drugs will develop positive antibody tests.

4. In Africa there is neither a new disease, AIDS, nor a new virus, "HIV".

5. The decrease in T4 cells is not the "hallmark" of either "HIV" infection or the clinical syndrome, that is the decrease in T4 cells is not "HIV" specific and is neither necessary nor sufficient for the syndrome to appear, that is, the clinical syndrome is not the result of decreased T4 cell numbers.

6. A most important prediction was that the tissues of AIDS patients and those at risk would be oxidised in general, and in particular they would have low sulphydryl (SH) group levels.

7. AIDS can be prevented and treated with antioxidants.

8. The phenomena which are said to prove infection with "HIV" are the result of oxidation and not infection with a unique retrovirus, "HIV".

In "Looking back on the oxidative stress theory of AIDS"
http://www.theperthgroup.com/CONTINUUM/lookingback.html all these predictions are discussed in detail and ample evidence is presented which indicates that the predictions have been fulfilled.

In our rapid response "More on oxidation - the primary cause for AIDS and "HIV", 1 February, we put to Nicholas Bennett 5 questions, to which he responded in his "Re: More on oxidation - the primary cause for AIDS and "HIV"", 2 February, where he wrote: "In response to their questions:

Q1 Does not make sense, since "oxidized tissues" does not necessarily equate to increased SH levels. However it does appear that cellular redox may be affected, if they want to use the correct terminology. A qualified yes."

We wonder if Nicholas Bennett has read and understood our simple question: "(a) The tissues of AIDS patients and those at risk are oxidised (have decreased SH levels)? Yes or no." Note that we wrote "DECREASED SH LEVELS" and not "increased SH levels" as Nicholas Bennett wrote. Surely anyone with even a rudimentary knowledge of SHs, redox and oxidation will realise the intimate relationship between the three. We find it incredible that these three terms have been used repeatedly in this debate and Nicholas Bennett still appears not to be aware of their definitions and relationships. Furthermore this doesn't seem to stop Nicholas Bennett making "authoritative" arguments

concerning them. Neither does he appear to be aware that AIDS patients and those at risk have decreased not increased SH levels.

Nicholas Bennett wrote: "Q2 SH levels only predict survival because...

Q3 SH levels are associated with low CD4 T cell counts, and CD4 T cell counts predict survival. So yes on both counts, but since HIV causes a loss of CD4 T cells due to rapid cycling this doesn't mean HIV doesn't cause AIDS. SH levels alone do not predict much since in the absence of HIV infection."

We are glad that Nicholas Bennett's answer is "yes" to both questions. However, we repeat would Nicholas Bennett please tell us where is the evidence that "HIV causes a loss of CD4 T cells due to rapid cycling"? Is the loss of CD4 due to "rapid cycling" killing or something else? (AND PLEASE PROVIDE US WITH WELL DOCUMENTED REFERENCES)

Nicholas Bennet wrote: "Q4 HIV can be detected in culture without any use of oxidants and doesn't require "antioxidants" to be inhibited. Most tellingly, since many seem to consider AZT as an oxidizing agent, it seems ironic that AZT inhibits HIV replication in culture of non-stimulated T cells [3] The answer is no on both parts. Please note this data is over 15 years old - one wonders if the Perth Group chose to ignore it during their extensive literature searches. It was the earliest paper I found in the single PubMed search I undertook to confirm this, so was hardly difficult to discover."

Would Nicholas Bennett please provide references to support his claim that "HIV can be detected in culture without the use of oxidised cells or oxidants and "antioxidants" do not cause its inhibition". From his statement we wonder if Nicholas Bennett thinks that both Montagnier and Gallo are wrong. [6 11] Regarding his reference 3, we wonder if Nicholas Bennett actually read either the paper or our analysis of it in our AZT critique. If he actually read our AZT paper he would have seen our detailed analysis of this paper. According to the authors, "AZT inhibits HIV replication" only at cytotoxic levels. They wrote "our results showed that complete DNA copies of the viral genome were formed in the presence of AZT…Whether virus spread occurs by cell-free virus or by cell-to-cell contact, cultures treated with 25mM AZT eventually produced as much virus as the non-drug-treated infected cultures."

Nicholas Bennett wrote: "Q5 I have not seen data looking directly at SH levels and viral load, but since SH levels correspond to rapid T cell cycling in response to HIV, one might agree that this could happen. Logically, yes."

We are amazed at Nicholas Bennett's response which is totally unscientific and irresponsible without looking at the data. So we wonder how he can simply "pull" his "logically, yes" out of his hat. Once again we ask Nicholas Bennett where is the evidence (a) that there is a rapid T cell cycling in "HIV" individuals; (b) the cycling is due to "HIV" and not to SH decrease?

Responding to our question: "If the answers to questions (a-e) [his Q1-Q5] are yes, does it not mean then that the presently available evidence provides significant support for our non-retroviral theory of AIDS and "HIV"? Yes or no". Nicholas Bennett responded: "Since the answers to Q 1 through 5 are not all yes, the Perth Group's conclusion does not follow. In fact, it would not follow anyway since they do not rule out the alternative possibility that HIV is causing the raised SH levels."

We wonder when Nicholas Bennett will realise that we have never nor anybody else (apart from him) claimed that "HIV is causing the raised SH levels". Since the reference he gave to support his "no" answer to Q 4 contradicts his claim, unless he has other references which actually support his claim, then surely it follows that the answer is to Q4 is "yes" rather than "no".

Given that:

(a) the predictions of the "HIV" theory of AIDS stated by Nicholas Bennett and others have not been realised (Nicholas Bennett himself has provided no scientific evidence showing these predictions have been realised) and Nicholas Bennett accepts that even today there is no evidence which proves the main tenant of the "HIV" theory of AIDS, that is "HIV" kills the T4 cells in AIDS patients and those at risk;

(b) the eight predictions of the oxidative theory of AIDS have been realised (Nicholas Bennett himself has admitted to the realisation of at least four of them);

(c) long after our oxidative theory was put forward and fully aware of it, Montagnier, the "discoverer" of "HIV", "*proposed*" that oxidation plays a "key" role in AIDS and "HIV" expression (emphasis Montagnier's);

then it is surprising that some scientists continue to "beat around the bush" in holding to the "HIV" theory of AIDS rather than pursue the oxidative theory of AIDS whose predictions are supported by scientific evidence.

