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N URGENT APPEAL FOR ACTION

PRESENTED TO:

THE UNITED NATIONS
COMMISSION ON HUMAN RIGHTS
Geneva, Switzerland

March/April 1998

BY:

HUMANITARIAN LAW PROJECT
INTERNATIONAL EDUCATIONAL DEVELOPMENT, INC.,
USA
and
INTERNATIONAL FORUM FOR ACCESSIBLE SCIENCE
IFAS
Switzerland

CO-SPONSORED BY:

CONTINUUM, England

GAY INTERNATIONAL ASSOCIATION (GAIA Trust), England

REGARDING:
THE "HIV-AIDS" DOGMA AND ITS CONSEQUENCES ON HUMAN LIFE

IN MEMORIAM

TONY RAGUSA

ORLANDO GARCIA

BARRY WEISS

MICHAEL CALLEN

MARK ALAMPI

JODY WELLS

KETTY OKULLA

and far too many more

&

ELSA WINIGER-GROEBLI

International Forum for Accessible Science (IFAS)
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Bern, Switzerland March 1998

To Those Who Care

„Nothing is more difficult and nothing asks for more character then to overtly oppose contemporary public opinion and to loudly say „NO!““ *(Kurt Tucholsky)*

As a member of the Non-Government-Organisation (NGO) ***INTERNATIONAL EDUCATIONAL DEVELOPMENT***'s delegation to the United Nation's Commission on Human Rights and Secretary General of the ***INTERNATIONAL FORUM FOR ACCESSIBLE SCIENCE, IFAS***, I would like to share a few words at the beginning about the intentions of going to the UN and how to best use this second edition of the *Public Information Dossier.*

Let us assume just for one moment that all we have learnt about „HIV" and „AIDS" is actually not factual and therefore not true. The consequences on scientific standards would be tremendous. No one would be able to distinguish fact from fiction any longer in future science. Any hypothesis could be claimed as „factual" without sufficient proof. The treatment approaches based on the assumption that „HIV" not only causes „AIDS" but is inevitably fatal would be detrimental. Not only would potentially harmful drugs be exposed to people who would be expected to die prematurely anyway, but under these false presumptions many people would be less careful about the intake of toxic substances. Toxic pharmaceuticals could even be made mandatory treatment under the false assumption that they have the potential to extend life. Such policies would leave those who do not want to take the recommended treatment for whatever reason unprotected from both health and financial viewpoints and could lead to more unnecessary suffering, health damage and premature death.

It is the intention of this document to dismantle both „HIV" and „AIDS", to show that the assumptions in the preceding paragraph are realities. Everything we commonly believe about „HIV" and „AIDS" is based on hypothetical predictions and mostly false.

Despite this „HIV" and „AIDS" remain a global problem. All issues concerning global situations should be disclosed in the highest forum possible. The facts given here refuting the claims of a „scientifically based" „HIV-AIDS" hypothesis surely need to be addressed on a platform where all or most countries' attention can be drawn and people can participate. These scientific conclusions do not take the „AIDS" problem away from us, but rather shift individual understanding from the common assumption of „HIV=AIDS=Death" to the detrimental global consequences growing out of this dogma, and make an international alert even more urgent.

IFAS feels the need to draw the attention of the United Nations to certain facts and their impact on world health, human rights and scientific standards.

Also this second edition of the *Public Information Dossier* is geared towards an understanding of the „HIV-AIDS" paradigm for all people interested. It is, however, written in a rather technical manner to meet scientific criteria in order to be useful to scientific and legal debates.

After a historical ride through the past seventeen years of „AIDS", this document will first address the failings of so-called „anti HIV treatments". This priority comes from the view that the drugs given to individuals labelled „HIV positive" pose the greatest threat to human life in the „AIDS"-struggle. The succeeding papers will then continue to dismantle both „HIV" and „AIDS".

After having carefully considered the information provided in this document, ***IFAS*** and ***INTERNATIONAL EDUCATIONAL DEVELOPMENT*** hope you agree that this data on „HIV" and „AIDS" and the consequences of the hereby refuted „HIV/AIIDS"-*dogma* needs to be addressed at the Commission on Human Rights.

IFAS invites all those remaining doubtful to check the conclusions drawn herein by asking national and international „AIDS" authorities to submit scientific papers which

- show „HIV-isolation" using the standard Pasteur protocol and
- demonstrate in vivo how „HIV" causes „AIDS" (suggesting Koch's postulate of identifying an infectious pathogen).

We would be grateful to be informed of the findings and interpretations of the data.

Sincerely yours,

Michael Baumgartner (editor)
Secretary General IFAS
Humanitarian Law Project
AIDS-co-ordinator

Christine Johnson (co-editor)
Member Public Access Board IFAS

IFAS questions the isolation of „HIV". Without „HIV's" presence the „AIDS" dogma cannot be maintained. For these reasons IFAS refers to both „HIV" and „AIDS" using quotation marks.

Views expressed in this document usually, but not necessarily, reflect the views held by IFAS. All reasonable care has been taken, but to protect itself from legal proceedings, IFAS will not be held responsible for any inaccuracies contained herein.

CONTENTS

SUMMARY

„The problem with the truth is that it is mainly uncomfortable and often dull." (H.L. Mencken)

Let us summarise the work done by experts in this document by including a few of our own thoughts and findings here.

In 1984, the „Human Immune deficiency Virus" („HIV") was declared the „probable" cause of the „Acquired Immune Deficiency Syndrome„ („AIDS") by Dr. Robert Gallo in a press conference called by US Secretary of Health Margaret Heckler. This announcement was made without prior publication of the data or discussion and debate in the scientific community. Shortly after, the word „probable" was dropped. The media simply stated what today is common belief: „HIV=AIDS", without any such proof.

Critics of this hypothesis have since personally invited Dr. Robert Gallo, Prof. Luc Montagnier, the US Centers for Disease Control and the World Health Organisation -- main proponents of the „HIV-AIDS"- hypothesis -- to submit scientific papers that would demonstrate how „HIV" causes „AIDS". **To date no such paper has surfaced.** When reminding them about the outstanding data to back up their claim, we were asked to take the „overwhelming epidemiological data" as such proof. For simple scientific reasons this is not acceptable:

1) **The correlation between „HIV" and „AIDS" has never been 100 %.** Gallo was **only** able to isolate what was considered „HIV" from 26 out of 72 AIDS patients (36 %). Antibodies could **only** be detected in 43 of 49 AIDS patients (88%) (see *Gallo* in *Science, Vol. 224, 1984).* Thousands of patients with clinical „AIDS", who are not found to be „infected" with „HIV" clearly prove, that whatever causes a chronic immune dysfunction can do so with or without „HIV" present (see *Duesberg* in *Biotechnology, Vol. 11, 1992).* Modern research indicates that maybe disease causes retroviruses to occur, rather than the opposite of retrovirus causing diseases in humans (see *Immunology Today, Vol. 16/No 4, 1995).*

2) **Correlation is no proof for causation.** The suspected pathogenic agent has to be found in **all** suspected patients, be **purely** and **properly** isolated and, when induced in a suitable model, causes the exact **same predictable** disease and not a selected number of unrelated ones. An infectious agent is highly active at the time when the patient is sick with the disease, causing the person to be sick. The correlation between „HIV" infected cells and the progression to „AIDS" does not match. „HIV" has been found in the same proportion, whether „HIV positive" individuals are healthy, sick with or even dying of „AIDS" (see *Duesberg 1992).* The correlation of the popular viral load counts and the compared counts of the immune cells CD4 does **not** match either. One can have simultaneously a high viral load and high CD4 count or a low viral load and a low CD4 count.

About 39% of the „AIDS"-defined diseases (wasting disease, Kaposi's sarcoma, dementia and lymphoma) are not due to low CD4-counts (considered „immune deficiency") and should therefore never have been attributed to the claimed detrimental effects of „HIV" (see *Duesberg* in *„Infectious AIDS - Stretching the Germ Theory Beyond Its Limits", International Archives of Allergy and Immunology, 1994:103:118-126).*

Close investigation from independent scientists from all over the world conclude there is no scientific proof of the „HIV=AIDS" model to date!

In 1973, the Pasteur Institute of France designed protocols for retroviral isolation (see *Sinoussi* in *Spectra 4, 1973).* The steps are:

1) Culture of putatively infected tissue.
2) Purification of specimens by density gradient ultracentrifugation.
3) Electron micrographs of particles exhibiting the morphological characteristics -- having condensed inner bodies (cores) and knobs -- and dimensions -- almost spherical in shape and a diameter of 100 - 120 nM -- of retroviral particles at the sucrose density of 1.16 gm/ml and containing nothing else, not even particles of other morphologies or dimensions.
4) Proof that the particles contain reverse transcriptase.
5) Analysis of the particles' proteins and RNA and proof that these are unique.
6) Proof that 1-5 are property only of putatively infected tissues and cannot be induced in control cultures. These are identical cultures, that is, tissues obtaining from matched, unhealthy subjects (similar to AIDS) and cultured under identical conditions differing only in that they are not putatively infected with a retrovirus.
7) Proof that the particles are infectious, that is, when PURE particles are introduced into an uninfected culture or animal, the identical particle is obtained as shown by repeating steps 1-5.

Only 10 years later the Pasteur Institute claimed the isolation of a new retrovirus -- „Lymphodenopathy **Associated** Virus" (LAV) from a **non**-AIDS-patient -- the virus later to be named „Human Immune deficiency Virus" („HIV"), **without** following their own protocol (see *Montagnier* in *Science, Vol. 220, 1983).* Not only has Gallo **never** discovered „HIV" himself, he and Montagnier do not agree on **which proteins should actually be considered „HIV"** (see *Papadopulos* in *CONTINUUM Vol.4/No.3, 1996).*

Despite there never having been proof of „HIV" causing „AIDS", nor an isolated exogenous entity which could be labelled „HIV", „AIDS" has been considered an infectious sexually-transmitted disease. From 1990 to 1992, the proportion of heterosexuals aged 18 - 49 in „high risk" American cities who reported multiple sexual partners **increased** from 15% to 19%, while condom sales **decreased** by 1%, and 65% of the respondents admitted they used condoms **either sporadically or not at all**. Americans are **not** practising safer sex and for this reason teen pregnancies and venereal disease are on the rise. Yet the fraction of Americans assumed to be „HIV positive" has **declined** from an estimated 1 million in 1985 to between 630 000 and 897 000 in 1995 (see *Catania* in *The American Journal of Public Health, Vol.85/No.11, 1995* and read *Hodgkinson's „AIDS, The Failure Of Contemporary Science" 4th Estate, 1996).* „AIDS" from it's first occurrence never behaved like a sexually transmitted disease and cannot therefore be considered one. This is confirmed by a study carried out in Chicago, where 336 out of 395 people diagnosed „HIV positive" (85%) initially reported as „heterosexually infected" had to be reclassified into different transmission categories. **„Most notably, 69% (272 of 395 cases) were reclassified into transmission categories that did not involve heterosexual contact...".** *(see Murphy in Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology 16:122-126; 1997)*

Demonstrating the dilemma of missing the virus, we now see laboratories basing the evaluation of their „HIV-antibody test" on the interpretation of sexual surveys rather than the expected detection of viral infection. Individuals who want to undergo „HIV-antibody testing" have to indicate their sexual orientation **prior** to testing. The Roche „HIV antibody test" used in Italy seems to use such knowledge to interpret indeterminate data (see the *Roche supplement* to test *COBAS Core).* It seems to us that either one is or is not infected -- with the agent to be found matching the specific antibodies (called Gold Standard) -- regardless

of one's sexual orientation!? The **only** way to prove that specific antibodies to „HIV" exist is to compare antibody reactions with the isolated „HIV". If these antibodies are specific, they will **only** occur in the presence of „HIV" and **never** in its absence. To date, such proof has not been reported because **no one** has provided evidence for the isolation of „HIV".

Infection with the „killer-virus of the century" seems only taking place in people's mind rather than physically. Because they have no access to accurate information, hence believe in the unsound information about a hypothesed dangerous infection with a new virus, they demand suitable drug. IFAS encounters the consequences of this lack of understanding which makes people follow hideous treatment protocols also designed for the new combination therapies including protease inhibitors. Risking one's life by undergoing such toxic ordeal is too high a price.

The suspected „HIV protease" is said to belong to the family of aspartyl protease, like pepsin (an enzyme in the stomach), cathepsin D (located inside the cell) and renin (regulating blood pressure). Considering the non-specificity of „HIV protease inhibitors" one might just get a glimpse of even more damage lying ahead for the health of people taking these drugs. The long list of possible toxic effects of pharmaceutical protease inhibitors now includes visual impairment, herpes zoster, abnormal bleeding, unusual fat deposits, changes in body composition: blood sugar, lipid levels and hemolytic anaemia etc.

Despite this questionable development, governments are discussing **mandatory treatment** with protease inhibitors in combination with nucleoside analogues. World-wide it is becoming common practice for AIDS doctors to **pressure** even healthy individuals to go on taking these combinations. It is known however, **only** about 50 % of people taking these cocktails can tolerate them to begin with and despite their toxicity there is still little known about their long-term toxic effects. Pharmaceutically-manufactured „HIV protease inhibitors" **repeat** in many ways the once hailed but seemingly failed „anti-HIV-drug" AZT. Dr. med. Daniel Oertle-Meyer -- a Swiss AIDS-specialist -- openly admits: *„The medications are still not toxic enough for the virus and too toxic for humans"*. The cost, for example in Switzerland, for these new drugs per patient per year is SFr. 20'000 (about $ 13'000). Since the approval is fast tracked - without the usual trials - people become publicly funded guinea pigs with individual financial contributions. We do not question efforts made to understand the mechanisms causing „AIDS". Yet, we do question the money spent on „HIV-research" - in England alone there are £ 290 000 spent for each person who died of „AIDS", while only £ 50 for every death from heart disease, a leading cause of death especially in men world-wide. And while we think support for people with „AIDS" is vital, we think that - again looking at figures in England - 3'400 „AIDS"-care workers for about 1'800 „AIDS" patients is also questionable, especially when looking at the kind of support and information these people actually get (read *Hodgkinson 1996)*!

People seem to fall sick due to a worn out immune system (formerly known as „bad" health) in higher numbers in certain groups and at unexpectedly young ages. That is why IFAS is -- taking this problem seriously -- have asked scientists from relevant fields to start separating fact from fiction, to outline the groundwork and indicate further research that needs to be carried out in order to help all people labelled „HIV positive" and „having AIDS".

It is this document's aim to dismantle the „HIV-AIDS" myth and indicate that the break-down of the immune function fighting disease can be attributed to other risk factors independent from „HIV".

Michael Baumgartner, Secretary General IFAS
March 1998

ABOUT THE AUTHORS

„Men with an extensive belief in their ideas and theories are poorly equipped to make discoveries and therefore make bad observations."
(Claude Bernard)

All papers published in this document are from authors who have challenged common beliefs with great human courage and sacrifice. They have carefully observed the evolving of the „AIDS" dilemma over the years and are well equipped to make sound reflections on the issues surrounding „HIV/AIDS". They have summarised their findings in the following papers, exclusively written for this dossier.

Dr. David Rasnick, has a PhD in chemistry and an extensive background in the making of protease inhibitors. He bases his paper on the presumption that „HIV" exists as a retrovirus - albeit never isolated in humans. He lives in San Francisco, USA. Dr. Rasnick is a member of the Group For The Reappraisal of the HIV/AIDS-Hypothesis.

John Lauritsen, has an extensive background as a survey research analyst. He has been active in the Gay Liberation Movement and he is now a full-time writer, living in Provincetown, USA. Mr. Lauritsen is a member of the Group For The Reappraisal of the HIV/AIDS-Hypothesis.

Dr. Eleni Papadopulos-Eleopulos, biophysicist at the Royal Perth Hospital and Chairwomen of the Board Of Scientists of IFAS and ***Dr. Val Turner,*** a emergency physician at the Royal Perth Hospital and their colleagues from Australia. Dr. Papadopulos and her team have published criticism of the „HIV-AIDS" model -- including the unspecifictiy of HIV-testing, HIV-isolation, AIDS and oxidative stress models and the AIDS-situation in Africa -- in scientific literature since 1983. Much of their outstanding work has been published in CONTINUUM magazine in England.

Prof. Alfred Hässig, is professor Emeritus of immunology and former member of the National Commission on AIDS in Switzerland. He is the founder of the Swiss-based Study Group for Nutrition and Immunity. He has published extensive work on explaining „AIDS" in the absence of „HIV". Prof. Hässig is a member of the Board Of Scientist of IFAS.

Christine Johnson is a freelance journalist with a professional background in science. She is living in Los Angeles and has closely followed the issues of HIV-test-specificity/cross-reactivity and HIV-isolation. Her eye-opening interview with Mrs. Papadopulos-Eleopulos bringing light into the non-isolation of HIV has been published in the English magazine CONTINUUM. Ms. Johnson is a member of the Public Access Board of IFAS.

Dr. Vladimir Koliadin is a senior research scientist at the Kharkov Aviation Institute. His field of work include statistics, mathematical modelling and mathematical foundations of data based inference. He has closely followed the definition changes and statistics of „AIDS" from early on. An extensive article on the issue by Dr. Koliadin has been published in the special issue of GENETICA. Dr. Koliadin is a member of the Board Of Scientist of IFAS.

Dr. Michelle Cochrane has a PhD in Medical Geography of the University of California at Berkeley. Her research includes environmental science. Writings on the subject of „AIDS" include in depth analysis of the dissenting voices on „AIDS" and „AIDS" in Africa. She has rewritten one chapter of her extensive dissertation „The Social Construction Of Knowledge(s) On HIV and AIDS" for this dossier.

Dr. Charles Geshekter, has an M.A. in African History and teaches such since 1968 at California State University in Chico, USA. He has challenged the AIDS situation in Africa as an insider for a number of years and is a well-placed expert in the „AIDS" situation in Africa, with frequent articles published on this topic. Dr. Geshekter is a member of the Group For The Reappraisal of the HIV/AIDS-Hypothesis.

Rosalind Harrison, is a practising eye-surgeon in Burton-on-Trent, England. She has a diploma in Tropical medicine and hygiene. After an extensive investigation she wrote together with her former husband, Richard Chirimuuta, the book „AIDS, Africa And Racism". (see literature list)

Simon Barker is a freelance writer from Seattle, USA. He has closely monitored the AIDS debate for a number of years. His particular interest -- studying Naturopathic Medicine -- is AIDS in children. He is working with the Seattle chapter of HEAL, an US organisation offering alternative information on HIV/AIDS.

Felix de Fries, is a long-term critical „AIDS" activist. As a critic of the common belief that „HIV" causes „AIDS" he founded the Swiss-based Study Group for AIDS-Therapy back in 1986, focusing on natural immune enhancing therapeutical interventions for people with an „HIV diagnosis".

Michael Urs Baumgartner, has been working in the field of „AIDS" as a counsellor and critical activist for the last 13 years. He is a licensed clinical social worker and worked as an „AIDS" Chaplain at San Francisco General Hospital for one year. He regularly writes on subjects relevant to the issues for CONTINUUM magazine in England. Michael Baumgartner is founder and Secretary General of IFAS.

HISTORY AND CONSEQUENCES OF THE "HIV/AIDS" HYPOTHESIS

(This paper outlines only some of the critical data on „HIV-AIDS", produced in the last 16 years, viewed by the author as crucial. It is therefore not exclusive.)

by Michael U. Baumgartner, Bern, Switzerland

1981 Increased clinical frequency of the skin neoplasm ***Kaposi's Sarcoma (KS)*** and the *pneumonia* ***Pneumocystis Carinii (PCP)*** in so-called "otherwise healthy" male homosexuals in the USA reported to the *Centers for Disease Control (CDC)*, a U.S. government health authority monitoring the spread of diseases, by Dr. Michael Gottlieb, of the University of Los Angeles (UCLA) *(MMWR No. 21/Vol. 30 June 5th, 1981).*

1982 The CDC announces this new phenomenon as ***Acquired Immune Deficiency Syndrome ("AIDS")*** *(MMWR No. 37/Vol. 31, September 24th, 1982)* despite the fact that ***KS*** is **not** due to an immune deficiency (low T-cell count).

1983 **(1)** ***Dr. Luc Montagnier*** (microbiologist) et al. at the Pasteur Institute, Paris isolate, from the lymphnode of a male homosexual **without** AIDS, "retrovirus particles" he terms ***Lymphodenopathy Associated Virus (LAV)*** which later will be called ***"Human Immuno- deficiency Virus" ("HIV")*** *(Science, May 20th, Vol.220, 1983).*

(2) ***Dr. Robert Gallo***, cancer researcher at the National Cancer Institute (NCI) in Bethesda, Maryland, USA, **revises** a paper written by **Dr. Montagnier** and **adds** in the introduction the suggestion that **"LAV"** is associated with the so-called ***Human T-cell Leukemia Virus*** family (***HTLVs***) *(Science, Vol. 220, May 20th, 1983)* - a type of virus he claimed credit for in the beginning of the 80s despite its having been first "identified" in Japan in the mid 70s *(John Crewdson, Chicago Tribune, November 19, 1989).* Gallo proposed "***HTLV I***" caused T-cell Leukemia, a rare form of cancer in adults, by infecting the T-cells (a type of immune cell which fights disease in the body), causing them to multiply uncontrollably. In AIDS-patients "***HTLV III***" is **claimed to do quite the opposite**, to destroy these very T-cells. **Both hypotheses have since been proven wrong.** Morphologically "HTLV I" and "II" are so different from "HTLV III" that they can hardly be considered the same family to begin with. The French therefore make a clear distinction between their virus "LAV" and the "HTLVs" *(Steve Connor, New Scientist, February 12th, 1987).*

(3) Dr. Gallo receives ***"LAV" sample*** from Dr. Montagnier with **patent right claim** for future **test kits** based on "LAV". Dr. Gallo states his lab is **unable** to grow "LAV". *(Steve Connor, New Scientist, 12th February 1987)*

1984 **(1)** Dr. Robert Gallo et al. continue to claim **"HTLV III"** causes AIDS by killing T-cells. At the same time **he takes patent** on the mass-production of "HTLV III" in **immortal** T-cell lines. (The same kind of cells that he claims simultaneously, are **killed** by "HTLV III/HIV".) *(Science, Vol. 224, May 4th, 1984).*

(2) Dr. Gallo publishes **pictures of his** HTLV III which **turn out to be** pictures of the French LAV which later will be called "HIV".*(Science, Vol. 224, May 4th, 1984/ Steve Connor, New Scientist, February 12th, 1987).*

(3) ***HTLV III infection*** is declared the "**probable cause of AIDS**" by Dr. Gallo at a press conference called by Dr. Margaret Heckler, Secretary of Health and Human Services under the Reagan administration, despite there **not** being any scientific paper published to back up such a claim, nor any scientific debate prior to the statement. The fact that he was only able to detect „HTLV III" in 26 out of 72 patients (**36% correlation**) and antibodies to „HTLV III" in 43 out of 49 patients (**88% correlation**) - using mainly the most unreliable ELISA test - has not been mentioned. *(L.K. Altman, New York Times, 24th April 1984).*

(4) The ***National Institute of Health (NIH)*** on behalf of the US government **takes worldwide patent** for the "HTLV III antibody test", known as the "AIDS test", with Dr. Gallo getting shares. *(Steve Connor, New Scientist, 12th February 1987)*

1985 DNA analysis **proves** Montagnier's **"LAV"** and Gallo's **"HTLV III"** to be **identical.** One of Dr. Gallo's samples has been **contaminated** with "LAV" accidentally of deliberately, as also happened in the case of the "Human Leukemia Virus 23" ("HLV23"), which Dr. Gallo also claimed credit for in 1975, but which turned out to be a cocktail of three monkey viruses *(Steve Connor, New Scientist 12th February 1987).*

1986 (1) The International Committee for the Taxonomy of Viruses names all the disputed virus ***Human Immunodeficiency Virus*** (**"HIV"**). *(Coffin et al. Science, Vol. 232, May 9th, 1986)*

(2) The ***French Government*** brings a **patent infringement** case against the ***U.S. Government,*** claiming that Dr. Montagnier is the discoverer of "HIV" and **not** Dr. Gallo. Dr. Gallo testifies he did **not** believe the French "LAV" was the cause of "AIDS" **before** he applied for **his own "HTLV III" antibodytestkit patent.**

(3) The American ***Phase II Study*** starts, to prove the therapeutic **antiviral** effect of the **toxic** early-60s **anti-cancer** drug ***AZT*** in people with "AIDS", and is prematurely terminated because it's claimed to "prolong life" and "improve quality of life" and must thus be available. This study is considered fraudulent by several scientists. *(John Lauritsen, The AZT Story: Poison by Prescription; Asklepios New York 1990).*

1987 ***Dr. Peter H. Duesberg***, molecular biologist at the University of California in Berkeley, **questions** the destruction of T-cells by "HIV" in humans (in vivo) and states **"no direct killing"** of CD4-cells (T-cells). *(Cancer Research, Vol. 47, 1987)*

1988 (1) Dr. Gallo **retracts** his hypothesis about direct killing of T4 cells by "HIV" but continues to support his **unproven theory** that "HIV" **causes** "AIDS". He now suggests "indirect mechanisms" triggered by "HIV" as being responsible for the decline of CD4 T-cells. *(Journal of Acquired Immune Deficiency Syndromes, No. 6/Vol. 1, 1988)*

(2) The ***Concorde Trial,*** a large Anglo-French study, starts in co-operation with the manufacturer of AZT, ***Wellcome***, to test the **prophylactic effect** of AZT as an **"antiviral"** on HIV-antibody positive individuals who have **no** AIDS-symptoms. Up till now AZT was given to people **at risk** from "AIDS" in the Western world.

1989 The US-recruit study terminates. From October 15, 1985 to March 31, 1989 1 141 164 US-recruits under the age of 20 were monitored for HIV-antibodies. Only 393 tested positive over a period of 3 1/2 years, that is **less than 0.035 %** *(JAMA, No. 15/Vol. 263, April 18, 1990).*

1990 Dr. Montagnier presents his findings that "HIV" itself does **not** kill T-cells at a press conference in San Francisco.

By 1991, ten years after the new syndrome was identified , the "HIV/AIDS" connection is ever more tenuous and questionable. Since there was never an isolation of what is called "HIV", there is no proof of a new retrovirus. All there seems to be are antibodies that, perversely and uniquely for medical history, are now an indicator of an early death due to about 29 old diseases under a new umbrella term, not to mention on toxic treatment with an old drug and an increasing number of new ones.

The hypothesis of direct cell destruction by "HIV" has been disproved, yet other hypotheses are and will be created with little reasonable foundation. If „HIV" were a retrovirus, it would like, all considered retroviruses, do no harm to its host, the human cell.

Despite the establishment's blindness, the number of "HIV/AIDS" critics is growing fast. Towards the end of the eighties there was an increasing number of agencies and publications in the USA, England, Germany, Switzerland, etc., addressing facts on "AIDS" rather than promoting the false and deadly "HIV=AIDS=death" hypothesis.

1992 **(1)** ***Dr. Kary Mullis***, an American biochemist and inventor of the ***polymerase chain reaction (PCR)***, to date the most precise way of testing for viral DNA, states "PCR has made it easier to see HIV. But human beings are full of retroviruses, and neither HIV nor any other retrovirus by itself poses any kind of threat, which is not to say that there is no such thing as AIDS - only that HIV does not cause it" *(Newsweek, August 1992).*

(2) ***The Committee of Research Integrity,*** a scientific body of the National Institute of Health, declares Dr. Gallo **guilty** of scientific misconduct on different occasions relating to the establishing of the "AIDS-virus" *(John Crewdson, Chicago Tribune, November 19, 1989).*

(3) Senior scientists and physicians from around the world are forming the publishing group ***Reappraising AIDS***, which proposes professional **re-evaluation** of the "HIV=AIDS=Death" hypothesis.

1993 **(1)** Evidence shows that the **majority** of people in the Western world diagnosed as HIV-antibody positive are **not** getting AIDS fifteen years after the assumed introduction of this retrovirus. US numbers by mid-1994 reveal more than one million "HIV-positive" individuals with around 350 000 "AIDS cases"; European figures reveal 0.5 million "HIV-positive" with between 50 000 and 100 000 "AIDS cases" including deaths. *(WER, WHO report No. 27, 1993)*

(2) The first intervention, *"An urgent appeal for action: On the effect of the unproven "HIV=AIDS" hypothesis and its effect on human lives"*, is made by ***People's International Health Project*** in conjunction with ***International Educational Development*** at the ***United Nations*** in Geneva, Switzerland *(Press Release HR 3358, Children's Issues Agenda Item 24 and HR 3360, Science and Technology, Agenda Item 14).*

(3) The termination of the Concorde Trial of AZT shows **no observation** of the hypothesised prophylactic effect, but a **higher mortality** in the AZT group than the placebo group. Still, the manufacturer Wellcome publicly reaffirms the drug's efficacy, citing a previously discredited Australian study and several Scandinavian studies which were too small to be significant *(Lancet, Vol. 341, April 3rd, 1993)*

(4) Dr. Eleni Papadopulos-Eleopulos, medical physicist from the Royal Perth Hospital, Australia, *et al.* demonstrate the **inaccuracy of all HIV-tests** - ELISA, Western Blot antibody tests and PCR viral detection. Their research shows that other conditions unrelated to "HIV", such as TB, malaria, leprosy, vaccination against Hepatitis B, even the common flu, or a flu vaccination can trigger a positive "HIV antibody" test result. *(Biotechnology Vol. 11, June 1993)*

(5) There are 4'621 **documented** AIDS cases clinically diagnosed - meaning from a risk group and with an AIDS-disease - **without** any indication of "HIV", at least 1'500 of them in the USA *(Biotechnology Vol. 11, August 1992).*

(6) The CDC again **adds** more elements to the "AIDS definition". Now 200 T-cells per microliter of blood or less, cervical cancer, recurring bacterial pneumonia, or any of the 25 other previously old disease in the presence of "HIV-antibodies" are called "AIDS" *(MMWR No. RR17/Vol. 41, 1993).*

(7) Dr. Kary Mullis is **awarded the Nobel Prize** for chemistry for the PCR and **continues to refute** the "HIV" causation theory of AIDS.

(8) Dr. Gallo is cleared of the accusation of scientific misconduct by a panel of lawyers **via alteration of the definition** of scientific misconduct.

(9) American Journalist ***Neenyah Ostrom*** of the only „AIDS" critical gay newspaper world- wide, ***The New York Native,*** publishes her book, suggesting the link between *Chronic Fatigue Syndrome* (CFS) and *Acquired Immune Deficiency Syndrome* (AIDS) *(America's Biggest Cover-Up; CFS and AIDS, TNM Inc., New York, 1993).*

1994 **(1)** ***Mrs Sue Threakell*** accesses ***Legal Aid*** in England to **prosecute** the pharmaceutical giant Wellcome over the death of her hemophiliac husband, which, she accuses, was caused by Wellcome's best selling "anti-AIDS drug" AZT (Retrovir) and not by his "HIV-condition". The case is pending.

