Valendar Turner
Do antibody tests prove HIV "infection?"

Montagnier’s ‘HIV’
I repeat, we did not purify!

Martin Walker
Nutritional health resources

changing the way we think about aids
November 1994 issue 34/10
CONTINUUM changing the way we think about aids

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On February 12, 1998 German virologist, Dr Stefan Lanka gave a public lecture entitled "AIDS: the biggest betrayal in medical history" at Imperial College, London, a stone's throw from Saint Mary's Hospital, one of the largest AIDS teaching hospitals in Britain. The event was part of the international "Refuse and Resist HIV-testing" campaign initiated by Continuum and including HEAL New York, HEAL Toronto, C.O.B.R.A. Barcelona, G.A.I.A (Gay International Association) London, and T.R.U.T.H. Int. Florida.

Dr Lanka's two hour lecture was introduced by Continuum Executive Committee member Rafael Ramos. The British AIDS and Gay press were invited as well as science journalists and service users of London AIDS organisations, HIV/AIDS diagnosed individuals and some alternative health practitioners.

With direct experience in viral isolation and in the field of HIV/AIDS, Dr. Lanka gave a scrupulous and well documented account of the principles of genetics, cancer research, cell biology, mitochondrial damage with antibiotics or other AIDS medication and a brief history of the HIV/AIDS scandal and antibody testing. A focal point of Dr. Lanka's lecture was his emphasis on encouraging those living with a positive diagnosis to unite and demand scientific proofs that HIV has been isolated and exists, and that HIV tests can ever accurately diagnose an infection. London's Thud news carried Continuum's Refuse and Resist HIV-testing press release, followed by campaign adverts in Scene UPDATE and Thud quoting UK Virologist Philip Mortimer, Prof. Gordon Stewart and Eleni Eleopulos on the non-specificity of the 'HIV' tests.
Heated press in Spain

Barcelona-based organisation C.O.B.R.A. hosted an extensive conference March 6 to 15 of anti-AIDS analysts and activists, health practitioners and diagnosed people from across Europe in preparation for attendance at the World AIDS conference in Geneva later this year. [A full report of the conference will be in the next issue].

Spanish dissent continues. Since December 2 '96 magazine Más allá de la Ciencia (Beyond Science) - a popular sensationalist investigative publication with a circulation over half a million - has been seeking scientific data including correct photos that prove HIV has been isolated and causes AIDS, from top Spanish AIDS officials and several state authorities, including the Minister for Health and Consumer’s Affairs, the Spanish Secretary for the National Plan Against AIDS, the President of the Spanish AIDS Interdisciplinary Society, the President of the Spanish Medical Research Council and the President of the Council of Pharmacology. On 18 December ‘97 the Secretary of the Medical Research Council, Antonio Entiste finally responded, “We have no data in relation to the documentation that shows that HIV has been isolated as causative of AIDS”. The Spanish Secretary for the National Plan Against AIDS, Francisco Parras Vázquez sent a non-specific photo and some pages from the “Textbook of AIDS Medicine” (eds Broder, Mergian, Bologna) which apparently includes some 550 references. German virologist Dr Stefan Lanka checked the proffered data stating, “This and other books - secondary literature - are used to convince the public that somewhere, there is scientific evidence ..... But no such affirmations have been proved... There are no scientific proofs that HIV exists”. Asked journalist José A. Campoy, “Do the Health Authorities of Spain consider Spanish journalists are idiots? Or as the Spanish Secretary of the MRC implicitly acknowledged, have they no proofs of any kind with respect to what they have been officially affirming for years and were simply rearguuting like parrots?” After further silence, Más allá approached the Spanish Head of State, Juan Carlos I and in October ‘97, His Majesty the King intervened giving orders to the Ministry for Health and Consumer’s Affairs to respond to the petition. November 18, ‘97 a Spanish Deputée in the Lower Chamber of Congress, F. R Rodriguez Sánchez, raised the issues of silence, lack of transparency or of any scientific debate by the Authorities. Más allá after Médicas Holisticas and Diario 16 is the third Spanish publication challenging the HIV/AIDS orthodoxy and giving extensive coverage to dissent views.

UN’s cheaper combos

The United Nations will collaborate with drug companies to make ‘aids medicines’ more affordable in ‘developing’ countries. The HIV Drug Access Initiative’s pilot phase will be carried out in Chile, Uganda, Ivory Coast and Vietnam. Glaxo-Wellcome, Roche and Virco will take part. Merck, Organon, and Bristol-Myers Squibb hope to join later. Subsidy made available by the manufacturers “will be defined by each company”, UNAids said but on some products are expected to be 50% or more. A World Bank report recently warned that it was too expensive to use new anti-HIV drugs in poor countries.

Traditional Ho

Dr David Ho of the Aaron Diamond AIDS Institute, believes more research into traditional Chinese medicine to find treatments for HIV was warranted, according to an article in the South China Morning Post. Dr Ho expressed a personal interest in a “very organised effort” to make such an investigation. He concluded: “I wouldn’t dismiss the use of traditional medicine but I think it has to be studied properly”. Reuters 12 Dec 97

Ugandan medical Prof. Charles Ssali claims his low-cost multi-herbal antioxidant and micronutrient treatment for AIDS called Mariandina was banned after a US$1million payment to his government by AZT producers Glaxo-Wellcome, which he sees as “little more than a bribe”. The joint decision by the NDA and the Ugandan Ministry of Health to ban Ssali’s treatment coincided with the Ugandan/ United Nations AIDS initiative to give cheap access to ‘anti-retroviral’ drugs. Ssali concluded: “I am convinced that the NDA and Ministry of Health have co-operated with drug companies in prohibiting my treatment.” Supporters of Ssali took to the streets in protest, and the authorities are looking at allowing the treatment, prioritising local successes over foreign intervention. Mariandina has apparently significantly helped over 18,000 people in Uganda so far.

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Cocktail Cult Still Popular in Canada

With few exceptions, the North American gay press has been reluctant to question the AIDS establishment in any way. All the more significant is a recent report of the Toronto gay magazine Xtra! that featured an article on "the failure of cocktail therapy." The piece took the form of an interview with the PWA Foundation's primary care director, Derek Thaczuk, who is now taking a version of the "magic bullet" cocktail. The piece took the form of an interview with the PWA Foundation's primary care director, Derek Thaczuk, who is now taking a version of the "magic bullet" cocktail. Thaczuk said that he was going to give up on AIDS and go to a conventional medical approach focusing on medication. The distinction between possibly beneficial and possibly therapeutic effects in the past and present treatment approaches focusing on HIV has been consistently unclear. IFAS agrees that the comprehensive range of information about HIV, AIDS, and treatments necessary to make proper decisions about policies, health care, research and treatment has not been made public. It is therefore IFAS' concern to make relevant information publicly accessible and oppose the long-standing censorship of HIV/AIDS critical voices. Members of IFAS agree to one of the following views about the HIV-AIDS-hypothesis:

1. There is to date no isolation of a retroviral entity called "HIV." Therefore "HIV" cannot be considered the cause of AIDS, thus the current focus of AIDS research and anti-HIV treatments has to be revised.

2. HIV has been cultured, is an infectious, exogenous retrovirus yet is insufficient and/ or unnecessary to cause AIDS, therefore the current focus of AIDS research and anti-HIV treatments is wrong. (c) HIV as an infectious, exogenous retrovirus has the potential with or without co-factors to cause AIDS but the established measurements and treatments are wrong.

3. IFAS has officially approached the organizers of the World AIDS Conference 1998, Geneva for a plenary session on the failure of "HIV" isolation, supported by two of the five sponsor organizations. People who would like to support such a plenary session should address communications to: Guy-Olivier Segond, Chairman Geneva World AIDS Conference 98, Fax +41 22 700 3311

For more information contact: IFAS, c/o Studiengruppe fur Ernahrung und Immunologische For national voices dissenting from an organisation aiming to link the growing number of international voices dissenting from the current scientific approach to HIV and AIDS. The International Forum for Accessible Science was founded on the initiative of Michael U. Baumgartner, a Swiss gay activist and internationally outspoken AIDS analyst, with the support of people from the fields of science, human rights, journalism, communication and education. Baumgartner saw the need for an umbrella organisation uniting the dissident views on HIV and AIDS at the same time as raising issues of human/ patient rights and ethics and bringing scientific standards into a field of medical research that has gone badly astray.

As an independent body of workers for the public good, headed by a board of scientists and a board of non-scientists and run by a Secretary General, IFAS is concerned about the focus of established AIDS science. In the past 15 years the international AIDS establishment has been almost exclusively absorbed researching HIV, with no conclusive model of how HIV is supposed to cause AIDS to date. Thus, it is necessary to question the research and anti-HIV treatments a few years ago to fund gp120 vaccine trials. Science Vol. 279 30 Jan 98
Coalition co-founder in “gross misconduct”

HIV/AIDS group The UK Coalition for People Living with HIV and AIDS received thousands of pounds in fraudulent payments from Brent Council workers and Coalition co-founder Terry White, who was later employed by the so-called Coalition, and whose lover was the director of the organisation, according to London’s mass circulation Evening Standard 6 Mar. The Coalition, called “Britain’s leading HIV lobby group” because its directors have gained direct access to government policy-makers on ‘HIV/AIDS’, in particular the office of ‘HIV minister’ Tessa Jowell, was revealed last year not to be a membership organisation but a limited company with a technical membership restricted to its Board of Directors. The Department of Health explicitly forbids local and national health funding for groups involved in political lobbying. The private, limited “Coalition” is implacably opposed to any investigation of the non-isolation of HIV, or non-HIV causes of AIDS, and with a more than 60% stake in the free full-colour monthly magazine Positive Nation, receives substantial support from drug companies. The new financial scandal, involving payments to London Lighthouse also, was uncovered when Brent’s Special Investigations Unit was tipped off that White had made unauthorised payments to an “HIV agency, and was working while “off sick” for a year. At least one senior Westminster figure is asking when the government will end its dubious and costly association with the discredited company.

Mandatory ‘HIV’ Tests

A community forum to discuss controversial calls for ‘mandatory federal HIV reporting’ was held in the Castro district in San Francisco last October. Eileen Hansen, legal policy director of AIDS Legal Panel, said that the legal referral group was “profundely disappointed” by calls for Mandatory Names Reporting (MNR) and said that the practice was not needed: “The HIV/AIDS community has had a long history of opposition to HIV names reporting, with good reasons ...There’s a planned effort to move this thing along as quickly as possible without generating any more community opposition than they can avoid.” POZ Feb 1998

AIDS downturn

The European Centre for the Epidemiological Monitoring of AIDS reported a decline in AIDS cases to 30 Sept 1997. The decline in numbers includes: Albania: 5 cases in 1995 to 1 in 1997; Croatia: 16 cases in 1995 to 9 in 1997: Estonia: 4 cases in 1995 to 2 in 1997; Iceland: 4 cases in 1995 to 1 in 1997; Israel: 47 cases in 1995 to 39 in 1997; Malta: 3 cases in 1995 to 2 in 1997; Monaco: 4 cases in 1995 to 1 case in 1997; and Norway: 70 cases in 1995 to 21 in 1997. Welt Nachrichten, ‘AIDS in Deutschland’, 27 Nov.97, reported their were only 35 AIDS-deaths in Germany for 1997. HIV/AIDS Surveillance in Europe, Third Quarterly Report ’97, no.5

Junk Science

The US Supreme Court recently affirmed the right of trial judges to disallow scientific testimony they believe to be flawed. The decision may cut down the use of “junk science” in the court room. This will give judges freedom to consider both methodology and conclusions, just as a scientist might in judging the quality of research. This means judges may now have to do more home work in scientific discourse. Nature 1 Jan 98

hiv project closes

The hiv project London will close on 31.3.98. Its Director, James Barrett stated: “Trustees have taken the decision recognising that we have to be realistic about the fact that the climate of funding in the sector is changing significantly”. hiv project letter 19 Jan 98

Family Values

At present anyone can buy Vitamin B6 in shops in Britain. But the government plans to limit its public sale to tiny quantities, on the advice of the Committee on Toxicity of Chemicals in Food (COT), based on just two studies. The author of one has criticised the government’s use of his results. The great majority of research finds nothing unsafe about B6 unless taken in huge quantities. Four points of interest are: (1) B6 is cheap and not very profitable. Its vast sales cause irritation in the drugs industry which sells remedies for the illnesses assisted by B6 at much greater profit. (2) Ten out of the 19 members of COT have declared a personal interest in large drug companies. Four who held an interest in Glaxo or Glaxo-Welcome. (3) The decision to restrict the sale of B6 was taken by, among others, public health minister with responsibility for HIV/AIDS Tessa Jowell, who last year also pledged UK E 23 million extra for state-funded “HIV combination therapies”. (4) Tessa Jowell’s brother, Dr James Palmer, is head of medical and regulatory affairs at Glaxo.

Leading AIDS Centre burns out

Flagship London AIDS-hospice The London Lighthouse is officially on the rocks. Said a widely-reported press release 10 Mar. arch, “We realise the closure of the residential unit and the proposed sale of the Lighthouse building will be a tremendous shock.” Chief Executive Susie Parsons last month denied reports in Positive Nation that her annual salary was in excess of £ 70,000.
The theory of vaccinating against a disease requires that one must find its true cause, and if it is an organism like a virus or a bacterium, a vaccine perhaps may be constructed which will raise prophylactic antibodies to neutralise the bug, should it infect, before it can cause the disease. None of this applies in the case of AIDS and ‘HIV’. AIDS is not a specific disease but a collection or syndrome of some thirty old diseases, the so-called AIDS-virus, ‘HIV’, has never been properly isolated, let alone shown to cause immunodeficiency; AIDS develops only after some ten years of an effective antibody defence against the constellation of some nine or ten proteins alleged to constitute the AIDS ‘virus’, which itself may not be a virus at all. The idea that an AIDS vaccine will be found at the end of the rainbow, like the proverbial pot of gold is merely a crock of shit.

Even the AIDS Junta are sharply divided over the use of an anti-‘HIV’ vaccine. One section are realistic enough to admit that there can never be a vaccine. The other group want to waste time and treasure in raising hopes whilst lining their own pockets. This latter group are getting ready to attempt the old vaccine scam. According to Nature (15.1.98) San Francisco-based veteran AIDS researcher Don Francis is embarking on a Phase III trial using some 7500 “healthy” volunteers in the USA and Thailand in a $20 million three year project using a vaccine based on glyco-protein 120, an alleged component of ‘HIV’; John Moore, of New York’s Aaron Diamond AIDS Research Centre, rightly describes this trial as a “total waste of time and money.” (Nature 15.1.98)

The vaccine scam works like this. Identify and magnify an ‘epidemic’ disease, whip up world panic, and devise a vaccine against the supposed causative agent. Administer the vaccine, preferably just before the epidemic starts to wane naturally, and then, when the cases of the disease start to diminish, claim the vaccine has worked and the pharmaceutical company who manufactures it will get the credit for saving mankind. There will be bouquets and Nobel prizes all round and every one makes a lot of money. One has only to look at the cases of the anti-polio and anti-smallpox vaccine campaigns to see the classic modus operandi in taking credit for ending epidemics, which in the manner of all self-limiting phenomena, were already dying out before the vaccine was introduced.

In the USA during the late ‘forties, there was a noticeable increase in polio cases. This prompted the authorities to pay a bounty of $25 to GP’s reporting any suspected case of polio, treating it as a notifiable disease. The numbers of cases of ‘polio’ shot up, causing a national panic. Any stiff neck or slight limp was reported. Curiously, at the same time, the official number of cases of aseptic meningitis, which shares some symptoms with polio, and previously reached some 25,000 annually nationwide, disappeared completely. A whole disease just vanished. Subsequently, when the polio epidemic had abated, the credit being given to Salk and Sabin’s polio vaccines (which frequently caused polio !) the numbers of meningitis cases returned to their previous level.

Professor Gordon Stewart explained to me that the same thing happened in India when people were paid a few rupees to report cases of smallpox during the WHO’s anti-smallpox campaign. As a result, official figures for chickenpox disappeared during the campaign, but reappeared with a bang after smallpox was declared eradicated in 1980. The trick is to make sure you get in with the vaccine just before the numbers of cases of the disease start to diminish. In the case of polio, the definition of the disease was later tightened up to exclude illnesses with similar symptoms - meningitis, encephalopathies etc. - and presto, there was a dramatic drop in the official polio cases. Hooray, the vaccine worked. Unfortunately for the AIDS Junta, the epidemic is clearly already on the wane, at least in the West, and there is the worrying little detail of the ten year incubation period. The latest official German annual AIDS-deaths figure for 1997 is 35 (thirty-five) out of a population of some 81,000,000 (eighty one million!) so does Germany really need a vaccine? Does anyone? Indeed, will it ever be possible to devise a single vaccine against a syndrome which seems to vary in intensity, incidence and symptoms throughout the world and even from city to city? Watch out for the inevitable redefinitions of AIDS, either to make the threat seem more widespread than it is, or to have just the reverse effect, by diminishing the number of AIDS-related illnesses and make it look as if AIDS is on the wane, thanks to a vaccine or the latest ‘miracle’ drugs.

In April 1984, the world was told that the ‘probable’ cause of AIDS had been found - a novel ‘human’ retrovirus called variously LAV, ARV, and HTLV-III. Subsequently, despite a complete lack of convincing scientific evidence, which persists to this day, that such an organism causes immunosuppression, in a pre-emptive move the claimed novel retrovirus was named ‘HIV’ (Human Immunodeficiency Virus) by an international committee (1986). It was accepted that after a period of latency, some infectees start to manifest symptoms of AIDS, and the average length of asymptomatic infection was some ten years. Therefore, any trial of an ‘anti-HIV’ vaccine must run for at least ten years to show whether the vaccine will prevent disease after infection with the alleged viral cause of AIDS. To date, none of the vaccine trials has run for more than a few months, and Francis’ trial is scheduled to run only three years.

Vaccination will be a singularly inappropriate method of countering ‘HIV’/AIDS. A vaccine uses either a killed pathogenic micro-organism or a specially weakened (attenuated) live form to induce a prophylactic antibody response. However, vaccination is primarily used to protect against pathogens which cause diseases of rapid onset. Organisms which replicate very rapidly to high titre may cause a disease before the body can mount a successful antibody response. Vaccination against a pathogen primes the body by tricking it into generating antibodies in advance of a natural infection. His antibody titre then subsides, but leaves behind memory cells which can mount an almost instantaneous defense reaction.

Nevertheless, in the smallpox epidemic of the mid-eighteenth century, it was found that the mortality rate was much reduced in those who had suffered a mild smallpox rash before it spread from the local population. (Smallpox was a highly infectious illness, the mortality rate in the modern world is around 0.1%) This observation led to the development of the smallpox vaccine, which was a live (attenuated) smallpox virus vaccine. The vaccine was initially effective in the late 1700’s and early 1800’s, but it was not until the early 1900’s that it was used on a large scale in Europe. The use of the smallpox vaccine led to the eradication of smallpox, which was declared by the World Health Organization in 1980. The vaccine worked because it induced a strong immune response against the smallpox virus.

The use of the smallpox vaccine is a prime example of a successful vaccine. It was effective in preventing the spread of smallpox and led to the eradication of the disease. However, it is important to note that the vaccine is not effective against all diseases, and it may be possible to develop vaccines against other diseases in the future. The key to the development of vaccines is to understand the mechanisms of disease and to develop vaccines that can be used to prevent the spread of the disease.
response on subsequent exposure, as opposed to having to wait for the body’s humoral immune system to analyse the intruding pathogen and then synthesise an appropriate antibody. However, according to the orthodox view of ‘HIV’/AIDS, disease only develops after the body has already been generating neutralising antibody against ‘HIV’ for roughly a decade. Indeed, it is these antibodies which are alleged to show infection with ‘HIV’ in the first place. All those rapacious AIDS researchers seem to be unaware of the irony that the very clue which reveals ‘HIV infection’ protects from all ill-effects allegedly caused by the ‘virus’. As Peter Duesberg explained eleven years ago (Cancer Research, 1:3’87) once you are producing the antibodies which give an ‘HIV’+ diagnosis, you are already vaccinated. However, we are now constantly being told of the lightning-fast mutability of ‘viral strains’, usually to explain why the latest miracle drugs cease to work. This has excused the failure of AZT, other nucleoside analogue drugs and protease inhibitors, and will no doubt be used to explain the failure of anti-‘HIV’ vaccines, including D on Francis’. Once again, the brilliant ‘HIV’ will have mutated to outwit the vaccine.

The difficulties surrounding the production of a vaccine against ‘HIV’ are too horrendous for it ever to be a possibility. The putative virus is alleged to mutate so rapidly that a single individual is said to produce many different strains of virus simultaneously. Which one do you vaccinate against? How could any vaccine protect against a constantly moving target? Should one use an attenuated live viral strain or a killed fully pathogenic one to generate an antibody response? It is also supposed, since a group of eleven ‘HIV’ infected people were found in Australia who have failed to develop AIDS after some 13 years, that not all strains of ‘HIV’ are pathogenic. Would a vaccine-derived antibody raised by using such a non-pathogenic strain in a vaccine afford protection against a fully virulent strain of ‘HIV’? What of those scientists like M ontagnier, who as early as 1985/6 was suggesting that the antibodies against ‘HIV’ were autoimmune antibodies and may themselves be the cause of the decline of the immune system? Could a vaccine cause AIDS, as the polio one undoubtedly did? Even in the more than unlikely event of a successful vaccine being devised, the implications are alarming for a country’s blood supply. If there is nothing to distinguish vaccine-generated antibodies from an acquired infection, and antibody-positive blood is screened out of the blood supply, this suggests that countries with widescale vaccination, i.e. in the Third World, will have large shortfalls in blood supplies for transfusion. Will vaccines be told not to donate blood, setting up a form of ‘positive’ discrimination?

What of the “healthy volunteers” in Francis’ study? Are they from the ‘high risk’ groups? The AIDS epidemics in the USA and Thailand have entirely different profiles, cases being more evenly distributed between the sexes in the latter area, but the linking factor amongst heterosexuals in both countries seems to be drug addiction, especially in sex workers. How will the volunteers be encouraged to challenge the vaccine? Will they be encouraged to have unsafe sex and indulge in heavy recreational drug use with shared needles in the hope that they will become infected with ‘HIV’, thereby challenging the vaccine to protect them against AIDS ten years - not the planned three years - down the line? This would obviously be stupid as well as completely unethical, but the fact remains that a vaccine trial must challenge the vaccine to protect the recipient. When W alter Reed wanted to demonstrate the efficacy of his yellow fever vaccine, he vaccinated himself and then deliberately infected himself with the mosquito-borne arbovirus which causes the disease to demonstrate that he was vaccinated. Are Francis “healthy volunteers” in ‘high-risk activity’ groups, or just Mr. and Mrs. Average? Mr and Mrs Average do not get AIDS. A recent feature article in the Sunday Times Magazine (14.12.'97) made much of a group of celebrity ‘viral strains’, with varying degrees of risk behaviour, but short of using deliberate injection with a strain of ‘HIV’, what risk do they have of ‘infection’? These people obviously mean well, but their altruistic gesture is as futile as nuns volunteering for a vaccine trial against syphilis. The fact remains, ‘HIV’ is not highly infectious like the polio or measles viruses, yet another reason why a vaccine is wholly irrelevant.

The only certain way to test the efficacy of a medicine or a vaccine is by double-blind placebo testing. Such testing is totally impracticable, ostensibly because it would be unethical to give a placebo to a group of volunteer controls when dealing with an allegedly fatal pathogen. This excuse is entirely specious. In a double-blind controlled study, the volunteers would have to agree to be infected with the pathogen - in this case ‘HIV’ - and then it could be shown whether the recipients of the vaccine received protection from ‘HIV’, whilst those who got the placebo would succumb to AIDS. According to the orthodox view it would take anything up to a decade or longer for AIDS symptoms to show up in those who didn’t get the vaccine after being infected with ‘HIV’, so such a trial would not only be unethical but also impractical from a time point of view. What the AIDS Junta really fears is that neither group of volunteers in a double-blinded controlled study would develop AIDS, because they are aware that ‘HIV’ is not the cause of AIDS but a marker for risk.
WWW.GODHATESFAGS.COM is the Internet web site of the Rev. Fred Phelps, pastor of the Westboro Baptist Church in Topeka, Kansas. His congregation consists almost entirely of family members, 39 of them. In their FAQ (frequently asked questions) they describe their activities as follows:

"The Westboro Baptist Church of Topeka, Kansas, engages in daily peaceful sidewalk demonstrations opposing the homosexual lifestyle of soul-damning, nation-destroying filth. We display large, colorful signs containing Bible words and sentiments, including: GOD HATES FAGS, FAGS HATE GOD, AIDS CURES FAGS, THANK GOD FOR AIDS, FAGS BURN IN HELL, NO TEARS FOR QUEER'S SIN & SHAME NOT PRIDE, FAGS=ANAL SEX=DEATH, FAG=AIDS=DEATH, GOD IS NOT MOCKED, FAGS ARE NATURE FREAKS, GOD GAVE FAGS UP, NO SPECIAL LAWS FOR FAGS, etc." [from the FAQ of the Westboro Baptist Church web site]

In addition to "homosexual parades" they picket the funerals of "impenitent sodomites", confronting the mourners with such signs as "[deceased] IN HELL", FAG FUNERAL, and "GOD HATES FAGS". Phelps has no patience with the notion that "God is love", to which he responds: "Try telling the people in hell that God loves them... FAGs are reprobate. God hates reprobates. Therefore, God hates fags... "The Bible preaches hate. For every one verse about God's mercy, love, compassion, etc., there are two verses about His vengeance, hatred, wrath, etc."

The maudlin, kissy-pooh, feel-good, touchy-feely preachers of today's society are damning this nation and this world to hell.

On the first of November 1997, the Phelps flock brought their message of Christian hate to Provincetown, Massachusetts, which was targeted as a prominent gay resort, and also because of a recent decision by the Provincetown school board to initiate an "anti-bias" curriculum to promote tolerance towards racial and religious minorities, women, gay men, lesbians, etc. Provincetown really is a tolerant community, and took the Phelps visit seriously. A special Provincetown Town Hall meeting was held, with the consensus reached to isolate and ignore Phelps, rather than give him publicity. A march and prayer service were held before Phelps's arrival, and the town was bedecked with yellow ribbons symbolizing "equality". The Provincetown Interfaith Coalition issued a statement, which began: "We are a community deeply rooted in our commitment to affirm the dignity and worth of every human being." As it turned out, the Phelps picket was uneventful. His daughters -- practising lawyers who organized the demonstrations -- sang hymns, but could not be heard over the din of the Bradford Street traffic. His young grandchildren held up signs saying "GOD HATES FAGS" and "AIDS CURES FAGS". A young hunk in sun glasses held up "P-TOWN FAG SHAME". Provincetown's response was subdued applause when the Phelps gang left in three cars after 90 minutes of picketing. Phelps was isolated in Provincetown. He himself admits being isolated from other right-wingers and other religious fundamentalists. Unfortunately, his ideas are shared by all too many Americans, hundreds of thousands of whom are affiliated with the Moral Majority and similar groups. These people put bumper stickers on their cars with such slogans as: "KILL A QUEER FOR CHRIST". Despite the apparent gains of the gay rights movement, half of the states in the U.S. still have sodomy statutes on the book, which make all sex between males a felony. It's not easy for a gay man in America to remain healthy. In addition to the crude death messages of the religious fundamentalists, there are subtler death messages emanating from AIDS organizations and counsellors of all kinds, from Public Health Service agencies, and from pharmaceutical propaganda, setting up the equation: GAY MAN = HIV-POSITIVE = AIDS = DEATH. The Phelps campaign and the AIDS Quilt are really two sides of the same coin: both are telling gay men that they ought to die.
Screening of Pregnant Women for HIV

by Rosalind Harrison

Rosalind Harrison is an ophthalmologist in Burton-on-Trent, Staffordshire, England. She was born in Brisbane, Australia and studied medicine at the University of Queensland and the London School of Hygiene and Tropical Medicine. She is co-author of the book AIDS, Racism and Africa and will be addressing the UN Human Rights Commission on AIDS in Africa later this year.