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Competing interests: None declared

Re: Let them be

22 March 2005

Theo HM Fenton,
Consultant Paediatrician
Mayday University Hospital, Croydon CR7 7YE

Send response to journal:
Re: Re: Let them be

A plea to Nicholas Bennett, Tony Floyd, and Peter Flegg: don't waste your time with the denialists -- you won't change their views.

Perhaps these dissenters are desperately trying to convince themselves that everything's okay. Why not leave them be?

Competing interests: None declared

Yet More on Oxidation – the primary cause for AIDS and "HIV"

23 March 2005

Eleni Papadopulos-Eleopulos,
Biophysicist
Department of Medical Physics, Royal Perth Hospital, Western Australia, 6001,

Valendar F Turner, John Papadimitriou, Barry Page, David Causer, Helman Alfonso, Sam Mhlongo, Todd Miller, Christian Fiala
Send response to journal:
Re: Yet More on Oxidation – the primary cause for AIDS and "HIV"

Yet More on Oxidation – the primary cause for AIDS and "HIV"

In his rapid response "Re: More on Oxidation – the primary cause for AIDS and "HIV" (2 February 2005), Nicholas Bennett wrote: "In reply to the Perth Group.

The cause of their "oxidative status" is simply rapidly cycling T cells, in response to chronic HIV infection."

We repeat, would Nicholas Bennett now agree with us that: (a) individuals belonging to the AIDS risk groups are constantly subjected to semen, factor VIII, recreational drugs, including nitrites; (b) all of them are strong oxidants. If so, how is it possible for them not to affect the cellular redox?

Would Nicholas Bennett please tell us where is the evidence that the T cells are rapidly cycling in response to chronic "HIV" infection and that this cycling is the cause of oxidation? It is a fact that individuals belonging to the AIDS risk groups are exposed to nitrites, factor VIII, recreational drugs and semen and that all of them are strongly oxidising agents. It is also a fact that one of the consequences of malnutrition is oxidation? One wonders why Nicholas Bennett and all the other "HIV" experts ignore this obvious fact to which we have been drawing their attention since the beginning of the AIDS era.

Nicholas Bennett wrote: "I am not aware at all that the very same kit (provided by the same manufacturer) is interpreted differently in different countries. The "same" test may of course be manufactured in different ways by various companies."

It is pitiful that Nicholas Bennet, a self confessed protagonist of "HIV" antibody testing, is not aware of this elementary and extremely important fact. He should make enquiries of any one of the companies that manufacture "HIV" Western blots antibody test kits. For example, one of the technical managers listed at Genelabs Diagnostics Pty. Ltd. 85 Science Park Drive #04-01, The Cavendish, Singapore Science Park Singapore 118259 Singapore.

Nicholas Bennett wrote: "HIV isolation is not the only way to ensure detection, since culture will only work if an entity is capable of replication.

It is the same reaction in some culture detection methods (others use RT-activity) but the reaction is of course reversed! To most people, being able to detect antibodies from the host AND antigen from cultures from the host is highly indicative of an infection. Most people clearly do not include the Perth Group!"

We repeat, would Nicholas Bennett please tell us how he and "most people" can obtain the "HIV" antigens without "HIV" isolation and determine the specificity of the "HIV" antibody test without comparing the antibody tests with a gold standard which can only be "HIV" isolation?

Note: We repeat that "being able to detect antibodies from the host AND antigen from cultures" are one and the same reaction. And thus "culture" is not synonymous with "HIV" isolation and cannot be used as a gold standard for the antibody tests.

Nicholas Bennett wrote: "Since the sequences detected in viral load are those of HIV, by definition viral load IS good evidence for ongoing viral replication."

This appears to be his reply to our request: "Would Nicholas Bennett please give us a few references where it has been shown that "viral load" means "HIV" infection and that a decreased "viral load" means inhibition of "HIV" replication." We wonder why he did not provide any references? Where is the evidence that "the sequences detected in viral load are those of HIV"? Would Nicholas Bennett please tell us if viral load proves "HIV" infection, why isn't anybody using it as a test for "HIV" infection? And why does the CDC recommend against this practice? Thus:

"*In adults, adolescents, and children infected by other than perinatal exposure, plasma viral RNA nucleic acid tests should NOT be used in lieu of licensed HIV screening tests (e.g., repeatedly reactive enzyme immunoassay)*" (emphasis in original).[1]

Nicholas Bennett wrote: "The Perth Group themselves have shown that antivirals inhibit HIV replication. In their paper "A Critical Analysis of the Pharmacology of AZT and its Use in AIDS " they cite literature showing reasonably well that the percentage of cells containing HIV-DNA does not change under antiretroviral therapy. However, they don't compare the situation with UNTREATED HIV patients, in which case the percentage of HIV-DNA containing cells increases [1]. Additionally, un-integrated DNA (a true measure of active HIV replication, as opposed to the more static proviral DNA load) decreases significantly while on therapy [2]."

Our paper was about AZT and we provided evidence that AZT does not inhibit "HIV" replication. The title of his reference 1 "Levels of HIV-infected peripheral blood cells remain stable throughout the natural history of HIV-1

infection" which refers to untreated "HIV" patients is self-explanatory. In this paper the text reads "PBMC HIV-1 DNA did not correlate with major indices of disease progression, including time following primary infection, time before reaching a CD4 cell count less than $200x10^6$/l, and time before death. The number of PBMC harbouring HIV-1 provirus was relatively constant throughout the clinical stages of HIV-1 infection".

In regard to treated patients, they wrote: "the mean HIV-1 DNA level of specimen obtained during antiretroviral therapy was slightly higher than in the absence of antiretroviral drugs."