(2) Fuller official data from the Concorde Trial is finally released **a year after the study's termination** and confirms the initial findings that it's **without prophylactic value** and **increases mortality** *(Concorde Coordinating Committee, Lancet, vol 343, April 9th, 1994).* Wellcome employees who were on the Coordinating Committee are told **not** to endorse the report.

(3) ***Dr. Max Essex,*** of the Harvard University School of Public Health, *et al.,* a leading originator of the "HIV=AIDS" theory publishes **evidence** that the **"HIV" antibody testing is non specific** with particular reference to the purported epidemic in Africa, where the numbers have been greatly over-estimated and revised by the World Health Organization (WHO) many times *(Journal of infectious Diseases Vol. 169, 1994).* This supports the published yet widely ignored work by Dr. Papadopulos-Eleopulos et al..

(4) Dr. Gallo **admits** "...**we have never found HIV-DNA in T-cells...**" at a meeting sponsored by the National Institute on Drug Abuse, May 23/24, in Washington DC *(Lauritsen, New York Native June 13, 1994).*

(5) Finally Dr. Monagnier gets **sole credit** for the discovery of "HIV" *(Newsweek July 1994).*

(6) A study carried out at the Royal Free Hospital London claims that 25 % of "HIV infected" hemophiliacs will **remain** AIDS free over a period of 20 years and 15 % will remain "AIDS free" over a period of 25 years *(British Medical Journal Vol.309, 1994).*

(7) The Inspector General's office of the US Department of Health and Human Services reports "...there was **no evidence** to support Dr. Gallo's claim of having independently discovered the virus or created the AIDS test...". "The claim that HTLV-3B (Gallo's HIV infected T-cell line) was contaminated by LAV comes into question since there appears to be **no evidence there ever was** a 3B to be contaminated" *(Ostrom, New York Native, Vol. 585, July 94).*

(8) ***Dr. Stefan Lanka,*** molecular biologist at the University of Konstanz, Germany, states that the existence of what has been called "HIV" has **not** been proven. **There has not been an isolated entity which may be called "HIV"**, only cellular proteins, among them an enzyme named reverse transcriptase (RT). It had been claimed RT was specific to so-called retroviruses , but as early as 1983/4 the enzyme could be detected in **all** living cells. Lanka claims what has been shown at the genetic level, instead of "HIV", is human endogenous (i.e. from within the cell) genetic material out of the pool of the as yet 90 % un-coded so-called repetitive elements of the chromosomes present in everybody. This stresses the **worthlessness** of the "AIDS-tests" and indicates these tests may only point out contact by one individual with human proteins from others, most likely from white blood cells, because the "HIV antibody test" is only made out of proteins from white leukemic blood cells produced in the lab: if someone has immunological contact with foreign human proteins and then produces antibodies, these are read as "HIV-antibodies" *(Fehldiagnose AIDS, Wechselwirkung, Dec. 94).*

At the tenth International AIDS Conference in Yokohama, Japan scientists now **claim** AIDS to be an auto-immune disease - the immune system turning against its own organism - yet most commonly known auto-immune disease are due to toxins, not viruses.

Meanwhile the prescription of AZT and other so-called "antiviral therapies" equally dangerous, continues to around 200 000 both asymptomatic "HIV-antibody" positive individuals and people with "AIDS" in the Western world, despite its **1000 times higher toxicity than initially claimed** - *a* **deadly treatment** that actually **mimics** the disease *(Project A.I.D.S. International, Public Information Dossier, March 15th, 1993).* Soon AZT under another brand name will be available in the so-called "third world".

Today it is the trend to give "cocktails" of so-called "anti-HIV-drugs". The treatment gets as indistinct as the diagnosis and deadly. **Healthy** individuals get treated with **highly toxic** substances based on a T-cell count of 200 or less despite the fact that these counts are **not** reliable markers for disease or early death *(Aboukler J.P. et al. Lancet April 1993/Farber Celia, SPIN-magazine, spring 1994).* The so called "side-effects" of those drugs (DNA chain terminators), some 50 in number, include the wasting, nausea, immune suppression, anaemia, disability and death common to medically perceived "AIDS".

After **22 billion** US dollars and ten years of "HIV" research and the claimed "world AIDS epidemic", there are **still people dying** of an erratic hypothesis summarized as "Acquired Immuno deficiency Syndrome. Despite the fact that 40 % of the "AIDS diseases" are **not** due to Immune deficiency, including KS, one of the two initial AIDS-defined diseases *(Lang Serge, HIV/AIDS; have we been misled?, Yale Scientist, Fall 1994).*

How much do we really know about "AIDS"? Studies and many experienced AIDS professionals state **intoxication** through combinations of factors such as intravenous and/or oral and/or nasal recreational drugs like heroin and/or cocaine and/or ecstasy and/or crack and/or poppers and/or MDA etc. often in combination with alcohol, and/or medical drugs like AZT and/or ddI and/or D4T etc. and/or prophylactic and/or excessive use of Septrin and/or antibiotics, etc. and/or extended mal nourishment and/or poor hygiene and/or misdiagnosis along with coercion and statistical zeal cause what we call "AIDS".

1995 **(1)** ***Dr. David Ho et al.*** from the Aaron Diamond AIDS research centre in New York City claim that „HIV" is producing billions of viral copies every other day and hereby exhausting the immune system by killing billions of CD4 T-cells *(Ho. et al., Science 373, p. 102).*

(2) Dr. Papadopulos et al. publishes evidence that there is **little or no** likelihood of HIV being in *Factor VIII* (clotting factor prescribed to haemophiliacs) and in the unlikely event of few particles surviving the Factor VIII manufacturing process they **could not** possibly be viable infectious particles. *(Genetica, 1995: 25 - 50)*

(3) Prof. Duesberg publishes evidence that the immune suppression found in haemophiliacs is **directly increased** by the contamination proteins found in clotting factors, which constitute 99 % of the product. **The immunosuppression is directly age and dosage related** *(Genetica 1995: 51 - 70).*

(4) Despite the serious scientific doubts and previous failures the CDC decides to introduce US-produced vaccination trials into the „3rd World" *(Jones Coleman, SPIN magazine, Jan. 95, p. 67 - 69).*

(5) In a revealing article ***Mark Craddock Ph.D.*** of the University of Sidney, Australia, lifts the magic of Ho's work and draws attention to the bad science on several accounts - such as mathematical errors, inaccurate measurement, no controls etc. *(Reappraising AIDS, Vol 3/no 5; May 1995).*

(6) After the US, Holland, Switzerland, England, Spain and Italy the next AIDS Dissident conference is held in Argentina.

(7) The latest treatment with the new „anti-viral" drugs - pharmaceutically produced protease inhibitors is launched as the latest hype even for clinically non symptomatic „HIV positive" individuals. No evidence of any clinical benefits, yet very costly, much like the once hailed drug AZT.

1996 **(1)** ***Prof. Alfred Hässig*** demonstrates a model of stress-related immune deficiency, explaining the CD4 decline **without** the presence of „HIV". This model has been t the focus of HIV-scientist Anthony Fauci in the 70s and dropped when „HIV" was established, despite its promising findings *(Hässig et al., CONTINUUM Vol. 3/No.5; jan/feb. 96).*

(2) In a petition process in Germany the German Minister of Health had to confirm - after initial denial - that they **never found** any trace of „HIV" in the blood serum of persons diagnosed HIV positive *(pet. 5-13-15-212-023; 08.03.96).*

(3) In an extensive study carried out at Royal Perth Hospital in Australia a team of scientists have demonstrated that the „Human Immune Virus, HIV" - the claimed cause for AIDS - has not only **never been isolated**, but just which of the different actins present in so-called „retroviral isolations" is actually supposed to indicate the „HIV-infection", has never been agreed on by Montagnier and Gallo *(Papadopulos-Eleopulos et al., CONTINUUM Magazine, Vol. 4/no. 3, sept/oct. 96).*

(4) ***Heinrich Kremer MD*** exposes at a conference held in Barcelona, Spain how a number of **medically prescribed** drugs including nucleoside analogues (f.e. AZT) and modern antibiotics (Septrim, Bactrim a.o.) cause cell death by damaging the mitochondrial DNA and hereby **mimic** the symptoms of the Acquired Immune Deficiency Syndrome *(Juan Luis Lopez/Heinrich Kremer; CONTINUUM; Vol. 4/No. 4; nov/dec. 96).*

(5) A team of Dutch researchers at the University of Amsterdam **disprove** the findings of Ho et al. They find **no evidence** of a rapid turnover in CD4 T-cells due to „HIV-infection" *Wolthers K.C. et al.; Science; Vol. 274; 1543 - 1547; 29. November 1996).*

1997 **(1)** Warnings from the FDA, other health officials and studies are surfacing, showing the dangerous effects and life-threatening complications in people who get treated with the pharmaceutically manufactured protease inhibitors *(Brău N. et al.; The Lancet, Vol. 349, 29th march 97; a.o.)*

(2) The *Urgent Appeal For Action* at the UN in Geneva continues, drawing attention to the flaws of the „HIV-AIDS" dogma and demanding an international halt to the established approaches to „HIV" and „AIDS".

1998 Papers published in *Immunology Today* and *Nature Medicine* call into question the distraction of CD4 cells by „HIV". *(Rosenberg et al.; Immunology Today, Vol. 19/No 1; 10-16; 1998; Gorochov et al.; Nature Medicine; Vol. 4/No. 2; 215-221, 1998)* „The last nail has been hammered into the coffin of this simple theory" so the German newspaper *Die Zeit* quotes. *(Schuh Hans, Die Zeit; 35; 26.02.98)*

The whole scenario closely mimics the 1970's Japanese SMON health scandal where the anti-diarrhoea drug *Clioquinol* (brand names *Entero-Vioform and Mexaform)* manufactured by Ciba Geigy and not the hypothesised *"Inoui Virus"* caused thousands of human deaths *(Channel 4 publication, Drug injury and what to do about it, The story of SMON by Joan Shenton).*

The consequences of ignoring these aspects of 16 years of medical history are massive with catastrophic dimensions for individual human life, human rights and the reputation of medical science and practice.

HIV PROTEASE INHIBITORS, MUTATIONS AND VIRAL LOAD

by David Rasnick, California, USA

Introduction

One of DNA's main functions is to provide the instructions for making proteins. Proteins are so versatile that they are the primary structural elements of all living things including viruses. With the exception of a few peculiar RNAs, all enzymes are protein. Enzymes are the biological molecules that get things done by acting on other molecules called substrates.

One of the largest classes of enzymes are the proteases. On the surface, proteases perform one of the simplest of biological reactions: they clip proteins into smaller proteins or peptides. Peptides are just short pieces of proteins. The point at which a peptide is elevated to the exalted status of a protein is arbitrary. Proteases that cleave primarily proteins are called proteinases, whereas proteases that cleave primarily peptides are called peptidases. For our purposes HIV protease can properly be called a proteinase since its primary substrate is the gag-pol polyprotein coded for by the HIV proviral DNA.

The 20 or so amino acids are linked together through amide bonds to make all the proteins and peptides that exist. The amide bonds of proteins have been given the special name of peptide bonds to signify that they belong to proteins and peptides. Peptide bonds are very strong chemical bonds and are difficult to break. Nonetheless, under the right conditions proteases can easily break peptide bonds. An everyday example is the very active cysteine protease called chymopapain that comes from the papaya plant and is the active ingredient in meat tenderizer. Chymopapain breaks the peptide bonds of meat before it is cooked. The aspartyl protease pepsin in the stomach and the serine proteases chymotrypsin and trypsin in the small intestine digest the proteins eaten. It turns out that proteases are far more complex and interesting than this simple picture implies. The vast majority of proteases are involved in the processing and regulation of other proteins including other enzymes.

Proteases are globular proteins with an indentation or cavity called the active site. Substrates fit into the active site of the protease where the enzyme catalytically breaks the specific peptide bonds to be cleaved. A large number of proteins act as specific inhibitors of proteases, regulating their activities. There are also many tens of thousands of small synthetic inhibitors designed in laboratories around the world to stick tightly to the active sites of many different proteases. The synthetic inhibitors are used most often as research tools and occasionally as potential therapeutic agents.

Proteases have been divided into four main classes according to the active site features that are common to each group: serine, cysteine, aspartyl, and metallo. These designations have nothing to do with the substrates the proteases cleave. **HIV protease belongs to the aspartyl family of proteases**. Examples of human aspartyl proteases are pepsin (a digestive enzyme in the stomach), cathepsin D (located within lysosomes inside cells), and renin (one of the proteases that regulates blood pressure).

HIV protease

Everything that is known about HIV has been constructed from *in vitro* experiments. It is important to recognize that all of the published statements concerning HIV's presence and activity are extrapolations from the *in vitro* studies and have not been directly observed in humans.

HIV, like all retroviruses, contains genes that code for structural proteins, envelop proteins and enzymes. During translation the individual proteins are all stuck together end-to-end via peptide bonds to form the Gag-Pol polyprotein. **It appears that the envelope proteins are severed from the polyprotein by a host protease and not by HIV protease**. The viral structural proteins (coded for by the gag region of the gene) and the viral enzymes (including HIV protease, coded for by the pol region of the gene) are connected by peptide bonds that must be processed (cleaved) by HIV protease.

HIV protease doesn't clip every peptide bond in sight; it is very particular about the sites of cleavage. Of the hundreds of possibilities, there are only eight specific peptide bonds of the gag-pol polyprotein that HIV protease must cleave for the virus to replicate and mature properly into infectious particles (1).

The production of infectious HIV particles is dependent on proper assembly of structural proteins into the core particle. The initial steps in assembly involve the association of the gag and gag-pol precursor proteins with the inner face of the membrane of the infected cell, followed by interaction of the precursors with each other. The membrane-based association of the precursor proteins precedes cleavage of the precursors by HIV protease. HIV protease is part of the larger gag-pol polyprotein and is only functional as a dimer. This arrangement allows the precursor proteins to arrive at the membrane in a coordinated manner and is largely successful in preventing premature activation of HIV protease. Once bound to the membrane, HIV protease cleaves the individual precursor proteins in an ordered, sequential manner (2-6) . If all goes well, budding and release of the mature, infectious virus particle occurs, leading to a new cycle of infection and viral replication.

Disrupting the proteolytic processing of HIV precursor proteins is an excellent strategy for blocking the production of infectious virus. Incomplete processing of the precursors by HIV protease still leads to budding but the viral particle produced is not infectious. **That is why in the presence of inhibitor infected cells are still capable of producing viral particles, but the virus produced is defective and not infectious** (2,7). The central question is will inhibiting HIV be of therapeutic benefit?

Inhibitor-resistant mutants: a mirage

Numerous *in vitro* experiments have demonstrated that impaired proteolytic activity, due either to the presence of protease inhibitors (2,7-9) or deleterious mutations of HIV protease (10-15) , results in noninfectious HIV particles. As a consequence of these studies and several human clinical trials (16-19) , a number of HIV protease inhibitors have recently been approved for clinical use. **The disappointing clinical efficacy of these inhibitors during the early trials led to the widespread belief that the HIV protease develops resistance to the inhibitors by mutating to less susceptible forms of the enzyme** (17-25).

To date, however, all of the inhibitor-resistant mutant proteases "identified" in clinical samples were obtained from traces of inactive HIV proviral DNA in a few cells of some people. These inhibitor-resistant mutant proteases have never been seen in viable, infectious virus. So far, only special laboratory conditions are capable of producing viral particles containing inhibitor-resistant mutant HIV protease. Even here, the viability and infectivity of these mutant particles relative to the so-called wild-type virus have not been reported (19,20,26,27).

The inability to find inhibitor-resistant HIV protease in infectious virus is readily explained. The minimum viable catalytic efficiency of HIV protease was found to be 2% of the wild-type

activity (11,12). The catalytic efficiencies of the mutant enzymes, however, are many orders of magnitude below the 2% level (28,29). The reason for the extremely low levels of activity is that both the inhibitors and the substrates bind to the same catalytic site of HIV protease. Since the wild-type HIV protease has evolved to the optimal level of activity, virtually all alterations to the enzyme's structure that affect catalytic efficiency are lethal to the virus. Mutations of the protease that reduce inhibitor binding result in an even more profound reduction in catalytic activity. This is because the overall catalytic efficiency of a mutant HIV protease is given by the product of the relative efficiencies of the mutant enzyme with respect to the wild-type for all eight obligatory cleavages (28). These eight cleavages can be thought of as an eight-number combination lock. Not only does HIV protease have to make all eight cleavages, but the enzyme must do it in the right order. **Therefore, even in the absence of inhibitors, the inhibitor-resistant mutant HIV proteases do not lead to viable, infectious virus**. That's not the end of the bad news for mutant HIV. As with the wild-type enzyme, the eight sequential cleavages force the inhibitor-resistant proteases to be exponentially sensitive to inhibitors, which more than compensates for their weaker binding (28).

Therapeutic doses too high

The extremely high doses of protease inhibitors that are being prescribed to patients in the hope of preventing the appearance of inhibitor-resistant mutants are unwarranted. The daily dose of around 1-2 grams of HIV protease inhibitors is well over an order of magnitude above what is needed to render HIV noninfectious and, therefore, nonpathogenic if it were true that HIV is pathogenic.

The toxicities of the protease inhibitors (so severe that 35-50% of patients cannot tolerate them) are well documented (30). The high doses of the inhibitors pose long-term risks to patients as well. The oral doses of protease inhibitors currently administered to patients are at minimum 50 times that needed to completely inhibit the intestinal aspartyl protease cathepsin D (calculation based on the Roche inhibitor Saquinavir; the Abbott inhibitor Ritonivar is 1000 times more potent against cathepsin D than Saquinavir). **The inhibition of cathepsin D in the intestines of patients my have clinical consequences since mice deficient in this enzyme (generated by gene targeting) develop normally during the first two weeks, stop thriving in the third week and die in a state of anorexia at day** 26 ± 1 (31). The fact that diarrhea is a common problem with all the protease inhibitors may be a warning of problems ahead. In light of the immune compromised state of AIDS patients, it is also important to note that the cathepsin D-minus mice suffered massive destruction of the thymus and spleen with fulminant loss of T and B cells. The maintenance of high blood levels of protease inhibitors makes their potential effects on the lymphocytes more than an academic concern.

Since all the protease inhibitors were approved under the FDA's accelerated approval process, the long-term safety of these agents is unknown (30). This is especially disturbing in view of the disclaimer attached to each of the HIV protease inhibitors approved by the FDA. The Merck entry is typical:

"Crixivan is not a cure for HIV or AIDS. People taking Crixivan may still develop infections or other conditions associated with HIV. Because of this, it is very important for you to remain under the care of a doctor. It is not yet known whether taking Crixivan will extend your life or reduce your chances of getting other illnesses associated with HIV. Information about how well the drug works is available from clinical studies up to 24 weeks."

Such a notice does little to inspire confidence either to prescribe or take the protease inhibitors, especially when faced with the known and potential toxicities of these compounds. **Finally, the specificity of the HIV protease inhibitors is not absolute and it is impossible to determine the toxic effects of new inhibitors just by looking at them.** For example, the Abbott HIV protease inhibitor unexpectedly inhibits a subset of the cytochrome P450 enzymes the liver uses to detoxify drugs, while high levels of the Merck compound cause kidney stones (30,32) and severe hepatitis (33). The FDA recently discovered 83 patients who contracted diabetes or hyperglycemia, high blood sugar, or had those diseases suddenly worsen after they began taking protease inhibitors (the Associated Press, 11 June 1997).

Infectious virus is the thing

There is also a problem with the surrogate marker used to evaluate the clinical efficacy of the protease inhibitors. The so-called viral-load test is claimed to measure pieces of HIV viral RNA (termed copy number), but the assay gives no indication as to the viability or infectivity of the viral particles presumed to be present in a patient's blood plasma. Even before antiretroviral therapy, 99.9% of the virus detected by the viral-load test was found to be noninfectious in the sole individual examined (34). (The 99.9% figure agrees with the known level of defective variants of the HIV genome (35,36). And after two days of protease inhibitor cocktail therapy none of the over 500,000 viral particles per ml in that patient's blood plasma was infectious (34) . Typically, in inhibitor naive patients, as well as in cell cultures, **only about 1 in 100,000 viral particles is infectious** (35,37-40). **The viral-load test, then, is measuring overwhelmingly noninfectious virus.** The significance of all this noninfectious virus has not been adequately addressed.

To further complicate matters, protease inhibitor therapy leads to defective, noninfectious viral particles (2,7-9) that are more stable than wild-type virus, thus adding to the already erroneously high measure of "viral-load." Confounding the issue even further, in 1993, Ho et al. reported 12 AIDS patients, including 8 who had AIDS "risk factors," who were totally HIV-free: "Specific antibody assays, viral cultures, and polymerase chain reaction (PCR) techniques" for HIV were all negative (41). **In short, no one has shown that the viral-load assay has anything to do with infectious virus**. In fact, the most that can be claimed for the viral-load test is that at best it is detecting viral debris.

Conclusions

The degree to which the mutant proteases are resistant to inhibitors is meaningful only in the context where the viability and infectivity of the mutant viruses are also quantitated. The general misunderstanding regarding the significance of the inhibitor-resistant mutants can be attributed in large part to the common practice of testing the mutant proteases against a single substrate without taking into consideration the eight sequential cleavages necessary for viable maturation of HIV (42). It is extremely unlikely that mutations of the enzyme, substantial enough to protect the protease against inhibition, will at the same time leave virtually unimpaired its proteolytic activity towards all eight cleavages. None of the inhibitor-resistant mutant HIV proteases reported so far (even in the absence of inhibitors) has come anywhere near the minimum level of overall catalytic activity necessary for infectious, viable virus. The conclusion of this analysis is that inhibitor-resistant mutant HIV proteases are very unlikely to contribute to viral viability *in vivo*. Therefore, traumatizing patients with the specter of drug-resistant mutants of HIV resulting from a failure to adhere to a Draconian regimen of medication is unjustified. It is important to emphasize that the protease inhibitors are toxic compounds with a growing list of serious and life-threatening consequences.

Finally, the deceptively named "viral-load" test does not measure infectious virus and should not be used to indicate the presence of HIV in the blood plasma of patients.

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THE AZT PHARMACOGENOCIDE

by John Lauritsen, Massachusetts, USA

Following is a brief report on a large-scale iatrogenic disaster: the prescription of AZT and other nucleoside analogues as treatment for the dubiously defined illness known as the "Acquired Immuno-Deficiency Syndrome" (AIDS).

The main points are: AZT is a highly toxic and worthless drug, which was approved by the U.S. Food and Drug Administration (FDA) on the basis of fraudulent research. Nucleoside analogue "antiretroviral" drugs have already caused the deaths of tens of thousands of people, many of whom were not even sick before taking these drugs.

Source references within the body of this report will be kept to a minimum. However, extensive references are contained in the Duesberg and Lauritsen books listed under "References".

What is AZT?

AZT is an abbreviation for azidothymidine, the proper chemical name of the drug. It is also known by its generic name, zidovudine, and by its brand name, Retrovir. Although AZT is described by its promoters as a "reverse transcriptase inhibitor", this description is misleading. The biochemical mechanism of AZT is extremely simple; it is a random terminator of DNA synthesis, the basic life process. This it does by substituting itself for thymidine, one of the basic building blocks of DNA (Duesberg 1996c, Meditel 1992). It was once claimed, based on research conducted by Burroughs Wellcome, the manufacturer of the drug, that AZT acted selectively against viral DNA synthesis as opposed to human cellular DNA synthesis. This claim has since been proven false by at least a half dozen independent studies, which found that AZT is up to 1000 times more toxic to human cells than was claimed when the drug was approved for marketing in 1987 (Duesberg 1996a) . Since reverse transcriptase requires DNA synthesis, it could be argued theoretically that terminating DNA synthesis using AZT would inhibit the process called reverse transcriptase by which the suspected retrovirus builds its DNA into that of the cell. But that would be like dropping a napalm bomb in someone's back yard, observing that insects had been killed, and then marketing napalm bombs as insecticides. AZT cannot distinguish between human cellular DNA and retroviral DNA, and is thus by its very nature cytotoxic -- a killer of human cells.

Developed as cancer chemotherapy in the 60s by Dr. Jerome Horwitz, head of a laboratory at the Detroit Cancer Foundation, AZT was designed to kill all growing cells through the termination of DNA synthesis. The rationale was that a DNA terminator might be an effective anti-cancer drug, since cells grow through the process of DNA synthesis and since cancer cells grow abnormally fast. However, AZT proved to be not only extremely toxic but totally ineffective in prolonging the lives of leukemic animals (Duesberg 1996c, Lauritsen 1990). Normally chemotherapy is given for only a few days or weeks. The hope is that the drugs will kill the cancerous cells in the patient's body before killing the patient himself -- and that, after the drugs have been stopped, the patient will be able to recover from the poisoning without the cancer. However, in the case of AZT therapy, the poison is administered for as long as the patient lives.

The fraudulent approval of AZT

The FDA approved AZT for marketing in 1987, based on the multi-center Phase II AZT trials, which were conducted in 1986. A two-part report on these trials was published in the 23rd July 1987 New *England Journal of Medicine* (Fischl 1987, Richman 1987). It was immediately apparent that there were serious problems with the report. The methodology section was incomplete and incoherent and the tables made no sense. The first part in particular, written by Margaret Fischl, was marred by contradictions, ill-logic and special pleading. This study, which would later be refuted by the Concorde study, directly led to the deaths of tens of thousands of people, who were not even sick before taking AZT.

Documents received from the FDA under the Freedom of Information Act showed that the trials were invalid on many counts: scientific rules were violated repeatedly, data were falsified, and the study was prematurely terminated. Although the study was designed to be, and legally required to be, "double blind" (where neither patients nor doctors knew who was getting the drug and who the placebo), in reality within a short time everyone knew who was getting what. [1] In his "Statistical Evaluation and Review", FDA statistician Lawrence Hauptman expressed dismay at the inadequacy of the Phase II trials. FDA toxicologist Harvey I. Chernov, in his "Review and Evaluation Of Pharmacology & Toxicology Data", reported that AZT was highly active in the Cell Transformation Assay and was therefore a "potential carcinogen". The animal studies were still in progress; however, even then "anemia was noted in all species (including man) in which the drug has been tested." **For these and other reasons Chernov recommended that AZT should not be approved.** Yet the FDA did approve AZT, as well as several other nucleoside analogue drugs which followed in its wake, for the treatment of „AIDS". These include ddI, ddC, d4T, and d3T. Although there are some differences in their toxicities, they all work on the same general principle as AZT.

The claimed mortality data -- that in only a few weeks 19 patients in the placebo group died, but only one patient in the AZT group -- were incredible and even preposterous. **The patients on AZT received no clinical benefits from the drug and experienced horrible side effects, and yet allegedly their lives were "extended".** On the other hand, the patients on placebo experienced a mortality rate many times in excess of what it should have been. In the ten years since the Phase II AZT trials were conducted, the "miracle" has never repeated itself. Never since then have there been results even remotely approaching the miraculous mortality data of the Phase II trials. It was clear even at the time that these trials could fairly be termed "fraudulent", inasmuch as FDA memoranda indicated that the investigators had deliberately used data which they knew were false.

The picture became clearer in 1992 when additional FDA documents were obtained under the Freedom of Information Act. These described in considerable detail the cheating that took place in Boston, one of the 12 centers. Patients who nearly died from the side effects of AZT were recorded as having experienced no adverse effects from the drug. Patients were recorded as having been in the study much longer than they were. The rules of the study (protocols) were violated right and left. And at least one patient on AZT was counted as a death in the placebo group. [2] The FDA team that uncovered the cheating in Boston recommended that the center be dropped from the study, and that none of the data be used. A high-level FDA meeting was held, at which the Commissioner of the FDA and top executives of Burroughs Wellcome were present, to decide what to do with all of the bad data, from the other centers as well as from Boston. An FDA woman shrewdly observed that if they were to throw out all of the bad data, there would be almost no one left in the study. The decision was then reached to eliminate nothing -- to throw in garbage along with

good data. For this reason alone the Phase II trials were fraudulent. It is never acceptable, under any circumstances whatever, to use false data knowingly (Lauritsen 1993).

Of all the hundreds of studies done on AZT, the Phase II trials remain the most important in one sense: they were the basis for FDA approval of AZT for marketing in 1987, and they were also the basis for AZT's approval in about 30 other countries, who followed the lead of the United States. For many years afterwards, the Phase II mortality data were still being used to claim benefits for AZT.

No scientifically credible benefits to AZT

In light of the cheating which took place in the *Phase II AZT trials* -- cheating which the manufacturer of the drug and the FDA knew about, condoned and covered up -- extreme skepticism is justified in evaluating studies claiming to show benefits of the drug. Any study which is paid for and/or controlled by *Burroughs Wellcome, Wellcome Pharmaceuticals,* or *Glaxo-Wellcome,* should be presumed to be fraudulent. At the very minimum, any such study should be thoroughly and independently audited before any credence is given it. In a letter to *The New York Times* (22. July 1995), Timothy H. Hand related having reviewed 25 major studies of AZT. **Those claiming benefits for AZT were funded and controlled by *Burroughs Wellcome* and were highly publicized. Those studies which found no benefits for AZT were not well publicized, although they had "good experimental design and independent funding".** Among other things, this episode shows that the peer review process of the leading medical journals is sadly inadequate. Not only did the *New England Journal of Medicine* publish the manifestly unsatisfactory Fischl/Richman articles, the editor of the journal, Arnold Relman, later refused to acknowledge that anything had been wrong with the study (Lauritsen 1993).

Protocol 019, the study that formed the basis of the FDA's approving AZT therapy for „asymptomatic" (that is, healthy) persons with positive results on the „HIV-antibody tests" (Volberding 1990), was a botched job, with meaningless tables, inadequate methodology description, gross mistakes in elementary statistics, and crude pleading for the drug (Lauritsen 1993). This study, which would later be refuted by the Concorde study, directly led to the deaths of tens of thousands of people, who were not even sick before taking AZT.

Although hundreds of papers have been published on AZT, there does not exist a single scientifically credible study establishing benefits of any kind from AZT therapy. **It is difficult to imagine how the health of a human being could be improved through the administration of a drug whose sole function is to terminate DNA synthesis.**

The toxicities of AZT

Azidothymidine can properly be called a poison, labeled as such with the skull-and-crossbones symbol by chemical supply companies (Duesberg 1996c).

The toxicities of AZT are extremely severe, and include anemia; myopathy (muscle disease, which manifests itself as muscular pain, muscular inflammation, and muscular atrophy); cachexia (wasting); nausea; headache; and damage to the kidneys, liver and nerves (Duesberg 1996 a-c, Lauritsen 1990, 1993). AZT is a known carcinogen: it is highly positive in a standard screening test for carcinogenicity, the Cell Transformation Assay; it causes cancer in rodents; and there is a strong correlation between long-term AZT therapy and cancer of the lymph system.