Antenatal testing for HIV was the leading topic of the 24th January edition of the British Medical Journal. In a paper by Angus Nicoll et al., results were presented of HIV-1 detection among pregnant women in the United Kingdom from 1988-96. Blood is collected routinely from all children born in the U.K., and about 70% of these samples were tested anonymously for HIV. As maternal antibodies pass across the placenta, antibodies in the baby are a surrogate marker for HIV positivity in the mother. Data of all known HIV positive children and their mothers for the same period is also presented in this paper. If these two sets of data are compared, only about a quarter of HIV positive pregnant women are identified by current voluntary testing programmes during pregnancy, and the authors argue that HIV testing must be made more available and accessible. The given purpose of identification of HIV positivity in pregnancy is to offer interventions that reduce the transmission of HIV from mother to child. These interventions include termination of pregnancy, abstention from breast feeding, and AZT.

Although not emphasised by the authors, the study confirms the virtual absence of spread of HIV positivity outside recognised risk groups. In London transmission of HIV positivity from mother to child occurs predominantly in African women, and in Scotland in IV drug users. Only 6% of births of reported HIV positive children (49/797) in the nine years of the study were to mothers “apparently infected heterosexually in the United Kingdom by a man without a known history of high risk”. From the anonymous screening there were 1,459 HIV positive babies of 3,080,632 tested, an average of 162 per year. 1,123 of these babies came from the London area. Seropositivity did not increase with time in Scotland, and the increase in the rest of the U.K. outside London was minimal. Seroprevalence for the total U.K. population of newborn babies tested was 0.047%. (If you check the paper by Nicoll et al. you will find the numbers in the totals column of Table 1 are incorrect or missing, but the correct figures can be calculated from the rest of the table).

Inevitably HIV positivity in African women is regarded as true positivity and acquired heterosexually. Unlike AIDS in other risk groups, the major AIDS-defining diseases in Africa are tuberculosis (29-44%) and bacterial pneumonia (21-35%). A study of patients with leprosy in 1994 found a high rate of false positivity to HIV tests due to cross-reaction with antibodies to proteins in the leprosy bacteria cell wall. The tuberculosis bacteria have the same proteins. Brazilian researchers, also in 1994, reported that 30% of repeatedly positive HIV ELISA tests in patients with tuberculosis were false positives. Antibodies to malaria have also been reported to be responsible for high rates of false positivity in Africans, and the incidence of both tuberculosis and malaria has been rising in Africa, and the same trends would occur in African immigrants. In the U.K., two consecutive positive HIV ELISAs are considered proof of HIV infection, and it is surely not difficult to appreciate that Africans in the U.K. who develop bacterial chest infections and tuberculosis could be diagnosed with a sexually transmitted immune deficiency. Now, if screening is increased, more pregnant African women will be given an HIV death sentence, and will be persuaded to have their pregnancies terminated or will be treated with AZT.
Do antibody tests prove HIV infection?

A blood-curdling interview with Dr. Valendar F. Turner

Dr. Valendar F. Turner is a member of the Perth group of HIV/AIDS dissidents. He graduated from the University of Sydney in 1969, is a Fellow of the Royal Australasian College of Surgeons and Fellow of the Australasian College for Emergency Medicine. He practises at the Royal Perth Hospital in Western Australia.
HC: Good afternoon Downunder.
VFT: Good morning Huw.
HC: The Perth Group publications\textsuperscript{1-13} seem to cover just about every facet of HIV and AIDS but what I want to go over again is the antibody tests.
VFT: Fine.
HC: I’m particularly interested in trying to make this subject plain and simple for ordinary folk who haven’t read the arguments published in the Group’s papers over the past decade. Or if they have, don’t quite understand. I mean it’s pretty much in-your-face to read an abstract telling you Eleopulos et al don’t accept HIV antibodies tests as proof of HIV infection in anyone.
VFT: I know but that’s how Eleopulos et al read the data.
HC: Could you start with an overview?
VFT: Sure. Let’s consider the two words ‘antibody’ and ‘test’. In this context ‘test’ has two meanings. The first is something you do in an attempt to indicate the presence or absence of some substance or property. For example, does a patient have appendicitis? Or is a woman pregnant? The second is something you do to ascertain something’s worth. For example, if you develop a blood test for pregnancy, how well does it perform?
HC: And antibodies?
VFT: Antibodies are proteins produced by cells of the immune system known as B lymphocytes. Not to be confused with T lymphocytes, the immune system cells which HIV allegedly kills making people immune deficient. The present theory of antibody production is that each B lymphocyte and its descendants, known as clones, elaborates one and only one unique antibody molecule.
HC: What switches B-cells into producing antibodies?
VFT: Two things. Firstly, when a B-cell encounters a substance known as an antigen. That word is derived from the letters of ANT IbodY GEnerating. Antigens are always large molecules and are often proteins. In fact proteins are the most powerful antigens. Even more so if they gain direct access to the blood stream.
HC: How does the antigen get the B-cell to make the antibody?
VFT: In the old days it was thought antigens instructed B-cells in the act of making antibodies. Like reading out a recipe while someone else makes the cake. But that’s no longer believed. Nowadays the theory is that each B-cell already knows the recipe. But for only one type of cake. Each is programmed to make a unique antibody. Many times over of course but all the same. It’s estimated B-cells have a combined repertoire of about one million distinct antibody molecules. It’s just a matter of an antigen meeting up with the right B-cell. When it does that’s the key which turns the switch as you suggest. The cell divides and produces a clone and out come the antibodies. That antibody then unites chemically with the antigen.\textsuperscript{14}
HC: W hat else induces antibodies?
VFT: B-cells can be stimulated non-specifically. You give the immune system a belt and an assortment of B-cells go into production. For all we know this might be quite common. The only way to find out is to test for antibodies to everything except what you used to belt the immune system.
HC: W hat is the biological purpose of the antibody/antigen union?
VFT: Supposedly antibodies neutralise the untoward effects of antigens.
HC: A re germs antigens?
VFT: Yes, but with some qualification. Obviously antibodies and antigens must combine at particular places on their molecules. It’s like hugging your grandmother. Your arms are only a small part of you and make contact only over a small part of grandma. The business end of the antibody molecule is called the combining site and the part of the antigen it joins on to is the antigenic determinant. There are many possible antigenic determinant sites on each antigen and any of these can induce a corresponding clone of B-cells to produce a particular antibody.
HC: So the antibodies produced to a germ are really a mixture of many different molecules to many different bits of the germ?
VFT: Yes. The technical term is that the antibody response is polyclonal.
HC: H ow do you give the immune system a belt?
VFT: Let loose with drugs or infectious agents or foreign proteins. Things to which all the HIV/AIDS risk groups are exposed. Of course these may act as conventional antigens but they can also act
on other B-cells. This may produce arcane reactions. A good example is that of Epstein-Barr virus, the virus that causes glandular fever.

**H C : W ha’s arcane there?**

**VFT :** Somehow the virus switches on a set of B-cells programmed to make antibodies which react with the red blood cells of horses. And another which makes antibodies to sheep blood. But these aren’t antibodies destined for EBV itself. They’re something completely different. One wonders why we would ever need to produce such antibodies but we can. In fact as doctors we make use of this to diagnose glandular fever. This is an antibody test but it doesn’t look for antibodies to the causative virus. Instead it looks for the horse blood antibodies.

**H C : C urioser and curioser. W hat’s the basis of using antibodies to prove HIV infection?**

**VFT :** The belief that because HIV is foreign it will induce the production of antibodies directed against HIV.

**H C : T he theory is that an antibody to a virus can only arise if B-cells have encountered that virus?**

**VFT :** Yes.

**H C : W hy not prove HIV infection by growing the virus?**

**VFT :** Antibodies are technically easier and a lot quicker and cheaper.

**H C : A nd you detect the antibody by taking some blood, mixing in some virus and seeing if the two react?**

**VFT :** That’s the theory but before we get to that let me explain something else very important. What we can call the age old antibody problem: why you can’t reason backwards from antibodies to germs. It comes about because a particular antibody may also react with an antigen or antigens that did not stimulate its production. This can be due either to non-specific stimulation or because antibodies cross-react.

**H C : W hat does cross-react mean?**

**VFT :** Two different antigens may share the same determinant. So the same antibody can get hold of either antigen by reacting with that part. Even though they’re otherwise different proteins. You can also prove the existence of cross-reactions by doing a little thought experiment. Antibodies are large proteins and can themselves act as antigens. So that’s at least two things an antibody can react with. The antigen that produced it and the antibody to it when it acts as an antigen.

**H C : W hy are these phenomena a problem?**

**VFT :** Because they spoil what would be a nice theory that a person who has an antibody to ‘X’ must automatically be infected with ‘X’. It’s scientifically impossible to make such a claim merely from a chemical reaction.

**H C : E ven if it is beyond question that ‘X’ is a constituent protein of a unique virus?**

**VFT :** Yes. You may never be infected with what your antibodies react with. Otherwise we’d have to say patients with glandular fever are infected with horse blood. As well as sheep blood. Or AIDS patients infected with laboratory chemicals.

**H C : A IDS patients have antibodies to laboratory chemicals? C an you name some?**

**VFT :** Off the top of my head I can name one. Trinitrophenyl antibodies.

**H C : A nd it’s not known how that arises?**

**VFT :** Not precisely.

**H C : H ow does one get around the antibody problem?**

**VFT :** First by realising the problem exists. If you like analogies, diagnosing infections using antibodies, that is, serological diagnosis, is like trying to identify objects from the shadows they cast on the ground. There’s a connection but clouds, buildings, trees and so forth all produce shadows that may look the same or similar. The way around the dilemma involves an appreciation of both meanings of that word ‘test’. According to the first meaning what we want is some method of finding HIV in the body - HIV infection. That’s what we’re really chasing. The best way to do that would be to find the actual object itself - HIV. Prove the existence of HIV in every patient by means that are unambiguous for a unique retrovirus. The gold standard. Any other way, including antibody tests, is indirect and must therefore be validated by comparison alongside the gold standard. The second meaning of ‘test’.

**H C : H ow?**

**VFT :** By running the two sets of data concurrently. The antibody test and whatever you do independently to prove the existence in the person of the virus.

**H C : V irus isolation versus the antibodies?**

**VFT :** Yes but there’s more to proving the existence of the virus than isolating a particle. After Eleni’s interview I’m sure your readers must be a full bottle on this topic.

**H C : I w onder! H ow is an antibody test for HIV actually done?**

**VFT :** As you said. Take some blood from a patient, remove the red cells and then add what’s left, the serum in which the antibodies are dissolved, to some proteins the experts claim are unique constituents of HIV.

**H C : W ha t do you see if the test is positive?**

**VFT :** If the antibodies react with the proteins there will be some detectable change in the solution or in whatever medium the test is performed. It may change colour or a precipitate may form. Or there is some other measurable effect.

**H C : T hings light up? T hat’s all there is to it?**

**VFT :** Basically. But there are refinements. For example, the ELISA versus the Western blot. The ELISA has all the proteins mixed together and in the Western blot you can see each reacting individually, side by side along a thin nitrocellulose strip.

**H C : H ow is the comparison with HIV gold standard done?**

**VFT :** Well everyone wants to know is whether the test can be positive when there is no HIV infection. In other words, is my test a false positive? So, what a scientist is obliged to do long before the test is introduced into clinical practice is to determine what’s known as the specificity of the test. That’s a measure of how often a positive test turns up given HIV is known to be absent. Proved by viral isolation. If the test is one hundred percent specific the answer of course should be never.

**H C : Y es. I think people tend to get confused here. C an we go over these two words, sensitivity and specificity?**

**VFT :** Sure. Sensitivity is a measure of how often a test is positive when there is actually a disease present. For example, if a thousand women are pregnant does the test diagnose them all? If it picks 980 then it’s only 98% sensitive. And it’s specific, in other words, is it ever positive when a woman is definitely not pregnant? For example, if, from a thousand women known not to be pregnant there was one positive test, the test would be 99.9% specific. You’d never dream of putting a pregnancy test into practice until you’d sorted out these parameters.

**H C : I f we take the HIV ELISA test, which is the first and sometimes the only type of test patients have performed to diagnose...**
HIV infection, how is the sensitivity determined?
VFT: First let's examine the way it should be determined. The correct procedure is to assemble a thousand people proven by HIV isolation to be infected with HIV and see how many have a positive ELISA. Now the ELISA is made positive because the solution in which the antibodies react turns cloudy and the degree of cloudiness can be measured with a special instrument that gives out a number.

H: Is any degree of cloudiness positive?
VFT: No because there is always some non-specific background activity. If you set the degree of cloudiness for a positive test very low then everyone might be positive. If it were a pregnancy test for example, even men could be pregnant. So you set some limit or sets of limits for the comparison.

H: How is this determined?
VFT: Here there are some very unscientific practices. Basically, a group of healthy individuals is tested to estimate the background activity. This will have a range of values and from this range researchers select an upper limit which is maybe two or three standard deviations higher than the mean value. Any reading greater than that is defined as positive.

H: It's arbitrary?
VFT: Yes.

H: They don't set the level according to the results of virus isolation?
VFT: No. And setting a level doesn't prove the antibodies are genuine anti-HIV antibodies. You can't say antibodies are to HIV just because there's more of them. Higher levels might just be more of the same that caused the lower level of cloudiness. Or lower levels might be the real thing. The only way to prove the antibodies are a reaction to something called HIV is first to prove you have the virus.

H: What about the sensitivity of the Western blot? How is this determined?
VFT: Again, you have to set criteria for what constitutes a positive test and then apply this to a population of known infected people. Again there are no such data for even one of the multitude of different criteria which are said to define a positive HIV Western blot. But, as I'm sure you know, the sensitivity is not of prime importance to the HIV experts because in most parts of the world the Western blot is put forward as a means of sorting out which positives ELISAs are due to HIV infection and which are not. What's important for the Western blot is its specificity.

H: How does one perform an experiment to measure specificity of the HIV antibody tests? ELISA and Western blot?
VFT: Take a large group of AIDS patients, as well as people who are sick with similar illnesses and laboratory abnormalities as AIDS patients, as well as those at risk and some healthy people, perform HIV isolation to prove none have the virus and amongst this group see how many are antibody positive by whatever criteria you set for each test.

H: What physiological standards do these people meet?
VFT: Because these tests measure antibody reactivity and you need lots of antibodies to produce lots of reactions to prove that the reactivity which defines a positive test is restricted to those individuals who are HIV infected.

H: What else, if sensitivity of either antibody test has never been measured against the guaranteed presence of HIV, has the specificity ever been measured against the certified absence of HIV?
VFT: No one has ever reported an experiment performed to draw this comparison. Not for the ELISA nor the Western blot. This is one of the greatest AIDS mysteries. However, if you look at Gallo's 1984 Science papers, what Gallo and his colleagues called HIV isolation was positive in only a third of their AIDS patients. Yet nearly three times that number had antibodies.28

H: That's a huge disparity. That's nearly twice as many people with antibodies and no virus as with antibodies and virus! It's a much better correlation between antibodies and absence of infection. So right from the start it should have been obvious the test was grossly non-specific?

VFT: Yes.

H: How did Gallo explain this discrepancy?
VFT: Gallo didn't admit to any discrepancy in virus isolation. Instead his group believed all the patients with antibodies were infected. They rejected the low yield of virus isolation on failure to receive or handle their tissue specimens under "optimal" conditions.

H: Yet the Gallo lab was considered expert in culturing retroviruses?
VFT: Over a decade of experience and nowadays it's claimed that the blood of untreated AIDS patients is teeming with HIV.

H: As the discrepancy between antibodies and HIV isolation narrowed over time?
VFT: Not in the least. If you remember our reply to Peter Duesberg, between 1992-93 several reputable, international laboratories in the UK, Germany and the USA tested 224 specimens from antibody positive individuals. These labs also claimed to have performed viral isolation but like all HIV researchers, they're forever perverting the meaning of that word. What they called HIV isolation was another antibody test. This time for detecting just one protein, p24. And under this guise 'isolation' was positive only 83 times. That's 37%.

H: Do HIV experts really refer to an anti-p24 antibody test as virus isolation?
VFT: Most of the time. And some report just finding reverse transcriptase as virus isolation.

H: Is the failure to perform the gold standard comparison the reason why the Perth group claims not one antibody positive person in the world is infected with HIV?
VFT: Principally on that basis we say there is no proof that one person is infected. Yes. But the other reason of course is that no one has yet proven the existence of HIV using the proper method. The method based on the definition of a virus and as discussed at length at the 1972 Pasteur Institute meeting.

H: Which Perth group was the first to argue over a decade ago?
VFT: Right from day one.

H: Nonetheless, it still seems an intrepid claim. No proof that even one antibody positive person in the world is infected.
VFT: Look. We just can't put the words "HIV" and 'antibodies' next to each other and claim you've proved they exist. Or a virus exists. All the test indicates is that some antibodies in patients react with some proteins present in cultures of tissues from the same patients. But given that information what a scientist is obliged to do next is make the comparison with the virus gold standard. Before pronouncing the test highly specific for diagnosing HIV infection. In fact, do you see that the origin of the proteins used in the tests doesn't matter? They don't have to come from HIV. I mean we diagnose Epstein-Barr virus infection without using proteins from the Epstein-Barr virus. Horse red blood cells are not constituents of that virus. What counts is the correlation between certain reactions and the presence or absence of the virus.

H: But surely it makes sense to use proteins from the germ?
VFT: It does because if there is a germ there is a possible connection, forwards, between the germ's antigens and the patient's antibodies. But just because you use the germ doesn't mean you can ignore the problem of antibody cross-reactivity and everything else.

H: So it's incorrect for scientists to say the HIV antibody tests are better nowadays because they use purer proteins?
VFT: That's right. It doesn't follow. Even if genetically engineered proteins are used in the test. You could take the purest protein in the world and find a patient with an antibody to that protein. That doesn't create an axiom that a person with that antibody is infected with a germ containing that particular protein. This is an extremely important but frequently unappreciated concept. In fact you could take a genetically engineered protein and make the test worse.

H: How?
VFT: Because every time you change the antigens there's a possibility you could introduce a new antigenic determinant. All antibodies know how to react and there might be an antibody lurking that links up with that determinant but whose presence bears no relation whatsoever with where you're testing for. For example, lots of humans have antibodies to things like hepatitis A and even Pneumocystis carinii. In fact by the age of four most children have antibodies to the PFC organism. Whether you're sick from the PFC organism. One of those antibodies might cross-react with the new determinant.

H C: Are patients tested for antibodies despite the fact that no one has done a gold standard comparison?

VFT: The tragedy is that these tests were introduced in the total absence of proof of their specificity. This is a fact. The moving finger has written and all our tears cannot wipe out a word of it.

H C: That's from Omar Khayyam?

VFT: Yes.

H C: The Perth group has claimed that the HIV proteins and antibodies as well as the existence of HIV are based on a circular argument. Could you explain that?

VFT: I'll try my best. When Montagnier and Gallo went hunting for retroviruses in 1983/84 they knew that merely finding a particle that looked like a virus, even if they were to isolate the particle and prove it could reverse transcribe RNA into DNA, did not prove the particle was a virus. That's because not all particles, even those that look like viruses, are viruses. And nothing that reverse transcribes is a retrovirus. Or even a virus. These phenomena are non-specific. And stringing together reverse transcription and particles doesn't cure the problem. The only scientific proof that a particle is a virus is purification and analysis followed by experiments to prove particles make more particles exactly the same. In other words, proof that the particles are infectious. These experiments have never been done. Proof of the existence of HIV is based on antibodies but unfortunately, picking up antibodies just added yet another nonspecific item to the list.

H C: But Montagnier and Gallo did discover antibodies from AIDS patients which reacted with certain proteins in their cell cultures.

VFT: Yes they found a few but that doesn't prove the proteins which reacted with these antibodies are the constituents of a virus. Or that the antibodies were induced by contact with a virus. If you'd like another analogy imagine this experiment. In place of that C-dinitrophenylated antigen someone hands you a test tube containing milks obtained from half a dozen different animals. In other words, a mixture of several different proteins but you don't know from which animals. Now in place of a mixture of antibodies from AIDS patients you obtain a second test tube containing a number of different acids. You add the mixture of acids to the mixture of milks and produce curdles. Now you claim you've isolated a cow. Or a goat. And not just any cow or goat. A completely new species of cow or goat. You'd never seen before. There, in the culture. And then you claim that only a particular selection of the acids in the mixture produced that curdle. So, getting back to HIV, proteins reacting with antibodies makes them into the HIV proteins. But since these newly discovered proteins react with these particular antibodies that means these antibodies must be the HIV antibodies. It's called chasing your tail. It's not the way a scientist should establish the existence of a virus or determine which are its antibodies.

H C: But almost everyone believes these antibodies are the HIV antibodies and they're highly specific to HIV.

VFT: True and that's because of virtually the same circular argument. AIDS, the clinical syndrome, usually but not always, is accompanied by antibodies which are interpreted as proof that AIDS-diagnosed patients are infected with HIV. Then the antibodies are used to prove that HIV is the cause of AIDS. In other words, AIDS proves it's HIV proves it's AIDS. Naturally the antibodies seem specific. They and AIDS run around the same circle. What's important for anyone in this debate to realise is that when you pare down what the experts claim proves the existence of HIV, they are all non-specific phenomena including antibody reactions. That's all. It's not isolation. No viral-like particles are separated and analysed and then added to fresh cells to see if exactly the same come out.

H C: But regardless of where these antibodies come from, doesn’t their relationship to AIDS in defining conditions mean something?

VFT: In the AIDS risk groups yes it does. If you have these antibodies you're at risk of either having or developing a number of diseases which constitute the AIDS clinical syndrome. But it doesn't prove the link is a retrovirus.

H C: Or that the illnesses are inevitable?

VFT: They may well not be inevitable. After all, we're taking statistics.

H C: All right. The Perth group has also written at length about the global variation in the HIV W estern blot antibody test criteria. It was first presented in the Bio/Technology paper of 1993 and Contiuum published your chart illustrating the same thing in the November 1995 issue. Tell us about that.

VFT: OK. The Western blot is a general laboratory technique for visualising individual protein/antibody reactions. The proteins are placed at discrete spots in a thin paper strip. In the case of HIV about ten. The human operator inspects the strip and declares which proteins react with antibodies. What you actually see is a series of dark horizontal rectangles called bands. You'd think that if there really were such things as HIV proteins, and that the HIV antibodies are highly specific, then just having one band light up would be proof that HIV is present. But according to the experts that's not the case.

H C: They say you need more than one?

VFT: With one single exception. The intriguing thing is this. Even if one or two bands are not sufficient to diagnose HIV infection there must still be a reason why they're there.

H C: Cross-reacting or non-specifically induced?

VFT: Right. Proteins in the tests lit up by part of the manegerie of antibodies present in AIDS patients. Or maybe a few present in a healthy person following some chance, B-cell stimulus. In fact, cross reactions is the explanation given by all the HIV experts for “non-infected” Western blots. Non-HIV antibodies produced by non-HIV stimuli. But if one or two bands in a Western blot can be caused by non-HIV, cross-reacting antibodies why can’t three or four, or five or six, or all ten bands be caused by cross-reacting, non-HIV antibodies?

H C: I don’t know. You tell me.

VFT: Well, a scientist must admit this possibility. And there’s only one way to find out. Compare your favourite combination of antibodies with HIV itself.

H C: But that has not been done?

VFT: No not done not done. Not even possible to do because no research group has ever presented evidence for the existence of HIV according to the proper rules. In Africa you need two bands but in France you need two bands but in France you need two bands but in the United Kingdom and Australia that wouldn’t count. In Australia you need four and under the US FDA and Red Cross rules you need three.

H C: What about the actual variation in the Western blot?

VFT: Another mystery. What is considered positive depends on where and by whom the test is done. Around the world different combinations of two or three or four of the ten possible bands are deemed proof of infection. In Africa you need two bands but in France, in the United Kingdom and Australia that wouldn’t count. In Australia you need four and under the US FDA and Red Cross rules you need three.

H C: This is the basis of the Group’s quip about emigration?

VFT: Yes. If you’re positive in New York City just get on a plane and come to Perth. You’ll never be positive.

H C: You mentioned an exception?

VFT: The US Multicenter AIDS Cohort Study or MACS. This excellent study began in the early 1980s and followed the fate of
5000 gay men. Under the study rules the Western blot could be positive with just one “STRONG” band. Although that later changed. But until 1990 one band was considered sufficient to diagnose HIV infection. That wouldn’t count anywhere else. Not even in Africa. So there are gay men out there HIV infected on this basis. And perhaps given antiviral drugs as a result.

H C: Let me get this right. We are always conscious of our new results and I think this is extremely important. You’re saying that even the experts concede that some numbers or patterns of bands in the Western blot are not indicative of HIV infection because they’re caused by non-HIV antibodies?

VFT: Yes. You can read what Anthony Fauci wrote about this in his paper. In 1996 we questioned this in a letter published in The Lancet. In light of the current Australian criteria we asked were the man or the four women still considered infected? In their reply the Australian experts defended the original claim of HIV infection because all five people had progressed to AIDS and died. This was because in 1985 the Western blot was in its “infancy”. They implied that the reason extra bands were not present in 1985 was because in 1985 the Western blot was in its “infancy”. They claimed that the reason extra bands were not present in 1985 was because in 1985 the Western blot was in its “infancy”.

H C: So it’s definite that non-HIV antibodies react in an HIV test? VFT: Yes. We know. There are plenty of examples. For instance, 30% of people transfused with HIV negative blood develop antibodies to p24. That’s regarded as one of the most specific HIV proteins and it’s present in the Western blot. And it was one way any one of those 5000 gay men could have scored a positive test in the MACS. So some gay men are infected with HIV on the basis of a test that turns up positive in one third of people transfused with blood that does not even contain HIV. We discussed this in our Bio/Technology paper.

H C: Food for thought. What other instances are there of cross reactions?

VFT: There are many more examples. Surely everyone knows of the dogs and the men in the MACS no one could have told the difference. There’s also the study co-authored by the Australian expert Dr. Elizabeth Dax. In 1991 her group re-analysed Western blot strips, not sera, performed in 1985 on sera originally obtained from ten intravenous drug addicts in 1971–72.

H C: What did that reveal?

VFT: Could I read the details from one of our unpublished papers?

H C: Go ahead.

VFT: Ten persons “with potentially positive WB patterns, when the more specific 1985 criteria were used”, were traced. One patient had died from a motor vehicle accident and there were “no lymphoreticular changes at autopsy, and a thorough retrospective analysis provided no evidence of infection”. Of the nine living addicts, two could not be assessed clinically, seven were not chronically ill, (one was in prison but in good health, one had been successfully discharged from a methadone program, one was enrolled in a methadone program, another sporadically consumed illicit drugs). “The two former patients whose 1971–72 WB results were most strongly reactive had current ELISA and WB assays that were negative. The immune function parameters were inconsistent with immune suppression”. Dax led the authors to conclude, “it is possible that antibodies to a non-pathogenic virus would have disappeared during the 17 to 18 years...follow up. Although this potential cannot be ruled out, it is more likely that the earlier results were false positives...definitive evidence of HIV infection in the United States’ addict population as early as 1972 is still lacking”.

H C: HIV antibodies can fade and even disappear over time? VFT: Yes. Despite the fact that we were told HIV is forever, here was not until The Lancet published our letter that the sera from the gay man and one of the women were retested. On these sera the gay man and the woman now did have four bands.
Our HIV Reference Laboratory admits that one quarter of HIV-free blood donors have one or more reactive bands on the HIV Western blot truly present.

H C: So although everyone admits to interference caused by non-HIV antibodies, no one has really sorted out the magnitude of the problem. As the Perth Group says, they may all be non-HIV antibodies?

