CONTINUUM
changing the way we think about AIDS

Noam Chomsky a duty to challenge

HIV origin of the specious

animal trials human tribulations
The orthodox view on AIDS holds that it is caused by a virus known as HIV that is transmitted through the exchange of body fluids. Once infected, a person will remain well for a time, though infectious to others, before going on to develop AIDS and dying.

Despite the huge sums of money spent on medical research, there is still no cure, just drug therapies said to slow the progress of the disease, and regular T-cell counts to measure health.

A whole industry has evolved around AIDS, on which many careers and businesses depend, but which offers little hope to those affected. It works on the premise that HIV=AIDS=DEATH.

Continuum began as a newsletter encouraging those affected to empower themselves to make care and treatment choices. As we look further, anomalies in the orthodox view continue to appear.

Are you aware, for example, that the link between HIV and AIDS has never been more than hypothetical? That a growing body of scientists and doctors throughout the world doubt that HIV causes AIDS?

At the onset of the “epidemic”, the hysteria that resulted from the linking of sex, death and an infectious virus created a climate where to question the “facts” was considered reprehensible. Many of those who dared to do so were silenced or ridiculed. Since the growth of the orthodoxy, those who question have also had to contend with the weight of vested interests.

Twelve years after HIV was first associated with AIDS many predictions based on the viral hypothesis are failing to materialise.

Continuum is a voluntary organisation dedicated to providing information we believe is necessary for the fuller understanding of HIV, AIDS and immunity. All our workers are unpaid and the organisation relies on subscriptions and donations to maintain its work. Your support in any way is greatly appreciated.
Grave implications for accuracy and honesty in HIV test results

Prejudice drives the production of songs that are free and in cyberspace, on the World Wide Web at www.theaterweek.com. Described as "A documusical" about an epidemic of lies, by composer Tom Steele and New York Native editor-in-chief Charles Ortleb, it draws attention to the work of two journalists, Hillary Johnson, and Neenayah Ostrom the Native’s investigative reporter who has written hundreds of articles on AIDS and the Chronic Fatigue Syndrome.

Says lyricist Ortleb, "These are songs about fraud and silence and include mothers singing about their dying sons, people singing about the government lies, and lovers singing about lost lovers." The production follows the story of Nathan Gate, publisher of a small newspaper, who discovers that much of what the public is being told about AIDS is untrue. Using music and biting lyrics, the story unveils details of "AIDS-gate" - the concealment of potentially life-saving information about AIDS and CFS by government, the scientific community and the media.

In the future, Web listeners - the RealAudio software for listening to the musical any time in real time can be downloaded for free - will be able to participate in the growth of the production and will be able to choose which characters to follow. Additional Web links, e.g. to the forthcoming Continuum Web site, will enable users to explore science and news on both AIDS and CFS, with chat rooms for discussion.

Says Ortleb, "The music industry may be unnerved in that there are some free songs, but this is an issue that the entertainment industry needs to get involved in."
A PhD student at Edinburgh University claims in The Lancet that it is “easy to document” HIV in CD8 cells – 15 years after the search for “HIV” began. In the week after an unexplained arson attack involving 30 fires at its offices, UK’s mass-circulation Pink Paper printed two stories on the subject, one by a staff reporter and one by researcher Wendy Livingstone herself, on the shakey assumption the claims were not a smokescreen.

Pressed for minimal details of their work however, Livingstone and supervisor Peter Simmonds revealed lax scientific standards in their work despite claiming that “HIV research has gone really well.” Using DNA-PCR, a technique to amplify genetic material in cells (rather than retroviral RNA), Livingstone looked for HIV by documenting the hypervariable region of a theorised HIV gene called env. Asked how the researchers were sure this highly variable region of cellular DNA was specific to an isolated retrovirus HIV, Simmonds asserted “infection equals viral isolation”. Scientists disputing this point output genetic material can be readily incorporated into cells whether it is viral or not. Livingstone says that having her first paper published in The Lancet has been “quite cool”.

CD8 “HIV”-infection inflames gay press

Poppers by another name

Sex shops and clubs in Britain are selling amyl nitrite, risking prosecution by the Royal Pharmaceutical Society which won an expensive case against their sale earlier this year.

The Crown Court ruled the “recreational drug” could only be supplied by doctors or pharmacists, after hearing evidence of its harmful effects on regular users. London’s Clone Zone currently sells four brands of the pungent inhalant, identifying it as “a room odouriser”. A Department of Health spokesman stated, “Any case of improper sales should be reported to the Medicines Control Agency and there are several investigations in progress.”

Use of amyl nitrite and its analogues has been linked with Kaposi’s sarcoma and immune suppression in “HIV” positive and negative gay men.

Infection verdict overturned in Argentine court

La Plata and Gente del Dia newspapers reported that a verdict has been overturned in the Court of Appeal and Criminal Justice in La Plata, Argentina, against five doctors of the Centro Modelo de Dialisis for infecting 34 patients with the “Aids-virus”, 14 of whom were said to have died through “HIV-infection”.

The Court maintained it has not been proved that the patients had acquired HIV and died as a result of negligence, and that the renal patients’ condition may have deteriorated due to treatment, immunological suppression and continuous infections, especially in the lungs. Except for one 45 yr-old all of those who died were aged over 70.

According to the Court it has not been scientifically shown that HIV is a virus that causes AIDS. One of the judges, Dr Horacio Piombo, stated: “You cannot condemn anyone while there are scientific doubts”. He added: “AIDS does not touch anyone brimming with health unless the person destroys his/her own immunological system through drugs, prescribed or otherwise”.

ACT-UP CHARGES

Three members of the pro-health AIDS activist group ACT-UP San Francisco have denied charges of criminal assault following their take-over of the city’s Davies Medical Center in August, and now face a full trial over the incident. They protested at the centre’s decision to stop them using a room for their meetings.

GAY RIGHTS MOVE

The Irish government is to submit a paper arguing for sexual orientation to be included in an anti-discrimination clause in the new EU treaty, says negotiator, Noel Dorr. The treaty needs unanimous acceptance, but several countries are known to be against such clauses, with some requiring a referendum.

SCHOOL TESTS

A private boarding school for members of a religious cult is insisting on an HIV saliva test for each of its 50 pupils every term. All visitors to the Osho Ko Hsuan school in Chawleigh, Devon, UK, are also required to present a recent certificate of being HIV-negative. Headmaster Suvendra said they had a strict hygiene policy “because we are not sure exactly how the virus is spread”.

PATENT DISPUTE

Swiss pharmaceutical firm Hoffmann-La Roche risks losing its rights to Taq DNA polymerase, the key enzyme used in the PCR test for HIV. A US federal court is to hear claims that the biotechnology firm Cetus falsely claimed to have originated the enzyme when it was patented in 1989. Roche bought the patent in 1991 along with the PCR methodology.

WOODSTOCK LEGACY

A new study into the prevalence of Hepatitis C suggests as many as 500,000 people in Britain are affected, many of them unaware of it while those who are keep it quiet. Dubbed the “Woodstock Generation”, they are theorised to have contracted it as IV drug users in the 1960s, but now have respectable lifestyles to protect.

GERSON THERAPY

The Gerson Institute, California, confirms policies in the US and Mexico forbid them accepting people with AIDS. They report two AIDS patents using Gerson Therapy on their own - one has since tested antibody-negative. The Institute treats thousands of patients for “incurable” cancer and other degenerative diseases using holistic and natural treatments, high quality nutrition, and improved detoxification.
“There’s a storm, blowing us into the future. This storm is called: progress.” – Laurie Anderson

Change is a challenge that requires some courage. At times only a small voice of reason and compassion which stretches like a silver thread into the future. At others, the roar of a wave that will redefine the landscape. In his interview Noam Chomsky says in our scientific age “error tends to be discovered and corrected pretty fast.” In this issue, the historic paper by Dr. Eleni Papadopulos-Eleopulos and colleagues demonstrating “HIV” has never been isolated and so is shown to exist, challenges all scientists and others working in this troubled field. Since 1988, Eleopulos and colleagues have achieved a brilliant list of publications in leading journals including Bio/technology and The Lancet. This phenomenal paper for Continuum may be the most important turning point in AIDS ever. Perhaps a little over a decade is “pretty fast” in scientific terms, but we have often had to measure the days of “HIV” in lives lost and futures clouded. Time is relative.

Workers, and their authors, they will not be held responsible for any inaccuracies contained herein. Inclusion in the magazine of therapy information or advertisements cannot represent an endorsement. Information should be used in conjunction with a trusted practitioner.

Whose antibodies are they anyway?

The AIDS establishment has managed to convince many people that the HIV antibody tests (ELISA, IFA and Western blot) are “99.5% accurate”. In this article, Christine Johnson, from HEAL Los Angeles, lists conditions documented in the scientific literature known to cause positives on these tests, and gives her references.
Factors Known to Cause False-Positive HIV Antibody Test Results

- Anti-carbohydrate antibodies
- Naturally-occurring antibodies
- Passive immunization: receipt of gamma globulin or immune globulin (as prophylaxis against infection which contains antibodies)
- Leprosy
- Tuberculosis
- Mycobacterium avium
- Systemic lupus erythematosus
- Renal (kidney) failure
- Hemodialysis/renal failure
- Alpha interferon therapy in hemodialysis patients
- Flu vaccination
- Herpes simplex
- Upper respiratory tract infection (cold or flu)
- Recent viral infection or exposure to viral vaccines
- Pregnancy in multiparous women
- Malaria
- High levels of circulating immunocomplexes
- Hypergammaglobulinemia (high levels of antibodies)
- False positives on other tests, including RPR (rapid plasma reagent) test for syphilis
- Rheumatoid arthritis
- Hepatitis B vaccination
- Tetanus vaccination
- Organ transplantation
- Anti-lymphocyte antibodies
- Anti-collagen antibodies (found in gay men, haemophiliacs, Africans of both sexes and people with leprosy)
- Serum zymosan-activated factor, antinuclear antibody (both found in rheumatoid arthritis and other autoimmune diseases)
- Autoimmune diseases
- Systemic lupus erythematosus, scleroderma, connective tissue disease, dermatomyositis
- Acute viral infections, DNA viral infections
- Malignant neoplasms (cancers)
- Alcoholic hepatitis/alcoholic liver disease
- Primary spherocytosis
- “Sticky” blood (in Africans)
- Antibodies with a high affinity for polystyrene (used in the test kits)
- Blood transfusions, multiple blood transfusions
- Multiple myeloma
- HLA antibodies (to Class I and II leuko-/anti-haptens)
- Anti-smooth muscle antibody
- Anti-parietal cell antibody
- Anti- hepatitis A IgM (antibody)
- Anti-Hbc IgM
- Administration of human immunoglobulin preparations pooled before 1985
- Haemophilia
- Haematologic malignant disorders/syndromes
- Primary biliary cirrhosis
- Stevens-Johnson syndrome
- Q-fever with associated hepatitis
- Heat-treated specimens
- Lipemic serum (blood with high levels of fat or lipids)
- Haemolyzed serum (blood where haemoglobin is separated from the red cells)
- Hyperlipidaemia
- Globulins produced during polyclonal gammapathies (which are seen in AIDS risk groups)
- Healthy individuals as a result of poorly understood cross-reactions
- Normal human ribonucleoproteins
- Other retroviruses
- Anti-mitochondrial antibodies
- Anti-nuclear antibodies
- Anti-microsomal antibodies
- T-cell leuocyte antibody antigens
- Proteins on the filter paper
- Epstein-Barr virus
- Visceral leishmaniasis
- Receptive anal sex

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REFERENCES


CONTINUUM VOL. 4, NO 3 5
Advertising watchdog whimpers

“In any battle, first conquer the eye”
— Tacitus (55-120 AD)

The Independent Television Commission (ITC) in Britain has been responding to numerous complaints about highly visual advertisements aired on Channels 3 and 4 for pharmaceutical multinational Glaxo-Wellcome, which seek to persuade viewers the company is the market-leader in HIV/AIDS research. Explaining, “We do not consider the manner in which the information is presented to be likely to mislead viewers”, the ITC is now publicly displaying its inadequacy to monitor the honesty and ethics of advertising on TV in an environment where technology and drugs are the new Tupperware.

The advert in dispute asks, “Why have thousands of our scientists, along with the universities and hospitals, spent twelve years and over 500 million pounds researching into something which measure one 10,000th of a millimetre?” The visuals, according to Glaxo-Wellcome’s transcript, “Open on a moving microscope slide of an Aids virus.” The picture shows how “It continually mutates”, while the soothing voice-over asserts: “Because this is the HIV virus that leads to Aids. Which has killed more people than died in the Allied forces in the whole of the First World War”. The “Camera starts to move in – one element of the virus looks like a poppy”, until the “Visuals clear to white” and viewers are asked to believe “Man has no greater enemy than disease. Disease has no greater enemy than Glaxo-Wellcome.”

“...it would appear that the majority of scientific and medical opinion believes that the HIV virus is causally linked to AIDS.” With classic myopia, the ITC has absorbed the assurances of Glaxo-Wellcome’s PR, derived in turn from the likes of Dr Graham Darby, International Therapeutic Director, Viral Diseases Research, Glaxo-Wellcome.

Many Britons will recognise the name Saatchi, who produced the advert...they’re doing – but it’s only distantly related to solving AIDS.

In his popular examination of the political economy of the mass media, Manufacturing Consent, Noam Chomsky discusses the relationship between propaganda and media: “The essential ingredients of our propaganda model...fall under the following headings: (1) the size, concentrated ownership, owner wealth, and profit orientation of the dominant mass-media firms; (2) advertising as the primary income source of the mass media; (3) the reliance of the media on information provided by government, business, and “experts” funded and approved by these primary sources and agents of power; (4) “flak” as a means of disciplining the media...”.

Many Britons will recognise the name Saatchi, the agency who produced the Glaxo-Wellcome advert, and might not expect truth to get in the way of their account margins. More disturbing is the complacency and subservience of apparently independent media professionals, among whom the ITC should certainly be counted — it is a body that seeks to define where standards of truth and reality should lie. Chomsky argues, “The elite domination of the media and marginalisation of dissidents that results from the operation of these filters occurs so naturally that media news people, frequently operating with complete integrity and goodwill, are able to convince themselves that they choose and interpret the news ‘objectively’...”. The same sadly can be said for the reporting of the authenticity of advertising.

Saatchi, the agency who produced the advert, evidence was presented in at least two complaints that HIV has never been isolated and so cannot be stained, sectioned or grown. Is it possible that McMurchie has overlooked a highly complicated technique, Data Review and Analysis, or “homework”? If the analogy is that AIDS patients are dying like cannon-fodder, asking Glaxo-Wellcome if they know what they’re doing is like asking arms manufacturers how far off peace is. Of course, Glaxo-Wellcome are clear what
how viewers are likely to interpret or react to advertising. The ITC requires the television companies to check any claims made in an advertisement before accepting it for transmission. "Anybody who thoroughly checked whether (i) ‘HIV’ causes AIDS or (ii) ‘HIV’ is known to exist, would arrive at negative conclusions. But how realistic is it to expect media workers at commercial TV companies to achieve this? Surely what is necessary is for the ITC to acknowledge that there is at least grave uncertainty in the scientific community about a relationship between a human immunodeficiency virus and AIDS, and put a hold on the gross profiteering of huge drug companies who may be causing untold damage. In the darkest of ironies, while claiming ‘Aids...has killed more people than died in the Allied forces in the whole of the first World War’,  Glaxo-Wellcome continue to market AZT (Retrovir) worldwide at substantial profit, inducing immune suppression and death in naive consumers. John Lauritsen states in his important book Poison by Prescription: The AZT Story, ‘The toxicities of AZT are firmly established. The drug is cytotoxic (i.e. it kills healthy cells); it destroys bone marrow; it causes severe anaemia, headaches, nausea, and muscular atrophy; it damages the kidneys, liver, and nerves; and it inhibits DNA synthesis. The consequences of AZT toxicity should not be taken lightly. When DNA synthesis is blocked, new cells are not formed, cells do not develop – the life process in effect comes to a halt. Joseph Sonnabend, a prominent New York city AIDS researcher and physician, expressed it succinctly: ‘AZT is incompatible with life.’ In August this year, Glaxo-Wellcome was ‘pleased to announce that the European Medicines Evaluation Agency (EMEA) has granted a licence for 3TC, now known as Epivir, to be used in combination with other antiretroviral agents for the treatment of HIV...’ A nucleoside analogue in the same class as AZT, it’s safety and efficacy trial was stopped early and it is now available.

Yet it seems to be the belief of the ITC that details, even when they spell death, are not the concern of the viewing public. If a process is ‘complicated’, it is to be unquestioned. We live in a climate of witless reassurance. Writes Chomsky, ‘Advertisers will want, more generally, to avoid programmes with serious complexities. Advertisers will want to avoid programmes with serious complexities’.

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Suicide Pact?

San Franciscan anti-AIDS activist Fred Cline comments on reports of gay euthanasia

Researchers reported at the 11th International Conference on AIDS in Vancouver that physician-assisted suicides among people with AIDS are probably far more common than previously believed.

A survey of 118 physicians who treat people with AIDS found that 53 percent had helped their terminal-stage patients die. A similar survey in 1990 found that only 28 percent said they had.

A British researcher also reported that between 10 and 20 percent of the patients with AIDS in British Columbia seek their doctors’ help in dying. The report also indicated that 83 percent of the people with HIV and AIDS in Canada believe they would consider suicide a valid option for themselves, and that 44 percent had made plans for some sort of euthanasia.

Comment: I am wondering what percentage of these AIDS patients are gay men? I would offer a suggestion of around 98%. In any event, the vast majority of AIDS patients are still gay men when they practically NEVER assist people with cancer or other terminal diseases to die. In fact, Dr. Jack Kevorkian has been called into court about a dozen times in order to try to hold him accountable for the assisted suicides he was involved in. I believe these were all straight people with diseases OTHER than AIDS. Has anyone heard of Kavorkian helping an AIDS patient to die?

I don’t think one needs to be very bright to see what is going on here. Surveys have already shown that the majority of medical doctors are uncomfortable with gay men (homophobic?). Is it any wonder that they are so ready to come up with the “final solution” at this point in time?

Those of us who do not believe in HIV as a pathogen see this as a continuum of increasingly lethal prescribed drugs ending in DEATH. How many would have never died if they had not fallen into the hands of their doctors? First the poisonous “cocktail” and then the coup de grace... “Cocktail” sounds so innocent, doesn’t it? As if we are going to a party...

What is so appalling is that these poor victims believe they are being kindly dealt with and that a gay newspaper can print this as a straight news story without seeing the obvious double standard that is taking place.

We have come a long way from the days in the 19th century (yes, my dears, it was just a little over 100 years ago that gay men were still being hanged for sodomy. Look it up.) Now they have invented a 100% fatal sexually transmitted disease and then brainwashed us into clamouring for the prescribed poisons and the outcome is always as it has been planned from the beginning—DEATH.

And all the while they continue to do research on the phantom virus, extracting billions of dollars from the US taxpayers who don’t have a due as to what is happening. It is such a perfect scenario that I believe it must have been invented. If it was not invented, then it is repressed homophobia which drives the whole thing.

Will history ever tell the truth about this one?
In June this year animal liberationists protested en masse in Washington. A debate on the role of animal experimentation towards a solution for AIDS crossed the pages of Britain’s gay press. London scribe Edward King admitted “Animal research played an important role in the development of every single one of the new anti-HIV drugs...” while Karl Burge condemned calls for “a change in research to protect rodents whilst never publicly calling for an end to so-called ‘anti-HIV’ antiviral research...”

George Orwell had his tyrant porker say in Animal Farm, “All animals are equal, but some are more equal than others.” Was this a vision of corrupt socialism or survival of the fittest, or should we resist trying to interpret fables in which animals can give sound-bites? There are those who argue our human self-respect is inseparable from our respect for fellow species. Yet is it an act of respect to identify animals in our own image, endowed with individualistic human moral ambitions and self-pity?

It’s argued by others that laboratory monkeys, for example, are the surest guarantee of their species – that like cows and sheep, and to a lesser extent household pets, species which expend members to satisfy humans will always have a place with humans, with an evolutionary advantage.

Three authors in the following articles look into the issues and alternatives as they arise the context of AIDS research.
being voiced increasingly by scientists who are highly critical of the huge expenditure on animal-based HIV studies by the Medical Research Council (MRC).

"The jury is still out on whether animal experiments are useful predictors of what happens with humans," according to one of Britain’s senior AIDS researchers, who requested to remain anonymous. "To spend so much money on this area (primate research) is almost criminally negligent".

What we do know for certain is that vivisectionists have found it impossible to inject animals with HIV and induce AIDS, except in one seriously disputed case. Conversely, the monkey immunodeficiency virus SIV does not produce AIDS in people. Critics say that setting aside so much of the AIDS research effort to studying non-humans means that far more important studies of the human immune system are being neglected and under-funded.

As long ago as 1992, scientists such as Professor Robin Weiss and Dr John Moore warned: “Is is a worrisome possibility that experiments on SIV may not be predictive”. Their view was echoed by the head of a private HIV research company funded by the MRC who suggested that “monkey models are not appropriate”. A similar opinion was expressed by Albert Sabine, the veteran scientist who developed the first oral polio vaccine: "Some of the MRC committee have evidence that other ideas are correct, but they are not publishing it”, he said.

The bulk of AIDS research revolves around HIV. The scientific focus is on understanding how HIV may destroy the immune system and on the development of a vaccine and cure. Leaving aside the contentious issue of the extent to which HIV could be the sole and exclusive factor in the AIDS equation, many current HIV theories and treatments are profoundly problematic in that they are based on experiments with other species. These have dubious relevance to people with AIDS, given the vast differences between human and non-human physiology. Even from within its own scientific parameters, much HIV research stands accused of using invalid animal research models.

Interestingly, a reported breakthrough in AIDS research in 1989 was part funded by an anti-vivisection organisation, the Dr Hadwen Trust for Humane Research, which is dedicated to the promotion of cruelty-free medical science.

It was the Trust’s funding that helped finance Professor Jonathan Weber’s team at St. Mary’s Hospital Medical School in London. This team produced new insights on the [theory of the -ed.] mechanism by which HIV [allegedly -ed.] enters human cells.

Professor Weber believes that this may never have come about if, like many other scientists, he had concentrated on experiments with chimpanzees, monkeys, rabbits, mice, cats and guinea pigs. "Understanding how HIV responds in humans is crucial to the development of a vaccine and cure”, says Professor Weber. “You’re unlikely to get that information from research with other species”.

Humans and non-humans have a quite distinct physical and immunological make ups. There’s also the uniquely human mental and emotional influences on illness. Our attitudinal response to disease produces a tangible psychological-immunological interaction that can affect the outcome. These factors don’t apply to other species. As a consequence, HIV and the drugs developed to treat it will inevitably react differently in people and animals.

Professor Weber also points out that tests on rats and mice failed to predict some of the adverse side effects of AZT and ddI on people with HIV. All that animal tests offer is a vague guide to toxicity, which may or may not reflect how the drug will affect humans.

The team at St. Mary’s Hospital believe that human volunteer trials can give fast and accurate results. Speed and veracity are the two factors of major importance to the millions worldwide diagnosed with AIDS.

The safety of new drugs can be assured using computer models, and human cell tissue and organ cultures. Another option is the administration of tiny, harmless doses to human volunteers and the monitoring of their internal effects by means of biopsies, lasers and ultrasound probes.

These test methods are usually a better indicator of a drug’s safety than experiments with other species. Arsenic and asbestos, for example, rarely cause cancer in animals, but often do so in humans. The use of Digitalis as a treatment for heart patients was delayed for many years because it was first tried out on dogs and resulted in their development of dangerously high blood pressure. Thalidomide tested safe on animals, yet it resulted in thousands of human deformities. Likewise, humans and other species react differently to anti-AIDS drugs. After experimenting on chimpanzees for 10 years, in late 1995 US AIDS scientist, Patricia Fultz, said she doubted that animal research can advance the understanding of HIV in humans or contribute to the development of successful new treatments: “I don’t think it (infecting chimps with HIV) will make any difference at all in vaccine development”.

Despite the emotive claim that animal research is necessary to save the lives of people with AIDS, there is not a single significant breakthrough in AIDS research that can be attributed to animal experimentation. Vivisection is a discredited, disreputable pseudo-science that is holding back a genuine understanding of AIDS and how most effectively to defeat it.  

Chrisie Hynde and The March For The Animals in Washington DC
The growing debate on human rather than animal testing in “HIV” research makes me aware of the need to address certain facts rather than myths in relation to “HIV”/AIDS experimentation. Long before I was involved in any AIDS-related work I was deeply concerned about what happened to animals in labs in the “interests of humanity”. At first I thought the case against vivisection could be handled simply by a romantic animal-liberationist approach, considering animals and humans equal as its basis. Yet looking into the issues in more depth, I learnt the basic argument against vivisection lies in science itself. I will not linger on the ethical issue here as I have been asked to keep my arguments short.

Those who proclaim “For queers to demand an end to homophobic oppression while supporting the oppression of other animals...”, imply homosexuals are animals and equate animals and humans. This is a fundamental mistake! Such a statement reveals more about the unconscious level of self-esteem of some homosexuals than about the ideals of self-identified political activists. I am not at any stage saying, animals are ‘only’ animals to excuse further cruelty in modern torture chambers, a.k.a. laboratories. The issue is that if we put humans and animals on the same plane by humanising animals or dehumanising humans, we invite the assumption that comparison is of scientific relevance when searching for treatments for humans. And here we are right in the middle of the scientific debate. Animals are not humans and can therefore not replace us when it comes to human testing. I am fully aware that there seem to be some grey areas, and I argue that even those can be solved without the disposal of our “brothers”, as Francis of Assisi (a Saint by Catholic standards) called animals. Defence of vivisection is primarily scientific, and most often indicates scientific expediency. Those who ‘humanise’ animals, be it in modern torture chambers or at home, do not understand their uniqueness and beauty and are probably not able to understand their needs. If so they can’t possibly understand what they tell us even in expensive test results. That is the main argument against vivisection: using animals to find human treatments is scientifically irresponsible! None of the findings of vivisection can be simply applied to humans. Medical history tells us of the shortcomings and horrors of such attempts. Many important books have been written on this topic by ethically and scientifically responsible people such as Hans Ruesch, F. and S. Delarue, Gerhard Buchwald, Pietro Croce, Bernhard Rambeck, etc.

Yet while anti-vivisectionists rightly point out that the animal model has not produced anything valuable in “HIV” research, they misunderstand the reason for that. It is not just that the animal model fails for reasons just pointed out; the “HIV”/AIDS model in itself is a total failure. I know some people do not like to hear that basic fact, but it needs to be said here again, especially to understand the endangered role people play in “HIV”-research, promoted to relieve us from the great “AIDS plague”. When scientists talk about human testing in so-called HIV-research they do not mean just tissue cultures – what they really seek is the “whole thing” i.e. the person. This must be pointed out very clearly! Human tissue cultures, to a great extent, would be a good alternative to animal testing, but they do not replace the human being as a whole, just as animals don’t. What researchers want is human guinea-pigs. Human wasting material is sought and not because there is scientific justification, but because science, which has got completely lost, covets it! And since homosexual men now offer themselves, that demand is no longer considered ethically unsound.

AIDS-activists might be unaware that human testing has been going on for quite some time now. One could argue that human testing has gone hand in hand with the development of - and is therefore as old as - our medical establishment. Although still more or less secret in the so-called Western world, the first widespread government-approved example was in the period of the Soviet Union called the Gulag from 1918 to 1956, where an estimated 10 million people were systematically killed, many homosexuals amongst them (homosexuality was illegal in the former USSR too) – most of them in government clinics and camps. Another regime torturing humans while claiming human benefits – already less secretively – was the
The following examples are from the pool of far too many, demonstrating the daily heritage of these fascist professionals: after the Second World War up to 1974 at least 23,000 humans underwent experiments with radioactivity, without consent! How much further knowledge was really needed after Hiroshima and Nagasaki! How was the “medical science” with their tax-funded presence. Plus ça change... The remainder were given new white coats so they could rebuild the post-war German medical establishment. No government really wanted to loose out on the possible value of their experiments. Much importance was placed being “scientifically” ahead in the new ways of terrorising citizens and killing possible future enemies. These “doctors” and “scientists” laid out what has become known as “modern medical science”. That is the true foundation of what so many people still seem to need to believe in today.

The human wasting material is still chosen out of minority groups, poor and disfranchised societies and from fewer than finally “will be saved”: though it’s human wasting, now at least it’s public-ised human wasting no longer done in secret, nothing to be ashamed about. Genocide pure and clean, in its way made socially acceptable.

Most queers volunteering in “HIV” experiments do not know the facts of either “HIV” or AIDS. If they would, they would hardly participate. They are so frightened and desperate that any outcome of their case-studies is so highly questionable as to be useless. Helping in understanding and coping with their fear should be considered therapeutically responsible, not an aid to make a personal fortune. Yet a responsible doctor in the AIDS disaster is no longer one who helps patients regain quality of life, but instead enrolls her/him in as many toxic trials as possible, with the outcome being death. Do AIDS-activists really believe a minority disease like AIDS, still restricted to risk-groups or borderline-groups - i.e. a certain fragment of homosexual men, drug addicts and economically poor people - is of common concern or in the long run generates significant profits? Please get off the cross, it is already occupied! Or in other words, if “queers” need to push the limits of their lives, at least take responsibility for it and accept the consequences and fight for something worth fighting for! When it comes to AIDS, become a dissident by first walking out on the doctors of death! Now that is what I call queer!

Sources
AIDS Inc. by Jon Rappoport
Human Testing by Peter-Ferdinand Koch
The Psychosocial Support of Victims of Torture by myself as well as literature by authors mentioned in the text.
Rights and Wrongs: from animals to humans

ALEX RUSSELL, founder of the underground anti-AIDS publication Death Camp, maintains that rights are conferred on animals out of sentimental humanism and asks how far off is cross-species organ transplantation.

But there will never be a vaccine against, or cure for, ‘HIV’ because it does not exist. Therefore, in the context of ‘HIV’ research, I argue against using both animal and human guinea pigs in pursuit of this phantom ‘virus’, on legal, scientific and logical grounds. ‘HIV’ science is fraudulent and catastrophically destructive to all life forms. The pharmaceutical-scientific-medical ‘HIV’ cartel have been responsible for gross human rights violations worldwide. 29 States of America make it a felony/misdemeanour to engage in ‘risky’ sexual activity if a person is tested positive for ‘HIV’ antibodies. Neenyah Ostrom recently reported: “On June 27th, 1996, the American Medical Association endorsed a resolution calling for the mandatory testing of pregnant women for ‘HIV’. Currently, the only individuals in the USA who are forced to be tested for ‘HIV’ are prisoners. Are pregnant women about to become prisoners?”

