# THE HIV-AIDS QUESTION

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## INTRODUCTION

The first descriptions of AIDS cases in the scientific literature are from the early 1980s. They suggest a possible acquired immune deficiency as the underlying factor responsible for the increase of particular recurring diseases among very marginal groups of people in certain American conurbations such as New York, San Francisco and Los Angeles.

All the members of these particular risk groups were identified as having health-hazardous lifestyles. The diseases were exclusively limited to the behavioural risk groups.

Acquired immune deficiency syndrome, as the USA's Centres for Disease Control (CDC) named it in 1981, was identified as a possible behavioural disease.

Up until 1983, AIDS was a disease relegated to big cities in the USA and Europe. In April 1984 it was announced that a newly discovered virus, HIV, was the cause of AIDS. Since then the world has lived under the threat of this permanent epidemic of potentially lethal consequences.

I have been asked by a group of concerned South African Muslims about the nature of AIDS and the bleak prospect of the AIDS epidemic in the African continent. In the media we see a constant update of the figures associated with AIDS. Apocalyptic estimates from different international health agencies predict a catastrophic future.

## I. THE CURRENT DEFINITION OF HIV-AIDS

The acronyms HIV and AIDS mean respectively '**H**uman **I**mmune-deficiency **V**irus' and '**A**cquired **I**mmune **D**eficiency **S**yndrome'. HIV is attached to AIDS, as in 'HIV-AIDS', to indicate that HIV is the cause of AIDS.

In any current medical text book we find that HIV-AIDS comes under the category of 'Sexually Transmitted Diseases' (STDs).[131]

### Epidemiology

Acquired Immune Deficiency Syndrome was first described as a clinical entity in 1981, and HIV was identified as the causative organism in 1983. In December 1997 the World Health Organisation (WHO) estimated that 29 million adults and 1.5 million children were already infected, and that up to 16,000 new infections occur daily world-wide. The WHO long-term projections estimated a cumulative total of over 40 million infections by the year 2000.

HIV is predominantly concentrated in developing countries among people in early adult life. Although HIV can be isolated from a wide range of body fluids and tissues, the majority of infections are considered to be transmitted via semen, cervical secretions and blood.

### The cause

HIV is considered to be the cause of AIDS. HIV is a lentivirus (slow-virus) of the family of retroviruses. There are at least two types, HIV-1 and HIV-2. HIV-2 is almost confined to West Africa although there is evidence of some spread to the Indian subcontinent. It is associated with an AIDS-type illness.

Retroviruses are characterised by possessing the enzyme Reverse Transcriptase, which allows viral RNA to be transcribed into DNA, and hence incorporated into the host cell genome (the DNA of the cell infected by the virus). Reverse transcription is a highly error-prone process with a significant rate of mis-incorporation of bases (wrong assembly of DNA bases). This, combined with the high rate of viral turnover, leads to considerable genetic variation and a diversity of viral subtypes.

**Transmission**

*a) Sexual intercourse*

World-wide, heterosexual intercourse accounts for the vast majority of infections, and co-existent sexually transmitted diseases, especially those causing genital ulceration, enhance transmission. The passage of HIV appears to be more efficient from men to women, and from men to the passive partner in anal intercourse, than vice versa.

Homosexual transmission still accounts for the majority of infections in the USA and Europe, but there appears to be an increasing rate of heterosexual transmission in developed countries. Up to 18% of infections in Europe are thought to be heterosexually acquired.

In central and sub-Saharan Africa the epidemic has always been heterosexual, and more than half the infected adults in these regions are women.

South East Asia and the Indian subcontinent are still in the early phases of a possible explosive epidemic, driven by promiscuous heterosexual intercourse and a high incidence of other sexually transmitted diseases.

*b) Mother-to-child transmission*

Vertical transmission is the most common route of HIV infection in children. European studies suggest that 15% of babies born to HIV-infected mothers are likely to develop AIDS, in the USA and Africa the rates of mother-to-child transmission are up to 40%.

Breast-feeding has been shown to increase the risk of vertical transmission by up to 20%. Vertical transmission can be reduced by the use of zidovudine (AZT) and the numbers of infected children have fallen in areas where zidovudine is used.

c) *Contaminated blood, blood products and organ donations*

Screening of blood and blood products was introduced in 1985 in Europe and the USA. Prior to this, HIV infection was associated with the use of clotting factors for haemophilia and with blood transfusions.

The practice of sharing needles and syringes for intravenous drug misuse continues to be a major route of transmission of HIV in Europe, USA, South East Asia and Latin America. Health care workers have a risk of approximately 0.3% following a single needle stick injury with known HIV infected blood.

## Pathogenesis

HIV attacks the immune system of the host. HIV recognises the CD4+ molecule in the surface of T4 lymphocytes. By interaction with this molecule, HIV enters the T4-cell. After transferring the viral genetic material into the cell DNA, it uses the cell machinery to produce its own viral particles, and finally destroys the T4-cell. Studies of viral turnover in HIV-positive individuals have demonstrated a virus half-life in the circulation of about six hours. Virus production by infected cells lasts for about two days and is probably limited by the death of the cell owing to direct HIV effects, linking HIV replication to the process of CD4+ destruction and depletion. Cell mediated (macrophage) immune deficiency is the major consequence leaving the host open to infections with intracellular pathogens, whilst the coexisting antibody abnormalities (decrease of T4-cells) predispose to infections with capsulated bacteria.

## Diagnosis

HIV infection is diagnosed either by detection of virus-specific antibodies (anti-HIV) or by direct identification of the viral material. These antibodies have no protective function and some of them persist for life, like IgG gp120, except in babies from HIV infected mothers, where they are lost gradually over the first 18 months of life.

Other antibodies like IgG p24 disappear as the disease progresses.

Viral p24 antigen (p24ag) is detectable shortly after the infection but has usually disappeared by 8-10 weeks after exposure.

## Laboratory

The blood abnormalities include lymphopenia (low count of lymphocytes) with atypical reactive lymphocytes, thrombocytopenia (low count of platelets) and raised liver enzymes. CD4+ lymphocytes may be markedly depleted and the CD4+/CD8+ ratio reversed.

Antibodies to HIV may be absent during the early stage of infection although the level of circulating RNA is high and p24 core protein may be detectable.

## Clinical latency

The majority of people with HIV infection (HIV-positive test) are asymptomatic for a substantial but variable length of time. However, the virus continues to replicate and the person is infectious. Studies suggest a median time of 10 years from infection to development of AIDS, although some patients progress rapidly and others remain symptom-free for up to 20 years.

Gender and pregnancy *per se* do not appear to influence the rate of progression.

## Clinical features of HIV infection

The spectrum of illnesses associated with HIV infection is broad, and is the result of both direct HIV effects and the associated immune dysfunction. The classification depends to a large extent on definitive diagnoses of infection, which makes it more difficult to use in those areas of the world without sophisticated laboratory support. As immune-suppression progresses, the patient is susceptible to an increasing range of opportunistic infections and tumours.

There are 29 diseases included in this syndrome, none of which is new, meaning that most of them have well-known causes other than HIV, like funguses, bacteria, mycobacterium, and viruses. Still others, like various cancers and neoplasms, have no established aetiology, and others like dementia or wasting syndrome have on their own many different causes. All of the following diseases are classified as AIDS-indicator diseases when there is a positive HIV-test:

1) Bacterial infections, multiple or recurrent (applies only to children)
2) Candidiasis of bronchia, trachea or lungs
3) Candidiasis of oesophagus
4) CD4+ T-lymphocyte count <200 cells/microliter (or a CD4+ percentage <14)
5) Cervical cancer, invasive
6) Coccidioidomycosis, disseminated or extra-pulmonary
7) Cryptococcosis, extra-pulmonary
8) Cryptococcosis, chronic intestinal
9) Cytomagalovirus disease other than retinitis
10) Cytomegalovirus retinitis
11) HIV encephalopathy (dementia)
12) Herpes simplex, with esophagitis, pneumonia, or chronic mucocutaneous ulcers
13) Histoplasmosis, disseminated or extra-pulmonary
14) Isosporiasis, chronic intestinal

15) Kaposi's sarcoma
16) Leukoencephalopathy, progressive multifocal
17) Lymphoid interstitial pneumonia and/or pulmonary lymphoid hyperplasia
18) Lymphoma, Burkitt's
19) Lymphoma, immunoblastic
20) Lymphoma, primary in brain
21) Mycobacterium avium or mycobacterium kansasii, disseminated or extra-pulmonary
22) Mycobacterium tuberculosis, disseminated or extra-pulmonary
23) Mycobacterial diseases, other disseminated or extra-pulmonary
24) Pneumocystis carinii pneumonia
25) Pneumonia, recurrent (within a 12-month period)
26) Salmonella septicaemia, recurrent
27) Toxoplasmosis of the brain
28) Tuberculosis, pulmonary
29) Wasting syndrome, HIV

Numbers 3, 4, 10, 15, 17, 21-25 and 27-29 are considered 'definitive diagnoses' or 'presumptive diagnoses'.

In Africa the clinical indication with or without a positive HIV test is: fever for more than two months + diarrhoea for more than two months + loss of body weight of more than 10% + persistent cough.

**Treatment**

Despite the introduction of new anti-retroviral drugs there is still **no cure** for HIV and AIDS, so the patient must live with a chronic, progressive, infectious and unpredictable condition.

The principal aim of anti-retroviral drug therapy is to suppress viral replication to as low a level as possible for as long as possible, in order to delay the progression of the disease. The evidence shows more benefits from anti-retrovirals used in combination. However, none of the present combinations eradicate HIV. Inhibitors of HIV-reverse transcriptase and of HIV-protease are so far the most developed.

The scientific rationale for early intervention—the 'hit early and hit hard' philosophy—is based on the rapid turnover that has been demonstrated throughout all stages of infection, and the view that the earlier and the more thoroughly viral replication is

suppressed, the less likely the immune system is to be damaged and the virus to develop resistance. Treatment is initiated at least with two or three drugs. The potential for adverse reactions and drug interactions is greater with more drugs.

However, it must be remembered that HIV has a long period of clinical latency despite continuing viral activity. None of the current therapeutic regimens is likely to be able to eradicate the virus from an individual, and there is a lack of data on the long-term effectiveness and toxicity of anti-retrovirals.

During pregnancy, HIV-positive mothers are treated with AZT to treat or prevent the development of AIDS, and also to reduce the risk of transmission of HIV to the foetus.

## II. EPIDEMIOLOGICAL APPROACH TO THE HIV-AIDS THEORY

HIV is attached to AIDS to indicate that HIV is the cause of AIDS, and that is because there was a time when there was only AIDS, and the cause of the syndrome was thought to be something other than HIV.

An accidental transitory disease that attacks large numbers of people at the same time and in the same country, region or place, is called an epidemic. Epidemiology is the science that deals with epidemics.

Medical science has long known about epidemics and has developed a very precise body of principles and rules by which it is possible, when applied to the phenomenon under scrutiny, to deduce by their distribution patterns the nature of the cause of a particular pathology which occurs at the same time among large groups of people.