VFT: Yes. For example, our HIV Reference Laboratory admits that one quarter of HIV-free blood donors have one or more reactive bands on the HIV Western blot. They concede these are caused by cross-reacting, non-HIV antibodies. Now, the way you get your cross-reacting, non-HIV-induced antibodies is to give your immune system a few belts. And the more belts, and the more closely spaced, the more likely a person tested will have cross-reacting antibodies. But we know that in places like Africa this kind of thing is happening all the time. And it happens across all the AIDS risk groups. So the very people you’re testing for HIV are those with the greatest chance of having cross-reacting or non-specifically induced antibodies. So we have this grotesque paradox. One quarter of pristine, well-fed, OZ* blood donors have one or more HIV WB bands, and that might include four or more reactive bands on the HIV Western blot. They concede these are caused by cross-reacting, non-HIV antibodies. Now, the way you get your cross-reacting, non-HIV-induced antibodies is to give your immune system a few belts. And the more belts, and the more closely spaced, the more likely a person tested will have cross-reacting antibodies. But we know that in places like Africa this kind of thing is happening all the time. And it happens across all the AIDS risk groups. So the very people you’re testing for HIV are those with the greatest chance of having cross-reacting or non-specifically induced antibodies. So we have this grotesque paradox. One quarter of pristine, well-fed, OZ* blood donors have one or more HIV WB bands, and that might include four or more reactive bands on the HIV Western blot. They concede these are caused by cross-reacting, non-HIV antibodies. Now, the way you get your cross-reacting, non-HIV-induced antibodies is to give your immune system a few belts. And the more belts, and the more closely spaced, the more likely a person tested will have cross-reacting antibodies. But we know that in places like Africa this kind of thing is happening all the time. And it happens across all the AIDS risk groups. So the very people you’re testing for HIV are those with the greatest chance of having cross-reacting or non-specifically induced antibodies. So we have this grotesque paradox. One quarter of pristine, well-fed, OZ* blood donors have one or more HIV WB bands, and that might include four or more reactive bands on the HIV Western blot. They concede these are caused by cross-reacting, non-HIV antibodies.
of HIV infection in the rest of the world is approaching that of Australia.

HC: Which is deemed to be one of the lowest in the world?
VFT: Yes.

HC: Obviously it's been made much easier to diagnose HIV infection in Africa compared to Australia.

VFT: The World Health Organisation criteria make it much easier to report a positive test in Africa. But that doesn't prove a positive test is an AIDS case.

HC: The criteria should be the most stringent in the so-called developing world?
VFT: No one knows the correct criteria anywhere in the world but everyone does know about cross-reacting antibodies. And they are what create the confusion. It's like losing your five year old kid at the pictures. If you had to take him to something Adults Only because your babysitter ran away, then it's simple. The theatre is most likely full of adults and any kid you see is likely to be your kid. But what if you took him to see Snow White? There's kids all over the place. You need far more stringent criteria before you can pick out your kid. If he had a look-alike, or even just dressed the same, you'd have to set the stakes higher still. If he had a twin brother you might need to take off his socks and look for the mole on his foot.

HC: So using only two bands in Africa means the test is worse quality than it is even in the West for example?
VFT: When you talk about tests you need to be careful with words. 'Quality' could refer to any test parameter. We don't know any of the test parameters because they've never been appraised against the gold standard. I must stress this again and again. Without knowing the sensitivity and specificity of the HIV antibody tests it is impossible to use the tests to prove HIV infection. But your question raises another interesting point. When you look at the mathematics of testing it's very easy to prove that where the prevalence of whatever you're testing point. When you look at the mathematics of testing it's very easy to prove that where the prevalence of whatever you're testing point. When you look at the mathematics of testing it's very easy to prove that where the prevalence is low the odds are stacked against a person even knowing the results of the test. That's because the odds are stacked before a person even stresses this again and again. Without knowing the sensitivity and specificity of the HIV antibody tests it is impossible to use the tests to prove HIV infection. But your question raises another interesting point. When you look at the mathematics of testing it's very easy to prove that where the prevalence of whatever you're testing point. When you look at the mathematics of testing it's very easy to prove that where the prevalence is low the odds are stacked against a person even knowing the results of the test. That's because the odds are stacked before a person even.

HC: Could an equal gender distribution of AIDS in sexually active adults prove sexual transmission?
VFT: It's consistent with sexual transmission but it's not sufficient proof. Equal numbers of sexually active adults develop appendicitis or meningitis. Or schizophrenia. Are there diseases sexually transmitted?

HC: Hasn't the Perth group recently published a paper reviewing cross-reacting antibodies?
VFT: Yes. Our last paper reported a considerable amount of data showing that antibodies to the types of organisms which infect 90% of AIDS patients may also react with all the putative HIV proteins. Including in the Western blot. So, if 90% of AIDS patients are infected with either a mycobacterium or a fungus such as Pneumocystis carinii, how is it possible to diagnose HIV infection in such persons, or to assert that HIV is the cause of their diseases? The paper also examined cross-reacting antibodies in relation to proof for the existence of HIV. In fact, as a caveat, we go into great detail to explain how virtually overnight the world's first human retrovirus, Gallo's HLV23A, became extinct when its antibodies were proved non-specific.

HC: And the Perth group posits a similar fate for HIV?
VFT: When someone finally takes on the isolation or specificity problem, they're really the same problem, we believe this is a distinct possibility.

HC: So compared to 1993, when the Bio/Technology paper was published, there's more evidence that positive antibody tests are caused by factors even the experts admit are non-HIV?
VFT: Definitely. The other thing that's important to remember is that patients are highly selected for antibodies before they ever get to the Western blot. WS are done on people who first of all feel the need to go to a doctor and then have sufficient antibodies to make the ELISA react twice in a row.

HC: T hey're preloaded with a selection of antibodies?
VFT: Right. You see Huw, when you say someone is HIV negative, the truth is they're not ELISA negative, WB negative. They are actually ELISA negative either once or one out two, and Western blot not done. A negative is not confirmed with a Western blot, only a positive. But by choosing this particular testing strategy the HIV/AIDS experts have maximised the chances for the appearance of cross-reacting antibodies.

HC: Maximised cross-reactions? Is there evidence for this?
VFT: Yes. In 1988 the US Army\(^4\) tested over a million soldiers and found that even in healthy military recruits, half of all the 12,000 first positive ELISA\(s\) were negative second time around. And after a second positive ELISA\(s\), two thirds failed to react on a first Western blot. And some first Western blots failed to react on a second Western blot. So, what you set up with two positive ELISAs before a Western blot is a great opportunity to introduce confusion caused by cross-reacting antibodies. Snow White in a test tube.

HC: Might those people who would test negative twice on ELISA and then positive on Western blot?

VFT: This happens but there is little data on how often because negatives usually aren't confirmed in this way.

HC: Are any other reasons put forward to justify the variation in the actual ELISA criteria?

VFT: None that I know unless of course HIV is endowed with some kind of global navigational system. It figures out where it is and then chooses which B-cells to engage. That skill would be extremely hard to encode in eight or nine or ten genes.

HC: Why eight or nine or ten genes?

VFT: It may be the most studied object in the universe but the experts still don't agree how many genes it has.

HC: In 1998 what advice would you give a patient wishing to know his or her HIV antibody status?

VFT: First of all, from the point of view of establishing the presence of HIV infection, I'd say don't have a test. Don't spread HIV testing. You wouldn't expect a woman who's missed a period to have a pregnancy test if you didn't know how well the test performed. So why this one?

HC: What if someone, say in a high-risk group, wants to know his or her chances of developing an AIDS-defining illness? Regardless of whether HIV is the cause?

VFT: I suppose there's two ways of looking at this. What are the chances of getting sick, which is how doctors tend to think, or what are the chances of remaining healthy? That puts a different emphasis from the point of view of the person. There's no doubt about the association between being in a risk group, having a positive test and developing certain diseases defined as AIDS. But that doesn't apply across the board. It's only statistical. So for an individual these two variables cannot be the whole story. Not at all such people get sick and the risk varies up to fifty times between the risk groups. So, if you put aside the retrovirus link and all that and look around for other factors. Now, like the ultimate causes of most diseases, some of these factors may be completely unknown and totally out of your control. But there might be some that are not unknown and are under your control. Maybe as simple as being in a risk group. You could, for example, decide to get out of your risk group or cease doing whatever is risky within your risk group. Remember what happened to the drug addicts. As far as explaining the association of the antibody tests is concerned, perhaps HIV researchers have inadvertently stumbled across a "something wrong test", like the ESR for example.

HC: What is the ESR?

VFT: The erythrocyte sedimentation rate. It's a test widely used in clinical medicine. It measures how fast a drop of blood falls to the bottom of a test tube of anticoagulant solution. The rate at which red blood cells sediment is affected by changes in the plasma in which they've been living, especially changes caused by alterations in the composition of the proteins. For example in inflammatory conditions such as rheumatoid arthritis and tuberculosis, although non-diseases such as pregnancy also produce a high ESR. In fact, in the old days, the ESR was used as a pregnancy test. The point is this. Our group has long argued lack of proof for a retrovirus as the cause of these antibodies. But nonetheless, something must stimulate their production and understanding that this is a possibility might lead people to things which could undo their possible harmful warnings. If the positive test is not caused by one of the actual diseases, maybe there are elements of the person's life which can be changed so that the stimulus to this warning system is turned down. Or even switched off. Again we come back to those drug addicts. They didn't have HIV, the experts say so, but they did have antibodies which reacted in an HIV test. Whatever the reason, when they altered their lives towards attaining better health, somewhere along the same road where they shook off their habit, they shook off their antibodies. I know the experts' explanation was that they never had "real" HIV antibodies but that, much more innocent interpretation, presents our side of the argument. These data are predicted by our theory. These data are a test of our theory and our theory has passed this test. The only difference is we say there are no proven, "real", HIV antibodies. So, maybe just the idea that these antibodies could have other causes might bring sufficient hope to neutralise the doom wrought by the explanation that they must be due to HIV. I think those of us who are not HIV positive cannot even begin to imagine how profoundly the psyche and health of an individual are affected by belief in the existence of a lethal retrovirus inexcusably eating away at the immune system. It must take extreme valour to even question what almost the whole of the rest of the world believes to be true.

HC: Would you study long-term survivors with HIV antibodies to delineate what factors lead HIV-positive individuals towards disease?

VFT: I'd say don't have a test. Don't spread HIV testing. You wouldn't expect a woman who's missed a period to have a pregnancy test if you didn't know how well the test performed. So why this one?

HC: What if someone not in a risk group is healthy but positive?

VFT: The only honest answer is that, from the antibodies point of view, there are no data upon which to pronounce a prognosis.

HC: Why do you say that?

VFT: Because from a purely scientific point of view, to determine whether these antibodies represent an independent hazard, one would have to take a hundred or so healthy, no risk, HIV positive individuals and follow them untreated for a number of years and see what happens. But you would not be able to tell them they're HIV positive.

HC: Why not?

VFT: Because, as we've just discussed, patients and physicians believe most fervently that being HIV positive is a death sentence. This belief and the possible administration of anti-HIV drugs may themselves produce illness. These two variables would severely confound the experiment.

HC: As a doctor yourself, what in particular would you say patients should ask their doctors?

VFT: Request scientific proof that the antibodies present in your body arise for no other reason than infection with a virus called HIV. It may be the most studied object in the universe but the experts still don't agree how many genes it has.

HC: What about people with actual AIDS-defining diseases?

VFT: As I said before, the diseases are elements of the person's life which can be changed so that the stimulus to this warning system is turned down. Or even switched off. Again we come back to those drug addicts. They didn't have HIV, the experts say so, but they did have antibodies which reacted in an HIV test. Whatever the reason, when they altered their lives towards attaining better health, somewhere along the same road where they shook off their habit, they shook off their antibodies. I know the experts' explanation was that they never had "real" HIV antibodies but that, much more innocent interpretation, presents our side of the argument. These data are predicted by our theory. These data are a test of our theory and our theory has passed this test. The only difference is we say there are no proven, "real", HIV antibodies. So, maybe just the idea that these antibodies could have other causes might bring sufficient hope to neutralise the doom wrought by the explanation that they must be due to HIV. I think those of us who are not HIV positive cannot even begin to imagine how profoundly the psyche and health of an individual are affected by belief in the existence of a lethal retrovirus inexcusably eating away at the immune system. It must take extreme valour to even question what almost the whole of the rest of the world believes to be true.

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According to Anthony Fauci, “the least likely explanation for an indifferent [insufficient bands for positive but not the complete absence of bands=negative] western blot is that the individual is infected with HIV...The most likely explanation is that the patient being tested has antibodies that cross react with one of the proteins of HIV.”

* http://www.virusmyth.com/aids/perthgroup/
* OZ - Australia
* Vegemite - A favourite Australian yeast-based sandwich spread.

REFERENCES

TANZANIA: A dream too far...

Since the 70's, many African countries have suffered a drastic degradation of their economic situation. Tanzania should have escaped this fate, but it is not the case. The first president, Julius Nyerere, intending to create a socialist state “with a Human Face” opened great expectations after independence. One hundred and thirty tribes were unified, self-reliance in agriculture was repelling famines, all children had access to school, and remote villages had a dispensary within reach.

The country dreamt for a while...

But the World Economic Order was not ready to finance any socialist experience. Beneath its “Human Face” Tanzania had no oil-field, no industry to barter for investments, no diamonds for which to be forgiven non-orthodox and non-aligned politics. A painful slip into deep poverty was only ended in 1985 when Nyerere himself gave up the leadership.

Tanzania should have escaped this fate, but it is not the case. The first president, Julius Nyerere, intending to create a socialist state “with a Human Face” opened great expectations after independence. One hundred and thirty tribes were unified, self-reliance in agriculture was repelling famines, all children had access to school, and remote villages had a dispensary within reach.

His successor, accepting the dictat of the International Monetary Fund (IMF), made poverty look different: glossy in towns, dull in villages.

Today, Tanzania is a “politically correct” nation of 25 million Africans mostly farmers, dreaming of what they could get instead of what they could be.
KAGERA: Dead-end to paradise.

The Kagera region, in north-west Tanzania bordering Lake Victoria and Uganda, was regarded as a little paradise at the beginning of this century. The local tribes, the Wahaya, had an easy life. Pleasant climate due to the elevation (1,200 meters), rains all year around, simple and permanent cultivation of bananas growing at the door-steps, plenty of fish from the lake and the surrounding swamps, large herds of cattle grazing freely in the low lands. With coffee became a valuable cash-crop, the Wahaya had an ideal location for the shrub, as it needs a shady environment: they introduced it in the shade of their banana trees. Soon, the region became one of the most important coffee producers. Cash-flow poured in, and without having to lose any of its autonomy, the small feudal Kagera jumped onto the consumer society. Bukoba, on the lake shore, which had never been more than a tiny trading post, became a large market. Kampala, only 300 km away, although in Uganda, became a capital for Kagera.

Wahaya children, who had been put in schools by the missionaries before all other Tanzanians, went to Uganda to continue their secondary education. They became the first educated generation, the first to enter public services, therefore the first ones to have access to power.

Such a boom supported the illusion that Kagera was connected to a permanent infusion of wealth, not obliged to sweat for its daily bread. The Gates of Eden were not kept open for long!

Other ethnic groups entered political competition. The coffee market slumped. Fuel prices rocketed. East-coast Fever wiped out most of the livestock. Nationalisation ruined schools and dispensaries. Amin Dada invaded the region during the 1979 war against Tanzania. Then...like a victim of a crash, the Wahaya farmer sat in shock amid his plantation, staring at the past, sobbing bitterly over his fate.

Essential commodities became scare. Malnutrition settled in. Lack of industry and infrastructure pushed the youth to migrate. Nowadays they are found all over East Africa's shantytowns, usually home once a year to put a roof on the family house, make pregnant one of their many wives, or fetch a "house-girl" among the teenagers.

Their last visit is when they come back in a coffin; many of them do not live long. They leave behind a flock of orphans, and their name is swallowed by the AIDS Control Programme for Tanzania.

For twenty years now the Kagera paradise has been sinking in this socio-economic storm.

AIDS: A label for an empty file.

Epidemiologists used to place Kagera region at the top of the hit-parade for AIDS in Tanzania: first in the number of deaths, first for orphans, first to have diagnosed the disease in 1985.

The Wahaya put the blame on their Ugandan neighbours, since it is hard to bear the flag of a shameful illness. They acknowledge having lost many young adults. With that fact, a question nonetheless remains: how to measure the increase of mortality and how to attribute it to AIDS? There is unfortunately not a single scientific tool to do that.

What about the test?

Almost nobody is tested for HIV. If people were, it would not mean much: the tests, developed in America and Europe, are of very poor predictive value in Africa, due to many cross reactions. Thus alternatively the epidemiologists have chosen to ignore the detection of an immune deficiency virus and are referring to the clinical condition instead.

A wasted patient with diarrhoea and prolonged fever is classified as an AIDS patient. In the course of time the list of symptoms has been stretched, but the method has not gained in specificity: out of the 25 symptoms it includes, not one is specific to a new disease in Africa.

What is the value of AIDS statistics built upon a definition which has nothing to do with the definition of AIDS?

What about a new pathogen?

The hypothesis of a new virus needs to be checked against the pre-existing mortality pattern. But there are no such data. Up to now, births and deaths have only been registered in the memory of the people. It is a very poor source to calculate the specific mortality per age group and per disease. One hundred years ago... .

A curve without coordinates is useless in mathematics.

What about the history of AIDS?

If it was the first epidemic of this type in the region, it could be of some meaning. But it is not the first time that many young adults have died here. In the 1940s, sudden and mysterious diseases (later qualified as syphilis), caused such havoc that the elderly went to the border of Burundi to look for women to repopulate their villages... those very villages which are said to be, now, devastated by AIDS.

In the collective memory, AIDS is nothing new.

There is a rampant, very subjective feeling that something has gone wrong with the health of the adults. Several decades ago, at a time when life expectancy was low, such a feeling may not have taken off. But since colonisation, people have seen hospitals, medicines, immunisations gradually shortening the usual course of their pathologies, and the present generation has forgotten the heavy toll that Life had to pay when natural immunity, through natural selection, was the only potion for longevity.

MORBIDITY: No need of a microscope.

The western look at health, after peering into so many microscopes, has become myopic. It does not see that, in Africa, it is not so much the tiny microbe which has to be tracked down as the conditions favouring diseases' proliferation.

In the absence of hygiene, treatments and prevention, why should a particular age group be exempt from common germs? To die in labour after nine months of malnutrition, to catch TB when you are an alcoholic, or genital ulcers when you don't look at your partners, should not come as a surprise in adults. No more than to be eaten alive by the billion parasites Africa is swarming with.

Let's leave the eyepiece for a while and look at the reality in real size, even if it is not easy: the dispensary introduced 30 years ago are closed, the hospitals left to the rackets of medical staff, the water supplies brought in by colonisation are all broken down, the pipes, the pumps stolen. The Primary Health Care programmes, deprived of funds and motivation, are piling up in dormant files.

It is not enough to say "We know". But to know the reality should be enough to stop looking frantically for the fashionable latest candidate pathogen. Fashion is a priority for the snobs, only.

NATURAL IMMUNITY: Impossible comeback.

Now that the western input in health is vanishing, could it be that natural selection is regaining its prerogative and coming again to play the moderator?

The field is open, indeed, but a hundred years of civilisation have completely changed the landscape.

In the remotest bush it has become a habit to fight boredom with a cocktail of local "bangi" (marijuana) and amphetamines.
Self-medication is the common behaviour of the many who cannot afford to see the doctor. A dulleder antibiotics are on the open market, cheaper than genuine drugs, and getting cheaper every time buying power breaks down.

During Socialist times, Medicare was free. Since the IMF became responsible for the Health budget, only vaccinations are free. Not only free, but hammered in: BCG, DPT-POLIO at birth, Measles at nine months, tetanus to girls in primary schools....No adult in Kagera has not got ten ritual injections. There are no roads, no sterilisation at the dispensary, no refrigerators for the vaccines on the way, but the magic Extended Programme of Immunisation (EPI) like Superman, is always in time. No matter how skinny, how feverish the buttocks - immunisation will not miss. Here comes the last, but not least, symbol of western magic!

Fighting back the environment with chemicals and immunisation may not only block the road to natural selection, it may also change the very nature of the environment. This is now the third generation of Africans using chemotherapy. Western research, in the hands of commercial labs, has no time to study the long-term effects of its products. It is for the consumer to pay and take the risks as well. In the States, girls develop breast cancer because of the contraceptive pill taken by their mothers. But who is ready to question the western approach to health? Who will take the time to study the impact of drugs on the immune system? The black continent, since it has always been a reservoir of guinea pigs for the “Institutes of Tropical Medicine”, could be a perfect cluster for such a study. Africans have already got their share of intoxication. Anyhow, a minimum of scientific curiosity should force us to open the debate, especially when AIDS, its diagnosis, its treatment, its evolution present to Science a challenge that billions of dollars are not able to meet.

SEX AND BABIES: The divorce.

Kagera, epicentre of AIDS, is in a serious deadlock. No hope of regaining the benefits of natural selection which renders epidemics temporary; no possibility to survive by producing a surplus of children because the only remedy to lethal disease now is a condom.

In such a trap, the collective consciousness is overcome by panic thrown into extreme behaviours, from denial to suicide. Africa has survived all the catastrophes of its history by having always associated sexuality and procreation. If now, because of a virus, unprotected sex turns out to be a death sentence, Africa is lost.

As long as the AIDS Control Programmes (ACPs) will not change the message about sexual transmission, they can only contribute to the general panic.

It is legitimate to fight STD’s. But when it comes to AIDS, ACP’s should know, and their mentors in Geneva should know, that a cent invested to create employment can do more for the young Tanzanian that the fortune spent in printing and lecturing morals.

It is common sense.

Unfortunately, common sense, simple reasoning, have a low resistance to the pressures of AIDS. The so-called “Lethal Sexually Transmitted Disease” has the power to trigger all the fantasies we associate with the coupling of SEX/DEATH. The brain freezes instead of reasoning, we issue feelings. In order to save Africa, we lock her in a brothel.

FIELD WITNESS: The elephant in the china store.

I have had the opportunity to be here at the “epicentre of AIDS” for 8 years and to observe, in total freedom, the slow crumbling of a society.

I don’t know more, I don’t know less than statisticians and epidemiologists, but in contrast to them, I live here, linked to 70 000 people at the border with Uganda.

The little money I can get thanks to a private organisation I spend to rebuild schools, to treat malaria, to offer work to the jobless.

I count up the living, I count down the dead. I try to understand.

Clinically confirmed immunodeficiency is seen at all ages, in both sexes. Some cases are transient, reversed by treatment or change of behaviour, even reverting spontaneously. Others die beyond remedy.

Others will never tell us what could have been their outcome: cases untreated, cases caught too late, cases poisoned by treatment ignorant of the dosage, the side-effects, the interaction between drugs. They are by far the most frequent.

Eight years of observations have me convinced of two things:

1. It is impossible to make a real prognosis in immunodeficiency.
2. It is impossible to speak of an epidemic of immunodeficiency. The number of cases is a constant proportion of the general mortality.

If “death from exhaustion”, resulting from the degradation of the quality of life, is called epidemic, then it remains to be proved that this “exhaustion” is sexually transmitted - before speaking of AIDS.

In the meantime...some predictions fall down, some correlations build up. For instance, the forest of tombs predicted 10 years ago is not there. Instead, what is every day more noticeable is the fast replication of HIV in the presence of money. MAJUTO NI MJUKUU is a Swahili saying, when tears come too late: Regrets turn into our children’s children. If the orphans of the next generation do not dare to accuse us for having invested fortunes in microscopes, they may well accuse us for having locked both eyes, and for so long, into molecular emptiness.

* prognosis: a forecast or advance indication
I have lived in this country England for ten years. I have never seen or heard of any Ugandan young or old dying of any illness other than so-called HIV-related illnesses in this country or even back home in Uganda. Why?

W henever you ask what happened (when someone dies) the answer is, “Why did he die?” I think that shows how ignorant our community is about these controversial issues. Ignorance kills. It has proved to be one of the major killers of all time in our community and around the world. This will not stop unless we educate our people. And education is never widespread when there is big money and politics involved.

W hile pharmaceutical companies are making a lot of money, many lives are being lost and others ruined. I think it is high time those who are classed as HIV-high-risk come together and examine that label that is sending them to early graves. Something has to be done and it is up to the “HIV-high-risk” groups themselves to take an active role. No-one will do it for you, no-one will help you do it unless you empower yourself and say no to the big-money-making liars. I am not an expert and therefore many of you might not take me seriously but there are things we have to realise. Experts try hard to tell us what to do and how to live our lives, but having a tablet for everything is not the answer. We have our natural common sense. One cannot fight nature, and this includes how the body works naturally.

Good doctors know that people have brains and try to give their patients enough support to be able to use their common sense with the help of their doctor’s medical knowledge.

The so-called HIV-high-risk groups refer to gay people, IV drug using people, people from sub-Saharan Africa, people with haemophilia. What is difficult to understand is why no-one ever gives a clear definition of the criteria used in determining these people as “high risk”. How on earth will this go on? How did the HIV/AIDS industry come up with this kind of classification? As far as I’m concerned malaria is malaria and syphilis is syphilis and so forth. The “HIV test” changes every other day, the life expectancy of its sufferers differing all the time. Yet we all know there is no “cure” offered apart from the toxic drugs that can be fatal. Their sickening side-effects make “HIV/AIDS” patients worse with new problems such as nausea, sickness, liver failure, bone marrow toxicity, severe reduction of red and white blood cells, kidney failure etc. and people still take them apparently to “postpone the onset of AIDS symptoms”!! Is it not abusive for doctors to encourage their patients to take these kinds of medications? No! It is not! Because being HIV-diagnosed definitely leaves the doctor with the power to conclude that you will suffer from AIDS and die anyway... I have seen many doctors complain that African men and women do not take their medications properly: they “become resistant” causing them to die quickly! Easy to say, and it suits very well with HIV/AIDS scientific practice. Ironically many Africans are unaware of the side-effects, and because of the language and cultural differences, and the advancement in new technology, they are not familiar enough to ask questions. Many Africans still believe the white race is superior so they (whites) do not make mistakes. I wish they (my fellow black Africans) acknowledged how horribly things can go wrong. Such as the public example of a recent scandal involving a London Health Authority and cancer tests.

M y wish is to see more and more Africans become assertive and avoid dying of passiveness. In this case passivity kills. The slogan of ‘confidentiality’ in relation to HIV/AIDS seems a great industry of its own. This causes more harm than good in those diagnosed HIV positive. It means the diagnosed person cannot trust anyone but his/her doctor and other ‘service providers’. The treatment at the hands of the service providers can sometimes be more cruel than the HIV-positive status itself. Questions upon questions planning one’s death alone with strangers can be very haunting and extremely frightening. The service providers continue to plan deaths one after another leaving the patient with little choice but to believe that no-one else has any knowledge to offer. By the time most of them remember to call upon their loved one or closest relatives it is often too late.

African families never talk to strangers about their personal problems. They are brought up to tell doctors the type of discomfort they feel, but not their private lifestyle: arriving in Europe, especially in the United Kingdom, every landlord/lady happy to take advantage of them for their housing benefit payments; living in horrendous situations: houses damp with no central heating, uncleaned surroundings, no ventilation and no hoovering facilities. Self-evidently people living in such a situation are prone to ill-health. In search of a better life, they end up working in the poorest conditions while being exploited by their employers. Some of these African people do not even know that a work-place can be hazardous. They have little or no knowledge of allergies. They start saying that white people have very awkward illnesses without realising they are in line. Things like pneumonia, influenza and pollution-related illnesses are rarely heard of in the tropics. Africans naturally do not expect to be affected by these. There is no information available to enlighten them about the different environment and its problems.

M ost doctors interpret symptoms they see any way they choose; this has created some of the serious problems within the Anglo-African community. The inability of most Africans to tell their doctors what they actually do, eat and are exposed to, leaves them in a deeply vulnerable situation. Come on, wake up and get a life!
I published the following picture in 1965 in a paper entitled "Viremia in Friend Leukemia: the electron microscope approach to the problem" which appeared in Pathologie-Biologie, vol 13, pp. 125-134. Transmission electron microscopy was used to verify the success of a method for virus purification which I had developed when working at the Sloan Kettering Institute in New York. The method was as follows: About 20 ml of blood from leukemic DBA/2 mice was collected, blood cells were removed by low speed centrifugation and the plasma was diluted 1/1 with cold heparinized Ringer's solution. The diluted plasma was cleared from contaminating debris by two consecutive steps of Millipore ultrafiltration, using pore size 0.65 µ first and 0.22 µ next. The second filtrate was then spun at high speed at 30000 g for 2 hours. The resulting pellet, about 1 mm in diameter, was immediately fixed with osmium tetroxide, embedded in epoxy resin and prepared for electron microscopy by routine thin sectioning methods. Aliquots of the unfixed pellet were resuspended in Ringer's solution and used for titration of the leukemogenic activity in adult DBA/2 mice, known to be 100% susceptible to the virus. It was that simple!