‘Rights’ are an ephemeral intellectual concept that we have sought to confer on animals out of liberal sentimental humanism. Marina Cronin, in a statement for OutRage! argues: “Even when faced with the horror of the AIDS epidemic, there can be no ethical justification for colluding with the victimisation of living, thinking, feeling creatures”. Thinking? Does she mean human beings or animals here? OutRage! blur the distinction between human and animal, promoting a kind of Disneyesque anthropomorphism of a most embarrassing kind. Peter Tatchell asks: “Where’s the moral consistency in wanting freedom for ourselves but denying it to other thinking, feelings creatures? All sentient animals – human and non-human – have a right to life, liberty and the pursuit of happiness, irrespective of their species, race, sex, class disability or sexual orientation”. This sounds like an animal equal opportunities charter. I find it hard to visualise a working-class, wheelchair bound Buddhist lesbian sooty mangaby “in the pursuit of happiness”, but others obviously don’t. Unless he is careful, Tatchell might be suspected not only of advocating anal sex with 14-year old chickens, but also guinea pigs and mangabies – with or without wheelchairs. Inanities about ‘animal rights’ display an intellectual immaturity and political naivety: how can ‘animal rights’ be given the same status as human rights? With Palestinian and East Timorese people being raped of their political and territorial rights it is a luxury to give animal rights the urgency and priority of human rights. OutRage!’s romanticism of animals is rooted in the 19th century; Beatrix Potter has a lot to answer for. OutRage! demand the development of a vaccine and cure for ‘HIV’. Would they volunteer to test an ‘AIDS’ vaccine? If so, how could they know it was working without deliberately practising the kind of ‘unsafe’ behaviour alleged to cause ‘AIDS’ - unprotected anal sex, lots of STDs, lots of antibiotics, and street drugs injected with filthy needles? A vaccine can be declared valid only after it has withstood the challenge of direct exposure to the pathogens known to cause the illness. How can you have a ‘cure’ or ‘vaccine’ for 29 different ‘AIDS’ illnesses?

If animal experiments could be shown to have any scientific, medical or biochemical relevance to the war on ‘AIDS’ I would have no objection to the use of animals to help save the lives of humans. How many activists have had no scruples about using a promising vaccine manufactured at the cost of an animal’s life? Fetal calf serum and horse serum are used in cell culture substrates for vaccine production. Polio and all the other common vaccines are made by sacrificing monkeys and other animals to obtain their kidneys and other organs for cell cultures; indeed it is certain that either animal cells obtained in this way, or pre-cancerous human ones, will be used to conjure the ‘AIDS’ vaccine they so misguidedly seek. Would anti-vivisectionists take this vaccine only if it can be guaranteed not to have been made using animal tissues obtained by vivisection?

While the presumed role of ‘SIV’ in infected monkeys is often used as proof of the ‘HIV/AIDS’ hypothesis, studies have shown that ‘SIV’ failed to cause disease in rhesus and mangaby monkeys. Tatchell rightly points out: “...vivisectionists have found it impossible to inject animals with HIV and induce AIDS...Conversely, ‘SIV’ does not produce AIDS in people”. ‘SIV’ has never caused any disease in wild monkeys, as ‘HIV’ has never caused any disease in wild homosexuals. Dogs that tested ‘HIV antibody positive’ are living testimony to the non-

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due to the shortage of human organs for transplantation and recent advances in surgical techniques, cross-species transplantation is becoming more widespread. Kidneys successfully transplanted from pigs into monkeys have brought the prospect of successfully transplanting a pig organ into a human a stage closer. Recently researchers have genetically engineered a herd of pigs so their organs would not be recognised as foreign when transplanted into primates. Anti-vivisectionists have rightly argued the risks involved in this xenotransplantation. Known pathogenic viruses that might pose a risk in xenotransplantation include many adenoviruses, papovaviruses, hepadnaviruses, morbilliviruses, and togaviruses. Whilst known viruses can be screened out, unknown varieties cannot. One has only to remember the panic caused by the contamination of hundreds of thousands of batches of Salk, Sabin, and Koprowsky polio vaccines in the '50s and early '60s with Simian Virus 40 (SV 40) to appreciate the ease with which hitherto unknown viruses can contaminate vaccines produced using animal cell cultures.

Many species carry multiple herpes viruses. It has been posited that xenotransplantation may cause the ‘epidemic’ spread of ‘new’ viruses. Anti-AIDS activists Act Up San Francisco condemned the introduction of baboon bone marrow cells into Jeff Getty; such experiments could transfer baboon viruses into the human population. Regarding ethics and rights, it can be argued that some animals enjoy more luxuries than humans when engaging in experimental medical trials. According to Prof. Suzanne Ildstad of the University of Pittsburgh Medical Centre: “Our baboons are treated better than some people in third world countries are. They have colour TVs and a social time. I think if animals can be treated ethically and are available for transplantation, then the potential benefit to humankind is significant.”

Xenotransplantation, as a new experimental medical procedure, involves risks that become assessed and manageable only with experience - as Dr David White, Director of Research, Imutran, argues: “It’s a fine judgement between advancing a technology that is going to save many people’s lives and putting at unreasonable risk the first few individuals who inevitably we will have to start with when we go into the clinic.” Demand for organ transplants has escalated with an estimated 40,000 persons in the USA currently awaiting transplants. With such an urgent global demand for organ transplants, xenotransplantation may become standard therapy in the near future. A pig’s kidney should be available for transplant into a human within three years. But who would refuse to take a pig’s organ if his/her life was on the line? With up to 50 people around the world dying everyday waiting for transplants, xenotransplantation is regarded by many as a risk worth taking. Dr Stephen Gundry of Loma Linda Hospital, California, contends: “Xenotransplantation is in fact nature’s way of one animal helping another. Animal to human transplants are inevitable. I think it is going to work as well, or perhaps better, than what we have in human to human transplant right now.”

Peter Duesberg has proposed to do nitrite inhalant studies on animals at the high dosage used by inhalant-users: “Exposure 100 mice, or cats, or monkeys to nitrite inhalants at doses comparable with human recreational use... I would predict this result: immunodeficiency, pneumonia, and pulmonary KS in animals.” These animals would be inhaling nitrites via test tubes to the nostrils mimicking human doses, although not engaging in the erotic homosex repertoire. Activists who designate mice as “these thinking, feeling, creatures... in the pursuit of happiness” should not object to nitrite studies being done with mice since they should get as much of a hard-on and a bang out of nitrites as humans do. The effects of nitrites on the human immune system using 23 volunteers were studied by E M Dax and colleagues, though Dax’s study was flawed due to the lack of a control group and low dosages of nitrites used. Since OutRage! see animal studies as ethically incorrect, maybe they could volunteer their members to take part in Duesberg’s proposed nitrite inhalant study, at least because drug trials on animals can lead to false conclusions about safety.

Had Penicillin been fully tested on rabbits prior to its release for humans, it would never have been released for human consumption and the subsequent history of the use and abuse of antibiotics might be very different today. Conversely, poorly trialled Thalidomide caused birth deformities not only in humans, but, as was only subsequently shown, in newborn mice. It is significant that AZT was cleared for human consumption months before the required animal studies were completed. AZT has killed tens of thousands of people although it was already known to cause ovarian cancer in rats. At present there is no animal model for testing protease inhibitors; however, out of fear and emotional coercion many humans have been conned into taking these untested drugs. ‘HIV’ organisations like NAM, NAT, THT and Project Inform must be held to account for human rights violations and corporate manslaughter for collaborating in direct medical killing (iatrogenic ‘AIDS’). Queer lives are now considered cheaper and more expendable than those of rodents according to Marina Cronin: “There are alternatives to the cruelties and inaccuracies of animal experimentation... human volunteers for drug trials.” Does she realise that recently a high-ranking public health official proposed using Michigan prisoners as guinea pigs for ‘anti-viral’ drug experimentation? While thousands of queers continue to die from iatrogenic-AIDS, OutRage! seem content to play charades with such niceties as saving rats. OutRage! must start saving queers’ lives from being slaughtered by ‘Murder Inc.‘: Glaxo-Wellcome, Bristol-Myers Squibb, and Hoffmann-La Roche.

References
3. BBC Panorama, July 1996.
4. ibid.
5. JAMA, 276:8, 28.8.1996.
You have said that in authoritarian conditions “most people internalise the values and then regard themselves as acting more or less freely.” HIV=AIDS=Death for many people generates values that are deeply held. Why might people be driven to choose profound limits to their freedom of actions?

NC: Under all conditions, people tend to internalise values and see themselves as acting freely. I’m not sure this is more true under more authoritarian conditions. Thus in a brutal state, people may adopt the values (perhaps out of fear) but not internalise them, whereas in a more democratic society, where forces are more hidden, there may be a tendency to internalise values without much awareness. To say that people have internalised “HIV=AIDS=Death” seems to me an overstatement, though perhaps some have. I suspect an investigation would show that many people have accepted the weaker assumption that HIV is likely to lead to AIDS which in turn is likely to lead to suffering and premature death. As to whether that assumption is correct, that is a different question. Rational people will look at the evidence and arguments, and decide accordingly. I am not convinced that people have some kind of “drive” that leads them “to choose profound limits to their freedom of action”.

Q: In Britain we have continuously been told there’s an AIDS epidemic, while on average less than 650 people per year have died with such a diagnosis. Do you think language is becoming less meaningful as society becomes increasingly sloganistic?

NC: I don’t know whether use of language is more or less “sloganistic” when hundreds of billions of dollars are spent every year to “control the public mind” (to borrow some terms of the public relations industry) or when people parrot rhetoric of organised religion, to select one of many examples. On referring to the spread of AIDS as an “epidemic”, the term “epidemic” is used to suggest that the problem is serious and should be a matter of grave concern. With that I agree.

Q: Technically of course “epidemic” need not refer either to infection or disease – it’s a phenomenon affecting people, unexpected in prevalence e.g. even an epidemic of lawlessness. According to a report in the Wall Street Journal the CDC is redressing a self-confessed public relations excess geared towards keeping the heterosexual population anxious and therefore vigilant about HIV/AIDS. The cumulative total for “HIV positivity” in the US was revised downwards in 1995 from some 1,000,000 cases to between 500,000 and 700,000. Do you believe in consistent full open accountability of government agencies?

NC: Of course, I believe in open accountability of government and other power systems, such as private corporations. In the former case it exists to a considerable extent, and citizen pressure has widened the boundaries, and should continue to do so. In the latter case, it barely exists at all. Whether the Wall Street Journal article you cite is accurate one has to evaluate in the usual manner: by investigation. If it is accurate, one then has to assess to what extent it is reasonable to use the term “epidemic” to suggest that the problem should be of grave concern.

Hundreds of billions of dollars are spent every year to “control the public mind”
Q: In 1986 the International Committee for the Taxonomy of Viruses formalised the name Human Immunodeficiency Virus for a collection of indirect molecular-biological markers which could be linked with at least transitory deficiency of the cellular immune system. Do names and language cast their own spell?
NC: Linguistic expressions carry all sorts of connotations. In technical usage one tries to divest them of such associations. The question that seems to be lurking here is a different one: namely, is the technical term that has been selected an appropriate one on scientific grounds? Maybe yes, maybe no, but that does not seem related to “the spell of language.”
Q: “Is the technical term... [HIV]...appropriate on scientific grounds?” – really, assuming that the Group for the Scientific Reappraisal of AIDS, numbering 500 official members including two Nobel laureates, aren’t all balmly, and given that plenty of examples are available of a far wider range of scientific skepticism sympathetic to the group over whether the proposition of a viral cause for AIDS is tenable – not least because of the enduring questions over whether the theory of the existence of retroviruses deserves to be held! – may we not argue that the 1986 adoption of the finite term HIV by the ICTV was a fascistic imposition?
NC: I think we agree that’s the core of the matter: To what extent is it reasonable to assume a viral cause for AIDS? To answer that question we have to investigate the facts, no trivial matter. The facts that 500 people including two Nobel laureates rejected the assumption is perhaps enough evidence to make one want to isolate. Does the media generally not articulate the majority. Do you think it’s to be challenged?
NC: I presume polls would show that most people would approve of “safe sex” rather than “unsafe sex”, and that inquiry would show that many choose the latter nonetheless. But I’m probably missing the point.
Q: Really? Current ideology is that sex can be unsafe because “the AIDS virus” can be transmitted that way. Despite many scientists doubting this, it is a felony or misdemeanour in 29 US states to engage in “risky” sexual behaviour if a person is “aware
The facts of the matter have to be investigated
he/she’s HIV positive”. Professor of law Philip Johnson of UC Berkeley has said of these laws, “Of course they’re irrational laws; they occur in the context of irrational fear.” Can one reasonably detect similarities between these legislations and their moral incitements and, for example, religiously motivated legislation in the issues of abortion, or homosexuality or racial equality?

NC: If you think the evidence is unpersuasive that “the AIDS virus” can be transmitted sexually, then by all means try to establish your conclusion and convince people of it. Citing a professor of law at Berkeley isn’t helpful. Laws are generally “irrational” in the sense of his comment. It doesn’t follow that all laws should be thrown out, just as the skepticism of some scientists doesn’t mean that all of science should be placed in the category of Papal Bulls. Again, one has to investigate case by case.

Q: Throughout human history, theorists and intellectuals like you have challenged official truths about science and human nature, putting their lives and careers at risk, often silenced or ridiculed. Can you describe some general principles of successful dissident organisation and struggle, beyond the obvious ones. About those, I have no more to say than the participants in popular organisations in the slums of Haiti, to go to the opposite extreme of privilege in the hemisphere where I live – and where quite impressive developments have taken place, of a kind that I’ve seen many times under conditions of extreme duress, around the world, and in sectors of privilege in the rich countries as well. There are no magic tricks. When we believe it is our duty to challenge orthodoxy, we don’t ask someone how to do it: we’ll get no useful answers. Rather, we do it. This isn’t quantum physics. It’s mostly a matter of using common sense. Whatever the issue, what is needed is not specialised knowledge or great insights, which are lacking in any event, but rather energy, dedication, courage, honesty - the simple virtues. We should also be aware of our extraordinary privilege, which offers us opportunities that are not available to the great mass of poor and struggling people throughout the world – opportunities to inquire, understand, and act.

As for challenging “preconceived ideas”, that is always appropriate.

Q: If public health systems aren’t working in the interests of the public, how might people respond to the possibility that their health is being compromised by economic forces?

NC: I think that honest people should seek to understand how the public health system works, and work to make it as responsive as possible to the informed decisions of the public – but that seems close totruism. How should this be done? By the usual mechanisms of popular organising and education, available to a considerable extent in relatively free societies like ours, with little personal risk or cost.

Q: HIV/AIDS self-help groups which started as dissidents and later founded the voluntary sector have provided information and support to change preconceived ideas about homosexuality and AIDS. Today, these organisations have become institutionalised and also promote state-sponsored drug-therapies. Do you think that in order to push for change Continuum must make compromises with the establishment?

NC: What tactics Continuum should follow, I can’t say. I don’t mean to suggest that tactical decisions are insignificant; on the contrary, they regularly have direct and often substantial human consequences, and therefore require careful thought and attention. But choice of tactics depends on goals and an assessment of the circumstances. As for challenging “preconceived ideas”, that is always appropriate, whether the ideas are about homosexuality or AIDS or anything else. A reasonable person will not easily adopt “preconceived ideas” on matters of any significance, but will try to find what seem to be the best ideas. As to whether standard ideas are the best ones, they usually are not, but that is a matter that requires specific inquiry.

Q: Do you feel the language of war and conflict translates successfully to the human body’s functions? “Defences compromised”, “titanic struggle” against ‘invaders’, ‘zapping’ microbes, etc. – where does the popular language of our biological identities derive from?

NC: I don’t see any particular problem in referring to “the body’s defences against disease” etc. The other terms you mention are picturesque, presumably used to attract attention. In a paper in a technical journal they would be out of place. In a public discouerce I don’t happen to like them much, but it’s a matter of taste.

Q: HIV/AIDS “dissident” concerns were first discussed 10 years ago at an international conference. The tenth such conference is due to be held next year. What are the best political purposes of conferences?

NC: The “political purposes” of any activity, including conferences, are (I suppose) to clarify our understanding, sharpen our agendas, and place them in the public arena as prominently as we think important in the particular case.

Questions by Huw Christie, Rafael Ramos and Michael U. Baumgarner

Titles by Noam Chomsky include:
Problems with Knowledge and Freedom (The Russell Lectures)
Language and Responsibility
For Reasons of State (in UK, from Serpents Tail, London)
Necessary Illusions – Thought Control in Democratic Societies (in UK, from Pluto Press, London)
Manufacturing Consent, The Political Economy of the Mass Media
Deterring Democracy (Vintage Press)
Origin of the Specious

NEVILLE HODGKINSON summarises the historic scientific paper which dispenses the illusion of HIV and forms the special supplement of this issue

Publication in this issue of Continuum of the article by Eleni Eleopulos and colleagues is an historic event. Their tremendous work refutes the idea that a new virus, “HIV”, which came to be known as the cause of AIDS by the scientific community and the world, has ever been shown to exist.

This is not idle word play. It is devastatingly important to people who have been told they are likely to die because of their infection with “HIV”; to people taking demonstrably dangerous and potentially lethal drugs in the hope of slowing the progress of this non-existent virus; to the doctors prescribing those drugs, and the medico-legal organisations who represent them; to politicians everywhere, but especially in poorer countries, who have switched scarce resources into fighting a mythical epidemic of “HIV disease”; to communities genuinely stricken by AIDS, who need to look again at the real causes of the syndrome; and to the world of science, which will need to address the question of how it can re-establish credibility with the public after not just allowing such a terrible mistake to occur, but actively obstructing efforts to question the “HIV” hypothesis.

Eleopulos’s disposal of the HIV illusion takes us into the deep waters of modern molecular biological techniques, where even the pioneers are in uncertain territory, though they all too rarely admit as much. But the paper, with its 251 references, is a definitive demonstration that not a single scientist has proven the existence of “HIV” as a unique and distinguishable molecular entity. It is the fruit of years of work. The authors are a group of scientists well-respected by their immediate colleagues, and with a history of publication in leading peer-reviewed journals, though on the “HIV” issue they have faced much obstruction, over many years, in seeking to make their views known to the scientific community. That community now has a clear obligation to respond to this paper, to grapple with its implications and answer its challenge.

The main isolation technique has been to spin the biological material to be examined through a density-graded centrifuge (the further the materials travel down the test-tube, the greater the density). This is a key step in separating and isolating any particles present, including viruses, since it causes them to band at characteristic densities. The particles can then be examined microscopically and biochemically, and used in biological tests to see whether or not they are infectious.

In the early 1980s, when AIDS was first recognised, scientists theorised that a new infectious agent might be responsible, and began a race to identify it. A front-runner theory was that it could be a retrovirus – a theoretical member of a family of microbes in which a special enzyme, called reverse transcriptase, allowed the genetic material of the virus to become integrated with the DNA of its host cell. Peter Duesberg, a world leader in the field, had won renown for linking an animal retrovirus with cancer. He later insisted however that the risk was brought about by a mutant human gene picked up by the retrovirus, and that essentially human retroviruses were benign, naturally present packages of
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leopulos demonstrates that such isolation has never been performed with "HIV". When cells taken from AIDS patients and AIDS risk groups are put through a variety of laboratory processes and then the fluid in which they have been grown is centrifuged, a lot of material, including gene segments of varying lengths, falls within the density band characteristic of retroviruses. Crucially, however, "HIV" researchers have never been able to find pure particles at that density with an electron microscope. Very occasionally, a stretch of genetic material with a length characteristic of retroviruses is found amidst all the other material, and fished out for analysis. These long stretches have formed the basis for claims about HIV's genetic structure. However, in 10 years of research, only 19 “full-length HIV” genomes of this kind have been sequenced, and no-one has ever established whether or not these too are an endogenous product of normal cells, or a genuine infectious entity. Their extreme rarity is strong evidence against the latter. Furthermore, not one such genome has ever been sequenced from cells taken freshly from patients; they have only been found in material subjected to drastic manipulation in the laboratory, mostly involving being cultured in cancerous cell lines. These are exactly the circumstances in which naturally present, endogenous mechanisms of genetic activation are most likely to take place.

Most analyses of so-called “HIV” genetic material are based on small segments of the purported virus genome, typically covering between 2% and 30% of it, since the longer sequences are so rarely found. There is not even any fixed pattern to the composition of these segments – they vary by 40% or more. No two identical “HIV”’s have been found, even from the same individual. In other words, there is no evidence for the presence of any unique molecular entity like a virus.

Peter Duesberg, in support of his retrovirology colleagues’ claims that “HIV” had been isolated, points to studies in which “full-length” genetic sequences have been taken from the soup of mutagenized lab cells described above, then passed into bacteria, and reproduced as pure copies. He claims that this means isolation “by the most rigorous method science has to offer”. Logic seems to have deserted him on this issue, however. The point is not to isolate a genetic sequence, which can in any case be done by picking it out of the banded materials, but to isolate virus, which means to obtain virus particles separated from everything else and show that they have the characteristics of an infectious, replicating, disease-inducing microbe.

Reproducing a stretch of genetic material through molecular cloning techniques does not mean you have an infectious microbe.

Duesberg further argues that because this cloned material reacts with antibodies used in the “HIV” test, that confirms the existence of “a unique retrovirus”. Eleopulos, however, has long ago demonstrated that the antibodies themselves are non-specific – that is, although they are certainly found much more often in AIDS patients and people at risk of AIDS than in healthy people, people can test positive as a result of activation of the immune system by a variety of microbial and toxic influences, including auto-immune processes.

So often, the correlation between the presence of “HIV” antibodies in a patient with AIDS itself has been taken as evidence for a viral cause of the syndrome, but this idea is torpedoed by a 1996 study cited by Eleopulos. Reinhard Kurth, a world leader in retrovirology at the Paul-Ehrlich Institute in Germany, showed that 70% of “HIV-positive” patients, compared with only 3% of blood donors, had antibodies which reacted with a molecular entity called HTDV/HERV-K. This “retrovirus”, however, is not only present in AIDS patients, but “in all of us”, as Kurth argues; – it is one of those endogenous retroviral-like gene segments referred to above. The fact that such segments are activated in AIDS patients, perhaps with concomitant appearance of antibodies, does not make them the cause of AIDS, nor does it mean the presence of an infectious virus.

In the late 1980s, to try to rescue the concept of an “HIV genome”, researchers began using the polymerase chain reaction (PCR) to look for “HIV” sequences. This immensely sensitive technique can find the genetic equivalent of a needle in a haystack, and multiply it into a stack of needles. On the basis of such methods, scientists such as New York’s David “It’s the virus, stupid” Ho (he once wore a badge proclaiming his faith to this effect) have developed theories that there is actually a mass of “virus” destroying AIDS patients’ T-cells, even though it cannot be detected by standard techniques. The mathematics through which he reached this conclusion have been demonstrated to be faulty, but even if that were not the case, PCR does not involve isolation, or even detection of “HIV”. It simply measures the presence of certain genetic segments, which have been postulated to belong to “HIV” but never demonstrated as such, because “HIV” itself has never been isolated.

Peter Duesberg argues that “HIV” is confirmed as a unique entity by the detection of HIV-specific genetic segments in most antibody-positive people, but not in “uninfected” humans. Eleopulos demonstrates that he has overstated the evidence from one study on this point, and failed to quote others that show less of a correlation. But in any case, the genetic probes for these segments have not had their specificity tested against the only meaningful measure, isolation of the virus whose segments they are supposed to be detecting. Failure to see this is one more example of the circular reasoning that has bedevilled “HIV” research from the start.

When in 1983 Luc Montaigner and his group first described the procedures and observations that made them believe they might have cultured an AIDS virus, Gallo did not believe them. Nor did Nature, which turned their paper down. Nor did the British virus expert Robin Weiss, who in a 1986 patent application referred to Montaigner’s “HIV strain” as a “so-called AIDS virus isolate”. But Gallo’s group did no better, other than to use leukaemic cells to produce a range of proteins that came to be known as characteristic of HIV, without proof that they were coded by “HIV” genes, or that they belonged to a retrovirus-like particle. Nevertheless, these described above, were used by Gallo and his group as the basis for numerous research papers in which they purported to demonstrate infection with a deadly virus, an unvalidated test that has brought unnecessary misery to millions.

There was no intent to mislead, but judgment became warped in what Gallo has called the “passionate” phase of the race to find “the virus” assumed by these virus experts to be causing AIDS. Although Gallo is now known to have twisted the facts in support of his claim to have come first in that race, he and Montaigner and Weiss believed in what they were doing. Fame and fortune were to follow, but they also hoped humanity would benefit from their findings. Unfortunately that has not been the case, and it is now time to end this tragic episode in the history of science.
The definite existence of any virus, including a retrovirus, can be proven only by isolating it. For nearly half a century retroviruses have been isolated by banding in density gradients. It is accepted that the procedures incorporated into this method, which is by no means perfect, have not been followed by the researchers who claim isolation of the human immunodeficiency virus, HIV-1. Nonetheless, it is said that at present, there is ample evidence that HIV has been isolated and shown to be a unique exogenous retrovirus.1

In this critique we have analysed the relevant data that purport to prove that HIV has been isolated. To simplify the presentation for readers of this article, the major arguments for HIV isolation (as presented by Peter Duesberg in Vol 4, No 2 of Continuum1) are used as the headings in the discussion. Since the topic is both complex and controversial it is necessary to present substantial original data and sometimes to repeat it in order to critically assess the basis for the view that HIV has been isolated.

1. “In 1983 Montagnier et al isolated a retrovirus”.

In the 1983 Montagnier et al study there is no proof of virus isolation by “the most rigorous method available to date”. Nor did they follow the “traditional.. Pasteur rules”. How then did they isolate a retrovirus? Even if Montagnier and his colleagues or others had followed the “Pasteur rules”, since “viral and cellular proteins, and cellular contaminants... copurify with virus purified by conventional density gradients”,1 there is no reason to accept any claim of HIV isolation by any research group who did not use “the most rigorous method available to date, i.e. molecular cloning of infectious HIV DNA”. However, to prove that HIV “has been isolated” by “the most rigorous method available to date”, virus cloning, one must start with HIV RNA (DNA). Since the propriety of naming an RNA “HIV RNA” is contingent upon prior isolation of a particle proven to be a retrovirus, on this basis alone, “the most rigorous method available to date, i.e. molecular cloning of infectious HIV DNA”, cannot prove HIV isolation.

2. “reverse transcriptase associated with such particles”.

There is not one single study which proves that the enzyme present in the “growth medium” or even in the material which in sucrose density gradients bands at 1.16 gm/ml, (the density which defines retroviral particles), and which catalyses the transcription of RNA into DNA, is a constituent of particles of any kind, much less of retroviral-like particles or a unique retrovirus. The only association between “particles” and “reverse transcriptase” (RT) arises from experiments which show that some cultures/cocultures with tissues from AIDS patients exhibit both particles, many of which are not even retroviral-like, and transcription of the synthetic RNA template-primer A(n).dT15. However, this does not constitute proof of the existence of RT or RT as a constituent of a retroviral particle. Furthermore, since:

(a) the presence of reverse transcriptase (RT) is proven indirectly, that is, by demonstrating transcription of the RNA template-primer A(n).dT15;  
(b) the template-primer A(n).dT15 can be transcribed not only by RT but by other cellular DNA polymerases. All the cellular DNA polymerases, a, b and g, can copy A(n).dT15. In fact, in 1975, an International Conference on Eukaryotic DNA polymerases, which included Baltimore and Gallo1 defined DNA polymerase g, “a component of normal cells”, “found to be widespread in occurrence”; whose activity can be increased by many factors including PHA stimulation, as the enzyme which “copies A(n).dT15 with high efficiency but does not copy DNA well”; it is impossible to say whether the polymerase in the “growth medium” or in the material banding at 1.16 gm/ml which catalyses reverse transcription of A(n).dT15 is RT or one of a number of other cellular DNA polymerases.

3. “…indeed, each of these criteria could reflect another retrovirus, and some of these criteria, eg, particles and proteins, could reflect non-viral material altogether”.

Although the HIV/AIDS experts, including Montagnier, Gallo and Barre-Sinoussi claim that RT is “unique to retroviruses” and “the hallmark of a retrovirus”4,5 this is not the case, a fact accepted by some of the best known scientists.6 “Reverse transcriptase (RT) was first discovered as an essential catalyst in the biological cycle of retro-
viruses. However, in the past years, evidence has accumulated showing that RTs are involved in a surprisingly large number of RNA-mediated transcriptional events that include both viral and nonviral genetic elements. . . the possibility that reverse transcription first took place in the early Archean is supported by a number of facts and the hypothesis that RNA preceded DNA as cellular genetic material. . . According to Varmus, Reverse transcription was assigned a central role in the replication of other viruses [hepatitis B and cauliflower mosaic viruses] and in the transposition and generation of other kinds of eukaryotic DNA. . . The hepatitis B viruses (HBVs) are small DNA viruses that produce persistent hepatic infections in a variety of animal hosts and replicate their DNA genomes via reverse transcription of their RNA intermediates, and from the RNA/ DNA hybrid viruses (ORFs, P) for pol, which is homologous to retroviral pol genes. . . Hepatitis B virus (HBV) resembles retroviruses, including HIV, in several respects. In particular, both viruses contain reverse transcriptase, and replicate through an RNA intermediate. Because of this, it has been suggested that hepatitis B infection should be treated with the same antiretroviral agents as HIV infection. . . At present, evidence exists which shows that although the major target organ for hepatitis B virus is the liver, cells other than hepatocytes—including peripheral blood lymphocytes and monocytes, may become infected with HBV. . . Lymphocyte stimulation in general and PHA stimulation in particular is associated with production of hepatitis B virus (HBV) peripheral blood lymphocytes in patients infected with HBV including "viral replication in chronic hepatitis B". . . The proteins from the culture supernatant which banded at 1.16 g/ml were also reacted with the sera but instead of the goat anti-p25 antiserum they used sera from another healthy donor. In the published strips it is difficult if not impossible to distinguish any reactive bands with any serum. In the text it is stated "three major proteins of umbilical cord lymphocytes were identified. "A(n).dT15. The finding of the same proteins in cultures of human umbilical cord blood, bone marrow or peripheral blood T lymphocytes, cultured with supernatants from the co-cultures, were extensively studied because retroviruses were said to be transmitted vertically (in the germ cell line) and because they were thought to play a significant role in differentiation. By the beginning of the AIDS era one or more of the following phenomena were reported from experiments with such cells: retrovirus-like particles, reverse 

4. "HIV antigens or proteins associated with such particles". To date nobody has presented evidence that the "HIV antigens or proteins" are constituents of retrovirus particle or even a retrovirus-like particle let alone a unique retrovirus, HIV.