Let us therefore start by approaching the phenomenon of HIV-AIDS from an epidemiological perspective.

That is to say, phenomenologicaly speaking, that we are going to look at its geographical features:—where it happens, when it happens, to whom it happens, how it happens—meaning, in Heideggerian terms, that we are looking for what is the "thereness" of the phenomenon. Let us see, then, what the phenomenon tells us from itself, as it stands alone as itself.

### The geography of AIDS

It was the U.S. Centres for Disease Control (CDC) which first reported in 1981 the growing number of male homosexuals, intravenous drug-users and some risk groups such as haemophiliacs and recipients of blood transfusion who were affected by previously known diseases such as: Kaposi's sarcoma, bacterial and fungal (pneumocystis and candida) pneumonia, oral yeast infections, diarrhoea, herpes, tuberculosis, weight loss, toxoplasmosis, and so on.

What was new was that the diseases were accompanied by a particular depletion of a group of immune cells—T4 lymphocytes—and that the epidemic spread non-randomly, selectively targeting a very specific group of people. Assuming, because of the T4-cell depletion, that immune deficiency was the common denominator, the Centres for Disease Control named it Acquired Immune Deficiency Syndrome, or AIDS (CDC 1981 1b).

A similar non-random epidemic was also reported in Europe by the WHO, affecting selectively the same group of people.

Then, in 1983, the French research group led by Luc Montagnier[5] and in 1984 the USA group led by Robert Gallo[6] presented papers published in *Science* claiming that both had isolated HIV.

The public announcement that HIV was the cause of AIDS was made in April 1984 at an international press conference in Washington by the Secretary of Health and Human Services, Margaret Heckler, and Robert Gallo, researcher at the National Institute of Health.

They announced that a new type of virus—a retrovirus—was the agent responsible for the epidemic.

As epidemiological features, viral and microbial epidemics have in common:

1) They rise exponentially and decline within weeks or months of infection. The classical epidemic curve is a bell-shaped curve that expresses the rise reflecting the exponential spread of the contagion, and the fall reflecting the resulting natural immunity of survivors.

2) The epidemics spread randomly in the population.

3) The resulting infectious diseases are highly specific, reflecting the limited genetic information of the causative microbe. As a consequence the viral diseases are typically more specific than those caused by the more complex bacteria or fungi.

4) The microbial and particularly the viral epidemics are self-limiting and thus typically seasonal, because they induce anti-microbial and viral immunity and also encounter genetically resistant hosts.

Being an infective agent, when we refer the HIV viral hypothesis to the epidemiological principles of infectious diseases, a particular contradiction begins to emerge. This is because it goes against two of the most fundamental epidemic principles of infectious diseases: firstly the HIV-AIDS epidemic is non-random, and secondly the development of HIV-antibodies by the host, after the encounter with the virus, gives no natural immunity.

**Non-randomness**

The first striking feature is the non-randomness of the epidemic. The *sine qua non* condition for an epidemic to have an infectious agent as a cause, is that it is random.

More astonishing is that from its beginning to this day (24 years), the AIDS epidemics of the USA and Europe have still remained highly non-random:

- "Approximately 90% of all patients in the USA and 90% in Europe are males.
- About 2/3 of all AIDS cases are male homosexuals (82% of all males).
- About 1/3 are male and female intravenous drug-users, 75% of which are male.
- One per cent are haemophiliacs and other transfusion recipients.
- One per cent are children born to drug-addicted mothers."

(World Health Organisation 2001 1a).

This also means that all of the heterosexual male cases are intravenous drug-users, except for a small fraction of haemophiliacs. And all of the women are intravenous drug-users.

According to the WHO (report 2001, 1b), since 1981 the AIDS epidemics of the USA and Europe increased steadily for a decade and, after reaching peaks in the early 1990s, all declined to currently about 1/2 of their peak levels. By 2001 the U.S. epidemic had generated a cumulative total of 816,149 AIDS cases, and the European epidemic 251,021 AIDS cases.

Not only the selective group distribution of the disease is non-random, but even the disease is distributed non-randomly among the selected risk groups:

- "Kaposi's sarcoma is exclusively diagnosed among the male homosexual risk group regularly using nitrite inhalants. The rest of the most common diseases in the homosexual group are lymphoma, dementia, weight loss, yeast infections and pneumocystic pneumonia.
- The children from mothers using psychoactive drugs during pregnancy commonly present: bacterial pneumonia.

- Among the intravenous drug-users and 'crack' (cocaine) smokers, tuberculosis, pneumocystis pneumonia and dementia are more prevalent than in any other risk group.

- Haemophiliacs and other transfusion recipients in the USA and Europe usually develop: pneumonia and yeast infections.

- The non-random distribution of these diseases in different risk groups, then and now, suggests risk-group-specific causes, rather than a common one."[106]

Another reason why the non-random feature of the epidemic is extremely interesting is because it implies that if the cause of the epidemic is the HIV virus, the virus, quite extraordinarily, is able to discriminate among its victims:

Discrimination by life-style (behaviour patterns): It has a particular tropism for a specific and limited group of people, as we have just mentioned (homosexuals, intravenous drug-users, haemophiliacs, and so on).

Discrimination by gender: 90% men (82% of whom are homosexuals), 10% women (almost all of whom are intravenous drug-users).

**No natural immunity**

All viruses are most pathogenic prior to anti-viral immunity—before the body's immune cells have identified them. Once the viral proteins have been spotted, the defence mechanism responds by producing a very specific group of antibodies, IgG, against these viral proteins, and the virus is neutralised. The whole rationale of immunisation is precisely to give the immune system the possibility to pre-empt the attack of a virus by creating in advance antibodies against the virus. So the antibodies are the confirmation that the patient has developed immunity to the virus.

By definition, HIV-AIDS is observed only after HIV immunity is established, that is, after the development of those antibodies which later are detected by a positive HIV test result. Ironically, in this case, antibodies do not mean immunity at all, but in fact quite the opposite: they are the confirmation, by definition, of a fatal disease.

Yet at the same time it is very difficult to find the virus in AIDS patients.

> "HIV can only be 'isolated' from rare, latently infected lymphocytes that are cultured for weeks *in vitro*, away from the antibodies of the human host."[107]

Even in patients dying from AIDS, less than 1 in 500 of the T4 lymphocytes that become depleted are ever infected by HIV.[108] Currently, this difficulty in finding the virus would be a confirmation of natural immunity, meaning that the antibodies are so effective that no HIV is detectable in the AIDS patient. Yet, in spite of the presence of the antibodies against HIV-proteins, the immune system continues to show low counts of T4 lymphocytes. If the decrease of T4-cells is due to the action of HIV, natural immunity in this case, for some reason, is no longer efficient.

Unless we now begin to revise the foundations of immune response, we may have to admit that either a) these antibodies are not against viral proteins and for that reason cannot kill the virus, or b) that there is no virus to be killed, meaning that the proteins which the antibodies are against, are not from HIV, and that is why HIV is so difficult to find.

Since 1984 researchers have been trying, still with no success, to develop an AIDS vaccine.

One might imagine that since the HIV virus has such peculiar characteristics, developing a vaccine would never be possible, because in the case of HIV natural immunity is inoperative.

These extremely specific features of the AIDS epidemics in the USA and Europe would suggest that it cannot be produced by an infective, transmissible agent.

Having said all that, the puzzling epidemiological picture becomes even more peculiar when we look at the epidemic in Africa.

The African epidemic, it is claimed, emerged in sub-Saharan Africa from 1984 onwards, and according to the WHO increased until the early 1990s, similar to the epidemics of the USA and Europe, but has since levelled off to cause about 75,000 deaths annually (WHO 2001 1b). By 2001, Africa had reportedly seen a cumulative total of 1,093,522 cases (deaths) (WHO 2001 1b).

The African epidemic contrasts in its features very sharply with its American and European counterparts:

- The first striking picture is that the African HIV virus no longer discriminates by gender, precisely one of the more peculiar characteristic of U.S./European HIV. In the sub-Saharan region of Africa, the AIDS epidemic is randomly distributed between sexes.

- The second striking feature to reveal an enormous difference between the U.S./Europe epidemic and the African one is that in Africa, the AIDS epidemic is not restricted to behavioural risk groups, whereas in the U.S./European case, the epidemic is almost entirely limited to a very specific risk group.

- The third difference of the African epidemic involves the diseases that the African AIDS produces. The African epidemic is a collection of long-established indigenous diseases, such as chronic fevers, weight loss syndrome (slim disease), chronic diarrhoea and tuberculosis. The predominant and most distinctive AIDS diseases in the U.S./Europe epidemic, pneumocystis carinii pneumonia and Kaposi's sarcoma, are almost never diagnosed in Africa.[106]

Reflection on this paradoxical epidemiological evidence suggests that the paradox may not be created by the evidence itself, but rather by the correlation made of this evidence under one single definition, that is, the affirmation that these diverse phenomena are caused by one single transmissible agent: the HIV retrovirus.

Indeed, there are no paradoxes in nature: only flawed hypotheses.

Having assessed that it may be the definition that creates the apparent paradox, we need to look now at the evidence that sustains that definition: the theory of HIV-AIDS, and more particularly its most defining tool of all, the HIV test, to see if the phenomenon can reveal itself, from itself, as itself.

## III. MORPHOGENESIS OF THE HIV-AIDS THEORY

### Cancer research

In modern medical text books we find, under the grouping of RNA viruses, the retroviruses. More specifically, the 'human lymphotropic retroviruses', whose name indicates their affinity to lymphocytes:

- HTLV-I and II are oncoviruses and are described as lymphocytic, meaning they stimulate the production of lymphocytes, causing a malignancy of the T CD4 lymphocytes called human T-cell leukaemia-lymphoma.

- HIV-1 and HIV-2 are lentiviruses (slow viruses) and are lymphopenic, meaning they destroy lymphocytes, causing AIDS.

All of these were discovered by the same man: Robert C. Gallo.

Robert Gallo was a man in pursuit of a particular type of virus: a retrovirus with cancerous capacity. Robert Gallo, researcher at the National Institute of Health, was one of the many virologists involved in President Nixon's decade of War Against Cancer.

In the mid 1970s Gallo claimed to have discovered the first human retrovirus in patients with leukaemia. He called the retrovirus HL23V.[1, 2]

Years later, when AIDS started to emerge, the first patients reported to have developed AIDS in the USA were young male homosexuals suffering from pneumocystic pneumonia and/or Kaposi's sarcoma, a rare form of cancer otherwise only observed in elderly people. The young age of the patients, alongside the disease's particular tendency to affect the lungs, made these cases even rarer. To oncovirus researchers like R. Gallo, these appeared potentially to be the work of a retrovirus.

Theory means 'to look at'. From where you look, you see. The platform from which this phenomenon was observed, was cancer research.

Retroviruses are the offspring of cancer research.