Many patients on AZT suffer from the "wasting syndrome" or cachexia. There can be no doubt that AZT plays a role in causing that cachexia -- by causing muscular atrophy, by damaging the intestines, by killing the friendly bacteria that are necessary for the absorption of nutrients, and by causing nausea. In an interview, the Swiss Professor of Immunology, Alfred Hässig, a leading authority on all aspects of blood transfusions, stated that damage to the bone marrow is not the worst of AZT's toxicities. According to Hässig, AZT has a deadly effect on the cells of the intestinal mucous membrane, the mitochondria in the cells and on the friendly bacteria in the intestines. [3]

Since AZT can directly cause several of the 30 "AIDS-indicator diseases" which form the official basis for an "AIDS" diagnosis in the U.S. -- and can indirectly contribute to causing most of the other 30 -- it logically follows that AZT can cause "AIDS" when administered to an a-symptomatic "HIV-(antibody)-positive" individual. For this reason it has been argued that since 1987 AZT has been the leading cause of "AIDS". (Duesberg1996 a-c).

The consequences of AZT treatment can easily be summarized: death! Unfortunately, there do not exist hard, actuarial statistics on AZT survival, which would tell us how long patients lived after beginning AZT therapy. At a 1991 meeting in London before the Broadcast Complaints Commission, representatives of Wellcome Pharmaceuticals admitted that no such survival data existed, although they were willing and eager to supply softer mortality data. They also admitted that they did not know how many patients in total had been prescribed AZT, and expressed contempt that anyone should want or need such information. And so, we can only state that thousands of patients with "AIDS" or with "HIV-(antibody)-positive" diagnoses have taken this costly toxin (as much as $ 12'000.-- per patient per year). This vague estimate is based on the yearly gross sales of AZT (about one-half billion dollars per year since 1987) in conjunction with the prevailing recommended doses of the drug.

When someone with an "AIDS" diagnosis dies, there is no official record kept of the drugs he was taking. It is highly likely that the great majority of them were taking AZT or a similar nucleoside analogue drug, but the official data do not exist. Obtaining such information should be high priority when an official investigation is begun into the AZT tragedy. It would be enlightening to gather statistics from the medical records of those who died under AZT therapy, especially the records of those who were still healthy before taking the drug. Did a decline in health accompany the initiation of AZT therapy?

Conclusion

A ruthlessly honest international investigation into all aspects of the „AIDS" phenomenon, including AZT crimes, should begin as soon as possible. This is not only necessary to save lives, but to save whatever remains of scientific and medical integrity.

Notes:

1. My detailed analysis of the Phase II AZT trials, "AZT On Trial", appears as chapter II in Poison By Prescription: The AZT Story. The FDA documents obtained under the Freedom Of Information Act are fully referenced here.

2. My report on the irregularities in the Boston center, "FDA Documents Show Fraud In AZT Trials", appears as chapter XXIX in The AIDS War. The additional FDA documents obtained under the Freedom of Information Act are referenced here.

3. Roger Müller, "Skepsis gegenüber einem Medikament, das krank macht: Azidothymidin, einst die 'erste Waffe gegen AIDS',AZT mehr und mehr im Kreuzfeuer der Kritik" ("Skepticism concerning a medication that causes sickness: Azidothymidine, once the 'first

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AZT IS INEFFECTIVE AND TOXIC TREATMENT FOR AIDS PATIENTS

by Eleni Papadopulos-Eleopulos, Val Turner et al., Perth, Australia

The genetic information of our own cells as well as that of many viruses is encoded by the linear sequence of the constituent building blocks of DNA. The building blocks are known chemically as nucleotides and there are four such nucleotides one of which is thymidine. Every set of three nucleotides in DNA signifies an instruction to insert a particular amino acid building block into a growing protein chain. To produce the protein chain the DNA is not used directly but is copied (transcribed) into an RNA chain which in turn codes for the proteins (translation). Some viruses contain only DNA and this DNA is used to produce the viral proteins via RNA. Other viruses including retroviruses contain only RNA and in some of these the RNA directly codes for the synthesis of viral proteins. According to retrovirologists, retroviruses are an exception in that their RNA is first copied „backwards" into DNA whereupon this stretch of DNA is inserted into the cellular DNA where it is carried as a DNA „provirus". There it lays dormant until the cell is activated, following which the proviral DNA is transcribed into RNA and thence the RNA instructions are used to construct proteins in the usual manner. To facilitate the copying of their RNA into the DNA provirus, retroviruses contain an enzyme (catalyst) called reverse transpcriptase.

The chemistry of producing DNA chains has two essential requirements:
1. The synthesis requires energy. This energy is carried in three phosphate groups attached to the 5'-OH group of each nucleotide. Thus every nucleotide next to be incorporated at the growing end of the DNA chain must be triphosphorylated
2. The last nucleotide in the growing chain must have a free 3' hydroxyl (3'-OH) group in order to attach the next nucleotide in the chain.

AZT is an analogue of the nucleotide thymidine, that is, AZT is similar overall in structure to thymidine, differing only in a small manner. The difference is that the 3' hydroxyl group of thymidine is replaced by an azido (nitrogen atom) group in AZT. Because of the similarity of AZT to thymidine, AZT can „trick" a growing DNA chain into taking it up in place of thymidine. However, since AZT lacks the 3'hydroxy group, once AZT is attached to the end of the DNA chain, no further DNA synthesis can take place.

In 1985 and 1986 researchers from the US National Cancer Institute (including Dr. Robert Gallo), Duke University, and the Wellcome Research Laboratories claimed to have shown that in cell cultures:
1. The cells are able to convert „inactive" AZT lacking the three phosphate groups into the „active" triphosphorylated form.
2. The triphosphorylated form of AZT inhibits the synthesis of HIV DNA. This means that no new cells can be infected with HIV and since cells already infected have a limited lifespan (days), any person taking AZT should rapidly become virus free and cured of infection.

Although the claims of these groups have been disproved by many other researchers [1-5], in 1986 AZT was introduced into clinical practice and still remains the most frequently used drug against HIV infection. However, since:
1. To act as an antiretroviral agent AZT must be triphosphorylated.
2. The AZT administered to patients is not triphosphorylated.
3. The cells of „HIV infected" individuals are not capable of triphosphorylating AZT

AZT cannot be an anti-HIV drug, either when used alone or in combination with other drugs.

As early as 1988 the HIV/AIDS experts including the researchers from the US National Cancer Institute, Duke University, and the Wellcome Research Laboratories expressed the view that due to its high toxicity AZT was of limited value in the treatment of patients who already had advanced disease, that is, AIDS [6]. At present, evidence exists showing that AZT causes „a significant increased risk of death among the patients treated early", that is, in patients who commence treatment when asymptomatic [7]. In other words, instead of curing patients or prolonging life AZT shortens it. Furthermore, since some of the clinical and laboratory manifestations of AZT toxicity mimic AIDS, it is impossible to differentiate between AZT toxicity and AIDS.

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IMPAIRED ENERGY PRODUCTION IN MITOCHONDRIA CAUSED BY NUCLEOSIDE ANALOGUES AS AZT (AZIDOTHYMIDIN, ZIDOVUDIN)

by Alfred Hässig, Bern, Switzerland

AIDS patients frequently show a significant weakening of their skeletal muscle. Until 1990 this was considered as HI-viral caused damage of muscles.

In 1991 a team of Japanese researchers showed that this muscle disease originated from damage caused to the mitochondria by AZT treatment: The excessive presence of oxygen free radicals has a detrimental effect on the mitochondria as to it's formation of ATP as a key substance of energy metabolism (1). The final sentence of this paper is: "However, for AIDS-patients it is urgently necessary to develop a remedy substituting this toxic substance AZT".

During the following years the toxic effects of nucleoside analogues in the treatment of viral diseases have been thoroughly studied. The multi-organ toxicity of these drugs was demonstrated on the heart muscles, the brain and nerve system as well as on liver and pancreas (2). In addition it has been demonstrated that successors to AZT, such as ddI and ddC, cause the same mitochondrial damage (3).

Both, pharmaceutical industry and registration authorities should have been obliged to seriously consider this mitochondrial damage caused by a long-term administration of nucleoside analogues as well as to prove that the mortality among AIDS patients is not connected to this drug treatment. In general, these obligations have been avoided and now create a serious new problem: Justified litigation claims and charges of intentional grievous bodily harm and homicide.

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HAS HIV BEEN ISOLATED?

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Anyone proposing that AIDS is caused by a unique, infectious retrovirus must have proof of the existence of such a retrovirus. Since the announcement of the discovery of certain laboratory phenomena claimed as proof of the existence of HIV we have critically analyzed the data and have always maintained that no such proof exists.[1-11]

A virus is a microscopic particle of particular size and shape (morphology) which contains particular constituents (biochemical properties) and which is able to replicate at the behest of living protoplasm. Replication of a virus-like particle is the property which defines the particle as being infectious. That is: **virus-like particle + replication = virus**. These defining data determine that the only way to prove the existence of a novel (new) virus is to (I) isolate viral-like particles, that is, first obtain the particles separate from everything else; (II) determine their morphological characteristics; (III) analyze their constituents (nucleic acid and proteins) demonstrating that such properties are those of retroviruses and are unique and (IV) prove that the particles are infectious, that is, when pure particles are introduced into non-infected cell cultures, new but identical particles appear.

Only then can the viral-like particles be deemed to a virus. **In the case of retroviruses, the steps in this procedure were developed over the half century that preceded the AIDS era and are described in Toplin and Sinoussi.**[12,13] These steps are:

Culture putatively infected cells demonstrating that such cultures contain retroviral-like particles which are almost spherical in shape and with a diameter of 100-120nM and having "condensed inner bodies (cores)" and surfaces "studded with projections (knobs)".[14]

1. Purify a sample in a sucrose density gradient. The latter is done by a process called density gradient ultracentrifugation. A test tube containing a solution of sucrose, ordinary table sugar, is prepared light at the top but gradually becoming heavier towards the bottom. A drop of supernatant (decanted) cell culture fluid is gently placed on top of the sucrose column and the test-tube is centrifuged for several hours at extremely high speeds. This generates forces many thousands of times gravity and particles present are forced through the sugar solution until they reach a point where their buoyancy prevents them from penetrating further. For retroviral particles, this occurs where the density reaches 1.16 gm/ml, the point where the particles concentrate or, in virological terminology, band. The 1.16 band can then be selectively extracted for further analysis.

2. Using the electron microscope (EM), photograph the 1.16 band proving there are particles of the correct morphology and no other material.

3. Introduce PURE particles into a virgin culture and, by repeating the above steps, prove that identical particles are produced.

To date, many electron micrographs of particles claimed to be retrovirus-like have been published. However, **not one of these micrographs demonstrates particles satisfying BOTH of the main morphological features of retroviral particles**, that is, a diameter of 100-120nM AND a surface studded with knobs. (HIV researchers are unanimous that the knobs contain a protein, gp120, which is essential for the particle to fuse with the membrane of an uninfected cell in order that the HIV particle core with its „HIV RNA" gains access to the cell, thus infecting it.[15]

To prove the existence of HIV, both Montagnier's group in 1983 and Gallo's group in 1984 banded supernatant in sucrose density gradients. However, for reasons unknown, until March this year (1997), neither these groups, nor anyone else, had ever published an electron micrograph of the banded (purified) material to show which, if any, of the particles seen in gross cell cultures are present at 1.16 gm/ml. **Indeed, until this year, it was not possible to know whether any structured material whatsoever was present at the density which defines retroviral particles.** Nonetheless, from the time of the Montagnier and Gallo studies in 1983/84[16,17], the material from culture supernatants banding at 1.16 gm/ml has been regarded as containing nothing else but PURE HIV particles. Acting on this premise, the proteins which are present in this band and which react with antibodies present in the sera of AIDS patients are claimed to be THE HIV proteins and the antibodies reacting with such proteins THE HIV antibodies. Similarly, a particular portion of the RNA banding at 1.16 gm/ml is claimed to be THE HIV-gnome. All conclusions have been drawn without ever proving that the proteins and RNA are structural elements of a particle, viral-like, retroviral-like or any other particle of any other kind, that is, without any scientific basis.

This March, two papers[18,19] were published with electron micrographs of sucrose density gradient banded material. Although the authors of these studies claim proof for the presence of HIV particles in this material, neither of these studies contains proof of retroviral particle isolation. To the contrary, the data in these papers support our claim that HIV has not been proven to exist and that banded specimens are *assumed* to be pure HIV particles: "Virus to be used for biochemical and serological analyses [using "viral" proteins to test for antibodies in patients] or as an immunogen [to produce antibodies in animals and test patients for "viral" proteins] is frequently prepared by centrifugation through sucrose density gradients. The fractions containing viral antigen [proteins] and/or infectivity *are considered to contain a population of relatively pure viral particles*"[19] (italics ours). That these data negate such assumptions and considerations is illustrated by the following:

1. The authors of both papers admit that the particles which are present in the banded material and which are said to be HIV particles represent only a very small fraction of the total material. Gelderblom *et al* state that the material contains "an excess of [cellular] vesicles with a size range 50-500nm, as opposed to a minor population of virus particles... cellular vesicles appear... to be a major contaminant of HIV preparations enriched by sucrose gradient centrifugation".
2. The particles do not appear to have on their surface, spikes (knobs), although the possibility that such projections many have been present cannot be excluded. (However, in other papers published by many researchers including Gelderblom and his associates, such projections are noted to be absent.[14,20]
3. The particles referred to as "HIV" are not spherical and have diameters exceeding 100-120 nM. In the EM in Gluschankof *et al*[19] there are arrows pointing to five "HIV" particles whose dimensions are 121 X 145; 121 X 169; 121 X 145; 121 X

145 and 133 X 145 nM respectively. In Bess *et al,*[18] there are a total of six "HIV particles" whose dimensions are 160 X 240; 200 X 240; 280 X 280; 208 X 250; 167 X 250 and 250 X 292 and nM respectively. Thus, by definition, the particles cannot be retroviral-like particles and even less, a unique retrovirus, HIV.

4. Assuming the Franco/German and US groups spun their specimens to equilibrium (the standard practice) and sought particles at the correct, retroviral density, then the particles found by both groups must have that same density, 1.16 gm/ml. If the major and minor diameters of the particles in the EMs they claim are HIV are measured and the average diameters taken, the Franco/German particles are 1.14 times larger than genuine retroviral particles (upper diameter=120nM) and the US particles are 1.96 times larger. This translates to 50% and 750% greater volumes respectively than retroviral particles. Also, the US particles are five times more voluminous than the Franco/German. Thus, since genuine retroviral particles have a density of 1.16 gm/ml and the Franco/German and US particles share the same density, they must contain 50% or 750% more mass than *bona fide* retroviral particles. If HIV contains a fixed amount of RNA and protein the Franco/German and US particles must contain significantly more RNA or protein or both. **Thus these particles cannot be identical to HIV.**

Conclusions

Indeed, the method adopted by all HIV researchers for proving the existence of HIV, that is, excluding proof based on purified particles with retroviral morphology but rather detection of antibody/protein reactions, does not satisfy any scientific principle and defies common sense.

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NON-SPECIFIC REACTIONS ON HIV ANTIBODY TESTS

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HIV antibody tests are the predominant means of diagnosing HIV infection and AIDS. Claims of their accuracy are made on the basis that one type of HIV antibody test will often reproduce the results of another. Test results are not compared to presence or absence of virus in the body of the patient (called a gold standard).

HIV antibody tests were originally developed by obtaining blood from a person with AIDS and growing it in tissue culture. **Without proof, it was assumed that this blood contained a retrovirus, HIV**,[1][2] and therefore whatever grew in the cultures was HIV. It was thought that virus budded out of the cells into the culture fluids, so the material in the fluid which had the density of retroviruses was broken up into a mixture of what was assumed to be HIV proteins (antigens). The two tests predominantly used today are the ELISA and the Western Blot. In the ELISA, this mixture of proteins is exposed to a blood sample and any antibodies in the blood that can bind to these proteins are allowed to do so. The clinician cannot tell which particular proteins have reacted, just that some proteins have. This is in contrast to the Western Blot, in which the proteins are separated onto strips (bands) on a gel plate an reactions to individual proteins show up as coloured bands.

The accuracies of HIV antibody tests require that these tests will not cross-react with antibodies produced by non-HIV agents. Indeed, the studies in which these tests were established[1] assume that 1) antibodies are specific, meaning that they react only with the antigens that elicited them)[3] and 2) proteins in the test kits (which are exposed to a patient's blood sample) are HIV proteins. However, since the publication of these papers, scientists have established **over 60 non-HIV entities and non-AIDS conditions which trigger antibodies that react with the proteins in HIV antibody test kits**. Included is a list of these entities and conditions, many of which are common among the groups officially considered to be at high risk for both AIDS conditions and positive HIV antibody tests (see below).

How hepatitis causes false-positive HIV antibody tests

The repeated appearance of hepatitis and its variants on the list below illustrates the problem of cross-reactivity in relationship to HIV antibody tests:

One of the major proteins of HIV is gp41 (which is found in the outer layer or "envelope" of the virus), and a positive reaction to the gp41 band on a Western Blot is considered to be fairly definitive for HIV infection.[4][5] However, it has been demonstrated that antibodies against actin, a ubiquitous cellular protein, react with gp41,[5][6] and that gp41 might actually be actin itself.[6] For example, well-studied retroviruses such as the Rous sarcoma virus have been shown to contain cellular actin.[6] Thus the "HIV" gp41 in test kits may not originate from HIV at all.

When replicating, hepatitis B and C viruses leave the infected cell, coating themselves with cellular actin. Antibodies are formed not only against the virus but against the actin as well.[7] That might explain why these viruses can cause anti-gp41 reactions on HIV Western Blots. Since AIDS risk groups have in common a high exposure to hepatitis B and C viruses, this problem should be of considerable concern.

Do HIV antibody test kits really contain HIV antigens?

Does a positive test indicate reactions with normal cellular proteins rather than reactions to HIV proteins? The answer may lie in recently published studies, one Franco/German and the other from the United States National Institute of Cancer, which describe the material from which HIV proteins are obtained for use as antigens in the test kits.[8][9] This material comes from HIV culture fluids and bands at the retrovirus density. The material in this band (or density gradient) is assumed to contain nothing but HIV, and for this reason is officially called "purified HIV." Proof that this is actually the case would be electron microscope (EM) photos of this density gradient which showed that the gradient's contents were exclusively, or nearly exclusively, material that fit the criteria by which retroviruses are defined. These two research teams took the first EM photos of an HIV density gradient ever to be published. However, **the photos didn't show anything that qualified as a retrovirus**! Prior to publication of these two papers, the only EMs available were of entire cell cultures or of culture fluids that had not been density-purified, both of which contained much non-HIV material. However, the density gradient EMs fail to prove that the retrovirus density gradient is exclusively inhabited by retroviruses (or inhabited by retroviruses at all!). An HIV density gradient is obtained by first extracting the culture fluids which supposedly contain the virus and spinning them in a high-speed centrifuge containing layers of sugar solution, each layer becoming progressively more dense. Because of their particular buoyancy, retroviruses are said to settle at one particular layer (1.16 gm/ml) of the density gradient, and in spite of much evidence that some cellular proteins have the same density it is still believed that this density gradient belongs to retroviruses alone. Since much other non-retroviral material bands at 1.16 gm/ml, it is necessary to analyse the gradient further. The US and Franco/German teams both performed purification using only density as a criterion, and they concede that most of the objects or particles in their pictures consist of cellular material: "microvesicles," or encapsulated cell fragments.

Retroviruses are strictly defined as possessing certain physical characteristics, not only a particular density, a particular size and shape, but a relative uniformity of their size and shape as well. Pictured in the density-purified bands are objects or particles considered to be HIV, yet these particles are too large, and of the wrong shape, and they also consist of a great variety of sizes and shapes, which does not support the view that 1) these particles represent members of the same species or 2) these particles are retroviruses.

Experts have agreed that retroviruses are spherical in shape, have a diameter of 100-120 nanometers (nm), and are covered with knobs, surface molecules which attach to cells in a way that facilitates infection.[13] However, the particles the two groups claim are HIV are not spherical, all have diameters of greater than 120 nm (in fact, many of them have diameters exceeding twice that permitted for a retrovirus), and none of them appear to have knobs. In terms of volume, the Franco/German "HIV" particles have 50% more volume than a retrovirus, and the US particles have 750% more volume.[13] The point is that any genuine retrovirus contains a fixed amount of RNA and protein, no more and no less. Too much volume means too much protein to be a retrovirus. On this poor showing, it is not possible to assume that the material extracted from the density gradient and used as antigen in the test kits actually represents HIV proteins.

Does cross-reactivity invalidate an antibody test?

Non-specific reactions (cross-reactivity) are a fact of nature when it comes to antibody tests, not just those for HIV. For example, Chrystie et. al noted that HIV-positive Africans were often testing positive for malaria as well, and wondered if the presence of anti-malarial antibodies could be used to "enhance the value of anonymous, unlinked HIV seroprevalence studies."[10] However, they discovered that 65% of 171 HIV-positive

Caucasians who had never travelled to malaria-endemic areas, and presumably had no reason to be exposed to malaria, nevertheless had positive antibody tests for malaria. The same was true for a more rare condition, leishmania. They concluded that anti-HIV antibodies were causing cross-reactions (false-positives) on the malaria and leishmania antibody tests.

The presence of cross-reactions does not automatically invalidate an antibody test. There are ELISA and Western Blot tests for many other microbes, not just HIV. All viral tests cross-react with entities other than what they are targeting, yet they are still of value as diagnostic tools. Why, then, is cross-reactivity such a big problem for HIV antibody tests? The main reason is because HIV antibody tests, unlike other antibody tests, have not been validated against viral isolation in fresh (uncultured) sera. For example, if the hepatitis ELISA test claims to be 99% accurate in people with hepatitis symptoms, it means that in validation studies, hepatitis virus can always be isolated from 99% of the people with hepatitis symptoms who test positive. So it wouldn't matter if a thousand things cross-reacted with the hepatitis ELISA test. If a patient has hepatitis symptoms and tests positive, there's a 99% chance the person has a hepatitis infection. **HIV antibody tests have not been validated against viral isolation**. For HIV ELISA tests, a 99% accuracy means that 99% of the time a positive test result can be duplicated by a positive result on a different type of HIV antibody test, typically the Western Blot. **"Accuracy" is determined merely on the basis of the ability to reproduce the test results on another test. At no time is demonstrated any direct evidence of an actual viral infection**. Furthermore, HIV test "accuracies" are assumed to hold for people with and without symptoms. It is recognised that valid viral tests have high accuracies in people expressing symptoms associated with the virus in question, and low accuracies in symptom-free people. Accuracies in symptom-free people are quite low, perhaps as low as the 20-30% range. HIV is the only instance where millions of tests are performed on people who have no symptoms, have not been exposed to HIV, and have very little, if any, potential for exposure to it. Concern has often been expressed about the unacceptably poor performance of HIV antibody tests in low-risk populations. [14][15] Tests for other viruses such as hepatitis B are not generally administered to people without symptoms or history of exposure, since it is recognised that positive results would more than likely be false-positives in this group, thus rendering the test useless in this context. The exception to this is people in identified special risk categories.

The other aspect to consider is that HIV tests probably cross-react with more common things than other tests, since all "HIV" components are probably normal cellular entities, such as actin.

HIV science relies on the existence of specific antibodies -- antibodies that react only with the HIV proteins. If there were such a thing, their data would work. But you'd need to isolate HIV first to prove that the antibodies were specific! If properly isolated HIV as a gold standard were used to verify the tests, the presence of cross-reactions and the unknown identity of the proteins in the test kits would not matter. You could use peanut butter as the test kit antigen if it could be established that peanut butter proteins would react with a person's blood if and only if HIV was present in that patient's body as proved by virus isolation. So far this has not been done.

Even if one is unwilling to accept that the reagents in HIV antibody test kits are composed of cellular proteins, not HIV proteins, there are other, more obvious, flaws to consider. In the United States, ELISA tests are used only as screening tests because they are considered to be so grossly inaccurate. According to Langedijk, almost all reactions on the ELISA represent false-positive results.[11] A diagnosis of HIV infection is never made on the basis of an ELISA test alone. The Western Blot test is considered to be more accurate

and is thus used to confirm the results of the ELISA. However, in England, it is just the opposite. The Western Blot is considered to be grossly inaccurate and therefore the ELISA is used as the definitive diagnostic test. One wonders how a test can gain in accuracy merely by being shipped overseas. An HIV Western Blot is read as positive when a certain number of major "HIV" protein bands are reactive with the patient's blood sample. Exactly how many of these protein bands must react before the test is read as positive? Well, it could be one...or two...or three...or four. **Such vastly different criteria can be used to read Western Blots that you can change from positive to negative simply by going to a different lab or taking your test in another country**. For instance, in Australia, four major bands are required for a Western Blot to be positive; in the United States, generally only two are required. A recent study noted that according to a test kit manufacturer's revised criteria for reading Western Blots, 8% of all positive Western Blots performed in 1991 and 1992 would now be re-classified as false-positives.[12] So, are these 8% infected or not?

ELISAs, on the other hand, are read according to the strength of the overall reaction. However, it's not a matter of a simple "yes" or "no," (reactive or non-reactive). **In fact, just about anyone's ELISA will react a little bit**. This small reaction is considered to be due to normally-occurring non- specific reactivity to non-HIV antibodies. Whether the ELISA is read as positive or not depends on *how much* it reacts and where the test kit manufacturer has arbitrarily set the cut-off point. Values above the cut-off point are read as positive and values below the cut-off point are read as negative. The cut-off point can be adjusted to increase or decrease the sensitivity of the test. If you make the cut-off point lower, more tests will be read as positive, and more people will suddenly be "infected with HIV."

Supposedly certain "patterns" in the strength of reactivity are associated with HIV infection, although in the absence of a gold standard to compare these patterns to (a sample of properly isolated HIV), this is a doubtful assertion. **And no one has explained why a tiny amount of HIV antibodies would not indicate HIV infection, but a larger amount would.** Why do these antibodies miraculously change into "HIV antibodies" simply because there are more of them and the strength of the reaction has exceeded a certain value? Persons with AIDS and third world persons considered to be at risk for AIDS share a common characteristic: they have been exposed to a multitude of foreign proteins and antigens, microbes and disease conditions which can give rise to cross-reacting antibodies. Bearing in mind the large number of factors causing potential cross-reactions, the more of these conditions that are present in any one person, the more likely there are to be some sort of cross-reacting antibodies, and hence a positive HIV antibody test. Although it goes against "conventional wisdom" on the matter, it is exactly the people in AIDS risk groups who are testing false-positive.

Conclusion

There appears to be no evidence that antibodies raised against proteins in HIV antibody test kits are antibodies triggered by retrovirus infections. Instead, the evidence provided by recent electron micrographs supports the view that positive HIV antibody tests are triggered by 1) antibodies to normal cellular proteins, or 2) antibodies to proteins associated with other, non-retroviral, diseases and conditions. People are being diagnosed as infected with a lethal virus on the basis of utterly arbitrary standards such as these. A careful examination of the scientific literature discloses no conclusive evidence that HIV antibody test kits contain HIV antigens or that people with AIDS really have HIV antibodies in their blood.

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DOCUMENTED CAUSES OF FALSE-POSITIVES ON HIV ANTIBODY TESTS

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- Hepatitis (1)(49)
- Hepatitis B vaccination (2)(3)(4)(22)
- Alcoholic hepatitis (4)(5)(6)(7)(21)(22)(23)(24)
- Q-fever with associated hepatitis (8)
- Gamma globulin or immune globulin (given as prophylaxis against infection) which contains antibodies from the donor (9)(6)(22)(25-29)
- Leprosy (10)(11)
- Tuberculosis (11)
- Mycobacterium avium (11)
- Renal failure (13)(6)(23)
- Hemodialysis (14)(5)(7)(30)(31)(49)
- Alpha interferon therapy in hemodialysis patients (1)
- Flu (15)
- Flu vaccination (16)(17)(18)(19)(6)(32)
- Tetanus vaccination (4)
- Herpes simplex I (20)
- Herpes simplex II (17)
- Upper respiratory tract infection (common cold) (17)
- Recent DNA virus infection or exposure to viral vaccines (4)(6)(21-23)(17)(43)
- Pregnancy (Multiparous women) (21)(6)(15)(22)(33)
- Malaria (34)(35)
- High levels of circulating immune complexes (34)(36)
- Hypergammaglobulinemia (high levels of antibodies) (4)(36)
- False-positives on other tests (such as RPR test for syphilis) are associated with false-positives on HIV antibody tests (5)(7)(23)(36-38)(49)
- Rheumatoid arthritis and samples positive for rheumatoid factor (21)(15)
- Renal transplantation (6)(14)(23)(40-41)
- Other organ transplantation (15)(42)
- Autoimmune disorders: (4)(5)(7)(12)(13)(22)(38)(39)(49)
  - Systemic lupus erythematosus
  - Diskoid lupus erythematosus
  - Scleroderma
  - Connective tissue disease
  - Dermatomyositis
- Malignant neoplasms (cancers) (4)
- Hematologic malignant disorders/lymphoma (6)(21-23)(41)
- "Sticky" blood (4)(44-45)
- Antibodies with a high affinity for polystyrene (used in the test kits) (4)(18)(47)
- Multiple blood transfusions (6-7)(15)(22)(31)(49)
- Multiple myeloma (5)(21-22)
- Hemophilia (5)(7)(49)
- Primary biliary cirrhosis (6)(21-23)
- Private sclerosing cholangitis (21)(23)
- Bilirubinemia (49)
- Multiple sclerosis (50)
- Dermatological disorders (50)

- Stevens-Johnson syndrome (6)(23)(41)
- Lipemic serum (high cholesterol) (7)
- Hemolyzed serum (7)
- Hyperbilirubinemia (5-6)(49)
- Globulins produced during polyclonal gammopathies (5-6)(23)(49)
- Poorly-understood or uncategorized cross-reactions in healthy individuals (5)
- Normal human ribonucleoproteins (6)(23)
- Non-HIV retroviruses (6)(23)(46)(51-52)
- Proteins on the filter paper (6)
- Heat-inactive (heat-treated) serum (7)(23)(53-55)
- Receptive anal intercourse (anti-sperm antibodies) (56-58)
- Candida albicans and other funguses (58)
- Epstein Barr (65)
- Visceral Leishmaniasis (66)
- Anti-carbohydrate antibodies (6)(59-60)
- Naturally-occurring antibodies (61)
- Anti-lymphocyte antibodies (14)(62)
- Anti-collagen antibodies (62)
- HLA antibodies (to Class I and II human leukocyte antigens) (5-7)(21-23)(48-49)(63-64)
- Serum positive for autoantibodies (including rheumatoid factor and anti-nuclear antibodies) (6)(21)(23)(46-47)
- Antimitochondrial antibodies (6)(23)
- Anti-microsomal antibodies (45)
- Anti smooth muscle antibodies (23)
- Anti parietal cell antibodies (23)
- Anti hepatitis A IgM (23)
- Anti-HBc IgM (23)
- T-cell antibodies (6)(23)

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HIV Positive? - It depends where you live

Take a look at the criteria that determine a positive HIV test result

The HIV antibody tests do not detect a virus. They test for any antibodies that react with an assortment of 'virus-proteins' that experts assure us are unique to HIV.