Note:

Since: (a) by definition all the anti-"HIV" drugs used at present do not inhibit "HIV" replication, they can decrease the "viral load" only by decreasing the number of newly infected cells, that is the "HIV" DNA; (b) there is no decrease in "HIV" DNA with "HIV" treatment[2] it means that the "anti-retroviral" do not have any effect on "HIV" or that either "HIV" DNA, RNA or both are not "HIV" specific.

Discussing their findings the authors of his reference 2 wrote: "Because of the limitations of our dataset (10 patients), additional studies are needed to assess the impact of HAART on the PBMC HIV-1 DNA in larger and more diverse populations…In a previous cross-sectional study, which analysed the integrated and total HIV-1 DNA load in resting CD4 T cells from infected patients receiving HAART, levels of unintegrated HIV-1 DNA 28-fold higher than integrated HIV-1 DNA were found"

Once again Nicholas Bennett fails to provide references supporting his claims and in fact "throws up" references that contradict his claims. The purpose of this debate is to solve a scientific problem, namely, "What is the cause of AIDS?" If he aims to argue for the "HIV" theory of AIDS, then his continuous contribution to this debate with long (sometimes unrelated) yarns and lack of scientific documentation are not helpful.

Nicholas Bennett wrote: "Rather ironically the Perth Group's AZT critique appears to support its use as an antiretroviral."

This is absurd. Would Nicholas Bennett quote where in our AZT critique have we presented evidence to " support its use as an antiretroviral"? Has Nicholas Bennett actually read our AZT critique?[3]

In his rapid response to James Whitehead "Re: Re: More on Oxidation – the primary cause for AIDS and "HIV"" (8 February 2005), Nicholas Bennett wrote: "I do not deny that AZT affects the mitochondrial control of the redox state, but I find it ironic that if so it cannot possibly fit with the Perth Group's assertation that HIV is only induced under oxidative conditions, since AZT inhibits HIV replication [1, 2, 3, 4]. Most importantly it does so selectively to cellular toxicity."

Similarly we wonder if Nicholas Bennett has actually read the references he has provided. The first three were analysed in our AZT paper.[3] Although we have not read the fourth we note that in the abstract there is no mention of the effect of AZT on "HIV" replication. Everybody should know that AZT used in clinical practice is non-phosphorylated and, according to the authors of Nicholas Bennett's first reference, "the unphosphorylated compound [AZT] does not inhibit reverse transcriptase ["HIV"] per se". Nicholas Bennett gave an incomplete title for his second reference, the complete title is: "Phosphorylation of 3'-azido-3'-deoxythymidine and selective interaction of the **5'-triphosphate** with human immunodeficiency virus reverse transcriptase" [emphasis ours]. There are important differences between the non-phosphorylated and triphosphorylated AZT, including redox. The sentence "Most importantly it does so selectively to cellular toxicity" is another example of Nicholas Bennett's inane and meaningless yarns.

In the same rapid response to James Whitehead, Nicholas Bennett wrote: "I cannot explain what is meant by oxidative stressors, since this seems to be a phrase most often used by the Perth Group!"

We have never used the phrase "oxidative stressors". Nor do we intend to use it. It is Nicholas Bennett who used it, and we have asked what does he mean by "stressors".

Getting back to his rapid response of 2nd February 2005, Nicholas Bennett wrote: "In response to their questions:

Q1 Does not make sense, since "oxidized tissues" does not necessarily equate to increased SH levels. However it does appear that cellular redox may be affected, if they want to use the correct terminology. A qualified yes."

We wonder if Nicholas Bennett read and understood our simple question: "(a) The tissues of AIDS patients and those at risk are oxidised (have decreased SH levels)? Yes or no." Note that we wrote "DECREASED SH LEVELS" and not "increased SH levels" as Nicholas Bennett wrote. Surely anyone having even a rudimentary knowledge of SHs, redox and oxidation will realise the intimate relationship between the three. We find it incredible that these three terms have been used repeatedly in this debate and Nicholas Bennett still appears not to be aware of their definitions and relationships. Furthermore this doesn't seem to prevent Nicholas Bennett making "authoritative" arguments concerning them. Neither does he appear to be aware that AIDS patients and those at risk have decreased not increased SH levels.

Nicholas Bennett wrote: "Q2 SH levels only predict survival because...

Q3 SH levels are associated with low CD4 T cell counts, and CD4 T cell counts predict survival. So yes on both counts, but since HIV causes a loss of CD4 T cells due to rapid cycling this doesn't mean HIV doesn't cause AIDS. SH levels alone do not predict much since in the absence of HIV infection."

We are glad that Nicholas Bennett's answer is "yes" to both questions. However, we repeat would Nicholas Bennett please tell us where is the evidence that "HIV causes a loss of CD4 T cells due to rapid cycling"? Is the loss of CD4 due to "rapid cycling", killing or something else? (AND PLEASE PROVIDE WELL DOCUMENTED REFERENCES)

Nicholas Bennet wrote: "Q4 HIV can be detected in culture without any use of oxidants and doesn't require "antioxidants" to be inhibited. Most tellingly, since many seem to consider AZT as an oxidizing agent, it seems ironic that AZT inhibits HIV replication in culture of non-stimulated T cells [3] The answer is no on both parts. Please note this data is over 15 years old - one wonders if the Perth Group chose to ignore it during their extensive literature searches. It was the earliest paper I found in the single PubMed search I undertook to confirm this, so was hardly difficult to discover."

Would Nicholas Bennett please provide references to support his claim that "HIV can be detected in culture without the use of oxidated cells or oxidants and that "antioxidants" do not cause its inhibition". From his statement we wonder if Nicholas Bennett thinks that both Montagnier and Gallo are wrong.[4 5] Regarding his reference 3, we wonder if Nicholas Bennett actually read either the paper or our analysis of it in our AZT critique. If he actually read our AZT paper he would have seen our detailed analysis of this paper. According to the authors, "AZT inhibits HIV replication" only at cytotoxic levels. They wrote "our results showed that complete DNA copies of the viral genome were formed in the presence of AZT…Whether virus spread occurs by cell-free virus or by cell-to-cell contact, cultures treated with 25mM AZT eventually produced as much virus as the non-drug-treated infected cultures."