The picture shows, at a magnification of 19500 x, an almost pure population of typical "type C" viruses (not yet called retrovirus in 1965...). Three arrows point at contaminating debris and microvesicles. The interpretation of these EM pictures was that virus purification was satisfactory and that contamination rate was extremely low.

Dangerously enough, EM was progressively dismissed in retrovirus research after 1970. Molecular biologists started to rely exclusively on various "markers", and what was sedimenting in sucrose gradient at density 1.16 gm/l was regarded as "pure virus". It is only in 1997, after fifteen years of intensive HIV research, that elementary EM controls were performed, with the disastrous results recently reviewed in Continuum. How many wasted efforts, how many billions of research dollars gone in smoke...

Horrible.

Errare humanum est sed diabolicum perseverare....

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Dr Etienne de Harven is emeritus Professor of Pathology, University of Toronto. He worked in electron microscopy (EM) primarily on the ultrastructure of retroviruses throughout his professional career of 25 years at the Sloan Kettering Institute in New York and 13 years at the University of Toronto. In 1956 he was the first to report on the EM of the Friend virus in murine (mouse) leukemia, and in 1960, to coin the word "budding" to describe steps of virus assembly on cell surfaces.
It is generally accepted that Peyton Rous discovered retroviruses in 1911 when he induced malignancy in chickens by injections of cell-free filtrates obtained from a muscle tumour. Similar experiments were repeated by many researchers and the tumour inducing filtrates became known as filterable agents, filterable viruses, Rous agents, Rous virus. However, Rous himself expressed doubts that the agents which caused tumours were infectious in nature. Indeed, Rous warned, “The first tendency will be to regard the self-perpetuating agent active in this sarcoma of the fowl as a minute parasitic organism. Analogy with several infectious diseases of man and the lower animals, caused by ultramicroscopic organisms, gives support to this view of the findings, and at present work is being directed to its experimental verification. But an agency of another sort is not out of the question. It is conceivable that a chemical stimulant, elaborated by the neoplastic cells, might cause the tumour in another host and bring about in consequence a further production of the same stimulant”.1 In 1928, AE Boycott, the President of the Royal Society of Medicine, Section of Pathology, in his Presidential Address entitled “The Transition from Live to Dead: the Nature of Filtrable Viruses”, said: “Another analogous phenomenon takes us, I think, a step further. The products of autolysis of dead cells in the body, in suitable concentration, stimulate tissue growth. It is a beautiful self-regulating mechanism in which the amount of stimulus is proportionate to the amount of cell destruction, and therefore to the amount of cell growth required, and it is obviously of the highest importance for survival - a far more potent factor in selection and evolution than any disease has ever been. As it normally operates in healing our cut

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**Eleni Papadopulos-Eleopulos**

Eleni Papadopulos-Eleopulos is a biophysicist and leader of a group of HIV/AIDS scientists from Perth in Western Australia. Over the past decade and more she and her colleagues have published many scientific papers questioning the HIV/AIDS hypothesis including the widely referenced critical study Is A Positive Western Blot Proof of HIV Infection in Nature/Bio/technology (now Nature/Bio/technology) in March 1993 and a comprehensive critique The Isolation of HIV: Has it really been achieved? The Case Against in Continuum, October 1996.
fingers, the final result is simply the restoration of the cells which were destroyed. But if the normal restraint exercised by neighboring tissues is evaded and use made of tissue cultures, the products of autolysis or metabolism (in the form of extracts of tissues, tumors, or embryos) stimulate growth indefinitely and a much larger quantity of tissue may be obtained than we started with. From the autolysis of this larger amount of stimulating substance may be obtained, and there seems no reason why this process of multiplication should have any limits: normal tissues in the physical isolation of tissue cultures are as immortal as malignant tissues in their physiological isolation from the rest of the body. These products of autolysis have not received nearly as much attention as they deserve, but they are probably of relatively simple and discoverable constitutions. Yet applied to cells they cause growth, and in so doing potentially increase their own quantity; this is very much what the Rous agent does. As to its origin, all the evidence seems to concur in indicating that the Rous agent arises de novo in each tumor. There is no epidemiological evidence that cancer comes into the body from outside; everything we know supports the classical view that it is a local autochthonous disease. Experimental sarcomas produced by embryonic extract and indol, arsenic or tar have been transmitted by filtrates. Epitheliomas are easily produced in mice by tar and in men by chronic irritation; and if we believe that all malignant tumors contain more or less of a carcinogenic agent akin to the Rous agent, it follows that we can with a considerable degree of certainty stimulate normal tissues to produce viruses.2

Twenty years later in an article entitled The Plasmagene Theory of the Origin of Cancer, D'Arlington, discussing the induction of cancer by the Rous agent, the filtrable viruses and the "self-propagating" particles transmitted by heredity but lying outside the nucleus found in plants and "known as plasmagenes", wrote: "These infections, it will be seen, are artificial, or at least unnatural. Now the distinction between natural and artificial infection has long been known, although little regarded, in the discussion of plant viruses. A number of aberrant conditions can be transmitted from stock to scion, and some even have arisen in a scion after it has been grafted on a healthy stock. These are artificial diseases; they are not transmitted in nature, but only by grafting. Some may have arisen by the mutation of self-propagating proteins in the cells of plants propagated over long periods by vegetative means (as tumors can be). Others have certainly arisen by the migration or transplantation of proteins from one organism to another. In either case they have a property of infection which they can reveal only in artificial circumstances. We make a great mistake therefore in calling them viruses; they are proviruses. One more question is worth answering: What form would the mutant protein be likely to take in the tumour cell? On account of its rapid multiplication it might well show a higher degree of aggregation than its progenitor. It would then appear as an alien particle in the mutant cell. This is borne out by the electron microscope observations on two chicken tumor agents of provirus type by Claude, Porter and Pickels (1947).3

The electron microscope observation by Claude et al is the first report of virus-like particles in a tumor, the first electron micrographs of the "Rous virus". Soon after many other researchers reported this type of particles in many tumors, and as Boycott predicted in "stimulated normal tissues", as far as D'Arlington's prediction that these particles may be due to "a higher degree of aggregation" of the cytoplasm it may be interpreted as noting that (a) for proteins, nucleic acids or protein/nucleic acid aggregation (condensation, contraction) to take place, oxidation is necessary;4 (b) tumor tissues are oxidized;4 (c) all the agents used to "stimulate normal tissues" to induce retroviruses are oxidizing agents.5-7

In the 1940s, following the development of the electron microscope (EM) and the technique of ultracentrifugation in density gradients, the particles observed in malignant tissues could be isolated and thus purified, that is, separated from everything else. Because these particles were seen in malignant tissues "it has been judged that the particles constitute the aetiological agent of the disease" and by the 1950s Rous's filtrable agents became known as oncoviruses (onco= tumor). The principal morphological characteristic of these particles is a restricted range of diameters and the main physical characteristic their density.8 When the ultrastructure of these particles was determined they were defined as particles with a diameter of 100-120nm containing "condensed inner bodies (cores)" and surfaces "studded with projections (spikes, knobs)".9 By the 1950s well-known retrovirologists such as JW Beard, recognised that cells including uninfected cells, under various conditions, were responsible for the generation of a heterogeneous array of particles, some of which may look like oncoviruses. This "particle problem" led to the opinion that to prove the existence of a retrovirus "the scheme of approach, as well illustrated by that devised and rigorously tested in investigations of viral agents, is relatively simple. This consists in (1) isolation of the particles of interest; (2) recovery (purification) of the particles in a given preparation that are homogeneous with respect to particle kind; (3) identification of the particles, and (4) analysis and characterisation of the particles for the physical, chemical, or biological properties desired". Beard also stressed that "identification, characterisation, and analysis are subject to well-known disciplines established by intensive investigations, and the possibilities have by no means been exhausted. Strangely enough, it is in this field that the most frequent shortcomings are seen. These are related at times to evasion of disciplines or to their application to unsuitable materials. As was foreseen, much of the interest in the more tedious aspects of particle isolation and analysis has been diverted by the simpler and undoubtedly informative processes of electron microscopy. While much can be learned quickly with the instrument, it is nevertheless clear that the results obtained with it can never replace, and all too often may obscure, the need for the critical fundamental analyses that are dependent on access to homogeneous materials".10 (italics ours).
preferred technique for purification of RTV". At a European meeting on the use of centrifugation in density gradients held at the Pasteur Institute in 1972 with Jean-Claude Chermann as its secretary, it was stressed that once the culture fluids (supernatants) are banded, the density band at which retroviruses are trapped (this varies slightly with the substance used to manufacture the gradients), must be thoroughly assayed.

The assays consist of the following:

"Assays for RNA Tumor Viruses
Physical
Electron Microscopy (neg stain and thin sect.)
Virus count
Morphology
Purity
Biochemical
Reverse transcriptase
60-70S RNA, total RNA
Total protein
Gel analysis of viral and host proteins and nucleic acids
Immunological
Gel diffusion
Complement fixation*
Immuno-fluorescence*
Biological
Infectivity in vivo
Infectivity in vitro

*With specific reagents for enveloped and internal antigens g and env".

(RReverse transcriptase is an enzyme first discovered in oncoviruses in 1970 hence their present name retroviruses, and 60-70S RNA, the viral RNA. Retroviruses are sometimes called RNA tumour viruses because their genome consists of RNA and not DNA).
any other infectious agent, all cells contain retroviral genomes which under appropriate conditions may be expressed in culture. That is, may lead to the appearance of retroviruses known as endogenous retroviruses. It follows that both the cells in the culture from which the original particles were obtained as well as the culture into which they were introduced may release identical retroviral particles even if the particles that were introduced were not infectious. Therefore it is absolutely imperative to have suitable controls.

Thus, to prove the existence of a retrovirus, one must isolate and analyse the retroviral-like particles twice. The first time to obtain and analyse the particle constituents released in the first culture. The second time to prove that the particles released, if any, by the cell in the second culture, are identical to the ancestral particles. The crucial caveat in this procedure is the use of experimental techniques to control for the effects of cocultivation, chemical agents and the many other factors which themselves may induce retroviral phenomena independent of exogenous retroviral infection.15-17

In conclusion, by the early 1980’s, retrovirologists agreed that to prove the existence of retroviruses one must first isolate (purify) candidate particles and the method to achieve this was by banding in a density gradient.

**SUMMARY OF MONTAGNIER AND COLLEAGUES 1983 SCIENCE PAPER**

In 1983 Luc Montagnier and his colleagues from the Pasteur Institute and other French researchers published a paper which is considered the first study in which the existence of “HIV” was proven. The paper is entitled “Isolation of a T-Lymphotropic Retrovirus from a Tumor of a Patient with Acquired Immune Deficiency Syndrome (AIDS)” with Françoise Barré-Sinoussi as principal and Jean-Claude Chermann as second author. The authors’ claim to have isolated a retrovirus and thus proven its existence was based on the following experiments:

1. **Lymphocytes from the lymph nodes of two patients with lymphadenopathies as well as peripheral blood mononuclear cells from these patients were put in culture medium with phytohemagglutinin (PHA), T-cell growth factor (TCGF), and antiserum to human interferon...In the mouse system, we had previously shown that antiserum to interferon could increase reverse virus production by a factor of 10 to 50.**

2. **Normal umbilical cord lymphocytes were cultured for three days (culture conditions not given), after which supernatants from the coculture and polybrene were added.** “After a lag period of 7 days, a relatively high titer of reverse transcriptase activity was detected”. (In fact the activity was relatively low, no more than 8,000 counts/min. Background activity as high as 4000 counts/min have been reported.) “Identical cultures” to which supernatant has not been added remained negative. (Since no supernatant was added the cultures could not have been identical. Since supernatant from non-infected cultures added to normal non-infected cells leads to the appearance of endogenous retroviruses this is also a significant difference). Commenting on the findings in the three experiments the authors wrote: “These two successive infections clearly show that the virus could be propagated on normal lymphocytes from either newborns or adults”. The data from the three experiments apparently were also considered proof of “isolation”, however, “That this new isolate was a retrovirus was further indicated by its density in a sucrose gradient, which was 1.16”.

3. **Normal umbilical cord lymphocytes were cultured for three days (culture conditions not given), after which supernatants from the coculture and polybrene were added. “After a lag period of 7 days, a relatively high titer of reverse transcriptase activity was detected”.** (In fact the activity was relatively low, no more than 8,000 counts/min. Background activity as high as 4000 counts/min have been reported.) “Identical cultures” to which supernatant has not been added remained negative. (Since no supernatant was added the cultures could not have been identical. Since supernatant from non-infected cultures added to normal non-infected cells leads to the appearance of endogenous retroviruses this is also a significant difference). Commenting on the findings in the three experiments the authors wrote: “These two successive infections clearly show that the virus could be propagated on normal lymphocytes from either newborns or adults”. The data from the three experiments apparently were also considered proof of “isolation”, however, “That this new isolate was a retrovirus was further indicated by its density in a sucrose gradient, which was 1.16”.

4. **The evidence from the sucrose gradients consisted of two parts:**

   (a) **The supernatant from the cord blood lymphocytes in which RT activity was detected was banded in sucrose density gradients. Maximum RT activity was reported at the 1.16g/ml band.**

   (b) **To the cord blood lymphocyte culture in which RT activity was detected [25S] methionine was added, that is radioactive methionine, an amino acid which is incorporated into growing protein chains and whose radioactivity allows detection of such proteins. Two types of experiments were performed with this culture, one with the cells and the other with the supernatant:**

   (i) **A cell extract was lysed (broken apart) and centrifuged. To parts of the cellular supernatant various sera (containing antibodies) were added and the proteins were electrophoresed (separated using an electric field) on a polyacrylamide-SDS slab gel. Many proteins were found to react, not only with the sera from the two patients with multiple lymphadenopathies but also with sera from a healthy donor and a normal goat.**

   (ii) **The culture supernatant was banded in a sucrose density gradient. Although no mention is made of EM studies of the 1.16g/ml band, it was claimed that the band represented “purified, labelled virus from patient 1”. The 1.16g/ml band was reacted with the sera of the two patients as well as two healthy blood donors and was processed in the same way as the cellular extract. Although published manuscripts it is virtually impossible to distinguish proteins reacting with any sera, even with the sera from the two patients, in the text it is stated that “when purified, labelled virus [the 1.16g/ml band] was analysed [reacted with the sera] three major proteins could be seen: the p25 protein and proteins with molecular weights of 80,000 and 45,000. The 45K protein may be due to contamination of the virus by cellular actin which was present in immunoprecipitations of all cell extracts” (italics ours).**

EM studies of the cord blood lymphocytes culture “showed characteristic immature particles with dense crescent (C-type) budding at the plasma membrane...The virus is a typical type-C RNA tumour virus.”
3. **HL23V, 1975**

Gallagher, R.E., Gallo, R.C.

“Today nobody, not even Gallo, considers "HL23V" as being the first human retrovirus or even a retrovirus.” [see p. 36]

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None published in the scientific literature

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5. **Banded, purified HIV, 1997**


“Purified HIV-1 preparations are contaminated by cellular vesicles. Purified vesicles from infected H9 cells (a) and activated PBMC (b)...or from non-infected H9 cells (c)”

Note: The authors themselves do not claim that (a) and (b) represent purified “HIV” but “purified vesicles”.

Luc Montagnier was born in Chabris, India in 1932. He obtained his doctorate in Medicine at Paris University in 1960. Since 1972 he has been head of the Viral Oncology Unit at the Pasteur Institute, Paris. In 1991 he became Head of the Department of AIDS & Retroviruses at the Pasteur Institute. In 1993 he became President of the World Foundation for AIDS Research and Prevention, and in 1997, Distinguished Professor at the Queen’s College, City University of New York. He became a member of the French Academy of Sciences in 1996.

Djamel Tahi has worked in the movie and television business for twenty years as a film and tape editor, and since 1990 as a writer and film director. He is author and director of a three hour programme about AIDS, including a 52 minute documentary about the AIDS controversy entitled AIDS: the doubt, broadcast on European TV channel ARTE in March 1996.

This interview will form part of a book to be published this year entitled A Conversation about AIDS with Professor Montagnier in which the French scientist talks to author Djamel Tahi on aspects of AIDS research.

The answers by Luc Montagnier have been numbered for easier reference to the analyses in the following article.

A group of scientists from Australia argues that nobody up till now has isolated the AIDS virus, HIV. For them the rules of retrovirus isolation have not been carefully respected for HIV. These rules are: culture, purification of the material by ultracentrifugation, Electron Microscopic (EM) photographs of the material which bands at the retrovirus density, characterisation of these particles, proof of the infectivity of the particles.

No, that is not isolation. We did isolation because we “passed on” the virus, we made a culture of the virus. For example Gallo said: “They have not isolated the virus...and we (Gallo et al.), we have made it emerge in abundance in an immortal cell line.” But before making it emerge in immortal cell lines, we made it emerge in cultures of normal lymphocytes from a blood donor. That is the principal criterion. One had something one could pass on serially, that one could maintain. And characterised as a retrovirus not only by its visual properties, but also biochemically, RT [reverse transcriptase] activity which is truly specific of retroviruses. We also had the reactions of antibodies against some proteins, probably the internal proteins. I say probably by analogy with knowledge of other retroviruses. One could not have isolated this retrovirus without knowledge of other retroviruses, that’s obvious. But I believe we have answered the criteria of isolation. Totally.

Let me come back on the rules of retrovirus isolation which are: culture, purification at the density of retroviruses, EM photographs of the material at the retro-
Did Luc Montagnier discover HIV?

“I repeat, we did not purify!”

virus density, characterisation of the particles, proof of the infectivity of the particles. Have all these steps been done for the isolation of HIV? I’d like to add, according to several published references cited by the Australian group, RT is not specific to retroviruses and, moreover, your work to detect RT was not done on the purified material?

I believe we published in Science (May 1983) a gradient which showed that the RT had exactly the density of 1.16. So one had a peak which was RT. So one has fulfilled this criterion for purification. But to pass it on serially is difficult because when you put the material in purification, into a gradient, retroviruses are very fragile, so they break each other and greatly lose their infectivity. But I think even so we were able to keep a little of their infectivity. But it was not as easy as one does it today, because the quantities of virus were nonetheless very weak. At the beginning we stumbled on a virus which did not kill cells. The virus came from an asymptomatic patient and so was classified amongst the non-syncytia-forming, non-cytopathogenic viruses using the co-receptor ccr5. It was the first BRU virus. One had very little of it, and one could not pass it on in an immortal cell line. We tried for some months, we didn’t succeed. We succeeded very easily with the second strain. But there lies the quite mysterious problem of the contamination of that second strain by the first. That was LAI.

Why do the EM photographs published by you, come from the culture and not from the purification?

There was so little production of virus it was impossible to see what might be in a concentrate of virus from a gradient. There was not enough virus to do that. Of course one looked for it, one looked for it in the tissues at the start, likewise in the biopsy. We saw some particles but they did not have the morphology typical of retroviruses. They were very different. Relatively different. So with the culture it took many hours to find the first pictures. It was a Roman effort! It’s easy to criticise after the event. What we did not have, and I have always recognised it, was that it was truly the cause of aids.

How is it possible without EM pictures from the purification, to know whether these particles are viral and appertain to a retrovirus, moreover a specific retrovirus?

Well, there were the pictures of the budding. We published images of budding which are characteristic of retroviruses. Having said that, on the morphology alone one could not say it was truly a retrovirus. For example, a French specialist of EM’s of retroviruses publicly attacked me saying: “This is not a retrovirus, it is an arenavirus”. Because there are other families of virus which bud and have spikes on the surface, etc.

W hy this confusion? The EM pictures did not show clearly a retrovirus?

At this period the best known retroviruses were those of type C, which were very typical. This retrovirus wasn’t a type C and lentiviruses were little known. I myself recognised it by looking at pictures of Equine infectious anaemia virus at the library, and later of the visna virus. But I repeat, it was not only the morphology and the budding, there was RT...it was the assemblage of these properties which made me say it was a retrovirus.

About the RT, it is detected in the culture. Then there is purification where one finds retroviral particles. But at this density there are a lot of others elements, among others those which one call “virus-like”.

Exactly, exactly. If you like, it is not one property but the assemblage of the properties which made us say it was a retrovirus of the family of lentiviruses. Taken in isolation, each of the properties isn’t truly specific. It is the assemblage of them. So we had: the density, RT, pictures of budding and the analogy with the visna virus. Those are the four characteristics.

But how do all these elements allow proof that it is a new retrovirus? Some of these elements could appertain to other things, “virus-like”...

Yes, and what’s more we have endogenous retroviruses which sometimes express particles - but of endogenous origin, and which therefore don’t have pathological roles, in any case not in AIDS.

But then how can one make out the difference?

Because we could “pass on” the virus. We passed on the RT activity in new lymphocytes. H. We got a peak of replication. We kept track of the virus. It is the assembly of properties which made us say it was a retrovirus. And why new? The first question put to us by Nature was: “Is it not a laboratory contamination? Is it perhaps a mouse retrovirus or an animal retrovirus?”. To that one could say no! Because we had shown that the patient had antibodies against a protein of his own virus. The assemblage has a perfect logic! But it is important to take it as an assemblage. If you take each property separately, they are not specific. It is the assemblage which gives the specificity.
But at the density of retroviruses, did you observe particles which seemed to be retroviruses? A new retrovirus?

At the density of 1.15, 1.16, we had a peak of RT activity, which is the enzyme characteristic of retroviruses. 9

But could that be something else?

No, in my opinion it was very clear. It could not be anything but a retrovirus in this way. Because the enzyme that F. Barre-Sinoussi characterised biochemically needed magnesium, a little like HTLV elsewhere. It required the matrix, the primer also which was completely characteristic of an RT. That was not open for discussion. At Cold Spring Harbour in September 1983, Gallo asked me whether I was sure it was an RT. I knew it, F. Barre-Sinoussi had done all the controls for that. It was not merely a cellular polymerase, it was an RT. It worked only with RNA primers, it made DNA. That one was sure of. 10

With the other retroviruses you have met in your career did you follow the same process and did you meet the same difficulties?

I would say that for HIV it is an easy process. Compared with the obstacles one finds for the others...because the virus does not emerge, or indeed because isolation is sporadic - you manage it one time in five. I am talking about current research into other illnesses. One can cite the virus of Multiple Sclerosis of Prof. Peron. He showed me his work a decade ago and it took him around ten years to finally find a gene sequence which is very close to an endogenous virus. You see...it is very difficult. Because he could not "pass on" the virus, he could not make it emerge in culture. Whereas HIV emerges like couch grass. The LAI strain for example emerges like couch grass. That's why it contaminated the others. 11

With what did you culture the lymphocytes of your patient? With the H9 cell line?

No, because it didn't work at all with the H9. We used a lot of cell lines and the only one which could produce it was the Tambon lymphocytes. 12

But using these kinds of elements it is possible to introduce other things capable of inducing an RT and proteins, etc.

Agreed completely. That's why finally we were not very ardent about using immortal cell lines. To cultivate the virus en masse - OK. But not to characterise it, because we knew we were going to bring in other things. There are MT cell lines which have been found by the Japanese (MT2, MT4) which replicate HIV very well and which at the same time are transformed by HTLV. So, you have a mix of HIV and HTLV. It is a real soup. 13

What is more it's not impossible that patients may be infected by other infectious agents?

There could be mycoplasmas...there could be a stack of things. But fortunately we had the negative experience with viruses associated with cancers and that helped us, because we had encountered all these problems. For example, one day I had a very fine peak of RT, which F. Barre-Sinoussi gave me, with a density a little bit higher, 1.19. And I checked! It was a mycoplasm, not a retrovirus. 14

With the material purified at the retrovirus density, how is it possible to make out the difference between what is viral and what is not? Because at this density there's a stack of other things, including "virus-like" particles, cellular fragments...

Yes, that's why it is easier with the cell culture because one sees the phases of virus production. You have the budding. Charles Dauguet (an EM specialist) looked rather at the cells. Of course he looked at the plasma, the concentrate, etc...he saw nothing major. Because if you make a concentrate it's necessary to make thinly sliced section [to see a virus with the EM], and to make a thin section it is necessary to have a concentrate at least the size of the head of a pin. So enormous amounts of virus are necessary. By contrast, you make a thin section of cells very easily and it's in these thin sections that Charles Dauguet found the retrovirus, with different phases of budding. 15

When he looks at the published electron microscope photographs, for you as a retrovirologist it is clear it's a retrovirus, a new retrovirus?

No, at that point one cannot say. With the first budding pictures it could be a type C virus. One cannot distinguish. 16

Could it be anything else than a retrovirus?

No.. well, after all, yes... it could be another budding virus. But there's a... we have an atlas. One knows a little bit from familiarity, what is a retrovirus and what is not. With the morphology one can distinguish but it takes a certain familiarity. 17

Why no purification?

I repeat we did not purify. We purified to characterise the density of the RT, which was soundly that of a retrovirus. But we didn't take the peak...or it didn't work...because if you purify, you damage. So for infectious particles it is better to not touch them too much. So you take simply the supernatant from the culture of lymphocytes which have produced the virus and you put it in a small quantity on some new cultures of lymphocytes. And it follows, you pass on the retrovirus serially and you always get the same characteristics and you increase the production each time you pass it on. 18
So, for isolation of retroviruses the stage of purification is not obligatory? One can isolate retroviruses without purifying?

Yes...one is not obliged to transmit pure material. It would be better, but there is the problem that one damages it and diminishes the infectivity of the retrovirus.

Without going through this stage of purification, isn’t there a risk of confusion over the proteins that one identifies and also over the RT which could come from something else?

No...after all, I repeat if we have a peak of RT at the density of 1.15, 1.16, there are 999 chances out of 1,000 that it is a retrovirus. But it could be a retrovirus of different origin. I repeat, there are some endogenous retroviruses, pseudo-particles which can be emitted by cells, but even so, from the part of the genome that provides retroviruses. And which one acquires through heredity, in the cells for a very long time. But finally I think for the proof - because things evolve like molecular biology permitting even easier characterisation these days - it’s necessary to move on very quickly to cloning. And that was done very quickly, as well by Gallo as by ourselves. Cloning and sequencing, and there one has the complete characterisation of the virus. This means: what are the proteins of which it’s composed?

But there comes a point when one must do the characterisation of the virus. This means: what are the proteins of which it’s composed?

That’s it. So then, analysis of the proteins of the virus demands mass production and purification. It is necessary to do that. And there I should say that that partially failed. J.C. Herrmann was in charge of that, at least for the internal proteins. And he had difficulties producing the virus and it didn’t work. But this was one possible way, the other way was to have the nucleic acid, cDNA, etc. It’s this way which worked very quickly. The other way didn’t work because we had at that time a system of production which wasn’t robust enough. One had not enough particles produced to purify and characterise the viral proteins. It couldn’t be done. One couldn’t produce a lot of virus at that time because this virus didn’t emerge in the immortal cell line. We could do it with the LAI virus, but at that time we did not know that.

So the stage of purification is not necessary?

No, no, it’s not necessary. What is essential is to pass on the virus. The problem Peron had with the multiple sclerosis virus was that he could not pass on the virus from one culture to another. That is the problem. He managed it a very little, not enough to characterise it. And these days to characterise means above all at the molecular standard. If you will, the procedure goes more quickly. So to do it: a DNA, clone this DNA, amplify it, sequence it, etc. So you have the DNA, the sequence of the DNA which tells you if it is truly a retrovirus. One knows the familiar structure of retroviruses, all the retroviruses have a familiar genomic structure with such and such a gene which is characteristic.

Gallo did it?

Gallo?..I don’t know if he really purified. I don’t believe so. I believe he launched very quickly into the molecular part, that’s to say cloning. What he did do is the Western Blot. We used the RIPA technique, so what they did was new was they showed some proteins which one had not seen well with the other technique. Here is another aspect of characterising the virus. You cannot purify it but if you know somebody who has antibodies against the proteins of the virus, you can purify the antibody/antigen complex. That’s what one did. And thus one had a visible band, radioactively labelled, which one called protein 25, p25. And Gallo saw others. There was the p25 which he called p24, there was p41 which we saw...

About the antibodies, numerous studies have shown that these antibodies react with other proteins or elements which are not part of HIV. And that they can not be sufficient to characterise the proteins of HIV.