5. "Antibodies against Montagnier's HIV strain - the global standard of all "HIV tests". In 1983 the paper entitled "Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS)". . . Montagnier and his colleagues reported the "isolation" of their "HIV" strain, cells from a lymph node biopsy of a gay man with lymphadenopathy (lymphadenopathy syndrome [LAS]) were put in culture and anti-serum to human interferon. (The latter had previously been shown in mice to lead to "increased retrovirus production by a factor of 10 to 50"). After 15 days RT activity was detected using the synthetic primer-template A(n).dT15. The reverse transcription of A(n).dT15 was considered proof that a retrovirus was present in the lymph node cells. The finding of the same activity in supernatants of a co-culture of the same cells with lymphocytes from a healthy individual was considered proof that the retrovirus could be transmitted. In another experiment, polybrene and the virus was isolated and how was it possible, before the virus was isolated, how was it possible to obtain rabbit antiserum "to HTLV-III" before Montagnier et al. Obviously, at the very best, the finding of phenomena such as virus-like particles in cell cultures, antibodies/antigen reactions and evidence for reverse transcription of A(n).dT15 can be considered proof only for detection of a retrovirus and then if and only if each are shown to be specific to the retrovirus. This cannot be done unless the retrovirus is isolated. Thus it comes as no surprise that Popovic, Gallo and their colleagues did not consider Montagnier et al.'s data as proof of "true isolation". In their 1984 papers Gallo and his colleagues defined isolation as detection of "more than one of the following": repeated detection of a Mg2+- dependent reverse transcriptase activity in supernatant fluids; virus observed by electron microscopy (EM); intracellular expression of viral related antigens detected with antibodies from seropositive donors or with rabbit antiserum to HTLV-III; or transmission of particles. (By transmission of particles was meant detection of RT or particles in cultures of human umbilical cord blood, bone marrow or peripheral blood T lymphocytes, cultured with supernatants from the "infected" cultures). Since this is no different from the experiments that Montagnier and his colleagues performed, it follows that Gallo and his colleagues did not prove "true isolation" either. In fact, Gallo et al.'s definition of isolation raises additional questions including: How was it possible to obtain rabbit antiserum to HTLV-III before the virus was isolated and how was it possible, before the virus was isolated, to ascertain that both the rabbit antiserum and the patient sera was subjected to test material from the cultures infected specifically with the virus? According to their definition, one can isolate HIV even if no RT is detected. How is this possible since RT is the "hallmark" of HIV? It is also significant that in his and his colleagues' 1986 patent application "Improvements relating to viral isolates and their use", Robin Weiss referred to Montagnier's "HIV strain" as "the material". A so-called Aids virus isolate was first reported in 1983 by Montagnier and his colleagues in France who named the material "Lymphadenopathy Associated Virus One". . . Furthermore, isolation of a retrovirus from the umbilical cord cultures is not proof that the retrovirus was introduced from the outside, that is, that it originated if no RT is detected. How is this possible since RT is the "hallmark" of HIV?"
transcriptase activity and retroviral antigens.\textsuperscript{24,26} Thus such findings cannot be proof for the existence of HIV.

Neither is the presence of antibodies in the AIDS patients, but not in the healthy controls, which react with the proteins which band at 1.16 gm/ml, proof that such individuals are infected with an exogenous retrovirus, HIV. For example, in a study published this year, one of the best known virologists, Dr. Robert Gallo, of the National Cancer Institute, reported the isolation of a retrovirus, without proof that they are coded by "HIV DNA", or that they belong to a retrovirus-like particle, the following proteins, gp160/150, gp 120, gp45/40, p34/p2, p14/p7, gp24. A large protein with a molecular weight of approximately 130,000 and a protein of 48,000 were also detected. Unlike Montagnier, Gallo's group also reported that, "With normal human serum, none of the antigens was detected (not shown)", and concluded, "These results show clearly that the anti-gens detected after virus infection are either virus-coded proteins or cellular antigens induced by the virus". Some of Gallo's colleagues also reported that of the proteins from the supernatant of the "infected" cultures which in sucrose density gradients banded at 1.16 gm/ml, only two proteins, p41 and p24, reacted with patient sera and concluded that "these molecules are the major components of the virus preparation. p24 and p41 may therefore be considered the viral structural proteins."

In the two years following their discovery of HIV, although Montagnier's group apparently made repeated attempts, unlike Gallo's group, they could not detect a "high molecular weight" protein which reacted with different sera but which was not present in the supernatant of uninfected control cells. In experiments reported in 1985, instead of using umbilical cord lymphocytes, they used "infected" CEM and H9 cells and cultured (labelled) them with radioactive cysteine, \textsuperscript{35}S cysteine, (an essential amino acid constituent of human proteins). They reported that in the supernatant a "protein of approximately 110-120K" could be specifically immunoprecipitated by sera from pre-AIDS or AIDS patients, in contrast to 54,000 daltons by calculation.\textsuperscript{33} Even if the molecular weight of the transmembrane part of the envelope protein complex, shows more than 80% conserved amino acids", but “gp41 should be about 52,000 amino acids long which may be glycosylated. The size of these predicted products agrees with the detection of a large glycosylated protein of M, 120-160K in HTLV-III-infected cells which is probably the glycosylated env gene precursor and a smaller, virion-associated gp41 which is probably the membrane protein."\textsuperscript{23} However, in a study published in 1987 by Gallo and his colleagues, where they performed a "Computer-assisted analysis" of "the amino acid sequences of the envelope protein complexes derived from the nucleotide sequences of seven AIDS virus isolates", it was reported that, "Although the overall sizes and structures of the seven surface proteins are rather similar, the deduced amino acid sequences differ in length and composition. They may reflect that even the amino acids are conserved in the exterior part of the protein...gp41, the transmembrane part of the envelope protein complex, shows more than 80% conserved amino acids", but “gp41 should be about 52,000 to 54,000 daltons by calculation.\textsuperscript{33} Even if the molecular weight of the glycoprotein predicted from the length of the "HIV" fourth ORF was found in the identical position of all the proteins in the Western blot (41,000), the claim by Gallo that the interaction of gp41 with antibodies found in AIDS patient sera is proof that gp41 is coded by the "HIV genome", and that both gp41 and the antibodies are specific to a retrovirus, is at odds with what Gallo was saying in 1981.

In the mid 1970s, Gallo and his colleagues reported the isolation of the first human retrovirus, \textsuperscript{23}HL23V, in further evidence for the "isolation" of \textsuperscript{25}HL23V surpassed that of HTLV-I and HIV in at least two aspects. Unlike HIV, Gallo's group:

(a) The culture supernatant and the cells cannot be considered synonymous with a retrovirus.

(b) Although Montagnier et al did not comment, their data shows that many proteins, including a p40 found in the supernatant of both "non-infected" CEM and H9 cells react with sera from the patients with lymphadenopathy. The proteins, without proof that they are coded by "HIV DNA", or that they belong to a retrovirus-like particle, the following proteins, gp160/150, gp 120, gp45/40, p34/p2, p14/p7 found either in cells, supernatants, or banding at 1.16 gm/ml in sucrose density gradients became known as the HIV proteins. In other words, contrary to all scientific reasoning, it was postulated that AIDS sera contain specific HIV antigens, and the proteins with which these antibodies reacted were defined HIV specific proteins.

5.4 The “HIV glycoproteins”, gp160, gp120 and gp41. (a) In 1983,\textsuperscript{20} and again in 1984 Montagnier and his colleagues\textsuperscript{29} claimed that although p45/41 reacted with patient sera, this protein was absent but the similar protein, gp41, was present. To demonstrate this, instead of using the CEM or the H9 cell line, they infected CEM or H9 cells with the virus preparation. p24 and p41 may therefore be considered the viral structural proteins.

In 1985, Gallo and his colleagues, comparing the fourth open reading frame (ORF) of the "HIV DNA", which they called env-lor with the env genes of other retroviruses, reported, "The predicted product of the env-lor open reading frame (ORF) shows a striking of which is a hydrophobic region near the middle of the protein...The amino-terminal domain of the translation product of the fourth open reading frame also resembles the env protein precursors of other retroviruses...we believe that the fourth open reading frame encodes the envelope precursor...in its mature form...in contrast to the envelope gene precursors of other retroviruses, the most striking of which is a hydrophobic region near the middle of the protein..."
munoprecipitation assays with proteins of mammalian type-C viruses including the internal (gag) and envelope (env) proteins of HIV, SSAV and BEV and concluded, “The serological studies presented here and by others provide indirect evidence that the infectious mode of transmission remains a real possibility in humans, and suggests that infection with an oncornavirus (retrovirus) may be extremely widespread and not limited to homo- and heterosexual transmission”.

In 1987 Henderson et al isolated the p30-32 and p34-36 of “HIV purified from non-banded material”. In 1981 Gallo accepted the evidence that the antibodies which reacted with proteins of HIV were directed not at the “HIV proteins” but against the carbohydrate moieties on the molecule that are introduced by the host cell as a post-transcriptional event, and which are therefore cell-specific and not virus-specific. This discovery was of such significance that today nobody, not even Gallo, considers HIV as being the first human retrovirus, or even a retrovirus. In fact, in 1981 when Gallo and his colleagues reported the presence of antibodies to a 30kD protein of human T-cell lymphoma virus. Gallo, considers HL23V as being the first human retrovirus, or even a retrovirus. In 1981 when Gallo and his colleagues reported the presence of antibodies to a 30kD protein of human T-cell lymphoma virus. In this paper Gallo and his colleagues demonstrated that the antibodies were “specifically directed at HTLVCR proteins and not at cell-specific determinants – in other words, the immunological reactions are not those reported in human sera against animal virus glycoproteins which, lacking virus specificity, are directed against the carbohydrate residues of the glycoprotein”.

(b) By 1989, researchers from New York showed that in Western blot analyses, “the components visualized in the 120-160 kDa region do not correspond to gp120 or its precursor but rather represent oligomers of gp41”. It was also shown that the WB pattern obtained is dependent on many factors including temperature and the concentration of sodium dodecyl sulphate used to disrupt the “pure virus”. “Confusion over the identification of these bands has resulted in incorrect conclusions in experimental studies. Similarly, some clinical specimens may have been identified erroneously as seropositive, on the assumption that these bands reflected specific reactivity against two distinct viral components and fulfilled a criterion for true or probable infection. It was later identified that the standards to be established for Western blot positivity: it may necessitate the reinterpretation of published results.”

5.6 The “HIV gag protein”, p24

As far as Montagnier is concerned, p24 is THE HIV protein, and for at least three years after the introduction of the “HIV” antibody test, a p24 band found in the WB was considered by most laboratories, including the CDC, as proof for HIV infection. At present there is ample evidence that antibodies which react with p24 are common in both uninfected and infected individuals. The development of many new monoclonal antibodies indicates that either p24, the antibodies, or both, are non-HIV-specific or a significant proportion of both humans and animals are infected with HIV. For example, if the p24 band in the WB is considered proof of HIV infection then about 30% of individuals who are transfused with HIV negative blood become infected as a result. Since, according to the AIDS Monitoring Committee (AMC) the number of HIV cases in the world of course, considers a reaction between the p24 in the WB and antibodies present in sera, as proof of HIV infection. Yet, when the same reaction takes place between an antibody to the p24 of the WB and a patient serum, it is considered proof of viremia, and when between an antibody to p24 and material present in a cell culture, the same reaction is considered proof of HIV isolation.

Obviously, the detection of a protein, even if known to be virus specific, in sera or even culture, does not constitute proof for isolation or viremia. That such a finding is non-specific can be best illustrated by a few examples. In 1992, Jorg Shupbach, the principle author of one of the first four 1984 papers published by Gallo’s group on the “HIV gag protein” of the “HIV” virus reflected a “precursor to gp120 and gp41” and unlike the latter two proteins, is only found in infected cells and not in mature particles; (ii) Although many EM have been published of virus-like particles in non-banded material, which gp120 is a precursor to gp120 and gp41 and unlike the latter two proteins, is only found in infected cells and not in mature particles; (ii) Although many EM have been published of virus-like particles in non-banded material, not even the CDC, or Hans Gelderblom and his colleagues who have most thoroughly studied the antigenic properties of the HIV gag protein recently described with human T-cell-related virus lymphotropic-endogenous sequence...The characterization of this 25-30kD protein may represent an important contribution to the detection of HIV-1-related endogenous retroviruses.”

The disagreement between Montagnier and Gallo about which proteins were actually “HIV” proteins was not limited to gp41 but included p24. Montagnier always mentioned that “no cross-reactivity...
 existed between HIV p24 and other antibodies including antibodies to HTLV-I, II. Until 1985 he also maintained that there was “a very close homology between LAV and HTLV-III but an absence of homology with HTLV-I and -II.” However, in 1985 he wrote, “We have also compared the deduced amino-acid sequences of LAV proteins with those of HTLV-I and other retroviruses and find no significant homology. Although proteins pol and gag which are generally conserved among retroviruses”.

Gallo always maintained that homology exists between the HTLV-I, II and HIV gag genes and the many features shared by all “human retroviruses” include a “small (p24/p25) major capsid protein; p24 cross-reactive antigenic determinant detected with either heterologous (rabbit anti-human monkey) or homologous antiserum. Indeed, gag stands for group specific antigens. As far back as 1974 Gelderblom and his colleagues wrote, “While the virus envelope antigens are primarily virus-strain specific, the bulk of internal proteins of the virion with molecular weight (mw) between 10,000 d and 30,000 d are group-specific (gs) for viruses originating in a given animal species.” The mammalian C-type oncornaviruses [retroviruses] with a molecular weight in the range of 30,000 d was found to possess, besides gs spec. antigen, an antigenic determinant that is shared by C-type viruses of many mammalian species including monkeys and was thus termed gs interspecies (gs-interspec.) antigen.”

If p24 detected in culture supernatants is a component of similar particles, antigenic expression and the cytopathic effect. Montagnier’s. Such evidence can be obtained only by isolating the retrovirus; infection with Epstein-Barr virus and Treponema pallidum. Montagnier’s HIV immunogenic proteins. For example, serological tests for both HIV-1 and HIV-2 are considered to be directed against “HIV”, that is, the only way to use the antibody test to prove infection with Epstein-Barr virus and Treponema pallidum.

At present evidence also exists that:
(a) there is an association between the redistribution of polymers in particle budding. (b) polymerisation of actin, actin-myosin interaction and cross-linking of polymers in general is regulated by redox state, oxidation leading to interaction;
(c) both AIDS patients and cultures derived from AIDS patients are subjected to oxidising agents. In fact, for the detection of “HIV” proteins and particles the cell cultures must be stimulated (treated with oxidising agents). Ten years ago Montagnier wrote, “Indeed, LAV infection of resting T4 cells does not lead to viral replication or to expression of viral antigen on the cell surface, while stimulation by lectins or antigens of the same cells results in the production of viral particles, antigenic expression and the cytopathic effect.”

In the presence of antioxidants no “HIV” phenomena can be observed. Ten years ago in the 1988 International AIDS Conference, researchers from Rome reported, “The results obtained using 3-ABA, NAC [antioxidants] and a combined treatment 3-ABA/NAC given together seem to confirm the role of intracellular redox balance in the modulation of the HIV expression. In fact, a significant reduction in the number of viral particles was observed in cultures which have received the combined treatment with NAC/ABA.”

Given the above data, may one be tempted to speculate that the “HIV” particles and proteins are nothing more than “non-viral material altogether”, induced by the agents to which the AIDS patients and cultures are exposed?

CONCLUSION—The statement “antibodies against Montagnier’s HIV strain—the global standard of all “HIV tests”...”, presumes proof of:
(a) the existence of more than one “HIV strain”, including one of Montagnier’s. Such evidence can be obtained only by isolating the retrovirus. However, Montagnier’s evidence does not prove the isolation of a retrovirus;
(b) the existence of “HIV” specific immunogenic proteins. Again, such proof can be obtained only by isolating the retrovirus;
(c) antibodies specifically induced by HIV infection. It is true that for detection of such antibodies one does not need to use HIV or the HIV immunogenic proteins. For example, serological tests for both infectious mononucleosis and syphilis employ antigens derived from human blood cells and ox heart respectively but nonetheless predict infection with Epstein-Barr virus and Treponema pallidum. However, the only way to prove that “HIV antibodies” are directed against “HIV”, that is, the only way to use the antibody test to prove HIV infection, is to present evidence which proves that the antibodies are HIV specific. Such proof can be obtained only by using HIV isolation as a gold standard. Since this has not been done it is not possible to say that “the global standard of all “HIV tests”...” proves HIV
infection.

6. “HIV DNA”
In debating the proof for the existence of a unique, exogenous retroviral agent one cannot adopt as an initial premise (“Full-length HIV-1 and HIV-2 DNAs...”) that is contingent upon proof of the argument (“With HIV are identified...”). The a priori designation of a particular fragment of DNA as “HIV DNA” merely begs the question under consideration.

6.1 MINIMUM EVIDENCE REQUIRED TO PROVE THE EXISTENCE OF HIV DNA
If “HIV DNA” is the genome of a unique retroviral particle then the most basic requirement is proof for the existence of a unique molecular entity “HIV DNA”, that is, unique fragments of DNA identical in both composition and length in all infected individuals. The claim that a stretch of RNA (cDNA) is a unique molecular entity which constitutes the genome of a unique retrovirus can be accepted if and only if it is shown that the particles have the morphological, physical and replicative characteristics of a retroviral particle. Proof of these properties can only be obtained by isolating the putative viral particles, that is, by obtaining them separated from everything else, extracting the nucleic acids and demonstrating that such particles are identical (their constituents including their nucleic acids are identical) and infectious. The correct procedures, now having been used for over half a century to achieve this proof, require demonstration that:
1. In “infected” cell cultures (cocultures) there are particles with a diameter of 100-120nM containing “condensed inner bodies (cores)” and surfaces “studded with projections (spikes, knobs)”; 82
2. In sucrose density gradients the particles band at a density of 1.16 gm/ml;
3. At the density of 1.16 gm/ml there is nothing else but particles with the morphological characteristics of retroviral particles;
4. The particles contain only RNA and not DNA and that the RNA consistently has the same length (number of bases) and composition no matter how many times the experiment is repeated;
5. When the particles are introduced into secondary cultures, but mindful of the critical caveat discussed below:
(a) the particles are taken up by the cells;
(b) the entire RNA is reverse transcribed into cDNA;
(c) the entire cDNA is inserted into the cellular DNA;
(d) the DNA is transcribed into RNA which is translated into proteins;
6. As a result of 5 the cells in the secondary cultures release particles into the culture medium;
7. The particles released in the secondary cultures have exactly the same characteristics as the original particles, that is, they must have identical morphology, band at 1.16 gm/ml and contain the same RNA and proteins.

The caveat is that while the introduction of the majority of infectious particles into cell cultures and subsequent release of similar particles is proof that such particles are indeed infectious, this is not the sufficient case for retroviruses. The basis of this exception is the fact that “one of the most striking features that distinguishes retroviruses from all other animal viruses is the presence in the chromosomes of normal uninfected cells, of genomes closely related to, or identical with, those of infectious viruses.”83 In fact, a cell may contain the genome of many retroviruses. As far back as 1976 retrovirologists recognised that “the failure to isolate endogenous viruses from certain species may reflect the limitations of in vitro cocultivation techniques.”84 In other words: finding a retrovirus in both the primary and secondary “infected” cultures/cocultures is not proof that the cells have been infected with an exogenous retrovirus.

One way which will suggest but will not prove that the cells acquired virus from the outside (exogenously acquired retrovirus, infectious retrovirus) and have not assembled a retrovirus HIV from information already existing in normal cells (endogenous retrovirus) is to conduct experiments that use controls, that is, to run in parallel with test cultures/cocultures control cultures/cocultures. The only difference between the test and control cultures should be the introduction of particles into the test cultures. In other words, apart from the introduction of particles, in every other respect control cultures must be dealt with in the same way as the test cultures. In example:
(a) because detection of RT and retroviral genetic sequences and release of retroviral particles depends on the metabolic state of the cells, the physiological state of the cells used in the control cultures should be as close as possible to those of AIDS patients;
(b) because the mere act of co-cultivation alone may lead to release of endogenous retroviral particles, if test cells are cocultured, so should the cells used in control experiments.85

(c) extracts, even from normal unstimulated cells when added to the cultures may increase endogenous retroviral expression.

Because of this, when cells are cultured with “HIV” (supernatant or material which bands at 1.16 gm/ml), the controls must be cultured with similar material from cell cultures originating from sick individuals with illnesses similar to AIDS, that is, matched individuals who are infectious depressed.

(d) the appearance of endogenous retrovirus can be accelerated and the yield increased a million fold by stimulating the cultures with mitogens,85 mutagens, chemical carcinogens and radiation.86 If test cultures are exposed to or employ such agents so should the controls.

(e) since AIDS patients and those at risk of developing the syndrome are exposed to strong oxidising agents, the control cells should also originate from such patients;

(f) to avoid observer bias and in the best interests of science, blind examination of test and control cultures/cocultures should be performed.

6.2 EVIDENCE FOR THE EXISTENCE OF “HIV DNA”
6.2.1 In 1984, in the first of two papers, Montagnier and his colleagues described the following experiment: “Because LAV can induce T-cell fusion and because EBV [Epstein Barr virus] is known to have fused cell lines with DNA, we performed co-infection experiments on uninfected lymphocytes (B and T) with both viruses. It was hoped that stable hybrids of LAV-infected T cells and of EBV-transformed B cells would be formed and that such hybrids would be able to continuously produce LAV. Several regimes were tried. The one that gave rise to continuous productive infection of LAV was the following: Whole lymphocytes of F. R. were first stimulated for 24 hours with PHA and then cultured with both and then infected with LAV obtained from a nasopharyngeal carcinoma. Five days later, half of this culture was infected with LAV as described (1) and then divided in two subcultures: one was cultured in medium lacking T-cell growth factor (TGF: interleukin-2), the other in medium containing TCGF. As expected, the TCGF-fed culture produced LAV as detected by a peak of RT activity appearing day 12 after infection...At the time of decline of LAV production, a subculture of the TCGF-fed cells received fresh T cells from the same donor: these T cells had been activated for three days with phytohemagglutinin (PHA)...Six days later (day 25), a new peak of RT appeared, but contrary to the first infection, it was not transient...At the time of the second LAV infection, large round cells transformed by EBV could be readily seen in this culture, as well as in the control culture not infected with LAV, indicating that immortalization of the B cells by EBV had already occurred. The immortalized B-cell line was termed RF8.”87 (Reference 1 to which Montagnier refers is the 1983 paper in which Montagnier et al described the first isolation of HIV type 1.)

In the second study, 200 ml of supernatant from the “HIV infected” FRB8 cells were banded in sucrose gradients, “Virus containing fractions were pooled” and centrifuged. (It is not stated how they determined the existence of “virus”, in which band(s) “virus” was found, how many bands if any were found to have particles, or why there were more bands than one (1.16 gm/ml) containing the “virus”). The pellet was incubated with several substances, dATP, dGTP, dTTP, dCTP including [32]dCTP and an oligo(dT) primer. From the CDNAS thus obtained, three clones “pLAV13, 75 and 82, carrying (d) the DNA is transcribed into RNA which is translated into proteins;
In May 1984, Gallo and his colleagues published four papers. To "isolate" HIV they used a leukemic cell line which they called HT. It is impossible to know with what tissues from AIDS patients this cell line was cultured. Reading the May 1984 papers one gets the impression that the HT cell line was cultured with concentrated (supernatant) fluids originating from individual, AIDS patient, stimulated T-cell cultures, and that the fluids were subsequently centrifuged. When the supernatant was found the HT cell line was cultured with concentrated fluids pooled initially from individual cultures of three patients and ultimately from the individual cultures of ten patients.92 The Gallo investigation found this procedure to be "of dubious scientific rigor." One scientist described the procedure as "really crazy."93 In 1985, Gallo and his colleagues wrote, "The HT cells..."73b..."were derived from uninfected T-cell line HT, following co-culture with T lymphocytes obtained from several AIDS patients, and contains many different HTLV-III forms".94

The detection of reverse transcription of A(n).dT15 in the supernatant, was considered proof of the HT cells being infected with a retrovirus, HIV, which originated from the patients' tissues. A clone, H9, of this infected cell line was described as "using sodium dodecyl sulfate as a helper for a donor as a feeder."95 The H9 cells were cultured with supernatant from the "HIV" infected HT cells. The H9 supernatant was banded in sucrose density gradients and the material which banded at 1.16 g/ml, without proof, Gallo and his colleagues considered to be synonymous with retroviral particles, was "lysed with sodium dodecyl sulphate (SDS), digested with proteinase K, and directly chromatographed on oligo(dT) cellulose column. The resulting cDNA of both infected and uninfected H9 cells as well as other uninfected human cell lines, only containing RNA purified from infected [that is, cells cultured with the virus], was used for the HTLV-III provirus was found to lack Xba I restriction enzyme activity, and contains many different HTLV-III forms".94

"Since the HTLV-III provirus was found to lack Xba I restriction enzyme activity, in a circular configuration containing a retrovirus, two long terminal repeats (LTRs); the upper broad band (>15kb) represents provirus integrated into the host cell DNA." In an additional experiment "whole-cell DNA from cells infected with ARV-2 was partially digested with Cc+RI; 9-15 kb cell DNA was cloned into an EMBL-4 bacteriophage l vector and recombinant phage were identified with the virus-specific cDNA probe". Among the recombinant phage obtained were I-8B and I-7A, each of which was 9.5 kb.96

**6.2.4 SUMMARY AND DISCUSSION**

It is obvious that although Montagnier, Gallo and Levy and their respective colleagues refer to virion or virus particles purification or isolation, none of these groups have presented evidence for the isolation of retrovirus particles or even the isolation of virus-like particles, the first and absolutely necessary step in proving the existence of a retroviral genome. (At the time of writing, neither has any other group of HIV/AIDS researchers). Finding some RNA which bands at 1.16 g/ml, selecting from it a poly(A) rich fraction, or a fragment of a genome, even if it hybridizes to the cDNA probe, does not give evidence for the presence of a retrovirus or even proof of isolation. Supernatants from these cultures were introduced into cultures of leukaemic or transformed cell lines. With the supernatants from these cultures they performed two types of experiments:

(a) Supernatants were banded in sucrose density gradients. At the 1.16 g/ml band (and sometimes at other bands) - at least in Montagnier's group's experiments, this is not made clear), they found fragments of RNA of certain lengths (although no two had the same length) or were rich in adenine, (poly(A) rich fragments), and called these "HIV RNA", the "HIV genome". Using a (dT) primer the "HIV RNA" was transcribed into a complementary DNA (cDNA).

(b) The supernatants were introduced into another set of the transformed and leukaemic cell lines as well as into stimulated cultures of normal T-cells. The DNA from these cells, as well as the DNA from the cultures to which no supernatant was added, were hybridised using probes from the cDNA. Positive results were obtained only with the DNA from the cells to which the supernatants were added. This evidence was interpreted as proving that the retrovirus, originated from the AIDS patients and in fact that these patient acquired it from the outside, that is, the retrovirus was exogenous.

There are many problems associated with these experiments and their interpretation. Among the many questions their conclusion raises the most obvious are:

1. HIV is said to be a retrovirus and retroviruses are particles...
which contain among other things, RNA. How then is it possible to claim that the RNA which banded at 1.16 gm/ml, “HIV RNA”, is the genome of a retrovirus without proof that it is a constituent of a particle, viral or non-viral which bands at this density? 2. RT is not specific to retrovirus and in fact A(n).dT15 can be reverse transcribed by all cellular DNA polymerases a, b and g. Is it possible that reverse transcription of endogenous human retroviruses is a prerequisite for HIV isolation or even detection of a retrovirus? Even if the process of reverse transcription were specific to retroviruses, can the detection of a process ever be considered proof for the isolation of an object, in this case, retroviral particles? 3. Cell culture supernatants will contain both DNA and RNA including non-viral cellular RNA (fragments) especially if cellular viability is not one hundred percent as is the case in cultures used by the three groups. The RNAs may include messenger RNA (which is adenine rich), as well as high molecular weight heterogeneous nuclear RNA. These RNAs, in addition to having high molecular weight and heterogeneity in size, also have poly(A), with the poly(A) attached at the 3’ end of the molecule, and may be RNAase resistant. Actinomycin inhibits its synthesis and also interferes with its proper processing and breakdown. 4. From animal virology it is also known that non-retroviral RNA and DNA also bands at 1.16 gm/ml. 100 How is it then possible to claim that just because a RNA bands at 1.16 gm/ml and is adenine rich or has a certain length, it is “HIV RNA”? 5. If this RNA is “HIV RNA”, then what is the other RNA and the DNA which also bands at this particular density? If the latter are cellular why not the poly(A)RNA as well? 6. By definition, retroviruses are infectious particles which contain only RNA. When they enter a cell the RNA is reverse transcribed into DNA, which is then integrated into cellular DNA as a provirus, which maintains its integrity and presence in the genome elsewhere. Yet many HIV experts including Gallo have shown that both the supernatants of “infected” cells and the “HIV particles”, that is, the material which bands at 1.16 gm/ml, contains “HIV DNA” which “may integrate directly into the host chromosomal DNA”. 101,103 The question then arises, is the “HIV DNA” the result of “HIV RNA” reverse transcription or is it vice versa? 7. It is accepted that the HIV RNA is localised in a condensed core surrounded by a “lipid-bilayered envelope derived from the cellular membrane of the host cell, studded with virally encoded gp120 and myristylated protein, p17. The so-called core-envelope link (CEL) attaches the core to the envelope”. 103 One of the best known facts in biology is that condensed cores (chromatin) is transcriptionally inactive. This is one of the reasons why viruses, including retroviruses, to multiply, must first enter cells where their chromatin is decondensed. However, in a paper published in 1993, Hui Zhang and colleagues including Poiesz, from Suny Health Science Center at Syracuse, New York, wrote: “We have shown that in the absence of detergent, large amounts of DNAase-resistant viral DNA can be synthesized within intact HIV-1 virions. This phenomenon is not dependent on perturbation of the viral envelope. [Not to mention decondensation of chromatin]. Nascent viral DNA synthesis also occurred in purified virions incubated at 37° in cell-free human physiological fluids including seminal plasma, breast milk, and fecal fluids”.103 This means that the intact HIV-1 virions “perform” a function that no other biological system with very condensed and protected chromatin can perform; or (ii) the “HIV RNA” found in the supernatants or in the “purified virions” is present in an unembodied form; or (iii) the “HIV RNAs” are de novo synthesised in the cell cultures (see 6.3.5). 8. At present there is ample evidence that any RNA or DNA present in the supernatant, irrespective of its origin, especially when cells are stimulated by polycations and oxidising agents, will be taken up by the cells (see 7.1). How is it then possible to claim that a positive hybridisation signal in cells cultured with the same “HIV DNA” con-taining supernatant as the supernatant from which the “HIV DNA” probe originated but not in other cells is proof that the “HIV DNA” is the genome of an exogenous retrovirus? 9. The first, absolutely necessary step in proving that the “HIV DNA” originated from the lymphocyte cells of AIDS patients and those at risk, is to perform hybridisation experiments using the DNA of the HTLV-III cell line which Gallo, Popovic, and their colleagues used was a new cell line and one which they established. The Gallo inquiry revealed that the HT (H9) cell line is the same as that used by Levy’s group, HUT78, a leukaemic cell line established in another laboratory. However, the abundant evidence for the expression of endogenous retroviruses has largely been obtained from experiments on leukaemic and transformed cells. Evidence exists that both H9 and EBV-transformed B lymphocytes release retrovirus-like particles even when not “infected with HIV”. 104 Furthermore, the HUT78 (H9) cell line was established from a patient with “malignancies of mature T4 cells”, a disease which, according to Gallo, is caused by the exogenous retrovirus, HTLV-I. Indeed, as far back as 1983, he claimed to have shown that the HT (H9) cell line contained HTLV proviral sequences. 105 According to some American researchers, EBV-transformed normal human peripheral blood B lymphocytes contain HTLV-I related transcripts. 106 Since all retroviral particles by definition band at 1.16 gm/ml, assuming that all the groups have retroviruses at a certain density, how is it possible to claim that the retrovirus originating from the HUT78 and EBV-transformed B lymphocytes is a new retrovirus HIV, and not one which was already present? Can one claim that the “HIV RNA” and thus the probes and primers originating from it are the RNA and probes and primers of a unique exogenous retroviral genome? 10. The biological dogma states that DNA is synthesised on a DNA template, RNA on a DNA template, and proteins on an RNA template. In other words, the only way for a cell to acquire new nucleic acid entities is for them to be introduced from the outside, exogenously either from another cell type, an infectious agent or a synthetic nucleic acid. If the biological dogma is correct then the “HIV RNA”, be it a cellular or viral molecular entity, should have originated either from the patients’ lymphocytes or the transformed and leukaemic cell lines. However, when “HIV cDNA” was used as a probe, not one of the groups reported positive hybridization results from any of the cells, not even from the lymphocytes of AIDS patients. The question then arises, does a unique molecular entity, “HIV DNA” exist? What does it mean and from where did it originate? 6.3. Speculations on “HIV DNA” If one wishes to speculate on the nature and origin of RNA (cDNA) derived from the cultures containing tissues of AIDS patients and those at risk, and which bands at 1.16 gm/ml, there are many possibilities including: 6.3.1 Although to date no such evidence exists, it is possible that the stretch of RNA, presently called “HIV RNA”, is the genome of an exogenous retrovirus, HIV. However, for this to be considered proven in addition to satisfying all the requirements in 6.1 one must also show that (i) the unique stretch of RNA can be obtained only from cells of particular individuals; (ii) when the RNA (or cDNA) is used as a probe to test fresh, uncul-tured lymphocytes, a positive test is obtained only from the fresh cells of individuals who also have a positive culture; (iii) that in animals or humans, the retrovirus is horizontally (animal to animal, person to person) transmitted. 6.3.2 The genome of an endogenous retrovirus, that is, a stretch of RNA with a corresponding DNA template present in the cellular DNA of uninfected animals and which is passed from generation to generation vertically (from parents to offspring via the germ cell line) and which under certain conditions can be expressed and incorporated into retroviral particles. For many decades it has been known that animal DNA contains sequences “closely related or identical with those of infectious viruses”. However, the human genome was considered to be an exception and as late as 1994, both Gallo and Fauci were of the opinion that “…there are no known human endogenous retroviruses”. 107 In fact, in the 1970s and in the 1980s after Gallo’s claim of the discovery of HL23V, HTLV-I and later HTLV-II, and especially after Montagnier’s claim of the discovery of HIV, considerably greater interest was engendered in retroviruses with the result that it became “increasingly clear that the DNA of man, like that of other vertebrates, contains many integrated retroviral elements”. 108,109 and that in many cases the genes are expressed, “including mRNA transcripts related to full-length endogenous retroviral DNA”. 109,110 with open reading frames for the gag, pol and env proteins. 111 By 1987, many researchers reported the expression of the genome of the human endogenous retrovirus, HERV-K, homologous to the mouse mammary tumor virus (MMTV). “In several cell lines, HERV-K genome was expressed as an 8.8 kilobase poly(A)+ RNA which appears to be the full-length tran-
script of this genome". When the human breast cancer cell line T47D was “grown in RPMI 1640 supplemented with 10% fetal calf serum, HERV-K genome expression was slight”. However, when the cells were treated with estradiol and then progesterone, they produced “retrovirus-like particles and soluble protein sharing antigenic determinants with MMTV env gene product”. In support of their thesis "that HTLV-I is not an exogenous infectious agent", Levy and colleagues' HUT78 cell line contains 50 copies of HERV-K. HERV-K is a human endogenous class I retroviral element that contains gag, pol, and env open reading frames...as well as intact LTR regions...Expression of a 9 kb genomic HERV-K RNA transcript was detected in human cell lines...We were able to show for the first time the expression of HERV-K pol gene in human cells and that HERV-K RNA was also observed in peripheral blood cells from two sets of non-leukemic individuals. The first set consisted of seven normal donors, while the second set consisted of 3 patients with PV, all of which expressed HERV-K pol gene. Five different nucleotide sequences were obtained from the seven normal donors. Four of the five normal sequences contained heterogenous proviral reading frames for pol as detected by both RT-PCR and RTNAase protection experiments, which normal cells did not express HERV-K proviruses, analysis of HERV-K pol gene from PV patient showed selective expression of a restricted family of related proviruses.111 By 1995, Gallo admitted that the human cell does contain retroviral genomes but he still insisted they are defective. "Retroviral proviral sequences or cellular elements (exogenous forms) or as infectious agents (exogenous forms). As do many other animal species, humans have both forms...The DNA of many species, including humans, harbor multiple copies of different retroviral proviruses. The human endogenous proviral sequences are virtually all defective, and comprise about one percent of the human genome”.112 The view regarding retroviral elements was further shared by Montagnier, with his colleagues, has extensively studied the human endogenous retroviruses113 and have shown that HERV-K sequences are transcribed and that a human teratocarcinoma cell line, GH, which contains these sequences, when examined by EM was found to produce “human teratocarcinoma-derived retrovirus (HTDV) particles”. By 1993 Kurth and colleagues reported that in the GH cell line, “Four viral mRNA species could be identified, including a full-length mRNA. The other three subgenomic RNAs are generated by single or double splicing events...Sequence analysis of expressed HERV-K genomes revealed non-defective gag genes, a prerequisite for particle formation. Open reading frames were also observed in pol and env. Antiserum raised against recombinant gag proteins of HERV-K retained HTLV-I electrophoretic mobility, linking the HERV-K to the HERV-K family” . Discussing their findings they wrote: "In Northern blots, expression of HERV-K could only be demonstrated in teratocarcinoma cell lines but not in other human lines. Preliminary RT PCR studies suggest, however, that HERV-K may be expressed in many if not all human cells at levels too low to be detectable in Northern blots. The basic similarity of herpesvirus sequences and the lack of differences in expression between teratocarcinoma cells and other cell lines is not clear. It is intriguing to speculate that a cellular factor(s) may regulate the synthesis of HERV-K mRNA depending on the cell type or the state of differentiation. In this context, it should be remembered that other retroid elements [ERV-9, RMLVL-H, LINE-1] all preferentially expressed in human teratocarcinoma cells”.114 It is of interest to note that Montagnier and his colleagues reported their “HIV genome” from a transformed cell line, that Levy and colleagues’ HUT78 cell line is a human leukaemic cell line and that Gallo and colleagues’ H9 cell line is one other than HUT78, and thus must have HTLV-I as well as endogenous retrovirus. It is equally important to note that although Kurth et al found no sequence homology between HERV-K and “human T-lymphotropic virus” or HIV, many researchers reported HTLV-I sequences in the human genome including in cell lines derived from teratocarcinoma.

In a paper published in 1985 researchers from a number of institutions in the USA including the Laboratory of Tumor Immunology and Biology, National Cancer Institute, Bethesda, reported that “Human endogenous proviral DNAs. In addition, human T-cell leukaemia virus type I-related sequences appear to be transcribed in both normal human T cells and in a cell line derived from a human teratocarcinoma”.115 In a paper published in 1989, researchers from the USA summarised their experimental findings as follows: “Human T-cell lymphotropic virus (HTLV) type I-related endogenous sequences (HRES) have been cloned from a human genomic library. HRES-1/1 is present in DNA of all normal donors examined. By nucleotide sequence analysis, HRES-1/1 contains two potential open reading frames capable of encoding a p25 and a p15. A 684 flanking region 5’ from the first ATG contains a TATA-box, a poly-A addition signal, a putative tRNA primer binding site, and inverted repeats at locations which are typical of a retroviral long terminal repeat...The HRES-1/1 genomic locus is transcriptionally active in lymphoid cells”, including EBV-transformed normal human peripheral blood lymphocytes, leukemic cell lines, melanoma cells and embryonic tissues.116 In a paper published in 1997 researchers from Hungary and Britain entitled “Human T-cell lymphotropic virus (HTLV)-related endogenous sequences, HRES-1, encodes a 28-kDa protein: A possible autoantigen for HTLV-I gag-reactive autoantibodies”, the “presence of a human T-cell lymphotropic virus (HTLV)-related endogenous sequence, HRES-1, in the human genome was documented. The HRES-1 genomic locus is transcriptionally active and contains open reading frames...Antibodies to HRES-1-specific synthetic peptides were noted in patients with MS, progressive systemic sclerosis (PSS), SLW, Sjogren syndrome (SJS), and essential cryoglobulinemia (ECG). The data suggest that HRES-1 may serve as an autoantigen and correspond to a natural target of HTLV-I core protein-reactive autoantibodies”.117

6.3.3 The genome of a retrovirus de novo assembled by genetic recombination and deletion of:
(a) endogenous retroviral sequences;
(b) retroviral and cellular sequences;
(c) non-retroviral cellular genes.
In the virological literature there is ample evidence which shows that when a cell contains two proviruses, progeny may be found that possess the genome of one but the structural proteins of either or both viruses present. Conversely, the RNA may be viral but at least some of the proteins may be cellular. In other instances, the particles do not have a genome at all, or one or more genes are missing (genetically defective particles). The genetic mixing can be between viral genomes or between viral and cellular genes. According to distinguished retrovirologists such as Weiss and Temin, new retroviral genomes may arise by rearrangement of cellular DNA caused by many factors including pathogenic processes, a view that proposes retroviruses as an effect and not the cause of disease. According to Varum, “Retroviral genomes recombine at high frequency (estimates range as high as 10 to 30% for each cycle of multiplication), and heterodimeric RNAs are thought to be intermediates, with recombination taking place during reverse transcription. Recombination appears to be strongly favoured by homology, but joining also occurs occasionally between unrelated sequences, e.g., due to errors in the reverse transcription by retroviruses. When viruses are grown in cells that contain related endogenous proviruses, packageable transcripts from those proviruses may participate in recombination reactions with the exogenous virus. This is most dramatically revealed by the repair of deletion mutations in the genome of an exogenous virus in a fashion that superficially resembles gene conversion”. In some animals proviruses have been acquired “during recent breeding of the strains in the laboratory” and “in a few
instances, endogenous proviruses have been established or increased in number during experimental observations\textsuperscript{121} (italics ours).

As far back as 1974, based on the then available evidence, Howard Temin proposed that the retroviral (ribodeoxyviruses) genomes originate from “normal cellular components. The relationship between these retrotransposons and the cellular components that reflect the relationships among the cellular components from which the viruses evolved and the convergent evolution of the viruses. In other words, there are relationships among ribodeoxyviruses because the ribodeoxyviruses evolved from cells which themselves had relationships deriving from common ancestors. A possible mechanism of this evolution is described in Fig. 5. In the legend to Fig. 5, Temin wrote: “A section of a cell genome becomes modified in successive DNA (W) to RNA (\textsuperscript{-}t) to DNA transfers until it becomes a ribodeoxyvirus genome. First, these sequences evolve as part of a cellular genome. After they have escaped as a virus, they evolve independently as a virus genome. The time scale may be millions of years in germ-line cells and days in somatic cells”.\textsuperscript{122} Temin reinforced his view in a more recent publication.\textsuperscript{124}

In 1975, Gallo, Gillespie and their colleagues wrote: “Even though RNA of class II [exogenous] retroviruses shows minimal homology to uninfected host cell DNA, hybridization of nucleic acids among class II leukemia viruses from different species gives a pattern which is the same as the phylogenetic relationships among their natural hosts...We have proposed other results for the intestine of the mouse and the rat...that all RNA tumor viruses are derived from cell genes, a proposal in agreement with the virology thought...By analysis of the RNA of virions infecting and replicating in a new host, evidence has also been obtained which indicates that the genome of type C viruses can be substantially changed by the host, probably by recombination with host DNA.\textsuperscript{125} A few years later, Coffin wrote: “The close relationship of virion proteins as well as overall nucleic acid homology must mean that both exogenous and endogenous avian tumor viruses [retroviruses] derive from a common ancestor”.\textsuperscript{126}

In 1991 researchers from the New York University published a paper entitled “Implications of the ‘Endogenous Retrovirus’”. Discussing the presently available data they wrote, “A recent detailed phylogenetic analysis of exogenous and endogenous retroviruses (including retrotransposons) strongly suggests that a pool of endogenous retroviral sequences periodically contributes to the generation of exogenous viruses, and that the presence of endogenous primate retroviruses is probably more directly related to exogenous viruses that might have been thought”.\textsuperscript{127}

6.3.4 The “novel” RNA found in the cell culture supernatant and the material from it banding at 1.16 gm/ml; the “HIV RNA”, may have nothing to do with a retroviral genome. It may be an RNA obtained by transposition, that is, by certain replicating DNA sequences (transposons) first being transcribed into DNA and then similarly being inserted into the genome. Reproposition can “use cellular mechanisms for passive retrotransposition, as well as retroelements containing reverse transcriptase”. The retroelements may be retrovirus-like elements or nonviral elements.\textsuperscript{128-130} Another strongly held view was the belief that the Endogenous Retrovirus. Discussing the presently available data they wrote, “A recent detailed phylogenetic analysis of exogenous and endogenous retroviruses (including retrotransposons) strongly suggests that a pool of endogenous retroviral sequences periodically contributes to the generation of exogenous viruses, and that the presence of endogenous primate retroviruses is probably more directly related to exogenous viruses that might have been thought”.\textsuperscript{127}

During the 1970s it was the discovery that the DNA base sequence, which coded for a given protein were not in a continuous stretch of DNA but may be interspersed with other, non-coding base sequences, that is, the genes are split, “genes-in-pieces”. A number of mechanisms have been postulated to account for this observation. In one such explanation it is hypothesised that the entire stretch of DNA is transcribed into a piece of RNA, then the non-coding regions (introns) are excised and the coding regions (exons) are spliced together to make the appropriate messenger RNA.\textsuperscript{131} There are no rules setting an upper limit on the number of introns in a “gene”, some genes may have up to sixteen or more introns. Nor are there any rules regarding the length of introns, although in general, introns are much longer than exons, the length of exons “peaking at about 40 of 100 amino acids...[itis]...the intron between 50 300 bases long, the longest extending out to some 50,000 bp”.\textsuperscript{132} According to Gilbert introns represent “hot spots” for recombination and new genes can be created “through the coupling of exons by intron-mediated recombination”, “introns are lost and more compli- cated exons are formed”.\textsuperscript{133} At present evidence exists showing that non-coding regions (introns) can become genetic elements, “chopped-up” or “chopped up” genetic elements, they self-splice, often contain reading frames capable of encoding a protein including “regions of homology to reverse transcriptase scattered over a roughly 250-amino acid stretch in the middle of each intron ORF”.\textsuperscript{134} The discovery of split genes “shows that the genetic apparatus of the cell is more complex, more dynamic than any of us had suspected”\textsuperscript{135}

Another strongly held view was the belief that all cellular reactions and the gene splicing were catalyzed by a protein enzyme. In the early 1980s it was found that RNA can cut, splice and assemble itself, as well as assemble RNAs other than itself: 136 137

6.3.6 One of the strongest held views in biology is the belief that nucleic acids have an inherent ability of instructing their own synthesis and that nucleic acids cannot be synthesised in the absence of a nucleic acid template. Manfred Eigen and his colleagues in Germany conducted extensive theoretical and experimental work on molecular self-replication.\textsuperscript{138} In their experimental work they used the bacterial virus (phage) Qb. In one of their experiments they showed that the molecular species of 4500 nucleotides, the virus has an RNA molecule of 220 nucleotides known as “Spiegelman's minivariant” which, like the genomic RNA, is reproduced in cell-free laboratory systems by an enzyme called Qb replicase. By mixing Mg\textsuperscript{2+} ions, the nucleoside triphosphates ATP, GTP, UTP, CTP, Qb replicase and template RNA, they could obtain RNA replication but a totally unsuspected finding was that even the absence of the template, RNA was still synthe-
sised. They performed many experiments to prove this phenomenon and to exclude the possibility of the presence of an initial RNA template and concluded, “Finally we were convinced we had before us RNA molecules that had been synthesised de novo by the Q₅ repli-case enzyme. What was most puzzling, the de novo product had a uniform composition which in each trial turned out to be similar to or even identical with Spiegelman’s minivariant.” When the template free mixture was then divided into several isolated compartments where optimal conditions for de novo synthesis were maintained they found that “each component had a uniform population of de novo product, the products differed from compartment to compartment. Further analysis revealed however that the different sequences were not completely unrelated... There was a definite, uniform final product for any set of experimental conditions, but here were as many different optimal products as there were different experimental conditions.

One of the optimal products appeared to be Spiegelman’s minivariant... Other products of optimization were adapted to conditions that would destroy RNAs, such as high concentrations of ribonuclease, an enzyme that digests RNA into pieces... Some variants bridge so well adapted to odd environments that they had a replication efficiency as much as 1000 times that of variants adapted to a normal environment... Any RNA formed by noninstructed chemistry would be reproduced by template-instructed chemistry at a rate proportional to the current RNA concentration. The result would be exponential growth. Furthermore, even if only a single template were formed initially by noninstructed synthesis, there would be a host of different sequences because errors (point mutations, insertions and deletions) would inevitably be made in the course of replication. Hence in each generation there would be not only a larger number of RNA strands but also a greater variety of RNA sequences. What would happen then to some of these variants would be copied more rapidly than others or would be less susceptible to errors in copying, and their concentration would increase more rapidly. Sooner or later these faster-growing variants would take over... Hence the results of the self-replication competition had to be the master sequence together with a huge swarm of mutants derived from it and from which it had no way of escape... We call this entire mutant distribution a quasi-species. It is the quasispecies mutant distribution that survives the competition among self-replicating RNAs and not just one master sequence or several equivalent ones that are the fittest genes in the distribution. The essence of selection then is the stability of the quasi-species.”

According to Eigen and his colleagues, the maximum length of an RNA master sequence is of the order of 10,000 nucleotides. 139, 141

6.3.7 A basic principle of molecular biology is that the primary sequence of RNA faithfully represents the primary sequence of the DNA from which it is transcribed. However, in the 1980s RNA editing, “broadly defined as a process that changes the nucleotide sequences of an RNA molecule from that of the DNA template encoding it,” was discovered. In these RNA sequences non-functional RNA, pseudogenes, were found, producing a translatable mRNA, or modifying an already functional transcript that lacked sufficient complementarity to hybridize to its own template. Sometimes editing is so extensive that the majority of sequences in an mRNA are not genomically encoded but are generated post-transcriptionally producing the “paradoxical situation of a transcript that lacks genetic complementarity to its own gene.” 142, 143 According to Nancy Maizels and Alan Weiner from the Department of Molecular Biophysics and Biochemistry at Yale University, “the central dogma has survived the test. The discovery of reverse transcriptase amended but did not violate the central dogma of how genes make proteins; introns qualified the conclusion that genes are non-overlapping with the proteins they encode; somatic rearrangement of lymphocyte DNA called stability of eukaryotic genomes into doubt... and catalytic RNA challenged the pre-emience of proteins and breathed new life into the ancient RNA world.”

However, the discovery of RNA editing “could come close to dealing it a mortal blow.” 143

6.3.8 CONCLUSION

The finding of a novel stretch of RNA or DNA and proteins in:

(a) lymphocytes of sick individuals or individuals who have been “shocked” with agents such as physical or chemical mitogens, carcinogens or oxidising agents in general as is the case with AIDS patients and those at risk; 70, 86
(b) lymphocytes in cultures or co-cultures (which could lead to the appearance of hybrids) which have been additionally “shocked” with sometimes multiple, similar agents; is not proof that the given stretch of RNA comes from the outside, irrespective of its length, the presence of poly(A) and number of ORF (“genes”).

From Montagnier’s, Gallo’s and Levy’s and their colleagues’ evidence it is not possible to conclude that the “HIV RNAs” they found are a “new species” of RNAs induced by “shocking” the cells or by one or more of the other phenomena which have come to light in the 1980s. Nor is it possible to conclude that their RNAs are the genome of an exogenous retrovirus as they did. However, a number of predictions can be made:

(a) if the “HIV DNA” is indeed the genome of an exogenous retrovirus then:

(i) there must be evidence to prove the existence of a unique molecular entity “HIV RNA”, and a corresponding fragment of DNA (“HIV DNA”) which has a unique length and unique nucleic acid sequences;

(ii) when the full length fragment of “HIV DNA” or “HIV cDNA” is used for hybridisation studies all infected people should give a positive result.

(b) if the selected RNA which was found to band at 1.16 gm/ml, the “HIV RNA”, is the genome of a retrovirus which exists “in all of us”, endogenous retrovirus, then again evidence must prove the existence of a unique molecular entity “HIV RNA”. Where hybridisation studies are conducted using the full length of the unique molecular entity as a probe, positive results should be found “in all of us”;

(c) if the RNA found by the three groups, “HIV RNA”, is the genome of a retrovirus assembled de novo from DNA already existing in the cells, as the result of in vitro or in vivo competition, evidence must also prove the existence of a unique molecular entity. When the whole length of the unique fragment of nucleic acids is used as a hybridisation probe, a positive result should only be found in cells which are subjected to exactly the same in vivo or in vitro conditions as those from which the “HIV RNA” at 1.16 gm/ml was obtained. Where only for an “amplified” RNA used for hybridisation, the probability of finding a positive result will increase;

(d) if the “HIV RNA” is a unique non-viral molecular species of RNA resulting from the transcription of a unique molecular species of DNA then when the whole fragment of “HIV RNA”, (“HIV cDNA”) is used a probe for hybridisation studies, a positive result should be found when the RNA is amplified using an amplified RNA or DNA of identical length and sequences;

(e) if the “HIV RNA” is neither the genome of a retrovirus nor a faithful transcript of a fragment of DNA present in the cells from which it has been obtained, but is the result of the “shock” to the cells that have been exposed, either in vivo or in vitro or both, or as a result of the phenomena discovered in the 1980s then:

(i) since it is not possible to exactly reproduce the conditions in vivo or in vitro to which the cells are subjected, it would prove difficult if not impossible to always obtain a unique molecular entity “HIV RNA”, that is, to always obtain a fragment of RNA or DNA of identical length and sequences;

(ii) when the full-length fragments of “HIV RNA” or “HIV cDNA” are used as hybridisation probes there will be only a low probability of finding a positive result. However, the probability will increase if only small fragments of the “HIV RNA” or “HIV cDNA” are employed.

6.4. EVIDENCE THAT THE “HIV RNA” BELONGS TO AN EXOGENOUS RETROVIRUS

The Montagnier, Gallo and Levy groups claimed that the special RNA which they selected from the total RNA which in sucrose density gradients banded at the density of 1.16 gm/ml was novel to the lymphocytes and that in fact belonged to an exogenous retrovirus. Although they did not present evidence to prove this assertion, the possibility cannot be excluded that indeed this may have been the case. Since at present their claim is generally accepted one would have thought that by now they or other researchers should have been able to provide ample confirmatory proof. This does not seem to be the case:

6.4.1 If the RNA originates from a retrovirus either endogenous or exogenous then evidence must exist which proves that such RNA is a constituent of particles which possess at least the most basic morphological and physical features of retroviruses, that is, “a diameter of 100-120 nm budding at cellular membranes. Cell-released virions contain condensed inner bodies (cores) and are studded with projections (spikes, knobs)” 87 To date not only has nobody shown that the “HIV RNA” belongs to such particles, there is no evidence that particles of any kind are present in the material from cell cultures/cocultures which bands at the retroviral density of 1.16 gm/ml and from which the “HIV RNA” is selected. Furthermore, although particles have been demonstrated in cultures, cultures contain many different types of particles but none display BOTH principal morphological characteristics, that is, “a diameter of 100-120 nm” AND surfaces which “are studded with projections (spikes, knobs)” 146.
6.4.2 If the “HIV RNA” is the genome of an exogenous retrovirus then, like the “exogenous animal retroviruses”, one should be able to find it in infected material without the necessity to revert to the use of co-cultivation or mitogenically stimulated cultures. However, none of the phenomena which are thought to prove the existence of HIV can be demonstrated. In one, one employs mitogens or co-cultures or both (and sometimes additional “shock”), a fact accepted by both Antinagier and Gallo.147

6.4.3 One cannot claim that “HIV RNA” is the genome of a unique retrovirus, HIV, unless evidence is presented to prove that “HIV” is a unique molecular entity.

By 1985 it was generally known that the “env genes of ARV and HTLV-II differ by more than 20 percent” and that “the Gallo group has sequenced another HTLV-III isolate and finds that it differs from the first by about as much as ARC”.124, 148 By 1986, Gallo and his colleagues accepted that the “HIV genome” has a “far greater variability” as “compared to HTLV” and in fact “The rate of genetic change for the HIV virus is much higher than for any other DNA viruses or RNA genomes and may even be tenfold greater than for some other RNA viruses including certain retroviruses and influenza A virus”.149 At present it is accepted that “no two isolates are identical. Each isolate contains many variants”.141 In one and the same patient the genomic data in monocytes differs from that in T-lymphocytes.151 There are “striking differences” between the proviral DNA and cDNA in one and the same PBMC sample “which could not be explained by either an artefact of reverse transcriptase efficiency or template selection bias”.152 The genetic data obtained in vitro do not correlate with the data obtained in vivo, “to culture is to disturb”.133 According to the researchers from the Pasteur Institute “an asymptomatic patient can harbor at least 105 genetically distinct variants of HIV, and AIDS patient the figure is more than 106”.144 The “HIV genome” varies with time; in one case where clones were obtained 16 months apart all the clones detected in the second sample were distinct from the clones in the first sample.156 It is also accepted that up to 99.9% of the “HIV genomes” may be defective.145

According to Levy, “The mechanism responsible for generating these varying strains of virions is puzzling. One theoretical possibility is that the unintegrated proviral copies of HIV that accumulate during acute replicative infection can undergo efficient genomic recombination leading to the evolution of infectious variants.”156 In Robin Weiss’ view, “the source of variation is the infidelity of reverse transcription, which has no editing mechanism for transcriptional errors”, as well as “genetic recombination” especially when cell fusion takes place.134 According to Levy, “at the end of this year...at least ten” (A-J) prevalent major (M) regions of the United States”. “Given the enzyme’s relative small size and the intra-genotypic gag distances averaged 7% whereas the inter-genotypic distances averaged 14%...The maximum level of variability in gag is still well below that observed for the envelope region of HIV-1”.146 “Two HIV-1 strains, designated ANTO70 and MVP5180 were isolated in 1987 and 1991 respectively from patients in Cameroon”. They were classified as HIV-1 subtype O. By 1994 evidence was presented which “indicated that subtype O was endemic in Cameroon and Gabon”.167 “DNA sequence analysis of MVP-5180 showed that its genetic organisation was that of HIV-1, with 65% similarity to HIV-1 and 16% similarity to HIV-2. The HIV-1 subtype was identified as subtype B”.168 Comparison of the MVP-5180 amino acid sequence with that of the Gabon chimpanzee virus showed similarities of 70, 78 and 53% in the gag, pol, and env respectively; similarities of 70, 76 and 51% to the Uganda HIV-1 (U455) and of 54, 57 and 34% to the HIV-2 isolate MVC-4 were found”.169

16.1. By the mid 1980s, researchers from the Pasteur Institute concluded, “it is increasingly clear that it will be very difficult to describe correctly the characteristics of HIV viruses using single molecular clones”. “It is evident that HIV, either in vivo or in vitro, is extraordinarily complex and that a population-based approach, a quasi-species approach, as proposed by Eigen, is required to define the true HIV.” They also added, “Even with a population-based approach, only small regions of the HIV genome can be studied... Given such complexity and the evident differences between quasi-species in vivo and in vitro, the task of defining HIV infection in molecular terms will be difficult.”160 The data which have been published since confirm this conclusion. Prior to the 1990s, the HIV sequences were classified as African and USA/European with sequence differences of 20-30 percent between these two groups.161 In the 1990s, HIV researchers started to divide the “HIV genome” into subtypes A, B, C, D, E, etc. The basis for this classification system is:

(a) subtypes are approximately equidistant from one another in env

(b) the env phylogenetic tree is for the most part congruent with gag phylogenetic trees;

(c) two or more samples are required to define a sequence subtype.

However, “Subtype naming problems have arisen for several reasons. A small but not insignificant number of viral sequences are hybrid, clustering with one sequence subtype in gag and another sequence subtype in env, for example; or, to take another example, clustering over different stretches with two or more subtypes in env. Naming becomes problematic when highly divergent forms of a given subtype arise: such forms are sometimes designated A’, B’, F’, etc. It is increasingly necessary to have sequence data from both gag and env for comparing sequences when a new form or subtype is being claimed.”162

By the middle of this year “at least ten” (A-J) prevalent major (M) and a low prevalence, O, HIV-1 genotypes were described and new genotypes are still reported.9, 161 According to researchers from the Henry M. Jackson Foundation Research Laboratory and Division of Retrovirology, Walter Reed Army Institute, USA, “The great majority of genotypic consignments for HIV-1 are based on subgenomic sequence segments, typically encompassing 2% to 30% of the genome”, and not by comparisons of the whole genome. This is because, “it remains impractical to obtain full length genomic sequences of HIV-1 isolates as a routine genotyping method, due to the low abundance of HIV-1 proviral DNA in clinical samples and virus cultures on PBMC substrate, and to the relative inefficiency of the polymerase chain reaction for amplification of HIV-1 subtypes.”163 The designation Human Immunodeficiency Virus Type-1 (HIV-1) encompassed an unanticipated complexity of viral forms.146 According to researchers from the Los Alamos National Laboratory, “while a subtype designation based on a gene or gene fragment may be correct, recombination may have occurred. Therefore, care should be taken to not overinterpret the data obtained from one gene or fragment.”164 The designation Human Immunodeficiency Virus Type-1 (HIV-1) encompassed an unanticipated complexity of viral forms.146 According to researchers from the Los Alamos National Laboratory, “while a subtype designation based on a gene or gene fragment may be correct, recombination may have occurred. Therefore, care should be taken to not overinterpret the data obtained from one gene or fragment.”164
lations of closely related genomes, referred to as a quasispecies. These include:

(a) Eigen and his colleagues developed the quasispecies model to describe the distribution of self-replicating RNAs. However, the “HIV RNA”, is said not to be a self replicating RNA, but replicates through a DNA intermediate.

(b) The self-replicating RNA of the RNA viruses appears to demonstrate remarkable stability in some situations. The type 3 Sabin poliovirus vaccine differed from its neurovirulent progenitor at only 10 nucleotide positions after 53 in vitro and 21 in vivo passages in monkey tissues. In 1977, H1N1 influenza A virus reappeared in the human population after 27 years of dormancy with sequences mainly identical to those of the 1950s virus. Although Eigen’s quasispecies model has been used to describe the genome of RNA viruses, even 1% sequence differences in these genomes are considered to represent “extreme variability”. Many selective forces may stabilize virus populations. These stabilizing factors may include the need for conservation of protein structure and function, RNA secondary structure, global variation in DNA, and the level of expression and stability of RNA-Protein complexes. Changes can be subject to selective pressures. Recently, remarkable conservation of certain protein domain sequences has been observed between completely unrelated RNA viruses. It is possible then to describe the “HIV DNA” even if it has variation of 10%, not to mention 20 or 30 or 40% as is the case, as a “population of closely related genomes, referred to as a quasispecies”.