Nicholas Bennett wrote: "Q5 I have not seen data looking directly at SH levels and viral load, but since SH levels correspond to rapid T cell cycling in response to HIV, one might agree that this could happen. Logically, yes."

We are amazed at Nicholas Bennett's response which is totally unscientific and irresponsible without looking at the data. So we wonder how he can simply "pull" his "logically, yes" out of his hat. Once again we ask Nicholas Bennett where is the evidence (a) there is a rapid T cell cycling in "HIV" individuals; (b) the cycling is due to "HIV" and not to SH decrease?

Replying to our question "If the answers to questions (a-e) [Nicholas Bennett's Q1-Q5] are yes, does it not mean that the presently available evidence provides significant support for our non-retroviral theory of AIDS and "HIV"? Yes or no". Nicholas Bennett replied: "Since the answers to Q 1 through 5 are not all yes, the Perth Group's conclusion does not follow. In fact, it would not follow anyway since they do not rule out the alternative possibility that HIV is causing the raised SH levels."

We wonder when Nicholas Bennett will realize that neither we nor anybody else (apart from him) has ever claimed there are raised SH levels in AIDS patients and those at risk or that "HIV is causing the raised SH levels". Since the reference he gave to support his "no" answer to Q4 contradicts his claim, unless he has other references which actually support his claim then surely it follows that the answer is "yes" rather than "no" to Q4 as well.

Nicholas Bennett wrote: "I'm surprised that the Perth Group state that "scientific thought does not count" when all they have provided to the world of HIV/AIDS research is opinions and scientific thought. One clearly has to add caveats to a dogmatic statement e.g. "HIV replication requires stimulation in some cell lines but not all". The caveat neatly destroys their hypothesis that stimulation is required for HIV expression - perhaps that is why they choose not to use it. Another would be "SH levels correlate with progression to AIDS, but perhaps because they are linked to CD4 T cell count which is itself independently linked to AIDS". The loss of CD4 T cells makes rather more immunological sense than a non-specific affect on global cellular redox."

Once again Nicholas Bennett takes only a phrase of what we wrote rather than quoting it in its proper context. We wrote "He [Nicholas Bennett] should not debate if he doesn't have any scientific evidence. Opinions and/or "scientific thought" do not count." So we repeat that without any supporting scientific evidence his "scientific" thoughts and/or opinions do not count.

We would like to point out to Nicholas Bennett that "stimulation is required for "HIV" expression" is not our hypothesis but an experimental fact. If he chooses to dispute this, would he please give us the references to support his "caveats". It appears that again Nicholas Bennett has missed the fact there is a better correlation between SH levels and progression to AIDS independent of CD4 T cell counts, than there is a correlation between CD4 T cell counts and progression to AIDS.[6]

Nicholas Bennett wrote: 'Perhaps the Perth Group can return the favour by responding to the following questions:

Q1 Why should a sexually transmitted disease be expected to be equally bi-directionally transmitted? There is no reason to assume this to be the case, especially considering the animal data [4], the fact that a male inoculum is far larger than a female's, and the fact that homosexual males are a largely sexually distinct risk group."

We never claimed that sexually transmitted diseases are "equally bi-directionally transmitted", nor do we expect such transmission. What we have claimed and supported with many references is that there is no evidence, even today, that "HIV" is bi-directionally sexually transmitted. Furthermore, we specifically gave Nicholas Bennett references showing that "HIV serology", like pregnancy, is sexually acquired but not sexually transmitted. Nicholas Bennett has failed to address this point. For more than 20 years the "HIV"/AIDS experts have claimed that humans are infected with a bi-directionally sexually transmitted virus, "HIV", and that by now more than 50 million individuals have "been" infected by this mode. Incredibly, the only reference Nicholas Bennett could provide (ref. 4) in support of his claim, is a paper on murine retroviruses.

Nicholas Bennet wrote: "Q2 Why do they say that Montagnier failed to distinguish retroviral RT from mitochondrial DNA polymerase, when his 1983 "isolation" paper clearly states the opposite?"

Montagnier detected RT activity in 3 cultures. The only statement regarding its relationship to "HIV" was "That this new isolate [RT activity] was a retrovirus was further indicated by its density in a sucrose gradient, which was 1.16". However, by now Nicholas Bennett should be fully aware that Montagnier's 1.16 band contained no particles which looked like retroviruses. This must mean the RT activity detected in the 1.16 band could not be "HIV".

Nicholas Bennett wrote: "Q3 Can they show that individuals treated with chemotherapy and "other oxidizing agents" develop a progressive, specific decline in the single subset of CD4 T cells which is reversed by the addition of nucleoside analogue (in the case of HIV, RT inhibitor) medications?"

Individuals exposed to oxidising agents do develop a decline in the subset of CD4 T cells. See HERE. It is difficult to see how the second part of Nicholas Bennett's question contributes to this debate. If he want to claim that increase in T4 cells, if any, after treatment with RT inhibitors proves that the cause of the decrease in AIDS patients is "HIV", then he is wrong. We and others (Rapid Responses for Mhlongo and Maduna http://bmj.bmjjournals.com/cgi/eletters/328/7438/523-b#55304) have presented evidence that the increase in T4 cells by antiretrovirals, if any, may be due to a mechanism unrelated to any "HIV" effects. If the increase in T4 cells in "HIV" infected individuals is due to an effect on HIV replication then how does Nicholas Bennett explain the increase of T4 cells by AZT in non-infected individuals? Which may approach double over pre-treatment levels? See

1. Levy JA, Ramachadran B, Barker E, Guthrie J, Elbeik T. Plasma viral load, CD4+ cell count and HIV-1 production by cells. *Science* 1996;271:670-671.