According to AIDS-experts, the ELISA test is not very specific and may react in the absence of HIV infection, and thus if positive is repeated, and if still positive warrants a third, but different, test called the Western blot. In the Western blot the 'virus-proteins', about ten of them, are located at discrete spots in a paper strip. Serum is added and whenever there is a reaction with some antibodies a colour change occurs which shows up as a dark band. The test is read by noting which bands show up, in other words, which proteins are reacting. Certain combinations of bands are defined as a positive test.

It is most peculiar that the location and number of bands required for a positive Western blot VARY around the world, and may even vary between laboratories within the same city. In Australia at least four bands are required, in Canada and much of the USA three or more. And in Africa two will do. If each indicates HIV infection then HIV must cause different populations of antibodies to appear in different places. Does that not sound very odd? Can HIV navigate? But at least it gives some Africans a way out: all an African has to do is retest in Australia because two bands would not be considered positive there.

Nevertheless, in spite of lack of standardisation and other problems such as reproducibility, the Western blot is promoted as greater than 99.9% specific, and if positive is regarded as synonymous with HIV infection.

A Martian might be forgiven for wondering whether wine-tasting was less subjective. ■

Western Blot 'virus proteins'	Africa	Australia	U.S. Food & Drug Admin	U.S. Red Cross	CDC (1)	CDC (2)	CON[1]	MACS[2]	UK
ENV gene: p160, p120, p41	ANY 2	1 OR >	1 OR >	1 OR >	p120/ p160 AND p41	p120/ p160 OR p41	p120/ p160 OR p41	ANY 1 Strong OR 3 Weak bands from: p15, p24, p32, p41, p45, p53, p55, p64, & p120. Score '1' for each weak band, and '3' for each strong band - total of '3' or greater is positive	1 OR >
POL gene: p68, p53, p32	OPTIONAL	ANY 3	p32	ANY 1			p32 OR		p31 (sic)
GAG gene: p55, p40, p24, p18			p24	ANY 1		p24	p24		p24

HIV – Western Blot Test Criteria in various centres around the world
With thanks to Dr Val Turner

1) Consortium for Retrovirus Serology Standardisation
2) Multi-Centre AIDS Cohort Study (USA)

WHAT CAUSES A POSITIVE TEST FOR HIV-ANTIBODIES

by Vladimir Koliadin, Kharkov, Ukraine

Introduction

In 1993, the widely accepted ideas that a positive test for HIV-antibodies is specific (i.e. means HIV- infection) and that HIV exists as an exogenous retrovirus were challenged [1]. Then, these views were developed by the same [2] and other authors [3,4]. Neither experimental nor even logical arguments were advanced by mainstream AIDS-scientists to disprove the central points in [1]. At present, it is still accepted in mainstream AIDS-science that existence of HIV and high specificity of HIV-antibody tests are ultimately proven scientific facts. Besides conformity of thinking and vested interests, this belief is maintained by the absence of clear-cut alternative explanations to the phenomena which are thought to prove the HIV-causes- AIDS theory. Some important questions have not been answered. If HIV does not exist, what causes a positive test for HIV-antibodies? Why almost all AIDS-patients are seropositive while almost all healthy individuals are not? Why is the positive test strongly associated with high mortality and susceptibility to opportunistic infections?

The heart of any HIV-antibody test is so called HIV-proteins. These proteins are produced by cell- cultures of human lymphocytes. According to the mainstream AIDS-science, these proteins belong to a new retrovirus HIV, by which these cell-lines are presumably infected. HIV-skeptics say that these proteins are of endogenous (intracellular) nature [1]. If to follow facts and basic principles of immunochemistry, the only conclusion which may be drawn from a positive test for HIV- antibodies is that some immunoglobulins in the tested blood-serum have bound to these "HIV-proteins". This is the case for both ELISA and Western Blot (WB) tests, as well as for any other immunochemical test. The point of disagreement is how to interpret such binding. Mainstream science insists that such binding is due to specific antibodies, produced by the immune system after its contact with HIV. The authors of papers [1,2] explain this binding by cross-reaction between "HIV-antigens" and antibodies to other non-HIV antigens, to which AIDS- patients and individuals from high-risk groups are exposed. Such cross-reaction is well known to occur when different antigens share common parts ("antigen determinants"). In this case, antibodies to one of such antigens, are capable to bind selectively to other antigen with the same antigen determinants. Nevertheless, the cross-reactivity hypothesis suffers from one problem: how to explain that almost all AIDS-patients and many people in high-risk groups have been exposed to some antigens bearing common determinants just with "HIV-antigens"? Why almost nobody beyond the risk-groups have not been exposed to such antigens, though anybody in course of his/her life have been exposed to many antigens (vaccinations, infections, etc.)?

Why HIV-antibody tests are thought to be specific

In the middle of 1980s, it was shown experimentally that a test for HIV-antibodies is positive in a great proportion of patients with diagnosis of AIDS or pre-AIDS, while it is negative in almost all perfectly healthy individuals. It is essential that AIDS-diagnosis in these patients was established from clinical symptoms, not from HIV-test itself. This undermines the "circular definition" argument advanced by some AIDS-dissidents. Moreover, such studies were carried out according to a blind and randomized protocol. This gave a great credibility to the HIV-causes-AIDS viewpoint. Do such studies actually prove that HIV-positivity is specific to AIDS? Even though it may seem "self-evident" at first sight, it is not so. An important principle of evidence-based science was obviously violated in these studies: **the control group was not matched to the AIDS group - perfectly healthy individuals were used as controls** [1].

Why is the principle of matched control so important? The following simple example provides some explanation. Let a disease is characterized by some feature, say by elevated body temperature. Let each of 1,000 patients with this disease demonstrates high temperature, whilst each of 1,000 healthy controls - normal (lower) temperature. Does it mean that body temperature is specific to this disease? Surely not - simply because the high temperature, though being highly unusual in healthy individuals, is typical to many other diseases. For the same reasons, the ability of HIV-test to discriminate between AIDS-patients and healthy individuals cannot prove specificity of HIV-tests. This obvious flaw in the studies of "specificity of HIV-antibody tests", spotted in paper [1], was ignored by the mainstream science. In respect to which symptoms the control group should be matched to AIDS-patients? It is not an easy question: many symptoms and signs are typical to AIDS. In paper [1] such symptoms were not specified. In the next section an attempt will be made to fill this gap.

There are also other studies, carried out with a great number of individuals (applicants for US military service) in which specificity of HIV-tests was reported as 1 false positive reaction in more than 135,000 [5]. Nevertheless, these studies have nothing to do with proofs of existence of HIV or specificity of HIV- antibody tests because just WB test-systems were used as the gold standard of HIV. The only thing shown by these studies definitely is that results provided by some ELISA systems in multistep testing algorithms are well compatible with results of WB tests, at least in a population with a very low prevalence of HIV-seropositivity. Just such question was addressed in these studies, not specificity of WB-tests themselves.

Non-specific immunoglobulins as the cause of HIV-seropositivity

The B-lymphocytes (a part of the immune system) normally respond to foreign substances (named "antigens") by increased production of special proteins -- immunoglobulins. Some of these immunoglobulins are capable to specific binding to the antigen -- that is, they bind strongly to the antigen but not to other substances. Such immunoglobulins are usually named "antibodies". Other immunoglobulins, which are capable to bind to a wide range of antigens, are named "non-specific immunoglobulins". Many authors name all immunoglobulins "antibodies"; to discriminate between the two types they use terms "specific antibodies" and "non-specific antibodies" (or "normal antibodies"). The ability of (specific) antibodies to bind only to the antigen which induced production of these antibodies plays a very important role in biomedical science and serological testing.

If HIV-antibody tests are positive in AIDS-patients and negative in healthy controls, does it mean that blood-serum of AIDS patients contains some factor which is absent in healthy individuals? Most scientists (not specialists in practical immunochemistry), let alone laymen, may believe that at least this fact was proven beyond any reasonable doubt by such studies. Nevertheless, this is not the case: **blood-serum from healthy individuals is also capable to provide positive WB. The only difference is that titers of this reaction are higher for AIDS-patients than for healthy controls.** The titer is the maximal dilution of the serum for which reaction is still noticeable. For example, Gallo and his group managed to obtain "specificity" (in respect to healthy controls) only after 500-fold dilution of the serum [6,7]. Thus, blood serum from healthy controls does provide a positive WB-test, but if diluted less than 1:500. In practical immunochemistry, such a phenomenon is explained by binding of non-specific immunoglobulins, which are present in any individual and bind to almost any antigen. **In commercial WB-systems this effect is not noticeable because sensitivity is artificially lowered to avoid positive reactions in healthy individuals.**

How to decide whether a test is positive because of binding with specific antibodies or only with non- specific immunoglobulins? In practice of immunochemical testing this problem is

apparently resolved by reduction of sensitivity (e.g. by dilution of the tested serum) up to the levels which guarantee negative tests in healthy controls. Such an approach tacitly implies that reactivity and concentrations of non-specific immunoglobulins are about the same in all individuals. Even though being widely accepted, this practice is dubious. If some states of organism are characterized by essentially higher levels of non-specific immunoglobulins or by their higher affinity (ability to bind), blood serum from such individuals can yield a positive test too. How to prove that a positive test for a given sample is caused just by specific antibodies, but not by non-specific immunoglobulins? By definition, specificity of antibodies means they are capable to bind only to one antigen (or very similar ones). Hence, it is necessary to check how this serum reacts with a great many of other antigens. If titers of these reactions are abnormally higher only in respect to the given antigen (as compared with titers of serum from healthy controls), it proves that reaction has been caused by specific antibodies. On the other hand, if the serum reacts with many other antigens in titers higher than normal, this is a strong reason to suspect that the test is positive due to non-specific immunoglobulins. Unfortunately, such multi-antigen tests are not currently used to confirm specific nature of a positive single-antigen test. This is partially explainable by much higher technical complexity and cost of such verification tests, absence of commercial toolkits, as well as by lack of interest of the mainstream immunology to non-specific phenomena.

There are several reasons to assume that tests for "HIV-antibodies" are positive in AIDS-patients due to non-specific immunoglobulins. First, **it is well known that total concentrations of non-specific immunoglobulins are much higher in almost all AIDS-patients than in healthy individuals**. This condition, named hypergammaglobulinemia, is highly non-specific and observed in many diseases and abnormal states of organism. (Causes of such a condition will be considered below.) Thus, **the principle of matched control is obviously violated** in the aforementioned studies - not healthy individuals, but just patients with hypergammaglobulinemia (but without AIDS) had to be used as controls. Second, in many AIDS patients, titers of "HIV-antibodies" are low. Perhaps for these reasons information about titers is conspicuously absent in AIDS-literature. For example, in Russia, due to shortage of test systems so called "pool-method" was frequently used - when blood from several individuals was mixed and tested. It was noted, that even such 2-8 fold dilution of the serum, may have resulted in a false negative test [8]. Thus, titers of putative "HIV-antibodies" in some AIDS patients are about 1:2 - 1:8. In immunology such low titers are usually not considered as significant at all [9]. Third, AIDS patients and individuals from groups with high risk of AIDS are known to be seropositive in respect to many other antigens (e.g. hepatitis-B virus, EBV, etc.). Even though this is usually interpreted as a sign of infection by these pathogens, it is also well explainable by non-specific reactivity of the serum of these individuals [3].

Non-specific reactivity should not be confounded with cross-reactivity [3]. Cross-reactivity is a specific phenomenon, well understood in immunology. Even though cross-reacting antigens differ, they share common determinants, and reaction is specific in respect to these determinants. Normally only a small proportion of antigens cross-react to antiserumagainst a given antigen. In contrast to the cross-reactions, neither mechanisms of production nor properties of non-specific immunoglobulins are understood in modern immunology. Textbooks of immunology either keep silence or say only a few words about their existence. It is also rarely mentioned in the textbooks, that **only a part, maximum 20-30 percent, of the immunoglobulins produced by B-cells in response to an antigen are capable to bind specifically to the antigen; other 70-80% of the immunoglobulins are non-specific** [10]. In [3] a hypothesis was advanced that affinity of non-specific immunoglobulins increases radically if the B-lymphocytes are over-stimulated, , and that just this effect leads to positive tests for HIV-antibodies.

The problem of non-specific immunoglobulins is exacerbated in the tests which are based on "sandwich" principle (as in ELISA and WB). In these test-systems, the antigen (e.g. "HIV- antigen") is attached to a surface and forms the first layer in the "sandwich". After exposure to the tested serum, immunoglobulins from the serum bind to the antigen and form the second layer in the "sandwich". To detect these immunoglobulins and estimate their quantity the two-layer sandwich is subject to one more procedure - it is being exposed to antibodies against human immunoglobulins (which are obtained from laboratory animals immunized by human immunoglobulins). These animal antibodies bind to the human immunoglobulins and form the third layer of the sandwich. Just the amount of the animal antibodies bound to human immunoglobulins, not quantity of the human immunoglobulins themselves, is being measured in test-systems of this sort [9]. It is tacitly postulated that affinity (ability to bind) of the animal antibodies to human immunoglobulins is about the same for all individuals and, hence, the amount of the animal antibodies bound is simply proportional to the amount of human immunoglobulins. But this is not the case if non-specific immunoglobulins from some individuals are capable to bind more strongly to various substances than immunoglobulins from healthy controls. Let, for example, such non- specific affinity of non-specific immunoglobulins increased 3 times in respect to various proteins, including both "HIV-antigens" and animal antibodies. Then, the number of molecules of human non-specific immunoglobulins bound per one molecule of the antigen is 3 times higher. After exposure to animal antibodies, 3 times higher number of molecules of the animal antibodies will be bound per one molecule of human immunoglobulins. Thus, the total amount of animal antibodies will be 9 times (3 multiplied by 3) higher, not 3 times. Hence, even a moderate increase in non- specific affinity of immunoglobulins may lead to radical increase in the titers and, hence, to HIV- seropositivity.

What increases the levels of non-specific immunoglobulins

As far as elevated levels of non-specific immunoglobulins and their higher affinity may lead to positive tests for "HIV-antibodies" it is reasonable to consider the causes of such conditions. It is widely accepted that hypergammaglobulinemia (which is typical to AIDS and many other conditions) results from polyclonal activation of B-lymphocytes caused by intensive stimulation by multiple foreign antigens. Individuals in groups with high risk of AIDS are abnormally frequently exposed to such multiple antigens [1]: multiple infections, including STD and AIDS-defining opportunistic infections, anal reception of sperm, contaminants of injected recreational drugs, foreign blood received either in course of sharing syringes for injection of such drugs or as transfusions, contaminants of the blood factors received by hemophiliacs as well as these factors themselves. Thus, the significantly higher frequency of positive tests for "HIV-antibodies" observed in the groups with high risk of AIDS is highly likely to be nothing but an indicator of such over-stimulation by multiple foreign antigens and, in many cases, an indicator of some diseases. **There are not any factual reasons to consider the antigen overload itself as a life- threatening factor**.

Along with the above mentioned factors, one more factor of antigen overload deserves a closer attention - prolonged use of broad spectrum antibiotics. Such an antibiotic abuse is closely associated with high promiscuity (frequent change of sexual partners): antibiotics are used to treat frequent STD as well as permanent prophylaxis of STD (a fashion popular among promiscuous individuals). Moreover, permanent use of broad spectrum antibiotics is prescribed to many HIV- seropositive individuals and to most AIDS-patients - as an apparent prophylaxis against opportunistic infections. Antibiotics cause antigen overload indirectly. These drugs suppress the friendly bacterial flora of the gut. These flora are of vital importance for digestion of food as well for stifling various opportunistic pathogenic microorganisms. Suppression of the friendly flora of the gut by antibiotics results in serious problems with digestion of food and in development of opportunistic intestinal infections [14]. Such intestinal abnormalities frequently cause increased permeability of the gut walls

("leaky gut syndromes") [15]. Proteins are essential parts of our diet, and they are very powerful foreign antigens. Why don't they cause antigen overload in healthy individuals? Normally, proteins are digested into short fragments which are not antigens (cannot induce immune response), and only these non-antigen short permeate the gut walls and run into the blood stream. Abnormally high permeability of the gut walls makes it possible for molecules of proteins themselves to run into the blood stream and to become a powerful factor of antigen overload. This mechanism provides a plausible explanation for the epidemiological association between promiscuity, diagnosis of AIDS, and HIV-seropositivity, mediated by a common factor - antibiotic abuse. It also explains the unusually high rate of HIV-seropositivity in Africa, where intestinal infections are rampant.

Besides the role of foreign antigens, there is another plausible hypothesis advanced in [11]: it holds that both typical signs of AIDS - hyperactivation of B-cells and T4-lymphocytopenia - are intrinsic features of the classical stress-syndrome. Stress-syndrome is a highly non-specific reaction of organism to various adverse factors: diseases, intoxication, psychological trauma, etc. This reaction is mediated by elevated concentrations of corticosteroids ("stress-hormones"), and it may be induced by direct injection of corticosteroids. (Incidentally, corticosteroids are frequently used to treat inflammatory conditions in AIDS-patients.) If to combine this hypothesis with the aforementioned one (that hyperactivation of B-cells results in increased affinity of non-specific immunoglobulins), it provides a coherent explanation of why people with severe opportunistic infections (named AIDS) usually demonstrate T4-lymphocytopenia and HIV-seropositivity.

HIV and mortality in Africa: another look at the facts

In a study conducted in a rural population of Uganda (Lancet 1994, 343, 1021-23), about 10,000 individuals were tested for "HIV-antibodies". In those aged 13-44 years, 9.6% were found HIV- seropositive. During follow up, mortality was estimated as 96/1000 man-years (=96 man out of 1000 in 1 year) in the HIV-positive group, and as only 1.4/1000 m.-y. in the HIV-negative group. Thus, mortality in HIV-seropositives was 60 times higher than in their HIV-negative counterparts. At first sight, these results seem to be a reliable proof for causal role of HIV in AIDS, specificity of HIV-tests, and reality of HIV. For this reasons these results are frequently referred to by proponents of the HIV-AIDS theory (see for example [12,13]).

Let us look at the data from another standpoint, and consider an alternative hypothesis - that a positive "HIV-test" is only a non-specific marker of various infectious diseases. Such a "marker-effect" is likely to be caused by the elevated concentrations of non-specific immunoglobulins and increase in their non-specific affinity associated with infectious diseases, especially with multiple infections and intestinal ones (see section 4). Such diseases are known to be the main cause of mortality in this region, at least in relatively young ages. If HIV-seropositivity is actually only a marker of these diseases, most sick individuals in this population have to fall into the HIV- seropositive group. Hence, mortality in HIV-seronegative group has to be much lower than the usual mortality rate in this region. On the other hand, if HIV-tests actually detect a new pathogen (HIV) and HIV causes additional mortality (as the mainstream AIDS science holds), mortality in the HIV-negative group should not decrease in course of the HIV-epidemic. Thus, comparison of mortality in the HIV-negative group with its usual rate, observed before the hypothetical HIV-epidemic, can easily discriminate between the two hypotheses.

What is the usual mortality in this population? Even though any reliable information about mortality in Uganda is absent, it is well known that a great proportion of population there usually die from various diseases in relatively young ages, and it was the case far before the hypothetical HIV-epidemic. Is mortality rate 1.4/1000 in HIV-negative group compatible with such information? If to compute the proportion of deaths in 30-year period (say, since

age 13 to 44) from such an annual rate of mortality, it gives only 4 percent of deaths in 30 years. It is not compatible with the above information that a great proportion of Africans die and died young. Thus, mortality in the HIV-negative group (1.4/1000 m-y) is significantly lower than the usual mortality in this region. In other words, HIV-test is capable to select a great proportion of the individuals who were to die even without the hypothetical "HIV-epidemic". It is in perfect agreement with the "HIV-is-only-a-marker" hypotheses.

Proponents of the HIV-AIDS theory [12] insist that the 9.3/1000 m-y mortality in the 13-44 age group at large is significantly higher than in other parts of the country, and this cannot be explained by causes other than HIV. Such argumentation is flawed - simply because the region was selected for this study just because mortality was higher than in other regions of Uganda. There are many other possible causes for the increase in morbidity and mortality from infectious diseases in some areas of an African country. Irrespective to the actual causes of this increase, both hypotheses predict much higher mortality in the HIV-seropositive group than in the HIV- negative one, and this difference cannot discriminate between the two hypotheses. Nevertheless, mainstream AIDS-scientists try to present this difference as a proof of the official HIV-causes-AIDS hypothesis. For some reasons, they also "forget" to pay attention to the mortality rate in the HIV-negative group, which is obviously lower than the usual rate in this region - what is in perfect agreement with the"HIV-is-only-a-marker" hypothesis and contradicts to the "HIV-causes-AIDS" one.

Other explanations for correlation between HIV-seropositivity and AIDS

The central goal of this summary is to show that there are no proofs that a positive test for HIV-antibodies is caused by any specific antibodies, and that non-specific immunoglobulins is highly likely to be the actual cause of HIV-seropositivity. Let us imagine that it will be shown experimentally that HIV-seropositivity is caused by specific antibodies - that is, blood serum from HIV-seropositive individuals reacts (in significantly higher titers than serum from healthy controls) only with the "HIV-proteins", but not with any other antigens. Surely, this would refute the hypothesis described here. Will such refutation be a proof that HIV-seropositivity is actually caused by HIV?

The main problem with the official HIV-causes-AIDS theory is that it has never been shown that "HIV-proteins" are related to a virus. The only thing known for sure is that these proteins are produced by human lymphocytes in certain conditions *in vitro* (in a test tube). To the same extent, they may be produced by the lymphocytes *in vivo* (in living organism). The "HIV-proteins" are likely to be of cellular (endogenous) nature [1] - that is, to be encoded in human genome by normally inactive genes ("inactive gene" means that the protein encoded by this gene is not produced by the cell). It is well known that only a small proportion of genes in human genome are active, and different sets of genes are active in different types of cells (even though genome is the same for all cells of a given organism). Even in cells of the same type some genes can be switched on (or off) if conditions change. The mechanisms which switch genes on and off are poorly understood in modern biology. If in some abnormal conditions lymphocytes start to produce the "HIV-proteins" (the corresponding genes are switched on), these proteins will be "foreign" for the immune system and specific antibodies will be produced against these proteins. Naturally, blood serum of such individuals will yield specific reaction to the "HIV-proteins". In other words, specific antibodies against "HIV-proteins" may be only a marker that corresponding genes have switched on due to some metabolic changes. Thus, existence of HIV cannot be proved by immunochemical and serological methods - irrespective to whether HIV-seropositivity is caused by non-specific immunoglobulins or by specific antibodies.

Even if to prove rigorously that HIV exists as an exogenous transmissible agent (this has never been done [1-4]) and that HIV-seropositivity is caused by HIV-infection, it still cannot

prove that HIV is the cause of AIDS. Correlation between HIV-infection and AIDS-defining diseases is equally explainable by the "HIV-is-a-marker" hypotheses, which hold that HIV is a benign passenger virus and can easily infect only individuals with some deviations from normal health status, thus, being only a marker of such deviations. In this case, morbidity and mortality may be much higher in HIV-positive individuals than in their HIV-negative counterparts, but not because the HIV-infection. Critical reevaluation of the studies carried out in Africa (see section) demonstrates that the results support "HIV-is-only-a-marker" hypotheses, and contradict to HIV-causes-AIDS one irrespective to the actual causes of HIV-seropositivity.

Conclusions

Specificity of antibodies cannot been established in serological studies with one antigen: it is necessary to prove that titers of the antigen-antibody reaction are significantly higher (than in healthy controls) only for this antigen, but not in respect to other antigens. As far as such multi- antigen serological studies have never been carried out, increase in non-specific affinity of immunoglobulins is the most likely cause of HIV-seropositivity. The problem is exacerbated by the use of "sandwich" tests (like ELISA and WB) - because they are abnormally sensitive to any increase in affinity of non-specific immunoglobulins. Besides the non-specific immunoglobulins, there are several other mechanisms which can lead to correlation between HIV-seropositivity and diseases, while not being compatible with the official "HIV-causes-AIDS" hypothesis. The main flaw of the mainstream AIDS-science is that no experimental or epidemiological studies have been designed and carried out to rule out such alternative explanations. **There are no reasons to consider HIV-seropositivity as a reliable marker of a deadly disease. The main danger of HIV-seropositivity is of iatrogenic (caused by medicine) nature: such individuals are at high risk of administering abnormally severe and prolonged "prophylactic" treatment (antibiotics, antiretrovirals)**. This iatrogenic effect is a direct consequence of the uncritical acceptance of the HIV-causes-AIDS theory.

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ERRORS AND FAILINGS IN PREVENTION AND TREATMENT OF AIDS

by Alfred Hässig, Bern Switzerland

The AIDS epidemic originated 1980 from Michael Gottlieb's report of immunodeficiency four homosexuals suffered from, all of them affected by Pneumocystis carinii pneumonia (PCP) and Kaposi's Sarcoma (KS). The Centres for Disease Control (CDC), to where the cases had been reported, promptly speculated about a virus causing the disease. Due to this presumption as well as for lack of an immunological analysis of the immunodeficient state of these patients the outbreak of a pandemic was proclaimed. It has never taken place. AIDS stayed restricted within the risk groups of a fragment of male homosexuals, drug addicts and hemophiliacs.

The second error was the assumption that the activation of the reverse transcriptase only occurs at the transcription of the genetic structure of retroviruses from RNA to DNA which then multiply in the process of DNA-RNA-protein transcription. It has been known since 1985 that the reverse transcription from RNA to DNA occurs in any eukaryotic cell in order to repair chromosomal damage. The assumption failed that immunodeficiency in AIDS be based on an activation of retroviruses.

The third error concerns the specificity of anti-HIV-antibodies. In infections caused by cell envelope hepatitis B and C viruses, in its immune answer the host develops not only aniviral antibodies but also antibodies against cell components, namely of the cytoskeleton. These autoantibodies react on the glycoprotein gp 41, gp 120 and gp 160. Accordingly, their titer in chronic active hepatitis is strongly raised. The standard anti-HIV test detects high titer autoantibodies against cytoskeletal proteins of the host cell. Low titer antibodies of the same specificity are frequent but not detected by the test. Therefor the anti-HIV test is not at all specific for retroviral antigens.

The major failing in the prevention and therapy of AIDS consists in administering nucleoside analogues such as AZT to suppress the activation of reverse transcriptase. These drugs, originated from cancer research, damage the mitochondria of the cells. The mitochondria energy production in form of ATP drops in all body cells. First, this leads to damage of tissue with highest consumption of oxygen: The skeleton and heart muscle system, the brain, peripheral nerves and the liver. The myo- and neuropathies attributed to retrovirus activity, root in an iatrogenic damage by nucleoside analogues. The ongoing administration of these drugs is the main cause for mortality among AIDS-patients.

Another failing is the establishment of synthetic mono-active protease inhibitors. At long-term administration they cause letal liver damage, kidney stones and serious diabetes activation. The administration of protease inhibitors per se is reasonable. Here, the administration of heparinoides is useful as in chronic inflammation they are able to specifically suppress the cascades of activated serum proteases of the coagulation and the complement system.

Conclusions

We point at successful possibilities of AIDS prevention. AIDS is caused by a persistent stress induced shifting of the neuro-endocrine control of the immune system in a catabolic direction. AIDS is a hypercatabolic disease and is, therefore, to be considered within the group of hypercatabolic diseases such as sepsis, toxic shock syndrome and protein calorie malnutrition. The psychic state of stress in AIDS as well as the toxic and infectious states of

stress are dominant. Together with decreasing these states of stress a sufficient administration of anabolic nutritional supplements such as polyphenols and polyanions is recommended.

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THE MAIN CAUSE OF OPPORTUNISTIC INFECTIONS IN AIDS

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Is the immune system the main protection from opportunistic infections?

The main clinical manifestation of AIDS is severe opportunistic infections (OI) with abnormally high mortality. These diseases are of endogenous nature: the OI-causing pathogens are present in healthy organism too, though only in small quantities. Why OI develop? A natural answer is that some factor X in healthy organism stifles opportunistic pathogens, and only if it fails, these ever-present pathogens proliferate and cause OI. What plays the role of this factor X? This question central to understand the causal mechanisms of AIDS and of other forms of susceptibility to OI. It is widely accepted at present that just the immune system normally suppresses opportunistic pathogens, and just immunodeficiency causes development of OI. Moreover, this notion seems to be "self-evident": if not the immune system, what else can protect from opportunistic pathogens?

The immune system is responsible for "acquired immunity" or "specific immunity", not for immunity in general. Specific immunity develop as reaction of the immune system to a certain pathogen, and it is directed only against this pathogen. The technical meaning of the term "immunodeficiency" is nothing but "deviation of some parameters of the immune system from the normal state", not a lack of immunity (resistance to pathogens) itself as many „non-specialists" believe. Immunodeficiency does not necessarily lead to decrease in immunity, and decrease in immunity is not necessarily caused by immunodeficiency. Besides the specific immunity, there are several forms of non-specific immunity, maintained by mechanisms not related to the immune system [1]. Failure of such mechanisms, technically not being "immunodeficiency", can result in increased susceptibility to pathogens (decreased immunity). The central idea in this summary is that the protection from OI is carried out by these non-specific mechanisms, not by the immune system. The failure of these mechanisms and not the failure of the immune system (immunodeficiency), is the actual cause of OI.

If other mechanisms of protection from pathogens, not related to the immune system, are well known [1], why is the immune system thought to be the main protection from opportunistic pathogens? Besides the "self-evidence", this belief rests on strong correlation between T-immunodeficiency and OI: many patients suffering from OI have immunodeficiency too, and this cannot be explained by random coincidence. But correlation cannot prove causation - simply because it may be caused by causal links other than "immunodeficiency causes OI": 1) OI cause T-immunodeficiency; 2) both OI and T-immunodeficiency are caused by a third factor; 3) correlation is overestimated because observations are biased. The main aim of the analytical study summarized below is to look at the known facts critically from such a vantage point, and to try to find alternative explanations to the observed correlation between T-immunodeficiency and OI. Oddly enough, after critical reevaluation, the current "immunodeficiency-causes-OI" postulate occurs to have neither experimental nor theoretical underpinning. Moreover, it even contradicts to tenets of immunology. An alternative view - that OI result from suppression of non-specific mechanisms of resistance - is capable to provide a coherent explanation to the whole body of facts known about OI, immunodeficiency, as well as about the pathogenesis and epidemiology of AIDS. Causal mechanisms of AIDS are well explainable at the level of knowledge which had been achieved by the middle of 1970s, or even middle 1960s, and need no any doubtful hypotheses about "deadly retroviruses".