(c) Defining the concept of a quasispecies Eigen wrote: “In the steady state that is eventually reached the best competitor, designated as the master sequence, coexists with all mutant sequences derived from it by erroneous copying. We designate this distribution of sequences as quasispecies”. However, to date, nobody has proven that:

(i) there is an “HIV” quasispecies which is ever in equilibrium;
(ii) the “closely related HIV genomes” are derived from a master sequence;
(iii) a master sequence has ever existed.

6.4.4 If the “HIV RNA” stretch is the genome of an exogenous virus which infects individuals with AIDS or those at risk, then this RNA (or cDNA) should be present in fresh uncultured tissue from all these individuals and in nobody else. Furthermore, if in these individuals there is massive HIV infection, as some of the best known HIV experts claim, Southern blot hybridisation should be more than sufficient to detect it. The first such study was conducted by Gallo and his colleagues in 1984. Using a Southern blot hybridisation technique they tested many tissues from AIDS patients, including lymph nodes. Summarising their finding they wrote, “We have previously been able to isolate HTLV-III from peripheral blood or lymph node tissue from most patients with AIDS or ARC”. They isolated it from 50% of patients referred to them by their colleagues. However, as lymph nodes are usually not detected by standard Southern blot hybridization of these same tissues and, when it is, the bands are often faint...the lymph node enlargement commonly found in ARC and AIDS patients cannot be due directly to the proliferation of HTLV-III-infected cells...the absence of detectable HTLV-III sequences in Kaposi’s sarcoma tissue of AIDS patients suggests that this tumor is not directly infected by HIV-1. However, when these same tissues and, when it is, the bands are often faint...the lymph node enlargement commonly found in ARC and AIDS patients cannot be due directly to the proliferation of HTLV-III-infected cells...the absence of detectable HTLV-III sequences in Kaposi’s sarcoma tissue of AIDS patients suggests that this tumor is not directly infected by HIV-1 infection” were examined using an HIV gag probe. “The anti-sense riboprobe hybridized to cells known to be infected with HIV-1. It hybridised to HIV-1-infected A3.O1 cells as well as splenic and renal lymphocytes obtained at autopsies from patients known to have AIDS. The probe did not, however, hybridize to neurons in the brain sections from 10 patients with AIDS...Surprisingly, when we applied the control sense HIV-1 gag probe to the brain sections from patients with AIDS, we observed specific hybridization to neuronal cells. Similarly, when brain sections from five individuals not infected with HIV-1 were examined, the HIV-1 sense probe detected transcripts in neuronal cells. Our Northern blot analysis confirmed these results and demonstrated the presence of a 9.0-kb polyadenylated transcript in both tissues.”

6.4.5 In the second half of the 1980s, in order to rescue the concept of an “HIV genome”, the HIV experts made extensive use of a newly discovered process known as the polymerase chain reaction (PCR). Although the PCR is a very useful tool in molecular biology there are many problems associated with its use in studying the “HIV genome”:

(a) PCR is an exponential amplification technique, where each round of amplification produces twice as many copies as the previous round, leading to a “PCR explosion”.

(b) The PCR method of detecting HIV DNA is based on the assumption that the presence of HIV DNA implies that the virus is present, but this assumption has not been proven to be true.

(c) Some of the best known HIV experts including Montagnier, Blattner and Gilderblom agree that the pol and gag genes may be highly conserved between subtypes of virus (see 5.6). In a paper published in 1996 by Gallo and colleagues, “Retrotransposons evolved in a variety of organisms ranging from protozoa to human beings. In these elements, RT genes are linked to genes that code for polypeptides with the potential to self aggregate and to form core particles. These proteins are the equivalents of the retroviral capsid proteins usually designated group-specific antigens (gag)”. Retrotransposons can be transcribed by RNA polymerase II and serve as precursors of retroviruses. Retroviruses differ from retrotransposons by the presence of at least one additional coding region, the envelope (env) gene. In 1984, Gallo’s group reported that the “HIV genome” hybridised with the “structural genes (gag, pol, and env) of both HTLV-I and HTLV-II. Obviously, the finding of a positive hybridisation is insufficient proof. The lack of detectability of the HIV gag or pol probe is no proof for the existence of the “HIV genome”;

In fact, at present evidence also exists which shows the presence of “HIV” sequences in non-infected tissues:

(i) although it is no longer accepted that HIV is transmitted by or is present in insects, in 1986 researchers from the Pasteur Institute found HIV DNA sequences in tsetse flies, black beetles and ant lions from Zaire and the Central African Republic;

(ii) in 1985 Weiss and his colleagues reported the isolation, from the mitogenically stimulated T-cell cultures of two patients with common variable hypogammaglobulinaemia, a retrovirus which “was clearly related to HTLV-III/LAV”. Evidence included positive WB with AIDS antigen and hybridisation of brain tissues;

(iii) DNA extracted from thyroid glands from patients with Grave’s disease hybridised with the “entire gag p24 coding region” of HIV;

(iv) In a study designed to address the question whether the neuronal cells of patients with AIDS dementia complex are infected with HIV, “the brains from 10 patients with AIDS and neurological evidence of viral encephalitis and the brains from five patients without HIV infection” were examined using an HIV gag probe. “The anti-sense riboprobe hybridized to cells known to be infected with HIV-1. It hybridised to HIV-1-infected A3.O1 cells as well as splenic and renal lymphocytes obtained at autopsies from patients known to have AIDS. The probe did not, however, hybridize to neurons in the brain sections from 10 patients with AIDS...Surprisingly, when we applied the control sense HIV-1 gag probe to the brain sections from patients with AIDS, we observed specific hybridization to neuronal cells. Similarly, when brain sections from five individuals not infected with HIV-1 were examined, the HIV-1 sense probe detected transcripts in neuronal cells. Our Northern blot analysis confirmed these results and demonstrated the presence of a 9.0-kb polyadenylated transcript in both tissues.”

6.4.6 As mentioned earlier, that is, HTLV-I and HTLV-II. However, at present even Gallo admits that the human endogenous proviral sequences “comprise about one percent of the human genome”;
There is no doubt that PCR can ‘amplify a DNA-needle into a DNA-haystack’ or even PCR cannot perform miracles. In a review of Neville Hodgkinson’s book, AIDS, The Failure of Contemporary Science: How a Virus That Never Was Deceived the World, Sir John Maddox wrote, “the virus that never was has been made more tangible” early in 1995 when “it became apparent that even in the earliest stages of infection by HIV, the virus is far from dormant.” Maddox is referring to two papers published in Nature in 1995. One by Ho et al. where the authors claim that even in patients who have not received antiviral treatment the “plasma viral levels ranged from...15 X 10^3 to 554 X 10^4 virions per ml” the other by Wei et al. in which it is claimed that the “plasma viral RNA levels in the 22 subjects at baseline ranged from 10^6 to 10^7 molecules per ml.” The conclusion of the study “suggests that virus expression per se is directly involved in CD4+ cell destruction. The data do not support an ‘innocent’ bystander mechanism of cell killing whereby uninfected or latently infected cells are indirectly targeted for destruction by absorption of virus proteins or by autoimmune reactions.”

These claims raise two obvious questions:

(i) “The majority of exogenous pyrogens are microorganisms, their products or toxins”, and “endogenous pyrogens are polypeptides produced by a large variety of nucleated host cells including monocytes/macrophages” and “lymphocytes, endothelial cells, hepatocytes, epithelial cells, keratinocytes, and fibroblasts, as well as other cells...generally in response to initiating stimuli triggered by infection or inflammation”. In addition, “many endogenous products result in the release of endogenous pyrogens, thereby causing fever. Such endogenous substances include antigen-antibody complexes, complexes with complement, complement cleavage products, steroid hormone metabolites, bile acids and some cytokines”. Since “the virus [“HIV”] is replicating 24 hours a day and from day one”, and “2x10^6 CD4+ cells [are] produced and destroyed each day”, and fever and “fever-inducing endogenous substances are triggered with the generation of pyrogenic cytokines, such as IL-1, TNF-alpha, or IL-6” it is indeed surprising that such “massive” infection and cellular destruction may remain largely, if not totally, asymptomatic for prolonged periods of time in HIV seropositive individuals.

(ii) “The 24 hours ‘HIV’ infection, why is it not detected by standard hybridisation procedures and why, in order to detect such ‘massive’ infection, did not the authors use PCR which can ‘amplify a DNA-needle into a DNA-haystack’ or even nested PCR but were obliged to define “Viral RNA” with novel assays, “modified branched DNA (bDNA) or RT-PCR assay and confirmed by QC-PCR” for which no data are given.

One of the many problems associated with the Ho and Wei studies and the methods they employ is illustrated in a presentation at the Xth International Conference on AIDS. Researchers from the Medical School, Camden, New Jersey took a single plasma sample at the XIth International Conference on AIDS. Researchers from the Pasteur Institute. Out of 30 positive samples, “34 were gag positive (90%) whereas env and LTR were detected in fewer cases 24 samples (63%) and 18 samples (38 positive samples, “34 were gag positive (90%) whereas env and LTR were detected in fewer cases 24 samples (63%) and 18 samples (22% of the 28 replicates, either the gag or an env sequence was amplified but not both”.

A PCR study of 40 individuals using primers from the LTR, gag and env regions was performed by French researchers including researchers from the Pasteur Institute. Out of 30 positive samples, “34 were gag positive (90%) whereas env and LTR were detected in fewer cases 24 samples (63%) and 18 samples (78% of the 28 replicates, the gag or env sequence was amplified but not both”.

One of the problems associated with the Ho and Wei studies and the methods they employ is illustrated in a presentation at the Xth International Conference on AIDS. Researchers from the Medical School, Camden, New Jersey took a single plasma sample from a patient with “a CD4 cell count of 123 cells/cmm” and divided it into ten aliquots. The RNA from each sample was reverse transcribed into cDNA and the cDNA was then amplified with an internal control DNA (mimic) using gag primers...cDNA was also pooled from the initial 10 patients into ten aliquots. The RNA from each sample was reverse transcribed into cDNA.

According to Maddox and Wain-Hobson both Ho and Wei and their colleagues were able to reach their startling conclusions only after a decade of HIV research because they teamed up with mathematicians and because they were able to use “new techniques for assaying the low levels of virus involved”! (italics ours). It is ironic then that the strongest criticisms of these studies have emanated from mathematicians such as Frank Buianoukas from the Department of Mathematics, and Computer Science, City University of New York, USA and Mark Craddock, School of Mathematics and Statistics, The University of Sydney, Australia. “What is this viremia of billions of RNA particles that can only be seen with an undetectable branch-PCR or PCR but not with a functional infectivity test?” “My question is this: just what exactly will it take to get people doing HIV research to turn away from such (unproven) methods, arcane speculations about molecular interactions etcetera etcetera and ask themselves ‘do any of us have the faintest idea what we are doing’?!” One can argue that criticism of the Ho and Wei papers by individuals from the HIV/AIDS dissmantlement movement is not to be unexpected but it is unheard of for one group of HIV experts to criticise another and it happens only with the Ho and Wei studies. In July 1995, as a result of “misgivings” about the claims of Ho and Wei and their colleagues, “two dozen AIDS researchers congregated in Berkeley. California...to challenge the establishment, swap copies of their own manifestos, and enjoy the bonhomie of hanging out for two days with fellow ‘alternative thinkers’ who concluded that Ho et al and Wei et al. were short on compelling evidence that their ideas were correct.”

According to researchers from the Walter Reed Army Institute of Research, “the extensive use of the polymerase chain reaction (PCR) to recover HIV-1 proviral DNA has favoured analysis of the short amplicons that are most efficiently recovered by this technique.” In fact, amplification results obtained with primers for different genes from one subtype are not in complete agreement. For example, in the first “HIV” PCR two primer pairs to amplify the gag gene were used and it was found that “some samples scored positive with only one of the two primer pairs”. It is said that in the USA and Europe individuals are almost exclusively infected with subtype B. Yet researchers from the University of Edinburgh found that “The results from two different centres are in general agreement. In 5 of the 28 replicates, either the gag or an env sequence was amplified but not both.” A PCR study of 40 individuals using primers from the LTR, gag and env regions was performed by French researchers including researchers from the Pasteur Institute. Out of 30 positive samples, “34 were gag positive (90%) whereas env and LTR were detected in fewer cases 24 samples (63%) and 18 samples (47%) respectively...10 of 40 samples were positive with three primer pairs, 16 with two primer pairs and 11 with only one primer pair”. Such discrepancies may be due to:

(i) “a false-positive reaction", which the authors themselves suggest but which they say is unlikely.

(ii) “the known genetic variability of HIV”. If this is the case then one cannot talk of the “HIV genome” as being a unique molecular entity. Indeed, if such variability is entertained then it may be only the lack of an immense variety of primer pairs that prevents all of Homo sapiens from being “infected with HIV”.

(iii) variable number of tandem repeats (VNTR)

(iv) “variable number of tandem repeats (VNTR) on the chromosome.

(v) “variable number of tandem repeats (VNTR) on the chromosome.

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possible to say what the “HIV DNA” sequences are, it follows that it is impossible to say what the “HIV DNA” sequences are, it follows that it is impossible to say what the “HIV DNA” probes are HIV, or DNA probes of an endogenous retrovirus or even an exogenous retrovirus HTLV-I; (iii) in a DNA (RNA) sample the primers bind only to HIV sequences and not to other HIV homologous or non-homologous sequences. Again, no such data exists.

Furthermore, given the facts that:
(a) “about one percent of the human genome” consists of endogenous retroviral sequences;
(b) homologies exist between the genes of endogenous and exogenous retroviruses, especially in the gag and pol genes, and between the gag and pol gene sequences. Again, no such data exists.

Even if (i)-(iii) are proven one must still determine the specificity of the PCR reaction, that is, show that no positive results are obtained in individuals who are not infected with HIV. This can only be determined by using HIV isolation as an independent gold standard, that is, by comparing PCR with the procedures listed above (see 6.1). This has not been done, a fact accepted by one of the best known HIV/AIDS researchers, William Blattner “One difficulty in assigning the specificity and sensitivity of human retroviruses [including HIV] is the absence of an accurate gold standard”. The finding of positive PCR in eosinophils has been interpreted as “infected” children which were analysed retrospectively 12 (6.7%) “cleared HIV infection”. Each child had at least two positive PCR results at two separate time points in the first year, followed by numerous (up to seven) negative PCR results. For PCR the investigators used primer pairs for the gag, pol, and env gene regions; and the test was considered positive “if at least two genes were amplified”. Commenting on their results the authors wrote, “Three different reactions with separate amplification circuits were used for DNA extraction, PCR-buffer preparation, amplification and blotting. Amplicons were never transferred in the area reserved for unamplified sequences. Thus, positive PCR results are unlikely to be due to contamination... Nevertheless, as our PCR assays are performed on unmanipulated cells, the presence of culture contamination leading to false positive PCR results is impossible... We therefore consider that the probability of repeated contamination on successive samples from the same child is rare”. The authors “could not find any correlation between either neutralizing or antibody-dependent cellular cytotoxicity-mediating antibodies and HIV clearance”. Of 139 children born to HIV positive mothers who were never exposed to HIV, none seroconverted or developed p24 antigenemia and “all of the infections must always include appropriate controls to ensure that no endogenous sequences contribute to positive signals. As previously noted, HIV unique primers corresponding to the highly conserved reverse transcriptase gene of HIV are not specific for HIV infection in the PCR amplification of HeLa DNA even at annealing temperatures around 60°... Another practical concern is that the use of PCR for determining the possible retroviral etiology of a variety of human diseases may be complicated by endogenous retroviruses. Even if cDNAs are used for PCR templates, the transcriptional activities of endogenous sequences considered to be inactive may be activated this year, where he discusses the laboratory diagnosis of “HIV infection”, Philip Mortimer wrote, “Other diagnostic methods, e.g. p24 antigen testing, and proviral DNA and RNA amplification exist, but these innovations in HIV diagnosis need to be matched against the anti-HIV test and should be rejected unless they fulfill a need that antibody testing fails to meet”. According to researchers from the University of London, “The use of polymerase chain reaction (PCR) for the diagnosis of HIV infection is becoming more widespread and although not yet entirely reliable compared with serology, has been of special value in HIV-seronegative intravenous drug users”. If PCR needs to be matched against the “HIV” antibody test because it is less reliable than serology then given the fact that at present there is no evidence which shows that a positive “HIV” antibody test is proof of HIV infection, one has no choice but to agree with Shoebridge et al that “until further molecular and biological studies are carried out, it will be unsure as to what detection of HIV-1 DNA, even when shown to be HIV-1 really means”. In analysing the “HIV molecular biology has helped to solve the enigma of Sir J ohn Madox, “Is there a danger, in molecular biology, that the data will eventually prove an encumbrance? Part of the trouble is that excitement of the chase leaves little time for reflection. And there are grants for producing data, but hardly any for standing back in contemplation”.

In a study of 327 health care workers exposed by needlestick injuries to the “human immunodeficiency virus”, four had “one or more positive” PCR tests. An additional seven had “an indeterminate PCR test result with an initial sample that gave a positive PCR result... “none seroconverted or developed p24 antigenemia” and “all of the subjects remained healthy”. While the evidence for such occurrence in adults is sporadic, it is much more often reported in children. However, PCR is not used for routine diagnosis of HIV infection in adults and rarely, if ever, is repeated. Unlike in adults, PCR is very often repeated in children, thus being the case because “diagnosis” is “complicated by persistence of poorly required material antibody”. By 1995 numerous studies in children revealed the conversion of a positive PCR to negative. One of the most recent reports was published in 1995 by French researchers. In a six year cohort of 188 “infected” children which was analysed retrospectively 12 (6.7%) “cleared HIV infection”. Each child had at least two positive PCR results at two separate time points in the first year, followed by numerous (up to seven) negative PCR results. For PCR the investigators used primer pairs for the gag, pol, and env gene regions; and the test was considered positive “if at least two genes were amplified”. Commenting on their results the authors wrote, “Three different reactions with separate amplification circuits were used for DNA extraction, PCR-buffer preparation, amplification and blotting. Amplicons were never transferred in the area reserved for unamplified sequences. Thus, positive PCR results are unlikely to be due to contamination... Nevertheless, as our PCR assays are performed on unmanipulated cells, the presence of culture contamination leading to false positive PCR results is impossible... We therefore consider that the probability of repeated contamination on successive samples from the same child is rare”. The authors “could not find any correlation between either neutralizing or antibody-dependent cellular cytotoxicity-mediating antibodies and HIV clearance”. Of 139 children born to HIV positive mothers who were never exposed to HIV, none seroconverted or developed p24 antigenemia and “all of the
CONCLUSION—The present data do not prove the existence of a unique molecular entity “HIV DNA” which constitutes the genome of a unique, externally acquired retrovirus, HIV. Neither is there any proof for the existence of an “HIV quasispecies”. Nor is it possible to say what exactly the different “HIV DNAs”, the probes and primers derived from these DNAs and the sequences in the cellular DNA with which they hybridise, represent.

7. “Isolation of HIV: The existence of a retrovirus HIV predicts that HIV can be isolated from the chromosomal DNA of infected cells. This prediction has been confirmed as follows: Full-length HIV-1 and HIV-2 DNAs have been prepared from virus-infected cells and cloned in bacterial plasmids (Fisher et al., 1985; Levy et al., 1986; Barnett et al., 1986). Such cloned DNA can be transcribed and cellular contaminants that copurify with virus purified by conventional density gradients. Indeed, these clones are even free of genomivc HIV RNA. Infectious HIV-1 and HIV-2 DNA clones productively infect human cells to initiate HIV replication (Fisher et al., 1985; Levy et al., 1986; Barnett et al., 1993). Such infected (“transfected”) cells can be isolated and provided with a plasmid containing a transcriptional promoter and HIV-specific antigens (Fisher et al., 1985; Levy et al., 1986), have diameters of 100 nm under the electron microscope (Fisher et al., 1985), as expected for retroviruses”.

7.1 Before the cited evidence is discussed in detail, to avoid misunderstanding, it will be helpful to define some terms including cloning of DNA, transfection and virus cloning, as well as to present evidence that must be presented to claim proof of these phenomena:

Plasmid—freely replicating, circular chromosomal elements present in bacteria. They duplicate independently of the main chromosomal element and are frequently used to “carry” a DNA fragment into a cell.

DNA cloning—the production of identical copies of a DNA fragment, any DNA fragment, from an ancestral DNA fragment by splicing it into a suitable cloning vehicle, for example, a bacteriophage or plasmid.

Transfection—the introduction of exogenous DNA into cells and its ability to replicate and express itself in these cells, that is, transcription of DNA into RNA, translation of RNA into proteins. The genetic material does not have to be of viral origin and transfection can be achieved by various methods. As far back as 1969 it was known that these methods may include “infection of cells with bacteria and viruses, formation of hybrids of two cell types by fusion, transplantation of isolated single nuclei in eggs and embryos, microinjection of nuclei and mitochondria fractions, and picnomic uptake of purified DNA”.

In that year Margit Nass from the University of Pennsylvania, taking advantage of “the phagocytic properties of mouse fibroblasts (L cells) grown in suspension culture” demonstrated that, “Mouse fibroblasts (L cells) in suspension culture incorporated isolated chloroplasts of spinach and African violets and isolated mitochondria of chicken liver...Green cells divided like normal cells. Green chloroplasts were followed for five cell generations or five days, at which time hybrid cells were greatly outnumbered by nongreen progeny cells.” By 1989 it was realised that the delivery of DNA into cells could be facilitated by polycationic reagents such as poly-DEAE dextran and poly-lysine. It was then realised that the delivery of DNA into cells from fibroblasts transfected in vitro under optimal conditions. The extent of expression from expression was readily detected in all cases, and no special delivery system was required for these effects. The extent of expression from expression was readily detected in all cases, and no special delivery system was required for these effects.

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mid. “The presence of HTLV-III sequences was demonstrated by Southern blot analysis” using “the molecular clone IBH-10, “an incomplete viral clone of HTLV-III”. “A 10-kb band, corre-
sponding to unintegrated linear virus, was detected in undigested DNA samples prepared 14 days after transfection. Digestion with XbaI revealed three distinct band at 11, 10 and 5.2 kb...these bands probably do not represent “Sequin” fragments...but are likely to be forms of unintegrated HTLV-III respectively. Digestion with HindIII, an enzyme which cuts the HTLV-III genome of pHXB-2D six times, yield-
ed bands at 4.5, 2.0 (doublet), 1.7 and 0.6 (a doublet)...This restriction pattern is clearly different from that of H9/HTLV-IIB...High relative molecular mass ‘smears’ were not observed when DNA was digested with BamHI. Therefore we had no direct evidence that transfected HTLV-III DNA is integrated in the host cell genome...In time-course experiments (Fig. 36), DNA isolated from a single culture 6, 11, 14, and 31 days after transfection with pHXB-2D, was digested with BamHI and analysed for HTLV-III sequences. Six days after transfection an 8.6 kb DNA fragment was detected as a faint band; 18 days after transfection, this cell line was able to produce virus. Digestion after transfection to the 8.6 kb fragment...No HTLV-III sequences were detect-
ed 31 days after transfection”. Despite these findings, the time-
course experiments were interpreted “as evidence that cells originally transfected with pHXB-2D are able to produce fully infectious virus which is then transmitted within the culture”.

(c) The pHXB-2D transfected umbilical cord lymphocytes were reacted with “monoclonal antibodies against the HTLV-III-gag-related proteins p24 and p15...maximum expression was observed 15 days after transfection, when 4-11% and 5-9% of cells were reactive with antibody to p15 and p24, respectively (data not shown)...In compar-
ison, among H9/HTLV-III cultures, a much larger proportion of cells (70-90%) was positive for p24 and p15”. In addition to the many prob-
lems associated with the interpretation of a positive antibody/antigen
reaction, especially with umbilical cord cells and the gag antigens
(antibodies), as proving HIV infection, it is also interesting to note that:
(i) maximum antibody/antigen reactions preceded maximum
reported RT activity and hybridisation bands.
(ii) mention should be made of the anti-correlation reactivity with the pSV2-neo transfected culture but “cord blood cells removed 18 days after transfection with pCH-Igpt (HTLV-I clone) were not labelled by these antibodies”. However, if as Gallo claims:
(a) the gag genes of HIV and HTLV-I are homologous;
(b) there is cross-reactivity between the p24 proteins of the
HTLV-I and HIV-2, then the reported finding that the “monoclonal antibodies against the HTLV-III-gag-related proteins” did not react with the pCH-Igpt trans-
sected cells is inexplicable.

Their immunological findings led them to write: “The finding that, at any stage, only a minor population of the transfected cells are apparently infected by the virus (<15% express viral proteins) sug-
gests that the cytopathic effects may not result solely from direct viral infection”. However, if the dramatic fall of viable cells in the pHXB-2D transfected cultures where only a minority of cells are “infected” is caused either directly or indirectly by “the clone of HTLV-III with bio-
logical activity” (cytopathic effects), why are such effects not also
observed in the H9/HTLV-III cell line where a much higher percent of cells are “infected” and where problems with “expression of antigens” and “late proteins gp160, gp120, gp41, and the gag proteins stall...”?

7.4 In 1993 Barnett, Levy and their colleagues published a paper entit-
led “Distinguishing features of an infectious molecular clone of the
human immunodeficiency virus (HIV-2).” This paper described a new
infectious molecular clone of HIV-2. The authors reported that “The virus recovered from
HUT-78 cells showed cytopathic effects (fusion, balloon degenera-
tion) typical of AIDS retroviruses”.

If the cytopathic effects are caused by an HI virus which appeared as a result of cloning then Levy et al managed to prove an effect of
HIV on HUT-78 (H9) which to date nobody else has managed to
demonstrate. (It is true that in 1986 nobody apart from Gallo and his colleagues knew that HUT78 is actually HT (H9)).
HIV strains. The proviral DNA sequence of UC1mc was found to be 10,271 bp long, and its overall genetic structure appeared to be similar to that of other sequenced HIV-2 strains... By sequence analysis, UC1mc appeared to diverge substantially from most other HIV-2 strains. The differences were most noticeable in the very low percent-ages of identity of the amino acids sequences of Env; viral regulatory proteins were found to be conserved for many years. Silvestri and Vpr. The divergence of UC1mc was more subtle but nevertheless sig-nificant in the generally more conserved Gag and Pol proteins (ital-ics ours).

7.5 COMMENTS

Absolutely necessary to claim cloning of a retrovirus, HIV. Nor was it possible for them to do so. To molecularly clone a retrovirus first one must obtain the retroviral RNA and this can only be obtained by isolat-ing the retrovirus. NO ISOLATION NO CLONING. However, to date not only has no researcher isolated a unique retrovirus from fresh tissues of patients with AIDS, but also no researcher has cultured infectious material from these patients but neither has any researcher proven the exist-ence of particles, viral or non-viral, which satisfy the principal mor-phological and physical properties of retroviruses.146 Fisher et al, Levy et al and colleagues, by various means, but with no proof that it belonged to a particle, any particle, selected fragments of DNA, no two of which were the same either in composition or length and called it "HIV DNA" (see 6.2). Subsequently one or two of these "HIV DNA" were introduced to the cells using well known techniques by which it is possible to introduce any DNA, viral or non-viral, into cells. Irrespective of what is meant by "HIV DNA", given the techniques they used, it is highly probable that they succeeded. However, proof can only be claimed if sequences of the "HIV DNA" both before and after cloning into the cells and none of these groups did so. The only evidence present-ed by the above workers to this effect and indeed to virus cloning was: (a) The detection in cell cultures of RT activity (transcription of A(n).dT15); (b) The finding in cells of proteins ("the envelope proteins gp160, gp120, and gp41", and gag proteins of molecular weight 55K, 25K and 16K) which react with antibodies to p24 and/or with sera from AIDS patients. However, thus far, nobody has proven that any of the above proteins which are present in cell extracts and which may react with AIDS patient sera are actually coded by the "HIV" env and gag open reading frames (see 5). Neither are the presence of viral-like particles in the culture supernatants nor transcription of A(n).dT15 proof for the exist-ence of HIV or of any retrovirus endogenous or exogenous (see 3.0). Even if there was proof that the particles were actually retroviral and that reverse transcription of A(n).dT15 was induced by a retroviral enzyme, the proteins were retroviral proteins and the antibodies were specifically directed against such proteins, their finding in cell cultures is not proof of retrovirus cloning. All of these phenomena may be caused by an endogenous retrovirus, especially if one considers the type of cells used, leukemic and umbilical cord lymphocytes, and the conditions, chemical stimula-tion and co-culture techniques. According to Kurth and his colleagues, "indirect evidence has accumulated over the past years that some endogenous proviral loci must also be expressed in humans." Expression of retroviral information was also suggested by the demonstration of reverse transcriptase activity and by the detection of antigens cross-reactive with animal retroviral antigens in a variety of human cells and tissues.

AIDS patients sera contain antibodies directed against many self and non-self antigens including lympho-cytic leukemia virus (i.e. HTLV-I). Following the "discovery" of "HIV DNA" in 1982, one of the main questions of "The viruses in all of us", that is, endogenous retroviruses.175 In a paper written by the above workers to this effect and indeed to virus cloning was: (a) The detection in cell cultures of RT activity (transcription of A(n).dT15); (b) The finding in cells of proteins ("the envelope proteins gp160, gp120, and gp41", and gag proteins of molecular weight 55K, 25K and 16K) which react with antibodies to p24 and/or with sera from AIDS patients. However, thus far, nobody has proven that any of the above proteins which are present in cell extracts and which may react with AIDS patient sera are actually coded by the "HIV" env and gag open reading frames (see 5). Neither are the presence of viral-like particles in the culture supernatants nor transcription of A(n).dT15 proof for the exist-ence of HIV or of any retrovirus endogenous or exogenous (see 3.0). Even if there was proof that the particles were actually retroviral and that reverse transcription of A(n).dT15 was induced by a retroviral enzyme, the proteins were retroviral proteins and the antibodies were specifically directed against such proteins, their finding in cell cultures is not proof of retrovirus cloning. All of these phenomena may be caused by an endogenous retrovirus, especially if one considers the type of cells used, leukemic and umbilical cord lymphocytes, and the conditions, chemical stimula-tion and co-culture techniques. According to Kurth and his colleagues, "indirect evidence has accumulated over the past years that some endogenous proviral loci must also be expressed in humans." Expression of retroviral information was also suggested by the demonstration of reverse transcriptase activity and by the detection of antigens cross-reactive with animal retroviral antigens in a variety of human cells and tissues.