2. Milazzo L, Vaira LM, Cremoni L. CD4+ lymphocyte count variations in HIV-negative subjects treated with zidovudine. *AIDS* 1996;10:1444-5.

How does he know the same mechanism does not operate in "infected" individuals? And what is his proof?

Nicholas Bennett wrote: "Q4 Do they accept the National Cancer Institute's statement that Kaposi's Sarcoma is massively increased in frequency in immunosuppressed people?"

We agree that Kaposi's Sarcoma is "massively increased in frequency in immunosuppressed people". But surely Nicholas Bennett is aware that everyone has accepted that the cause of Kaposi's Sarcoma is not immunosuppression.

Nicholas Bennett wrote: "Q5 Do they accept that a RT activity level in a culture spiked with virus compared to an uninfected culture is therefore due to the presence of the virus as per the method of Potts? [5]"

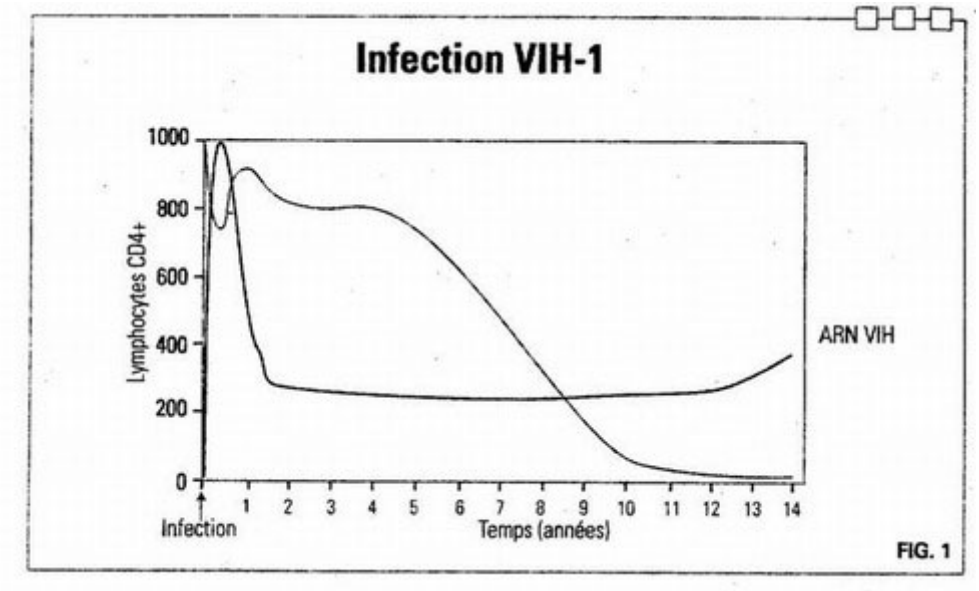
The answer is yes provided that you put in a virus which has been shown to have RT and you put nothing else which also has RT or can activate cellular RT.

Nicholas Bennett wrote: "Q6 If the anti-HIV antibodies are non-specifically induced and are caused by the same thing that causes AIDS, why does lower anti-HIV antibody levels correlate with worse progression? [6]"

If by this question, he implies that lower anti-"HIV" antibody levels lead to increased "HIV" levels and thus worse progression, then he contradicts himself as seen in one of his responses to Peter Duesberg where he wrote "The thing is, antibodies aren't the main antiviral response so to use them as the prime rationale for assuming immune control of a virus is wrong…The fact that antibodies may exist and may bind doesn't mean they will necessarily neutralize nor control infection." [http://deanesmay.com Extract Dean's World 8 February 2005] We agree that antibodies do not neutralize viruses. Responding to Peter Duesberg, Nicholas Bennett also stated that " …the antibody levels drop just prior to clinical AIDS" and that this "may also be simply an effect of the B cell dysfunction." [http://deanesmay.com Extract Dean's World 8 February 2005]

Nicholas Bennett wrote: "Q7 Why does HIV-specific RNA levels correspond to the rate of CD4 T cell loss? [7]"

We agree with James Whitehead's response to this ("Re: Re: More on Oxidation – the primary cause for AIDS and "HIV", 7 February 2005). Montagnier, the "discoverer" of "HIV" does not seem to agree with Nicholas Bennett. At the European Parliament Meeting, 8 December 2004, he showed the following graph:



As can be seen from the 2nd to the 11th year the "HIV" specific RNA levels are approximately constant while the T4 cell number decreases approximately 30-fold from about 800 to 30.[7]

Nicholas Bennett wrote: "Q8 If non-HIV stimuli cause spontaneous antibody, RNA and antigen formation coincident with CD4 T cell decline, what possible genetic mechanism can explain this spontaneous appearance in a subset of host cells?"

We have repeatedLY claimed that oxidation will lead to genetic and PHENOTYPIC changes and have presented scientific evidence to support this claim. See http://www.theperthgroup.com/CONTINUUM/pgvsduesbergreward.html

Nicholas Bennett wrote: "Why not consider an infectious retrovirus?"

An infectious retrovirus has been considered for more than 20 years wasting a lot of people's time and billions of dollars and still nobody including Nicholas Bennett can give any scientific evidence showing that the "antibody, RNA and antigens" are those of a retrovirus "HIV" or that "HIV" causes a decline in CD4 T cells.