The hypothesis that infectious agents could produce cancer had been on the scientific horizon for quite some time. Research on animal tumour-cells by Ellerman and Bang in 1908, and by Rous in 1911, led to the first description of an agent separable from the tumour cells that could induce, following inoculation, leukaemia or sarcoma in healthy animals. In neither case did the malignancies occur naturally, rather the experiments

entailed the production of acellular ultra-filtrates from artificially leukaemia-inbred laboratory chickens or induced sarcomas on a laboratory fowl. Since the ultra-filtrates contained no cells, the information had to come from genetic material, and the hypothesis was that the agent had to be a virus.

In the 1950s Ludwick Gross found retroviruses that caused tumours in mice and chicken.

All the animals in the retrovirus studies were inbred laboratory strains. Moreover, many of the infections were congenital.

From 1955 onwards, under the direction of James Shannon, the USA's National Institute of Health (NIH) promoted cancer research and received extraordinary budgets from Congress to finance major programmes in the fight against cancer. These included the search for oncoviruses and the development of cancer drugs.

In 1960 the feline leukaemia virus (FeLV) was discovered by W. Jarret. The virus was capable of causing not only malignancies in blood cells, but also aplasias (insufficient growth of affected cells) and an immune deficiency.

All of these animal oncoviruses are now classified as retroviruses.

All of the retroviruses had been found in lymphocytes. They were the outcome of laboratory experimentation on animal lymphocytes, diseased by induction or naturally.

New hypotheses were developed in the 1960s by Duesberg that a virus could carry a cancer gene (oncogene), and its introduction into the cell DNA would later be responsible for that cell becoming cancerous. Peter Duesberg, a brilliant scientist from Berkeley, had mapped a particular mutation to a single nucleotide in what was to become known eventually as an oncogene. Duesberg was named California Scientist of the Year for developing the theory that oncogenes might be introduced by viruses into humans and cause cancer. Years later Duesberg found flaws in his own theory and announced this to his surprised colleagues, who were working on demonstrating that it was highly unlikely. Despite this, research on the viral oncogene hypothesis continued fruitlessly for the next ten years.

It is important to point out at this stage that these viruses were thought to be exogenous viruses, meaning they come from the outside. However, the human organism also contains internal, or endogenous, viruses. Our bodies contain countless retroviruses, which have been in the human genome since the beginning of human life. Endogenous retroviruses are regarded as evolutionary genetic remnants, bits of DNA or RNA seen as viruses, whose genetic material is attached to the main DNA of the cell and has been found in the chromosomes of many animal species. They are transmitted to other

members of the species through ordinary Mendelian inheritance, or directly from the mother in the form of new viruses—infectious viral particles that can pass from mother to foetus. In 1969, Robert J. Huebner and George J. Todaro of the National Cancer Institute proposed that the activation, by carcinogens, of these normally silent endogenous sequences was the mechanism of all malignancies.

**Retroviruses**

A virus is not a cell but a microscopic particle with a coat made of a few proteins (a given species of virus is always of the same form and size), strung around a piece of RNA or DNA that contains genetic information (for a given species of virus, this is always of the same length).

Retroviruses are viruses that have RNA as a genetic material.

Retroviruses are incredibly small—about 100 nanometres in diameter—and can only be seen through an electron microscope. They are almost spherical, and have an outer envelope covered with knobs and an inner core consisting of some proteins and RNA.[9]

Retroviruses are classified into three Subfamilies:

- Spumavirinae
- Lentivirinae (HIV I, HIV II)
- Oncovirinae—type A, B, C and D particles

In the 1970s such particles were frequently observed in human leukaemia tissues, cultures of embryonic tissues,[53, 54] and in the majority of, if not all, human placentas.[55]

These findings led to a very interesting view among certain retrovirologists, which was this: because retroviral genomes may arise from the rearrangement of cellular DNA caused by numerous factors, including pathogenic processes, retroviruses may be more of an effect than a cause of disease.[56, 57] "The human genome carries DNA sequences related to endogenous retroviral genomes that are subdivided into families according to sequence homology. Some are present in only a few copies, whereas others are present in hundreds to thousands of copies."[58]

Viruses are stable, because they have to leave cells or even the organism to infect other cells or organisms anew. Unlike cells, viruses do not have any of the cellular structures (mitochondria, ribosoma, etc.) that allow a cell to survive and reproduce. Therefore, in order to reproduce themselves and express the genetic information that they carry, they need to parasitise cells.

The protective coat of the viral particle fuses with the cell membrane, then the particle passes inside. Once inside, its genetic information gets into the cell nucleus, becomes part of the cellular DNA, and takes over the cell's metabolic machinery for its own benefit, by which process the virus particle is disassembled. Then, using the same machinery, separate pieces of new virus are synthesised. Later, all the viral components are put together and the new virus particles are ready to free themselves, either by destroying the host cell or, in the case of retroviruses, by a more conservative process of budding out of the cell membrane. This particular characteristic of needing to parasitise cells makes viruses a difficult target for specific therapeutic agents. Anti-bacterial drugs like antibiotics attack their bacterial targets with tremendous specificity (until, of course, the bacteria develop antibiotic resistance!) Nevertheless, by killing specifically bacteria, they do not cause too much damage to the host's body. In the case of viruses, an anti-viral drug would need to discriminate between the proteins and DNA made for viruses, and those proteins and DNA made for their human hosts.

**The discovery of reverse transcriptase (RT)**

For most of the cells in all living things, the direction of information flow is from the DNA of the nucleus to the RNA in the cytoplasm. In the nucleus the DNA duplicates itself, then this copy, after being trimmed by an enzyme, is released into the cytoplasm and becomes RNA, by which the genetic message is transported from the nucleus to a cytoplasmic structure called ribosome, where the protein synthesis will take place according to the instructions that the RNA carries.

Retroviruses have their name because, unlike other viruses, the transfer of information is 'backwards'.

In the 1970s there were many scientists engaged in the 'War Against Cancer'. This experimental work led to two crucial discoveries: the finding of reverse transcriptase in 1970, and of interleukins in 1976.

In 1970 the enzyme reverse transcriptase (RT) was discovered in oncoviruses, which from then on became known as retroviruses. Howard Temin was awarded the 1975

Nobel Prize for Medicine for that discovery. The popularity of this unique retroviral enzyme (as it was then assumed to be), caused many virus researchers to switch to the retroviral quest. One of the major reasons for the interest was that this fundamental enzyme could be a specific target for the drugs against the virus. So, while there were people looking for diseases that might be caused by retroviruses, alongside, the pharmaceutical industry was already developing anti-retroviral drugs, drugs that could interfere with reverse transcriptase. The famous AZT, the first drug claiming to treat AIDS, was produced in 1964.

Retroviruses do not carry DNA as a genetic material, rather RNA.

Retroviruses do not use their RNA blueprint directly to make more viruses. Once they enter a cell, retroviruses first make a DNA copy of their RNA. This process, called *reverse transcription*, is catalysed by the enzyme called reverse transcriptase (RT). This DNA then moves into the cell nucleus where it becomes part of the cellular DNA. This string of DNA is called a *provirus*, and sits there, hibernating as it were, for a long time, perhaps several years, until something activates the cell. Then the pro-viral DNA is copied back into RNA and it is this RNA, not the original RNA that entered into the cell when it was parasitised by the virus, which instructs the production of the necessary proteins to make new viral particles.

So the detection of reverse transcriptase became the *blueprint* for the confirmation of the existence of a retrovirus. "Since reverse transcriptase is unique to retroviruses, finding it in tumour cells would show that such a virus was there." (R Gallo, 1986, 'The first human retrovirus', *Scientific American*, p. 91). In the same article the author mentions that from leukaemia cells of a few patients, they purified DNA polymerases that seemed to have all the properties of reverse transcriptase. He then goes on to say, "The enzymes might have been unusual cellular polymerases detectable only because their numbers are increased in diseased cells," meaning natural enzymes from the main tumour cells.

Ever since then, the standard technique for detecting retroviruses has been the analysis of the activity of reverse transcriptase in cell cultures.

Unfortunately, in the 1970s, the biochemical function of reverse transcription did not fit the dominant world picture of genetics and was thought to be exclusive to the new class of viruses, the retroviruses, which is why the detection of reverse transcription was henceforth accepted as a 'proof' of the presence of a retrovirus. But in 1990s it was established that the biochemical process of reverse transcription is a natural cellular function that reflects a repair mechanism for damage to cellular genetic material.[102]

**The discovery of interleukins**

The discovery of reverse transcriptase as the supposed indicator of the presence of a retrovirus heightened the search for human retroviruses, and led to the development of molecular biology techniques aimed at detecting low-levels of reverse transcriptase activity.

The hypothesis on human retroviruses was that they were slow viruses that required a long time to emerge, and it was therefore thought necessary to keep the cells alive for long periods of time in order to detect low levels of virus expression. Human blood cells, normal or cancerous, are difficult to grow in the laboratory. Prior to 1976 it was impossible to grow lymphocytes for long enough. The discovery of growth factor proteins was the answer. Gallo and his colleagues found that after the stimulation of T lymphocytes with a protein called phytohemaglutinin (PHA) derived from plants, T-cells would release a growth factor, now called interleukins 2 (IL-2), that would induce T-cells to divide and mature.

This was a crucial step. The discovery of IL-2 could be used to grow T-cells in the laboratory long enough to see if any retroviral presence would emerge.

**Standard identification of a retrovirus**

Three steps must be fulfilled in order to prove the existence of a retrovirus:

1. Culture cells and find a particle that looks like a virus. Physical-electron microscopy for virus count, morphology and purity.

2. Use the standard method to isolate the particle, break it into pieces, and analyse its make-up. Biochemical reverse transcriptase activity, viral and cellular RNA, total protein, gel analyses of viral and host proteins and nucleic acids.

3. Prove that the particle in question can make faithful copies of itself, in what is known as ‘replication’. Biological infectivity *in vivo* and *in vitro.*

Explanation:

Identifying a retrovirus requires an electron microscope and a high speed centrifuge. Retroviral particles have a physical property—their *buoyancy*—which enables them to be separated from other material in cell cultures. This is utilised to purify the particles by a process called ‘density gradient centrifugation’.

The identification process begins by growing cells that are believed to contain retroviruses. The retroviral particles will then be released from the cells into the culture fluids. After decanting a specimen of culture fluids, a drop is placed on top of a solution of sucrose in a test tube. This is a solution that is light at the top but gradually becomes more dense towards the bottom. Then the test tube is spun at extremely high speeds. This generates tremendous forces, and particles present in the drop of fluid are forced through the sugar solution until they reach a point where their buoyancy prevents them from penetrating any further, meaning a place at which their own density is the same as that of the sugar solution. They 'band' at a point where the density is 1.16 g/ml. That band can then be selectively extracted and photographed with an electron microscope. This band, however, is not specific to retroviruses alone, but only to a particular molecular weight.

The next step is to disrupt the particles, find out what proteins and RNA they contain, and prove that one of the proteins is reverse transcriptase that turns RNA into DNA.

Finally, PURE particles have to be added to a culture of un-infected cells to see if they can replicate themselves and produce the same particles with the same constituents.