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8. "IDENTIFICATION OF HIV"

8.1 The existence of HIV predicts that infected cells contain a unique, virus specific DNA of 9150 nucleotides which cannot be detect-ed in the DNA of "uninfected" human cells and tissues. The RD cell line used by Levy is a human rhabdomyosarcoma cell line derived from a normal human embryo with a high frequency of reverse transcriptase activity and to release retroviral-like particles.227 For cloning, one assumes that the "HIV DNA" is indeed retroviral, for which there is no proof. It cannot be "HIV DNA" because neither in the HUT78 cell line nor in any of the H9 cell lines was there any evidence of reverse transcriptase activity and by the detection of cDNA which is the genome of a unique retrovirus particle, HIV-1, which can only be obtained by isolating the retroviral particle. At present there is no such proof. Fisher et al and Levy et al selected a portion of the RNA which from the supernatant of "infected" HUT78 cells banded at 1.16g/ml or had a certain length, reverse tran-scribed it and called it "HIV-1 DNA" (see 6.2.1.5). However, since neither they nor anybody else before or after them has shown that this RNA (cDNA) was even the constituent part of a particle, any partic-le retroviral or otherwise, the claim that the DNA is "Full length HIV DNA" is, at the very least, difficult to substantiate. In the cell extracts of "transfected" cells Fisher et al and Levy et al found some proteins with molecular weights similar to the "HIV proteins" which reacted with AIDS patient sera. They also found reverse transcription of A(n).dT15 in the cell supernatant but presented no evidence that the proteins or the RT were constituents of a particle, viral or otherwise, and thus they cannot claim that they have proven that the "transfected" cells "produce particles that contain reverse transcriptase, HIV specific antibodies". Although Fisher and colleagues had an electron micrograph showing virus-like particles in the culture supernatant, they did not prove that the particles were indeed retroviral particles, or even that they had some of the basic morphological and physical features of animal retroviruses and thus they "could reflect non-viral material altogether." Fisher et al, Levy et al and Barnett et al did not start with RNA (cDNA) proven to be the RNA of a retrovirus and did not obtain retro-viral particles proven to contain the same RNA, a most basic require-ment for cloning. In fact, given their evidence they cannot even claim transfection of cells with a DNA, viral or non-viral.
insects (see 6.4.4). It is a fact that:

(a) hybridization of nucleic acids of "exogenous retroviruses" "from different species gives a pattern which is the same as the phylogenic relatedness among their natural hosts",229 a relationship which led retrovirologists including Gallo to conclude that exogenous retroviruses are "derived from cells genes".

(b) Endogenous retroviruses has been "shown" using hybridization probes derived from endogenous and exogenous animal retroviruses.

If this is the case and if "HIV DNA" is the genome of an exogenous human retrovirus, the non-infected human genome should contain sequences which will hybridise with "HIV DNA" probes. There can be two reasons why such findings have not been seen:

(a) Most HIV researchers ignore one of the most fundamental requirement of basic experimental research, that is, controls. In the rare instances where controls are used, they are not suitable (see 6.1). In the 1970s, Gallo, Gillespie and their colleagues were saying that the success of the "hybridization assay appears to depend on the biological state of the cells",125,228 In a large study published in 1975 entitled "Relationship between Components in Primate Rna Tumor Viruses and in the cytoplasm of Human Leukemia Cells: Implications to Leukemogenesis", the aim was to show that human leukemia cells but not normal cells have properties associated with retroviruses including retroviral genomic sequences. It was reported that "The human leukemic blood cell cytoplasmic particles is that contains reverse transcriptase activity capable of synthesizing DNA in vitro, using endogenous RNA DNA as template and primer. This endogenous activity has been used to learn about the nature of the particle itself. Many intracellular cytoplasmic particles or organelles (described generally in Table 8) can carry out endogenous DNA synthesis in vitro. These include mitochondria, small cytoplasmic particles of low density, 1.10-1.16 g/cc in sucrose density gradients, and small cytoplasmic particles of higher density, 1.17-1.19 g/cc in sucrose density gradients...Small particles have been detecting in the cytoplasmic fraction of phytohemagglutinin-stimulated lymphocytes from normal donors...These particles carried endogenous DNA sequences, and were RNA population contained sequences related to genomes of RNA tumor viruses...". Viral-related sequences were found in patients with several types of leukemia, including AML, CML, MCL-A and CLL...Attempts to detect viral sequences in RNA of leukemic cells by hybridizing DNA synthesized by animal viruses to RNA isolated from cytoplasmic small particles (the reciprocal hybridization experiment) in our hands fails to find differences in sequences in RNA of leukemic and dividing normal [PHAs stimulated] human peripheral white blood cells. It has been reported that others that radioactive DNA probes synthesized by MuLV, hybridize to cytoplasmic RNA from leukemic, but not normal white blood cells. A difference between our experiments and those previously reported is that the normal human cells used as a source of RNA are actively dividing which were not in the previous studies".

(b) The "HIV RNA" is not the genome of either an exogenous or an endogenous retrovirus or even the transcribed DNA fragment present in un-"shocked" cells.

8.1.2 Most of the positive results in "uninfected cells" have been found by using probes and primers for one or at most two genes or even gene fragments. The "great majority" of HIV studies, encompass "2% to 30% of the genome". However, finding fragment of a gene or even a gene is not proof for the existence of the HIV genome.

8.1.3 Montagnier and his colleagues reported the "HIV DNA" to be 9 ± 1.5 Kb whereas Gallo and his colleagues reported that "The overall length of the HTLV-III provirus is approximately 10 kilobases". In Levy and colleagues' first study of the "HIV genome", the "broad band (>15 kb) represents provirus integrated into host cell DNA". In 1995, Pasteur researchers reported that "The complete 9193-nucleotide sequence of the probable causative agent of AIDS, lymphadenopathy-associated virus (LAV), has been determined. The sequence 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". In the same year, Gallo and his colleagues reported their results on the "HIV" nucleotide sequences using clone BH10 but also added, "The sequence of the remaining 102 bp of the HTLV-III provirus, not previously sequenced before (BH10, 1984, RNA primer binding site and a portion of the header sequence) was derived from clone HXB2...Of note is the presence of a fifth open reading frame (nucleotides 8, 344-8991) designated 3' off. present in clone BH8 but truncated in BH10". They concluded, "The complete nucleotide sequence of two human T-cell leukemia type III (HTLV-III) proviral DNAs each have four long open reading frames, the first two corresponding to the gag and pol genes. The fourth open reading frame encodes two functional polypeptides, a large precursor of the 3' major envelope glycoprotein and a smaller protein derived from the HTLV-III provirus is approximately 10 kilobases". In "2% to 30% of the genome". However, finding fragment of a gene even gene fragments. The "great majority" of HIV studies, encompass "8.1.2 Most of the positive results in "uninfected cells" have been found by using probes and primers for one or at most two genes or even gene fragments. The "great majority" of HIV studies, encompass "2% to 30% of the genome". However, finding fragment of a gene or even a gene is not proof for the existence of the HIV genome.

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(c) there are structural and functional abnormalities in the lymphocyte genome of AIDS patients. "AIDS patients have shown increased levels of spontaneous DNA repair synthesis (three times higher), increased quantity of single-stranded DNA breaks (11-18%), decreased ability to repair DNA damage (2-2.5 times lower) compared to healthy persons".
(d) According to Chermann and his colleagues, "Different populations of distinct HIV-1 DNA fragments of highly variable size ranging from 600 bp to full length provirus were present in PBMC from HIV-infected persons... Defective genomes tended to gradually disappear after activation of PBMC by phytohemagglutinin".
(e) According to the HIV experts, the defective genomes are "rescued" by recombination and this recombination is one of the main causes of "HIV DNA" complexity. If this is the case one may ask:
(i) can one exclude the possibility that the 19 “full-length HIV genomes” described so far, even if they all had the same length of 9,150 bp and identical sequences are nothing more than a chance finding among the many molecular species present in the cultures, or even the uncultured lymphocytes, which have nothing to do with a retroviral genome and which appeared as a result of either in vivo or in vitro conditions or both and of natural selection?;
(ii) if there is such a high rate of recombination between the HIV genomes, is it not also possible that the same process takes place between the endogenous retroviral genomes? If this is also the case, how does one know that the 19 “full-length HIV genomes” are nothing more than recombinations between endogenous retroviral sequences and cellular sequences, for example, non-retroviral retroelement?

As has been pointed out, HIV researchers seldom use controls and to date those that have, failed to use appropriate controls, that is, tis-
sues or cultures derived from similarly sick, non-AIDS individuals in which experimental techniques and conditions employed are identical apart from the presence of putative retroviral material. However, if HIV researchers or others capable of mounting such experiments were encouraged to put as much effort as they put into studying “HIV” from lymphocytes of at risk patients into studying lymphocytes from untreated subjects (a) who are exposed to agents (other than “HIV”) and doses similar to those in the high risk groups; (b) which have similar structural and functional abnormalities as lymphocytes from AIDS patients or those at risk; (c) using exactly the same methods and culture conditions as those used by researchers who can one exclude the possibility that in another ten years time these researchers will not be able to report “19 full-length HIV genomes” in these individuals?

8.2. “For example, Jackson et al. have tested blood cells of 409 antibody-negative, but in none of the 131 antibody-negative people (Jackson et al., 1990)."

8.2.2. Apparently up until 1987 Jackson et al considered the detection of RT (reverse transcription) determined by transcription of A(n).dT15 in cultures, synonymous with HIV isolation! However, they had an “isolation rate of 57% in patients with acquired immunodeficiency syndrome”. By 1988 the “reverse transcriptase assay was replaced with the Abbot Laboratories HIV-1 antigen assay, which “primarily detects the p24 core antigen of the virus”. If no supernatant samplings were positive, with the later sampling showing greater activity! “HIV-1 was isolated from the PBMC of 141 (99.3%) of 142 HIV-1 antibody-positive patients”. In their 1990 paper Jackson et al reported that “Between February 1987 and October 1988, peripheral blood mononuclear cells (PBMC) from 409 antibody-negative and 20 antibody-positive for HIV-1 by Western (immuno) blot (56 AIDS patients, 88 patients with ARC, and 265 asymptomatic individuals) were cultured. Using a sensitive technique previously described, the p24 assay noted above, they reported that “HIV-1 can be isolated from 100% (56 of 56) of AIDS patients, 99% (87 of 88) of ARC patients, and 98% (259 of 265) HIV-1 antibody-positive asymptomatic individuals”. Not one of “131 HIV-1 antibody-negative individuals has a positive culture”. Using the same p24 assay (Abbot) they tested the serum from 403 of the 409 individuals. The test was positive in 23/56 (42%) AIDS patients, 31/88 (37%) ARC patients and 44/259 (17%) asymptomatic antibody-positive individuals. For untested reasons a positive serum test is considered proof for the detection of “HIV-1 antibody-negative, but in none of the 131 antibody-negative people (Jackson et al., 1990).”

The fact that in experiments with “serial dilution studies of culture supernatants” the p24 test is more likely to be positive than RT is not proof that the p24 test is “at least 100-fold more sensitive that reverse transcriptase assays”. Sensitivity for HIV can only be measured by the use of HIV isolation as a gold standard.”

(b) There are no scientific reasons and indeed no commonsense reasons why reactions such as reverse transcription or antibody/antigen reactions, even if specific for retroviruses, can be considered proof for “isolation” of HIV. Placenta or heart from laboratory-to-laboratory variation due to differences in techniques and operations” and that “in some reported studies there is no proof that the p24 test is “at least 100-fold more sensitive than reverse transcriptase assays”. Sensitivity for HIV can only be measured by the use of HIV isolation as a gold standard.”

8.2.2. To improve on the p24 assay, the DNA extracted from frozen uncultured PBMC of their seven “antibody-positive culture negative subjects” and “23 healthy heterosexual HIV-1 antibody-negative, culture negative individuals” was assayed by PCR. In addition, “In order to compare the sensitivity and specificity of the two tests, PCR and culture, the PBMC of 59 seropositive and 20 seronegative individuals were tested by both methods inoculated with identical aliquots of blood by using a primer pair, SK38-39, which amplifies a 115-base-pair conserved region of the gag gene (nucleotides 1551 to 1665 of HIV SF23: GenBank accession no. K02007). The amplified product was detected by oligomer hybridization, a technique in which a 24-mer labeled probe (SK19) to the nucleotide 1595 to 1635 gag region hybridizes in solution to one strand of the amplified sequence. The probe-target duplex was then resolved by electrophoresis in 1% agarose gel and autoradiographed”. None of the seronegative individuals was reported to have a positive PCR test. “All initial DNA samples from the seven HIV-1 antibody-positive, culture-negative patients were reported positive. When the PCR and culture tests were compared, there was no evidence of positive cultures in the 20 antibody-negative patients that had a positive culture. The two PCR negative individuals had positive cultures and the two culture negative individuals had a positive PCR. The authors concluded, “We isolated HIV-1 or detected HIV-1 DNA sequences from the PBMC of all 409 HIV-1 antibody-positive individuals. None of 131 HIV-1 antibody-negative individuals were HIV-1 culture positive, nor were HIV-1 DNA sequences detected by PCR in the blood specimens of 43 seronegative individuals. In addition, HIV-1 PCR and HIV-1 culture were compared in testing the PBMC of 59 HIV-1 antibody-positive and 20 HIV-1 antibody-negative hemophilics. Both methods were found to have sensitivities and specificities of at least 97 and 100% respectively... Our ability to directly demonstrate HIV-1 infection in all HIV-1 antibody-positive individuals provides definite support that HIV-1 antibody positivity is associated with present HIV-1 infection”.

In other words, Jackson et al used the antibody tests as a gold standard for both the culture and PCR tests and the PCR and culture tests as a gold standard for the antibody test.

Jackson et al’s claims are not even confirmed by other laboratories. According to Jackson et al, up until 1990 only three small studies reported “100% isolation rates of HIV-1 from AIDS patients”. In all the other studies, “HIV-1 was not isolated from 6 to 50% of HIV-1 seropositive AIDS cases reported. The culture recovery rate of HIV-1 from HIV-1 antibody-positive asymptomatic patients has generally been even lower, only 20 to 42% in some studies”. The most recent small study of 60 patients who were HIV-1 DNA positive published by Jackson et al, between 1992-93 224 specimens were collected in Brazil, Rwanda, Thailand and Uganda from asymptomatic “HIV positive” individuals. Serostatus was first confirmed in the country of origin and then at the “centralized laboratories responsible for confirming serology, virus isolation, virus expression, and detection of reagents (George-Schuyler Hans Chemotherapeutische Forschungsinstitut (GSH) in Frankfurt, Germany; National Institute for Biological Standards and Control (NIBSC) in London, United Kingdom; and DAIDS/NAID at Bethesda, Maryland, United States). Using the method of Jackson et al. “of a total of 224 virus cultures, 83 were positive (isolation rate=37%)”.

Jackson et al’s PCR results, like their culture results, are not reproducible in other laboratories. For example, in the study conducted by Defer and her colleagues, where the same samples were tested in “Seven French laboratories with extensive experience in PCR detection of HIV DNA”, the data revealed that of 138 samples shown to contain “HIV DNA”, 34 (25%) did not contain “HIV antibodies” while of 262 specimens that did not contain “HIV DNA”, 17 (6%) did contain “HIV antibodies”! Unfortunately, the PCR and culture techniques are “exceedingly labor intensive and suffer from laboratory-to-laboratory variation due to differences in technique and operations” and that “in some reported studies there is no correlation between p24 antigen levels and measurements of infect-
tious virions. Similarly, a decrease in p24 antigen level is not necessarily associated with a positive clinical outcome". Because of this, to “Monitor Human Immunodeficiency Virus Type 1 Burden in Human Plasma”, the authors used the “branched DNA signal amplification assay” which, “offers improved sensitivity” and compared it with the “two other standard assays for viral burden: end-point dilution plasma culture and immune complex-dissociated (ICD) serum p24 antigen assay”. They reported that “HIV-1 DNA and ICD serum p24 antigen assays were done on serum samples from 102 seropositive (Western blot-confirmed) patients who were being screened for enrollment in clinical trials...of the 102 patients, 75 (74%) were positive for HIV RNA by the bDNA assay and 61 (60%) were positive by the ICD p24 assay. Only a subset of these patients had gp 120-antibodies and 36 (70%) were tested for plasma viremia by viral culture; 34 (61%) were culture-positive, while 50 (89%) were positive by bDNA assay and 39 (70%) were positive by the ICD p24 assay”. How is it then possible to claim that “virtually all people who contain HIV DNA also contain antibodies against Montaguer’s HIV strain” and “most, but not certainly all people who lack HIV DNA contain no such antibodies?".

CONCLUSION AND COMMENTS—Since Jackson et al did not test all 409 patients and all 131 antibody-negative individuals for the presence of “HIV DNA” using PCR, but tested only 66 patients and a maximum of 43 “antibody-negative” individuals; did not sequence the amplified segments which they sequenced did not yet use the only valid gold standard, HIV isolation, it was not possible for them to report “HIV specific DNA subsets...in 403 of the 409 antibody-positive, but none of the 131 antibody-negative people”. Furthermore, Jackson et al acknowledged that their PCR method did not prove the existence of the full-length HIV genome but only “that AIDS patients as well as HIV-positive asymptomatic individuals harbor HIV-1 genetic material”. In addition, for their PCR determinations, Jackson et al used a small fragment of the gag gene as a primer. But: (a) since the best known HIV experts agree that the gag genes of retroviruses are homologous, Jackson et al’s negative PCR results in all 43 “antibody-negative” individuals must at least have had the retroviral gag genes, if the PCR specific for the gag gene was effective; and (b) finding a positive PCR result using a small fragment of the gag gene as a primer is not proof for the existence of the “full length HIV genome” or even for the existence of the “full length HIV gag gene”. As has been already mentioned, by 1989 researchers at the Pasteur Institute concluded that “the task of defining HIV infection in molecular terms will be difficult”. In fact, as far back as 1973, retrovirologists were aware that the unusual nature of retroviruses "will prove a stumbling block to any genetic analysis of RNA tumour viruses”. Yet, at least some HIV experts, including Jackson et al insist on defining HIV infection in genetic terms. On the other hand, an analysis of the presently available data on retroviruses shows that all retrovirologists seem to agree that the single most decisive factor in proving the existence of "HIV" is morphologic. Thus is the virus, its importance well illustrated by the history of the discovery and subsequent demise of HL23V (see 5.4). As far as HIV is concerned, it is well known that the only evidence considered to prove the HIV theory of AIDS is a correlation between the clinical syndrome and a positive antibody test. Less well known is the fact that in the four papers published in Science between 1981 and 1984, Gallo and his colleagues claimed that in contradistinction to Montague and his colleagues, he and his colleagues achieved “true isolation”. However, it is of pivotal significance that the only difference between the experiments performed by the two groups is that Gallo’s group employed a leukemic cell line from which they were able to obtain abundant “HIV antigens” and thus could perform significant "more antibody tests." Given the crucial status retrovirologists accord to specific antibodies proving the existence of a unique retrovirus and its pathogenicity, proof of antibody specificity would appear to be mandatory. The specificity of the HIV antibody tests can be determined only by the use of HIV isolation as a gold standard. To date this has not been done and the method was adopted because no one has fulfilled even the first step in the only scientifically valid method for retroviral isolation, that is, electron microscopic demonstration of the particles with the morphological characteristics of retroviruses banding in sucrose density gradients at the density of 1.16 gm/ml. In addition, “HIV” can only be “isolated” from a minority of individuals who have a positive antibody test. Yet an even more substantial criticism is that in the case of HL23V, there is evidence that the antibodies present in human sera which react with “HIV proteins” are also non-specific: (a) “One half of the molecular weight of gp120 is represented by oligomannosidic oligosaccharides...Polyclonal antibodies to mannan from yeast also recognise the carbohydrate structure of gp120 of the AIDS virus”241; (b) “The immunochromatographic determinants of the antigenic factors of Candida albicans display a high identity with the glycoprotein (gp) 120 of HIV-1: they contain (a1→2) and (a1→3) linked mannose terminal residues”242; (c) antibodies to the mannans of Candida albicans “block infection of H9 cells by HIV-1” as well as the binding of lectins to gp 120;242; (d) recognition of gp120 by antibodies to a synthetic peptide of the same antigen was “partially abolished if it was absorbed with the total polysaccharide fraction of C. albicans” while the antigen recognition by antibodies to “gp120 from human T cell lymphotropic virus type 111B”, “was totally blocked”. From these data the authors conclude: “These results indicate that mannose residues of C. albicans can serve as antigens to raise neutralising antibodies against HIV infection”242; (e) “normal human serum contains antibodies capable of recognising the carbohydrate moiety of HIV envelope glycoproteins...from 100ml of human serum approximately 200mg of MBigG was recovered (MBigG=mannan-binding IgG). MBigG bound to HIV envelope glycoprotein gp 120;243; (f) researchers from the University of Rome infected healthy mice with an E. coli lipopolysaccharide (LPS) and reacted their sera with two synthetic peptides, one encompassing gp 120 V3 loop of “HIV-1 MN” and the other “representing a gp41 immunodominant epitope” (V Colizzi et al., personal communication).

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Collective Fallacy

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eaders will be aware that there have been a number of claims for, and responses to, Continuum’s offer of a reward for “The Missing Virus”. These have ranged from requests for clarification as to the type of proof required, through dismissive comments that the proof sought was an irrelevance, to an outright claim to the prize by Professor Peter Duesberg. Readers will recall that the point of the whole exercise was consistent with my article explaining that HIV does not actually exist, as opposed to the more frequently asked question whether HIV is responsible for AIDS or not.

The distinguished Australians led by Dr Eleopulos-Papadopulos have already provided a detailed reply to the Duesberg claim, so I shall endeavour to explain how the erroneous concept of retroviruses brought about the present situation.

Duesberg’s enormous services to mankind are beyond dispute. It is he who for nearly 10 years now, has steadfastly and at great personal cost, been the anchor of sanity and decency in a world driven mad by the simple-minded HIV theory. Whether HIV exists and whether it causes AIDS is largely academic: when did you last come across a “normal” heterosexual – someone who does not derive his living from perpetuating the panic – who their work might be. Even worse, is the habit of making countless ad hoc adjustments to the original theory, which completely distort the original hypothesis. Correct science demands that there should be a radical re-think when this happens. If there isn’t, as in this case, a fundamentally flawed concept goes haywire ending in disaster. Duesberg went along with mainstream AIDS researchers, limiting his objections to the relatively minor aspect of whether HIV could cause AIDS or not, whereas he really ought to have smelt a rat regarding the whole concept of retroviruses, given his earlier, courageous stance in admitting the mistaken role of retroviruses in causing cancer long before anyone else did even though he was deeply enmeshed for a long time in that fallacy, too. In my view, he could reasonably have been expected to satisfy himself that there was in fact such an entity as a retrovirus at all. Instead, he has allowed himself to be bedazzled by the technical feat of "retrovirologists" who manage to reproduce in a consistent manner certain phenomena peculiar to particular biological constituents of cells. In so doing, he allowed himself to be misled into believing that this was due to a virus. It is a complete non-sequitur. This lack of intellectual rigour has, in a contemporary metaphor, debased molecular biology to a virtual science, leading to the deplorable state of having a disease (AIDS) without a virtual definition, due to a virtual pathogen (HIV). Unfortunately for humanity, AIDS is not unique in this regard, but represents merely the tip of a much larger iceberg.

Lack of intellectual rigour has debased molecular biology to a virtual science

takes the slightest notice personally of the official story? In practice, Duesberg’s claim to our unqualified gratitude has been his long standing and unwavering opposition to AZT (and its analogues), whose use always ends in death.

That said, it is unfortunately also true that Duesberg is himself the victim of another collective fallacy (the Denkkollektiv of Ludwig Fleck), which he himself helped to formulate and which he is now apparently locked into.

Retroviruses were postulated as being that species of micro-organism which caused reverse transcription to occur, which, as a working hypothesis at the time in the early 1970s, was entirely reasonable. The mistake was to elevate the hypothesis to a dogma. Early gene detection techniques lent some credence to the existence of an entity that could be transmitted from one cell to another, which was unfortunate, because this, too, turned out to be wrong. Errors of this kind occur whenever technology makes available for general use a new experimental procedure, which propels a whole army of researchers into mass producing experimental data, heedless of what the biological significance, if any, of

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o the astute observer, it should have been apparent as early as 1973 that the working hypothesis of ascribing the experimentally observed phenomenon of reverse transcription to retroviruses had become untenable, when it became known that reverse transcription was anything but rare; by 1980 at the latest, the hypothesis should have been abandoned by everyone. Indeed, the extraordinarily artificial and circumscribed conditions under which reverse transcription could be induced in the laboratory should have alerted everyone to the extreme improbability of such exclusively laboratory conditions having any bearing whatsoever on naturally occurring phenomena. All the more so, as no retrovirus was ever shown to exist – for example, by being able to isolate and characterise it, and to demonstrate its transmissibility. These failures (obviously not for want of trying) should have sufficed to kill off the whole concept. It may be hard to believe that all maps purporting to represent a whole retrovirus, including HIV, are always compilations, many bits and pieces cobbled together by their authors to the best of their beliefs. They are collages. No complete retrovirus nor its RNA in its entirety has ever been proved to exist either in vivo or in vitro.

A further difficulty for the hypothesis was that it had never proved possible to show that the experimental observations attributed to retroviruses were exogenous to the cells used in experiments, i.e. that they came from outside of the cell; indeed,

It is a dire example of how a distinguished scholar who has contributed much to the advancement of science, now impedes further progress by his stubborn adherence to a dogma of his own creation.

If he did not feel himself obliged to repeat things that are untrue just because they were once said, they would have become quite different people.

Johann Wolfgang von Goethe,
Maximen und Reflexionen, Textstelle 586
all the evidence pointed to the opposite, i.e. that they were endogenous (inherent) to the cells themselves. Part of the evidence was that the so-called retroviral activity could only ever be induced experimentally in one type of cell, whereas HIV is supposed to infect many different types of cell in the body. The two contentions are clearly incompatible. The whole theory is rendered even more implausible when it is remembered that retroviral concentrations are always extremely low, which is why a huge excess of cellular material from "patients" is needed to be able to demonstrate any replicating virus at all. This, incidentally, is also the basis for the claim that HIV has only a low rate of infectivity: a much more rational explanation is that there is no virus there at all.

HISTORY furnishes an unhappy precedent for this form of research. At the turn of the century experiments were conducted using highly in-bred laboratory animals. Under strictly circumscribed conditions, these displayed higher disease susceptibilities than animals which were not in-bred; the phrase 'highly in-bred' was forgotten about, and generalisations about viral infectivity were made which turned out to be wrong, from which medicine has not recovered to this day.

In like manner, experiments are nowadays performed with cell cultures instead of whole animals, for the very good reason that they greatly speed up experiments. The disadvantage is that this limits experimentation to just one of a few cell lines, which are always cancerous, because only they will grow continuously in the laboratory. History is repeating itself: generalisations are made about the behaviour of normal cells on the basis of results obtained from highly abnormal cells.

Such cells can incorporate extraneous pieces of DNA (into their own DNA) when added to growth medium (as normal cells can, too, only more slowly). Cells, which have incorporated such DNA, will obviously manifest characteristics for which that DNA coded, making it appear that a virus had been at work, when nothing of the sort had happened. It is easy to see, therefore, how the bizarre notion of 'infectious' DNA arose, and to conclude (wrongly) that a virus is the only possible explanation. You are told that 'it means what I say it means.'

Yet this is the basis of Duesberg's claim. In his retrovirological zeal he does not seem to appreciate that 'infectious DNA' is a contradiction in terms. For what else is a virus but that? Folded up DNA wrapped in a protein coat so that the DNA can be transmitted from one cell to another, is what is normally called a virus. A loose strand of DNA could not do this by itself: it would be exposed to enzymic degradation; it would become entangled with other components. How would it identify its target; how would it get there; how would it enter it without a specific mechanism?

A man of Duesberg's abilities should need no prompting that replicating (=ioning) something in a test tube and then detecting that something (=molecularly cloned DNA), in a place where you have previously put it, is a circular argument, and therefore no argument at all. But then, tautologies are an indispensable part of all retrovirology, as I was at pains to point out when explaining the fallacy inherent in HIV antibody tests.

CONCLUSION

The rules demonstrating the existence of HIV (and retroviruses in general) were never adhered to by those who devised them nor were they ever validated. It is now easier to understand why people felt it necessary to enquire of this magazine what the rather self-evident term 'isolation' meant: suitable synonyms might be 'pure' and/or 'free of contaminants'. They clearly had a slight worry at the back of their minds all along that the term was used in retrovirology rather as in Alice in Wonderland – "it means what I say it means."

Until AIDS was invented, retrovirologists were a minority sect who were happy to accept each others' flights of fancy without being too critical. They could fiddle around to their hearts' content, safe in the knowledge that "retroviruses were the least dangerous of all viruses". Well-meaning and credulous colleagues, as well as aspiring virologists, journalists and, through them, laymen, were mesmerised by incomprehensible jargon into believing that the mass of data on HIV and retroviruses somehow meant something. Each property relating to HIV, and retroviruses generally, can be shown to pertain to the cells used in the co-cultivation experiments. At no time have there ever been any credible grounds for thinking that these properties and components had anything to do with viruses in general, nor with "HIV" in particular.

No particle of HIV has ever been obtained pure, free of contaminants; nor has a complete piece of HIV RNA (or the transcribed DNA) ever been proved to exist.
Keith Haring: Journals
Introduction by Robert Farris Thompson
Preface by David Hockney
Published October 7 by Fourth Estate, London, £20.

Keith Haring (1958-1990) is considered by some to be one of the most important and popular artists of the 20th century, whose work is still as historically and socially relevant as when he died with an AIDS diagnosis six years ago. The publication of his journals this month has given NIGEL EDWARDS a chance to assess the significance of the artist’s contribution to the HIV/AIDS debate, measured against Haring’s understanding of his own role as an influential artist.

“To be a victim of your own knowledge is not understanding what your knowledge is and what its result is,” writes Keith Haring on October 14, 1978. It is one of the earliest entries in his journal, and one of the most poignant and ironic. “Thinking you know the answer is as dangerous as not thinking about the possibilities of no answers,” comes a few sentences later.

Haring wrestling with his deepest thoughts on the power of art in society, and his role as an artist in possibly influencing changes in society, is one of the themes that makes the most impact on anyone reading his journals. The entries begin in Pittsburgh on April 29, 1977, a few days before his 19th birthday and as he anticipates hitchhiking to a Grateful Dead concert in Minnesota. The last, on September 22, 1989, five months before his death, is written in Milan by someone who is himself as internationally famous as any rock star and who is about to set out for Pisa to create a fresco on a 1,000-year-old building. The pages inbetween, filled somewhat sporadically with his intimate and professional reflections, chronicle a career that started out as a graffiti artist in the New York subways, drawing with white chalk on the black paper pasted over unused advertising spaces. His subsequent work is in the permanent collections of museums around the world.

Haring’s artistic style and legacy is probably better known than Haring himself, something which Haring was certainly aware of and which serves to remind us of the power of art when it seeks to convey ideas and influence people. The 21-page introduction to the journals by Robert Farris Thompson spells out plainly what is perceived to be Haring’s artistic contribution in the HIV/AIDS field. “Never knowing, after 1988, when AIDS might take him, Haring painted in the late Eighties to save others and keep himself alive,” he writes. “Characteristic, he enriched the documents of alarm with variations of astonishing strength.”

Thompson relates how Haring’s first paintings and posters were straightforwardly activist, such as the 1989 Silence=Death composition, in which a pink triangle is filled with ghostly silver figures, covering their eyes or closing their ears. The Haring “probes the terror in extreme promiscuity” with a graphic image of “beings who have fucked themselves to death.”