Mechanisms of non-specific immunity to opportunistic pathogens

Human organism is permanently exposed to a great many of species of microorganisms which are present in environment. Nevertheless, most of these species cannot cause a disease because the whole set of physical and chemical conditions in organism is normally not suitable for their reproduction [1]. Such a set of conditions is determined genetically. Thisis perhaps the most important (but less noticeable and poorly understood) factor of resistance. This form of resistance is frequently named "innate non-specific immunity" (and by many other terms [1]) and lies beyond the scope of the current immunology. Naturally, if metabolism of the host deviates radically from the normal state (e.g. in course of a disease, intoxication, stress, etc.) such a change can made conditions suitable for some ever-present opportunistic pathogens, and OI will develop. Even though such mechanisms of innate immunity protect organism from a great many of species, this protection is far from being universal. Many areas of the organism, especially that connected to environment (skin, mucous membranes, gut, etc.) are still suitable ecological niches for thousands of species of microorganisms, including many potentially pathogenic ones. Without some additional protection infections would be inevitable. Nature has found an elegant way to overcome this vitally important problem: competition between various species had been effectively harnessed [2]. Since the first hours and days of life of the host, these ecological niches are colonized and, then, permanently populated by many species of microorganisms. The system of these species is named "resident microflora" or "normal microflora". It is of vital importance to the host that only non-pathogenic species normally dominate in its microflora and keep potentially pathogenic ones (opportunistic pathogens) at low and safe quantities. Thus, these non-pathogenic microflora protect the host from ever-present opportunistic pathogens [2,3]. Such a symbiosis rests on the ability of the host to maintain conditions which guarantee selective advantage for non- pathogenic flora. This ability of the host is determined genetically and emerged in course of long- term natural selection.

What and how causes opportunistic infections

There are two types of opportunistic pathogens: the ones which are normally dormant because conditions in the host are not suitable for their reproduction, independently on presence of other species (type I), and the pathogens which are capable to replicate in conditions of healthy organism, but are normally suppressed by other species of micro-organism -- by non-pathogenic resident flora (type II). Hence, the causes of opportunistic infections may be also subdivided into two groups: radical changes in metabolism of the host, and some factors which suppress the non-pathogenic resident micro-flora - antagonists of the type II opportunistic pathogens.

Which factors are known to be capable to suppress non-pathogenic resident flora? At least three groups of such external factors are well known: 1) antibiotics, including bacteriostatic drugs (e.g. sulphonamids) and other antibacterial drugs; 2) drugs with cytotoxic effect (e.g. drugs used for anti-cancer chemotherapy, for immunosuppression) ; 3) radiation [2-5]. The most frequent causes of the OI observed in clinical practice are antibiotics, especially broad spectrum ones [5-8]. **Non-pathogenic micro-flora, which normally dominate and overgrow pathogenic species, are more sensitive to these drugs than some opportunistic pathogens (e.g. fungi). Therefore, antibiotics create selective advantage for these pathogens by suppression of their usual antagonists-non-pathogenic bacterial flora** [1, 5-8]. If two species are antagonists, even a small reproductive advantage of one of them, after a few tens of generations inevitably leads to complete dominance of this species. Thus, **even a small influence may result in radical changes in balance of microflora**. Such an influence should not necessarily increase reproductive ability of one

species and decrease that of another. It is enough, for example, to stifle both species, but to a different extent, and the less stifled species overgrows another. Surely, in reality such interrelations are much more complex because not two but thousands of species are involved in this process. But the main feature of this ecological system remains the same -- external factors may lead to radical changes in proportions of various species in microflora [5].

For the same reasons, along with the external factors affecting microflora directly, there are indirect mechanisms leading to changes in the balance of normal microflora. As far as microflora are essentially dependent on the host's metabolism (they obtain many nutrients and growth factors from the host), significant changes in the metabolism of the host (especially in catabolic direction) may lead to radical changes in the balance of microflora. If these changes are advantageous for at least one opportunistic pathogen, this species will proliferate and cause OI. Such metabolic changes can also cause OI through violation of the "innate resistance" determined by peculiarities of the normal metabolism of the host. Opportunistic viruses are likely to be activated by such metabolic changes: viral reproductive cycle essentially depends on cellular metabolism. Thus two groups of factors may cause OI: suppression of the resident non-pathogenic flora by external factors (see above), and the factors capable to cause radical changes in metabolism.

The most frequent cause of such metabolic changes in catabolic direction is stress-reaction. Such reaction is highly non-specific -- it can be caused by a wide range of adverse factors: any severe disease, intoxication, psycho-trauma, protein-calorie malnutrition. It is known that stress-syndrome is mediated by elevated concentrations of corticosteroids ("stress-hormones"). Besides the stress-factors themselves, most features of the stress reaction may be induced by direct injection of corticosteroids. These hormones and their synthetic analogues are used in medicine to cope with acute inflammations (and they are frequently used to treat AIDS-patients).

Why opportunistic infections correlate with T-immunodeficiency

The T-immunodeficiency is well known to be an essential feature of the stress-syndrome. In the middle of 1970s it was also shown experimentally that just T4-lymphocytes (a subtype of T- lymphocytes) are most sensitive to stress-hormones corticosteroids. Hence, T-immunodeficiency at all and T4-immunodeficiency in particular are highly non-specific symptoms -- they appear almost always when stress-reaction develop. It is natural that this forms of immunodeficiency are present in almost any severe enough disease or other abnormal states of organism. Mainstream AIDS-science tries to ignore these reliably established facts -- mainly because the T4-immunodeficiency is promulgated as a specific feature of AIDS (see [13,14] for details).

There are several explanation for the correlation between OI and T-immunodeficiency. First, severe OI (e.g. induced by antibiotics) cause stress-syndrome. Hence, T-immunodeficiency has to be expected in such cases -- as a consequence, not the cause of these OI. Second, both OI and T- immunodeficiency have common causal factors: cytostatic and cytotoxic drugs, and radiation. In individuals exposed to such factors, for example, in patients receiving anti-cancer or immunosuppressive therapy, OI can develop due to suppression of the non-pathogenic resident flora by these factors. Third, radical changes of metabolism in the catabolic reaction (e.g. caused by any form of stress, or by protein-calorie malnutrition) suppress both normal non-pathogenic flora (this causes OI) and the immune system (this causes T-immunodeficiency). Such a mechanism explains why people with severe and long-lasting diseases frequently suffer from OI as well as have signs of T-immunodeficiency. Administration of corticosteroids, even though these immunosuppressive drugs don't suppress resident microflora directly, depletes microflora indirectly - through the

catabolic changes in metabolism these drugs inevitably induce. This explains why OI frequently develop in course of such therapy by corticosteroids. Thus, the strong correlation between OI and T-cell immunodeficiency is easily explainable by mechanisms other than "immunodeficiency-causes-OI". The correlation between OI and T-immunodeficiency is likely to be overestimated due to observation bias. To establish the fact of T-immunodeficiency it is necessary to carry out some laboratory tests. Such tests are normally used only if physicians already suspect immunosuppression in this patient. As far as "immunosuppression-causes-OI" postulate is believed to be self-evident, mainly some signs of OI made physicians suspect immunosuppression and force them to carry out tests for immunodeficiency. If immunodeficiency is found, this finding is perceived as confirmation of the causal role of immunodeficiency in development of OI. **Naturally, most cases of T-immunodeficiency, not associated with OI, remain unnoticed**; and correlation between OI and T-immunodeficiency seems apparently high.

Belief in "immunodeficiency-causes-OI" as the cause of iatrogenic OI

Perhaps the most frequent cause of OI in developed countries is of iatrogenic (caused by medicine) nature. As far as immunodeficiency is thought to be the main cause of severe OI, almost any patient with severe immunodeficiency (or presumably high-risk of immunodeficiency) is put on permanent prophylaxis by broad-spectrum antibiotics. Such a prophylactic measure is thought to be capable to reduce risk of OI. Nevertheless, **such long-term intake of broad-spectrum antibiotics is known to be the main cause of iatrogenic OI**, and it is known that antibiotics induce OI because suppression of normal microflora [3, 6-8], not of the immune system. It is not an easy task to understand what has caused OI in such cases -- immunodeficiency or antibiotics. Interpretation of the facts depends mainly on preconceived ideas. Even if OI have been caused by antibiotics, immunodeficiency is usually accepted as the "actual" cause.

Thus, the uncritically accepted "imunodeficiency-causes-OI" postulate is capable to increase dramatically the number of cases of OI through inadequate administration of broad spectrum antibiotics to some categories of patients. Naturally, this creates actual correlation between OI and immunodeficiency. Oddly enough, the same belief conceals the real causes of these iatrogenic OI: these diseases are being explained by immunodeficiency and the correlation is perceived as an additional proof for the "immunodeficency- causes-OI" dogma. Such an iatrogenic effect essentially depends on subjective estimates of the risk of OI shared by physicians. If the risk is believed to be unusually high (e.g. in AIDS patients or HIV-positives), very severe regimen of broad-spectrum antibiotics is being chosen. Naturally, probability and severity of OI is in direct relation with intensity and longevity of such "prophylaxis". In any other situation, doctors would avoid such intensive and prolonged use of antibiotics, but the belief in high risk of severe OI, forces them to leave usual precautions. It is hardly surprising that severe OI will finally develop in these patients (due to destruction of their normal microflora by antibiotics) as well as T- immunodeficiency (as stress-reaction to the OI themselves, intoxication, and psychological trauma).

Can the immune system stifle opportunistic pathogens?

If to accept that T-immunodeficiency is the main cause of OI, one should also admit that just the immune system plays the leading role in permanent suppression of opportunistic pathogens in healthy organism. How does the immune system carry out such protective functions? Any clear-cut explanations are conspicuously absent in medical literature. If to try to answer this "naive" question, serious inconsistencies immediately appear. Some of them are summarized below.

1) Opportunistic pathogens are not able to induce any noticeable acquired immunity even in experiments [4]. How can the immune system protect from OI if it works mainly through mechanisms of acquired immunity? 2) It is completely unexplainable how the immune system can discriminate between pathogenic and non-pathogenic species and to suppress just the pathogenic ones: both types are presented in microflora by thousands species, and both types are foreign for the immune system. 3) Opportunistic infections begin to develop in areas not easily accessible by the immune system, at least in healthy organism. How can the immune system be the factor which normally suppress opportunistic pathogens in these areas? 4) Since the first minutes of life organism is being colonized by microorganisms, and just non-pathogenic species normally dominate in this process. This means the pathogens are stifled by some factor since the very birth. How can the immune system be this factor if development of specific immune reactions need some time (at least a few tens of hours)? Moreover, the immune system is dormant in newborns. 5) Cytotoxic T-cells, a central mechanism of the cell-mediated acquired immunity, cannot stifle opportunistic pathogens - simply because these immune cells can attack only the cells with the same genetic makeup, having the same MHC-antigens as the host [1], but microorganisms don't carry such antigens. 6) A great number of potentially pathogenic species ever-present in microflora poses one more problem: **it has never been shown experimentally that specific immune reactions can be activated in respect to hundreds of foreign antigens simultaneously.**

How epidemic of AIDS became possible

The epidemic of severe iatrogenic OI, misleadingly named AIDS, was triggered by a "fashion" of long-term use of broad-spectrum antibiotics as permanent prophylaxis of sexually transmissible diseases (STD). This fashion became popular in late 1970s in most promiscuous (having many sexual partners) gays as well is in some groups of heterosexual population with specific lifestyle marked by high level of promiscuity, heavy abuse of recreational drugs, etc. Gradual destruction of the resident flora by antibiotics resulted in development of OI, as well as in long-term diarrhea, maladsorption, weight loss -- classical symptoms of antibiotic abuse [3-7]. In cases of severe enough OI T-immunodeficiency developed as a natural stress-reaction to the opportunistic disease. These cases of severe OI in gays were spotted by the CDC in 1979-1981 and erroneously explained by acquired T-immunodeficiency as the primary biological cause. Association of these cases with homosexuality and promiscuity was interpreted as a sign of infectious nature of these disease. Nobody doubted whether T-immunodeficiency was actually the cause of these severe OI. All efforts were focused at finding some external factors causing the immunodeficiency, not at the causes of OI themselves. In 1981, cases of "unusual infections" (in reality caused by antibiotics), if observed in gays, were announced as first signs of new and deadly immunodeficiency [10]. Intensive permanent prophylaxis by broad-spectrum antibiotics was prescribed to such individuals. Thus, **instead of a cure, they received just the disease-causing factor; this made their OI recurrent, severe, and seemingly incurable**. In 1980s, the mainstream science promulgated highly non-specific symptoms as signs of AIDS (lymphadenopathy, a positive test for HIV- antibodies). This expanded dramatically the range of victims of the dangerous practice to prescribe permanent use of broad-spectrum antibiotics. Naturally the number of cases of severe OI was rapidly increasing in 1980s (and this seemingly supported the official "infectious" hypothesis). Besides the wrong deciphering this syndrome as immunodeficiency, the „HIV=AIDS=death" belief was also an important causal factor of the iatrogenic epidemic. **This belief forced physicians to use abnormally severe regimen of antibiotics** (see section). In 1990s, this fear gradually alleviated, and this resulted in noticeable decrease in new cases of AIDS in the middle of 1990s.

Which hypothesis explains facts on AIDS better?

The hypothesis on causes of OI described in sections 2-6, if applied to AIDS, is capable to explain basic features of pathogenesis and epidemiology of AIDS. But the officially accepted HIV-AIDS hypothesis also provides its own explanations. Below, some arguments are summarized which could help to decide which hypothesis is better compatible with facts.

1) Abnormally long and intensive use of broad-spectrum antibiotics is a factor common to all AIDS patients and many HIV-positives, irrespective to the group of population they belong too. On the other hand, such regimen of antibiotics is extremely rare in other categories of patients and in population at large.

2) In many cases of AIDS, severe OI are observed earlier than any noticeable T4-immunodeficiency. As for mild OI, such as thrush, these infections are observed earlier than immunodeficiency in most cases of AIDS. If the immunodeficiency is actually the cause of OI, how can the consequence precede the cause?

3) Such typical symptoms of AIDS as diarrhea, maladsorption, weight loss are also typical to suppression of the friendly microflora of the gut. This suppression is a natural effect of antibiotics. It also may be caused by substances with cytostatic effect (AZT is an example of such a substance). As for the HIV-AIDS hypothesis, no any coherent explanations were provided how HIV can cause these symptoms.

4) If immunodeficiency is actually the main problem in AIDS, why do the patients suffer mainly from opportunistic (endogenous) but not from usual (exogenous) infections?

5) The official HIV=AIDS hypothesis fails to explain why male homosexuals are the main victims of AIDS. The mainstream science tries to explain this essential feature of AIDS by radical difference in probability of transmission of HIV during homo- and heterosexual contacts. Even if to assume that HIV is transmissible, the difference is maximum 2-fold [11]. Anal sex is quite popular in heterosexual pairs, and heterosexual population is many times larger than homosexual. Nevertheless, AIDS is not associated with heterosexual anal sex [15].

6) The HIV-causes-AIDS theory cannot explain why AIDS is highly associated with promiscuity. Probability of transmission of HIV is estimated as 1-2 in 1,000 contacts. With such a low rate of transmission in one intercourse, spread of infection should not depend on promiscuity. For example, if one infected person changes sexual partners after each intercourse (maximal level of promiscuity), another infected person - only after 100-200 intercourses (low level of promiscuity), the average number of partners infected after a great number of contacts will be almost the same for the two infected individuals [12]. On the other hand, promiscuity is directly associated with antibiotics (treatment and/or permanent prophylaxis of STDs).

7) The official hypothesis fails to explain why AIDS is distributed very unevenly over the globe. For example AIDS (not HIV-seropositivity) is extremely rare on the territory of the former Soviet Union in spite of higher incidence of STD and about the same number of male homosexuals as in the USA. It is essential that long-term prophylactic use of antibiotics is very unpopular among physicians in the former Soviet Union.

8) Drug-resistant strains of microorganisms are frequently detected in AIDS patients, and just this drug- resistance is partially responsible for the frequent lethal outcome of opportunistic infection incurable by antibiotics. There is no visible link between immunodeficiency and drug- resistance of the microorganisms. On the other hand, drug-

resistance is a natural consequence of antibiotic abuse -- it results from natural selection of drug-resistant mutants under the selective pressure of antibiotcs [3]

Conclusions

The ever-present opportunistic pathogens are normally stifled by mechanisms of non-specific resistance -- mainly by non-pathogenic microflora, not by the immune system. Failure of these mechanisms, for example suppression of the resident flora by antibiotics, is the main cause of opportunistic infections. The widely shared postulate, that T-immunodeficiency causes opportunistic infections, has neither theoretical nor factual underpinning. T-immunodeficiency is well known to be a secondary phenomenon and a typical symptom of catabolic stress. Epidemic of AIDS is of purely iatrogenic nature - its main cause is antibiotic abuse. The epidemic resulted from wrong interpretation of the syndrome as acquired T4-immunodeficiency in early 1980s. Non- specific symptoms were announced as first signs of these disease, and individuals with such symptoms were put on permanent prophylactic use of broad spectrum antibiotics. All the symptoms of AIDS, including severe opportunistic infections, are caused by this abuse of antibiotics.

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THE FIRST NINE GAY MALE AIDS CASES IN SAN FRANCISCO

(Edited summary of chapter VII, page 411 - 415, of the dissertation „The Social Construction of Knowledge(s) on HIV and AIDS" by Dr. Michelle Cochrane)

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At this point, I would like to take a moment to summarise what I have documented regarding the first nine homosexual/bisexual men that were captured and reported with AIDS in San Francisco as of July 1981.

Although San Francisco had the largest population of gay men (on a per cabita basis) in the entire United States, and despite extensive surveillance of this population by the SFDPH prior to AIDS (as a result of the Hepatitis B vaccine trails and efforts to curtail „gay bowel" syndrome), **no emergent health problems were noted among these men until the CDC catalysed surveillance efforts in the city in the summer of 1981**. Immediately subsequent to this alert, via contacts with physicians and dermatologists and a review of death certificates in the city, the CDC and the SFDPH were able to discover evidence of exactly nine homosexual/bisexual men in the previous year who had developed clinical symptoms or died of conditions suggestive of AIDS. [e.g. Karbosi's sarcoma or Pneumocystis carinii pneumonia – these were the only two opportunistic infections seen in SF in the first several years].

Following my review of documentation on these cases I have concluded that the health department's records on these patients, and the subsequent characterisation of these patients in the press and in popular narratives regarding the epidemic, is flawed in several respects. Premier among these misrepresentations was the inaccurate reporting of risk factors for the disease; for example, although three of the nine (one-third of the initial cohort) were intravenous drug users, **none of them were initially reported with that risk.** It took five years for the SFDPH to properly reclassify Case No. 0004 as a GIVDU; the other two gay IV drug users were captured as a result of my review of their medical charts. This finding is consistent with my argument throughout this text, **that drug use (and all other HIV/AIDS risks, e.g. transfusion, hemophilia etc) among gay male AIDS cases has always been, and continues to be, significantly under-reported.**

My second point is that census tract data used by the SFDPH for reporting these patients **was inaccurate for five of these men (55% of the total), and that it demonstrated a consistent bias towards over-emphasising the „gayness" of this disease.** For example, when an AIDS case had no address, had an out-dated address, or had several places of residence reported (even several cities of residence), then the SFDPH captured the case as a San Francisco resident (thus increasing the per-capita incidence of the disease) and assigned a census tract with correlated to a gay bathhouse or a predominantly gay neighbourhood in the city. This finding suggests that there are profound problems with the integrity of geographical analysis of AIDS in San Francisco as published by the Health Department and used methodologically, in subsequent epidemiological studies in the cities.

As a third point, my review of these cases indicates that the socio-economic status of the majority of these men was very tenuous, a conclusion which is contrary to the popular characterisation of early AIDS patients. For instance, two of these earliest patients were homeless with no means of support (case No. 1005 and No. 1008), a third (case No. 1004) had no known occupation. Four other patients were employed in low-end administrative or service positions (case No. 1001, No. 1003) or had only vague occupational information reported; one „retired", one disabled paralegic (case No. 1006, No. 1009). In summary, that

leaves just two men who were presumably middle-class individuals with a guaranteed source of income (No. 1002, No. 1007 death certificate review for the latter). My conclusion regarding the generally low socio-economic status of these cases is also borne out of the fact that apparently only one of these nine men (No. 1007 – death certificate) had private health-care in the city, and **of this patient the patient the least information is available as why he was as captured as an AIDS case following his death**.

My retrospective analysis of the material evidence regarding these early AIDS cases in San Francisco is barely consonant with the oft-quoted characterisations of these patients in various popular publications. According to Shilts, (Randy Shilt's *And The Band Played On),* AIDS first emerged among moderately „Guppies" who summered on Fire Island and lived the high and fast night-life in New York City and San Francisco. They were politically well-connected individuals who were often engaged in long-term relationship and enjoyed access to social and medical support systems. In Shilts' estimation, it was only as the epidemic evolved out of this core group of moderately affluent gay men, that AIDS then began to appear in the „corridors of poverty" associated with marginalized populations in urban centres on the East Coast. To the contrary, **I am suggesting that the epidemic began, and to a large extent remains to this day, overwhelmingly (though not exclusively) concentrate among impoverished or disenfranchised inner-city populations**; a population with does not *ipso facto* exclude homosexual/bisexual men.

But as I mentioned, Shilts is not alone in his characterisation of the privileged socio-economic status of the early gay male AIDS cases. In a book chronicling the daring history of the Centers for Disease Control, Elizabeth Ethridge reviewed the CDC's initial case control study of AIDS-patients in the late summer of 1981. She described the various epidemiological investigations launched by the CDC investigators throughout the country and gave an account of a trek to San Francisco by one of the key members of the KS/OI Task Force, Dr. Harold Jaffe. Jaffe's comments regarding early AIDS patients were as follows:

„We were struck by how sick these men really were. Many were obviously dying and were just wasting away ... Secondly, we were struck that these men did lead a particular kind of life style. These were not gay men who were in long-term monogamous relationships. These were highly sexually active gay men. Often they were well-to-do, had good jobs, travelled a lot. They tended to have sexual partners in many parts of the country, often anonymously ... They tended to use a lot of drugs along with this ... (in the) fast track life style."

Although my own research, as well as that published in early articles on AIDS patients, supports Jaffe's characterisation of the heavy recreational (and intravenous) drug use among early cases, his socio-economic characterisation applies to just two of the nine gay men that I have discussed. Of course, it is possible that my case study is significantly biased and that San Francisco AIDS patients are atypical in the regard; but then again, one cannot reach such a conclusion on the basis of Jaffe's own contemporaneous (1982 and 1983) epidemiological research and academic publications on early AIDS patients.

In 1983, when the CDC got around to publishing an analysis of their „National Case-Control Study of *Karposi's Sarcoma* and *Pneumocystis carinii Pneumonia* in Homosexual Men" Jaffe et al summarised their research of the various ways in which AIDS cases differed from matched homosexual controls. The authors concluded that AIDS cases were more likely to have had a greater lifetime consumption of specific drugs, greater numbers of sexual partners, and more frequent exposure to several sexually transmitted infections.

But what is significant for my argument above, is that the authors also noted that among all of the men in the study (both cases and controls), only one-third had an „income over $ 20'000 in (the) past year". So, although this data cannot be used to argue that AIDS was *likely* to be associated with poverty, **it certainly does not support an argument that homosexual men in general, or AIDS cases in particular, were „well to-do" (and) had good jobs".** In fact, the opposite was true, as the majority of these men, who resided in two of the most expensive cities in the US (New York City and San Francisco), were barely getting by. And because the CDC's case-control income data was never disaggregated, nor presented with a high/low range for each category, it is impossible to determine whether or not the 64% of the AIDS cases that earned less than $ 20'000/year had significantly lower incomes than the 63% of the controls who earned under $ 20'000/year. Provisionally, this would be my argument, given that my review of the first nine cases reported in San Francisco showed that a third of these men were homeless and unemployed. But again, the political-economy of this disease was never a concern at the CDC; instead their explicit focus was on the „fast track" promiscuous gay „life style".

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AIDS, UNDERDEVELOPMENT AND SEXUAL STEREOTYPES: RETHINKING AIDS IN AFRICA

by Charles L. Geshekter, California, USA

Defining AIDS in Africa

History will show that AIDS became one of the leading bio-medical research controversies in the late 20th century. Because Africa plays a major role in the dire predictions about increased AIDS incidence, it is important to distinguish between a virus (HIV) and a syndrome (AIDS) to understand how knowledge about AIDS is created and disseminated. Before international donors pour more money into African AIDS research, or conduct another knowledge-attitude-practice survey, or advocate modifying traditional sexual behavior, they should subject the basic suppositions about AIDS cases in Africa to the same standards of consistency, testability, and parsimony used in the empirical sciences.

"African AIDS" refers to a cluster of clinical symptoms (weight loss, chronic diarrhea, fever, and persistent coughs), none of which are new or uncommon on the African continent. Since 1985, however, these symptoms have been defined as a single "syndrome" and made part of an infectious disease theory that assumed they were caused by a contagious virus (HIV). It was also assumed that this virus could be easily transmitted through sexual contact.

How can a virus cause 29 heterogeneous "AIDS indicator" diseases almost entirely among males in Europe and America but afflict African men and women in equal numbers? The answer is that the World Health Organization uses a definition of AIDS in Africa that differs decisively from the one used in the West. The origins of the former definition are illuminating.

Joseph McCormick and Susan Fisher-Hoch were physicians from the U.S. Centers for Disease Control (CDC) who were instrumental in convening the WHO conference in the Central African Republic in 1985 that produced the "Bangui Definition" of AIDS in Africa. They recently explained the motivation for the conference and the rationale behind the definition that resulted from it:

"We still had an urgent need to begin to estimate the size of the AIDS problem in Africa....But we had a peculiar problem with AIDS. Few AIDS cases in Africa receive any medical care at all. No diagnostic tests, suited to widespread use, yet existed...In the absence of any of these markers [e.g., diagnostic T4/T8 white cell tests], we needed a clinical case definition...a set of guidelines a clinician could follow in order to decide whether a certain person had AIDS or not. [If we] could get everyone at the WHO meeting in Bangui to agree on a single, simple definition of what an AIDS case was in Africa, then, imperfect as the definition might be, we could actually start to count the cases, and we would all be counting roughly the same thing.[emphasis added]

The definition was reached by consensus, based mostly on the delegates' experience in treating AIDS patients. It has proven a useful tool in determining the extent of the AIDS epidemic in Africa, especially in areas where no testing is available. ***Its major components were prolonged fevers (for a month or more), weight loss of 10 percent or greater, and prolonged diarrhea..."***

The doctors wanted to refute the ugly moralism of the 1980s that AIDS was a "gay plague" by convincing the American government that "AIDS was a plague all right, but that no one was immune." McCormick and Fisher-Hoch recalled that:
"experts in STDs continued to regale us with tales of the excessive and often bizarre sexual practices associated with HIV in the West...we were also beginning to see a direct correlation between the number of sexual partners and the rate of infection...Compared to the West, heterosexual contacts in Africa are frequent, and relatively free of social constraints - at least for the men....There was every reason to believe that, having found heterosexually transmitted AIDS in Kinshasa, we were likely to find it everywhere else in the world."

It was upon these grossly unscientific claims, inaccurate clinical generalization, western notions of social amelioration, and 19th century racist stereotypes about Africans that AIDS became a "disease by definition" and Africa was assigned a central role in the premise that AIDS was everywhere and everyone was at risk. By 1986, "people were falling over one another to get involved in AIDS research," recalled the couple. "They realized that AIDS represented an opportunity for grant money, training, and the possibility of professional advancement...A certain bandwagon mentality took hold. Careers and reputations were riding on the outcome." Since they had no proof that AIDS was sexually transmitted, McCormick and Fisher-Hoch relied on a narrow survey conducted by Kevin DeCock, another CDC epidemiologist. DeCock wanted to determine what had happened to the five Africans out of 600 who had been tested for Ebola virus in 1976 in the small town of Yambuku (northern Zaire) and whose serum, when tested retrospectively ten years later, showed antibodies to HIV. According to McCormick and Fisher-Hoch, "three of the five were dead. To determine if their deaths were attributable to AIDS, Kevin interviewed people who had known them. The friends and relatives of the deceased described an illness marked by severe weight loss and other ailments that left little doubt in Kevin's mind that they had succumbed to AIDS." It is not known how many of the 595 HIV negative subjects had died or what was their clinical cause of death.

This kind of presumptive diagnosis known as a "verbal autopsy" is widely accepted in Africa, where "no country has a vital registration system that captures a sufficient number of deaths to provide meaningful death rates." While medically certified information is available for less than 30% of the estimated 51 million deaths that occur each year worldwide, **the Global Burden of Disease Study (GBD) found that sub-Saharan Africa had the greatest uncertainty for the causes of mortality and morbidity since its vital registration figures were the lowest of any region in the world - a microscopic 1.1%.**

These 1997 findings prompted The Lancet to acknowledge editorially that "current strategies to improve the world's health may need to be reassessed" and to ponder "how much more money is spent on research into HIV infection [the 30th cause of death] than into the causes of suicide [#12] or the prevention of road-traffic accidents [#9] and why should this be."

Racism and African Sexuality

The conventional thinking asserts that AIDS in Africa is somehow explicable by Africans' sexual predilections. Such insinuations merit close scrutiny since generalizations about African sexual practices are analytically useless for an internally diversified continent of 650 million people. Nonetheless, at the 10th International AIDS Conference in Yokohama (August 1994), Dr. Yuichi Shiokawa claimed that AIDS would be brought under control only if Africans restrained their sexual cravings. Professor Nathan Clumeck of the Universite Libre in Brussels was skeptical that Africans will ever do so. In an interview with Le Monde,

Clumeck claimed that "sex, love, and disease do not mean the same thing to Africans as they do to West Europeans [because] the notion of guilt doesn't exist in the same way as it does in the Judeo-Christian culture of the West." Such myths about the sexual excesses of Africans are old indeed. Early European travelers returned from the continent with tales of black men performing carnal feats with unbridled athleticism, with black women who were themselves sexually insatiable. These affronts to Victorian sensibilities were cited, alongside tribal conflicts and other "uncivilized" behavior, as justification for colonial social control.