No! Because we had controls. We had people who didn’t have AIDS and had no antibodies against these proteins. And the techniques we used were techniques I had refined myself some years previously, to detect the src gene. You see the src gene was detected by immunoprecipitation too. It was the p60 [protein 60]. I was very dexterous, and my technician also, with the RIPA technique. If one gets a specific reaction, it’s specific.

But we know AIDS patients are infected with a multitude of other infectious agents which are susceptible to...

Ah yes, but antibodies are very specific. They know how to distinguish one molecule in one million. There is a very great affinity. When antibodies have sufficient affinity, you fish out something really very specific. With monoclonal antibodies you fish out really ONE protein. All of that is used for diagnostic antigen detection.

For you the p41 was not of viral origin and so didn’t belong to HIV. For Gallo it was the most specific protein of the HIV? Why this contradiction?

We were both reasonably right. That’s to say that I in my RIPA technique...in effect there are cellular proteins that one meets everywhere - there’s a non-specific “background noise”, and amongst these proteins one is very abundant in cells, which is actin. And this protein has a molecular weight 43000kd. So, it was there. So I was reasonably right, but what Gallo saw on the other hand was the gp41 of HIV, because he was using the Western Blot. And that I have recognised.

For you p24 was the most specific protein of HIV, for Gallo not at all. One recognises thanks to other studies that the antibodies directed against p24 were often found in patients who were not infected with HIV, and even in certain animals. In fact today, an antibody reaction with p24 is considered non specific.
It is not sufficient for diagnosing HIV infection. 27

No protein is sufficient.

No protein is sufficient anyway. But at the time the problem didn’t reveal itself like that. The problem was to know whether it was an HTLV or not. The only human retrovirus known was HTLV. And we showed clearly that it was not an HTLV, that Gallo’s monoclonal antibodies against the p24 of HTLV did not recognise the p25 of HIV. 28

At the density of retroviruses, 1.16, there are a lot of particles, but only 20% of them appertain to HIV. Why are 80% of the proteins not viral and the others are? How can one make out the difference?

There are two explanations. For the one part, at this density you have what one calls microvesicles of cellular origin, which have approximately the same size as the virus, and then the virus itself, in budding, brings cellular proteins. So effectively these proteins are not viral, they are cellular in origin. So, how to make out the difference? Frankly with this technique one can’t do it precisely. What we can do is to purify the virus to the maximum with successive gradients, and you always stumble on the same proteins. 29

The others disappear?

Let’s say the others reduce a little bit. You take off the microvesicles, but each time you lose a lot of virus, so it’s necessary to have a lot of virus to start off in order to keep a little bit when you arrive at the end. And then again it’s the molecular analysis, it’s the sequence of these proteins which is going allow one to say whether they are of viral origin or not. That’s what we began for p25, that failed ... and the other technique is to do the cloning, and so then you have the DNA and from the DNA you get the proteins. You deduce the sequence of the proteins and their size and, you stumble again on what you’ve already observed with immunoprecipitation or with gel electrophoresis. And one knows by analogy with the sizes of the proteins of other retroviruses, one can deduce quite closely these proteins. So you have the p25 which was close to the p24 of HTLV, you have the p18...in the end you have the others. On the other hand the one which was very different was the very large protein, p120. 30

Today, are the problems about mass production of the virus, purification, EM pictures at 1.16, resolved?

Yes, yes. Absolutely. One can see them, one even sees visible bands. 34

Do EM pictures of HIV from the purification exist?

Yes, of course. 31

Have they been published?

I couldn’t tell you...we have some somewhere .. but it is not of interest, not of any interest. 33

Today, with mass production of the virus, is it possible to see an EM, after purification, of a large number of viruses?

Yes, of course. 32

So for you HIV exists?

Oh, it is clear. I have seen it and I have encountered it. 35

end

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Between the lines

a critical analysis of Luc Montagnier’s interview answers to Djamel Tahi

by Eleni Eleopulos and colleagues

1. If “culture, purification of the material by Ultracentrifugation, Electron M Icroscopic (EM) photographs of the material which bands at the retrovirus density, characterisation of these particles, proof of the infectivity of the particles” is not isolation, then why did Montagnier and his colleagues claim in 1983 to have isolated “HIV” by either performing or claiming to have performed all but one (no EM photographs of the banded material) of these procedures? Why in the 1984 paper where they claimed the first isolation of “HIV” from haemophiliacs, as well as in their other studies that year in which they also claim “HIV” isolation, have they either not performed or claimed to have performed all but one of these steps? Why in their study entitled “Characterisation of the RNA dependent DNA Polymerase of a new human T lymphotropic retrovirus (lymphadenopathy associated virus)” did they state that the virus was “purified on sucrose gradient using isopycnic centrifugation (8)? Reference 8 is the paper presented by Sinoussi and Chermann at the 1972 Pasteur Symposium where they stressed the importance of showing that the banded material contained nothing else but particles with “no apparent differences in physical appearance”.

2. The finding of some or all of the phenomena Montagnier outlines are not proof of isolation. These phenomena can be considered only proof for viral detection and then, if and only if, they are specific to retroviruses. The word “isolation” is derived from Latin “insulatus” meaning “made into an island”. It refers to the act of separating an object from all the extraneous matter that is not that object. Here the object of interest is a retroviral particle. The words ‘isolation’ and ‘passing’ have different and distinct meanings. ‘Isolation’ means to obtain an object, a retrovirus particle for example, separate from everything else. ‘Passing’ means to transfer an object (which may or may not be isolated) from one place to another, for example, from one culture to another. Therefore, even if one assumes that the “something” which Montagnier and his colleagues passed from one culture to another by means of transferring cells or culture supernatants was a retrovirus, and that it was passed to an infinite number of successive cultures, it still is not evidence for isolation. For example, if one has a series of bottles containing water in which the first has a dye added, then takes part of the first and puts it in the second, and from the second passes a sample into the third etc, clearly this procedure has not isolated the dye from the water. A culture contains a myriad of things and thus by definition is not evidence for isolation of an object. The only way possible to claim that one has “made a culture of the virus”, is to have had proof for the existence of the virus before making a culture. The only thing which Montagnier and his colleagues have proven is the emergence in the co-culture with “lymphocytes from a blood donor” of RT activity. Detection of an enzyme in a culture, even if specific to retroviruses is not evidence for isolation. For example, the measurement of cardiac or liver enzymes in cases of myocardial infarction or hepatitis respectively cannot be construed as “isolation” of the heart or liver. The finding in the culture of particles with the morphological characteristics of a retrovirus and of reverse transcriptase activity either in the culture or the 1.16g/ml band, even if “truly specific of retroviruses” is not evidence for retroviral isolation. Even if Montagnier and his colleagues knew beforehand that some of the proteins present in the culture or the 1.16g/ml band were retroviral, and the patients had retroviral antibodies which reacted with these proteins, such a reaction is not evidence for isolation. Argument based on analogies, or even on knowledge of other retroviruses, cannot be construed evidence for isolation. For example, observing something in the ocean which looks like a fish (even if it is a fish), is not equivalent to having the fish in your frypan separate from everything else that occurs in the ocean.

3. We agree with Gallo that Montagnier et al did not present proof for “true isolation” of a retrovirus, any retrovirus, either old or new, exogenous or endogenous.

4. The “knowledge of other retroviruses” shows that not all particles with RT activity and “visual properties of retroviruses” are viruses. This is a fact acknowledged even by Gallo well before the AIDS era. It also shows that RT is not “truly specific of retroviruses”. Non-infected cells as well as bacteria or viruses other than retroviruses have RT. According to some of the best known retrovirologists including its discoverers, as well as Nobel Laureate and Director of the US National Institutes of Health, Harold Varmus, reverse transcriptases are present in all cells including bacteria. Indeed RT activity has been reported in many of the cell lines from which “HIV” is “isolated”, including H9 and CEM as well as normal lymphocytes even when they are not infected with “HIV”. Montagnier, Barre-Sinoussi and Chermann themselves have shown that RT activity is not specific to retroviruses. In their 1972 paper Barre-Sinoussi and Chermann wrote: “There was significant activity in the sample zone and the fastest sedimenting peak, consisting mainly of cell debris. This enzymatic activity can be explained by the presence of some virus particles in these regions, and, since similar polynuclease activity has been found in normal cells, may be mainly ascribed to the cellular enzyme”. In this interview, Luc Montagnier answering question 14 says: “For example, one day I had a very fine peak of RT, which F Barre-Sinoussi gave me, with a density a little bit higher, 1.19 and I checked! It was a mycoplasma, not a retrovirus”. How is it then possible for Montagnier to say that RT is specific to retroviruses? We agree that RT activity is characteristic of retrovirus. However, ‘specificity’ does not have the same meaning as ‘characteristic’. Hair is characteristic of human beings but not every animal with hair is human.

5. Isolation means to obtain an object separate from everything else. Retroviruses are particles and no amount of “analogy” can
prove that one has isolated a retroviral particle. "Knowledge of other retroviruses" can be of assistance in choosing the best method to obtain isolation. The "knowledge of other retroviruses" shows that the best, but by no means perfect method to isolate and prove the existence of retrovirus, is to perform isopycnic (identical density of particle and portion of the gradient) banding and to perform all the assays specified at the 1972 Pasteur symposium. The "knowledge of other retrovirus" also shows that there is nothing specific about the morphology of retroviral particles, protein-antibody reactions or even banding at the density of 1.16 g/ml in sucrose density gradients. R retroviral particles band at the density of 1.16 g/ml but not everything at that density, including particles with the morphology of retroviral particles, is a retrovirus. To remind ourselves this is the case, one needs go no further than to consider the "first" human retrovirus, "HL23".

In the mid-1970's Gallo and his colleagues reported the isolation of the first human retrovirus. In fact the evidence for the isolation of "HL23" surpassed Montaginner et al and everybody else's evidence for "HIV" in at least three important aspects [see p. 29]. Unlike "HIV", in the case of "HL23" Gallo's group (a) reported the detection of RT activity in fresh, uncultured leucocytes; (b) did not need to stimulate their cell cultures with various agents; (Both Montaginner and Gallo concede that none of the phenomena which they say prove the existence of "HIV" can be detected unless the cultures are stimulated with several agents); (c) published an electron micrograph of virus-like particles banding at a sucrose density of 1.16 g/ml. However, today nobody, not even Gallo, considers "HL23" as being the first human retrovirus or even a retrovirus. (For a more detailed discussion see Papadopulos-Eleopulos et al.)

One also must not forget the following additional knowledge in relation to retroviruses:
(a) the lesson of the enzyme adenine triphosphatase. Like RT, this enzyme was considered to be specific to retroviruses and at least in the 1950s was used not only for their detection and characterisation but also for their quantification. Y et at present it is accepted that this is one of the most widely spread enzymes.
(b) a much higher percentage of sera from AIDS patients and those at risk react with proteins of endogenous retroviruses than the sera of healthy people, 70% versus 3%.

2.1. It is true that Montaginner and his colleagues found a peak of RT activity at the density of 1.16 g/ml. However, finding this peak is not proof that the band was made up of retrovirus particles either pure or impure. They therefore this evidence cannot be considered that "one has fulfilled this criterion for purification".

2. In the same issue of Science where Montaginner and his colleagues published their study Gallo pointed out that "the viral envelope which is required for infectivity is very fragile, it tends to come off when the virus buds from infected cells, thus rendering the particles incapable of infecting new cells". Because of this Gallo claimed that "cell-to-cell contact may be required for retroviral infection". At present all "HIV" experts agree that for "HIV" infectivity gp120 is absolutely necessary.

In 1993 Montaginner himself said that for the "HIV" particles to be infectious they must first bind to the cellular C D 4 receptor and that "The gp120 is responsible for binding the CD4 receptor". However, to date nobody has published EM of cell-free particles having the dimension of retroviral particles and also knobs, spikes, that is gp120, not even Hans Gelderblom and his colleagues from the Koch Institute in Berlin who have conducted the most detailed electron microscopy studies of the particles present in culture/ co-cultures containing tissues derived from AIDS patients. In one of their latest publications where this matter is discussed they estimate that immediately after being released, "HIV particles" possess an average of 0.5 knobs per particle but also pointed out that "it was possible that structures resembling knobs might be observed even when there was no gp120 present, i.e., false positives". This means that neither Montaginner and his colleagues nor anybody else subsequently could infect the cultures with cells from healthy donors, umbilical cord lymphocytes or any other cultures with the "purified HIV" or, even the cell-free fluids (the culture supernatant) even if the "purified" virus contained nothing else but particles. In other words, it is impossible for Montaginner and his colleagues to have had any infectivity even "a little" with either the culture supernatant or the "purified labelled virus". For the same reason the "second strain" could not be amplified by the "HIV-4" method. In Montaginner et al provided Gallo with cell-free supernatants, it would have been impossible for the Gallo cultures to be contaminated with BR U, LA1 or a mixture.

3. Montaginner's "virus" did not come from an asymptomatic patient but a patient with "lymphadenopathy and asthenia". Neither in their study nor even today, after nearly fifteen years of "HIV", is there proof for the existence of a human retrovirus which has the ability to "kill cells". The study which at present is most often quoted as proving "HIV" kills T4 cells, considered to be the "hallmark" of AIDS, was published in 1984 by M ontaginner and his colleagues. They cultured CD4+ (T 4) cells from a haemophilic patient who was an asymptomatic virus carrier", "in the presence of phytohaemagglutinin (PHA) followed by IL-2". In the culture they detected RT activity and "virus particles characteristics of the small eccentric core". The number of T4 (CD4+) cells in the culture were measured by counting the number of cells able to bind a monoclonal antibody claimed specific for the CD4 protein. The number of cells which were able to do so decreased with time. Discussing their finding they wrote, "This intriguing phenomenon may be due to virus-induced modulation at the cell membrane, or by steric hindrance of the antibody binding site", that is the decrease is not due to cell killing. Given their data, the conclusion that the decrease in T 4 cells is not due to cell killing is not surprising. However, their conclusion that the effect may be induced by "the virus", is surprising. Montaginner and his colleagues were aware of the experimental evidence which showed that under certain conditions, (including exposure to PHA, IL-2 and other oxidising agents) decrease in T4 cells appears in the absence of "HIV". In this type of culture, T-cells lose their CD4 marker and acquire other markers, including CD8, while the total number of T-cells remains constant. Furthermore, they had evidence that in "infected cells, this phenomenon cannot be detected unless the culture is stimulated by substances such as PHA or antigens. (Proteins such as the "non-HIV" proteins present in the "infected" cultures) Given the above facts it is even more surprising that Montaginner and his colleagues did not have controls, that is, cultures of T 4 cells originating from patients who were not at risk of AIDS but who nonetheless were sick and to which they added PHA and IL-2. Such experiments were reported in 1986 by Gallo and his colleagues. They had evidence on three cell cultures which contained 34% CD4 cells to begin with: One culture was "infected" and stimulated with PHA, the other was not infected but was stimulated with PHA and the third was neither infected nor stimulated. After two days of culture, the proportion of CD4+ cells in the stimulated-uninfected and stimulated-infected culture was 30% and 28% respectively, while at 6 days the number was 10% and 3%. The number of CD4+ cells did not change significantly in the non-infected non-stimulated culture. By 1991 Montaginner and his colleagues had performed experiments with uninfected, unstimulated cells when they studied "HIV" induced apoptosis, which was said (and is still said by many), to be the principle mechanism by which "HIV" kills cells. They showed that in acutely "HIV infected" CEM cell cultures in the presence of mycoplasma removal agent, cell death (apoptosis) is maximum at 6-7 days post infection, whereas maximal virus production occurred at Days 10-17, that is, maximum virus production predate the maximum "infected" cells. The "infected" CEM cells and the monocytic cell line U 937, no apoptosis was detected although these cells produced continuously infectious virus. In CD4 lymphocytes isolated from a normal donor, stimulated with PHA and "infected with HIV" in the presence of IL-2, apoptosis becomes detectable 3 days post infection and clearly apparent at 4 days. Intriguingly, on the 5th day apoptosis became detectable in "uninfected", PHA stimulated cells. They concluded: "These
results demonstrate that HIV infection of peripheral blood mononuclear cells leads to apoptosis, a mechanism which might occur also in the absence of infection due to mitogen treatment of these cells.45

In conclusion, all the presently available data shows that “HIV infection” in the absence of stimulating agents neither decrease the T4 cell number, nor induce apoptosis, while stimulating agents (similar to those to which patients at risk of developing AIDS are exposed) do so in the absence of “HIV”. That is, neither the “HIV”, which Montagnier and his colleagues “stumbled” at the beginning, nor any other “HIV” since then has been shown to "kill cells".

3 Retroviruses are not exotic, nuclear or cosmological notions whose postulated existence can only be inferred by indirect observations. They are particles which can be seen, albeit not with the naked eye. Since Montagnier and his colleagues admit to not seeing particles at the 1.16g/ml band having the morphology of retrovirus, to claim the presence of a retrovirus much less a “purified virus” is totally unsubstantiated and defies belief. The 1.16g/ml band can be likened to a fishing net. The difference is that the band traps objects according to their density, not their size. Imagine a fisherman who sees in the ocean many different objects some of which may be fish. He throws the nets, waits, and upon retrieval of the net performs a thorough examination of its contents and shows that it contains many sea creatures but nothing that looks like a fish. Yet strange as it may seem, he claims to have caught fish. In fact, he claims that the net has nothing else but pure fish.

4 Although budding from the cell membrane is the manner in which retroviral particles appear, this process is not virus specific. In other words, just because a particle buds and has the morphological characteristics of retroviral particles does not prove it is a retrovirus. That this is the case can be illustrated by two facts and by quoting two of the best known retrovirologists: “Budding virus-like particles” have been found in non-infected T-cell lines CEM, H9 and C8166; in 2 lines of EBV transformed B-cell lines and in cultures of primary human lymphoid cells from cord blood, which were either PHA stimulated or not and grown with or without serum and in cord lymphocytes directly after Ficol separation.46 (italics ours). Following an extensive, in vivo study conducted by O’Hara and colleagues from Harvard, “HIV particles” were found in 18/20 (90%) of patients with enlarged lymph nodes attributed to AIDS. However, identical particles were also found in 13/15 (87%) of patients with enlarged lymph nodes not attributed to AIDS and at no risk for developing AIDS. These data led the authors to conclude, “The presence of such particles does not, by themselves indicate infection with HIV”.47 In 1986 Gallo and his colleagues discussing the “First isolation of HTLV-III” wrote: “At the time we obtained LAV it was the contention of several experts in virus morphology that the particles shown in the electron micrograph published in Science by Barre-Sinoussi et al was an arena virus...Since we considered the mere detection of virus particles in cultures from AIDS and ARC patients to be insufficient to confirm scientifically our hypothesis that such particles were implicated in the aetiology of the disease, we decided first to obtain specific reagents against the new virus in order to publish definite results concerning AIDS aetiology”.48

According to Peter Duesberg the “HIV” “parties and proteins which could reflect non-viral material altogether”.49

5 In their study Montagnier and his colleagues wrote: “Electron microscopy of the infected umbilical cord lymphocytes showed characteristic immature particles with dense crescent (C-type) budding at the plasma membrane...This virus is a typical type-C RNA tumor virus”. In 1984 Montagnier, Barre-Sinoussi and C Hermann reported that their virus was “morphologically similar to D particles such as those found in M aen-Pfizer virus or the virus recently isolated from simian AIDS”.50 (By 1984 researchers from the primate research centres in the United States claimed the existence of AIDS in monkeys and that the cause of AIDS was a type-D retrovirus similar to the M aen-Pfizer virus, a typical type-D retrovirus and suggested that the monkey AIDS and these retroviruses could be helpful in the study of human AIDS and “HIV”). In the same year, in yet another publication, Montagnier et al claimed that the “HIV” particles had “morphology similar to that of equine infectious anaemia virus (EIAV), and D type particles”. The EIAV and the visna virus are neither type C nor type D retroviruses but lentiviruses, that is, viruses which have totally different morphology and said to induce diseases long after infection. (By the time this paper was published it was realised that patients who had a positive “HIV” antibody test did not develop AIDS immediately, that is, there was a delay between the positive test and the appearance of AIDS.) It is most astonishing that the morphology of one and the same virus is able to change genus from a typical type-C to a typical type-D particle and then to a completely different subfamily, namely a typical lentivirus, apparently at will. (The family Retroviridae is divided into three subfamilies, Oncovirinae, Lentivirinae and Spumavirinae. Oncovirinae are in turn divided into genus type-B, -C, and -D particles. These findings are analogous to describing a new species of mammal as human, a gorilla and an orang-utan).

6 1. Apart from retroviruses other particles may possess “the assemblage of properties” (the density, RT, budding and the analogy with the visna virus). It follows that the detection of particles having this “assemblage of properties” is not proof that the detected particles are retroviruses. In fact, Montagnier and his colleagues did not report the detection of “HIV” particles having this “assembly of properties”. Since Montagnier and his colleagues could not find particles with the morphological characteristics of retrovirus at the “density” of 1.16 gm/ml even after “a Roman effort”, it follows that the evidence for the existence of “HIV” from the density gradient was not only non-specific but was non-existent. (This fact alone is sufficient to dismiss any claim of proof for the existence of a retrovirus, no matter what else they found anywhere including budding particles from the cell surface, retrovirus-like particles in the culture, RT at that density or proteins at the same density which react with patient sera).

2. It is true that Montagnier et al reported RT activity at the density of 1.16/14 ml but since:

(a) Barre-Sinoussi and C Hermann accept that cells and cellular fragments also have RT activity;

(b) at the 1.16g/ml band no particles with the morphological characteristics of retrovirus were seen;

(c) at that density Montagnier et al found cellular fragments, it follows that the evidence for the existence of “HIV” by detecting RT activity at that density was not only not specific but non-existent.

Given the facts that:

(a) there are significant differences in the nature of the budding processes between type-C, type-D particles and lentiviruses and that in 1983 Montagnier et al reported their retrovirus as type-C and in 1984 as either type-C or type-D, and even later that year as EIAV;

(b) visna virus and EIAV are lentiviruses, it follows that at least up mid 1984 Montagnier et al evidence for the existence of “HIV” (if “HIV” is a lentivirus) from “pictures of budding” and the analogy with EIAV and visna virus was not only non specific but non-existent.

Given these facts the culture conditions which Montagnier and his colleagues and all other “HIV” researchers use to detect
"HIV" together with the presently available data on "HIV" and AIDS, it is more probable that "HIV" (if proven to exist) is an endogenous retrovirus rather than an exogenous retrovirus. Part of the data related to the culture conditions can be summarised as follows. In culture, cells sooner or later start to release endogenous retrovirus. The appearance of endogenous retrovirus can be accelerated and the yield increased up to a million fold by stimulating the culture with mitogens, co-cultivation by adding this culture supernatant to HIV-infected cells. Mitogens, typically PHA which was present in the "HIV" preparation. As experiments proved beyond doubt that the decrease in T4 cells (of at least in vitro the observed decrease in T4 cells after "HIV" infection is not due to cell killing but decreased binding of the T4 (CD4) antibody to the cells. Two years later the Gallo team's experiments proved beyond doubt that the decrease in T4 cells (of the CD4 antibody binding) was not due to "HIV" infection but to the PHA which was present in the "HIV" preparation. As mentioned, at the beginning of the AIDS era there was ample evidence that treatment of cell cultures with PHA and other oxidising agents leads to decreased binding of the CD4 antibody and to increase binding of the CD8 antibody, that is, a decrease in T4 cells was accompanied by increase in T8 cells, while the total cell number remained constant. AIDS patients and individuals belonging to the AIDS risk groups are continuously exposed to strong oxidising agents. At present it is accepted that in both AIDS patients and those at risk, the decrease in T4 cells is accompanied by an increase in T8, while the T4 + T8 cell number remains constant. Also, it is of interest to note that as far back as 1985 M ontagnetier wrote: "This syndrome [AIDS] occurs in a minority of infected persons, who generally have in common a past of antigenic stimulation and of immune depression before LAV infection" or, that is, Montagnier recognised that in the AIDS risk group, immune deficiency precedes "HIV" infection. In 1984 M ontagnetier and his colleagues including Barre-Sinoussi and Chessman; stated that "AIDS virus has been isolated in a cell line from a new species and in a viral model in which such viruses [LAV, HTLV-III=HIV] could induce a disease similar to AIDS." Up to today, no such model exists. When pursued by the Nobel Laureate Kary Mullis for even one scientific paper proving the HIV theory of AIDS, Montagnetier himself showed that a positive antibody test reverts to negative and a low T 4 cell count to normal by stopping anal intercourse, which means that the positive outcome is not due to a retrovirus. Having partners who acquired this infection through drug use (Padian herself says that this means that the women may also be IV users); (iii) the presence in the female of ST D S. (antibodies to their own oxidising agents may cross-react with the "HIV" proteins). A negative male partners of positive female index cases only two seroconverted. They estimated that the likelihood of female-to-male transmission was 8 times lower than for male-to-female. As Padian questioned the validity of these two cases. For the first one she gave several reasons in 1991, when this case was reported for the first time. In the second case they mentioned the fact that "chlamydia was transmitted simultaneously or close to the transmission of HIV is striking", that is, the positive "HIV" antibody test appeared at the time when he became infected with chlamydia. In the prospective study, starting in 1990, "We followed 175 HIV-discordant couples over time, for a total of approximately 282 couple-years of follow-up...The longest duration of follow-up was 12 visits (6 years). We observed no seroconversions after entry into the study...At last follow-up, couples were much more likely to be abstinent or to use condoms consistently...N nevertheless only 75% reported consistent condom use in the 6 months prior to their final follow-up visit." Note: Not only seroconversion were reported only in the cross-sectional study but all the cases were diagnosed before 1990. However: (i) All the "HIV" experts agree that the specificity of the test kits used then was inferior to those used at present; (ii) T he W B criteria used to define "infection" are not sufficient at present. Even if one accepts Padian et al data from the cross-sectional study, they have estimated the risk to a non-infected male of acquiring "HIV" infection from his infected female partner per contact is 0.00011 (1/9000). This means that on average, males having sexual intercourse daily with an infected female partner for sixteen years (that is, 6000 contacts at 365 per year), would score a 50% probability of becoming infected. If sexual intercourse takes...
place on average weekly then it would take one hundred and fifteen years to reach the same probability. Under such circumstances one must question how “HIV” could become epidemic as the result of bi-directional heterosexual transmission.

1. In the Montagnier et al 1983 study, the detection of nothing else but RT activity in the stimulated cultures of lymphocytes from a healthy blood donor was considered proof of passing the retrovirus from the gay man’s lymphocytes to the donor’s lymphocytes and also for virus isolation. However, passing an activity (RT) is not the same as passing an object (retrovirus). Furthermore, since non-“HIV” infected lymphocytes as well as many bacteria and viruses other than retrovirus possess RT activity (RT activity has been reported in many “non-HIV” infected cell lines used to isolate HIV such as H9 and CEM and as far back as 1972 in normal, PHA stimulated lymphocytes), finding RT activity in successive lymphocyte cultures each of which contains material which originated from the preceding one, is not proof even for passing RT activity. To illustrate what Montagnier and his colleagues have done, let us return to the analogy of the fisherman and his net: Assume the fisherman casts his net and catches some sea creatures. He leaves a few in the net as bait and then throws it out again. This time, in addition to sea creatures he catches some fish as well. He removes the fish, leaves some sea creatures in the net, throws the net again and this time he catches even more fish. He repeats the procedure several times and every time he catches more fish. Let Montagnier et al who remove the cells and re-use the supernatants, the fisherman removes the fish and re-uses the sea creatures (“the bait”). Clearly the fish caught in the net are not offspring of the “bait”. The purpose of the “bait” is to create the right conditions for fish to appear in the net. (Indeed, real fishermen spend a lifetime determining the right conditions. All the fisherman is “passing” is the means for catching the fish, not the fish themselves. Similarly, Montagnier et al appear to be “passing” the conditions to generate RT activity thus generating the illusion of “passing” RT activity.

2. Having a peak of RT activity is not proof for having “replication” of a retrovirus. Keeping track of RT is not the same thing as keeping “track of the virus”.