Haring’s most significant statement is where he “dared to personify the virus – as demonic sperm” which “bursts from an egg, like a giant horned insect. Its horns break the frame of crimson, as if escaping from the paper. Haring locates the lairs of the virus: drug addicts’ needles, uncovered penises and vaginas.” And Thompson concludes: “If this series is important, it is because the artist expressed the presence of a killer by a radical combination of elegance and shock.”

Thompson, of course, views all this as the strength of Haring’s claim to artistic immortality. “He shows that in the spirit of his art, not his doomed body, his durability must be sought.” But the tragedy of Haring is that not only did he fail to live up to his own aspirations, so clearly expressed in memorable passages throughout his journal, but that he completely failed to recognise that he was failing.

For it is clear that Haring accepted and purveyed a scientific view of HIV and AIDS that he never once appears to have questioned. Haring believed that HIV was a virus that inevitably led to AIDS and death. This was despite the following journal entry of June 26, 1987: “Contemporary man, with his blind faith in science and progress, hopelessly confused by the politics of money and greed and abuse of power, deluded by what appears to be his ‘control’ of ‘the situation,’ etc., etc., believes in his ‘superiority’ over his environment and other animals.” Haring goes on to assert that, because of this, it is “so important” for artists to “interfere” with established ideas about the purpose and meaning of life and to change them “through the insertion of aesthetic manipulation.”

So Haring sought to influence the world towards accepting viewpoints and concepts regarding HIV and AIDS that he himself had never properly explored and challenged. His own acceptance of the inevitability of his own death is graphically illustrated merely by noting the frequency and length of his journal entries. Before 1987, these are very uneven. After, we become aware of a Haring who, conscious of limited time, seeks to leave his literary and philosophical legacy alongside his artistic contribution.

He adopts what can best be described as the politically correct attitude towards HIV and AIDS of the average gay American of his generation. He records how he wore safer sex T-shirts, flirted with the concept that HIV might be a biological weapon developed and covered up by the US Government, considered ideas for designing condoms, and reacted angrily to attempts by journalists and friends to inquire into his HIV status. A phone call from New York Newsday asking him to comment on the rumour he had AIDS was condemned as “obnoxious”. But while he tries to get on with life without dwelling on it, he notes on Valentine’s Day 1989 that he never once appears to have questioned. Haring believed that HIV was a virus that inevitably led to AIDS and death. This was despite the following journal entry of June 26, 1987: “Contemporary man, with his blind faith in science and progress, hopelessly confused by the politics of money and greed and abuse of power, deluded by what appears to be his ‘control’ of ‘the situation,’ etc., etc., believes in his ‘superiority’ over his environment and other animals.” Haring goes on to assert that, because of this, it is “so important” for artists to “interfere” with established ideas about the purpose and meaning of life and to change them “through the insertion of aesthetic manipulation.”

As a self-acknowledged influential artist who came to believe he had a personal right to pronounce on HIV and AIDS it is ironic to read (June 26, 1987): “Art is important, and important artists (respected ideas) are important because their ideas and creations are widely discussed and dispersed. The responsibility that carries with it is, for me, mind-boggling.”
Taoism, Sex and Health

ANDY SAICH co-facilitates the Sensual Touch Workshop in London with Gary Hampson. Here he outlines the principles and practices that inform their work. The Sensual Touch Workshop is an introduction to Taoism, Sex and Health.

Two thousand years ago Taoist physicians studying sex concluded that lovemaking is necessary to the physical, mental and spiritual well-being of men and women. Also a man whose lovemaking skills enabled him to enjoy prolonged and frequent sexual play is more highly rated than a man who is merely young and handsome.

According to Taoist belief energy and momentum are the sources of all life. In the universal picture humans are relatively small. In order to maintain a dynamic balance of health we must be in harmony with these sources that are the infinite power of nature.

Today we are taught to look outside ourselves for entertainment and constantly seek new stimulation to satisfy this need. Yet hidden within ourselves are unlimited electrifying ecstasies that we can never tire of.

Taoism is neither a religion nor a path to salvation. The Tao is the infinite force of nature, the path of the heart or put simply ‘the way’. The raw materials needed for the Tao can be found at any given moment in our lives. The Taoists, being practical, proposed that we begin with the most accessible energy which is the feeling of sexual attraction.

Unlike Tantra, Taoism never took on secret rituals and invocation of religious deities – sex was more openly used in China as a medicinal form of healing and natural way to spiritual balance.

There are two main goals for which Tao techniques of energy cultivation can be used. The first is to improve worldly happiness, to increase physical, emotional and mental satisfaction. For example strengthening love partnerships, alleviating sexual frustration, impotency and premature ejaculation and relieving boredom with sex, increasing longevity and good health. The second is for someone on a spiritual path who wishes to integrate sexual desire with his spiritual belief or meditative practice. The Tao is a sex-positive approach that helps to better integrate sexuality with spiritual growth.

The beginning stages of Taoism if you have a lover are:

1. A man learns to sustain erection for as long as desired and regulates his ejaculation.
2. Both partners re-direct sexual energy through the body into higher regions of the heart and brain.
3. The lovers exchange their super charged energy with each other.

If you do not have a lover you follow what is called ‘single cultivation’; this is where sex energy is channelled creatively in daily life and used to heal the mind, body and spirit and so enjoy life without sexual frustration.

Taoists see sexuality as the primary source of power behind love. The aim is not to fulfil the human ego with its insatiable desires but to quiet the ego and calm the mind so as to first observe and then cultivate the body’s energies.

Many in the West have heard of the concept of abstaining from ejaculation as Taoist. In fact the Tao views male semen as a vital essence and actually urges all men to develop ejaculation control. Depending on your age and physical condition a man should discover and master his own ideal ejaculatory frequency. This should not be more than two or three times in ten coitions (sexual incidents ).

It is generally assumed in the Western world that ejaculation is the climax of pleasure. The Taoists taught that male orgasm and ejaculation are not the same thing.

After the brief sensation of ejaculation a man is tired. By reducing and regulating ejaculation your body will be strengthened. Though at times he might seem to have denied himself an ejaculatory sensation, he will be more energised, he will have more confidence by savouring the energy generated in sexual contact, his immune system is stimulated and his love for his partner will increase, as if he could not have enough of him. This is true lasting pleasure.

The Tao shows how to balance the Yin (feminine energy) and the Yang (masculine energy). Heterosexual couples would seek to balance their feminine/masculine essence from each other through lovemaking. Gay partners able to experience physically both masculine and feminine roles have a more direct opportunity to balance these energies within. Androgyny in this respect could be considered a spiritual state of being as it represents a physical manifestation of a Yin/Yang balance.

The Sensual Touch Workshop is an introduction to Taoism, Sex and Health.

Savour the energy created in sensual contact
Sensual Touch and Sexual Feeling

Continuum Administrator TONY TOM PSETT was invited to attend a Sensual Touch workshop recently and describes how he felt after the experience.

I reckon we all consider ourselves accomplished and knowledgeable when it comes to sex. And of course, there is a wide range of sexual practices, some of which would make your grandmother cringe, but which are commonplace in certain areas of society today. But, I dare say that something that isn’t taught or mentioned much is sensuality, and, whilst we’ve all heard the word, I wonder if we really know what it means, or ever put it into practice?

Often our natural sex drive carries us away and we go through the motions of sex, reaching the climax we think we wanted, only to find that it wasn’t as fulfilling as we thought and the next night we need that fix again, whether it’s a quickie out of duty with your partner, a casual pickup when you’re a bit drunk, or even a long session that was prearranged and meant to be enjoyed. So often, sex is just sex.

When the invitation came to find out more about sensuality I jumped at the chance hoping to gain something that I might put into practice in my own life. I was naturally a little apprehensive, as the brochure mentioned words like Tantra, Taoism and Shamanism, and as I didn’t know much about these things I wasn’t sure what to expect.

Anyway, I arose early one Sunday morning and arrived at a delightfully sunlit studio in Central London, where the gathered group numbered some 22 gay men of all ages, most of whom, during the introduction, expressed what I too was feeling. The outline of the day was explained and we were made to feel perfectly safe with a get-out clause if we felt we didn’t want to participate in any particular activity. I don’t think anyone opted out at all.

We were given a basic introduction to the Eastern ideas of opening up the mind and body to allow energies to flow and be concentrated on what we can feel – surely an erotic but relaxed setting, and it certainly takes the idea of enjoying sex and sensual feelings into a new realm, taking away any urgency that we normally feel. The climax of the day was the exercise that combined the erotic massage and breathing methods. This was done in small groups, where one man would be touched and massaged by at least two others. It really was a wonderful experience to lie down with my eyes closed and, relaxing and concentrating on my breathing, to let others touch and stroke me, giving me a sensual massage that immediately aroused my natural energies and feelings, allowing me to direct and enjoy the pleasure, but without the pressure to reach orgasm. The sense of warmth that engulfed my body was both calming and powerful – something I shall remember for a long time and hope to incorporate into my life many more times.

I would certainly recommend these workshops to anyone, however unsure they are, as they are conducted in a professional and structured way where you feel completely safe. It is a brave but very interesting and enjoyable experience to put oneself into the hands of others in an erotic but relaxed setting, and it certainly takes the idea of enjoying sex and sensual feelings into a new realm, taking away any urgency that we normally have, and allowing us to relax and concentrate on what we can feel – surely the true meaning of the word sensuality (using the senses).

This was only an introduction to Taoism but I became aware of the concept of channeling the natural energies we have in order to achieve relaxation and heightened sexual pleasure. Whilst there was not too much ‘spirituality’ in how and what we were taught I appreciate that there is a wealth of experience in other traditions which we can usefully take on board and which I am sure can assist us to add another dimension to our enjoyment of life and each other.

Further reading:
*T’ung Hsuan Tzu. chapter 12 by Li Tung Hsien
Tao Te Ch’ing by Lao-Tse
Secret of the Golden Flower by Wilhelm and Jung
Taoist Secrets of Love by Mantak Chia
The Tao of Love and Sex by Joan Chang

For details of the Sensual Touch Workshop phone 0181-746 3599. Any readers with personal experiences of Taoism, sex and health who wish to comment on this subject should write in or fax.
ANGLÉ LOPEZ studied Sociology at the Complutense University, Madrid. After 10 years in business he lived in Paris where he combined literature and commerce studies and worked in the fashion industry.

In general terms one could say that sadomasochistic activities have a very bad reputation in our Western society, and perhaps more precisely here in Britain. I'm referring to a number of convictions carried out in 1990 in this country by the police, “Operation Spanner”, against certain individuals for engaging in SM practices. These practices took place in private, among adult friends exercising consensual sadomasochism. It appears that this kind of activity frightens people -why? After all, it had little to do with the most extreme situations described by the Marquis de Sade (1740-1814) - whose fiction was anyway more excessive than his sexual life - and involved people who chose and knew what they were doing. To some extent, these “disciplines” were domesticated and adapted to liberal-bourgeois thinking. In this case, it seems to me that arrest and condemnation stem more from prevailing fear of sexuality itself, particularly in this repressive society of Victorian heritage which tolerates with great difficulty diverse forms of sexual delights and “refinements”. Paradoxically, this repression creates a syrup of an excellent harvest, although certainly not the only one for more daring sexual fantasies.

Perhaps the most outrageous character of SM practices comes from its metaphorical value. This is because sex also has a violent component. SM yields dominant and submissive scenarios manifested in physical and theatrical forms among its practitioners. These relationships are also a product of elaborate mental constructs. When these are in the realm of consciousness, one seeks to satisfy one’s own restrained fantasies and desires. According to Camille Paglia: “Sade’s highest erogenous zone is the mind. His works, like Genet’s, are autoerotic prison dreams creating a perverse Universe of new sensations and sexes”.

People may consider power relationships are respectable when they are funnelled into productive, economic ventures. Explicit power relationships appear unacceptable when they are used for gaining sexual satisfaction. In Richard Tristman’s words: “All sexuality entails some degree of theater” and in sado-masochism this theatricality is developed and plays a fundamental role. There is no place for simplicity in the sexual SM act, in contrast with unsophisticated sexual expression in a bland domestic environment.

To channel pleasure through power or submission expresses one’s own aggres-

The first astonishing aspect while queuing up to get in (four o’clock in the afternoon) on Saturday 15th September at London’s “Dungeon in the Sky” was the contrast between Bloomsbury’s traditionally elegant neighbourhood, and the people hanging about. The SM cult uniforms, where black fetter leather and rubber prevail, were combined with multiple tattoos and piercing together with studs and other skewers. Touches of fantasy evoked “Merry-widow” operetta style thanks to the girls wearing peacock’s comb-type feathers and transparent embroideries.

The opposition became accentuated rather than mitigated once inside the University of London Union’s headquarters where the party took place throughout this four-storey building. The intense neon lights and decor, persistently cold and ‘academic’, did not seem very conducive to SM and otherwise generally hidden habits.

I personally found interesting the fist-fucking inducements, although sometimes their sanitary aspects outweighed that SM and otherwise generally hidden habits.

I thought that widespread activity was perhaps reserved for the evening “Spanner Ball” under The Arches of Southwark Street - an appropriate place for this kind of gathering due to its underworld nature, like a catacomb. I found myself with a Carnival à la mode whose main theme was more like SM fantasies, people dancing and displaying their original attire, everything very much like a “show off”. Nevertheless some of the girls on show left me quite impressed with their piercing techniques, to the point of having the impression of being sometimes in a surreal happening.

The party had little resemblance to those SM parties in Paris or Amsterdam where the ‘ceremony’ is more prone to excitement and its subsequent activities. The outfits sometimes seemed more appropriate for a Gaultier cat-walk in Heaven than an SM party where people are able to realise their sexual cravings. Curiously, it seems to me that the longing wishes were not precisely what was missing at this party - rather more, how to reach out to someone and spend a good time in the company of people with similar sensibilities.
I became a dissident in a round-about way. Researching my book Dirty Medicine, I looked at Welcome and the marketing of AZT at the same time surveying those whose work touched upon AIDS and who had been attacked by the medical and scientific AIDS establishment. So it happened in a negative way that I came to recognise all those therapies, thinkers and practitioners that the AIDS establishment saw as a threat to their profitable HIV theory.

Working on the assumption that my enemies’ enemy is often my friend, I assumed that the dissident world would be full of people who held co-operative and overlapping theories about the immune system, viral overload, nutritional depletion, chemical toxicity and iatrogenic drug damage. I believed that there would be a considerable exchange of information between those who researched ME (believed in some cases to be a post-viral syndrome aided by excessive use of antibacterials and antibiotics), organophosphate toxicity those exposed to pesticides develop a range of illnesses from respiratory to cardio-vascular to cancers) and most of all Multiple Chemical Sensitivity (MCS) (an increasing number of well documented cases of people who suffer almost complete immune system failure).

After six years of mixing with AIDS dissidents, however, I still find that many of those involved are reluctant to place the illnesses which are wrongly attributed to HIV in the context of either chemical sensitivity or the range of ‘new illness’ which medical orthodoxy is unable to diagnose. Even Peter Duesberg despite his well argued case that Poppers (amyl nitrate) and AZT are responsible for large numbers of ‘AIDS cases’, is reluctant to introduce information about other immune-suppressing chemicals or discuss the history of clinical ecology (environmental illness). In his excellent book, Inventing the AIDS Virus Duesberg gives a contextual history of iatrogenic drug induced illness, concentrating on SMOH. He also discusses illnesses brought about by nutritional deficiencies. But he gives no information for example about the substantial work on pesticides and cancer (which, in the case of DDT, use in Australia, Canada and New Zealand in the 1950s, also appeared like an outbreak of fatal infectious illness) or the basis of a hidden political agenda; although it appears that the NHS is an organisation which monitors and maintains the health of the people, in fact, it is a handmaiden of the State, in hock to the pharmaceutical companies; orthodox professional scientists can not always be trusted to forgo the profitable abstractions of their science, increasingly as a consequence of the funding crisis they too are handmaidens of the chemical and pharmaceutical companies; workers and those at the lower end of the economic scale – ultimately workers in the developing world – often women, suffer most from the damaging effects of the chemical industry; finally, the chemical, pharmaceutical and processed food industry is a global industry which has to be fought internationally.

A community based movement against chemical toxins is growing and becoming organised. It is evolving its own institutions, user friendly experts and legal advisers as well as its own media. This is not to say that there have not been, and will continue to be, failed campaigns and damaged and uncompensated individuals. Nor is it to say that the growing movement is not still naive.

Organisation and a rising consciousness in the community inevitably create a backlash from the chemical industry. This is already happening in America, where there has been an increase in groups providing expert scientific witnesses to argue against Multiple Chemical Sensitivity sufferers, populist corporation front-organisations which harass environmentalists, and pseudo-scientific Institutes which provide ‘tobacco research’ studies.

The movement, however, is coming of age and its naivety is slipping away. At the workshop on the enforced fluoridation of the water supply, presented by the National Pure Water Association, the man who asked if he could put the case for fluoridation was told with authority, ‘This is not a debate!’ Professor Samuel Epstein, Professor of Occupational and Environmental Medicine at the School of Public Health at Illinois University, and author of The Politics of Cancer seemed at times to tower over the conference. He delivered a massive and sustained attack upon the chemical industry and those who have obscured the ongoing health damage, especially with respect to cancer for which it is responsible. At one point, answering a question, he said unequivocally, ‘When professionals prevaricate or try to avoid the environmental cause of health damage then we should believe the sufferers and their intuitive concerns about their local environment.'
environment”.

The same message was delivered by Dr Andrew Watterson, who gave a talk and a workshop on what has now come to be called Community Epidemiology. Although it is possible to quibble about the title – Jo Anna Ibarra of Community Hygiene Concern (which fought a successful campaign for a natural alternative to organo-phosphate treatments for head lice) said to me that she thought she had been involved in community epidemiology for some time, calling it Action Research – community epidemiology,

People have to collect their own evidence

intends like all good community movements to educate and empower the people to carry out their own research into the environmental causes of illness.

Researching the issues is still some way from fighting multi-national corporations or becoming legally empowered inside and outside the courts. One point of department does, however, often run directly into the next. Doug Cross, now an environmental consultant and previously a community activist in Camelford during the aluminium exposure crisis there, rightly laboured the point that people have to collect their own evidence. If they do not, they should not be surprised if later it is lost or on analysis, proves the case for the chemical companies.

I found myself most affected by the paper given by was Sarojeni Rengam, executive director of PAN the Pesticide Action Network. Sarojeni had coordinated a three year programme on the impact of Pesticides on women in Asia. This work was carried out in countries such as Indonesia, the Philippines and Korea. In these countries, workers, already suffer under the yoke of unjust domestic regimes, poverty, poor food, lack of work and low wages. The final brand of oppression is their endless subsistence level work, in sweat shops, factories and often, simultaneously, in the plantations and fields spraying deadly pesticides on food and flowers destined for a distorted global market. In these countries the terror of pesticides, eats away at the very heart of the community, often affecting all members of the family.

I came away from the conference, thankful to the Green Network for having organised it, but also with the feeling that I had witnessed the beginnings of a movement and that all those isolated groups of people concerned about diverse chemical health damage, were now, at last, beginning to weld themselves together into a progressive international movement.

In 1935 it was found that a product called suphanilamide from the red dye prontosil rubra protected mice from streptococcal infection. Hundreds of similar drugs have now been produced and tested for anti-bacterial activity. They belong to the sulphonamide group.

These drugs stop bacteria multiplying by interfering with the use of folic acid in the bacterial cells. Sulphonamides were prescribed extensively by doctors which led to the appearance of resistant bacteria and a high incidence of harmful effects, yet to be fully understood.

As penicillin and other antibiotics were introduced, the popularity of the sulphonamides decreased and their use was limited to the treatment of certain disorders such as infections of the urinary tract. More recently they returned as a common prescription in the form of SEPTRIN (BACTRIM) and CO-TRIMOXAZOLE for people with “HIV” diagnoses.

Harmful effects of sulphonamides are relatively common, and vary according to the particular drug used and the susceptibility of the individual. People who break down certain drugs slowly in their livers may be more at risk. Generally, harmful effects are related to the length of treatment and not always the dose. Reported harmful effects include:

Allergic reactions and skin rashes serious skin rashes, conjunctivitis, sensitivity of the skin to sunlight, nettle rash, itching; drug fever and chills, severe allergic reactions; serum sickness-like symptoms (fever, swollen glands, painful joints); bruising and bleeding into the skin (purpura); inflammation of arteries (periarteritis nodosa); systemic lupus erythematosus (an immune disorder producing inflammation of the skin, blood vessels, heart, lungs, nerves and joints); severe dermatitis, and a skin rash with conjunctivitis and ulcers of the eyes, mouth and urethra (Stevens-Johnson syndrome).

Blood disorders damage to the bone marrow and thus effects on red blood cell production, causing anaemia, and white blood cell production causing reduced resistance to infection. They may damage red blood cells directly, causing haemolytic anaemia, and interfere with the body’s use of folic acid, producing folic acid deficiency anaemia.

NOTE sulphonamides may, rarely, knock out red cell and/or white cell production completely.

Mouth, stomach and intestinal disorders sore gums, sore tongue, loss of appetite, nausea, vomiting, abdominal pains, diarrhoea, colitis.

Kidney disorders causing crystals in the urine and severe kidney damage, leading to kidney failure.

Liver disorders hepatitis and jaundice.

Disorders of the brain and nerves headache, drowsiness, dizziness, noises in the ears (tinnitus), vertigo, numbness and pins and needles in the arms and legs, incoordination of movements (ataxia), convulsions and meningitis.

Mental disorders hallucinations, depression, apathy and nervousness.

Other harmful effects include painful joints, painful muscles, weakness, fatigue, insomnia, bad dreams and confusion. Individuals who accept long-term treatment with sulphonamides should have regular blood tests to check their white and red cell counts and to check for other blood disorders.

Anyone taking sulphonamides should drink plenty of fluids in order to prevent crystals forming in the urine. Sulphonamides should be given with caution to people with severe allergies or asthma, and with great caution to people who are deficient in folic acid or who are sensitive to sunlight.

Because of bacterial resistance and the availability of more effective and less harmful antibiotics, sulphonamides until AIDS were no longer used to treat or prevent infections of the throat and chest, bacillary dysentery and meningococcal meningitis. Although the use of sulphonamide drugs alone has gone out of general fashion the combination of a sulphonamide drug (sulphamethoxazole) with trimethoprim is in fashion. This combination, known as co-trimoxazole, is widely used even outside AIDS medicine, and it is important to be aware that it contains a sulphonamide and that all the warnings on the use of sulphonamides also apply to co-trimoxazole.

HUW CHRISTIE

Source


For advice and information on Septrin contact The Septrin Action Group at P.O. Box 16, Wigan, WN6 9QZ, UK.
Kanne drink

For years people in Germany have been able to purchase Kanne Bread Drink not only as an aid to the promotion of good health but also to assist in the recovery process after illness. Seven million bottles of these products are sold each year. For the first time Kanne drink is now directly available in Britain.

Dirk J. Petersen, Doctor of Chinese Medicine, presents the product as follows:

“In recent decades medicine has made incredible progress in the field of technology. Operating techniques and methods of diagnosis have reached an amazing level, but great successes have also been achieved in the field of infectious diseases, through the controlling of smallpox, cholera, typhoid, polio and other scourges of humanity.

“Nevertheless there are many illnesses and health problems which obviously cannot be successfully treated by the methods of modern medicine. There are too many patients who are treated by conventional scientific methods and who, however, retain their symptoms. Not everything can be treated with cortisone, antibiotics or chemical drugs – this is all too clearly demonstrated by the large number of patients who do not get better.

“Since I particularly wanted to help those patients, I have taken an interest in alternative methods of treatment alongside conventional medicine. And so, after studying in Germany I went to Taiwan, to study traditional Chinese medicine there. Later, on extended trips to many countries, I attempted to become acquainted with the interesting treatments of primitive peoples. As a result I was then often able to help patients in my practice in Germany with, for example, a Chinese treatment, if Western medicine failed, or with a South American one, if the patient was rather neglected.

“Both healthfoods have supported my treatment in many cases. It was frequently even enough to introduce these two healthfoods, without further treatment, in order to cure patients of symptoms which have been present for a long time.

“Because they are convinced of their products which so many are using, the Russian Health Minister initiated in 1993. He has children, who had been affected by radiation, treated with the KANNE products. The results were so spectacular, that now a large-scale trial with 1000 patients is beginning in Chernobyl!”

Kanne products are not medicines but healthy nourishment based on grains fermented in lactic acid and rich in trace elements and minerals whose positive effect of people’s health was more or less discovered by chance.

The product has been found to aid recovery in people suffering from a wide range of illnesses and skin disorders, including: lack of vitality and energy; circulatory disturbances in small blood vessels; hyper tension; many forms of skin disease; diabetes; many forms of gastro-intestinal illness; hypercholesteremia; rheumatic and arthritic illnesses; digestive problems and dysentery; low levels of potassium, calcium and magnesium; osteoporosis; wounds, injuries and insect bites.

A booklet is available containing testimonials and scientific documentation recording the successes experienced in using “Kanne Bread drink” (see box below). Some recipes for nutritional drinks similar to Kanne drink can be found in wholefood literature. Often very good, their production requires a commitment to regularly culturing the drink.

For an information booklet about Kanne Bread Drink send 4 x 1st class stamps to the address below.
The drink is available by cash and carry and mail order and sold at £2.49/bottle.

Human Nature
25 Malvern Road
London NW6 5PS

Please make cheques payable to: Mr Nari Sadurham and send to the address above, OR for credit card orders call 0171 328 5452 or 0131 818 846. Door to door 4-day delivery (mainland) costs: 6 bottles - £8.50; 12 bottles - £10.50; 18 bottles - £12.50; 24 bottles - £14.10 (£5 surcharge for Scotland).
any people are confused by the messages that they hear on news reports and in magazines are so often contradictory. This is because they focus on the 'latest scientific research'. Most of the answers you really need have been around for a long time. Still one of the most nutritious meals that you can have is a soup which Hippocrates made famous in the fifth century BC and it was he who said 'Let food be your medicine and let medicine be your food.'

What is the point of paying to see a nutritional consultant when you can see an HIV dietician for free on the NHS? The point is that with a nutritional therapist you can address the very problems which are causing you to suffer from certain symptoms. The dietician will give you food which (apparently) will help you to cope with/alleviate the symptoms, but will not address the root cause. Someone who has trained in nutritional medicine will arrange an appointment which usually lasts one hour. They will ask you lots of questions about your life, health and ill health. Your whole life will be looked at in terms of your medical history, what illnesses you had, how they were treated or if they were left alone to run their course, what major events have happened in your life of both the stressful and life enhancing varieties, what jobs you have done and any toxins or problems which you might have been exposed to and of course what food you have eaten. You will not be asked to produce a list of all the foods you have ever eaten, more what eating patterns have you had and your attitudes towards them, for example were you brought up on meat and two veg? How long have you been eating processed foods? Have you been a heavy tea or coffee drinker for years? At what age did you become a heavy tea or coffee drinker for years?

How long have you been eating processed foods?

vegetarian? How many years did your daily round of cheese on toast with tomato sauce last? They will look for any habits that caused you to take in a lot of one kind of food and whether or not your body had the ability to deal with it at the time. Your current diet will be looked at in much closer detail and you might be asked to produce a short food diary.

Once your whole life story has been established the practitioner will be able to work out what toxins you have accumulated in your body, what your current symptoms are being caused by, any mineral imbalances that you may have and the depth of your disease. Armed with all this information she or he will prescribe a diet for you, tailored to your individual needs at a level with which you can cope, some supplements to support your body in its healing efforts and probably some additional things which you can be doing as well as eating your new diet and taking your nutritional supplements. This could be a kind of exercise, skin brushing, acupuncture, aromatherapy or a whole host of other supportive measures.

Follow up sessions will happen every four to eight weeks and will assess your progress, adjust the eliminatory pressure of your supplements, change your diet or whatever is appropriate. Your course of treatment will always remain unique to you, because whatever disease you are suffering from, the way in which you manifest it will be unique to you.

Nutrition is the foundation for all healing. If you do not provide your body with the nutrients which it needs in order to carry out its normal functions then you certainly will not be giving it the extra boost it needs in order to heal itself. The person who coined the phrase ‘you are what you eat’ was right in a lot of ways, the very bodies that we walk around in are made from the food and drinks which we have put inside us. A more accurate phrase to start quoting, however, might be you are what you don’t shit!

How does this nutrition business actually work then? It is based on the premise that the body, given the right energy and the correct micronutrient support will heal itself. This energy is the life force which pervades us all and has been recognised for thousands of years. It has many pseudonyms such as God, Prana and universal energy. Our bodies would be just a bunch of chemicals if we did not have a life force and they wouldn't hang around together for very long and we would cease to exist. The question of what this energy is has intrigued philosophers and healers throughout the ages. Centuries ago, the predominant point of view centred on the philosophy of ‘vitalism’, which postulated the intelligence and power to govern the myriad of processes involved in both health and disease. Some animating force or principle enters the organism at the time...
of conception, guides all the functions of life, and then leaves at the time of death. What does occur at the moment of death? The organism is structurally intact, cells are busily functioning, chemical reactions are still proceeding, yet a sudden change occurs and the body begins to decompose. Reflection upon this fact renders the concept of vital forces not only understandable, but appealing.

Our life force takes a battering if we are run down or depressed, if we have an accident, a friend dies or you go through any major stressful event. We can increase and support our life force by eating foods which are vital, those which contain this life-giving energy. Any food which is fresh from the ground, which has not been through umpteen processing units and had chemicals added to it is vital. You know that feeling when you have a carrot juice or a fresh salad and you feel great? That is the benefit of eating life-giving foods. Every living organism is made from cells which are a part of the whole. The nutritional interactions of the organism takes place on a smaller level within each cell. The human body is made up of a frighteningly large number of cells, each with its own nutritional requirements and demands, proportional to its size and workload. The total amount of food a human needs to consume is simply the sum total of what the cells need.

As with any living thing, a cell has to control what goes in and out of it. This is done by a highly specialised membrane, acting as a barrier, which can selectively import and export different molecules. Without this barrier the cell could not control its chemical balance and it would die.

Damaging the genetic material can prevent successful multiplication of a cell, kill it, or in a worse scenario, it could proliferate unchecked, in the form of tumours. A lack of oxygen or damaging molecules involved in protein synthesis can also kill off a cell.

In order to improve your health it is essential that you remove toxins from your body and decrease the amount that you are exposed to. These toxins come in the form of those produced during metabolism, those taken in with food and other poisons which enter your body from the air we breathe, water we drink etc. To remove toxins from your body you have to start by getting them out of your cells. If the toxins are not removed from the individual cells then the toxins will accumulate in the tissue or organ which the cell is a part of and will damage them - causing disease either now or in the future. Once the toxins are out of the cell you need to remove them from the body. The main system involved in moving toxins from the environment surrounding the cell they have just been thrown out of to the organs of elimination is the lymph. It is important that you exercise to move your lymph around or manually move it around with massage.

Some toxic eliminations occur daily, such as defaecation, urination, sweating etc. Some occurs less often like the once a month release of menstrual blood. Other eliminations take a while for your body to build up to and then are very effective, for example vaginal discharges, vomiting and fevers. Fevers are characterised by a high temperature which is a sign that cells are working incredibly hard to throw off toxins and you will feel this cellular effort as heat.