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Competing interests: None declared

Reply to Fenton: The Outlandish Anomalies of the 'HIV' Hypothesis

24 March 2005

Alexander H Russell,
Writer/artist/philosopher
WC1N 1PE
Send response to journal:
Re: Reply to Fenton: The Outlandish Anomalies of the 'HIV' Hypothesis

Theo HM Fenton stated: "…the views repeated ad nauseam by the HIV- denialists (and the anti-vaccine propagandists) are so outlandish that nobody could possibly take them seriously."
On the contrary: the views expressed by the 'HIV' Monotheists are so outlandish that nobody could possibly take them seriously as you will notice when reading the following questions concerning the anomalies, contradictions and paradoxa regarding the redundant 'HIV/AIDS' hypothesis:
a) Why is 'HIV' supposed to spread like wildfire in Africa horizontally (heterosexually) but not this way in the West? Why is 'HIV' prejudiced against 'infecting' white heterosexuals in the West? Why is 'HIV' so racist?
b) Young girls in the UK are practicing 'unsafe' sex yet are hot becoming 'HIV' positive - why not? (Sky News: 24th March. 2005). Teenage STD rates are rising in the UK and USA but there is no endemic 'HIV' epidemic in this group. Why not?
c) Why is 'HIV' alleged to have an elastic incubation period of 10 to 30 years? If 'HIV' is meant to be a 'lenti-virus' why is 'HIV' suddenly supposed to be a 'quick-virus' in Africa? How would a 'lenti-virus' survive in nature? Can they explain the eternally long, and unpredictable, incubation period of 'HIV' between so-called 'infection' and 'disease'?
d) Why does 'HIV', unlike any other pathogenic virus, only causes disease in the presence of neutralizing antibodies?
e) What is their evidence that 'HIV' is destroying t-cells by 'infection'? There is still no empirical evidence or scientific reference paper for this hypothetical assumption - it is no more than pure speculation and wishful (non) thinking.

Supporters of the 'HIV/AIDS' Hypothesis - like Fenton, Bennett, Flegg, Floyd (just like politicians and theologians) – can never directly answer questions put to them by the dissenting 'HIV' critics and:

1. say blatantly untrue things
2. say irrelevant things and make unfounded assertions
3. engage in emotive ad hominem attacks
4. speculate and make assumptions
5. ignore and distort points made by the opposition
6. invent outlandish science fictions
7. shift the goal posts and make up the rules of the game

Fenton unwittingly demonstrates that 'HIV' Momtheism is more about blind faith than hard science. Today the 'HIV' Industry has much more to do with religion and politics than science, as Phillip Johnson stated: "For essentially political reasons, HIV science has been ruled by unexamined assumptions. It is time at long last to have the scientific debate that wasn't allowed to occur ten years ago. Let the politics be put aside, and let the science begin."
I would like to point Fenton to a typical example of what happens when one directly questions the simple-minded 'commonsense' folk-law of 'HIV' Belief as Kary Mullis, the 1993 Nobel Laureate in Chemistry, did. Mullis was writing a report on the use of his PCR invention for 'HIV', when he came across the phrase, 'HIV is the probably cause of AIDS'. In his own words:
"I asked the guy sitting beside me, "What is the support for that, what's the reference?" And he said, "You don't need a reference, everybody knows that."

"I assumed there must be such a reference, and that there might be a controversy over who got credit for it, because I was under the impression that Gallo and Montagnier might have been fighting over who had first shown that HIV was the cause of AIDS.... I went back over their early papers, and found that neither of them had shown that HIV was the probable cause of AIDS."
"And then finally, Luc Montagnier came to San Diego, and gave a talk, and I thought, this guy will know. [laughter] After the meeting I asked him, and he first mentioned the CDC report, and I said I had already looked at it, that it wasn't what I was looking for - that I wanted a scientific paper that would support the notion that HIV is the probable cause of AIDS, not the consensus of a bunch of people who'd already begun looking at it.
He said, "Well, let's see ..." (and there was a little knot of people around us at that point, thinking, the man must have an answer to that question), and he said, "Why don't you quote the SIV work?" And I said to myself, "Oh my god! There really isn't such a paper, there can't be, or he wouldn't have to refer ... to a virus that might kill a monkey ... to illustrate the probability that HIV is the cause of AIDS!"
(Reference: The Presentations at the HIV Symposium at AAAS Conference; Charles A. Thomas Jr., Kary B. Mullis, and Phillip E. Johnson. "What Causes AIDS: It's an Open Question". Reason, June 1994).

Competing interests: None declared

Re: Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

24 March 2005

torsten engelbrecht,
journalist
20359 hamburg
Send response to journal:
Re: Re: Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype
dear mr. bennet,
thanks again for your reply. please allow me to explain why you are mistaken again:
1. the question if the hiv hypothesis and its basis has been proven is the most important issue in aids science. so don't "coil up", just answer it aor admit that that there is no such proof!
2. the supporters of the hiv hypothesis seem to love putting the responsibilities upside down. in fact, those who make the claim that the hiv hypothesis has been proven/is correct bear the burden of proof. so again: don't "coil up", just deliver the clear-cut proofs (studies)!
3. again: don't "coil up", just deliver the clear-cut proofs (studies)!
4. the issue if the hiv hypothesis makes sense or not is not a quastion of originality!!! again and again: don't "coil up", just deliver the clear-cut proofs (studies)!
5. again and again and again: don't "coil up", just deliver the clear -cut proofs (studies)! just one single study for the 4 most important theses of the hiv hypothesis!
6. okay, here we are. obviously, we agree that there is no clear-cut proof for the hiv hypothesis. epidemiology is not such clear-cut proof because it's not causation it's just correlation. the problem: even the correlation of the hiv hypothesis is a very bad one!
6.again and again and again and again: don't "coil up", just deliver the clear-cut proofs (studies)!
7. there is not even one study that proves that hiv kills cd4 cells. or do you know one?
8. gall speaking at that nida-meeting was referring to oppers, not to hhv8! "it has been shown that llicit drugs may alter the normal immune functions", you can read even in the journal of aids a couple of months ago. so it's just fact that poppers, crystal meth or so suppress the immune system.
concerning your "pseudo-science"-attack. i think it makes no sense at all to shout. just let us stick with the facts. put them on the table and that's all.
concerning your claim "it is also odd that the use of anti- retrovirals DOES prolong life". there are many proofs to DISprove your statement. to make it short: the indispensiable precondition for this statement is just missing: placebo- controlled studies! so you just

cannot say that haart or whatever anti- retroviral therapy prolong live(s). there is practically just one placebo study: the fischl trial in 1987. but this trial was totally flawed, or as the swiss newspaper "weltwoche" wrote: "the fischl trial was a gigantic botch". the only thing for sure is that's highly toxic and immune suppressive, and that it can kill and that it kills people - you can even read that on the package inserts. so instead of taking these toxins people should think about their lifestyle: did i take too many recreational and/or medical drugs for too much time? and/or what about my eating, sleeping etc. habits? of course, many poor people in poor countries don't have the choice: they have to stick with bad and few food. but in this case politicians and mighty poeple should think about their politics: e.g., why do we spend 900 billion $ a year worldwide for war "toys"? keeping also in mind that only a small part of that incredibly huge amount of money was needed to give all people on earth enough good food to eat.
best
torsten engelbrecht
www.torstenengelbrecht.com
Competing interests: None declared