3. Let us assume that one has isolated and proven the existence of a retrovirus in cultures with tissues originating from humans. “The first question put by Nature is Is it an endogenous retrovirus?” Only when one has evidence that it is neither an exogenous nor an endogenous human retrovirus does the question of “laboratory contamination” with animal retroviruses arise.

4. What the patient had was antibodies which reacted with a protein which in sucrose density gradients banded at 1.16g/ml. Since at that density Montagnier and his colleagues could not find particles with the morphological characteristics of a retrovirus, the evidence that this protein was retroviral was non-existent. In fact, they had no evidence that the protein was embodied even in non-retroviral particles, any particles whatsoever present at that density.

5. If Montagnier and his colleagues somehow knew beforehand that the protein which banded at 1.16g/ml and reacted with the gay man’s serum was the protein of a retrovirus which was present in his lymphocytes (and not the lymphocytes of the healthy donor or the umbilical cord), and at the same time that the antibodies were directed against “his own virus”, why was it necessary to have all these experiments to prove its existence?

9 Even though they had RT activity at the density of 1.16g/ml they had no evidence for the existence of retroviral particles and thus the activity could not be considered proof for the existence of such particles.

8

10 In 1983, Montagnier, Barré-Sinoussi and Chermain and their colleagues proved the existence of the enzyme reverse transcriptase “using the ionic conditions described for HTLV-I”, that is, “5mM Mg2+” and “poly (A).oligo-(dT)12-18 as template primer”. These conditions and this template primer may be characteristics for retroviruses but they are not specific for retroviral RT nor indeed any RT. Even before the AIDS era it was known that this template-primer, under the conditions used by Montagnier et al and their colleagues, can be transcribed not only by RT but by cellular DNA polymerases as well. Suffice to mention the study entitled: “Characteristics of the RNA-dependent DNA polymerase (RT) of a new human T lymphotropic retrovirus (lymphadenopathy associated virus)” (“HIV”) in which Montagnier, Barré-Sinoussi, Chermain and their colleagues “characterized” the “HIV” RT. There they used the same ionic conditions as in 1983 and three template primers “Activated DNA”, poly (A).oligo- (dT)12-18 and poly C.oligo-dG 12-18. They reported that while poly C.oligo-dG 12-18, “a reverse transcriptase specific template primer” was transcribed only by the “HIV infected” cells, “Activated DNA” and poly (A).oligo- (dT)12-18 were transcribed by both infected and non-infected cells. In other words, finding RT activity by using the template primer An dt 12-18 is not even proof for the existence of RT and even less for the existence of a retroviral RT.

11 No comment.

12 No comment.

13 We agree with Montagnier that when using lymphocyte cultures infected with exogenous retroviruses such as MT2, MT4 and H9 (HUT-78), all of which originated from patients with “adult T4-cell leukemia”, said to be caused by HTLV-I, it “is a real soup”. However, given the existence of endogenous retroviruses, when one uses lymphocytes from normal individuals and umbilical cord lymphocytes, the result is still “a real soup”. Maybe a different soup, but nonetheless still “a real soup”.

14 We agree that patients with AIDS and those at risk are infected with a “stack of things”. Furthermore, the cultures with tissues from these patients in addition to these agents may also be infected in vitro with other agents, such as mycoplasma.

15 It may be true that sometimes it is easier to detect a particle with the morphological characteristics of a retrovirus in the culture than in the plasma. However, since the viral “concentrate” is obtained from the culture supernatant and since by definition a “concentrate” would have more particles per unit volume than the culture supernatant, it follows that it should be much easier to see a particle in the concentrate than in the culture. Since Montagnier and his colleagues “saw nothing major” in the “concentrate”, that is, in the 1.16g/ml band, then why in their 1983 paper did they state the “concentrate” not only contained viral particles but “purified” virus? In the electron microscope picture which Montagnier and his associates including Charles Dauget published there are buds on the cell surface, some of which are more pronounced than others. But what is the evidence that they are virus or they are in the process of becoming a virus?

16 A similar phenomenon was described for the AIDS related “Kaposi’s sarcoma” in which the way of presentation is different for AIDS patients than for immunosuppressed patients.
However, there are particles which are **not** viruses (including retroviruses) that exhibit identical morphological features as retroviruses. Therefore from morphological considerations both the buds and cell-free particles cannot be considered to be retroviruses. Furthermore, cultures of tissues derived from AIDS patients contain a plethora of viral-like particles with diameters ranging from 65-250nm, shapes which are spherical, angular and tear drop, surfaces with and without spikes, and which contain cone shaped, bar shaped, centrosymmetric and tubular cores, as well as double cores and a mixture of cores. Like the several particles of varying taxonomy deemed the "HIV" particle, none of these particles have been purified and characterised and, like "HIV", their origin and role must remain conjecture.\[9\] \[10\] \[11\] 

**1.** If they did not purify the particles why did they claim to have done so and continue with the same claim up to this interview? 
**2.** It is true that they reported the peak of RT activity at the density of 1.16g/ml, that is, at the density in which they claimed to have "purified, labelled virus". However, how is it possible to claim that the RT activity "was soundly that of a retrovirus" when they "didn't take the peak...or it didn't work", that is when at that peak they did not even find retrovirus-like particles, not to mention retroviruses? To pass a retrovirus from one culture to another, one must have proof for the existence of a retrovirus in the first culture. "Passing" non-specific phenomena is no proof for passing a retrovirus. Furthermore, since all the phenomena which Montagnier and his colleagues considered as proof for the existence of a retrovirus, including RT activity and virus-like particles, could arise denovo in the cultures, especially under the culture conditions they used, they cannot claim proof for passing anything. How did Montagnier and his colleagues know that if they had suitable controls, the same phenomena would not have occurred in the blood donor's culture as well as in umbilical lymphocytes even if they were not "infected" with "HIV"? 

**1.** If the stage of purification (isolation) is not necessary, then why did Montagnier and his colleagues claim to have proven the existence of "HIV" because they "isolated" it, "purified" it? 

2. Since any piece of DNA can be cloned and amplified, cloning and amplifying a piece of DNA provides no information whatsoever in regard to its origin, that is, if it is retroviral or not. Neither is it possible by sequencing a piece of DNA to say that it is "truly a retrovirus" unless prior proof exists that these sequences are present in a retroviral particle and nowhere else. There is nothing specific about the "structure of retroviruses". If indeed there is a unique "sequence of DNA" indicating "it is truly a retrovirus" and "all the retroviruses have a familiar genomic structure with such and such a gene", then no such proof exists for the "HIV genome". \[12\] \[13\] \[14\] Suffice to mention that to date no two identical sequences for the "HIV genome" have been published. One and the same patient may have different "HIV DNA" sequences. According to researchers from the Pasteur Institute, "an asymptomatic patient can harbour at least 10⁹ genetically distinct variants of HIV, and for an AIDS patient the figure is more than 10⁹.\[15\] \[16\] \[17\] The genetic differences may reach 40%.\[18\] (Compare this to the 1-2% difference between human and chimpanzee DNA). The length of the "HIV DNA" has been reported to be between 9-15kb. In 1983 the Pasteur researchers reported that "The deduced genetic structure is unique; it shows, in addition to the retroviral gag, pol, and env genes, two novel open reading frames we call Q and F.\[19\] In 1990 the "HIV" genome was said to consist of ten genes;\[20\] in 1996 Montagnier reported that "HIV" possesses eight genes\[21\] and, according to Barré-Sinoussi,\[22\] "HIV" has nine genes. 

**2.** We agree that to transmit a retrovirus one does not need pure material. However, to transmit something, one first must know what one is transmitting, that is, one must have proof for its existence. For retroviruses such evidence can only be obtained by isolating (purifying) the particles, determining their physical and chemical properties and proving they are infectious. 

**1.** Montagnier and his colleagues, even after a Roman effort could not find even retrovirus-like particles at this density thus, from his experience (experimental evidence), there are zero chances and NOT 999 out of 1000 that RT activity at the density of 1.15, 1.16 represents a retrovirus in their case. 

2. We agree that it could be a retrovirus of different origin. The existence of endogenous retroviruses, together with the presence in AIDS patients and those at risk of antibodies which react with their antigens, means that even if M. Montagnier et al had proven the existence of a retrovirus, it would have been impossible to say that the retrovirus originating in the gay man and not in the donors or umbilical cord lymphocytes. 

3. The "molecular biology", the "cloning and sequencing" of the "HIV" genome has been discussed in detail elsewhere.\[23\] \[24\] Suffice to mention here that: 
- (a) proof for the existence of "HIV" and indeed for its causative role in AIDS was claimed before any "molecular biology", "cloning and sequencing"; 
- (b) since any piece of nucleic acid can be cloned and sequenced, cloning and sequencing of a piece of nucleic acid cannot be used to prove the existence of a retrovirus or of its genome. To the contrary, proof for the existence of viral nucleic acids (viral RNA and cDNA) can be accepted if and only if it is shown that the RNA is a unique molecular entity belonging to particles with morphological, physical and replicative characteristics of retroviral particles. This can only be done by separating the particles from everything else, by purifying them. Instead, Montagnier and Gallo used "a real soup" of cultures and co-cultures (Montagnier's group) or "a real soup" of cells infected with Epstein-Barr virus. The supernatants from these cultures were then sieved in sucrose density gradients. From all the RNA (and DNA) which banded at 1.16g/ml they arbitrarily chose some RNA using totally non-retroviral specific criteria and called it "HIV RNA", without any proof that the band contained even retroviral like particles\[25\] \[26\] \[27\] (c) the first, absolutely necessary step in proving that the "HIV RNA", retroviral or not, originated from the lymphocytes of "infected" individuals, is to perform hybridisation experiments using fresh, uncultured lymphocytes and the "HIV DNA" (obtained by reverse transcription of the "HIV RNA"), as a probe. It is hard to understand why M. Montagnier and his colleagues did not report such experiments. Gallo's group did and the results were negative. In 1994 Gallo was quoted in this magazine as saying: "We have never found HIV DNA in the tumour cells of KS...In fact we have never found HIV DNA in T-cells." At present there is no study proving the existence of even one single copy of the "full-length HIV genome" in the fresh T-cells even of...
a single AIDS patient or a patient at risk of AIDS;
(d) Currently the number of “HIV” particles in the plasma is
quantified by measuring “HIV RNA”, the viral load which is
reported to be “15 x 10^3 to 554 x 10^3 virions per ml.” 23 Any
studies claim proof that the “viral load”, the “HIV RNA”, can be
determined to undetectable levels by the use of both RT and
protease inhibitors. However, since:
(i) it is accepted that the “HIV RNA” is a transcript of the “HIV
DNA”;
(ii) by their nature neither the RT nor the protease inhibitors have
any effect on DNA transcription, they only inhibit infection of
new cells, that is, the decrease in “HIV RNA” is a consequence of
the decrease in “HIV DNA”; one would expect that the effect of these drugs would be deter-
mined by measuring the level of “HIV DNA”. Yet hardly any
such studies have been published. The very few which exist show
that neither RT nor protease inhibitors have any effect on “HIV
DNA”.74-76 which means that no relationship exists between “HIV
RNA” and “HIV DNA”.

4. In 1984 M Montagnier and his colleagues reported that “pre-
activation of T4+ lymphocytes with three different monoclonal
antibodies directed at the T4 glycoprotein blocked cell infection
by LAV”, that is, blocked the detection of RT activity in T4 cells
“infected” with “HIV”. They concluded their “findings strongly
suggest that the T4 glycoprotein is at least associated with all or
part of the receptor for LAV”.38 However, blocking a non-
specific “HIV” phenomena, namely RT activity, cannot be
considered proof of blocking “HIV” infection or association of
“HIV” with T4 cells.

22 We agree that “analysis of the proteins of the virus
demands mass production and purification. It is
necessary to do that”. In this respect they have not just
partially failed, but TOTALLY FAILED. If the
“analysis of the proteins of the virus demands mass production and
purification”, so does the analysis of “nucleic acids, cloning etc.”
If one fails to purify the virus then it fails:
(a) to characterise the viral antigens and to obtain a gold standard
for the antigen-antibody reaction, that is, one cannot use antibody
tests to define infection with the retrovirus
(b) to obtain and characterise the retroviral nucleic acids, R N A
(cDNA) and thus probes and primers for hybridisation and PCR
studies, that is, one cannot use molecular tests to define retroviral
infection. That this is the case is accepted by Donald Francis, a
researcher who with Gallo, played a significant role in developing
the theory that AIDS is caused by a retrovirus. In 1983, Francis,
then the chief collaborator of the AIDS Laboratory Activities, US
Centers for Disease Control and former chief of the WHO
smallpox program, speculated on a viral cause for AIDS: “One
must rely on more elaborate detection methods through which, by
some specific tool, one can “see” a virus. Some specific substances,
such as antibody or nucleic acids, will identify viruses even if the
cells remain alive. The problem here is that such methods can be
developed only if we know what we are looking for. That is, if we
are looking for a known virus we can vaccinate a guinea pig, for
example, with pure virus... Obviously, though, if we don’t
know what virus we are looking for we are unable to raise antibodies in
guinea pigs, it is difficult to use these methods...we would be looking for something that might or might not be there using techniques that might or might not work” 77 (italics ours).

23 It is impossible to characterise two viral unknowns,
namely its proteins and the antibodies directed
against them, by the formation of an
antibody/antigen complex let alone characterise the
“virus”. By what means did M ontagnier know that someone
had antibodies against the proteins of the virus and that the
proteins with which the antibodies react were viral? It is a scient-
ific impossibility to know that someone has antibodies to a virus
and at the same time, the 1.16g/ml band contains proteins of the
same virus before one has proven its existence.

24 1. It is true that M ontagnier had controls but the
controls were not suitable. M ontagnier and his
colleagues reacted the proteins which banded at
1.16g/ml with the sera from two gay patients with
lymphadenopathy. The patients with AIDS and those at risk were
already known to have a plethora of antibodies, all with potential
for cross-reactivity. Therefore, one would have expected that
M ontagnier et al to have used as controls sick individuals who did
not have AIDS or pre-AIDS and who were not at risk for AIDS
but who also had a plethora of antibodies, all with potential for
cross-reactivity. Instead their controls consisted of two blood
donors whose state of good health is characterised by lower levels of
antibodies.

2. M ontagnier et al did not obtain proof for “a specific reaction”.
The sera from the patients and the donors were reacted with both
the “purified virus”, that is the 1.16g/ml band, and extracts from
the “infected cells”. In their published strips, with “purified
virus”, it is not possible to distinguish any reacting proteins with
any of the sera. In the text they state: “When purified, labelled
virus [the 1.16g/ml band] from patient 1 was analysed...three
major proteins could be seen; the p25 protein and proteins with
molecular weight of 35,000 and 45,000”. No such reactions were
reported with the donors’ sera. In the published strips with
extracts from the “infected cells”, it is obvious that many proteins
reacted with both the patients’ and the healthy blood donors’ sera.
One year later M ontagnier and his colleagues confirmed that “sera
from some AIDS patients bound a lot of cellular proteins...This
banding was apparent in the RIPA and only sera which specifically
precipitated the p25 were regarded as positive”. In other words,
for some unknown reason, they concluded that from all the
reacting proteins only p24 (their p25) was retroviral and from all
the antibodies only the one which reacted with p24 was directed
against the retrovirus. Even if one considers the reaction between
the p24 which bands at 1.16g/ml and the antibody present in the
sera specific, that is not due to cross-reactivity, from such a
reaction it is impossible to draw the conclusion that p24 is retro-
viral protein and the antibody is elicited as a result of infection
with this retrovirus. Indeed given the fact that M ontagnier et al
could not even detect retrovirus-like particles at 1.16g/ml, their
considerations regarding p24 and the antibody reacting with it
completely defies scientific reasoning.

25 1. No antibodies, not even monoclonal antibodies
are “very specific” or even specific.78,84 Indeed
there are instances where “cross-reactive antigen
binds with higher affinity than the homologous antigen itself...The
most obvious fact about cross-reactions of monoclonal antibodies
is that they are characteristic of all molecules and cannot be
removed by absorption without removing all reactivity...Even
antigens that differ for most of their structure can share one deter-
minant, and a monoclonal antibody recognising this site would
then give a 100% cross-reaction. An example is the reaction of autoantibodies in lupus with both DNA and cardiolipin.” 80
However, “It should be emphasised that sharing a “determinant”
does not mean that the antigens contain identical chemical struc-
tures, but rather that they bear a chemical resemblance that may
not be well understood, for example, a distribution of surface
charges”. 81 It is of importance to note that “HIV” experts concede “cross-reactivity” as the reason for “indeterminate
antibody reactivity seen in the “HIV” Western blot, as well as for,
example, reactivity between monoclonal antibodies to the “HIV
p38 protein and dendritic cells in the lymphatic tissues of a variety
of patients with a number of non-AIDS related diseases and
normal tissues taken from “non-HIV” infected individuals.” 45 No one to be convinced that all “antibodies [including monoclonal]
are polyclonal, that is, they are able to react with various disim-
ilar antigens such as “proteins, nucleic acids and haptenes”, “they
are able to react with more than to self or non-self antigens, often
without any apparent antigenic similarities”, all one has to do is to
read the scientific publications of the researchers from the Pasteur
Institute such as stalis Avrames” 44-47.
2. It cannot be concluded that a protein which bands at 1.16g/ml is viral merely because it reacts with an antibody present in the patient's sera even if somehow one knows that the antibodies present in the sera are monoclonal. Let us assume an ideal situation where:
(a) all the antibodies present in the patients' sera are monoclonal and "very specific";
(b) the 1.16g/ml band contains in addition to the many unembodied and microvesicles, embodied proteins of cellular origin and maybe of bacterial, fungal and viral origin (constituents of the many infectious agents, other than retroviruses, present in the culture and the patients) and, as shown in a 1997 Franco/German study, a number of retrovirus-like particles. Even in this ideal situation, it is NOT POSSIBLE TO CLAIM that just because a protein such as p24, p41, or others is found in this band and reacts with the sera, the protein is a constituent of the retrovirus-like particles.

3. The reality is that:
(a) all AIDS patients and those at risk have a plethora of antibodies including auto-antibodies. The auto-antibodies include anti-lymphocyte, and as M. Montagnier and his colleagues have shown anti-actin and anti-myosin antibodies, that is antibodies to the two ubiquitous cellular proteins actin and myosin.
(b) all the antibodies present in the sera have the potential of cross-reactivity.
(c) the proteins from the supernatant of non-infected lymphocytes which in sucrose density gradients band at 1.16g/ml, the mock virus, include proteins having the same molecular weights as the "HIV" proteins.
(d) animals inoculated with the mock virus develop antibodies which react with the "SIV" proteins, a "retrovirus" whose proteins share the same molecular weights as the "HIV" proteins and is said to be the closest relative of "HIV".
(e) AIDS patients and those at risk are repeatedly subjected to allogenic stimuli including allogenic lymphocytes which is not HTLV is retroviral.
(f) up till 1997 no evidence existed showing that the 1.16g/ml band contained even retrovirus-like particles.

Given this reality, to claim that just because a protein bands at 1.16g/ml and reacts with antibodies present in the patients' sera is at best no different than the following:
(i) A researcher has two bowls, one of them contains a mixture of raw eggs, some known and maybe some unknown, and maybe some milk originating from several animals. The other contains several acids. Again some known and maybe some unknown. Once the contents of the two bowls are mixed he gets a precipitate. He claims that the precipitation proves the existence in the bowl of milk from a previously unknown animal and an unknown acid and that the reaction is between the unknown animal and a protein of the unknown milk.
(ii) This claim is scientifically impossible since any protein in the eggs could have reacted with any acid to produce the observed precipitate. Thus, given the reality as outlined in (a) to (f) above, it is completely unscientific to claim that the reaction between proteins which band at 1.16g/ml and react with antibodies present in the patients' sera is proof of the existence of "HIV" proteins. To claim that the reaction between proteins which band at 1.16g/ml (in the absence of evidence that the band contains even retrovirus-like particles) with antibodies present in the sera indicates not only the band contains retroviral proteins, but proteins of a new retrovirus, is no different than the following: A fisherman has sea creatures but no fish in a net. He throws some animals into the net. The fisherman observes that the animals eat some proteins present in the net and claims that the proteins were not just fish proteins but the proteins of a completely new fish, a fish which nobody has seen before, a golden fish.

27

The p24 protein is not sufficient for diagnosing "HIV" infection because it is not specific. Indeed, no other "HIV" protein not even p41 (p45/43) has been reported to react more often with sera from healthy (at no risk of AIDS) individuals. Neither has a monoclonal antibody to any of the other "HIV" proteins been found to react more often with proteins present in non "infected" cultures or sera from individuals at no risk of AIDS. According to M. Montagnier because:
(a) "there are cellular proteins that one meets everywhere - there is a non-specific background noise";
(b) one such protein, having a molecular weight of 45,43, is actin;
(c) this protein reacted with sera from individuals at no risk of AIDS; the p45/43 represents a cellular and not a viral protein. However, since:
(i) myosin is as ubiquitous as actin.
(ii) myosin has a light chain with a molecular weight of 24,000.
(iii) the cytoskeletal proteins (of which actin and myosin are the most abundant) have been reported in "pure HIV".
(iv) myosin and actin are said to play a crucial in budding and release of the "HIV" particles;
(v) M. Montagnier has shown that patients with AIDS and at risk of AIDS have anti-myosin antibodies. Why should not one consider the p24 band as representing myosin?

28

We agree that no protein is sufficient to diagnose "HIV" infection. The problem then, as it is today, was not "to know whether it was an HTLV or not", but whether it was retroviral or not. Not everything which is not HTLV is retroviral.
which band at 1.16g/ml are “HIV” proteins. The only reason that 20% of the proteins which band at 1.16g/ml are said to be “HIV” is that this fraction of proteins is found to react with different AIDS patient’s sera at some time or another.

2. We agree that with the technique used by Montagnier’s group, one cannot prove which proteins (nucleic acids) are cellular and which are viral.

3. We agree. The only way one can prove the existence of the viral protein (nucleic acids) is “to purify the virus to the maximum”, that is, to obtain density gradients which contain only particles with the morphological characteristics of retrovirus and nothing else. This has never been done to prove the existence of the “HIV” proteins and nucleic acids.

4. If one always “stumbles on the same proteins” in successive gradients, this is no proof that these proteins are viral and the ones which disappear are cellular.

30. No matter how many times the banding is repeated, if one starts with no retrovirus-like particles one will end with no such particles. Some times, by successive bandings, one may be able to eliminate non-retroviral components and obtain a band which contains nothing else but particles with morphological characteristics of retroviruses. However, to be able to do so, even after the first banding, one must begin with a relatively high proportion of retrovirus-like particles.

2. Once again, the origin of the proteins cannot be determined by molecular analysis, that is, by sequencing the proteins.

3. We agree that if the proteins of a retrovirus are coded by its genome, as is generally accepted, then it may be possible to characterise the retroviral proteins by its genome. However, to do this one must first prove that the RNA (cDNA) is a constituent of a retroviral particle. This has not been done for the “HIV” genome. In fact even today there is no proof that the “HIV” RNA is a constituent of a particle, any particle viral or non-viral.

4. To date there is no proof of a relationship between the sequences in the “HIV” RNA (DNA) and the sequences in the proteins “observed with immunoprecipitation or with gel electrophoresis”. In fact there is no relationship even between the size of the proteins coded by the “HIV” genes and the size of the proteins “observed with immunoprecipitation or with gel electrophoresis”. For example, in 1987 Gallo and his associates performed a “computer-assisted analysis” of the amino acid sequences of the envelope protein complexes derived from the nucleic acid sequences of seven AIDS viruses isolated, and concluded that “gp41 should be about 52 to 54 daltons by calculation”.

5. One of the many puzzling aspects of “HIV” is the following: (a) “HIV” experts agree that no two “HIVs” have the same genomic sequences and the difference may be as high as 40%; (b) They also admit that the vast majority (99.9%) of the “HIV” genomes are defective, that is, either part of a gene(s) or whole gene(s) are missing; How then is it possible: (i) to measure the viral burden (“HIV DNA”) and the viral load (“HIV RNA”) by using one and the same hybridisation probes and PCR primers? (ii) to perform antibody tests for all the different “HIV” by using kits containing the same antigens?

6. Indeed, the history as to how “HIV” researchers have tried to prove the existence of p120 and how they ultimately agreed on its existence is very interesting. However, given the fact that the p120 protein is said to be present only in the knobs, no cell-free “HIV” particles possessing knobs have been reported so far. It follows neither the particles in the culture supernatant nor the “pure” virus will have gp120. In other words, it is impossible for either the RIPA or the WB strips to have a “HIV” protein of molecular weight 120,000.

1. Prior to March 1997 no group of “HIV” researchers had published even a single electron micrograph of material banding at the density of 1.16g/ml in a sucrose density gradient. The first EMs of material banded in sucrose density gradients appeared in 1997 in two publications, one Franco/German and the other from the US National Cancer Institute (NCI). The Franco/German EMs are from the 1.16 gm/ml sucrose density gradient whereas it is not possible to tell from which density the NCI data originate. The data from both studies reveal that the vast majority of the material is “non-viral”, “mock” virus, cellular “microvesicles”, that is, the banded material is virtually all cellular. These particles, like the retroviral particles, contain nucleic acids in addition to proteins but they are not as condensed.

2. The EM micrographs in both studies also contain a small minority of particles which have morphologies more closely resembling retroviral particles than the “mock” particles. Both groups claim the fewer particles are “HIV”.
33 Pictures of the 1.16g/ml are of profoundly significant interest. How else can it be known that there are retrovirus-like particles there, especially since even M ontan gier admits that other things may band there. For any scientist who claims proof for isolation, purification of a retrovirus using sucrose density gradient banding, it is vital and absolutely necessary to make a micrograph of the 1.16g/ml band showing nothing else but retrovirus-like particles.

34 If this is the case, why is such data not available in the scientific literature?

35 In one of their 1984 papers M ontan gier and his colleagues wrote, “Several characteristics indicate that LAV or LAV related virions belong to the retrovirus family. Budding particles at the plasma membrane have been observed in electron microscopy. The density of the virus in sucrose gradient is 1.16 and a g^2 dependent reverse transcriptase activity has been found to be associated with RNA containing virions.”

36 Furthermore, in this study they showed that DNA polymerases beta and gamma and of non-infected cells reverse transcribe An.dT12-18 in the presence of M g^2+. Thus, M ontan gier’s own conditions and data do not prove his claim that what he has “seen” and “encountered” is a retrovirus. If “HIV “exists”, and it is “clear” to M ontan gier that he has “seen it” and “encountered it”, where is his proof?

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VIRUS CHALLENGE

CONTINUUM vol 5, no 2

For many challenged by an HIV antibody diagnosis the possibility of long-term survival has become a reality. Many of us are aware that not only are we very much alive and well after many years but we have discovered that there are challenges, from within the scientific community to the fundamental HIV/AIDS science, that may explain our continued existence.

Unfortunately, it has been left to individuals and organisations to disseminate the information, with many of the aids organisations purporting to represent the interests of the individual either repressing, indifferent to or totally ignorant of that knowledge. How can someone make decisions affecting their future if not fully aware of the facts? The simple fact that the isolation of the putative virus has been challenged could play a vital role in an individual's appraisal of their future. Incredibly, the sixty or so accepted viral antigens have not been tested differently, and ignorantly produced cross-reactions to the test are not otherwise. But why should we provide the counter-evidence? - It wasn't our hypothesis. In any case, it is particularly difficult to prove survival when the goal posts keep moving; as the latent period extends or the death sentence is relaxed.

While there have been several scientific studies of long-term survival published, a comprehensive study embracing some of the factors many survivors themselves believe are responsible has not been forthcoming, nor have most of the previous studies accessed those outside conventional medicine. A study is currently being prepared to redress that urgent need. Initiated by the group Action positive Switzerland (APS) the study will be carried out by several organisations, including Gay International Association Trust (GAIA) and Continuum, hopefully accessing as many diagnosed as possible with a questionnaire covering aspects of health history of the individual. The study evaluates health prior to testing as well as the various strategies for survival after. The study has been planned for completion in twelve months with questionnaires being distributed as widely as possible in several countries and analysed by a panel of various professionals, research physicians, clinical psychologists and clinical social workers plus long-term survivors.

Continuum is eager to participate in the study in the hope that it will provide insight into the many factors that affect health but also hopefully awaken the closed-minded to the fact that survival is a reality. The timing of this is all the more important as the push, aided by an ignorant media, is towards having us believe that HIV is no longer a threat.