Replication is paramount in order to be 100% certain that a retrovirus has been identified, because retrovirus-like particles are not the only material that may find its way into this band of the density gradient. Very small cellular particles, some recognisable as internal cellular structures, or just cellular debris, can also band at 1.16 gm/ml, and some of this material can even enclose nucleic acids (DNA and RNA) and take on the appearance of retroviral particles.

## IV. ANALYSIS OF THE EVIDENCE OF THE HIV-AIDS PHENOMENON

A modern clinical medicine textbook, under the heading 'Diagnosis of HIV infection', indicates:

> "HIV infection is diagnosed either by detection of virus-specific antibodies (anti-HIV) or by direct identification of viral material:
>
> - Detection of IgG Antibody to gp120 and its subunits. This is the most common marker of infection. The routine tests used for screening are based on ELISA techniques that may be confirmed with Western Blot assays. Up to 3 months may elapse from initial infection to antibody detection (serological latency). These antibodies to HIV have no protective function and persist for life. As with all the IgG antibodies, the anti-HIV antibody will cross the placenta. All the babies born to HIV infected women will thus have the antibody at birth. In this situation, the anti-HIV antibody is not a reliable marker of active infection [i.e. the development of AIDS diseases] and in un-infected babies will be gradually lost over the first 18 months of life.
>
> - Detection of IgG antibody to p24. The anti-p24 antibody can be detected from the earliest weeks of infection and through the asymptomatic phase. It is frequently lost as disease progresses.
>
> - Antigen assays are nucleic acid-based assays that amplify and test for components (PCR, viral load) of the HIV genome. All are based on HIV-1 subtype B material, and there are potential inaccuracies with other subtypes, especially group O variants. These assays are used to aid diagnosis of HIV in the babies of HIV-infected mothers, or in situations where serological tests may be inadequate.
>
> - Viral p24 antigen (p24ag). This is detectable shortly after infection but usually disappears by 8-10 weeks after exposure. It can be a useful marker in individuals that have been infected recently but have not had time to mount an antibody response. It may reappear at low levels intermittently during the period of clinical latency, and in some people as infection progresses.
>
> - Isolation of virus in culture. This is a specialised technique available in some laboratories to aid diagnosis and as a research tool."[131]

## A) HIV IDENTIFICATION

As we have mentioned before, the three known human retroviruses were reported by the same man: Robert C. Gallo. They are HTLV-I and II, and HTLV-III or HIV-1.

HTLV-I and II are oncoviruses and described as causing lymphocytosis, meaning they stimulate excessive production of lymphocytes.

HTLV-III, better known as HIV, is a lentivirus (slow virus) and is lympholytic, meaning it destroys lymphocytes. Since a second strain of HIV has been reported, mostly in west Africa, HIV is now being described as HIV-1 and HIV-2.

R. Gallo is important because, historically, he was a man looking at the phenomenon from a particular perspective, and he was fundamental in the development of the arena in which that perspective unfolds, which is the laboratory technique of the continuous growth of leukaemia T-cells in the artificial medium of a test tube.

The experiment and the experimenter are part of the same reality. In this sense one could say that R. Gallo developed his own method of identifying the virus.

In the mid-1970s Gallo claimed to have discovered the first human retrovirus in patients with acute myelogenous leukaemia. He called the retrovirus HL23V.[1, 2]

Gallo used nothing more than antibody reactions to 'prove' which of the proteins (antigens) in the cultures were viral proteins. This is an indirect way of identifying a virus. The presence of the antigen revealed by the antibody does not prove the origin of the antigen itself, therefore it may not be specific to retroviruses. Because it avoids the proper standard scientific method of isolation and identification of the retroviruses, the evidence is not conclusive.[7,8]

Not long afterwards, other researchers claimed to have found the same antibodies in many people who did not have leukaemia. A few years later these same antibodies were shown to occur naturally and to be directed against many substances that had nothing to do with retroviruses.[3, 4] It was realised that HL23V was a mistake.

In 1980, Gallo and his group presented the discovery of another retrovirus, and this time he called it HTLV-I.[2b] The procedure utilised for this 'isolation' was the same one as before. He claimed that it caused a particular rare form of leukaemia that was then named 'adult T4-cell leukaemia' or ATL, in which T4-cells are present in excessive amounts. Apart from being medically irrelevant, the HTLV-I discovery was again nothing more than experimental data from leukaemia patients, as was his former HL23V. The greatest prevalence of HTLV-I was reported in Africa and Southern Japan, and that

is where it remains prevalent. Yet although this virus is said to cause leukaemia, less then 1% of persons who test positive to the *test* ever develop leukaemia. In 1982 a new subtype of human T-cell leukaemia virus (HTLV-II) associated with a T-cell variant of hairy cell leukaemia was discovered by Gallo and collaborators.[2c]

The reason why retrovirologists became interested in AIDS was that Kaposi's sarcoma (KS), a rare form of cancer only observed in elderly people, was one of the first of the most common shared diseases among those young male homosexuals suffering from AIDS in the United States. The young age of the patients, as well as the new peculiarity that KS was affecting the lungs in all of these cases, made it even more exceptional. So to oncovirus researchers, these cases, sharing as they did the same rare type of cancer, and all being promiscuous young male homosexuals, indicated that the cancer might be transmitted—sexual transmission perhaps—therefore an infectious agent. And that would fit the retroviral hypothesis.

The actual identification of the retrovirus HIV, surprising though it may be, does not follow the standard method of retroviral isolation explained earlier, but instead by-passes major steps, casting doubt on the evidence itself.

It is important to go back to the experiments that were done in 1983-84, because everything believed and taught about HIV is founded on what happened then.

In 1983 Luc Montagnier from the Institute Pasteur in Paris presented a paper[5] claiming that he had isolated a retrovirus from a lymph node of a homosexual decorator with AIDS. He called it the 'lymphadenopathy associated virus' (LAV). Note: to this French researcher, the virus was not yet a causative agent but an associated one. The causative agent view only emerged some time later, when Gallo patented the HIV-test made with proteins that allegedly came from Montagnier's sample. Montagnier had sent supernatants from LAV-1 'infected' cultures to Robert Gallo at the National Institute of Health in the USA for evaluation. Later on Montagnier was to sue Gallo for misappropriating his strain.

Gallo's isolation was almost a carbon copy of his first two failed attempts to isolate a retrovirus in his war against cancer, HL23V, and HTLV-I and its later variety HTLV-II. In fact, some of these early efforts were scientifically more soundly conducted than the infamous HIV procedures.

The identification of the HIV virus stands on three main evidences:

1: Electron micrographs of retroviral particles

2: Finding reverse transcriptase

3: Antibody reaction to HIV proteins

## 1: Electron micrographs (EMs)

In 1973 the Pasteur Institute hosted a meeting attended by a number of scientists, some of whom are now among the leading HIV experts. At that meeting the method of retroviral isolation was thoroughly discussed, and the photographing of the 1.16 band of the density gradient was considered absolutely essential.

Surprising though it may seem, Luc Montagnier and Robert Gallo published electron micrographs (EMs) of a few particles in cultured fluid that they claimed to be the HIV retrovirus,[5, 6] and everyone subsequently called it 'pure HIV'. However, they did not publish any EMs of the material at the 1.16 gm/ml band density gradient as the retroviral scientific method requires, let alone prove what the particles were.[10] Even more puzzling is that, up until March 1997, all of the electron microscope pictures published were from un-purified cell cultures, not from the density gradient.

Animal retrovirologists are well aware of the importance of obtaining retroviral particles without disrupting the cells in order to avoid cellular contamination, which is why they strongly advise handling the cultures gently and regularly topping them up with nutrients to keep the cells alive so that they do not disintegrate. But in most of the HIV experiments the cells are deliberately broken up by the experimenter as part of the experiment.

Moreover, if HIV is cytopathic as it is believed to be, it means that it destroys cells, Lymphocyte T4-cells, and it would be very difficult to claim that the putative virus particles are the only things likely to be floating around in cultured fluids or at the 1.16 gm/ml density gradient.

Finally, in 1997, two groups, a Franco-German one[12] and one from the U.S. National Cancer Institute[13] published pictures of the density gradient band. The Franco-German study pointed out that cell membrane vesicles are a major contaminant of the gradient-enriched human immune deficiency virus type-1 (HIV-1) preparations, and their pictures revealed that the vast majority of the material in the density gradient is cellular, therefore non-viral. The American study, in which it is impossible to tell from which density gradient the pictures are taken, concede that those micro vesicles (encapsulated cell fragments) are a source of contamination of cellular proteins found in purified HIV-1 preparations. So both say that their samples might be contaminated.

Yet the researches claim that the few particles that appear in the pictures are retroviral HIV particles. They do not give any evidence why. Also, it would be expected that if the sample were to contain retroviruses, billions of particles would be found, not just a few—yet that is not explained either. Although these few particles may look like retroviral particles, they may also not be. The only definitive proof that they are retroviruses would be if they were capable of self-replication, which again was not done in any of the studies.

Regarding the morphology of retroviruses, most retrovirologists agree that retrovirus particles are almost spherical in shape, have a diameter of 100-120 nanometres, and are covered with knobs.[9, 11] But in these two studies the particles that are claimed to be retrovirus are not spherical, the diameters exceed twice that permitted for retroviruses, and none appear to have knobs.

The Franco-German particles are 1.14 times larger than the known retroviral particles and therefore have 50% more volume than a normal retrovirus and the American particles are 1.96 times larger, meaning they have 750% more volume, so the American particles are five times more voluminous than the Franco-German ones.[109] This is particularly crucial: because density is the ratio of mass to volume, if the volume is up, to keep the same density, the mass has to go up by the same amount. Any genuine retroviral particle will contain a fixed amount (mass) of RNA and protein—no more, no less—yet the particles presented in these two studies are made up of much more material. This means that if these different sized particles are truly HIV, then HIV cannot be a retrovirus, and the specific 1.16 gm/ml density band which is characteristic of retroviruses and which is employed to determine HIV, can no longer be used, unless we begin to redefine retroviruses and their means of identification. This is the band on which all the major research on HIV has been done for the last 15 years, and, moreover, it is the band used to obtain proteins and RNA as diagnostic agents in the HIV-test to prove HIV infection.

The other possibility, of course, is that the electron micrographs of that study are not from the 1.16 gm/ml band, in which case the particles are obviously not retroviruses.

Another problem arises related to the absence of knobs in the particles of these two studies. All of the AIDS experts agree that the knobs are absolutely essential for the HIV particle to lock on to a T-cell. The knobs of retroviruses contain a protein called gp120, which is the hook in the knobs that grabs hold of the surface of the cell that it is about to infect.[14] So, if the HIV particles do not have knobs, it is very difficult to explain how they can attach themselves to a cell and parasitise it.

On the evidence provided by these pictures it is impossible to explain the claim that this material is pure, or that it contains even retrovirus-like particles, let alone the specific retrovirus HIV.

**2: Finding reverse transcriptase (RT)**

The current way to prove the presence of RT is indirect. Reverse transcriptase is not isolated as such, but if a template of RNA is introduced into a solution suspected of containing RT, the enzyme will reveal its activity and therefore its presence by transcribing the RNA into DNA.