If the organs of elimination are not functioning properly, other cells and organs can be literally bathed in the blood and/or lymph containing all these toxins. This is an unfortunate position to be in and is known as an aggravation. It is like stirring up a muddy pond and finding that the water goes murky until the bits of mud have settled on the bottom again. If you stir your toilets they will float around for a while, causing you pain and upset and if you don't release them from your body then they will probably go right back to where they came from.

It is easy to confuse the elimination of toxins with disease and be worried that you are sick. A disease which is related to toxins is one where the body that you are losing itself from unwanted toxins is known as an acute condition. A chronic condition is one where the body can no longer heal itself. To return to good health from a chronic condition you must go through the acute stage - you will get 'ill' as you start to get better and release toxins. It is important to be aware of this and to thank your body for helping you, even when you feel lousy with a throbbing headache and blisters all over your skin, or whatever. Before entering the acute stage in the recovery process from a chronic state you may feel really well. Do not be deceived and think that the hard work is over, this is your body preparing for a big piece of work - a healing crisis.

A healing crisis is an acute reaction taking place in a supported body. It is a very good sign that the natural healing powers of the body have been evoked and are fully functioning. Crises are regarded by most people as a sign of ill health, not recovery. The path to better health is the reverse of what made you ill and it is necessary to repeat past diseases. All those conditions that you have had which have contributed to the state you are now in will be removed from your body and you will feel it. It is a fantastic thing and afterwards you will be amazed at how well you feel.

If the eliminations are prevented from removing toxins it is known as suppression. Most drugs which you get from pharmacists 'work' by suppressing natures healing reactions. The reason that your rash goes away or your spots disappear is because they are pushed back inside your body only to come back more virulently next time. It does seem worth taking the time to go through elimination and reappear stronger and less toxic. Always bear in mind that it takes a long time to create a chronic condition, to actually damage your body that much - whether it is a lot of hamburgers or too many late nights - and undoing that damage will take some time too. You will revel in the difference.
“When I first found out about DNCB, I thought it was the most stupid thing I had ever heard about. I still think the concept of putting something on your skin to control AIDS is stupid (and I don’t blame others for thinking that it is stupid) but the evidence is there and the science supports my observations.”

Bill Goldberg

The DNCB Files

by GARETH JAMES

DNCB supports and strengthens cell-mediated immunity

When I first found out about DNCB, I thought it was the most stupid thing I had ever heard about. I still think the concept of putting something on your skin to control AIDS is stupid (and I don’t blame others for thinking that it is stupid) but the evidence is there and the science supports my observations.”

Bill Goldberg

The way in which we choose to treat a disease defines our understanding of its nature and its cause. Therefore, the effective use of DNCB in the treatment of HIV/AIDS heralds a potential renaissance in our understanding of the processes involved in developing the syndrome.

The DNCB Files is a compendium of literature, edited by AIDS activist George Delmerco from ACT-UP San Francisco, which describes itself as a “rag-tag scrapbook of E-mail, dense research, news stories and polemic.” As the pages turn, such an obvious under-selling sharply reveals it to be a compelling rethink of modern immunology and orthodox medical treatment strategies. Spanning 200 pages, its central claim is that weekly use of DNCB will specifically support and strengthen precisely that branch of the immune system which tends to fail during HIV/AIDS - the cell-mediated immune response. In contrast, The DNCB Files critiques and condemns the use of “anti-HIV” drugs (AZT, protease inhibitors and the usual suspects) arguing that they are not only short-lived in their successes but that they further suppress the cell-mediated immune response. Well presented, convincingly argued, The DNCB Files is, of course, a pharmaceutical company’s nightmare.

The opening pages boldly state that “It is evident that every single theory about how AIDS is caused and how it should be treated has been proven wrong during recent years. The truth about AIDS, rather than the dogma, is as follows: (1) HIV does not cause AIDS by killing T-cells; (2) CD4 (T4) cells are an invalid surrogate marker for disease progression; (3) CD8 (T8) cells (and the DTH Multitest) are valid surrogate markers; (4) antibody-based vaccines are immunosuppressive, toxic and don’t stop people from dying (except for PCP prophylaxis, which extends survival); (7) there is no latency period in AIDS; (8) all AIDS opportunistic infections (except PCP) are intracellular in nature; and (9) activating the cellular immune response is the only way to control HIV and intra-cellular infections.”

So, what is DNCB? DNCB is a chemical used to manipulate the immune system. The letters stand for dinitrochlorobenzene. It is a crystal, dissolved in various strengths of acetone, which is then swabbed on to a 2-inch square of the skin with a cotton bud where it becomes absorbed. The purpose of this seemingly bizarre exercise is to try and prompt a small, red and initially itchy rash. If successful, DNCB has started to produce a pure, systemic cellular immune response in the body. It is exactly this cellular immune response which is typically eroded and suppressed during HIV/AIDS.

The theory of how it works goes something like this: The small amount of DNCB absorbed through the skin is picked up by immune cells which then try to rid the system of the DNCB antigen and any other pathogen-infected cells. In 1993, AIDS scientists Mario Clerici and Gene Shearer established that people testing ‘HIV-antibody positive’ may start to induce a switch between two different types of CD4 cells. They found that there was an ongoing decline in CD4 cells which stimulate the cell-mediated branch of the immune system and an increase in CD4 cells which induce antibody responses instead. These two differing types of CD4 cells are usually referred to as TH1-type and TH2-type. Although no-one is clear as to how HIV causes this to happen, progression to AIDS is often characterised by this switch from cellular immunity (TH1) to antibody production (TH2). Weekly use of DNCB starts to reverse this process back towards TH1-type CD4 cells, thereby reconstituting the cell-mediated response.

The real point made by The DNCB Files is that with the exception of PCP, all the microbes which cause the opportunistic infections of AIDS, are harboured inside the body’s cells. Some immune cells can actually serve as reservoirs of microbes. Antibodies can only neutralise microbes once they have emerged from the cell. The only effective way to control cells which are infected is via the cell-mediated branch of the immune system.

The theory (if not the treatment) has attracted supporters from the most unlikely quarters of the AIDS industry. Eminent AIDS scientist Dr. Jay Levy has not only criticised our focus on killing HIV but has also lent his authority to supporting the restoration of cell-mediated immune responses. In a 1995 issue of The Lancet, Levy stated: “I submit that the lack of efficacy in present approaches to finding a solution to AIDS reflects a focus on the wrong target.” Levy has also stated that “Cellular immune activity against virus-
infected cells is the most important response for protection of the host and that "one could essentially control HIV and the consequences of HIV infection with a strong cellular immune response."

In March of this year, an independent report commissioned by the NIH urged a 'back to basics' policy for future funding priorities within the AIDS field. Although sparing no back-slapping for the previous decade's work (albeit fruitless), the report criticised funding efforts to help develop AIDS drugs 'as they appear less likely to have a long-term impact on the epidemic' and instead urged that funding should be redirected to gain a greater understanding of the pathogenesis of AIDS and to support promising avenues of research that have previously been under-funded or neglected. The DNCB Files comes in direct response to this call.

Since 1984, the 'one microbe - one disease - one cure' ideal has dominated our attempts to unravel the mysteries of AIDS. Accelerating competition amongst pharmaceutical companies to patent for profit that elusive 'magic bullet' has further obscured alternative treatment strategies for controlling AIDS. However, such a crystal clear admission by the NIH of our complete failure to understand (and successfully treat) AIDS in this exclusive context, lends tremendous weight to the DNCB message. The DNCB understanding of AIDS relies upon re-interpreting AIDS as an immunological disorder rather than treating it as a virological disease.

This sobering switch of perspective also fits snugly with many of the non-HIV based theories of AIDS. All the damaging risk-factors associated with the syndrome, namely: psychological stress - from receiving an HIV-positive diagnosis or living with AIDS-related illnesses; multi-microbial stress - from an accumulation of microbes other than HIV; toxic stress - from recreational drug use, certain vaccines and prescription drugs; and nutritional stress - from depleted antioxidant reservoirs, all specifically suppress the cell-mediated immune response.

AIDS scientist, Prof. Robert Root-Bernstein places the greatest emphasis on the role of risk-factors in suppressing this cell-mediated immune response. Published in the science journal Genetica in 1995, he notes that "a significant proportion of people repeatedly exposed to HIV become PCR positive (a test for the 'virus' itself) but remain antibody negative and healthy." He further states: "It follows that the lack of immunity is symptomatic of a failure of T-cell immunity (cell-mediated immunity). The issue in understanding AIDS now becomes that of establishing what causes the failure of T-cell immunity. Since this failure does not occur in a large proportion of people exposed to HIV... it is unlikely that HIV is, itself, the cause of this failure. HIV is more likely an opportunistic or synergistic infection that becomes manifest only in people predisposed to or with on-going causes of immune suppression." Root-Bernstein also acknowledged that "these data strongly suggest that the primary line of defence, and the only effective one against HIV is a T-cell response."

Coupled with the recommendations in the NIH report, incorporating risk-factor theories into the current model of AIDS should become a priority for healthcare professionals and seriously underscores our need to respect AIDS as a multifactorial phenomenon.

Unlike many alternative and non-orthodox treatments, DNCB has over the last few years attracted sufficient interest from the scientific community to merit a number of small trials and studies. The results have been extremely impressive and we now have a wealth of study data validating the claims and endorsing its use as a treatment for HIV/AIDS.

DNCB induces a gradual and consistent increase in cell-killing (cytotoxic) CD8 cells. These cells primarily clear pathogen-infected cells from the body. One established common trait for long-term survivors and non-progressors is that they all have high levels of cytotoxic CD8 cells. DNCB increases the numbers of CD4 cells though the increases are not as great as the CD8 cells. Natural killer cells, responsible for destroying tumours - Kaposi's sarcoma - and other foreign substances in the body, also increase in number during DNCB treatment. A major reduction in opportunistic infections and increases in weight are typically documented for DNCB trial participants. Subscribers to the modish wonders of viral load testing should also have no argument here. The latest trial data on DNCB presented at this summer's annual AIDS Conference in Vancouver demonstrated a mean drop in viral load of about 1 log over a 3-4 month period. Many trial participants experienced a reduction well over 1 log and the total average drop was from 150,000 to below 20,000.

The DNCB Files lists over 15 studies which testify to the efficacy and safety of this treatment. The only US-based study to concur with the findings of existing studies was conducted by the National Institutes of Health (NIH) itself, which announced DNCB failed to ameliorate disease progression. It comes as little surprise to discover the trial was deeply flawed in its design.

So, amid such positive reporting, where's the catch? Well in this case, the drawbacks of DNCB use appear slight indeed. Using DNCB alongside antiretrovirals is contra-indicated as these drugs are such powerful suppressors of cell-mediated immunity. Similarly, standardised herbal 'shotgun formulas' containing high levels of polysaccharides are also contra-indicated as they favour humoral immune responses and suppress cell-mediated immunity. Although there are no reported side-effects, sporting a 2" square rash every week somewhere on the body may deter the would-be DNCB user. However, in the words of George Delmerico: "I've gotten really annoyed with those who whine and mope about a small and almost always painless pink patch on their skin - usually in a place hidden by clothing in the first place. I want to slap these guys and ask, 'would you rather have Kaposi's blisters on your face? Get real!'" Perhaps best of all, DNCB costs next to nothing. The commitment to DNCB therapy by ACT-UP San Francisco means they will ensure anyone wanting to use DNCB can do so irrespective of their ability to pay. The Heal Trust also supplies DNCB at a cost of £3.00 for the complete starter kit (which should last about six months - postal service available throughout the UK).

DNCB is certainly not a cure for AIDS but even if your cellular immunity is already significantly compromised, DNCB may still be an appropriate treatment option. As treatment strategy is fast becoming recognised as the key to longevity and survival, then a weekly pulse of DNCB to strengthen cell-mediated immunity could become one of the most valuable tools in the survivor's tool kit. The DNCB Files, itself, is an articulate, first rate user's guide to a new paradigm for AIDS immunology.

Anyone who has been given a positive HIV-antibody test result or is immune-compromised should completely immerse themselves in this 'rag-tag' collection of data before making any decision about their treatment choices - it is an absolute must.

The DNCB Files and DNCB starter kits are available from:

The DNCB Treatment Group (ACT-UP San Francisco) Tel: ++1 415 954 8896 e-mail at <geodel@west.net> & The Heal Trust, Heal House, 375 Kennington Lane, London SE11 5PG Tel: 0181 265 3989, Fax: 0181 265 3972.
S
o, I finally gave in and went for the much dreaded CHEST X-RAY. The result? A 5cm cavity in the back of my left lung. My GP was so sure that this was “HIV-positive” Tuberculosis that he made an emergency appointment with the chest specialist at the Royal Bournemouth Hospital, Dr Williams.

Next day, off to Royal Bournemouth and Dr Williams to have another CHEST X-RAY to be sure. His office seemed to be filled with consultants talking over my X-RAY. I was taken to another room where I was told by Dr. Williams that he had ordered me a private room for 2 o’clock that afternoon. I was also told what I had was LIFE THREATENING and if I didn’t check into the hospital I would surely die. My buddy took me back to my flat to collect a few important things and we went on to an experience that I’ll never forget as long as I live.

Checking into the hospital I was assigned to the third ward and a private room – there were only elderly people on ventilators and me – it was funny at the time. The nurses seemed to be nice but I was to find out different later.

No sooner had I settled in my room when the Medicine Cart arrived and here were four pills for me to start taking in case I did have TB. I wonder what harm these drugs did to my system.

Eventually it was time to eat. At first I was excited but very soon I disliked this time of day or anytime that had to do with food, because within 10 minutes of eating I began to up-chuck everything. The only explanation I can think of was my body was fighting back all the different drugs that I wasn’t used to. After about three days of the same thing happening the nurses began to get the idea that maybe something was wrong. But the doctor said that my body was just getting used to the drugs and not to worry as a drip was to be put into me and if it were necessary I would be fed that way. There were more drugs for me to take anyway IV-style. I was on 11 different medications. They wanted to change the time I took my epilepsy medications to 6.00p.m. instead of at night when I have always taken them. Knowing what happens at dinner time I said no to that idea. This seemed to anger them as they had no control over me.

At night when I was burning up with fever I had my covers removed and the ice packs arrived. Each night I heard “I’m going to open your window so you can feel the fresh cold night air!” I thought: if they don’t kill me with drugs...

Two weeks passed, all my test results were returned – and they were all negative! I was taken off the TB drugs but in the two weeks I was on them I had lost 2 stone – and for me that’s a lot. The nutritionist would come into my room and tell me different things to eat that would put weight on me. What she didn’t tell me was that any food I would eat had such a bitter taste now that it was impossible to swallow.

Following this fiasco it was time for the doctors to do their rounds which meant you were a guinea pig and the student doctor who could figure out what was wrong with you won two weeks in Ibiza. Five of them stood around wondering what to do. One said “Let’s put a tube in him and drain the cavity.” All seemed to agree with the young handsome doctor and as this was his idea he was elected to tell me. He entered my room bringing some paperwork – a consent form for me to sign. I was so doped up the words were blurred on the paper so I had to ask questions such as:

Q. What does this mean? A. It releases the hospital from all liability. Q. What does that mean? A. Well if you should perish on the table we aren’t responsible. Q. Is this a difficult procedure? A. No. Q. Have many people lost their lives over this before? A. No.

After a lot of thought and with the assurance I would be given Holy Communion beforehand, I signed. It was Friday morning and by noon the priest was there to give me communion. Even though I have a Guardian Angel I was afraid. This had never happened to me before and I swear it will never happen again. After the priest left I was off to the specialist who would be putting the tube into me. I was assured there would be no pain even though they would not put me to sleep.

How stupid can one be to believe that when they cut a hole in your back and insert a tube there will be no pain?? The tube was put where they thought it should go and I was put in a corridor to wait for someone to fetch me. Half an hour passed and no one came. They were too busy or at lunch or something so a student finally came to take me back. She said that when I got to my ward they would attach a bag for fluid. That never happened. About an hour after these procedures I began to cough up mouthfuls of blood. When I rang for the nurse she assured me that this was a natural thing to happen. She said that whenever a foreign object was placed in the body you cough up blood. I said “Oh”, and when she left taking with her my blood I knew she was truly Full of Shit.

During the weekend I was given muscle relaxers and sleeping pills but none of these things seemed to work. Friends came to see me including Margaret Houghton, a social worker from E.A.S.T., an AIDS charity. She was alarmed when she saw me and reported it to the nurses who paid no attention. Other friends that came to see me were afraid that I was going to die then and there.

After the weekend had passed in which the nurses were injecting saline solution into the tube and getting back blood mixed with water, they realised that this wasn’t working as the tube was in the wrong place! I was so pumped full of drugs I didn’t know whether I was coming or going. After the three days of having the tube in me they decided to remove it. Mind you I was given nothing for the pain – they just pulled it out!

All seemed to agree with the young handsome doctor

T
he following day I was told that I would be discharged the next day - I was still not sure why, but I do know that the nurses were told I’d had a seizure and had slipped into dementia or something like it and the reply was “We’re too busy. The doctor has seen Mr. Heesey and there is nothing wrong with him.” When asked, “Why is he behaving so strangely?”, the nurses’ reply was “He’s play-acting to get attention. We’ve seen him do this before.”

Amazing! When I was released from Royal Bournemouth Hospital there still seemed to be a blank as to what was wrong with me. It was not AIDS-related and I thank God for the friends who took it upon themselves to get me out of the Hospital from Hell so I could begin what I’ve now achieved – a proper recovery.

by

MICHAEL HEESY
In years past, whilst I still believed the 'party line' about HIV and AIDS, I couldn't understand why Western women weren't dying in the numbers Western men, and African men and women, were said to be. After all, bi-sexuality is extremely common, and other 'sexually-transmitted' diseases never discrimi-nate.

You've taught me that HIV tests are complete bollocks, HIV doesn't lead to AIDS (if it exists at all) and the main components of AIDS are lies, damn lies and AIDS statistics. I think that anybody who tests positive should take a tip from Groucho Marx and refuse to be a member of any club who'd have them as a member!

When 'AIDS' cases first appeared in the States the quacks at the time said gay men were coming down with diseases of birds, cattle, etc., which were unknown in men. What was THAT all about? and where have all those 'unknown in men' diseases gone? Any ideas? I haven't!

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I started taking the recom-mended dose of 2 vials a day. I noticed that the Candida at the back of my throat was actually starting to spread all around my mouth and tongue. I assumed this was part of the process.

I very quickly into the treat-ment (2-3 days) I started to develop a very sore throat. When I asked if this could be the Allicin burning the throat membrane, I was told that it was more likely to be thrush 'digging in' for dear life, as it was under threat. The pain in my throat became so bad after one week that my sleep was continually broken, and my appetite diminished because of the sheer pain of swallowing – even yoghurt, soup, etc.

After 10 days other symptoms started to manifest – severe night sweats and day fevers, 'burning' sensation throughout the whole of my body, a sense of total debility, feelings of despair, despondancy and depression, breathing difficulties, tight constricted chest, dry cough, wheeiling, rapid shallow breathing.

I checked myself into Ealing Hospital for extensive tests to be a member of any club who'd have them as a member!

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LIVE, LIVE, LIVE!

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If you are an avid Continuum reader you will remember the life story on the last page of the October (95) issue by a person named GOLDIE GLITTERS. Well, we are one and the same, and I am going to start using that name for my column. I'm always asked where did I get my name. I got it from GOLDIE HAWN a long time ago, when she was on Laugh In. There are many Michaels but there is only one GOLDIE GLITTERS.

I'm moving house. Yes, since my ordeal with the hospital I have been offered a ground floor flat in Canford Cliffs. I'm lucky as I'm moving in with a friend who owns the flat and this isn't far from here. I do dread packing everything. Of course it's a chance to find things!

As I sit here watching the sun set I must tell you of two more wonderful things that are great for my and your health.

First, I've started taking acupuncture. It's the greatest, and I go to a woman doctor who has a lot of HIV+ patients. So she knows what energies to channel. This means no drugs whatsoever.

The second thing is a liquid you can get at your own health food store, called ECHINACEA PURPUREA. It's made from a flower and you mix the liquid with fruit juice. I take it and feel like a new person. Very soon it's going to be eighteen years since the battle of the bullfrogs, and I would hate to go back to the bullfrog days."

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I checked myself into Ealing Hospital for extensive tests to find out the cause of my demise. PCP, TB, extensive blood tests all came back negative, much to my relief.

I stopped taking Allicin as of yesterday morning. Last night was the first time in weeks I did not sweat. Although I do not feel 100% my normal energised self, I have a sense of regaining my positive attitude, and physical/spiritual well-being. I

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Lust for Life

Rafael Ramos travelled through different cultures and unexpected events following his antibody diagnosis, discovering how some people hide from life behind their confused assumptions.

We met mid-summer this year, high on ecstasy, dancing hedonistically to the rhythm of orgiastic-hum-drummed-music. There was little time for ritualistic courtship, other than the usual smile and an irresistible erotic attraction. We knew what we wanted and later spent long hours kissing and melting our bodies in amazement. I ended up with very raw lips and a red nose that lasted almost a week.

Our second encounter was spent in bed followed by a romantic Sunday walking for miles in the countryside, including a sunset punting in Oxford. Thereafter uncertainty began with big questions marks. He had just finished with his partner of 12 years and asked me if I had taken the AIDS test. Oh no, it’s so soon! I thought. How can I get around this? I better be economical with the truth and change the subject - I don’t even know what is going to happen next. In any case, how can I expect anyone to suddenly digest the information I have absorbed over the past two years since I was diagnosed?

How can I convince ingrained believers like him that HIV is not a death sentence or truly dangerous? More to the point, how can anyone comprehend that the presence of HIV in the blood has not been clarified and that testing positive does not show any specific pathogen? What it means is, perhaps, exposure to some endogenous or exogenous agents (like Candida, Glandular Fever ... ), stress factors or oxidising factors.

If only lab-technicians were allowed to reveal the limits and flaws of the HIV-tests kits, many threats to life could be avoided. Why then tell anyone about my unspecific HIV-status? Who needs to spread a tidal-wave of “health-fascism” – this tendency to unknowingly encourage blame, guilt, pity, paranoia, distress, anger, hatred or rejection by promoting a genetic entity as a maker for ill-health, when in fact, ‘it’ has not been biologically isolated. Is it not true that a positive diagnosis for Hepatitis B means that the body or a vaccine has created a defence mechanism against yet another ‘virus-like-parcical’, which has not been properly defined? Can anyone really explain to me then, how any of them actually operate?

My efforts to disentangle my own psyche from the shackles of the HIV-myth began soon after I became diagnosed positive in June 1994. My immediate reaction was to blame my promiscuous lover, yet I decided to contact my friend Edward instead. Looking back now, I consider myself fortunate. My initial shock had been clearly perceived by Edward. We walked and discussed the politics and controversies of the HIV/AIDS-hypothesis. After a few hours, he could see I was able to stand on my own two feet, enough for me to realise that this life-threatening ‘diagnosis’ I had been hoisted by, was only a transitory condition. I had understood his message and the seeds of hope and reason had been planted. Eventually, my friend’s efforts paid-off and would lead to a Grade A on my final year’s dissertation.

From that moment on I had a lot of homework to do. I needed to tell my lover and waited two weeks for the moment to arise. He took it calmly and we wept and hugged in bed. Three days later, bang, wham, shit hit the fan. He started tearing up photos and anything else he could get his hands on. The fear and anger had to come out somehow, like a baby burstng out crying just to find some peace minutes later. The following weeks and before we were ready to go on holidays to Spain, I visited Meditel and read volumes on the subject of AIDS. The more I read about the way pharmaceutical companies had coaxed the politicians and medical research, the more angry and disconcerted I became. I stopped reading. Soon after we arrived in Spain I came down with Candida and exhaustion. Travelling through the heart of my own country, especially in Cuenca, one of the most enchanting and haunting places of Castille, I realised how beautiful and precious life can be. My boyfriend had taken some self-healing books by Louise L Hay and Dan Millman. I began soaking up and practising their wisdom without delay. I also knew that I had a whole year ahead of me while studying French in Strasbourg and there I could set out to discover and use this new concept of personal development.

Once in France I joined Aides Alsace, a voluntary federation, somewhat equivalent to Body Positive. I wanted to know what it was like to hear about the ‘disease’ from the best horse’s mouth in town. Basically, I needed to understand why people take drugs like AZT which pretty reliably will make them sick. What drives good-hearted volunteers and paid staff to work with HIV/AIDS-sufferers and death? How self-help groups which initially began as dissident groups to educate society about homosexuality and “safer-sex”, later and unlike Continuum, became enmeshed in politics with little choice but to follow conventional wisdom. And, why positive diagnosed people within this type of organisation knowingly and happily took and promoted AZT, isn’t AIDS an immune-related syndrome? Don’t you know that AZT effectively destroys the very system you’re trying to help heal, I kept asking? In general, I found these chari-
Lust for Life continued

table people were helpful, warm and
generous. They were doing what they
could but unaware that they were
encouraging a belief-system of fear
and despair.

To some extent, I too became
convinced of Montagnier’s co-
factors story. Yet my life-long
voyage of self-discovery and change
had begun. I was determined
to slowly overcome my barriers and go
beyond, towards a more peacefull
existence, both internal and external.
I swam 3-4 times a week, drank and
smoked less and ate better. I started
meditation, massage, re-birthing, urine-
therapy and practiced Louise’s
techniques as a stepping stone
to improve the quality of my thinking.
I met a wonderful woman, Rose, a
volunteer herself who lodged me in her
beautiful apartment and introduced me
to nutrition and more. By then, my
partner in London had surprised me by
finding someone else and the damned
Candida had flared up once again.
Rose took me to a nutritional G.P. who
put me on mega-doses of injectable
vitamins and other supplements. In the
end this fungus and its symptoms
disappeared.

When I returned to London, I knew I
had to confront my ex and dreaded the
moment. More drama lay ahead and I
could not allow him to run my head.
Deep inside, I was aware he had done
me a favour, our relationship had done
to my emotions what nicotine does to
my lungs. I realised ages ago, that co-
dependency can be as difficult a habit
to kick as smoking. More than a habit –
a obsession, a physical addiction, an
oral fixation. I enjoyed sex with him,
more than with anyone before him. He
was playful, funny, crazy-fun, but
mostly verging on the absurd. He used
to nick-name me: my "Chupa-Chus"
(my lolly-pop), then Camel-face and
later Carrot-top, which my friends and I
found hilarious. Predictably, after
another dramatic break-up, we did not
speak or see each for months. But
eventually I learnt to swallow my
Spanish-pride and accepted my
responsability for what had gone wrong
between us. We have now resolved our
differences and are on friendly terms.
Today, I've finished my degree and am
happily volunteering full-time at
Continuum and one evening a week at
the London Lighthouse.

Becoming HIV-diagnosed triggers a
host of different responses. Among
other things, people need support in
their post-diagnosis, and perhaps
counseling. Generally, we need confi-
dence that there is clear and well-
formed information about the cause
and effects of a hypothesis like HIV,
and what is good-health – not just toxic
therapies like AZT plus. Unfortunately,
the Establishment, HIV/AIDS organisa-
tions and the media alike, have failed to
tackle or answer the questions
raised by Duesberg and Eleni. I
strongly believe therefore that the
simple antibody test for HIV is toxic, it
poisons the mind with the potential of
terifying and destroying anyone who is
possessed by ‘it’.

Fortunately, I come from a humble
background. I grew up in a farming
community of “agropecuarios”
(“somewhere out in the sticks”), and
although my parents could not possibly
show me a clear path towards uncon-
ditional love and acceptance, they did
provide me with the necessary tools to
grow and develop a healthy living.
Moreover, animosity towards doctors
and medication runs in the family which
helped me to learn the basis of good
health, i.e. nutrition and to take respon-
sibility for my own actions. After all, my
little sister died due to a negligent
doctor and my parents’ lack of under-
standing.

When adversity confronts a man with
himself, he has nowhere to run but to
face himself. First find love within
yourself to be able to share it with
others, then choose the pathway to
self-contentment, which ever that
might be. When a prospective lover
asks you: have you ever taken the
AIDS-test? (Oh God, not again), well
just pay attention to what you may say,
because you might scare him/her
away. Or, you might ask yourself, who
do I want to victimize – myself? In the
past, similar situations have arisen,
even with friends. They tend to
overreact, sometimes cry and others act
irrationally when sperm falls on their
skins. There are no specific recipies for
big silly questions like this, but be
aware of the devil you might unleash.
One thing I have learned from the
English culture is to take everything with
a pinch of salt, and later respond when
and if the circumstances are more
conducive.

In my case, no potential lover has
materialised. It was a beautiful
encounter and after some residual
interest on his part, he ran away
scared. Perhaps of himself. He did say
that the life of his HIV+ friends had
been fucked up by the diagnosis and
therefore he would never take the test.
In the end, I did not think that
announcing a ‘condition’ I have been
labelled with was going to alter any
value. Not without at least having been
given the chance of being loved for
who I am. The pursuit of happiness
begins when you are ready to listen to
yourself – there is however an indepe-
dent yet unhappy world out there. And
because life is deep and precious, in
this story, love is the only truthful logic.

Ah Love! could thou and I with Fate
conspire
To grasp this sorry Scheme of Things
entire.
Would not we shatter it to bits - and then
Re-mould it nearer to the Heart’s Desire!

-Omar Khayyám

by MAVIS CRUET
the (Welsh) Fairy

It was Canada’s turn to host the massive
pharmaceutical industry bash which
laughingly passes for The International
Conference on AIDS in July. Over four
days, a variety of pious pseudo-experts
were given platform to air the “latest
breakthroughs” (sic) in what passes for
medical research. Wisely, Prime Minister
Jean Chrétien stayed well away from
the whole affair.

Oh, how we laughed at the science
fiction routines of Scott Hammer,
Martin Markowitz and David Ho et al.,
Paul Parren and his performing mice,
Martin P Cranage and his macaques (in
fact there were several star turns
involving monkeys), Antony Fauci and
Jay Levy doing a duet; and Robert
Gallo’s rendition of “My Way” was his
usual Vegas-style ham performance.
And where did they get all those brilliant
foreign comedians from?

Following the traditions of all music
hall acts before them, there were no
subjects too ludicrous or dangerous to
 tackle – how about home testing? A
little job of spit or urine (fluids which
have never been implicated in transmis-
sion of this “clever virus” before) and
away you go down the Yellow Brick
Road to a brighter, triple or quadruple-
drug regime future! Or how about:

“Fancy a change? Try Protease – it’s
new, it’s theoretical, we’ve got drugs
for it!”

If the whole shebang was not so very
frighteningly obvious in its efforts to
get those poor sick (sic) bastards
[victims] out there to ingest higher
doses of untried, untested drug combi-
nations and make the fat cats in the
biotech industry even fatter, it would
indeed have ranked as one of the
funniest shows off Broadway.

But, as we see yet again promotional
material by drug manufacturers being
passed off as real “news” – such as
recent Ritonavir coverage on national
radio and in the papers – the whole
macabre spectacle of this annual fiasco
seems more and more reminiscent of
Les Misérables: a tragic overblown
fantasy which goes on and on without
critical challenge or real reason to be
taken seriously, except of course by itself!!!