For more information contact Clair at Continuum (see index page)
Cultural hypnosis, group fantasy and psychogenic death have profound social functions. They collectively serve to keep everyone's attention off anything in life that matters like real love, health and happiness. They specifically serve to keep our attention off anything which threatens the focus of this paper - the Big Lie that: within a world in crisis, everything is OK.

Experts and officials also have a social function. If you think about it, when dealing with personal Big Lie issues, people not only expect to be lied to, but they depend upon it. As long as the lies have a seed of 'truth' it gives those who need it an opportunity to dismiss messy things like the role of self-responsibility in health and illness, not to mention things like economic injustice, political exploitation and most of all medical murder. And the experts all happily oblige; after all, they need the Big Lie too. Without it they would have to acknowledge their racism, sexism, homophobia, exploitation and countless other crimes against humanity.

PROTECTIVE STUPIDITY

When dealing with this sort of group think, the only thing we can be sure of is that nobody is thinking. This is confounded by the fact most hypnotized people can not appreciate that they are already in a hypnotic Big Lie-protecting trance. In fact most people become irritated, defensive or even hostile at the mere suggestion, because once hypnotized there is a spontaneous impulse to defend the resulting mythology. Orwell called this “protective stupidity”. (Orwell, G. “1984”)

This defensive reflex is in itself evidence of the trance as without the trance a person would simply consider the information being presented. When in a trance however, one will dismiss as out of hand “dangerous information”, i.e., any information which threatens the Big Lie view of the world or which suggests taking responsibility and/or action around issues of one's own health and happiness, particularly in the social realm. This is what makes it so very challenging to help people realize that they have been culturally brain-washed throughout their lives (hypnotized without their knowledge or consent). Without the Big Lie, the true state of the world would be emotionally devastating. The group fantasy ends up serving a powerful survival-istic and anxiety-regulating function. One is not even permitted to think about it let alone discuss it.

THE GROUP FANTASY NEED FOR ‘HIV’

The very ideas of group fantasy, cultural hypnosis, epidemic hysteria and psychogenic death are so unsettling that they almost always produce an “I can't believe that!” response. Put simply, group fantasy is the social agreement that black is white, up is down, and that the emperor is wearing clothes. It is used to mask rather than unveil the Big Lie and the identity of all who participate in perpetuating it.

‘Epidemic hysteria’ is the psychophysiological bridge between the group fantasy and the development of clinical psychogenic symptoms. Both have very important functions in that they are unconsciously used by the group to purge the social body of poisonous feelings that have been generated by the Big Lie in the first place. (C. Schmidt. M.D. Group Fantasy Origins of AIDS. The AIDS Cult - Essays on the Gay Health Crisis edited by John Lauritsen and Ian Young).

Epidemic hysteria does, however, require a seed of
Which brings me to the AIDS Zone. The AIDS Zone is a phenomenon that requires the existence of people who are actually sick to get started - people who are in fact sick because of the Big Lie. To dissociate this connection between sickness and the lie, we pull out the old standby: 'viral' scapegoats - like the group fantasy object 'HIV'.

**'HIV' AND THE BIG LIE**

But how does one tell whether, in the case of epidemics of illness, one is dealing socially with hysteria, psychogenics and protective stupidity, or with an actual effort to curb social and physical illness? It depends on how the truly sick are dealt with. If their illnesses are used to direct attention to the Big Lie, health and better social conditions will result.

If on the other hand, it is being used to direct attention away from the lie, as in the case of AIDS/'HIV' and as evidenced by the many defensive reactions triggered by exposure of the fraud, there will be an escalation of both illness and social tension, i.e., a golden opportunity for the critically urgent psychosocial purge. This explains why most of us have had such a hard time seeing that the perception of an 'epidemic' of 'HIV disease' particularly in the gay community is a group fantasy. As AIDS analyst Michael Baumgartner points out, more gay men will die of heart disease this year than 'AIDS'. And yet no-one is putting any energy into heart disease. Why is it that people are not up in arms over this? Without seductive and titillating triggers like sex, anal sex or the threat of sexual transmission, there is no opportunity to bring to a climactic head pre-existing social tensions. There is nothing titillating about heart disease. With AIDS however there is enough to arouse in everyone a hysteria. This mass hypnosis allows people to unconsciously act out their preconditioned roles, roles which are essential to perpetuating the Big Lie. If you’re tranced ‘HIV+’ your part is to get sick and die; if you are a doctor your role is to test for an antibody, make healthy people sick and sick people die, and then blame an alleged ‘virus’; if you’re a gay AIDS activist your role is to ensure that unproven treatments get into everyone’s body and that everyone wear a condom as if your role is to deliver ‘HIV+s’ to the pharmaceutical ovens and to the pharmaceutical industry, who questions the insanity; and if you’re not in any of these groups your role is to wear a red ribbon, a latex condom and act like you care.

‘AIDS’ works because everyone has something to do. It justifies the multi-billion dollar AIDS War industries which reinforce through their mystery the manufactured belief that we need military experts, covert operations and weapons of mass destruction to protect us from the ‘Red menace’. Today, the collective ‘Red menace’ has been replaced by the much more individual ‘viral menace’. The social function of the ‘viral menace’ is also to scapegoat all the social problems in our lives. Economically it justifies the multi-billion dollar AIDS War industries which reinforce through their mystery the manufactured belief that we need military experts, covert operations and weapons of mass destruction to protect us from (the Big Lie) ‘HIV’.

**THE REALITY OF PSYCHOGENIC ILLNESS**

Recently there was a brilliant essay published in CONTINUUM ("Communicable disease" - Alex Russell, vol 4/no 6 June/July '97) designed to help ‘victims’ liberate themselves from the AIDS Zone. It was a masterpiece that offered readers a free pass out. This was followed by "Dissenting view" (Whose Hysteria?, by Cooper and Walker, vol 4/no 5 June/July '97) designed to help ‘victims’ liberate themselves from the AIDS Zone. It was a masterpiece that offered readers a free pass out.

What we get in this essay is a conditioned reflex based on the authors’ unwillingness to address the staggering implications raised in Russell’s article. The reality of a transmissible hypochondria and subsequent psychogenic death was just too much for them. Make no mistake about it, if you are branded ‘HIV+’ you do not have the luxury of ducking this urgent consideration. For you, understanding the nature and scope of the AIDS Zone and psychogenic disease (and how to escape both) is a matter of life and death. There are many concurrent epidemics of hysteria raging today and the psychogenic factor, particularly in the case of pseudo ‘HIV disease’, becomes clearer when we differentiate between the physiological factors of illness and the psycho-social factors of illness - that is, the core group of people getting sick and the much larger shadow group of people getting interpreted as identically ill if they really are ill, or as at risk of illness.

**THE AIDS AND OTHER ZONES**

Which brings me to the AIDS Zone. The AIDS Zone is a phenomenon that requires the existence of people who are actually sick to get started - people who are in fact sick because of the Big Lie. To dissociate this connection between sickness and the lie, we pull out the old standby: ‘viral’ scapegoats - like the group fantasy object ‘HIV’.

"It's better not to know that you can escape from the AIDS zone"
SOCIAL HEALTH RISKS

Gulf War Syndrome (GWS) is an excellent example of what I am talking about. The primary social health risk is the stress of war itself. From here things break up into two groups. In addition to the stress of war, the core group of people developing GWS symptoms suffer very serious exposures to very serious stressors, i.e., experimental vaccines, deadly pesticides, chemicals and possibly weapons of biological war - the very probable physiological factors of their illnesses. But the much larger shadow group unconsciously identifies with the core group and, in the absence of the same complex of stressors, begins to generate physical, psychosomatic versions of these symptoms to express the intense emotional trauma of war. This is also the case with ‘AIDS’. The core group of people who develop ‘GRID/AIDS’ indicator diseases and conditions suffer the physiological impact of very serious stressors, i.e., experimental vaccines, deadly medicines, street chemicals and sexually transmitted biological stressors (not ‘HIV’!). The much larger group of people who are developing symptoms perceived by themselves and others as AIDS-related symptoms are suffering a combination of hypochondria and normal flu, colds etc. But within the AIDS Zone, these take on a life of their own. They were once called ‘ARC’ - AIDS-related complex, the precursor to AIDS, but are now called ‘HIV disease’ because in the absence of treatment a majority of ARC cases never develop into ‘AIDS’. Those actually presenting psycogenic symptoms though are suffering from the psychological consequences of the destructive social lies nobody wants to talk about - alienation, homophobia, racism, poverty, malnutrition, self-hate, medical consumerism and drug use.

The unaddressed emotional tension finds a substitute form of expression by gravitating to and identifying with the physical syndrome manifested by the core group - this, in addition to the medical choices based on the hysteria, can lead to death. In the case of pseudo ‘HIV disease’ the major emotional stressors, the major psycho-social stressors relate to being gay, non-white, and/or labelled ‘HIV+’. Simply put, it’s the core group that defines the badge and the others who later adopt it. If that sounds unbelievable consider this:

1) There are well documented case studies of people who are loosely called “the worried well”. Even though they have repeatedly tested ‘HIV’ negative and have been repeatedly assured they are not at risk for AIDS, these people present a whole range of pseudo ‘HIV disease’ symptoms like dramatic weight loss, low grade fevers, skin disorders, diarrhea, low T-cell counts and chronic flu like symptoms. The American Psychiatric and Psychological Associations named the disorder AFRAIDS (Acute Fear of AIDS) deeming its symptoms psychogenic (R edotz. Considering the psycho-social aspects of AIDS. M. Hosp. jnl. 8/86, Vol.22 No.8) Of course such studies have failed to explore an equation between those who test negative and think they are ill and those who test positive and think they are ill. Since the ‘HIV’ test is unvalidated, both groups are actually dogged by the same beliefs and victims of the same ‘HIV’ hex.

2) Among certain cultures there is a powerful phenomenon called “bone pointing”. Its similarity to an ‘HIV-positive’ diagnosis is a crucial and long over due consideration because among those cultures, the belief that the bone can kill is enough to cause death. The bone has no physiological power in the same way that ‘HIV’ has no physiological power, but inside the Zones the belief in the either the bone or ‘HIV’ can be deadly.

WE NEED AIDS!

Which brings us back to the mass hypnosis. Ever notice how defensive some people become when you point out that the non-specific ‘HIV’ tests are not proof that anyone is infected with the putative ‘HIV’? Or that there is compelling evidence that low T-cell counts can reflect merely a chronic psychocorticophysiological redistribution of T-cells to the skin and other organs, not an indication of one’s actual state of health or illness? Or that long-term antibiotic and chemotherapy/’anti-viral’ drug use are classically recognized to cause ‘AIDS’?

Instead of being relieved, most people are insulted! How dare anybody suggest ‘HIV’ is any less than the brutal killer ‘virus’ that everybody knows it is! Without ‘HIV’ we might have to look at the true nature of the world around us, and perhaps even our responsibility in it all. No. It’s better not to know that you can escape from the AIDS Zone; it’s better not to lose ‘HIV’ solidarity and the social tolerance one gains within an unaccepting society. (Note that you’re tolerated as long as you die!) They react like many people who are told their GWS problems and symptoms are the result of post war stress. They are hurt and offended because like everyone else they have been culturally conditioned to seek the legitimacy of the medical industry for their health complaints, post war stress just isn’t an industrially or socially sanctioned disorder - we might have to consider things like economic injustice, political exploitation and emotional murder! As communities we have been persuaded and programmed to ignore and/or de-value both psychogenic disease and the intense distress of the war experience!

We have become unknowingly programmed to surrender our power to experts, germs and other government officials who know what’s best for us; to ignore anything that challenges our necessary illusions. We believe in the entity of ‘AIDS’ though the same mechanisms as we believe in the marvels of modern medicine: brainwashing, protective stupidity and the social function of the lie.

THE DOCTOR WILL KILL YOU NOW

Modern medicine exploits the unconscious pain of a wounded society to obscure the real causes of its
The Zones are the only atmosphere in which pseudo ‘HIV disease’ and this massive cover-up could thrive. In other words, everybody has been hypnotized to turn off their thoughts and feelings and, instead of living, we are mindlessly sleepwalking from one Zone to the next. This is our social function - evading the Big Lie. Is there a broader social function - that simultaneously serves its role of self-responsibility in generating one’s own health and happiness - that simultaneously serves its broader social function - evading the Big Lie. Is there a better explanation?

AIDS AND PSYCHOIATRIC STRESSORS

Hysteria and self-hypnosis explain why the group fantasy object ‘HIV’ is as seductive as it is menacing; why so many gay men wear their seropositivity like a badge of honor, why the public has dully accepted the psychosis, Giraldo points out that the scientific community has ignored when these people are sentimentally written off as victims of a mythical ‘AIDS pandemic’.

Roberto A. Giraldo, MD has written a penetrating exposé of the ever increasing industrial (and other) stressors in our daily lives. I wholeheartedly recommend interested people read Giraldo’s AIDS and Stressors to get a comprehensive understanding of what is really making us sick and make sense of the really serious threat this is to everybody’s health. In closing I highly recommend that anyone testing ‘positive’ read The AIDS Cult - Essays on the Gay Health Crisis edited by John Lauritsen and Ian Young. This, combined with what it is Everything you Thought You Knew About AIDS Was Wrong? by Christine Maggiore is your passport out of the Zone. Reading CONTINUUM on an ongoing basis is essential to staying out of the Zone as it provides the only international forum in which the free exchange of this kind of information is possible. I also recommend boning up on Pavlov, Chomsky, Orwell, Freud, Lacan and Showalter for a better understanding of group dynamics, psychological contagion and hysteria. Thank you for your consideration.

Stressing health

Roberto Giraldo’s AIDS and Stressors

Roberto A. Giraldo’s radical book, AIDS and Stressors (Impresos Begon, 1997:205p, USA, $15.00 Published in Medelin, Colombia) is paradigm-shifting, gut-wrenching and awe-inspiring. Well-researched with more than 800 references and citations, Giraldo’s thesis links ‘AIDS’ to iatrogenic, psychogenic and immunological stressors. The book contains a foreword by Angel Galeano, President of the Art and Science Foundation of Medelin, a preface by the editor, and six chapters: Chapter 1: Worldwide rise of immunological stressors; Chapter 2: A proposal for the pathogenesis of AIDS; Chapter 3: A proposal for the Natural History of HIV; Chapter 4: The Real Meaning of HIV; Chapter 5: AIDS is neither an infectious disease nor is sexually transmitted; Chapter 6: AIDS Crisis in the scientific method.

Giraldo, an expatriate Colombian and formerly one of that country’s experts on infectious diseases, was an organiser of the Conference ‘AIDS Without HIV: Myth or Reality’, which took place from 2-5 October, 1997. The Conference was held under government auspices at the University of Santander in Bucaramanga, Colombia and featured Drs Stefan Lanka, David Rasnick, Prof Peter Duesberg and Kary Mullis. Giraldo nominates ‘AIDS’ as “the maximum state of deterioration that the human immune system can reach; it is a toxic-nutritional syndrome caused by the alarming worldwide increment of immunological stressor agents”. Giraldo argues that ‘AIDS’ should be understood as a severe acquired immunodeficiency due to repeated, chronic and multiple exposures to immunological stressors. The immunological stressor agents create immunotoxic or immunogenic effects, or both, which generate a state of oxidative stress in cells and in metabolic reactions of the immune system.

Giraldo points out that the scientific community has been wrong many times in one considering as infectious diseases that are not: pellagra, scurvy, beriberi... Giraldo sees his AIDS/Stressors thesis as a practical tool for the medical authorities and diseased (and other) communities to fight ‘AIDS’. Only by abandoning the redundant and futile ‘HIV/AIDS’ hypothesis and implementing strategies for the AIDS/Stressors model can we hope to solve ‘AIDS’. This mind-blowing, life-saving book must be read as soon as possible...it’s a wake-up call.

Alex R. Russell

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They are what they write
You are what you read
We’re all what we eat

Martin J. Walker reviews a contemporary treasure trove of nutritional health texts

The British public is being exposed to good nutrition late in the day. Despite the fact that over the last two hundred years, Britain has been home to some of the great herbalists and medical nutritionists, the public and the patient have been the constant victim of the professional dietitian. Dietitians are low grade health workers, usually women, pushed to the periphery of medicine by the male dominated medical profession. Their work and their education is often sponsored by the food industry. Their clarion call is for a balanced diet.

With the contemporary depletion of the nutritional value of food, the rise in environmental toxins, the burgeoning rates of food-borne illness and an increasingly individualised society where people present greatly different nutritional needs, the rubric of ‘a balanced diet’ has come to be more or less meaningless.

Orthodox medicine, the pharmaceutical industry and the processed food industry have such power in Britain, that for almost a century, alternative views about the medical effects of food, have been suppressed. Like, Homeopathy, acupuncture and herbalism, nutritional medicine has become part of an essentially secret history of healing.

Albeit in the shadow of orthodoxy, changes have occurred in the last ten years. One reason for these changes has been the constant rise in cases of cancer, AIDS-associated illnesses and allergic responses such as asthma and chemical sensitivity. Orthodoxy has met these rises with a mesmeric inability to offer either preventative philosophies or safe and life enhancing curative therapies. For those who suffer from these illnesses, it has become only a short step, from the black hole of high-tech science to the reassuring embrace of more empathetic and optimistic alternative approaches.

Today’s nutritional therapist is quite different from the dietitian, interested in specific questions about nutrition and the body’s metabolism of food. Interested in how nutrients are absorbed; in the major and minor deficiencies of vitamins or minerals which exacerbate or cause medical conditions. Such nutritionists will probably believe in supplementation, although good advocates and practitioners will not suggest that supplementation should take the place of food. Perhaps most importantly, today’s nutritional therapist will be aware that each human organism is unique, with its own history of toxic challenges, illness and nutritional demands.
If Dr Stephen Davies is the father of nutritional medicine, Patrick Holford, is the heir apparent. In The Optimum Nutrition Bible, published in 1997, Holford has written a book worthy of a title contender. While Stephen Davies’ book tends to be formal and conservative in style, Holford’s is a dazzling display of graphic pyrotechnics. While it has a narrative textural core, diagrams, drawings, charts (even self-fill questionnaires to test vitamin deficiencies), jump off every page.

Patrick Holford founded the Institute for Optimum Nutrition in 1984, which from its small independent beginning has now grown to be one of the largest and most up-to-date nutritional teaching establishments in Britain. Holford’s approach to nutritional education has more in common with the campaigning zeal of Ralph Nader the American consumers’ rights advocate, than it has with typically retiring conservative English expertise.

The Optimum Nutrition Bible is divided into nine parts. The first six parts over two hundred and fifty pages recount in great detail the full past and present of nutrition. The last fifty pages of the book are made up of two A-Z sections, the first dealing with nutrition and a wide range of illnesses and health problems, the second dealing with vitamins and their properties. This is the perfect book for those who want to ‘get into nutrition’ and commit themselves to a healthier lifestyle.

In choosing a title for his book Patrick or his publishers appear to have followed the example of Samuel Epstein, the notable American writer about the politics of cancer who in 1995 published The Safe Shoppers’ Bible. Despite the fact that a bible can be just an authoritative book, it also means the book, and those who are serious about their writing must always know that the book is still to be written.

I have to admit to being a convinced fan of Robin Needes’ book, You Don’t Have to Feel Unwell; Nutrition, Lifestyle, Herbs and Homeopathy, A Home Guide. It wasn’t instant - when I was first given a copy of the book my unreconstructed Stalinist consciousness revolted against the cover depicting the silhouette of a happy, healthy person jumping for joy before a sunset festooned with hedgerow berries and nutritious fruits. I soon got over my problem with the cover however when I discovered the extensive, well written and clearly laid out contents of the book.

You Don’t Have to Feel Unwell moves easily between different modalities and spends time explaining therapies and their history. While the book provides an eclectic and exemplary therapeutic guide to a wide range of illnesses from allergies to Angina, Needes also introduces the history and philosophy of health dissents.

Part one of the book looks at such interesting conflicts as those between Vitalists and Mechanists, Bechamp and Pasteur; it looks at the history of homeopathy and discusses its estrangement from allopathic medical orthodoxy. Part Two looks at the general and finer points of Nutrition, its seventy pages deal with a wide variety of nutritional health models such as vegetarianism, food combining and naturopathy. The last part of the book, almost two hundred pages, looks at 40 different disease complexes and gives detailed plans about how they might be controlled. For every illness a wide range of diverse advice drawn from a variety of categories is given, including advice about diet, supplementation, herbs and homeopathy.

Continuum readers might at first be disappointed in Needes’ consideration of HIV and AIDS. In the first instance, he appears to take for granted the HIV=AIDS hypothesis. But on reading further it is clear that he is willing to pick up every corner of the carpet and look beneath it. The construct which he finally advances is one in which those who survive the longest, are those who work at their nutritional health, avoid toxic medical drugs, and develop a survivor’s mental profile. He follows this with four pages of treatment protocols, which covers diet, supplementation, herbs and homeopathy.

Linda Lazarides is one of Britain’s most experienced and strategically intelligent nutritional campaigners. For the last ten years, she has been at the forefront of battles with the EU, MAFF and the Medicines Control Agency to stop laws and regulations which deprive us of choice in the area of vitamin and mineral supplements. This campaigning work has not earned her either the money or the authoritative profile enjoyed by some other, mainly male, nutritional experts.

The Nutritional Health Bible by Lazarides was published in 1997; it is reassuringly populist while containing plenty of scientific data. That two of Britain’s best nutritionists whose views are at variance, could both publish bibles, is not only ironic but lends some credence to my observations above.

Linda’s book contains none of the entertaining stories of either the old or the new testament and is laid out in such a way that from the beginning it lacks contextual and narrative content. The first two hundred pages are in A-Z form which inevitably turn the book into a reference work rather than one which might hook the reader with a self-interest in nutritional health. The next two sections of the book which cover 111 pages deal firstly with the nutritional causes of illness and then everyday family circumstances which relate to nutrition and health. The last ten pages contain appendices, which deal summarily with important subjects like ‘The rules of healthy eating’.

There is a wealth of useful information in The Nutritional Health Bible but...
Since the nineteen twenties the relationship between diet and cancer has been a minefield of conflict. From this time onwards the multinational processed food industry began to create hegemony in the area of food and health. Also, after the 1920’s, the pharmaceutical companies and the medical profession began to gain monopoly control of therapeutic practices.

Dr Rosy Daniel is the Medical Director of the Bristol Cancer Help Centre, as well as being a holistic medical practitioner. The Bristol Cancer Help Centre has always had an interest in diet, principally due to the influence of Gerson and Issels on their early philosophy. Rosy Daniels has also been involved with Sandra Goodman - founder and editor of the monthly magazine Positive Health - in compiling one of the largest existing data bases on nutritional research and cancer.

In her book Healing Foods, Rosy Daniels addresses the question of healthy eating. It is refreshing to read a nutritional book which is almost solely about food and deals with supplementation in eight pages. There are suggestions for meals and combinations of food to keep generally healthy and tackle cancer and the damaging effects of cancer chemotherapies specifically. The book’s message is supported by a bibliography of cookery books rather than academic and scientific references, although there are plenty of these throughout the text.

It is not possible to discuss food, honestly, in contemporary society without addressing the issue within a political and economic context. Rosy Daniel tackles the health dangers of the modern diet in the first twenty pages of the book, giving a political and economic context to bad food, vitamin and mineral deficiencies, agribusiness, pesticides and food processing.

Although this book is only one hundred and fifty pages long, it is an ideal introduction to the much larger subject of diet and cancer for the lay person and particularly anyone who has cancer or is being treated for cancer.

People come to nutrition as they come to any philosophy or therapy by differing routes. Dawn Taylor trained to be a nutritional consultant, at the Raworth College in Dorking. In 1996 she became a pupil of Gerald Green, a highly regarded medical herbalist. Green is part of a long tradition of self-taught healers and herbalists and has committed his working life to treating respiratory illnesses and inflammatory bowel disease. Dawn Taylor and Gerald Green have a rare voluntary relationship uncommon in today’s increasingly professionalised world.

In 1996, Dawn decided to write a herbal and nutritional handbook, which made public the work and ideas of Gerald Green. What she eventually published was a spiral bound hand-book for self-therapy which described a non-traditional therapeutic approach to Crohne’s ulcerative colitis, candida and multiple sclerosis. The treatment plan is based upon an anti-candida, gluten exclusion diet, supplemented with herbal remedies and an organic food diet which strengthens the immune system. In thirty short sections over 67 pages, Dawn presents a mosaic of the many influences which affect these illnesses.

Self-published as it was, Get Your Life Back was only a

Wide ranging therapeutic hand-book. The basic hypothesis behind the form of Overcoming AIDS is that health is not just dependant upon taking a remedy which alleviates a symptom but upon the individual’s overall attitude to his or her own life. In the case of AIDS-defining illnesses a part of this total approach is an understanding of the social construction of the illnesses, their historical and political context.

Overcoming AIDS can be divided into four basic parts and it is the internal logic of these sections which make the book interesting and easy to read. Byrnes introduces his subject through his own eyes and the condition of a new partner who came into his life carrying the baggage of an HIV+ diagnosis and AZT treatment.

The first three chapters trace the history of the HIV idea and look at the arguments for and against HIV being the cause - or the sole cause - of AIDS. This part of the book also examines in considerable detail the detrimental effects of a wide range of chemical substances, including various pharmaceuticals.

Chapters four to ten look at a number of treatments and remedies for a wide range of AIDS related conditions, as well as listing and explaining different approaches to health care. Overcoming AIDS discusses the effect of common health-threatening illnesses for people who are immune compromised, conditions such as salmonella, pneumonia and cryptosporidiosis. A wide range of detailed protocols to treat these different conditions are discussed. The book also reviews the most well-established ‘grand’ alternative therapies, such as Ozone and Vitamin C, advocated for AIDS and HIV+ conditions. Each therapy is given an evaluating sign.

Overcoming AIDS takes you on a journey, from Byrnes’ first personal confrontation with the effects of an HIV+ diagnosis, through a process of conscious analysis; reading, questioning and discovery to some kind of equitable understanding of a life strategy to deal with the effects of that diagnosis.
limited edition, which sold out very quickly. Since that time, despite updating the book, Dawn has been unable to fund another issue. Get Your Life Back lacks the sophistication of high profile nutritional books despite being a good, eclectic introduction to its subject which informs the reader of nutritional patterns and relationships of which they might previously have been unaware.

The book makes up for its lack of contextualising political or philosophical structure and academic and scientific references, with a genuine feeling of discovery and a gritty concern for an important area of public health.

There is an argument that to change content is to change little; real change, it is said, can only be affected by changing form. For AIDS analysts, this apparently slight philosophical question is especially important. If we dissent from the scientific construct of HIV and AIDS, do we also question the mind and the social organisation of individual scientists, the form of whose knowledge is shaped by their work in ivory laboratories?

A part of the great debate about HIV and AIDS, centres upon the conflict between the non-clinical, distanced objectivity of scientists and groups of individuals who have experienced, or might in the future, experience illness. If we do begin to question the way in which scientific knowledge is produced, how far do we take this campaign? Do we for example retain the analytical tools and methods - such as double blind clinical trials - used by scientists to evaluate new therapies? Do we write in the culturally denuded language which so many scientists employ? Or do we shift the ground of our enquiry, taking an altogether more subjective, intuitive and qualitative approach which is based upon the voice and the experience of those with illness? Do we look more closely at how disease and illness are constructed and experienced in our society?

All these questions are relevant to books about health and nutrition. The best books about nutrition are those which have an implicit understanding of the journeys which people undertake when they begin to empower themselves and get involved in self treatment. There has been a continuous debate within social science over the last fifty years about the nature of narrative and the place of the author in work about social constructs. The most progressive view suggests that because the individual is both affected by and affecting the subject of the text it is best that the author is introduced into the work.

Our approach to the food which we consume is intimately interlocked with all the other aspects of our life in a capitalist society. The only way that we can begin to challenge the way our society works, is to challenge the whole nature of our lives. Changing our care for our own bodies is a good place to begin that revolution.

THE BOOKS

Nutritional Medicine; the drug-free guide to better family health  Dr Stephen Davies & Dr Alan Stewart. Pan Books 1987.