It is of the greatest importance, when trying to detect the activity of reverse transcriptase, that the natural genetic messenger material is used—the RNA-genome of the virus that should be there if the virus exists. Despite this, all HIV researchers always use, without any explanation as to why, synthetic messenger material templates. This is so crucial because those templates are not specific to reverse transcriptase alone: on the contrary, they are efficiently recognised and transcribed by normal, common, cellular genetic material-producing enzymes such as DNA-polymerases.

Gallo's group began culturing lymphocytes from AIDS patients, but none of the cultures produced enough reverse transcriptase to prove that a retrovirus was present. Then Gallo and another researcher from his group, Mikulas Popovic,[6] made a preparation consisting of a mixture of culture fluids from ten AIDS patients and added that to a culture of leukaemia cells, HUT78 cell, which had been obtained years earlier from a patient with malignancies of mature T4-cells, called adult T-cell leukaemia (ATL). However, these leukaemic cells were supposed to already contain the retrovirus HTLV-1 of which Gallo had presented the genetic sequences in a paper published a year earlier (1983) in *Nature*.[15] This time enough reverse transcriptase was produced for them to believe they had a retrovirus. It is difficult to know if the reverse transcriptase was from the presence of HIV, or HTLV-1, or neither of them.

Popovic also made a clone of the same leukaemia cell line, the HUT78 cell line, called the H9 clone. Evidence exists that the H9 cell line releases retrovirus-like particles even when not 'infected with HIV'.[103] The H9 clone has since then been widely used, both in research and commercially for producing what are regarded as the HIV proteins for use in the antibody test kits. Interestingly enough, although HIV is thought to kill T4-cells, the leukaemic cell line as well as its H9 clone are both immortal even when infected with HIV. So these cells permit what is believed to be HIV to grow indefinitely. The proteins

produced from these cells are the ones patented by R. Gallo as the antigens for testing antibody reaction to HIV. All of the test kits for HIV come from these proteins.[110, 111]

As mentioned above, the existence of reverse transcriptase is proven indirectly by introducing a synthetic piece of RNA (An.dT15) into a culture and seeing if DNA bearing the corresponding sequence appears. In all HIV research, the copying of the template-primer An.dT15, when incubated with supernatant or the material that bands at 1.16 gm/ml from AIDS cultures/co-cultures, is considered proof of HIV reverse transcriptase activity. So it is measured by demonstrating the process of reverse transcription, which is what the enzyme does, but not by measuring the actual enzyme itself. That is why the same template is also copied when incubated with material that bands at 1.16 gm/ml from leukaemia T-cell cultures,[49] and normal non-infected spermatozoa.[50] Both An.dT15 and Cn.dG15 are also copied by material that bands at 1.16 gm/ml (the retroviral band) originating from normal non-infected but mitogenically stimulated lymphocytes.[49, 51] Furthermore, An.dT15 is copied not only by reverse transcriptase but also by two (beta and gamma) of the three cellular DNA polymerases. In fact, DNA polymerase is a cellular enzyme that copies An.dT15 with high efficiency but which does not copy DNA well.[52] Thus, the copying of the template An.dT15 cannot be considered synonymous with the presence of HIV reverse transcriptase.

There is a problem emerging here, because the laboratory techniques that are supposed to be direct and specific as the hallmark of retroviral-HIV identification, are not:

Firstly, reverse transcriptase is not unique to retroviruses. Reverse transcriptase is present in normal cells and also in bacteria.

Secondly, reverse transcriptase is not the only substance capable of producing reverse transcription. Normal cellular enzymes can also do it, as we have mentioned above. In fact they do it very well with the very same synthetic RNA (An.dT15) that all HIV researchers introduce into their cultures to copy into DNA.[16] It is known also that some of the chemicals that are an obligatory component of these cell cultures cause normal lymphocytes to reverse transcribe.

Even more important is the fact that leukaemia cells can also produce reverse transcription unaided, when not cultured with such chemicals or cells from AIDS patients.

In fact, reverse transcription is now known to reflect a repair mechanism for damage to cellular genetic material in normal cells.[102] So it is perfectly obvious that finding reverse transcriptase, less still by merely showing that reverse transcription is taking place, is not enough to prove the presence of a retrovirus.

Retrovirus-like particles are found practically everywhere. In the 1970s such particles were frequently observed in human leukaemia tissues, in cultures of embryonic tissues, and in the majority of animal and human placentas. These findings are of extreme significance because the H9 cell line (the clone cell line which is the source of all of the proteins for the HIV test) is made up of leukaemia cells. They are also important because Luc Montagnier obtained his EMs from cultures produced using umbilical cord blood lymphocytes. In 1988, a study by researchers from Harvard[17] reported the finding of retrovirus-like particles, or 'HIV particles' as they were called, in 90% of enlarged lymph nodes from both AIDS and non-AIDS patients.

### 3: Antibody reaction to HIV proteins

The Gallo hypothesis is that there is a virus causing AIDS, and it is foreign, so when it infects a person that person develops antibodies to the virus.

Using antibodies to prove the existence of a virus is the key part of the problem, since it is an indirect method that in itself only proves that a reaction is taking place, but does not confirm the origin of the protein that triggers that reaction.

Viral proteins are those proteins that come out of particles proven to be viruses. But in order to define the proteins of a retroviral particle, first it has to be proven that the particle in question is a retrovirus. What may not be done is to *assume* that the protein comes from a retroviral particle, and to then use the antibody reaction to the protein as *proof* of the identity of the particle if we do not yet know the identity of the protein in the first place.

Antibodies are imprecise because they do not react only to single proteins.[18, 19] In what immunologists call cross-reactions, an antibody reacting to a protein in a culture could just as well be an antibody made to counter something totally unrelated. And if we are trying to define which proteins are unique constituents of a retroviral particle, it cannot be proven by performing chemical reactions on what is essentially a culture soup. In that case antibodies are completely irrelevant.

What the experiments reported in the first Gallo paper really tell is that some antibodies present in a patient with haemophilia, as well as in rabbits, reacted with some proteins in H9 cells (a clone of leukaemia cells) cultured with lymphocytes from AIDS patients.[6] These reactions do not say anything about the identity of the proteins coming from the H9 cells (are they cell proteins or viral proteins?) nor about the specificity of the antibodies from the haemophilic patient (are they against a cell protein or a viral

protein?) Antibodies can only be specific to HIV if, and only if, they are present ONLY when HIV is present. This means that we need to have proteins that we are 100% certain belong only to HIV.

It is therefore quite clear that it is impossible to prove the origin of a protein by an antibody reaction, if you have not first identified the source of the protein.

What we have are some cultures of tissues derived from AIDS patients that react with antibodies present in the serums of AIDS patients. We know that AIDS patients are infected with many different agents. So if these agents, or bits of them, are present in AIDS patients, they will also be present in their cell cultures. Everyone agrees that AIDS patients have myriads of antibodies to all manner of things, including antibodies to human T-cells, the cells that make up the cultures. If we add some antibodies from the same kind of patients to these cultures, all we see are reactions: we cannot tell what is reacting to what.

A good example of this is the hepatitis B virus (HBV). Many AIDS patients, and in the case of haemophiliacs virtually all of them, are infected by HBV. And HBV does not just infect liver cells, it also infects T-lymphocytes. Also, strange though it may seem, HBV has a reverse transcriptase enzyme and people make antibodies against this virus.

The other puzzling aspect of the Gallo experiment[6] is that he claims he had serum from rabbits that contained antibodies specific to HIV. They say that they had prepared rabbit antibodies by repeatedly infecting rabbits with HIV.

Before he had a virus there was no way of knowing in advance that antibodies to HIV existed at all. Anywhere. So if they were preparing antibodies to HIV they would have had to inject rabbits with pure HIV, which means they would have had to have isolated already that which they were in fact attempting to do for the FIRST time.

What Gallo and Popovic injected was their culture material, which at very best was a banded 1.16 gm/ml specimen (a debris of proteins), something akin to what we see in the Franco-German and U.S. National Cancer Institute pictures,[12, 13] which they and everyone else have since regarded as pure HIV. Gallo and Popovic would have exposed their rabbits to a multitude of cellular proteins. Proteins are the most potent antibody-producing substances available, even more if they are introduced directly into the blood-stream. The rabbits would have then produced antibodies to all those proteins, and when they added these antibodies back to the material they injected in the first place, there would be reactions as you would have expected. That, however, does not make that material injected into a virus.

And even to begin to talk about specific antibodies to specific HIV proteins, first it has to be proven that the proteins are constituents of a retrovirus-like particle, and that that particle is able to *replicate*. The virus is needed BEFORE looking for proteins and antibodies, not the other way around.

Science is about evidence and proof. But for some unexplained reason the traditional, logical, reliable, common-sense method of proving the existence of a virus has been abandoned in the HIV era.

## B) THE HIV-ANTIBODY TEST

The antibody test is the same procedure that was used to prove the existence of HIV in cultures from AIDS patients by the French in 1983 and by the Americans in 1984. But it is also the same procedure that Gallo and his colleagues used to prove the existence of HL23V in the mid seventies,[1] which later proved to be a big mistake.

The problem with using antibodies is that there can be two types of antibodies. One type is *specific*, meaning antibodies caused by HIV and nothing else, and reacting to HIV and nothing else. The other type is *non-specific*, meaning antibodies caused by other agents or stimuli, which certainly react to those agents, and also react to HIV.

The test is nothing more than a chemical reaction. Something changes colour. So when a person's serum is added to some of the so-called 'HIV proteins' in a culture or in a test kit, and a reaction appears, it is impossible to tell which type of antibody is responsible for the reaction. There are three possibilities: all the antibodies might be the specific type, or none of them, or there might be a mixture. The only way to tell would be to test for antibodies in all sorts of patients: some with AIDS, some who are ill but who do not have AIDS, and some healthy people as well. And in all the experiments, at the same time, HIV would have to be used as the adjudicator to determine what type of antibodies they are. Only then could the test be introduced into medical practice as a diagnostic tool. Yet that experiment has never been done.

Gallo only managed to isolate a retrovirus in 36% out of 72 AIDS patients, but 88% of the patients had antibodies, which means that there were more patients with antibodies but without viruses than there were patients with viruses. Yet Gallo filed a patent for the antibody test the very same day that the idea of HIV as the cause of AIDS was launched into the world, 23 April, 1984.