AIDS researchers added new twists to an old repertoire: stories of Zairians who rub monkeys' blood into cuts as an aphrodisiac, of ulcerated genitals, and of philandering East African truck drivers who get AIDS from prostitutes and then go home to infect their wives. **A facetious letter in The Lancet cited a passage from Lili Palmer's memoirs as evidence for how a large male chimpanzee's "anatomically unmistakable signs of its passion for [Johnny] Weismuller" on the Tarzan set in 1946 "may provide an explanation for the inter-species jump" of HIV infection.** No one has ever shown that people in Rwanda, Uganda, Zaire, and Kenya - the so-called "AIDS belt" - are more active sexually than people in Nigeria which has reported only 1148 AIDS cases out of a population of 100 million or Cameroon which reported 3072 cases in 10 million. **No continent-wide sex surveys have ever been carried out in Africa. Nevertheless, conventional researchers perpetuate racist stereotypes about insatiable sexual appetites and carnal exotica.** They assume that AIDS cases in Africa are driven by a sexual promiscuity similar to what produced - in combination with recreational drugs, sexual stimulants, venereal disease, and over-use of antibiotics - the early epidemic of immunological dysfunction among a small sub-culture of gay men in the West. The research from Africa suggests nothing of the sort. In 1991 researchers from Medicins Sans Frontieres and the Harvard School of Public Health did a survey of sexual behavior in Moyo district of northwest Uganda. **Their findings revealed behavior that was not very different from that of the West.** On average, women had their first sex at age 17, men at 19. Eighteen per cent of women and 50% of men reported premarital sex; 1.6% of the women and 4.1% of the men had casual sex in the month preceding the study, while 2% of women and 15% of men did so in the preceding year.

The media misrepresentations that link sexuality to AIDS have spawned inordinate anxieties and moral panics in regions of Africa already afflicted with extreme poverty, ravaged by war, and deprived of primary health care delivery systems. **The "disaster voyeurism" of tabloid journalism enables them to use AIDS to sell "more newspapers than any other disease in history. It is a sensational disease - with its elements of sex, blood and death it has proved irresistible to editors across the world." Public health seems to require salesmanship, not skepticism.** The media's appetite for scary scenarios and its disdain for alternative perspectives enables it to treat Africa in apocalyptic terms. This marketing of anxiety helps to promote behavior modification programs to "save Africa." Disregarding the morbidity and mortality data from the Global Burden of Disease Study, journalists maintain that "AIDS is by far the most serious threat to life in Africa."

Several serious consequences of claiming that millions of Africans are threatened by AIDS make it politically acceptable to use the continent as a laboratory for vaccine trials and the distribution of toxic drugs of disputed effectiveness like ddI and AZT. Campaigns that advocate monogamy or abstinence and ubiquitous media claims that "safe sex" is the only way to avoid AIDS inadvertently discourage Africans from visiting a public health clinic for fear of receiving some "fatal" AIDS diagnosis. And even Africans "with treatable medical conditions (such as tuberculosis) who perceive themselves as having HIV infection fail to seek medical attention because they think that they have an untreatable

disease." Some Western scientists, such as Dr. Luc Montagnier, the French virologist who discovered HIV, claim that the practice of female circumcision facilitates the spread of AIDS. Yet Djibouti, Somalia, Egypt, and Sudan, where female genital mutilation is the most widespread, are among the countries with the lowest incidence of AIDS. AIDS cases in Africa are thought to constitute a "heterosexual paradox" because the sexually equal distribution contrasts sharply with the 12-1 ratio of men to women with AIDS in North America and Western Europe. Most frightening to people of the developed world is what the African example seems to augur for them. Whereas AIDS in the industrialized countries is almost exclusively a disease of a small percentage of homosexuals, intravenous drug users, and recipients of tainted blood transfusions, AIDS in Africa is said to be as general and indiscriminate a killer as such long-time African curses as malaria, schistosomiasis, and sleeping sickness (trypanosomiasis). In Africa, where women contracted "Slim Disease" in numbers roughly equal to males, there is no evidence of any correlation between being immune deficient and having engaged in promiscuous homosexual intercourse. Intravenous drug use is uncommon among impoverished African villagers - even among city dwellers. Did this mean, deductively, that heterosexual intercourse could put anyone at risk for AIDS? Did the "AIDS epidemic" in Africa portend the future of the developed world? The scientific establishment certainly thought so. **Biomedical funds that had been earmarked to fight African malaria, tuberculosis, and leprosy were diverted into sex counseling and condom distribution, while social scientists shifted their attention to behavior modification programs.**

Good Intentions, Bad Science: HIV Tests and Disease

A reappraisal of AIDS in Africa should also recognize that HIV tests are notoriously unreliable among African populations where other endemic microbes and bacteria cross-react to produce ludicrously high false-positive results. **A 1994 study on central Africa reported that the microbes responsible for tuberculosis, malaria, and leprosy were so prevalent that they registered over 70% false HIV-positive tests.** HIV tests also register positive results in people whose immune systems are compromised for a wide variety of reasons, including chronic parasitic infections and anemia brought on by malaria.

By definition, all viruses that cause a "disease" infect over 30% of the cells they target, are present in the blood at concentrations in excess of 10,000 per milliliter, and are contagious. HIV is such a weak retrovirus that when detected at all, it is present in such low concentrations (about one per milliliter) that only its antibodies can be detected. This is the probable explanation as to why it is barely transmissible, requiring an average 1000 unprotected vaginal sex contacts with an antibody-positive person for someone to "get" HIV. Most HIV tests do not detect a virus but rather viral antibodies that are read with an assortment of proteins that are not even unique to HIV. The notion that antibodies are a prognosis of death defies all classical experience with viruses, microbes, and antibodies. An investigation reported in The Lancet was equally revealing. In Uganda, 9,389 individuals with unequivocal blood test results were enrolled in a study. After two years, 3% had died, 13% had left the area, and 84% remained. **There had been 198 deaths among the seronegative people and 89 deaths in the seropositive ones.** Medical assessments made prior to death were available for 64 of the HIV-positive adults. Of these, five (8%) had AIDS as defined by the WHO clinical case symptoms. **The self-proclaimed "largest prospective study of its kind in sub-Saharan Africa" had tested nearly 9400 people in Uganda, the so-called epicenter of AIDS in Africa. Yet of the 64 deaths recorded among those who tested positive for HIV antibodies, only five were diagnosed as AIDS-induced.** A highly touted 1995 report on the Mwanza region of Tanzania claimed that "improved STD treatment reduced HIV incidence by about 40%...[in] the first randomized trial to demonstrate an impact of a preventive intervention on HIV incidence in a general

population." **This occurred despite the fact that "no change in reported sexual behavior was observed in either group."** And on close inspection of the data, one realizes how the 40% reduction was measured. Of the individuals who initially tested HIV-negative, in the intervention group 48 out of 4149 (1.2%) were HIV-positive two years later; 82 of 4400 (1.9%) in the comparison group tested HIV-positive. **The researchers arrived at the "40% reduction" figure merely by comparing the difference between 1.2% and 1.9%.** The Africans in this study had tested positive or negative for antibodies to HIV but the source of their "infection" was unknown. **While the research suggested that a regimen of antibiotics reduced the prevalence of HIV-antibodies, the investigators always assumed, without any evidence whatsoever, that it had somehow reduced the transmission.**

AIDS researchers in Africa assume that there is a correlation between clinical symptoms - weight loss, chronic diarrhea, fever, persistent coughs - and sexual activity. **Correlation - whether one phenomenon is found in tandem with another - is not causation.** Proof of causation requires that we control all variables in order to isolate one variable as a cause, not merely an associated factor. The clinical symptoms that define an AIDS case in Africa are expressed in roughly equal numbers among men and women, not because of alleged heterosexual transmission, but more likely because the socio-economic conditions that give rise to these symptoms are caused by environmental risk factors to which many Africans are regularly exposed. A literature review in the World Journal of Microbiology and Biotechnology pinpointed the methodological flaw in the belief that AIDS is sexually transmissible:

"Since AIDS is a panoply of diseases or symptoms and signs, the minimum requirement to prove that AIDS is spread by sexual activity is to take an index case, isolate the putative agent, trace the sexual contacts of that case, and then isolate the same agent. To date, no data anywhere of this type has ever been presented either in Africa, or anywhere else.

In the whole history of medicine there has never been an example of a sexually transmitted disease which is spread unidirectionally, and certainly not one that is spread unidirectionally in one country and bidirectionally in another.

Indeed, given this and the other differences between AIDS in the West and Africa, it is necessary to postulate that HIV must possess unique features...[and] be able to distinguish the gender and country of residence of its host. The only other alternative is to agree with African physicians that positive HIV antibody tests in Africa do not mean infection with HIV and that immnuosuppression and certain symptoms and diseases which constitute African AIDS have existed in Africa since time immemorial."

Nor is there evidence of widespread secondary or tertiary transmission of HIV/AIDS among heterosexuals in the West either. "This is an important point to consider," warns Michelle Cochrane, "because the foundation of orthodox AIDS science and epidemiology rests upon the premise that HIV/AIDS is relatively frequently transmitted from an index AIDS case (the primary individual) to a secondary AIDS case either through an exchange of semen or blood. In turn, this secondarily 'infected' individual must, per force, be capable of transmitting HIV/AIDS to a third individual (tertiary transmission) by the same means, or an infectious disease epidemic cannot be sustained." In a meticulous review, Cochrane juxtaposed the central tenets of AIDS orthodoxy against the material record of San Francisco AIDS patients' charts. **She found that public health officials persistently over-estimated the risk of contracting HIV/AIDS through sexual activity "while simultaneously under-estimating the proportion of the HIV/AIDS caseload that were attributable to intravenous drug use and/or socio-economic factors which condition**

access to healthcare and prevention services." Cochrane showed that health officials conspicuously failed to investigate all risk factors for immunological dysfunction among heterosexual adult females. In their surveillance studies, it was considered sufficient for

"a heterosexual female merely to claim that the source of her infection was sex with an IV drug user or another man at risk for HIV/AIDS...A percentage of the 187 female AIDS cases [out of 24,371 cumulative cases in San Francisco] attributed to sexual transmission would, with proper investigation, be attributable to IV drug use. Epidemiological research in the United States and Europe has never proven that a female has sexually transmitted HIV to a man. [Because] heterosexual transmission of HIV from a male to a female happens with difficulty and very infrequently...all AIDS surveillance statistics on female AIDS cases have been gathered without rigorous scrutiny of the woman's risk for disease and with a bias towards including as many women as possible.[emphasis added]"

The a priori assumptions that directed AIDS surveillance activities in the United States subsequently allowed predictions about an exponential spread of the disease to survive as "common knowledge" despite the lack of empirical data. These are critical points to consider when reviewing epidemiological data on "AIDS" cases in Africa. For the period 1984-95, the WHO compared estimates of HIV seropositivity with the actual numbers of AIDS cases in its Weekly Epidemiological Reports. **The cumulative result is that 99.95% of all Africans do not "have AIDS" and 97% of those who are presumed to be HIV-positive had not developed AIDS.**

AIDS and the Medicalization of Poverty

Primary health care systems in Africa will remain hampered until public health planners systematically gather statistics on morbidity and mortality to accurately show what causes sickness and death in specific African countries. During the past ten years, as external financing of AIDS programs in Africa dramatically increased, support for other health sectors remained static even though deaths from malaria, tuberculosis, neo-natal tetanus, respiratory diseases, and diarrhea grew at alarming rates. **The political economy of underdevelopment and environmentally caused endemic sickness pose the gravest threats to African health.** Poor harvests, rural poverty, migratory labor systems, urban crowding, ecological degradation, social mayhem, the collapse of state structures, and the sadistic violence of civil wars are the primary threats to African lives. When essential services for water, power, and transport break down, public sanitation deteriorates and the risks of cholera, dysentery, and respiratory infection increase.

WHO Director General Hiroshi Nakajima warns emphatically that "poverty is the world's deadliest disease." Indeed, the leading causes of immunodeficiency and the best predictors for clinical AIDS symptoms in Africa are impoverished living conditions, economic deprivation, and protein malnutrition, not extraordinary sexual behavior or the trace measurements of antibodies for a mysterious virus that has yet to be isolated. The so-called "AIDS epidemic" in Africa has become the medicalization of poverty to justify Western medical intervention in the form of vaccine trials, drug testing, and almost evangelistic demands for behaviour modification. AIDS scientists and public health planners must recognise the role of malnutrition, poor sanitation, anemia, and parasitic and endemic infections in producing the clinical AIDS symptoms that are manifestations of non-HIV insults. Socio-economic development, not sexual restraint, is the key to improving health care systems in Africa.

Phillipe and Evelyn Krynen, medically trained charity workers employed by the French group Partage in Kagera Province of Tanzania, report that when "appropriate treatment was

given to villagers who became ill with complaints such as pneumonia and fungal infections that might have contributed to an AIDS diagnosis, they usually recovered." In Kenya, a former surgeon, Father Angelo D'Agostino who founded Nyumbani, a hospice for abandoned and orphaned HIV-positive children had experiences that corroborated those of the Krynens:

"People think a positive test means no hope, so the children are relegated to the back wards of hospitals which have no resources and they die. *They are very sick when they come to us. Usually they are depressed, withdrawn, and silent....But as a result of their care here, they put on weight, recover from their infections, and thrive. Hygiene is excellent [and] nutrition is very good; they get vitamin supplements, cod liver oil, greens every day, plenty of protein. They are really flourishing."*

Conclusion

People can be encouraged to behave thoughtfully in their sexual lives if they are provided with reliable information about condom use, contraception, family planning and venereal diseases. Multilateral institutions and African AIDS educators should familiarize themselves with the literature that demonstrates the contradictions, anomalies, and inconsistencies in the HIV/AIDS orthodoxy. They have a major responsibility to consider the non-contagious explanations for "AIDS" cases in Africa and to stop the proliferation of terrifying misinformation that equates sexuality with death.

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21) Thomas Bass, Reinventing the Future: Conversations with the World's Leading Scientists (Reading, Massachusetts: Addison-Wesley, 1994), p. 40.
22) Oscar Kashala, et. al. "Infection with HIV-1 and Human T Cell Lymphotropic Viruses Among Leprosy Patients and Contacts...," Journal of Infectious Diseases, Vol. 169, (February 1994), pp. 296-304.

23) Eleni Papadopulos-Eleopulos, et. al., "Is A Positive Western Blot Proof of HIV Infection?" Bio/Technology, Vol 11 (June 1993), pp. 696-707 explains why there is no correlation between a positive HIV antibody test result and the isolation of HIV itself. The authors conclude that "the use of HIV antibody tests as predictive, diagnostic and epidemiological tools for HIV infection needs to be carefully reappraised." See also, Eleni Papadopulos-Eleopulos et. al., "The Isolation of HIV: Has It Really Been Achieved?" Continuum, Vol. 4, #3 (September/October 1996). Another recent study reports that even if HIV-1 is detected in the blood or cervical secretions of an HIV-seropositive woman, "the amount of HIV-1 excreted in the cervicovaginal fluid is independent of the quantity of virus present in the blood cells or plasma." Suraiya Rasheed, et. al., "Presence of Cell-Free Human Immunodeficiency Virus in Cervicovaginal Secretions is Independent of Viral Load in the Blood of Human Immunodeficiency Virus-Infected Woman," American Journal of Obstetrics and Gynecology, Vol. 175, #1 (July 1996), p. 123. Richard Strohman, Professor Emeritus of Molecular Biology at University of California (Berkeley), points out that "HIV science has always been based not on detection of real infectious units (real virus) growing under some reasonable standard condition in living cells in the lab. Rather it is based upon a high tech series of assays constructed so that disappearingly small quantities of the virus, or some part of the virus, or some trace (aura) of viral presence may be measured. We have substituted the measurement for the real thing, like substituting the menu for the meal." (E-mail message, July 7,1997)

24) Daan W. Mulder, et. al., "Two-Year HIV-1-associated Mortality in a Ugandan Rural Population," The Lancet, Vol. 343 (April 23, 1994), pp. 1021-23.

25) Heiner Gosskurth, et. al. "Impact of Improved Treatment of Sexually Transmitted Diseases on HIV Infection in Rural Tanzania: Randomized Controlled Trial," The Lancet, Vol. 346, (August 26, 1995), pp. 530-36. The a priori assumptions of the research team are evident in an exchange with Richard Hayes (London School of Hygiene and Tropical Medicine), the corresponding author for the research group. On October 14, 1996, I sent a series of nine questions to Hayes to clarify the group's findings. Hayes' responses on March 14, 1997 are indented after each question below:

a) Among the twelve village health centers on or near Lake Victoria where "annual HIV incidence" was 1%, what techniques did researchers use to distinguish between the incidence or prevalence of HIV and the transmission of HIV? What method was used to determine that HIV was actually "spreading" or that the incidence of new cases had decreased?

1. "We measured the incidence of HIV infection by following up a random sample of adult residents over two years. The annual incidence is the proportion of seronegative subjects who seroconvert, divided by two (because of the two-year follow-up period). In the 'comparison communities' (which did not receive the improved STD services), 1.9% seroconverted over two years, giving an annual incidence of about 1% as stated. In the 'intervention communities' (which did receive the improved services) only 1.2% seroconverted, so the incidence of new infections was about 40% lower, presumably as a result of the intervention."

b) The survey suggests that improving the STD case-management brought about a 42% reduction of HIV incidence. What percentage of the patients who were initially diagnosed with a sexually transmitted disease also initially registered positive for HIV-antibodies?

2. "I do not understand this question. What do you mean by 'the patients initially diagnosed with an STD"? If you mean those presenting for STD treatment at the health units, HIV testing was not carried out on these patients. The point of the study was to make available the improved STD services for everyone living in the intervention communities (regardless of HIV status). We then measured the HIV impact by testing a random cohort of individuals at baseline and follow-up. These HIV tests were carried out in a population-based survey, not through the health units where the STD treatment was provided."

c) The intervention group received a proper supply of antibiotics to treat bacterial STDs and were actively encouraged towards health care-seeking behavior. Wouldn't that suggest that people who receive better health care are less likely to register HIV+?
3. "Of course the whole point of our trial was that improved health care (specifically improved STD treatment) reduced HIV incidence in the general population. However, I am not sure what you mean by 'less likely to register HIV+'? Do you mean that the antibiotics given to STD patients would interfere in some way with the HIV serological tests? There is no evidence of any such effect, as far as I am aware."
d) There was no discernible difference in the reported sexual behavior or frequency of condom use in the intervention and control communities. While the intervention of drug therapies may have played a role in reducing HIV seroprevalence, what would that necessarily suggest about HIV transmission?
4. "Transmission implies the occurrence of new cases as the virus is spread from one individual to the next. This is measured as the 'incidence' of new infections, as explained above, and our results showed a clear effect of the intervention on incidence. We assume the explanation for this is that it is much easier for the HIV virus to be transmitted from one sexual partner to the other if one of them has another STD (this is the so-called STD Cofactor Effect). By treating STDs promptly and effectively, you should be able to reduce their duration and hence prevalence, so that it becomes much more difficult for the HIV virus to be transmitted." [see footnotes #13 and #14 above]
e) What did the research team identify as the "measured risk factors for HIV infection"? Which of those risk factors were significantly reduced?
5. "Question not understood. What part of the paper does this refer to?"
f) The baseline HIV prevalences were 3.8% for the intervention group and 4.4% for the comparison group. The prevalence of active syphilis was 8.7% and 8.3% respectively and The Lancet article states that the "treatment regimens would be expected to achieve cure in over 90% of cases of sexually transmitted diseases...Over the two years of follow-up there were 48 seroconversions (1.2%) in the intervention group and 82 (1.9%) in the comparison group... HIV incidence...was consistently lower in the intervention community..." Did fewer people register HIV-positive antibody results because they received antibiotics to treat their STDs, quite apart from any changes in sexual behavior?
6. "See 3. above. You are correct that the effect on HIV is attributed to the improved treatment of STDs, and not to any change in sexual behavior. In fact no change in sexual behavior was seen."
g) The Lancet report states that the difference between 1.2% and 1.9% is the "overall reduction in HIV incidence of about 42% over two years of follow-up...[and]...the most plausible explanation for our results is that the STD treatment programme reduced HIV incidence by shortening the average duration of the STDs, thus effectively reducing the probability of HIV transmission" even though actual sexual behavior involving condom usage did not change at all. Did the researchers report the incidence of new STDs in either group two years later?
7. "We only surveyed our cohort at baseline and at follow-up two years later. You would need much more intensive follow-up to accurately measure STD incidence during this period. However, we were able to measure the prevalence of STDs at follow-up, and could demonstrate a significant effect of the intervention on active syphilis and on symptomatic male urethritis. These findings are consistent with the explanation given in 4. above. A paper setting out the STD data in more detail is about to be submitted for publication."
h) Although the report states that the STD intervention program "evidently had a substantial effect on HIV incidence in this rural population," since there was no noticeable change in "risk behavior" among the intervention communities, why wouldn't researchers conclude that improving the health care makes people healthier even in the absence of demonstrable changes in the sexual behavior practices thought to facilitate HIV transmission?

8. "That is exactly what we did conclude. Improved health care (specifically improved STD treatment services) reduced HIV transmission without any change in the sexual behavior of the population." [Actually the report showed that it putatively reduced the incidence or prevalence, not transmission.]
i) Please elaborate on the "cofactor effects" mentioned in the conclusion: "the impact of improved STD treatment depends on the proportion of HIV infections in the general population that are attributable to the cofactor effects of STDs..."
9. "This relates to the 'STD Cofactor Effect' mentioned above (see 4.). Obviously, in a population where STDs are present at a very low level, few HIV infections would be attributable to the enhancing effect of STDs, and so removing STDs from the population would have little effect on the HIV epidemic. Conversely, in parts of the world where STD prevalences are very high, it is possible that a large proportion of HIV infections could be avoided by removing or reducing the prevalence of STDs through improved treatment, and this is what our results suggest in Mwanza."
26) For a small sample of articles that uncritically apply the contagious HIV/AIDS theory to Africa, see: John C. Caldwell and Pat Caldwell, "The African AIDS Epidemic," Scientific American (March 1996), pp. 62-68; Simon Gregson, "Will HIV become a Major Determinant of Fertility in Sub-Saharan Africa?" Journal of Development Studies, Vol. 30, #3 (April 1994), pp. 650-79; and Kelly Lee and Anthony B. Zwi, "A Global Political Economy Approach to AIDS: Ideology, Interests and Implications," New Political Economy, Vol. 1, #3 (1996), pp. 355-73.
27) Eleni Papadopulos-Eleopulos, Valendar Turner, John Papadimitrou and Harvey Bialy, "AIDS in Africa: Distinguishing Fact from Fiction," World Journal of Microbiology and Biotechnology, Vol. 11 (March 1995), pp. 141-42.
28) Michelle Cochrane, "The Social Construction of Knowledge on HIV and AIDS: With a Case Study on the History and Practices of AIDS Surveillance Activities in San Francisco," Ph.D. dissertation, Department of Geography, University of California, Berkeley, April 1997, p. 253. Cochrane's dissertation is a case study of the emergence of AIDS and the creation of a bureaucracy for AIDS surveillance in San Francisco. Orthodox epidemiological and surveillance knowledge on AIDS in San Francisco played a key role in the construction of a global consensus on AIDS historiography and science. According to Cochrane, this knowledge displays a remarkable coherence and internal consistency that is marshaled to refute any critique of its assumptions about the etiology, epidemiology, and history of AIDS. The AIDS Sero-epidemiology and Surveillance Branch in San Francisco constitutes the greatest repository in the world for primary documentation on AIDS. It includes the medical charts and case files for every one of the 24,371 AIDS patients cumulatively reported since 1981 in the city. Cochrane demonstrates how the vested interests of institutions, organizations, and individuals perpetuated the orthodox consensus that HIV causes AIDS, "a conclusion which persists despite the presence of multiple lacunae or anomalies that the theory has not resolved." (pp. 322-24)
29) Cochrane, op. cit., p. 7.
30) Ibid., pp. 259-60.
31) For instance, even though South Africa reported only 1,120 AIDS cases in 1995 but 90,292 cases of tuberculosis in 1994, AIDS was accorded a much higher national profile and larger budget so that it now dominates clinical practice across all medical fields ranging from pediatrics to neurology. World Health Report 1996, p. 130; "South Africa: Country Profile," The Lancet, Vol. 349 (May 24, 1997), p. 1542.
32) World Health Organization, Bridging the Gaps: The World Health Report 1995 (Geneva: WHO, 1995), Table 5 (p. 18) and Table A3 (p. 110); and World Health Organization, Fighting Disease, Fostering Development: The World Health Report 1996 (Geneva: WHO, 1996), Table 4 (p. 24) and Table A3 (p. 127).
33) WHO, The World Health Report 1995, v.

34) This is further elaborated in Charles Geshekter, "Outbreak? AIDS, Africa, and the Medicalization of Poverty," Transition, #67 (Fall 1995), pp. 4-14; and Cindy Patton, Inventing AIDS (New York: Routledge, 1990), especially Chapter 4, "Inventing African AIDS."
35) Cited in Neville Hodgkinson, "Cry, Beloved Country: How Africa Became the Victim of a Non-existent Epidemic of HIV/AIDS," in P. H. Duesberg (ed.), AIDS: Virus- or Drug-Induced? (Amsterdam: Kluwer Publishers, 1996), p. 353.
36) Hodgkinson, op. cit.. pp. 350-51.
37) Recent critical studies include: Richard and Rosalind Chirimuuta, AIDS, Africa and Racism (London: Free Association Books, 1989); Neville Hodgkinson, AIDS: The Failure of Contemporary Science (London: 4th Press, 1996); Elinor Burkett, The Gravest Show on Earth (Boston: Houghton Mifflin, 1995); Hiram Caton, The AIDS Mirage (Sydney: University of New South Wales Press, 1994); Robert Root-Bernstein, Rethinking AIDS: The Tragic Cost of Premature Consensus (New York: Free Press, 1993); and Peter Duesberg, Infectious AIDS:Have We Been Misled? (Berkeley: North Atlantic Books, 1996).

For an expose of the CDC's deceitful scare campaign in the United States, see Amanda Bennett and Anita Sharpe, "AIDS Fight is Skewed by Federal Campaign Exaggerating Risks," Wall Street Journal (May 1, 1996) and David R. Boldt "Aiding AIDS: The Story of a Media Virus," Forbes Media Critic (Fall 1996). The CDC believed that exaggerating the risks to the entire American population was the only way to drum up widespread support for measures and funding to combat AIDS. Thus, the theme of its public service ad campaign launched in 1987 was, "If I can get AIDS, anyone can." But from 1990 to 1992, the proportion of heterosexuals (aged 18-49) in high risk American cities who reported multiple sexual partners increased from 15% to 19%, while condom sales decreased by 1%, and 65% of respondents admitted they used condoms either sporadically or not all. Americans are not practicing safe sex and for this reason teen pregnancies and real venereal diseases are on the rise. Yet "AIDS" cases continue to decrease sharply and even the fraction of Americans that is assumed to be HIV-antibody positive has declined from an estimated 1 million in 1985 to 700,000 in 1996. See, Joseph A. Catania, et. al., "Risk Factors for HIV and Other Sexually Transmitted Diseases and Prevention Practices Among U.S. Heterosexual Adults: Changes from 1990 to 1992," American Journal of Public Health, Vol. 85, #11 (November 1995), pp. 1492-99.

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AIDS IN AFRICA: THE WRONG DIAGNOSIS AND THE WRONG TREATMENT

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It is widely accepted that the most important health problem facing many African countries is an epidemic of sexually transmitted immune deficiency. As a consequence, African Ministries of Health, assisted by non governmental agencies funded by Western countries have concentrated their energies and resources on limiting the transmission of immune deficiency, at the expense of combating other infectious diseases known to affect millions of Africans each year. Yet if the scientific evidence for African AIDS is unsound, so will be the policies founded on this science, and many Africans may be suffering and dying needlessly from preventable and treatable diseases.

The scientific method may be the best way of discovering how the physical world functions, but it is far from flawless. Many naively believe that scientists begin their work by making observations and measurements, then elaborate hypotheses that, if confirmed by experiment, enter the body of scientific knowledge. **Yet even the gathering of data is determined by the pre-existing views of the scientists, who will seek evidence for the hypotheses they wish to verify and may ignore evidence to the contrary**. In order to reduce such subjective bias it is generally accepted that scientific hypotheses should undergo critical review, to the extent that evidence for the contrary hypothesis should be considered and investigated.

Many Africans were sceptical when scientists proposed that AIDS originated in African monkeys. For example, Yinka Adeyemi, the science and health correspondent for the Nigerian Weekly, Concord, wrote in July 1985: „To the average European researcher in virus cancers, the notion that the Acquired Immune Deficiency Syndrome (AIDS) had its origin in Africa is now a scientific fact... Yet, arguments by such scientists whose minds are made up about the African connection are replete with fundamental loopholes and illogicalities that render them not plausible..." Gallo, who first identified the AIDS-causing virus in man, said at the Dakar conference: "Viruses closely related to HTLV, but distinct from it, have been isolated from Old World monkeys. This and other facts led us to propose that the ancestral origin of HTLV is in Africa." Comments such as this immediately raise problems because of the socio-historical implications. To the ordinary man, Gallo will be understood as saying that: "We (European scientists) conclude that AIDS originated from Africa because we found AIDS virus in monkeys, and Africans are closer to monkeys." (1) Yinka Adeyemi's scepticism was well founded. Retroviruses in nature are species specific, and a retrovirus in one species of monkey will not pass to another, let alone to a human. If a retrovirus had mutated in a monkey to a form infectious to a human, it is most unlikely it would remain infectious to the monkey. Transmission of a retrovirus between monkey and man in nature is, therefore, extremely improbable, and Gallo and his fellow retrovirologists must have known this. Indeed, subsequent experiments on retroviruses that supposedly proved a the link between monkeys and humans were found to be due to laboratory contamination. (2,3,4,5,6,7,8)

Other scientists claimed that AIDS was an old disease of Africa. For example Kevin De Cock wrote in 1984: „This report proposes that the infectious agent causing AIDS... is endemic and unrecognised in parts of sub-Saharan Africa, from where it recently disseminated into external populations...In rural Africa diagnosis is often inexact. Fever is readily attributed to malaria without confirmation, and pneumonia is often assumed to be pneumococcal or tuberculous... In such a situation immunodeficiency would go unrecognised... As Kaposi's sarcoma was a feature in about one third of reported cases of

AIDS, it would seem mandatory to look for AIDS where Kaposi's sarcoma has its highest incidence in the world, equatorial Africa... The incubation period of AIDS is thought to be one or more years. The first American cases are likely to have become infected in the early to mid-1970's, a time when tourism from the United States to Africa was developing as a result of heightened cultural interest..." (9) The historical ignorance displayed here is quite breathtaking. For many centuries before the Portuguese sailed around the Cape powerful west African kingdoms conducted trade across the Sahara to the Mediterranean, and every year many thousands of west Africans made the pilgrimage to Mecca. On the east African seaboard there were city states that flourished on trade between the central and southern African kingdoms such as Monamatapa in Zimbabwe and Asia as far as Ming dynasty China. With the advent of the Portuguese began four hundred years of the African slave trade, during which many millions of Africans were transported to the New World and Europe, and when African women were regularly raped from the time of capture. Following the demise of the slave trade came the scramble for Africa, when almost the entire continent was colonised by the European powers. (10,11,12,13) If AIDS was the cause of a tumour as common as Kaposi's sarcoma in equatorial Africa, the disease would have spread to the rest of the world hundreds if not thousands of years earlier. **Even if spread were recent, AIDS would have appeared in Europe before the United States.** (14,15) Nonetheless attempts were made to find an "isolated tribe" harbouring HIV without success.