Re: Re: Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

24 March 2005

Nicholas Bennett,
Infectious Disease Postdoc/Clinician
Department of Pediatrics, University Hospital, Syracuse NY

Send response to journal:
Re: Re: Re: Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

With regards to Herr Englebrecht's responses:

If referring him to previously outlined posts covering the past year is "coiling up" then so be it, but I suggest he read the entire list of rapid responses before assuming that anything I say is unsupported.

For example a very quick search for HIV mediated cell death revealed the following paper.

"Killing of Primary CD4+ T cells by non-syncitium inducing macrophage -tropic human immunodeficiency virus type 1".

Yu et al, PNAS Vol 91 pp 10237-10241 Oct 1994

Once again I suggest that if he (or others) believes something to be true simply on the basis of a few dissident websites, that perhaps they should look to see if they have not been seriously mislead.

Another example is Getchell et al, J Clin Microbiol. 1986 Apr;23(4):737-42."Continuous production of a cytopathic human T- lymphotropic virus in a permissive neoplastic T-cell line." They cloned a cell line that is resistant to HIV-mediated cell death, even though over 90% of the parental cells were killed after infection.

The latter paper is available online at
http://www.pubmedcentral.nih.gov/picrender.fcgi?artid=362827&blobtype=pdf

I would also say that statements like "epidemiology is not such clear -cut proof because it's not causation it's just correlation" also highlight the problem. The correlation is PART of the causation proof. You cannot simply choose to ignore each small aspect of the proof and then ask for the proof to be presented without key components. It's a bit like saying

that a car engine is useless without the wheels, so the engine cannot make the car move. Now show me a car moving without an engine.

The fact that HIV infection precedes a CD4 T cell decline that is not seen in any other human condition is proof enough. It isn't even required that HIV directly kills the T cells, even though it does. Smoking after all predisposes people to lung infections, but it doesn't directly introduce the bacterial pathogen... This is why epidemiology is in fact far more than "just" correlation. You can prove beyond reasonable doubt that a factor is bad or good without having a clue as to why that might be the case.

The studies requested have all been previously discussed here. http://bmj.bmjjournals.com/cgi/eletters/326/7387/495 The post entitled "HIV mechanism revisited" is a fully referenced explanation as to how HIV causes AIDS, according to my current understanding.

Nick Bennett njb35@cantab.net

Competing interests: None declared

Re: Re: Re: Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

1 April 2005

Torsten Engelbrecht,
journalist
20359 Hamburg

Send response to journal:
Re: Re: Re: Re: Re: Re: Analysis: the properness of the HIV hypothesis is a media hype

"measurement of t- and b-cells and their subsets in diseases has no clinical meaning." thomas fleming and david demets

"that hiv uses cd4 as its primary receptor, and that cd4+ t-cell numbers decline during aids, are an unfortunate coincidence that have led us astray from understanding the immunopathogenesis of this disease." mario roederer

dear mr. bennett,

the following:

1. i think it makes not that much sense to switch from one question to the other! the main question is: is there evidence for "hiv" directly killing t cells? and the answer is no! you couldn't present any single study, either. so the supporters of the hiv hypothesis are in big trouble because more than 160 billion(!) $ are spent for hiv=aids-research that is based on pure speculation as well as ignorance because no single dollar or euro is spent in order to see if other factors than "hiv" might cause aids(-symptoms) or at least might contribute to the development of (well- known) diseases like aids-ks.

2. there may be some hints that "hiv" kills cd4 cells. but there are also studies showing that cd4 cell counts make no sense in the context of aids diagnosis. not only the biggest aids study, the concorde study, called the critical use of cd4 cell counts into question. in this context please allow me to quote from david rasnick's compilation "an abuse of surrogate markers for aids – cd4 cell counts and pcr viral load tests": "at the beginning of the aids epidemic, it was already recognized as probably a mistake to use CD4 as a marker of aids or even a measure of therapeutic effectiveness. in 1981, james goodwin, md, wrote what he called “a diatribe against the measurement of t-cell subsets in human diseases” [1].

his “diatribe” began: 'it’s starting again. the t- and b- cell measures – having run through the sick, the elderly, the young, the pregnant, the bereaved – had finally run out of diseases. each condition was the subject of many reports; so that now, to give but one example, we can conclude with some assurance that t-cell numbers are up, down, or unchanged in old folks. and it’s starting all over again, this time with t -cell subsets.”

'what will they find?' he asked. ‘sometimes the suppressor cell markers will be up and helper cells down; sometimes the suppressor cells will be down and the helper cells up; sometimes they’ll be unchanged--and various combinations of the aforementioned. my strongest argument is this: measurement of t- and b-cells and their subsets in diseases has no clinical meaning.'