Get Your Life Back a limited edition self-published book by Dawn Taylor with an original price of £9.50. Dawn Taylor can be contacted at; PO BOX 3029, Sherbourne, Dorset, and a copy of her book can be negotiated.

The Nutritional Health Bible Linda Lazarides, Thorsons 1997.


Healing Foods; How to nurture yourself and fight illness Dr Rosy Daniel published by Thorsons for Bristol Cancer Help Centre  London 1996

Positive Health 51 Queen’s Square, Bristol BS1 4LH Tel 0117 983 8851

Journal of the Asociacion de Medicinas Complementarias, Madrid, Spain. Tel. 351 21 11 Fax 351 21 71

CONTINUUM vol 5, no 2
Letters

Farewell
My brother Michael’s illness started in 1989. It was hard for me and with the stress of living up to what he believed were other people’s expectations of him and when he had any physical symptoms he would always fear the worst. But his meditation and spiritual beliefs were a great comfort to him.

For months, years in fact, M. was torn over the decision of whether to undergo conventional treatment for being HIV+. He knew I was against it personally and in his heart I think he was. But the relentless pressure of his peers, society and his consultants made it very difficult for him to justify his own convictions. His weekly counsellor would say to him things like, “Eventually when you like it or not you will be needing to come to us for treatment.”

On 14th January Michael was particularly in pain with his chest and anal herpes. Our sister went with him to see his consultants. Michael described in his diary how Carol was side-tracked and diverted by Dr. M. so that Dr S. could speak to him alone. Dr. S. then shouted in a loud voice at him, thumping his fist on the table. Michael, exhausted. “It’s not what your brother thinks, it’s not what your sister thinks, IT’S WHAT YOU THINK YOU SHOULD DO!” Michael agreed to undertake the treatment. When C. heard the news she was in tears and said could he have one day to think about it. Dr. S. said, “No, absolutely not!”.

However his mental state then diminished rapidly; he was only able to take in a cupful of drugs every two hours or so and for 4 days before he died he was sick every day, getting almost no nourishment. He was on 3 sets of antibiotics and a drip, his oxygen level was low and he had high levels of potassium. He died having collapsed on the way to the bathroom after soiling himself in bed. The death certificate said bilateral pneumonia although personally I think it was a toxic overload.

About a week later I went to Dr S. for more information. He said we don’t know what happened in the end but if he had started the recommended treatment earlier he might have had a better chance. I asked if in retrospect it was right to have given him so many drugs in such a weak condition. He said yes, it was the conventional treatment that we have to give. This made me realise what convention really means - it is a prescribed treatment which overrides any patient circumstances (other than diagnosis of HIV). The Health Authorities have no alternative but to stick to the convention in order to protect themselves.

Anyway I am not bitter towards the health profession. Michael was suffering and is no longer suffering in a body which was failing him. Please continue with Michael’s subscription to Continuum because I read it more than Michael used to.

Yours faithfully,
Chris L.
Newcastle, England

Going it alone
I am a long term survivor of 18 years. I sero-converted in 1979. I’ve been sick several times but have never taken antivirals of a chemical type AZT, DDI, 3TC, etc.

I have however done lots of other things when needed - Ayurvedic medicine, Acupuncture, good healthy living and herbal compounds such as Composition A from I.T.M. of Portland, Oregon.

Please feel free to contact me if you have any interest in my survival long term. Nobody here seems to be at all interested. I can’t imagine why. If I were a medical person I would leave no stone unturned. Much thanks for your wonderful and hope-filled magazine. The magazine POZ is more than half-filled with full-page glossy ads from pharmaceuticals and the rest pretty much is depressing news. Your publication is a ray of light. I’m so grateful.

Adrian M ontagano,
Massachusetts, USA

PWA’s Princess
I just wanted to write a brief note to express my profound admiration for the truly excellent article you wrote in the last edition of Continuum, “The PWA’s Princess.” It was far and away the most thoughtful piece I have read about this whole sad affair of Diana, and to parallel it as you did with the whole sad affair of AIDS, really brought home the fact that we are living in times and which amply corroborate the irrational belief that can be sustained all the while people are motivated solely by money.

I doubt the hysteria your article will doubtles, provoke in certain quarters is raging as I write, but I want you to know that I stand shoulder-to-shoulder with you in all you wrote, because it was thoughtful, honest, and, in a strange sort of way, far more compassionate than so much of the somewhat theatrical “concern” expressed by those who have bought into the AIDS industry and its mythology.

Dave Godin
Sheffield, England

Dolce Vita
You may be interested to know that I finally spoke to an Italian organization that is wholly dedicated to alternative treatments (and philosophies) on the question of the supposed HIV diagnosis and its subsequent treatment, and they receive Continuum. This was good news, and marks an end to my “isolation”, since the last time I dropped into one the AIDS organizations here, ASA (Associazione Solidarieta Aids), they seemed uncritically excited about the protease cocktail. Personally, I have chosen to pursue an immunity-oriented approach, with homeopathic treatment complemented by an attentive diet and lifestyle, the results of which amply corroborate the philosophies expressed in Continuum.

By coincidence, the day of the magazine’s arrival the Italian television network broadcast a special report on the drug companies’ monopoly and manipulation of AIDS treatment round the world. The program was forceful, well paced, and duly critical; notably, the program’s editors received no response to their invitations to interview representatives from Roche et al. to state their side of the argument. However, the main pro-drug doctors were represented by the “Gallo” of Italy Dr. Aiuti, who was, as can be expected, evasive and at times even superficial.

Andy Ellis
Milano, Italy

Kenyan work
Thank you for your consideration to register us on your mailing list. I want to assure you that Continuum magazine has become part of KAIPPTI’s archives on AIDS education, prevention and training. Through this publication we have managed to read much of Prof. Peter Duesberg’s research work. We need much information on truth about AIDS, especially the kind of research being done in developed world is unheard in less developed countries.

Dr. H illary S. M aloba
Kenya AIDS Intervention Project
M umias, Kenya

French Translation
With the authorization of the author, I have translated the Eleopulos interview of Voix/No1 into French.

I pity my fellows who don’t read English and are deprived of such good material. It’s my pleasure to send you a copy, although you may have no use of it. For your archives...

Philippe K yren
Bukoba, Tanzania
Co-enzyme Q10: sustainer of life and energy?

from an article edited by Rohit Mehta

Rohit Mehta B.Sc. is Director of the Hale Clinic, Regents Park Crescent, London

Also known as ubiquinone because it’s ubiquitous, meaning it exists in every one of the body’s cells, coenzyme Q10 (CoQ10) is essential, acting as a catalyst in the creation of energy that cells need for life. The body can’t survive without CoQ10, state Emile Bliznakov M.D. and Gerald Hunt, authors of The Miracle Nutrient Coenzyme Q10 (Bantam). Once body levels of this nutrient become more than 25% deficient, many diseases may begin. These can range from high blood pressure and heart disease to immune system deficiencies and cancer. Moreover, if CoQ10 levels in the body drop much below 75% deficiency, life can no longer be sustained.

CoQ10 supports cellular energy production by helping create adenosine triphosphate (ATP), the body’s primary source of energy. Cells, particularly muscle cells, produce ATP with fuel released from foods - glucose, fatty acids, amino acids, various enzymes, oxygen and CoQ10. CoQ10 is also an important antioxidant, providing protection from oxidative damage occurring in fat-soluble media such as cell membranes, which are composed of fatty acids. As such, it also works with vitamin E to prevent damage to lipid membranes and plasma lipids. Like other antioxidants, CoQ10 also offers protection against accumulation and deposit of oxidised fats in blood vessels, which can lead to arteriosclerosis (Molecular Aspects of Medicine, 1994, vol. 15).

In medicine, CoQ10 has shown therapeutic value in treatment of heart disease, high blood pressure, high blood cholesterol, periodontal disease, immune deficiency, diabetes and ‘AIDS’.

CoQ10 is also an important antioxidant, providing protection from oxidative damage

The body makes CoQ10 from the amino acids tyrosine and methionine. Although CoQ10 is available in food, the therapeutic amounts needed far exceed what the body can make or absorb from food. CoQ10 sources include fish, fish oils, vegetable oils, organ meats and the germs of whole grains, which are also the best source of vitamin E, which synergistically enhances CoQ10’s effects.

CoQ10 exists more abundantly in the cells of some organs than in others. Organs that require the largest supplies of energy to function, such as the heart and liver, have high concentrations of CoQ10. Body levels of CoQ10 are influenced by factors such as stress, cold, illness, hormone concentrations, drugs and physical activity.

Periodontal Disease

Periodontal disease which affects the tissues that support the teeth, including the gums, accounts for more lost teeth in adulthood than any other dental problem. This condition can cause facial disfigurement, pain, an inability to eat leading to malnutrition and the anti-social stigma of profound halitosis. Up to 9 out of 10 adults in the West will suffer some form of periodontal disease in their later lives and as a result 1 in 4 of them will lose all their teeth before the age of 60.

A constant feature of periodontal disease is a deficiency of coenzyme Q10 in the gum tissue cells. This finding led many researchers to study what would happen if coenzyme Q10 were given to restore gum levels to normal. The results were quite enlightening in that most of those treated responded dramatically to the therapy combined with regular periodontal care.

Drugs and CoQ10

Many drugs adversely affect the production of CoQ10, and supplementation with CoQ10 can reduce the adverse effects associated with these medications. Drugs commonly used to lower cholesterol such as Lovastatin also inhibit the manufacture of CoQ10. Common psychotropic drugs (drugs that modify mood or behaviour), including antidepressants, have also been shown to inhibit CoQ10-dependent enzymes. And CoQ10 may help prevent some of the side effects of beta-blockers, drugs that help decrease blood pressure.

Sports Nutrition

Q10 may provide extra help for athletes, who experience higher oxidative stress. Tissue levels of CoQ10 are known to increase with endurance training (Journal of Applied Physiology, 1987, vol. 63). In one study, healthy men aged 20 and up were supplemented with 60 mg of CoQ10 over the course of eight weeks, resulting in improved exercise capacity (Biomedical and Clinical Aspects of Coenzyme Q10. 1981. vol 3).

Coenzyme Q10 and Breast Cancer

In 1993, Dr Karl Folkers, a leading researcher on Coenzyme Q10, reported that people with breast cancer had lower levels of Coenzyme Q10 and that those with cancer of lung, colon and prostate lived longer when they took supplemental doses of this coenzyme. A study published in Biochemical and Biophysical Research Communications (1994:199) shows a benefit for women with breast cancer. Thirty-two women with breast cancer were supplemented with antioxidants, fatty acids, and 90mg of Coenzyme Q10. Six of the patients showed some partial tumor regression. Two of these women were given a daily dose 300-390mg of Coenzyme Q10. In two months their tumours disappeared. Other cases also showed dramatic results.

The Need for Supplementation

Although there are metabolic pathways for the body to make coenzyme Q10, supplementation becomes necessary when synthesis becomes impaired. This may occur as a result of a nutritional deficiency in one or more of the components required by the body to make coenzyme Q10. There may be a genetic or acquired defect in the ability of the body to manufacture it. Alternatively, there may be an increased body need for coenzyme Q10 as a result of a particular medical state or tissue need. Apparently, one of the key factors is simply ageing.

Which CoQ10?

When taking a dietary supplement of Q10 it is important to use a formulation that the body can readily absorb. Scientists at the University Hospital in Copenhagen, who have considerable experience with Q10, use only Q10 which has been dissolved in soya oil and made up in a soft gelatine capsule. Studies have shown this formulation produces optimal and reliable bio-availability. One experiment showed that Q10 in soft gelatine capsules increased the level in the blood 2.7 times. Granular or powder forms were much less effective and Q10 in tablet form had no measurable effect on blood levels.

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I am writing this in a sixteenth century castle in France. The weather is splendid and the autumn hues have never been so beautiful or haunting. Each day I put on my galoshes (wellies) and go walking in the forest or through the beautiful tiny village called Gouvix. Who could have predicted in the sense-crazed seventies, when we were all going wild discovering how necessary it seemed to have lots of drugs and multiple sex partners, that now late into the nineties we would have lost most of our swinging gay friends to a "lethal, incurable world virus"?

There is an interesting trail of events that leads to the fact that I am quaintly labelled "a long term survivor". In this Aids Struggle I am indeed a survivor, but for reasons very different from those which the Aids Establishment may wish to know or accept. When I first quit the sunny shores of Australia in 1968 I had great expectations - to get away from our quasi-isolated continent and discover the rest of the world in all its then perceived mysterious glory - in so-doing I wanted to discover who I was. After all, I was only twenty three years old and knew nothing. Now thirty years later I think I can talk with some experience under my belt. Ironically it took the arrival of whatever Aids is to teach me some of the most important things about the world and myself, and they are not always easily palatable.

As an early baby-boomer I was an Aussie who discovered the sexual revolution not in the sixties but in the seventies. We were in those days a little behind in world movements, but we certainly freed ourselves with a vengeance from our Victorian heritage. With the benefit of hindsight I now see the sixties and seventies as periods of enormous social change in contrast with the eighties and nineties which I feel were an interval to reassess the effects, both positive and negative, of these changes. Overall I feel Aids phenomenon is just one of the factors enabling us to prepare properly for the gigantic changes in store for us in the next century. This may sound horribly simplistic but it is the result of quite a few years of research and observation. I am one of the increasing number who have totally "lived the life" but who remain healthy; I'm also one who has had hundreds of friends who have died with Aids diagnoses - iatrogenically, I strongly believe.

First I would like to clarify my position in this very minor group which is quite separated from what is called by some the "Aids Zone". Like the founder of Continuum Jody Wells, I am an Aids dissident, that is one who doesn't believe the majority opinion that an "hiv" causes "aids". "Hiv" is in quotes because, like my compatriots Eleni Eleopulos and Val Turner who have published a much unheeded but superbly researched proof of the non-existence of this "virus", I also believe that "hiv" is nothing more than a biochemical reaction arrived at through procedures which at a certain time show a "positive" result. The so-called Aids test I believe is the single most misleading and dangerous marker for anyone to take, and incidentally has been the cause of innumerable deaths simply through its application. In 1983 I believe my body secreted antibodies to something or other; five years and two negative tests later I received a positive result exactly at the time that I had contracted a simple bout of the clap. Who knows what this test meant? If I am to trust my body's signs I for one am sure that it is no direct marker of ill-health. But back to my story which I believe may elucidate some of the reasons that the Aids scenario exists.

In 1968 the war in Viet Nam was the biggest political hot potato in Australia. American soldiers were visiting Sydney in droves on Rest and Recreation (R & R) leave. Little did we then know that the drug industry was laying strong foundations for its future market. Hundreds of thousands of brain-washed soldiers were returning to the US, men who would contribute to the gigantic drug culture which was to permeate the country leaving a tragic scar. The Golden Triangle in Burma and Saigon, ironically along with the drug barons of the legitimate sort who were selling antibiotics by the million
to the ever-fearful masses were set to make a killing (forgive the pun). I travelled to the US in January 1969 with no idea of what was going on. The first night I spent in the city of Portland, Oregon where I witnessed a body being carried out from my hotel. I was told by the locals, “just another O.D.” I should have realised the symptoms were already obvious - this was just the beginning. Haight Ashbury, the district in San Francisco fabled for hippies and flower-power, was already strewn with heroin casualties. The “epidemic” in its insidious way had already started, but I didn’t know that fifteen years later scientists were to label it the new pandemic.

On a later visit to New York in ’79 I witnessed the increasing state of sexual madness that immediately preceded the crisis that appeared soon after. So...circa 1981 when the imminent state of sexual madness that immediately preceded the crisis which were to grow around it. But I couldn’t have predicted that appeared soon after. So...circa 1981.

Well they’ve been wrong before. It was Gazzard who in the last year last November, Professor Brian Gazzard, President of the British HIV Association (BHIVA), after a laconic stroll through the topic ended his speech by saying that perhaps 100% of what he had said could be wrong - perhaps 10% was right - only history would tell which 10%.

Well they’ve been wrong before. It was Gazzard who in the days when he was enthusiastically defending his treatment of Freddie Mercury and countless other patients with AZT, complained to the New Scientist that some of his patients had stopped their treatment before it was even half way through and proves nothing.

Double therapy, triple therapy and now quadruple therapy... The pressure is on to try whatever new combination of protease inhibitors and reverse transcriptase inhibitors the drug companies may recommend. But what are these recommendations based upon? Surrogate markers. And what are they? Well they are certainly not evidence of better health and recovery. They are simply interpretations of “viral load” and “antibody responses” to these new drugs based on one paltry study which was stopped before it was even half way through and proves nothing. Wouldn’t it be nice if it were true that PIs (protease inhibitors) really did hold the key to recovery from “AIDS”? Wouldn’t it be nice if it were true, as the media persists in telling us, that AIDS wards are emptying out and people are suddenly restored to health and their old full time jobs? Unfortunately, the few well publicised Lazarus cases have been short-lived. David Roemer was on the front page of the New York Times when, after combination therapy, he was able to take up his bed and return to his job in the Justice Department in Washington, but two months later he was back on the front page - dead.

A visit to some of London’s drop in centres reveals that half the people on combination therapy have not been able to tolerate the regime or the side effects, others have died soon after onto combination therapy and the few who say they feel great are usually recovering well from treatment to a specific “AIDS related” condition.

Attendance at some of the well-meaning PI talk-ins organised by Treatment Action Task Force (TAT) make one shudder at the ignorance and lack of confidence amongst the speakers. At the Royal College of Physicians last November, Professor Brian Gazzard, President of the British HIV Association (BHIVA), after a laconic stroll through the topic ended his speech by saying that perhaps 100% of what he had said could be wrong - perhaps 10% was right - only history would tell which 10%.

Well they’ve been wrong before. It was Gazzard who in the days when he was enthusiastically defending his treatment of Freddie Mercury and countless other patients with AZT, complained to the New Scientist that some of his patients had suspended their treatment after seeing our film critical of AZT (Continuum’s)

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CONFERENCES

TORONTO, Canada - HEAL (Health Education AIDS Liaison) Toronto will be hosting a HEAL International conference on April 11th & 12th 1998 in Toronto, Canada. The conference will focus on fostering greater solidarity between chapters and establishing goals. Topics already on the Agenda include:

- Improving internal communication
- Developing media strategies
- Fundraising
- Establishing an international constituency database.
- Supporting international dissent activities.

Twelve Chapters have already confirmed their participation. Also in attendance will be Vancouver publicist and communications strategist Kevin Dale McKeown. Kevin is the proprietor of Festival Communications, and has directed media campaigns for many high-profile organizations and individuals.

We hope representatives from as many chapters as possible will be able to attend and, while this is specifically a conference for and about HEAL chapters, we do welcome supporters of HEAL’s work.

In conjunction with the conference, on the evening of the 11th, at the George Ignatieff Theatre, HEAL Los Angeles founder Ignatieff Theatre, HEAL Toronto has even arranged to welcome supporters of HEAL’s work. In conjunction with the conference, on the evening of the 11th, at the George Ignatieff Theatre, HEAL Los Angeles founder Ignatieff Theatre, HEAL Toronto has even arranged to welcome supporters of HEAL’s work. In conjunction with the conference, on the evening of the 11th, at the George Ignatieff Theatre, HEAL Los Angeles founder Ignatieff Theatre, HEAL Toronto has even arranged to welcome supporters of HEAL’s work.

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We hope representatives from as many chapters as possible will be able to attend and, while this is specifically a conference for and about HEAL chapters, we do welcome supporters of HEAL’s work.

In conjunction with the conference, on the evening of the 11th, at the George Ignatieff Theatre, HEAL Toronto has invited HEAL Los Angeles founder Christine Maccio to speak at what will be the first in a series of free public lectures introducing dissent speakers to Toronto audiences. We welcome her and look forward to publicizing the AIDS controversy and her remarkable story at this important public lecture. For information please call:

Carl Strygg, Toronto, Canada Tel: +1 416 778 4207, (e-mail: carl@total.net)
or HEAL Toronto Tel: +1 416 406-HEAL, (e-mail: endads@hotmail.com)
Confrencean Agenda suggestions may be be sent to:
mckeown@uniserve.com-Improving internal communication

AFRICA - preliminary interest in a conference on the real health problems in various areas of Africa vs. the failed HIV hypothesis. A national government has expressed interest and some meetings have taken place in London and Switzerland. For info contact Michael Baumgartner @ International Forum for Accessible Science Tel: +41 31 332 9373 or Huw Chriddle @ Continuum Tel: +44 171 713 7071.

SAN MARINO, Europe - International Congress on “AIDS and N atural Medicine”, March 20-22nd. Hosted by the non-profit Poiesis Centre after five years of “work and bibliographic research on medical plants tested and used in HIV infection”, in collaboration with Ministry of Health and Social Security of San Marino (the oldest extant republic), and World Health Organization Traditional Medicine Programme.

For info contact Dominique Hug email: poiesis-ita@www.exodus.it

I.F.A.S. - A growing number of people realise the dead-end street of the established HIV/AIDS approach based on the still unproved HIV-dogma. More people living with HIV/AIDS diagnoses, frustrated with the toxic outcome of 15 years of retrovirological AIDS-research, are looking for alternatives. AIDS analysts have accumulated well-investigated, highly interesting information important for understanding the origin of the HIV-dogma, and the causes of conditions wrongly attributed to “HIV” and called AIDS. The International Forum for Accessible Medicine (IFAS) plans to host an international gathering to make accessible this information to an interested public.

For more information contact International Forum for Accessible Science (IFAS)
c/o Studiengruppe für Ernährung und Immunität, Elisabethanstrasse 51, 3014 Bern, Switzerland. Fax + 41 31 348 1636 Tel + 42 32 332 9373

PUBLISHING

HIV/AIDS and ethics - submissions invited. Journal of Medical Ethics (BMJ publications) seeks articles for first ‘ themed’ issue, Deadline 1st April. Up to 3,500 incl. references preferred. “We would particularly like to see papers focusing on contemporary perspectives.” For info or to contribute contact The Editorial office, Journal of Medical Ethics, Analytic Ethics Unit, Imperial College of Science, Technology and Medicine, Exhibition Road, London SW7 2AZ. Mark clearly ‘HIV/AIDS AND ETHICS’

WORKSHOPS

LONDON - RESEARCHING THE PHARMACEUTICAL COMPANIES: THREE PARTICIPATORY SEMINARS FOR STUDENTS, ACTIVISTS AND AIDS DISSIDENTS

O n three dates in March, Martin Walker, writer, activist and author of Dirty Medicine, will run three ninety minute seminars on researching and investigating pharmaceutical companies. These sessions will look only briefly at academic research but might still be useful to students doing first degrees or post graduate work on issues of conflict and critical social studies. The seminars will be specifically geared to helping people with practical projects or campaigns around drugs, medical research and chemical or pharmaceutical companies.

The seminars will be held at 7.00pm over 3 consecutive weeks in April/May. Times and dates will be set according to number or participants. Attendees have to subscribe to all three seminars, the cost of which will be £ 50 inclusive for professional and waged individuals with concessions to be negotiated for those on grants or social security.

There will be twenty places only on each of these seminars. To register call Continuum on 0171 713 7071 before the last day in March.

An outline of the seminars and more detailed information will be sent to you as soon as you register.

LONDON - The Bryna Trust’s Gift of the Heart seminar, 1-5th April. “A life-enhancing experience. Participants learn valuable techniques and find ways to enhance the quality of their life regardless of the challenges confronting them.” Endorsed by Louise Hay author of “You Can Heal Your Life”. A bit muddled on “HIV/AIDS” but focus on health and living.

For info contact Rena Pearl telephone 0181 455 7661 email rena.pearl@virgin.net

WEBSITES

- German translation of Eleopulos interview with Christine Johnson from Continuum vol 5 No 1 at http://www.virusmyth.com/aids/data2/continuum.htm
- R eappraising AIDS website at http://www.virusmyth.com
Copies are available for all back issues of the magazine. Where we have no stock of original copies, articles reproduced from these issues are available individually. The index below details the contents of recent issues (available as complete magazines). A list of contents of earlier issues is available on request. To order please use the form overhead.

**Recent back issues**

**Vol 5, No 1 Autumn 1997 44pp**
Focus: Christine Johnson interviews leading AIDS analyst biophysicist Eleni Papadopulos-Eleopulos
Healthy Options: Michael eliner on how to choose a doctor in the age of AIDS
Viruse Challenge: Karl Krafeld says scientists always knew HIV was an invention
Hospital Watch: Nursing AIDS patients can be an ethical challenge says Kevin Corbett
CounterCulture: Witchboys: Confession, Possession, Obsession by Alex Russell
Nutrition: Linda Lazarides on the importance of the liver and detoxing
Dissenting View: Whose hysteria?
Plus: News, HIV Watch, Lust for Life, etc

**Vol 4, No 6 June/July 1997 40pp**
FOCUS: Antibiotics: Geoffrey Cannon looks at the magic bullet concept
Micro-ecology: Heinrich Kremer asks some evolutionary questions
Antibiotic alternatives discussed by Leon Chaitow
Interview: Immunologist Prof. Alfred Hässig on politics, risks and therapies
Immune Suppression in Hypercatabolic Diseases, by Alfred Hässig
Conference Report on the Chemotherapy of AIDS, by David Rasnick
Nutrition: The vital role of minerals
FEATURE: HIV, AZT, big science and clinical failure: Martin Walker on the history of an AIDS-defining drug
Escaping the AIDSzone: a new column
Dissenting View: The provocative work of Elaine Showalter

**Vol 4, No 5 February/March 1997 40pp + 24pp Supp**
FOCUS: Protease inhibitors (PIs): PIs in Provincetown: John Lauritsen wonders how hope can exact such a price
From Hope to Mediation: Recent research has led to serious caution
SUPPLEMENT: Peter Duesberg and David Rasnick's The Drugs-AIDS Hypothesis
Conference report: Alternative therapies in France
Interview: Holistic doctor Leon Chaitow, on wide-ranging health

The forces that produce the CONTINUUM magazine and its international network were born out of the necessity for human justice around the absurd death prognosis promoted throughout the AIDS-era.

Fourteen years after the proposal of HIV as the "probable" cause of AIDS, highly toxic medication is still marketed and huge sums of money are spent on biased medical research with little or no hope for the future. Similarly, powerful companies have grown into ever larger pharmaceutical corporations capable, in some ways, of superceding the "richest" nations on Earth. These corporations have substantial financial interests in controlling disease management, diagnostic tests and so-called terminal illnesses.

Naive patients - mostly homosexuals, drug abusers, blacks, US Latinos, haemophiliacs, babies and the destitute - have become free willing guinea pigs condemned to die young after being labelled with HIV. In contrast, images and voices of resistance of many analysts - including scientists, Nobel laureates, medical doctors, researchers and health activists - worldwide have been abruptly erased and silenced by the mass media for questioning the HIV/AIDS hypothesis.

CONTINUUM is a life-affirming organisation mostly run by and for people whose deepest desire is to remain healthy after an HIV/AIDS diagnosis.

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FOCUS: Pneumonias & Lung Diseases: Aquired Iatrogenic Death Syndrome: Dr. Heinrich Kremer examines the real causes of PCP and other lung diseases that are usually labelled "HIV-associated"
Counter Culture: Ian Young's The AIDS Cult and its Seroconverts: part 1
Sexual Health: Anal Sex and AIDS examined by Fred Cline
Lifestyle: E for Ectasy or 'ealth? Club culture from a sociopsychological perspective
Nutrition: Knowing your Immune System
Science Speak: Prof. Alfred Hässig on Hepatitis viruses
Viral Load & the PCR: Christine Johnson explains why they can't prove "HIV" infection
Review: Toxic Sludge is Good For You! Lies and the public relations industry
DrugEffects: Corticosteroids
SWARL: World AIDS Day hype and MDR-TB
Dissenting View: The UK's long-term survivors study under the microscope
PLUS: News, HIV Watch, Lust for Life, etc.

CONTINUUM magazine is a unique forum for those in the scientific and health communities challenging the AIDS orthodoxy. We're a voluntary organisation dedicated to providing information we believe necessary for the fuller understanding of HIV/AIDS and immunity. We aim to encourage those whose lives have, in some way, been touched by the hypothesis, to unite and demand scientific proofs that HIV has been isolated, that it exists and that it causes AIDS. Our workers are unpaid and the organisation relies on subscriptions and donations to maintain its work. Your support in any way is greatly appreciated.