AIDS-like cases allegedly acquired in Africa before the appearance of the epidemic in the West have also been cited as evidence that AIDS was an old disease in Africa. These cases include a British and a Norwegian seaman who sailed all over the world, and a Danish surgeon who worked in Zaire. (16,17,18) In fact pre-epidemic AIDS-like cases can be found in many countries, and the Danish surgeon is a most striking example of the selectivity of AIDS scientists. (19) On the opposite page of same journal where she is reported there is an account of a German homosexual who died of an AIDS-like illness in the same year (1976), but the Danish surgeon's case has been cited many times, and the German case has not been mentioned. (20) **Later it was found that the Danish surgeon's blood was negative for HIV.** (21)

When blood tests were developed for HIV these too were used to prove an African origin for AIDS. Alarmingly high rates of positivity were found in stored blood samples, but the results were challenged by other scientists. (22,23,25,26,27,28) One critic, Professor Hunsmann, head of virology and immunology section and professor of medicine at the German Centre of Primate Research at Gottingen, published results of his studies in medical journals. In an interview shown on British television he said the following: „We... had several thousand serum samples frozen and saved in our refrigerated stock. When the news came that there was another, and new human retrovirus discovered, the AIDS virus... we could immediately search among our stock and probe for an earlier presence of this virus in Africa... These tests quickly and clearly gave results, namely, that the first "positive" probes which we could find among our more than 7,000 serum samples are dated only after the beginning of the 'eighties, from the years 1982-83; and that among samples from before that date- and we had quite a lot of that earlier time in our stock- not a single one proved positive." (24) Later in the same interview when asked why AIDS is not considered to have originated in the United States, Professor Hunsmann made the following comment: „Testing of the kind being done in Africa and to that volume has never been done by anyone in America. Nobody has looked at the stocked blood serum in the USA and there certainly is much more there than in Africa. Nor has anyone asked what happened to the general population. Only one single group, the homosexual community in San Francisco, has been analysed and the results showed a high percentage of HIV positivity already by the mid 1970's. But no other samples have been tested to the extent done in Africa. I think this should be clearly said." (24)

It would seem reasonable to expect that questions about the reliability of blood tests for a disease as serious as AIDS would be investigated thoroughly. Indeed false positivity could have been anticipated, as it is well known that African serum can give false positive reactions to a variety of blood tests, a phenomenon called the "sticky serum syndrome" which is largely due to chronic malaria. Eighty percent of the worlds cases of malaria occur in Africa, and although it is a major cause of disease and death, most Africans survive because they develop immunity at an early age. This immunity does not normally eliminate the parasite or prevent reinfection, but does reduce the number of parasites circulating in the blood stream. (29) Sufferers can develop very high levels of antibodies (proteins responsible for immunity) to malaria, and these can react with foreign proteins that have nothing to do with malaria (a false positive reaction). Each type of antibody is made to react strongly with a specific infectious agent, but may react weakly with other infectious agents. If there are very high levels of a particular antibody, even a weak, non-specific reactions will register as a positive test, and this may well be the case with malarial antibodies and HIV tests. Paradoxically a degree of cell mediated immune suppression (the type of immune suppression found in AIDS) can also occur. (30) AIDS researchers have claimed that although early tests for HIV were unreliable, these problems have been resolved, but these claims are not supported by evidence. (31)

When European doctors went to Africa in the early stages of the AIDS epidemic they did not go because African doctors or Ministries of Health had noticed a new phenomenon and requested their assistance. They went to search for cases of AIDS because they believed that AIDS came from Africa, and initially they were regarded with scepticism and even hostility. (32) Without difficulty they found African patients with cell mediated immune suppression who had reduced numbers of helper T lymphocytes (T4 cells) and although diseases common in Africa such as malaria and tuberculosis are known to deplete T4 cells, they concluded that these patients were suffering from heterosexually transmitted AIDS. (33,34) With this experience the WHO proposed the following clinical case definition for AIDS in Africa:

WHO clinical case definition for AIDS in Africa (an adult must have at least 2 major and 1 minor signs)

1. Major signs
   (a) weight loss >10% body weight
   (b) chronic diarrhoea >1 month
   (c) prolonged fever >1 month (intermittent or constant)
2. Minor signs
   (a) persistent cough >1 month
   (b) generalised pruritic dermatitis
   (c) recurrent herpes zoster
   (d) oro-pharyngeal candidiasis
   (e) chronic progressive and disseminated herpes simplex
   (f) generalised lymphadenopathy (34)

Like the blood tests used to diagnose AIDS in Africa, this case definition is non-specific, and would easily describe a patient presenting with tuberculosis or other common infectious diseases. Either or both the blood tests and the case definition have been used to document the epidemic of AIDS in Africa, and many African doctors and public at large have come to accept their validity. But is this tenable?

The definition of AIDS has been expanded and changed several times. (35,36) The earliest definitions included only uncommon or rare opportunistic infections such as Pnuemocystis carinii pneumonia, cytomegalovirus eye infections, and tuberculosis (TB) occurring outside

the lungs, for it was the appearance of these diseases in homosexual men that led to the recognition of the AIDS epidemic. (37) In 1993 pulmonary TB (i.e. tuberculous pneumonia) and recurrent bacterial pneumonia (the common form of pneumonia) were included as AIDS-defining diseases. (38) These diseases have always been more common in third world countries. (39) A 1997 study of all the reports of AIDS in Africa to date showed that tuberculosis and bacterial pneumonia were the predominant infectious complications of AIDS, and tuberculosis remained the leading cause of illness and death. (40) Even Kaposi's sarcoma, a tumour that has a higher incidence in parts of Africa than anywhere else in world, was not a common feature of African AIDS. (41) Thus African patients diagnosed with AIDS are not for the most part presenting with rare or uncommon diseases that occur in Western AIDS patients, but have diseases that have always been common in Africa.

According to the WHO definition of AIDS, patients with tuberculosis are diagnosed as having AIDS only if they also have a positive HIV test. A study of Zairean leprosy patients published 1994 found that antibodies to a protein in the cell wall of mycobacteria, the family of bacteria that cause leprosy and tuberculosis, could cause a false positive HIV test. (42) Researchers in Brazil studied over 1,000 tuberculosis patients and found a high rate of false-positivity for HIV infection. (43) Patients with tuberculosis commonly have cough, fever and weight loss, symptoms that fulfil the clinical case definition of AIDS in Africa. Tuberculosis itself depresses cell mediated immunity, and T4 lymphocytes can fall to levels found in AIDS patients. (44) Thus neither the symptoms nor signs of the disease nor any of the commonly used tests can distinguish a patient with tuberculosis from a patient with AIDS. At the very least one would expect that doctors would be warned to these diagnostic pitfalls, but this evidence has been almost completely ignored.

From the beginning of the AIDS epidemic in the West, AIDS has been confined to certain risk groups, namely homosexuals, intravenous drug users. and haemophiliacs and other recipients of blood or blood products. When the epidemic was attributed to the Human Immunodeficiency Virus (HIV) it was assumed that AIDS would spread inexorably from the high risk groups into the general population, and Africa was held up as the mirror of the future. After more than fifteen years of the epidemic the heterosexual explosion in the West has yet to occur. (45) There has been some spread of HIV positivity into the heterosexual partners of members of the high risk groups, but a recent study reported that male to female per contact infectivity was 0.0009, i.e. it took an average of 1,000 acts of sexual intercourse to spread HIV from an infected man to a non-infected woman. If the woman was infected and the man uninfected, transmission was eight times less efficient i.e. required an average of 8,000 acts of sexual intercourse. (46) In Western countries, AIDS has occurred in very few cases where neither sexual partner has belonged to a high risk group, and the number of these cases has not risen with time. (47,48)

It is probable that there is a small epidemic of "true" AIDS in Africa associated with the same risk factors as for AIDS in the West: exposure to blood and blood products, occurring in Africa in hospitals and clinics without resources to screen blood transfusions or sterilise needles, and unprotected anal intercourse amongst male and female prostitutes. But if HIV is so difficult to transmit by heterosexual intercourse in the West, how can HIV have spread so widely in Africa? And how can a disease that is so much more efficiently transmitted from man to woman than woman to man by heterosexual intercourse equally affect men and women in Africa? Without evidence scientists have stated that Africans are more promiscuous and engage in more unusual sexual practices than people in the West. (49) Increased transmissibility of HIV has been attributed to higher rates of other sexually transmitted diseases (STD's) in Africa, yet gonorrhoea and syphilis, for which there is at least a 20% chance of transmission in one sexual act, are less common than the reported incidence of HIV. (50,51,52) These convoluted and implausible explanations for African AIDS can only remain credible if the alternative explanation is ignored: **That the diseases**

diagnosed as AIDS in the great majority of Africans is not caused by a sexually transmitted virus.

Although Africa is portrayed as the epicentre of the world AIDS pandemic, of more than 6,000 publications on AIDS listed in the United Kingdom of Medline, the most widely used computer database of medical publications, just over 1,000 were about AIDS and Africa. African academics have few resources to undertake research without financial support from Western government and academic institutions who can then control the content of the research projects they fund. Without substantial and reliable research it is very difficult to assess the real picture of disease and death in Africa. If it is true, as some observers report, that the death rate is rising, particularly amongst young adults, it is inappropriate to believe without further evidence that this is due to sexually transmitted immune deficiency. Increasing disease and death are the inevitable companions of economic decline. (53) With progressive devaluation of the currency of many African countries agricultural production has been diverted to cash crops and nutritional standards have deteriorated. Unable to survive on the land, people have moved to the cities to live in overcrowded and unsanitary shanty towns where infectious and parasitic diseases flourish. Governments crippled by foreign debt, declining revenues from taxation and increasing costs of imported goods have little to invest in sanitation and preventive health programmes, and for those who become ill the cost of treatment is prohibitive. Real solutions for African ill health will come from economic development that will provide resources for investment in nutrition, sanitation, housing, and quality medical care.

It is a cruel form of victim blaming to attribute a patient's ill health to his or her sexual behaviour without good evidence. When such victim blaming is extended to a whole continent the consequences are far-reaching. People struggling to live under adverse circumstances are told they face mass death, and are being subjected to trials of vaccines and toxic drugs that are more likely to be harmful than beneficial. Real solutions for African ill health will come from economic development that will provide resources for investment nutrition, sanitation, housing and quality medical care.

Conclusions

In summary, Western scientists have propagated the belief that Africans are suffering from a major epidemic of sexually transmitted immune deficiency, but the scientific basis for this is unsound. The clinical criteria and blood tests used to diagnose immune deficiency in Africa are non specific, and cannot distinguish between AIDS and diseases such as tuberculosis that have always been common in Africa. Resources are being allocated to AIDS prevention at the expense of programmes to prevent and treat diseases such as malaria and tuberculosis that are killing millions of Africans each year. The fundamental causes of African ill health are due to economic underdevelopment, yet Africans are being wrongly terrified with the prospect of mass death from a fatal sexually transmitted disease. Inappropriate and even harmful treatment is the inevitable consequence of a wrong diagnosis, and when faulty science is applied to a whole continent, millions can suffer and die. Surely it is time to reappraise AIDS in Africa.

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The two main HIV antibody tests are the ELISA, which is used as a screening test, and the Western Blot, which is used as a confirmatory test. Diagnostic tests need to be specific, that is, they need to register negative in people who aren't infected. Test kit manufacturers "verify" the specificity of their tests (specificity is a measure of how often false-positives will occur) by testing several thousand random blood donors (by definition at low risk for AIDS or HIV infection), with 20 or 30 subjects thrown in who represent several of the more commonly recognised cross-reacting conditions such as rheumatoid arthritis or systemic lupus erythematosus. (The 60 or so other known cross-reacting factors8 including the Africa-endemic diseases of tuberculosis, malaria, leprosy, tapeworms, parasites, etc. are not added to the equation.) Since it is assumed that random blood donors are not infected, if any positives occur in this group, they are considered to be false-positives. For instance, if 4000 random blood donors are tested and 4 of them test positive, the specificity of the test would be calculated as 99.9% (3996/4000).

This practice of omitting Africans from the test sample, either healthy Africans or Africans with other similar non-AIDS conditions that might elicit cross-reactions, results in a picture of test accuracy which fits only the type of population in the test sample. This creates severe bias and overestimates test specificity.9 Constantine stated, "Test parameters thus obtained with this sort of a biased sample cannot be validly extrapolated to assess a test's performance in different diagnostic situations."10 In other words, **an HIV antibody test kit developed in the West will yield different results in Westerners and Africans.** ELISAs with estimated specificities in the high 90s have been used in Africa, with very poor results, for exactly this reason. Constantine reports "unsatisfactory test performance has been described in studies with east African serum from Tanzania and Egypt."10 Indeed, the specificity of one test dropped to an abysmal low of 51% when used in Africa.11 (In terms of HIV antibody tests, a specificity even of 95% would make the test hopelessly inaccurate.) Confounding this is the widely-acknowledged propensity of antibodies to one retrovirus to cross-react with the antigens of another retroviruses.12,13 Gallo and his colleagues have repeatedly stated that the p24 protein of HIV and of two other human retroviruses, HTLV-I and HTLV-II, which Gallo claims to have isolated from humans, immunologically cross-react.14 Since HTLV-1 is endemic in sub-Saharan Africa,1 people infected with HTLV-I may be mis-diagnosed as being HIV infected. In addition, in Africa (and other third world regions) diagnoses are often made on the basis of ELISA alone without Western Blot "confirmation." This is countenanced and even recommended by WHO, as Western Blots are expensive and well beyond the budgets of many countries. Yet in the United States, this is not an acceptable practice. Indeed, when concerns are expressed as to the multitude of factors which can cause false-positives on HIV antibody tests, it is often argued that using a confirmatory test will solve this problem.

Are the new "third generation" test kits any better?

The World Health Organisation (WHO) attempted to deal with this problem by organising a "sentinel surveillance" program in key centres to improve training and the quality of serological testing. Local clinics were set up to offer testing using techniques that employed genetically engineered HIV antigens called recombinant antigens (as opposed to the usual whole viral lysate antigens which contained many cross-reacting contaminants), and local lab techs were trained to use these tests properly. Gordon Stewart, a British epidemiologist had visited one such centre in Nairobi where all tests were performed with recombinant antigen, and registrations were vetted by a visiting Danish epidemiologist in co-operation with local, specially trained staff. But he suspected nevertheless that "Much of the testing in Africa was probably unsupervised and invalidated," and that positive results were not usually questioned. Indeed, recently Panafrica News Agency correspondent Eliezer Wangulu described a part of Kenya where "most health facilities have dysfunctional laboratories that have also run out of reagents."

Whether or not a significant portion of African populations has access to properly run and equipped labs and testing programs, the use of recombinant antigen test kits will not solve the problem. It is claimed that these "third generation" test kit antigens are "purified" to the extent that unwanted cross- reactions (and thus false-positives) will not occur. However, recombinant HIV antigens are derived from E. coli and may contain bacterial epitopes (a small section of the microbe which elicits a specific antibody). In test sera from some individuals with antibacterial antibodies, HIV antibody false positives may occur as a result of the interaction of antibacterial antibodies with the antigens of the enteric bacilli that contaminate the HIV test kit.15,16 Other false positives can occur for reasons unique to recombinant technology, e.g., immunoreactive epitopes may rely on either primary amino acid sequence or conformational shape for antigenicity and therefore, non-specific reactivity may result if similar epitopes exist on different viruses (such as the common flu virus).15 The fact that other microbes share epitopes with HIV has often been documented.17,5 Test systems based on recombinant HIV antigens have yielded positive results much more often than those based on whole viral lysate.16 **A study of two groups of blood donors showed false positives to occur twice as frequently in the group tested with recombinant-antigen-based tests** (617/119,004) **as in the group tested with lysate-antigen-based tests** (246/119,178).18 Another study done in the former USSR to determine the positive predictive value (how often a positive test result indicates a true infection) of various confirmatory tests yielded the following results in AIDS high-risk groups:19

Whole viral lysate antigens: 99.4% specificity Recombinant peptide antigens: 95.1% specificity Synthetic peptide antigens: 86.1% specificity

As mentioned above, a specificity of 95% indicates a very inaccurate test in terms of potential false-positives.

Yet another study demonstrated cross-reactions between the sera of people with autoimmune disorders (for example, systemic lupus erythematosus and Sjogren's syndrome) and synthetic peptides or recombinant gp120, gp41, and p24 proteins.17

The purity of the antigens is not the issue. Regardless of the source of antigens, **all serological tests are subject to non-specific and unpredictable reactivity**.15 It does not matter whether the HIV antigens are natural or engineered, or not even derived from HIV itself (e.g., a serological test for infectious mononucleosis employs sheep red blood cells). What does matter is whether the reactions of patients' sera with these antigens are shown to be specific for the presence of HIV in vivo. A fundamental principle of antibody testing is that for a test to be valid, regardless of time of development, generation, or appellation, its specificity must be authenticated by the use of an independent gold standard.20

Mycobacterial diseases can cause false-positive HIV antibody tests

In 1994, Essex found significant levels of false-positive reactions on both ELISA and Western Blot in people with leprosy, a disease associated with Mycobacterium leprae infection.5 Antibodies to the carbohydrate structures found in the mycobacterial cell wall, lipoarabinomannan (LAM) and phenolic glycolipid (PGL), were noted to "[yield] significant cross-reactivities with the HIV-1 pol [p31] and gag [p24] proteins." Essex stated that the "data suggest that mycobacterial cell wall antigens may share common epitopes with HIV" and warned that "ELISA and Western Blot may not be sufficient for HIV diagnosis in AIDS-endemic areas of Central Africa where the prevalence of mycobacterial diseases is quite high."

These carbohydrate-containing antigens are also present in other mycobacteria, in particular Mycobacterium tuberculosis. It is particularly significant to note:

1. Of the 661 million people in sub-Saharan Africa, 2-3 million have active TB with an annual mortality of 790,000;21

2. TB has now become an AIDS-defining illness, and 30-50% of African "AIDS" deaths are from TB;21

3. "HIV infection" as evidenced by positive HIV antibody tests does not precede TB infection but rather follows it;21

4. In a tuberculosis santitorium in Kinshasa, Zaire, half of the suspected pulmonary cases, one-third of the confirmed cases and two-thirds of the confirmed extra-pulmonary cases had a positive HIV Western blot test.21,22

The presumption is that HIV infection leads to tuberculosis as an AIDS indicator disease, but from the above data it is more reasonable to conclude the opposite, that 1) antibodies present in people with tuberculosis causes cross-reactions and false-positives on HIV antibody tests, 2) HIV-positive TB patients have tuberculosis alone and are not infected with HIV.

Anti-carbohydrate antibodies cross-react with HIV proteins

It has been recognised at least since 1980 that naturally-occurring anti-carbohydrate antibodies cross-react with HIV proteins.23 Healy speculated that false-positive Western Blot gp41 bands were actually due to anti-carbohydrate antibodies, since gp41 and non-viral proteins share similar antigenic structures.24 Tomiyama stated that "normal human serum contains antibodies capable of recognising the carbohydrate moiety of the HIV envelope glycoproteins (gp41, gp120 and gp160)."25 This is of particular significance when one realises that African criteria for reading Western Blots allow a positive diagnosis based on two envelope bands alone. Eleopulos states "Not only mycobacteria (M. leprae, M. tuberculosis, M. avium-intracellulare) but also the walls of all fungi (Candida albicans, Cryptococcus neoformans, Coccidioides immitis, Histoplasma capsulatum including Pneumocystis carinii), contain carbohydrate (mannans). One hundred per cent of AIDS patients (even those with 'No candida clinically') have Candida albicans antibodies...Since antibodies to mannans react with the 'HIV proteins' then, as Essex and his colleagues have pointed out for mycobacterial infection in Africa, one would expect the sera of all people infected with fungi and mycobacteria to cross-react with the 'HIV-1 glycoproteins as well as to cause 'significant cross-reactivities with HIV-1 pol and gag proteins.'"17

The vast majority of opportunistic infections experienced by AIDS patients in the US/Europe are due to PCP, candidiasis, cryptococcosis, coccidioidomycosis, histoplasmosis, tuberculosis or Mycobacterium avium-intracellulare disease (88% of AIDS cases diagnosed between 1988 and 1992 had one or more fungal or mycobacterial infections).17 At the very least tuberculosis and histoplasmosis26 are endemic in many parts of Africa, and if AIDS in Africa and AIDS in the US/Europe are the same disease, it can be presumed that many African AIDS patients will be infected with the above organisms.26

A mannose-type oligosacharride is furanose, from which the antibiotic nitrofurantoin is derived. O.M. Mulugheta, an African who had worked for two years as a tropical medicine doctor in Malawi, was concerned about possible exposure from his practice and decided to take an HIV antibody test. It was positive (both on ELISA and Western Blot). Three weeks prior to the testing, he and his wife had both taken the antibiotic nitrofurantoin for a minor

urinary tract infection. Several weeks later they both experienced the symptoms of polyneuropathy, dermatitis, allergic pneumonitis, herpes zoster, and severe headaches. AIDS was immediately suspected by other local physicians, but Mulugheta was convinced he wasn't infected with HIV. Refusing to accept a diagnosis of HIV infection or AIDS, and a prognosis of death, he researched his problem and developed the theory that it was the furanose sugar (of which nitrofurantoin is made) or its metabolites the furans, which lead to reactive HIV antibody tests in Africans. Mulugheta pointed out: "Furanose is found in the husks of maize, barley, and oats. Maize is a dietary mainstay of the Central African states and much, if not all, of their local home-brew is made from maize. To concentrate the alcohol content, the husks are mainly employed."

It is currently believed that 15 million Africans are infected with HIV. A recent headline in the Washington Post proclaimed that Africa is being "ravaged by [the] virus."27 According to Stewart, WHO bases this estimate on the numbers of seropositives and AIDS cases reported by member states, "accepted at face value and, with rare exceptions, unvalidated." It is assumed that a huge hidden and unreported AIDS epidemic exists in Africa (and in other third world countries as well). Attempts are made at estimating the true extent of this alleged hidden epidemic by using flawed mathematical models which assume that the numbers of actual AIDS cases (as opposed to reported AIDS cases) can be predicted from the number or estimates of seropositive results and the much lower number of reported cases.

Conclusion

On reading the testing literature, it soon becomes apparent that there is no consistency whatsoever -- there is a huge range of specificity values for test specificity that change according to a multitude of factors including type of antigen or population tested--and that determining the true specificity of HIV antibody tests for anyone, anywhere, is impossible using current methods. This situation is unlikely to change unless virus isolation is used as a gold standard with which to verify the tests. In the meantime, Africans represent a population which is particularly susceptible to false-positives on HIV antibody tests.

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PEDIATRIC AIDS: VICTIMS WITHOUT VOICES

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The first cases of pediatric AIDS were described in 1982-4 (1-4), not long after the reports of AIDS in male homosexuals, hemophiliacs and intravenous drug users and shortly before the news conference which established the dogma of HIV as the cause of AIDS. (5) Even before this event, researchers were working under the assumption that a microbe was responsible for the syndrome. The problematics of an infectious cause of AIDS and the likelihood of extraneous causes such as drugs and other factors leading to oxidative damage have been scrupulously described elsewhere. (5-7) Immune deficiency in children is of particular concern because a) their immune systems are immature, b) the course of their illness is generally shorter than that of adults (8), and c) they are dependent on the choices of others for their well-being.

The population of children with AIDS has been described as bimodal. (9) There are those who develop severe disease in the first year or two of life and those who survive for several years before getting sick. (9-11) The high mortality of children under the age of two is not surprising given what is known about the development of the immune system. Few long-term studies of HIV+ children have been published, but those that have indicate a group of children who get sick later in life, while most remain stable or improve. (9-11) Children with AIDS in Africa, as their adult counterparts, have dissimilar disease processes, and their symptoms can easily be explained by the infections and malnutrition which are widespread. (12,13) **Many diagnoses of AIDS are made clinically in Africa using the WHO clinical case definition. In one study, it was seen that less than half of the children who met these criteria were HIV+.** (14)

All cases of AIDS in children, as in adults can be explained by factors other than an infectious agent. (6) **In addition, there are many cases of HIV+ infants who remain healthy** (9-11) **and HIV- infants who become sick in the same manner as HIV+ infants seen in the same studies**. (11,15) Young hemophiliacs with AIDS have more in common with other hemophiliacs than AIDS babies - they do not show the same pathologies as pediatric AIDS
patients and their sickness stems from the immune dysfunction associated with hemophilia and its treatment, as has been clearly shown elsewhere. (16)

It is clear that AIDS cases in children other than hemophilacs are due to one of four things (notwithstanding a role for real congenital infections such as syphilis, cytomegalovirus or toxoplasmosis):

1) Maternal drug use
2) Malnutrition
3) Poor environmental conditions
4) Aggressive postnatal drug treatment of the child

1) The use of legal or illicit drugs prior to or during pregnancy is known to have profound effects on the mother and the fetus. (17-25) These drugs include, but are not limited to, cocaine, heroin, methadone, methamphetamine, alcohol, tobacco and AZT and other "anti-retroviral" treatments (26-27). The majority of HIV+ mothers in American and European studies are admitted drug users (11,28-30) (and it is clear that many addicts will deny drug use to their doctors). (21) The majority of the characteristics of AIDS babies in the West can be directly attributed to drug use, particularly low birth weight (21,23,25), short gestation

(17,21), failure to thrive (17,24), impeded neurological development (17,22), diarrhea (21-22,24), increased susceptibility to infection (24-25) and congenital abnormalities (the so-called "HIV dysmorphic syndrome.") (18,21,29) It has been shown that while maternal HIV status has no effect on perinatal outcome, maternal drug use has a profound negative effect in studies controlling for both. (31-34)

2) Malnutrition is widely acknowledged as the most common cause of acquired immune deficiency worldwide. (35) Protein-energy malnutrition and associated micronutrient deficiencies have been clearly implicated in a variety of impairments in immune function. (36-37) The "epidemic" of pediatric AIDS in Africa and Asia is a chimera based on ignorance, racism and politics. (13,38) The children with immune deficiency on these continents are suffering in the same way and from the same diseases that people have been suffering from for centuries, (13) and it is seen that AIDS cases have a seasonal bias that correlates with times of lowest food availability. (39) **Many studies show that it is impossible to distinguish an AIDS case from a non-AIDS case without antibody testing.** (12-13,40-41) This lack of difference underlines the fact that factors other than HIV are responsible for AIDS without even discussing the serious problems of cross-reactivit on HIV antibody tests in Africa. (13,42) In the West, malnutrition plays a major role in childhood AIDS, either alone or more commonly in combination with drugs and the child's environment. (43) Many nutritional deficiencies leading to immune dysfunction have been characterised in children with AIDS. (43-44)

3) Probably the least recognised factor in the etiology of pediatric AIDS is the child's environment. Poor socioeconomic conditions can lead to drug problems and malnutrition and can themselves have serious adverse effects on health. Neglect and abuse of children can lead to illness and impaired immune function. (45-47) The application of maternal touch or therapeutic touch have been shown to positively affect behavioural and physiological parameters. (48)

4) The use of AZT and other toxic drugs to treat "HIV infection" in children is a particularly disturbing phenomenon. AZT has been associated with a plethora of deleterious, potentially fatal, effects in adults and children. (49) AZT directly suppresses the immune system and has been shown to cause many of the conditions associated with AIDS in children including neutropenia, (49-51) anemia, (49) decreased CD4 counts, (52) wasting (30) and heart damage. (53) **Recently, a trial with protease inhibitors in children showed no clinical benefits and numerous severe side effects.**(54)

Conclusions

Given the above mentioned information, it is clear that the scientific and medical approach to AIDS in general – and pediatric AIDS in particular – needs to be re-evaluated. It has long been necessary to alter the infectious paradigm to researchers and doctors in this field have been labouring under for so many years. This is not only because of its scientific inaccuracy but also because of its sweeping effects on the medical treatment of those involved.

The most important clinical priorities to emerge from this situation are those which require a restructuring of medical practices with regard to pediatric AIDS patients. The following dangerous and unnecessary practices based on an inadequate understanding of a presumed viral infection should be halted immediately:

1. Cesarian sections for HIV+ mothers;
2. The prohibition of breastfeeding by HIV+ mothers; (9,55) and
3. The prescription and trials of AZT and other toxic drugs.

Instead, the underlying causes of immune deficiency in these children should be addressed and finances moved towards supporting the health of these infants rather than exposing their immune systems to further damage.

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THE GREAT NUMBER GAME: HIV-AIDS

by Vladimir Koliadin, Kharkov, Ukraine

Has transmission of HIV ever been proven rigorously?

The widely held belief - that AIDS is contagious and HIV is transmissible - rests on CDC's reports, epidemiological observations, and some studies devoted to establish "networks of HIV transmission". Interpretation of these observations and studies as indisputable proofs of transmission of HIV is based on several methodological flaws.

One source of confusion is that the CDC report cases of AIDS by "exposure categories": "Men who have sex with men", "Injecting drug use", "Heterosexual contact", etc. For most, it sounds as if the very fact of transmission and its route had been established by rigorous scientific methods. Nevertheless, this CDC's classification has nothing to do with any proofs of transmission - the exposure categories are ascribed on the basis of questionnaires, and the very procedure of classification is based on the assumption that AIDS is caused by HIV, and HIV is transmissible [1, p.22-23].

Another sort of "proofs" rests on observation that rates of HIV+ individuals are much higher in the specific groups of population with high risk of AIDS - mainly male homosexuals and injecting drug users, than in healthy heterosexuals [2]. Such "proofs" seem to ignore a basic principle of the evidence-based science - that the case and control groups should be attached in respect to all factors other than that under investigation (i.e. HIV+, in this case). Because homosexuals, injecting drug users, promiscuous heterosexuals differ from general population in many respects, such observations prove nothing but that positive tests for HIV antibodies are caused by some factors which are more frequent in the "risk-groups" than in the general population.

One more source of confusion are the multiple studies which report that "sources of infection were identified" - that is, an HIV+ or high-AIDS-risk individual was found among the sexual partners (or donors) of an AIDS patient. Such "identification" rests on the assumption that HIV is transmissible. This sort of findings cannot prove transmission without rigorous proof of their statistical significance - i.e. that such results cannot be explained by random coincidence.

The are several frequent logical errors with estimation of statistical significance in studies carried out be mainstream AIDS science.