'nonimmunologists have naturally assumed that any subject occupying so much journal space must be relevant in some way--a logical but incorrect assumption. and while the identification of t-cell subsets in mouse and man represents a major breakthrough in the understanding of immunoregulation, the enumeration of these subsets in myriad diseases largely represents a waste of time.'

as recently as 1998, mario roederer of stanford university confirmed goodwin’s assessment that an obsession with t-cell subsets in aids patients has been a mistake: '[t]he facts (1) that hiv uses cd4 as its primary receptor, and (2) that cd4+ t-cell numbers decline during aids, are an unfortunate coincidence that have led us astray from understanding the immunopathogenesis of this disease' [2].

prior to roederer’s remarks, the use of the cd4 (t-cell counts) as a surrogate marker of disease progression was also criticized by the authors of the concorde Study, the largest clinical trial evaluating the use of azt: the authors concluded that: 'the small but highly significant and persistent difference in cd4 count between the groups was not translated into a significant clinical benefit. thus, analyses of the time until certain concentrations of cd4 were reached (eg, 200/µL, 350/µL, or 50 % of baseline) revealed significantly shorter times in the def[erred] group. had such analyses been regarded as fundamental, the trial might have been stopped early with a false-positive result. this discrepancy in the differences between imm[ediate] and def groups in terms of changes of cd4 count and of long- term clinical response casts doubt on the uncritical use of cd4 counts as ‘surrogate endpoints’ in trials...' [3].

thomas fleming and david demets have stated that, 'the use of surrogate end points has probably been more intensely discussed in the design and analysis of clinical trials of hiv infection and aids than in any other area' [4]. however, “predictions having an accuracy of approximately 50%, such as the accuracy seen with the cd4 count in the hiv setting, are as uninformative as a toss of a coin.” With regards to clinical trials and fda approval of anti-hiv drugs, fleming and demets have warned that, 'Surrogate end points are rarely, if ever, adequate substitutes for the definitive clinical outcome in phase 3 trials' [4].

indeed, a summary result from a 1993 state-of-the-art conference had previously concluded that the effect of treatment on the most popular surrogate, cd4 cell count, did not accurately predict the effect of treatment on the clinical outcomes, that is, progression to aids or time to death [6].

Nevertheless, with the exception of the early azt clinical trials, all subsequent anti-hiv drug trials and fda approvals have relied exclusively on the measurements of these surrogate markers and not on the real clinical outcomes, such as morbidity and mortality, that matter to most people.

a year later, fleming stated that, 'it is very apparent one cannot simply consider establishment of statistically significant treatment effects on cd4 cell counts to be a valid

surrogate for either of the two clinical endpoints. when the progression to aids/death endpoint was positive, the cd4 endpoint appropriately was significantly positive in 7 of 8 trials; unfortunately however, the cd4 endpoint was significantly positive in 6 of 8 trials in which the progression to aids/death endpoint was negative. the relationship of cd4 effects and survival is even more unsatisfactory. the cd4 endpoint was significantly positive in only 2 of 4 trials in which the survival endpoint was positive; yet it was significantly positive in 6 of 7 trials in which the survival endpoint was negative. in three other trials, survival trends were observed which were in the opposite direction of significant treatment effects on CD4' [7].

3. you and all the other people who take the hiv hypothesis for granted (of course, without having any proof for its basic theses) should also not forget that only hiv=aids-research is being financed. so for example, instead of asking "can mr. russell explain the fact that hiv seropositivity (i.e. infection) produces disruption of lymph node architecture?", you should have asked: what have been done in order to study other factors than "hiv" that may explain this? or: does seropositivity always produce disruption of lymph node architecture? if no, how can that be?

4. moreover, i can only repeat myself: the supporters of the hiv hypothesis are not able to make a neutral approach to the issue. saying: "hiv seropositivity (i.e. infection)..." already implies that (1) hiv has been proven, that (2) antibody tests prove hiv, and that (3) positive test results mean that a person is infected by hiv. but none of these claims have been proven.

5. concerning correlation and causation: first, i am "happy" to hear from you that you admit that there is no clear-cut proof for the hiv hypothesis because there is no single study proving the hiv hypothesis. second, you write that "correlation is part of causation". but the problem with that statement is that the correlation of the hiv hypothesis is a very bad one. instead the hiv hypothesis is highly inconsistent. to make it short, please have a look under alexander russell's recent rapid response (here in this rapid response section) "reply to fenton: the outlandish anomalies of the 'hiv' hypothesis" from 24 march 2005. on the other hand, the drug hypothesis saying that aids can be explained by (1) illicit drugs, (2) anti-retroviral and other medical drugs, (3) malnutrition, and/or re-definition of well-known diseases like tbc into aids is very consistent. so relying on correlation as "proof" would mean accepting the drug hypothesis as the hypothesis that needs to be financed urgently!

6. you wrote: "If referring him [= i] to previously outlined posts covering the past year is 'coiling up' then so be it, but i suggest he read the entire list of rapid responses before assuming that anything y say is unsupported. for example a very quick search for hiv mediated cell death revealed the following paper: 'killing of primary cd4+ t cells by non-syncitium inducing macrophage-tropic human immunodeficiency virus type 1' by yu et al, pnas, vol 91 pp 10237-10241, Oct 1994" but: doing a pubmed search for keywords is very easy. but the important question in this case is: was the "hiv-1" that was added to these cells purified, or was it just the supernatant of a culture, or some product of that after filtering and centrifugation? and also: i am not "assuming" that anythiny you say is unsupported, but i just can't see an answer to my simple questions like: where is the study that proves that hiv causes aids? where is the study that proves that hiv kills cd4 cells? so i am not assuming, but i have to state that you don't have a proof for all these theses!

7. you wrote: "the fact that hiv infection precedes a cd4 t cell decline that is not seen in any other human condition is proof enough. it isn't even required that hiv directly kills the t cells, even though it does. smoking after all predisposes people to lung infections, but it doesn't directly introduce the bacterial pathogen... this is why epidemiology is in fact far more than 'just' correlation. you can prove beyond reasonable doubt that a factor is bad or good without having a clue as to why that might be the case." so my questions are: are you saying that hiv infection always precedes a cd4 t cell decline? how do you

define an "hiv infection"? what proof do you have that any one hiv test has been validated against purification of the virus?

torsten engelbrecht journalist www.torstenengelbrecht.com

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Competing interests: None declared

(Quelle: http://bmj.bmjjournals.com/cgi/eletters/326/7387/495?ck=nck#101125)