For the test, the patient's blood is mixed with proteins acting as antigens extracted from H9 or other cell cultures, and put all together in a test tube or separately at discrete

points along a thin paper strip. The first is called ELISA and the second Western Blot. If the proteins react with the blood then the patient is reported HIV positive. The ELISA test is used to screen for antibodies, and then 'confirmed' by the more specific and highly sensitive Western Blot. The leaflet accompanying one such test kits says:

> "The test for the existence of antibodies against the AIDS-associated virus is not diagnostic for AIDS and AIDS-like diseases. Negative test results do not exclude the possibility of contact or infection with the AIDS-associated virus. Positive test results do not prove that someone has an AIDS or pre-AIDS disease status nor that he will acquire it."[20]

**The antigens: the viral proteins**

The proteins considered to represent HIV antigens are obtained from mitogenically stimulated cultures in which tissues from AIDS patients are co-cultured with cells derived from non-AIDS patients—usually established leukaemia cell lines. Following the detection of the enzyme reverse transcriptase in the cultures (actually, not detecting the enzyme itself, but the reverse transcription process with synthetic RNA), the cell lysates are spun into density gradients. The material that bands at 1.16 gm/ml is considered to represent 'pure HIV' and consequently the proteins found at that density are considered to be HIV antigens. The immunogenic HIV proteins are thought to be coded by three genes:[21]

**Gag** gene: codes for a precursor p53/p55, then cleaved to p24/p25 and p17/p18.

**Pol** gene: codes for proteins p31/p32.

**Env** gene: codes the precursor protein p160, which is cleaved to p120 and p41/p45.

Montagnier's group considered p24 sufficient to define a positive Western Blot (WB), whereas Gallo's group considered p41 sufficient. Most laboratories use the criteria recommended by the Centres for Disease Control (CDC), namely the presence of a band at either p24 or p41.

— The **p41** protein

p41 is one of the proteins detected by both Gallo's and Montagnier's groups in the first HIV isolates. However, Montagnier and his colleagues observed that AIDS sera reacted with a p41 protein both in HIV and HTLV-I infected cells, as well as non-infected cells, and concluded that the p41 band "may be due to contamination of the virus by cellular actin which was present in immunoprecipitates of all the cell extracts."[5]

Actin is a ubiquitous protein found in all cells as well as bacteria and several viruses. It is also known that the oxidation of cellular sulphydryl groups, as in the case of AIDS patients,[23] is correlated with the assembly of polymerised actin,[24] and that the level of actin antibody binding to cells has been proposed as "a sensitive marker for activated lymphocytes."[25]

Platelets from healthy individuals also contain a p41/45 protein that reacts with sera from homosexual men with AIDS and immune thrombocytopenic purpura (ITP) and which "represents non-specific binding of IgG (antibodies of AIDS patients) to actin in the platelet preparation."[26]

— The **p24/25** protein

Detection of p24 is currently believed to be synonymous with HIV isolation and viraemia. However, apart from a joint publication with Montagnier in which they claim that the HIV p24 is unique, Gallo and his colleagues have repeatedly stated that the p24s of HTLV-I and HIV immunologically cross-react.[27]

Genesca et al[28] conducted Western Blot assays in 100 ELISA negative samples of healthy blood donors. 20 were found to have HIV, with p24 being the predominant band (70% of cases), leading them to conclude that "most such reactions represent false-positive results."

Antibodies to p24 have been detected in 1 out of 150 healthy individuals, 13% of randomly selected, otherwise healthy patients with generalised warts, 24% of patients with cutaneous T-cell lymphoma, and 41% of patients with multiple sclerosis.[29]

97% of sera from homosexual men with immune thrombocytopenic purpura (ITP) and 94% of sera from homosexual men with lymphadenopathy or AIDS contain an antibody that reacts to a p25 membrane antigen found in platelets from healthy donors and AIDS patients, as well as a p25 antigen found in green-monkey kidney cells, human skin fibroblasts, and herpes simplex cultured in monkey kidney cells. This reaction was absent in sera obtained from non-homosexual patients with ITP or non-immune thrombocytopenic purpura.[26]

— The **p32** protein

In 1987, Henderson isolated the p30-32 and p34-36 of "HIV purified by double banding" in sucrose density gradients. By comparing the amino acid sequences of these proteins with Class II histocompatability DR proteins, he concluded that "the DR alpha and beta chains appeared to be identical to the p34-36 and p30-32 proteins respectively,"[22] and were therefore non-HIV proteins. (Class II proteins expressed on the surface of macrophages, B-lymphocytes and activated T-lymphocytes).

An antibody test becomes meaningful only when it is standardised, that is, when a given test result has the same meaning in all patients, in all laboratories, in all countries. From the first antigen-antibody reactions performed by Montagnier's[5] and Gallo's[6] groups, it was found that: not all the 'HIV proteins' react with all sera from AIDS patients or even sera from the same patients obtained at different times; and that sera from AIDS patients may react with proteins other than those considered to be HIV antigens.

The **Western Blot (WB)** test kit

In this test the 'HIV proteins' are dissociated and placed on a polyacrylamide gel slab. After electrophoresis, which separates the proteins by molecular weight and charge, the proteins are transferred to a nitro-cellulose membrane by electro-blotting. After adding the patient's serum to the proteins, the reaction is interpreted visually as coloured bands, each of which is designated with a small 'p' and a number (p for protein and the number is its molecular weight in kilodaltons).

In 1987 the Food and Drug Administration (FDA) licensed a WB kit manufactured by DuPont. The DuPont kit remains the only licensed WB kit. It specifies "extremely stringent" criteria for a positive result, namely "specific bands representing three different gene products: p24(**gag**), p31(**pol**), and an **env** band, either p41, p120 or p160."[30]

The American Red Cross defines a positive result as presence of antibodies to at least one gene product from each of the **gag**, **pol** and **env** genes, without specifying which bands.

The Association of State and Territorial Public Health Laboratory Directors/Department of Defence/CDC, consider a WB positive if two out of p24, gp41 and gp120/160 are reactive.

The Consortium for Retrovirus Serology Standardisation (CRSS) defines a positive WB as the presence of antibodies to at least p24 or p31/32, and gp41 or gp120/160.[31]

All the other major USA laboratories for HIV testing have their own criteria.

In the scientific literature, no strips have been published of a standard positive WB. As the instruction manual from Bio-Rad, a manufacturer of WB test kits states: "Each laboratory performing Western Blot testing should develop its own criteria for band interpretation. Alternatively, band interpretation may be left to the clinician."

It is obvious that a lack of standardisation creates problems of interpretation. When FDA criteria are used to interpret the WB, only a minimal number (less than 50%) of AIDS patients have a positive WB. If the criteria of the CRSS are used, the percentage of AIDS patients testing positive increases to 79%.

On the other hand the scientific data reveals major doubts about the specificity of the proteins used as antigens for the antibody reaction. The finding that the p31/32 band represents a cellular protein,[22] and that p120 and p160 are oligomers of p41,[32] reduces the criteria of the CRSS and that of the American Red Cross to two bands, p21 and p41, which are "less than perfectly specific."[33] But even at the present, the p160, p120 and p41 bands are considered to represent distinct viral envelope glycoproteins. The current WHO guidelines consider a serum positive for HIV-1 antibodies if "two envelope glycoprotein bands with or without other viral specific bands are present on the strip."[34]

Despite this, the general consensus is that proof of the specificity of the HIV-antibody test is firmly established.

**Non-specific antibodies**

Sick individuals with disorders of the immune system are prone to have all kinds of antibodies that produce cross-reactions when tested with antigens for different diseases. This is called a 'biological false positive' test (BFP) and is illustrated by the serological tests to syphilis. BFPs to syphilis occur in patients with auto-immune haemolytic anaemia, systemic lupus erythematosus (SLE), idiopathic thrombocytopenic purpura, leprosy, and drug addiction.[35] Significantly, 14% of AIDS patients are also found to have BFP syphilis serology.[36]

In a study conducted in 1986, 1129 serum samples from intravenous drug-users and 89 control samples from non-users were tested by two commercial ELISAs and a Western Blot. All the samples were collected during 1971-1972. The result was that 17 of the drug-users tested positive and all of the non-drug-users tested negative. They concluded: "On the basis of our positive Western Blot data, it appears that parenteral drug-users may have been exposed to HTLV-III (HIV) or a related virus as early as 1971. An alternative but equally viable explanation is that the HTLV-III (HIV) seropositivity detected in these specimens represents false positive or non-specific reactions."[37]

It is known that all antibodies, including MCAs (monoclonal antibodies), are polyspecific and are capable of reacting with immunising antigens as well as other self and non-self components.[39, 40]

In 1980 Gallo discovered HTLV-I, which he and his associates claimed causes adult T-cell leukaemia. Up to 25% of AIDS patients have antibodies to this retrovirus.[41] However, AIDS patients do not develop leukaemia any more often than the general population. This can only be interpreted as meaning either that HTLV-I does not cause adult T-cell leukaemia, or that some retroviral antibodies detected in AIDS patients are *non-specific*.

Sixty-three sera obtained from 23 patients before and immediately after immunoglobulin (IgG) infusions were tested for HIV antibodies using WB. Of the 63 sera, 52 (83%) were found positive. "Several samples tested in an HTLV-III (HIV) p24 radio immuno-assay were also positive. The amount of antibody detected was greatest immediately after infusion and decreased between infusions."[42]

Regarding immunisation, another study involved the administration at 4-day intervals of six 5 ml injections of Rh+ serum from a donor negative for HIV antibodies. The blood taken from the recipient after the first immunisation was still negative to the HIV-antibody ELISA and Immunoblot assay. But after a second immunisation a weak signal was monitored on ELISA. "After the third immunisation the signal was strong and the

immunoblot revealed distinct interaction with p17 and p55 proteins. An even stronger signal was monitored after the fifth immunisation. Interaction with p17, p31, gp41, p55 and some other proteins was evident."[43]

Individuals from the main AIDS risk groups—homosexual men, drug-users and haemophiliacs—are exposed to many foreign substances such as semen, drugs, factor VIII, blood and blood components, and commonly develop infections unrelated to HIV. These individuals have high levels of antibodies directed against antigens other than HIV. At present, evidence exists that individuals with AIDS, AIDS-related complex (ARC) and those at risk, have immune complexes, rheumatoid factor, anti-cardiolipin, anti-nuclear factor, anti-cellular, anti-platelet, anti-red cell, anti-actin, anti-DNA, anti-tubulin, anti-thyroglobulin, anti-albumin, anti-myosin, anti-trinitrophenyl and anti-thymosin antibodies.[44, 45]

It is also known that serum IgG levels are higher in Black blood donors than in Caucasians.[46]

Rodriguez and his colleagues[47] found that Amazonian Indians who have no contact with individuals outside their tribes, and have no AIDS yet, have a 3.3-13.3% HIV WB seropositivity rate, depending on the tribe studied.

Venezuelan malaria patients were found to have a 25% to 41% positive WB result, but no AIDS.[48]

In the light of this data it can be concluded that HIV testing is a *non-specific antibody reaction*, a marker that may identify an immune disorder:

The main pillar of the HIV hypothesis is that people with AIDS have a positive antibody reaction to the proteins of HIV. The fact that many people with AIDS test positive shows a correlation *per se*, but not a causation. It is therefore an indirect test.

Examples of indirect tests:

— *Cardiolipin* (extract of ox heart) is used as an antigen to predict the development of syphilis. Antibodies from a patient with syphilis react with cardiolipin, producing a positive test. Cardiolipin antibody-reaction is an indirect indicator of the disease, but cardiolipin is not the cause of syphilis.