1. Selection bias of AIDS patients are reported among whose partners (donors) at least one HIV+ of "high-risk" individual had been found. Such omission of negative findings results in false statistical significance. Studies [3,4] carried out by the CDC provide a typical example. Results of these studies are considered as indisputable evidence of transmission of HIV via blood transfusions [10]. **Only 7 AIDS-patients out of 18 under investigation [3] and 24 of 143 [4] were reported to have at least one "high-risk" or HIV+ donor. Nevertheless, the results were presented as if "identification" had been successful in 7 of 7 [3] and 24 of 24 cases [4]. The remaining cases were omitted on the basis that donors investigations "had not been completed" (equivalent to "HIV+ or high-risk donor(s) had not been found").**

2. Improper choice of the "base rate" of HIV+. The rate of "high-risk" or HIV+ individuals among partners (donors) of AIDS-patients is usually compared to the rate in healthy

heterosexuals from general population. If the former is significantly higher than the latter, the findings are reported as proofs of transmission. This is another typical violation of the principle of matched control. **The rate of HIV+ depends very much on the place of living, lifestyle, ethnic and racial group, income, etc. [5]. As long as AIDS patients and their partners (donors) are from endemic regions, and belong to ethnic, racial, lifestyle groups with higher rate of HIV+, the choice of the average national rates of HIV+ almost inevitably leads to false statistical significance.**

3. Another sort of bias stems from inclination of the researchers to find "at least one" HIV+ individual among partners or donors of AIDS patients (see for example [3,4]). When one such partner is found, the further search is either discontinued or at least carried out with much lower persistence. This reduces drastically the chances of finding more than one HIV+ partner (donor) for an AIDS patient, even if he/she has had actually more than one HIV+ or "high-risk" partner. On the other hand, just the presence of more than one HIV+ partner would have reduced statistical significance of the finding of „at least one partner" as an evidence of transmission. Thus, such sort of bias inevitably leads to overestimation of the statistical significance.

For many categories of AIDS patients, networks of transmission were not reported at all -- even with non-rigorous proofs of statistical significance. For example, high rate of HIV+ in homosexual men (up to 50-70% [2,5]) and great number of sexual partners make any finding of the hypothetical "networks of transmission" highly insignificant statistically: due to pure chance, among tens of partners at least one will be HIV+ with probability close to 100% (see [6], p.84-85 for details). This is also the case for injecting drug users, among whom rates of HIV+ amounts to 30-70% [2,5]. Networks of transmission have never been established for hemophiliacs - who receive blood factors derived from blood of hundreds and thousands donors. The widely publicized attempts [7] to prove transmission of HIV through invasive medical procedures, have also been shown to be inconclusive [8,9].

Conclusions

Although transmission of HIV is widely accepted as the ultimately proven fact, it has never been proven by rigorous methods. The belief in transmission of HIV rests on several logical flaws in interpretation of some epidemiological observations and studies. **If HIV is actually transmissible, it is highly unlikely that this fact had not been proven rigorously for more than a decade of intensive research, especially taking into account the immense intellectual and material resources consumed by the mainstream AIDS science. This striking fact seems to have a natural explanation: "transmission of HIV" is nothing more than a speculation.**

Expansion Of The Definition Of AIDS In 1993: A Vital Necessity Or Dangerous Trickery?

In December 1992 CDC published a new surveillance definition of AIDS in which the range of the health conditions classified as AIDS was expanded dramatically [11]. Now 200 T-cells per micro-liter of blood or less, cervical cancer, recurring bacterial pneumonia, or any of the 25 other previously old diseases in the presence of "HIV antibodies" are called "AIDS".

Tens of thousands of asymptomatic HIV-positives in the USA became "AIDS patients" on this formal basis. Such re-definition of AIDS boosted the numbers of new cases of AIDS reported in the US national statistics 2-2.5 times. **After the purely formal re-classification into AIDS patients, many of the asymptomatic HIV-positives were put on medication by the toxic drugs normally used to treat AIDS.**

The study was focused on the following questions: Did the asymptomatic HIV-positives actually gain from the purely formal change of their medical status in 1993? How did the expansion of the definition of AIDS affect dynamics of the epidemic of AIDS reported in the national statistics? What is the actual trend of the epidemic of AIDS in the USA?

Direct analysis of the CDC's computer data set [12] revealed some interesting details related to the expansion of the definition of AIDS. These details are not well known to the public and even to most scientists, who rely mainly on the aggregated numbers reported in the US national AIDS statistics and scientific papers, but not on the initial statistical data themselves. As long as the data set [12] represents the picture of the AIDS epidemic in the USA more or less adequately, the following conclusions may be made.

1) The cases of AIDS'93 - i.e. the conditions which meet only the new 1993 definition of AIDS but not earlier definitions - showed about 4-8 times lower rates of mortality than cases of the classical AIDS in the period preceding 1993. At that time, these individuals were classified and treated as "asymptomatic HIV-positives", not as "AIDS patients" yet.

2) The purely formal re-classification of asymptomatic HIV-positives into "AIDS patients" in late 1992 - early 1993 coincides in time with abrupt growth of mortality in this group. During a year annual mortality rate increased 2-2.5 times. This trend is conspicuous in individuals whose onset of AIDS'93 was established retrospectively in 1989-1992, but absent for the patients with classical AIDS diagnosed at the same time (i.e. in those who were not affected by the expansion of definition of AIDS in 1993).

3) This upward trend in mortality does not depend on the time elapsed since the official date of diagnosis, but only on the calendar time itself. This peculiarity testifies that this increase in mortality in 1993 was not due to a natural course of "HIV-infection", but was caused by some external factors. These factors started to exert their influence in late 1992 - early 1993, and affected only the AIDS'93 group.

The above three observations are in perfect agreement with predictions of the heterodox hypotheses shared by many AIDS dissidents -- that it is not HIV but just the toxic drugs used to treat HIV-positives and especially AIDS patients that cause the gradual deterioration of natural defense systems and development of full-blown AIDS in some proportion of these individuals [13-16].

The following observations are related to the essentially misleading method used by the CDC to present information on the epidemic of AIDS to the public and policy makers.

In spite of the dramatic difference in the severity of AIDS'93 and the classical AIDS, the CDC reports the new cases of the classical AIDS and AIDS'93 taken together. No explications of this fact were given to public and policy makers.

If considering only the cases of classical AIDS, the incidence of AIDS in the USA reached its peak in the first half 1992. Since then, the incidence has been declining rapidly. During a year the incidence of the classical AIDS reduced to its level observed 4 years before the peak had been reached. Thus, AIDS incidence declines far more rapidly in the 1990s than it was growing in the 1980s. In the first half of 1995, the number of new cases of the classical AIDS were twice as low as the peak level observed in 1992.

The downward trend in the incidence of AIDS was disguised by inclusion of AIDS'93 cases in the official AIDS statistics. In the beginning 1993, only 55 percent of the officially reported new cases of AIDS met pre-1993 definitions of AIDS (i.e. were cases of the "classical AIDS", in our terminology); in the second quarter 1995 -- less than 40 percent. Thus,

implementation of the new definition of AIDS helped to conceal the actual downward trend of the AIDS epidemic as well as to boost the numbers of newly registered cases of AIDS 2-2.5 times.

Conclusions

All these findings considered, the following hypothesis might be set forth. **Main reasons to expand definition of AIDS in 1993 were direct budgetary and commercial interests of the AIDS establishment, not the health of HIV-positive individuals. Expansion of the definition of AIDS helped to conceal the fact that the AIDS epidemic is declining rapidly in the USA.** As for the HIV-positives who became "AIDS patients" only due to the new definition of AIDS, this purely formal reclassification resulted in a significant increase in their mortality rates. Iatrogenic hypotheses on AIDS pathogenesis explain these observations quite well. **According to these hypotheses, just the toxic drugs used to treat AIDS patients, not HIV itself, are the main cause of the deterioration of health and high mortality observed in AIDS [17].**

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PREVENTION THERAPIES FOR „HIV POSITIVE" INDIVIDUALS

Summarised by Felix De Fries, Zürich, Switzerland, from papers by Heinrich Kremer MD, Gerhard Orth PhD, Alfred Hässig MD

The multiple immune deficiency disease called AIDS is a consequence of persisting inflammatory reactions in order to resist external pathogens, simultaneously decreasing internal control and removal of aged cells and pathogenic agents. This allows pathogens to multiply. At the same time, increasingly released „self" enveloped proteins are embodied in enveloped viruses, e.g. hepatitis viruses. Consequently, antibody production is activated which ultimately, by the HIV antibody test is detected as specific antibodies against a so-called HIVirus. This although, the proponents of the HIV-AIDS-hypothesis have not yet managed to isolate HIV and showed it to be an infectious agent in organs and blood preparations.

Persistent inflammation reactions are caused not only by ongoing psychic and toxic stress but certainly also by a persisting oxidative stress, as it occurs with the mitochondria damage of their DNA structure. Mitochondria, an early unicellular organism, in a symbiotic relationship with human cells mutate oxygen for „nutritive supplement" from respiratory air in order to detoxify and supply cells with energy. A weakening of the genetic structure results in immune and organ failure especially in organs with raised energy consumption (brain, muscles, lymph tissue, liver, kidney, bowels and lung). As toxic oxygen disintegration can not longer be sufficiently dissolved bacteria multiply causing a switch to a oxygen free metabolism. Phagocytes release inflammatory agents. These induce stress hormones which drive the resistance to external infectious agents to the point where auto-immune reactions develop. These auto-immune reactions identify the body's own structures as foreign and attack them. The daily internal scavenging of aged cells which show retroviral components of the genetic material at their surface, e.g. HI retrovirus, is strongly restricted.

New antibiotics (aminoglycosides, Monobactames, Quinolones, chloramphenicol, glycopeptides, polymyxides, sulfonamides, Trimethoprim, tetracylines and antimycotica such as Amphotericin, Ketoconazol and Nystatin) damage the DNA of benign bacteria and fungi (Toxoplasma and Pneumocytis) which then mutate to aggressive, antibiotic resistant pathogens. As nucleoside analogues (AZT, ddI, ddC and 3TC) affect the unprotected genetic structure within mitochondria, therefore the energy supply within the body is no more guaranteed. Consequently, viruses that embody „self" enveloped proteins can no more be eliminated or hindered in their mutation.

Kidney, liver and pancreas dependent on a raised protease formation are harmed by synthetic protease inhibitors (Invirase, Indinavir, Ritonavir a.o.) which should inhibit the rapid multiplication of so-called HIV particles. Already after a short time they lead to drug treatment resistance and the number of s.c. viral particles is increasing again.

Preventive Treatment

- Natural occurring protease inhibitors (heparines and heparinoides from sea weed, kelp and spirulina), which activate „self" antiproteases can slow down a persistent inflammation reaction with increased cell division.
- Plant antioxidants (e.g. the effective Tibetan herbal mixture PADMA 28) are capable of binding toxic oxygen free radicals and promote their scavenging and, therefore, correct the weakened activity of mitochondria caused by persisting oxidative stress as well as continuing inflammation reactions (e.g. hepatitis).

- The co-enzyme Q10 (e.g. Q-Min Q10) can ameliorate the electron transport in the respiratory chain which stimulates the crucial energy supply within cells.
- N-Acetyl-Cysteine (e.g. NAC Ratiopharm) can ameliorate the formation of glutathion molecules which are essential for the energy supply to cells.
- If not too much progressed mitochondrial DNA repair can be supported by folic acid and low doses of selenium (contained in brewer's yeast) and zinc.
- Liver activity can be activated f.e. by thistle (Mariendistel).
- The intestinal flora can be built up by lactatic-acid containing drinks (e.g. Kane Brottrunk)
- Bacterial and fungus infections can be treated with pineapple extract (Citricidal) and locally by gargling with apple vinegar-honey.

Persistent inflammation reactions can be avoided by:
- specific reduction of stress (autogenic training, stretching)
- refraining from frequent drugs in order to shift psychic and physic efficiency (coffee, alcohol, nicotine, heroin)
- refraining from chemical drugs (cocaine, poppers a.o.)
- avoidance of inflammation and injuries
- a healthy respecting of „safer-sex rules"
- an utterly sugar poor and bulk rich nutrition containing whole carbohydrates, plant antioxidants (vegetables, curry (curcumine), herbal and green tea), virgin oils, lactate products, soya protein, a sufficient supply of trace minerals and no iron rich nutrition.

The success of this prevention and immune system support therapy can be detected by measurement of the stress hormone profile, the T4/T8-cell ratio, the macrophage activation (Neopterin Test) and cutaneous anergy test.

Further information can be obtained at the following address:

Study Group for Nutrition and Immunity, Elisabethenstrasse 51, 3014 Bern, Switzerland

CONCLUSIONS

Based on the tremendous work done by only a few scientists, some of their findings summarised in this document, IFAS concludes the following:

- Critical analysis of crucial data strongly indicates that „AIDS" does not behave at all like a sexually transmitted disease and sexual activity is not the crucial factor to explain the clinical condition called „AIDS"
- Scientific proof demonstrating *in vivo* how „HIV" causes „AIDS" does not exist.
- To date there has been no isolation of the „Human Immuno-Deficiency Virus", according to the Pasteur rules and therefore no proof of „HIV"'s existence.
- A valid „AIDS-„ or „HIV-„ or „HIV-antibody test" does not exist.
- The labelled „anti-HIV antibody tests" do not show infection with a novel retrovirus.
- All current treatments -- namely pharmaceutical nucleoside analogues and protease inhibitors -- are dangerous due to their high toxicity and should be banned as so-called „anti-HIV-treatment"

For these reasons IFAS places the following requests to both national and international legal and/or health bodies/authorities:

1) Grant immediate access to non-toxic treatment options for all diseases classified as or associated with „AIDS" in the presence of an „HIV-diagnosis", for all people labelled „HIV positive" and/or having „AIDS", regardless of their „HIV-status".
2) Demand an immediate halt to all „HIV-therapies" such as nucleoside analogues (AZT, ddI, ddC etc.), pharmaceutically-produced „HIV protease inhibitors" and any other treatment to be introduced based on the assumption that „HIV" causes „AIDS", until the mechanisms of chronic immune dysfunction are fully understood by documented scientific standards, justification for the use of possible treatments are fully established, and possible side-effects are understood and documented in terms of overall risk-benefit ratio.
3) Request the immediate application of the standard protocols for virus isolation as established by the Pasteur Institute in 1973, using pure cell cultures from „AIDS-patients" and suitable controls (patients without AIDS diagnosis, yet similar disease: oxidised and immune deficient), with absolute secrecy concerning risk status of the former to avoid bias in results interpretation (conducted blindly).
4) Urge an immediate cessation of use and thorough reappraisal of all so-called „AIDS tests" (ELISA, Western Blot, PCR, etc.) to check for validity and specificity, accuracy of diagnosis and standardisation to international „gold standard" level. If „HIV" can not be identified as a virus, we request the name „AIDS" to be no longer referred to in order to stop further confusion and panic.
5) Carry out studies which evaluate the health and well-being of so-called „long term survivors", people who have tested „HIV positive" some years ago and remain healthy in the absence of any conventional treatment.
6) Carry out studies which evaluate the relation between a so-called „HIV positive diagnosis" and being at higher risk for illnesses, taking in account all existing implicative data.
7) Initiate a full reappraisal of the whole „HIV-AIDS"-hypothesis, by an international, independent scientific committee, having due regard to qualified scientific dissenting opinions and supported by appropriate data.
8) Publicly address and disclose all previous errors in „AIDS-research", with no concealment of scientific misconduct where it can be proved.
9) Provide full access to legal representation for possible litigation claims to all individuals suspected of having been damaged by the consequences of the „HIV-AIDS" dogma.

IFAS, in conjunction with other concerned organisations, will take all necessary steps to make sure these requests are heard, taken in account and responsibly followed.

IFAS will furthermore assist individuals and organisations working in the direction of the steps outlined herein in getting their voice heard, according to the resources of our organisation.

Michael U. Baumgartner
Secretary General IFAS
Geneva, Switzerland, March 1998

Amendments to these conclusions and requests may be made upon occurrence of new information.

IFAS

ACCESSING SCIENCE

The ***International Forum for Accessible Science, IFAS,*** based in Bern, Switzerland is an independent world-wide body of scientists, workers for the public good, human rights activists and organisations concerned with the following principle which constitute the aim of ***IFAS***:

To make unbiased science in general and medical research in particular accessible to the concerned public.

Mission-Statement

Health and health care must not to be controlled
by profit-oriented and other inappropriate interests,
and so may not be dictated
by market forces or exclusively controlled by government.

Academic and industrial medical research
with possible public health and/or treatment benefits
and policies for health maintenance
must be subject to a public discourse
to carry the authority of public confidence.

This should also apply for any such data about results of research
which are relevant to the formation of public opinion
on health risks, disease prevention, treatment possibilities and health care policies.

IFAS was founded 1997 on the initiative of Michael Baumgartner and the support of a few people from the fields of science, human rights, journalism, communication and education. Mr. Baumgartner saw the need for an umbrella organisation uniting the dissident views on „HIV" and „AIDS" and at the same time connecting accessible science with human/patient rights. ***IFAS*** is headed by the ***Board Of Scientist (BOS)*** and the ***Public Access Board (PAB)*** and operated by the ***Secretary General***. The two governing boards are chaired by the distinguished Mrs. Eleni Papadopulos-Eleopulos, Biophysicist from Australia (BOS) and the distinguished Ms Karen Parker, Human Rights Attorney from the US (PAB). Mr. Baumgartner acts as ***IFAS*** first Secretary General.

United ***IFAS*** aims to help those mostly affected by the outcome of biased scientific research.

IFAS has officially approached the organisers of the *World AIDS Conference 1998* in Geneva, Switzerland. The request for an appropriate forum for the dissident voices on „HIV/AIDS" to address the issues of „HIV's non-isolation" and „AIDS-causation" has been placed. During the World AIDS Conference in Geneva (June 26th to July 3rd) ***IFAS*** prepares a supplementary symposium („*Concluding AIDS*") for a closer look at the 17 years of „AIDS".

For information contact:
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„The most important breakthroughs today often do not happen where people intend them, but where individuals work anyway"
(M. Baumgartner)

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It is important that we do not remain silent, when there is so much harm caused by the "HIV-AIDS" myth!!!

WE WILL CONTINUE!!!

Michael Urs Baumgartner
Geneva, Switzerland; March 1998

LIST OF LITERATURE

- ***What If Everything You Know About AIDS Is Wrong*** *by Christine Maggiore, HEAL Los Angeles (us)*
- ***Positively False*** *by Joan Shenton, IB Tauris (engl.)*
- ***Lieben Wir Gefährlich*** *by Christian Fiala, Deuticke. (austrian)*
- ***AIDS And Stressors*** *by Roberto A. Giraldo, Fundacion Arte Y Ciencia (colombian/us)*
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PUBLICATIONS/ORGANISATIONS:

- ***CONTINUUM***, quarterly international AIDS-magazine, 172 Foundling Court, Brunswick Centre, London WC1N 1QE, England,
- ***REAPPRAISING AIDS***, monthly news-update, 7514 Girard Avenue, 1-331, La Jolla, CA 92037, USA
- ***Zenger's***, monthly periodical, P.O.Box 50134, San Diego, CA 92165-0134, USA
- ***MuM,*** PIROL-Verlag, Postfach 1210, 85066 Eichstätt, Deutschland

For videos on the subject contact:

- ***MEDITEL***, 172 Foundling Court, Brunswick Centre, London WC1N 1QE, England,

To order the easy reading booklet ***What If Everything You Know About AIDS Is Wrong*** *by Christine Maggiore* write to:

HEAL (Health-Education-AIDS-Liaison) 11684 Ventura Boulevard Studio City, CA 91604, USA

HEAL chapters exist in different cities in the US, Canada and Germany. Information is provided in the booklet.

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How the „HIV-AIDS" dogma is endangering women's lives

(Agenda item 5 „women")

Presentation to the UN Sub-commission on Human Rights by Michael U. Baumgartner; Humanitarian Law Project (International Educational Development) on August 14th, 1997.

Mr./Ms. Chairperson

International Educational Development is extremely concerned about the situation of women especially from African countries, who are considered to be carriers of what has become to be known as „HIV". Although we address other specific issues related to „HIV" and „AIDS" under other agenda items, this statement is not about discrimination of people labeled „HIV positive", but about the right to life, liberty and **security** (Universal Declaration of Human Rights, art. 3) of women and children in danger because of forced or involuntary medical treatment, having no medical justification, violating article 5 of the Universal Declaration of Human Rights: „No one should be subject to torture or **cruel**, inhuman or degrading **treatment** or punishment". We are especially concerned that after the US and Europe, now women and children from African countries are exposed to governmentally funded trials with the extremely toxic substance azidothymidine (AZT), based on unsound scientific work. It came to our attention that the governments of Zimbabwe and South Africa are looking into the options of such trials. In view of the facts being outlined, we would strongly advice against that. Besides a tremendous amount of psychological distress caused by the finger pointing effect of an „HIV-positive" diagnosis, it poses the great threat of possible iatrogenic - meaning medically induced - damage, even death! Since women and children especially those in economically deprived circumstances, have less access to appropriate information about the toxic effects of AZT, they must be considered especially at high risk.

In 1984 the „Human Immunodeficiency Virus" („HIV") was declared the „probable" cause of the „Acquired Immune Deficiency Syndrome„ („AIDS") by Dr. Robert Gallo. This announcement was made without prior publication of the data or debate in the scientific community. Shortly after, the word „probable" was dropped. The media simply stated what today is common belief, „HIV=AIDS", without any such proof. Rather than on scientific facts, important treatment decisions were made based on this belief.

Critics of this hypothesis have personally invited the main proponents of the „HIV-AIDS"-hypothesis to submit scientific papers that would demonstrate how „HIV" causes „AIDS". To date no such paper has surfaced. We expect the scientific community to demonstrate the mechanisms by which a disease is caused, in order for the right treatment approach to be developed. However, when reminding them about the outstanding data to back up their claim, we were either harshly criticized for insisting on getting an answer, or we were asked

to take the overwhelming" epidemiological data as such proof. For simple scientific reasons this is not acceptable.

Not only was Gallo not able to isolate what was considered „HIV" in more than 26 out of 72 of his initial „AIDS" patients, but thousands of patients with clinical „AIDS", who are not found to be „infected" with „HIV" clearly prove, that whatever causes a chronic immune dysfunction can do so with or without „HIV" present. Just because something is present in a diseased person's body, does not mean it actually causes the disease! This would be like saying the witness present after a car accident happened actually caused the accident! Before the inaccurate „HIV-AIDS" hypothesis, research had a more scientific approach in identifying a disease causing agent. Even so 39% of the „AIDS"-defined diseases are not due to immune deficiency - meaning low immune cell count - and can therefore not be attributed to „HIV" killing those very cells. About 30 unrelated disease were taken together to create an epidemic, based on no more than „common belief"?!

„HIV=AIDS" became a dogma. Anybody questioning it was silenced, ridiculed or considered irresponsible and therefore of great danger to human life. It was just that dogma, however, that led to the use of toxic drugs with the misleading label „antivirals", like AZT and its successors. Today these drugs are favorably given to pregnant women and their infants. In African, the claimed epicenter of „AIDS", where „HIV" is supposed to have originated from, especially women and children are now in danger of getting killed by AZT treatment.

The conventional thinking asserts that „AIDS" in Africa is somehow explicable by Africans' sexual predilections. Such insinuations merit close scrutiny since generalizations about African sexual practices are analytically useless for an internally diversified continent of 650 million people. The statement made by Dr. Yuichi Shiokawa at the 10th international conference on AIDS in Yokohama, claiming that AIDS would only be brought under control if Africans restrained their sexual cravings. This demonstrates well the view in which the sex-negative Judeo-Christian culture sees the „AIDS" problem in Africa and people from African countries, despite studies showing that e.g. Ugandan sexual behavior is not different from let's say, North American sexual behavior. A letter in the scientific journal The Lancet cited a passage from Lili Palmer's memoirs as evidence for how a large male chimpanzee's „anatomically unmistakable signs of its passion for Johnny Weismüller" on the Tarzan set in 1946 „may provide an explanation for the inter-species jump" of „HIV"!? Ms./Mr. Chairperson, this is the racist attitude that upholds this myth of „HIV's" origin. Again it is mainly put in the hands of women to take the necessary preventive measures to solve the „AIDS" problem in Africa. The pressure put on women, who in many parts of Africa are the main family supporter, is crucial and indicates in our view the patriarchal notion of „blaming the victim".

While the political authorities continuously fail to protect the most vulnerable members of society like mothers and children, the medical authorities violate the highest medical standard, the Hippocratic oath, by prescribing the drug AZT, discredited on several accounts. Based on unsound unscientific assertions and fraudulent drug licensing processes women get exposed to treatment with AZT as a presumed way of preventing the suspected transmission of „HIV" from mother to child. The concluded treatment with AZT proves especially dangerous for pregnant women.

The biochemical mechanism of AZT - also known as zidovudine and Retrovir - is extremely simple; it terminates the making of DNA, the basic life process. It was once wrongly claimed, based on research conducted by Burroughs Wellcome, the manufacturer of the drug, that AZT only attacked viral DNA synthesis, leaving the human cellular DNA synthesis untouched. This claim has since been proven false by at least a half dozen independent studies, which found that AZT is up to 1000 times more toxic to human cells than was

claimed when the drug was approved for marketing in 1987. AZT cannot distinguish between human cellular DNA and retroviral DNA, and is thus by its very nature a killer of human cells.

Scientists have stated that damage to the bone marrow is not the worst of AZT's toxicities. In 1991 a team of Japanese researchers showed that the significant weakening of the skeletal muscle - until 1990 considered a damage caused by „HIV infection" - originates from the damage caused to the mitochondria of human cells by AZT treatment. During the following years the toxic effects of nucleoside analogues in the treatment of viral diseases have been thoroughly studied. The multi organic toxicity of these drugs was demonstrated on the heart muscles, the brain and nerve system as well as on liver and pancreas. In addition it has been demonstrated that successors to AZT, such as ddI and ddC cause the same mitochondrial damage. Since AZT can directly cause several of the "AIDS-indicator diseases" and can indirectly contribute to causing most of the others - it logically follows that AZT is iatrogenically - medically induced - „AIDS", when administered to asymptomatic "HIV-(antibody)-positive" individuals.

AZT is tremendously toxic to fast-replicating cells, like those in fetuses and infants. That puts women and children at a special risk of such iatrogenic damage - including possible birth defects - or even death! We know today that only about 50 % of the infants born to „HIV positive" mothers test positive at birth themselves to begin with. Studies carried out show furthermore that the fast majority of the infants testing „positive for HIV" at birth sero-convert within about two years, without any therapeutic intervention at all, including AZT. That leaves only a small number of babies remaining „HIV+". There is a tremendous problem, however, with the consideration of „HIV positivity".

The French Pasteur Institute had outlined the steps for retroviral isolation a decade before coming up with a new retrovirus, Lymphodenopathy Associated Virus (LAV) from a **non**-AIDS-patient, the virus later to be renamed „Human Immunodeficiency Virus" (HIV**), without following their own protocol**. Independent research since has shown that up to March of this year none of the claimed papers on „HIV" actually show an isolation of „HIV". Not only has neither Gallo nor Montagnier followed the rules designed for retroviral isolation but **there is a disagreement between Gallo and Montagnier on which proteins should actually be considered „HIV"**. These documented facts call to question all currently used falsely called „AIDS tests". The only way to prove that specific antibodies to „HIV" exist is to compare antibody reactions with the isolated „HIV". If these antibodies are specific, they will only occur in the presence of „HIV" and never in its absence. Such an experiment has never been reported and to date could not be performed because no one has provided evidence for the isolation of „HIV".

Verified stories have surfaced whereby mothers in Europe and the US get threatened to lose custody over their newborns if they refuse immediate toxic treatment with AZT. A mother's children got taken away from her, because her ex-husband exposed her „HIV status". Being labeled „HIV positive" made her an unsuitable mother. African refugee mothers shortly after immigration to England were enrolled in AZT studies, without having had sufficient knowledge about the effects of the drugs. While told being enrolled in a drug trial would protect them from a possible deportation, some mothers claimed that they did not know the syrup given to their children contained AZT. The toxic effect many children suffered from was attributed to „HIV" rather than the well documented detrimental effects of AZT. Many of the mothers and the children in discussion are clinically „asymptomatic", meaning healthy; however, due to another change of the AIDS definition in 1993, considered as having „AIDS" in order to give them AZT. The longest and largest study - the Anglo-French CONCORDE study - carried out by Wellcome to prove the benefits of giving

AZT to people who are asymptomatic, showed that there not only are no benefits of early AZT administration but even a higher mortality in the group of asymptomatic individuals taking the drug compared with those in the placebo group and should therefore not be given to simply „HIV positive" individuals without „AIDS". By shifting the goal post again, these women and children are now suitable candidates for being poisoned with AZT. Ms/Mr. Chairperson, is the scientific community given a license to kill?

Both pharmaceutical industry and registration authorities should have been obliged to consider the documented damage caused by administration of nucleoside analogues as well as to prove that the mortality among AIDS patients is not connected to this drug treatment. In general, these obligations have been avoided and now create a serious new problem: Justified litigation claims and charges of intentional grievous bodily harm and homicide.

Health authorities world wide gave in to the pressure of lobbying groups and media propaganda without carefully checking references, clarifying the mounting evidence of the inaccuracies of the „HIV-AIDS" hypothesis.

We urge the health authorities to do so now by asking national AIDS authorities to submit scientific papers which

- show „HIV-isolation" using the standard Pasteur protocol and
- demonstrate how „HIV" causes „AIDS".

The document on which this presentation is based shows that „AIDS" can be attributed to other risk factors independent of „HIV".

While we are waiting for an accurate response to the „HIV" crisis, as outlined in our document, we urge all countries to stop putting people's lives at risk - especially pregnant women and infants - by being exposed to useless even dangerous pseudo-treatments that create much suffering adding to the problems already faced in particular by mothers in economically deprived circumstances.

Thank you Mr./Ms. Chairperson.

[This presentation was based on the first edition of the UN Public Information Dossier prepared and put together by Michael Baumgartner and presented to the UN Sub-Commission on Human Rights on behalf of CONTINUUM, 172 Foundling Court, Brunswick Centre, London, WC1N 1QE, England]

THE MICROBE

The Microbe is so very small
You cannot make him out at all,
But many sanguine people hope
To see him through a microscope.
His jointed tongue that lies beneath
A hundred curious rows of teeth;
His seven tufted tails with lots
Of lovely pink and purple spots,
On each of which a pattern stands,
Composed of forty separate bands
His eyebrows of a tender green;
All these have never yet been seen –
But Scientists, who ought to know,
Assure us that they must be so ...
Oh! Let us never, never doubt
What nobody is sure about!

Hillarie Belloc.
from MORE BEASTS FOR WORSE CHILDREN
(first published in 1897)