— Another non-specific test is the measurement of the *erythrocyte sedimentation rate* (*ESR*). When high, this indicates the presence and intensity of morbid processes within the body, and has the capacity to predict a likelihood of death within the next several years far above a normal *ESR*.

An HIV-positive test is an evidence of the result of the disease, not of the cause of the disease.

**PCR and viral load**

These very specialised laboratory tools are used to indirectly confirm viral *presence and replication*. As indirect tools they have the same problem as the previous ones if the diagnosis of HIV is based on their results.

Polymerase chain reaction (PCR) is a very specialised laboratory technique by which sequences of DNA or RNA found in tissues can be amplified by the use of primers from DNA or RNA of the same type of tissue.

Primers claimed to represent segments of RNA or its complementary DNA (cDNA) from HIV are used to amplify similar sequences found in the tissues of AIDS patients.

Having seen the way in which HIV has been 'isolated', there is no real proof that the RNA of the primers used in the PCR test are constituents of a viral particle. Endogenous sequences could be the source of these bits of RNA. Therefore, transcriptional activities of endogenous sequences must be considered.

Humans are born with DNA nucleotide sequences known as endogenous retroviral sequences. Unlike genomes of bacteria, viruses other than retroviruses and other infectious agents, which if present in humans are clearly exogenously acquired, retroviral RNAs (proviral DNAs) are present in all of us and are known to constitute at least 1% of the human genome.[141] When expressed, these DNAs give rise to retroviruses known as endogenous retroviruses.

With regard to the HIV viral load tests, which are used to quantify HIV in plasma, researchers from the Massachusetts School of Medicine expressed the problem concisely: "Plasma viral [RNA] load tests were neither developed nor evaluated for the diagnosis of HIV infection. Their performance in patients who are not infected with HIV is unknown, and their use leads to mis-diagnosis of HIV infection."[141]

According to the manufacturer Roche: "The Amplicor HIV-1 [RNA] monitor test is not intended to be used as a screening test for HIV-1 or as a diagnostic test to confirm the presence of HIV-1 infection." (Roche Diagnostic Systems, 06/96, packet insert)

## C) IMMUNE DEFICIENCY

Immune deficiency is a pivotal aspect of the theory of HIV-AIDS, because it is the expression of the cytopathic action of the virus. The destruction of the T CD4+ lymphocytes causes a depletion of a very important part of the immune defence mechanism. Cell mediated immune deficiency leaves the host open to infections by intracellular pathogens and capsulated bacteria. Immune deficiency is the source from which the vast array of 30-odd diseases springs.

### Lymphocytes and immune responses

Human immune response is carried out by a group of blood cells called leukocytes or white cells. Lymphocytes are a type of these. Lymphocytes are the effector cells for immune responses to immunogens, that is, to materials not recognised as 'self' and therefore triggering reactions designed to neutralise or destroy 'non-self'. Immune responses combat the invasion of the body by organisms, cause the rejection of organ transplants between individuals with different histocompatibility antigens, and may also suppress the growth of malignant cells.

There are two types of immune responses: cell-mediated and antibody-mediated.

Cell-mediated response means that the immune cells kill off bacteria directly by phagocytosis. This kind of response is carried out by a type of white cell called granulocytes (neutrophils, eosinophils and basophils) and monocytes (macrophages). They come from a common myeloid stem cell in the bone marrow.

Antibody-mediated response means that for the immune cells to react, they need a recognition of the target first, and that is facilitated by an antibody. This very specific response is the action of lymphocytes and macrophages (monocytes).

The lymphocytes arise from a lymphoid stem cell in the bone marrow and are further processed into immunologically competent cells in the thymus (T-cells) and bone marrow (B-cells). They are of three functional classes: T-lymphocytes (T-cells), B-lymphocytes (B-cells), and a small group called natural killers (NKs).

We are interested in T-lymphocytes, which regulate and mediate immune reactions and also regulate antibody synthesis.

In our cells, a group of genes from chromosome 6 produce two types of molecules that show on the surface of the cell as the 'ID' of the cell. These are named Class I and Class

II molecules (major histocompatibility complex). These molecules are the tissue blue-print of an individual and largely determine whether organ or tissue transplants are recognised as self or foreign and, therefore, accepted or rejected. Class I molecules are in the surface membrane of most of the nucleated cells, Class II molecules are expressed normally by only a few cell types: hemopoietic progenitor cells, B-lymphocytes, macrophages and activated T-lymphocytes.

T-lymphocytes possess a cell surface T-cell antigen receptor, with the capacity to distinguish between non-self and self Class I and II proteins (MHC). This receptor contains a molecule, CD3+, which all mature T-cells posses. As a means of identifying mature T-lymphocytes in tissues and body fluids, monoclonal antibodies have been developed that recognise CD3+. The binding of the antibody to the CD3+ molecule is used as the counting tool for mature T-lymphocyte population.

While being processed by the thymus, T-cells also acquire surface glycoprotein molecules that determine whether the antigen receptor on the surface of the T-cell will react with an antigen of Class I or II molecules.

With monoclonal antibodies, two such surface glycoproteins can be identified, CD4+ and CD8+. CD4+ T-cells recognise Class II molecules, and CD8+ T-cells recognise Class I molecules.

T4 lymphocytes, or helper cells, activate macrophages to destroy affected cells and also B-lymphocytes to secrete immunoglobulins, and induce all lymphocyte sub-populations to proliferate.

T8 lymphocytes, suppresser cells, suppress the proliferative response of other T-cells and of B-cell immunoglobulin production and secretion. T8 suppresser cells keep the immune responses from going out of control.

The T4 lymphocyte is the agreed target of HIV. The progressive destruction of T4 by HIV reduces the capacity of the immune system to respond to other immune attacks. The immune deficiency then allows the development of a diversity of diseases, and although most of them are not fatal, the patient eventually dies from them.

**The CD4+ T-lymphocyte cell count — T4/T8 cell ratio**

In the 1970s it was known that patients who were treated with immune-suppressive drugs, or who suffered from ‘immune-suppressive illnesses’, had relatively high frequencies of malignancies and opportunistic infections (OIs).

Following the frequent diagnosis of Kaposi's sarcoma (KS) and OIs among homosexual men, intravenous drug-users and haemophiliacs, it was realised that when T-lymphocytes from these patients were reacted with monoclonal antibodies (MCA) to the CD4 antigen, the number of CD4 antigen-bearing cells diminished. For that reason it was thought that the high frequency of these diseases in these groups was due to the death of T4-cells and was the direct result of suppressed cellular immunity defined by diminished numbers of T4 helper lymphocytes. The newly postulated Acquired Immune Deficiency Syndrome (AIDS) was defined in 1982 by the Centres for Disease Control (CDC) as "illnesses in a person who:

1. Has either biopsy-proven KS or culture-proven life-threatening OI;

2. Is under the age of 60;

3. Has no history of immune suppressive underlying illness or immune suppressive therapy."

Then, in 1984, Gallo and his colleagues claimed that AIDS was caused by the HIV retrovirus. It was postulated that HIV is a cytopathic retrovirus and causes immune deficiency by destroying T4-cell (helper) lymphocytes. The destruction of T4 lymphocytes is necessary and sufficient for the appearance of the clinical syndrome.

The technology for counting T-cells appeared in 1980. According to the HIV theory of AIDS pathogenesis, "The Human Immune deficiency Virus (HIV), the etiologic agent of the Acquired Immune Deficiency Syndrome (AIDS), has the capability of selectively infecting and ultimately incapacitating the immune system. HIV-induced immune-suppression results in a host defence defect that renders the body highly susceptible to 'opportunistic' infections and neoplasms."[97] Lower counts of T4 lymphocytes were reported in AIDS patients and currently the selective depletion of CD4-bearing helper/inducer lymphocytes has become the hallmark for the assessment of active AIDS. Decrease of T4-cells to approximately 200 x $10^6$/l leads to the development of 'constitutional symptoms', and less than 100 x $10^6$/l to opportunistic diseases.[98]

However, neither Montagnier nor Gallo in any of their papers were able to present any proof of such cytopathic action.[5, 6]

**The in vitro tests**

Most of the hypothesis of how the virus acts has been drawn from experimental *in vitro* tests, in a laboratory test tube.

In a paper from 1986 Montagnier wrote, "Replication and cytopathic effect of LAV [the French name for HIV] can only be observed in activated T4-cells [meaning T4-cells that have been immunologically stimulated]. Indeed, LAV infection of resting T4-cells does not lead to viral replication or to expression of viral antigens on the cell surface [no HIV proteins], while stimulation by lectins or antigens of the same cells results in the production of viral particles, antigenic expression and the cytopathic effect (cell death)."[72]

In trying to prove how the action of HIV could account for the decrease of T4-cells, a hypothesis was put forward in 1991 of a "single unique mechanism, activation-induced T-cell death (programmed cell death, PCD, or apoptosis) that can account for both the functional and numerical abnormalities of T4-cells in HIV infected patients."[71] In support of their theory they reported that stimulation of peripheral blood mononuclear cells of asymptomatic HIV infected individuals with pokeweed mitogen or staphylococcal enterotoxin B was followed by cell death, whereas no death was observed at 48h in the unstimulated cells. Death was only observed in the CD4+ enriched population and not in the CD8+ lymphocytes.

So, in order to produce an experimental model of what the HIV virus *might* be doing to T4-cells, all the experiments absolutely required an immunological stimulation in order to induce cell death.

Up until 1991 very little was presented regarding the effects of the laboratory activating agents themselves on cell survival. However, Montagnier and his colleagues showed in 1990 that activation, in the absence of HIV, can induce the same cytopathic effects.[73] If this shows that HIV is neither necessary nor sufficient for the induction of cytopathic effects observed in the experimental model of HIV infected cultures, these cytopathic effects are most likely to be caused by the many activating agents to which the cultures are exposed.

Activation (stimulation) is induced by oxidation. Evidence shows that oxidising agents, including all mitogenic (activating) agents, can induce: reversible cellular changes, cellular activation, malignant transformation, mitogen unresponsive cells, and cellular death, including death by apoptosis.[74]

Apoptosis occurs under both healthy and pathological conditions, is frequently prominent among the proliferating cells of lymphoid germinal centres, and can be enhanced by numerous agents including radiation, cytotoxic drugs, corticosteroids and calcium ionophore A23187. Apoptosis is cellular death characterised by morphological criteria: cellular condensation, DNA fragmentation, and plasma membrane 'blebbing' leading to the release of 'apoptic bodies' which vary widely in size and some of which contain

pyknotic chromatin (genetic material) surrounded by intact membranes.[75-78] These changes are induced by increased concentration of $Ca^{+++}$, which in turn induces contraction of the cytoskeleton, whose main components are the proteins actin and myosin.[79-83] Intracellular free $Ca^{++}$ concentration is regulated by the cellular redox state. Oxidation leads to an increased and reduction to a decreased $Ca^{++}$ concentration.[84] Cellular surface blebbing, chromatin condensation and apoptosis are the direct result of cellular oxidation in general and of cellular sulphydryl groups in particular. In a paper from 1992, Montagnier explains how an anti-oxidant prevents apoptosis and early cell death in lymphocytes from HIV infected individuals.[85]

From another test-tube model, suspicions can arise about the cytopathic action of HIV, meaning the effect that HIV has on the cells that it is cultured in. The H9 clone is widely used, in both research and commercially, for producing what is regarded to be the HIV proteins used in antibody-test kits. What is interesting in this case is that although HIV is thought to kill T4-cells, the leukaemic cell line, as well as its H9 clone in which the HIV is grown, do not exhibit any cytopathic effect from HIV, in fact both are immortal even when infected with HIV. So these cells allow what is believed to be HIV to grow indefinitely. The proteins produced from these cells are the ones patented by R. Gallo as the antigens for testing antibody reaction. All the test kits for HIV come from these proteins.[110, 111]

**Immune suppression and antigenic stimulation**

In 1985 Montagnier wrote: "The clinical AIDS syndrome occurs in a minority of infected persons, who generally have in common a past of antigenic stimulation and of immune depression before LAV [HIV] infection."[86] This means that in the AIDS risk group, acquired immune deficiency appears before the 'HIV infection', before testing 'positive'.

In another paper from 1991, Montagnier and colleagues showed that in acutely HIV CEM cultures, in the presence of mycoplasma removal agent, cell death (apoptosis) was maximum at 6-7 days post-infection, "whereas maximal virus production occurred at 10-17 days."[87] That is, maximum effect precedes maximum cause.

A study of intravenous drug-users in New York showed that "the relative risk for seroconversion among subjects with one or more CD4 count below 500 cells/ul, compared with HIV-negative subjects with all counts above 500 cells/ul, was 4.53."[88] A similar study in Italy showed that "a low number of T4-cells was the highest risk for HIV infection."[89] The decrease in T4-cells is the risk factor for seroconversion and not vice

versa, and therefore factors other than HIV lead to both T4 decrease and 'HIV positive' tests.

Data presented from the Multicenter AIDS Cohort Study (MACS 1993) shows that HIV seropositive homosexual men at least 1.67 to 3.67 years prior to a clinical diagnosis of AIDS, as well as HIV seronegative homosexual men—although the frequency in the latter is lower—suffer from a wide variety of complaints including fatigue, shortness of breath, night sweats, rashes, coughs, diarrhoea, headaches, thrush, skin discoloration, fever, weight loss, sore throats, depression, anaemia and sexually transmitted diseases.[100] Some of the diseases that occur in these individuals, or the agents that cause them, including EBV and CMV, are immune-suppressive. Many of the agents used in treatment, including corticosteroids and some antibiotics, as well as recreational drugs like cocaine, heroin and the wide-spread use of amyl nitrite, are also known to be immune-suppressive.

Very important to the pathogenesis of AIDS is the homosexual practice of anal intercourse, regarded as immune-suppressive due to the oxidative effect of semen that brings foreign proteins directly into the blood stream by rectal absorption. The critical structural difference between the epithelium of the rectum and vagina reveals how biologically unnatural anal intercourse is. The vagina is lined with a thick stratified squamous epithelium that makes ulceration and penetration of semen into the vascular lamina unlikely. In contrast, semen in the rectum is separated from blood vessels and lymphatics by a single layer of cells that is easily penetrated and ulcerated during anal intercourse.

Studies of haemophiliacs receiving factor VIII, and by that token known to be exposed to immune-suppression, concluded: "We have thus been able to compare lymphocyte subset data before and after infection with HTLV-III (HIV). It is commonly assumed that the reduction in T-helper cell numbers is a result of the HTLV-III (HIV) virus being tropic for T-helper cells. Our finding in this study that T-helper cell numbers and helper/suppresser ratios did not change after infection supports our previous conclusion that the abnormal T-lymphocyte subsets are a result of the intravenous infusion of factor VIII concentrates per se, not HTLV-III (HIV) infection."[101]

In the light of the above studies, an immune-suppressive action on the immune system, by whatever means, and not HIV, is more likely to be the cause of the depletion of T4-cells and the cause also of the posterior seropositive conversion (HIV+ test), which in fact is a non-specific antibody marker for a particular immune disorder.

> "In TB as well as in lepromatous leprosy, an immune-suppressive state will frequently develop in the host. This state is characterised by T lymphopenia

> with a decreased number of T-helper cells and an inverted T-helper/T-suppresser cell ratio. [...] Immune-suppression induced by the infection with M tuberculosis can persist for life, even when TB is not progressive."[96]

Patients who have malaria have severe immunoregulatory disturbances including a decrease in T4-cells. A significant number of these patients also test positive for HIV but they do not develop the AIDS clinical syndrome, so "exposure to HTLV-III/LAV (HIV) or related retroviruses and the occurrence of severe immunoregulatory disturbances may not be sufficient for the induction of AIDS."[99]

On the other hand, many HIV-positive individuals continue to have normal T4-cell counts years after the seroconversion.[90] Most of these individuals remain healthy. In these cases the 'positive' test is another example of antibody cross-reaction.

**T4/T8 ratio**

T-cells are lymphocytes produced by the thymus. They are part of what is called the cell-mediated immune response. T4+ T-cells provide helper functions for optimal development of cytotoxicity in cell-mediated lympholysis. In addition, the T4+ subset produces a variety of helper factors that induce B-cells to secrete immunoglobulin and all lymphocyte sub-populations to proliferate. T8+ T-cells suppress the proliferative response of other T-cells, and B-cell immunoglobulin production and secretion.

From the beginning it was realised that in AIDS patients the decrease in T4 lymphocytes is accompanied by an increase in T8 lymphocytes, "with kinetics that mirrored the loss of CD4+ cells, resulting in a CD8 polarisation,"[91] while the total T-cell population remains relatively constant. There is an interesting explanation which reveals that the "loss of either CD4+ or CD8+ T-cells is detected by the immune system only as a decrease in CD3+ T-cells (as total mature T-cells). The compensatory response to such a selective decrease, then, is to generate both CD4+ and CD8+ T-cells in order to bring the total CD3+ T-cells back to a normal level. The consequence of this non-selective T-cell replacement after a selective depletion of one T-cell subset would be an alteration in the CD4 to CD8 ratio after normalisation of the total T-cell count with a polarisation towards the subset that had not been initially depleted. [...] Repeated events of selective CD4+ T-cell killing will result in higher and higher CD8+ T-cell counts and lower and lower CD4 T-cell counts."[91-93]

Knowing that T4 and T8 cells are identified and counted among the T-cell population by the expression of their surface antigens, which are molecular markers of differentiation,

there have been a number of studies showing that *in vitro* stimulation of T-cells by PHA, ConA, radiation PMA and polybrene, all of which are oxidising agents, leads to a possible change of phenotype (change of the type of protein on the T-cell surface). The experiments[94, 95] recorded antigenic shifts between T4, T8 and T10. So it can be argued that *in vivo*, due to the exposure of AIDS risk groups to many oxidising agents and well known mitogens, the selective decrease of T4 and the increased proliferation of T8 cells may not be the result of the destruction of the T4 subset, but of a loss of T4 surface markers and an acquisition of T8 surface markers.

**From evidence to proof**

It is necessary to point out that all the above evidence comes from laboratory counting devices and procedures. But, in the case of AIDS, these laboratory tools only produce indirect evidence and are therefore non-specific. The evidence is circumstantial, and cannot be taken as a means of discrimination. We are not saying that the counting is not accurate, but it tells us nothing about the identity of what is being counted.

When a phenomenon can be produced by different causes, in order to know with certainty which one of them is responsible, we need procedures that can reflect direct and therefore specific characteristics of the phenomenon to leave no doubt as to its cause. Only then does the evidence become proof.

Herein lies the problem, because in the case of HIV-AIDS, all of these accurate indirect methods are used for direct identification of HIV. By bypassing the standard method of identification with Gallo's short-cut, the electron micrographs become irrelevant because they do not reveal the identity of what is depicted *per se*. Reverse transcription is meaningless because it is not exclusive to retroviruses and therefore is not an identification of them. HIV proteins might not be viral proteins, but rather cellular ones, and are therefore no proof of a virus, nor does the positive HIV-test made with these proteins constitute proof. Antibody lymphocyte counting of the CD4+ subset is an indirect method of counting T-cells, and becomes even more indirect when the counting is done under conditions of immune-suppression—that is, while the patient is under immune suppressive drugs or practices.

These laboratory procedures are biological amplifiers of microscopic aspects of life, but not like a magnifying glass or microscope. Their amplification is 'blind' because it only *signals* that certain reactions are taking place. Their answer is not specific, so we cannot ask them. All of the procedures of HIV identification are in themselves not direct or specific proof of HIV, and are therefore irrelevant as a means of diagnosis.

## D) SEXUAL TRANSMISSION

Besides blood transfusion, infected intravenous needles and mother-to-child transmission, sexual transmission is considered to be the main mechanism of infection.

In the mid 1970s, sexually transmitted diseases (STDs) were rising at a terrifying rate among homosexual groups in many cities of the USA and Europe, with an alarming increase in gonorrhoea and syphilis. Promiscuity meant continuous re-infection, for which continuous antibiotic treatment as a prophylaxis was the norm. But parasitic infections were also appearing, as were persistent fevers, swollen lymph glands, chronic skin eruptions, and multiple viral infections (CMV) that would not clear up.

One of these diseases was the particular parasitic disease pneumocystic pneumonia, caused by the *pneumocystis cariini.* This is relevant because it normally only occurs as a complication produced by the use of chemotherapy to suppress the immune system in the treatment of leukaemia in children (ATL). It led to the suspicion that an immune deficiency could be behind the problem. In 1981, for the first time, blood from 30 homosexual men was tested with the newly emerging technique of T-cell counting, by D. Purtillo, a pioneer of the technique at the University of Nebraska. Ten among the 30 had extremely low counts. A blind test correlated the 10 men with a low T-cell count to high promiscuity, in comparison with the other 10 with 'monogamous' relationships and normal T-cell counts (Lancet, 1982). Promiscuity was depressing the immune system.

Epidemiological studies show that the clusters of cases of pneumocystic pneumonia had in common that they occurred in big cities (New York, Los Angeles, and San Francisco), that all were young male, very promiscuous homosexuals involved in the long-term use of antibiotics for their shared tendency to contract venereal diseases, and chronic users of nitrite inhalant and other recreational drugs for sexual stimulation.

Another of the particularly rare features of the collapse of the health of the people involved in these sexual practices was a rare form of cancer, Kaposi's sarcoma, only observed before in old men, that was now appearing among young men, and particularly affecting the lungs. Cancer researchers looking for viruses that could transmit cancer had spotted the new candidates for their oncoviral theory. The lymphocytes of these patients, they believed, must contain retroviruses. After the 'identification' of HIV, the now leading HIV-experts (formerly cancer research experts) required a means of transmission for the virus.

Sex, or to be more precise anal sex, seems to be the common link between these people. If we then introduce the idea (1984) of a virus, the equation is closed: sex must be the transmission mechanism.

The next step is to prove that heterosexual transmission is possible. However, there is no direct proof of it. There is not one single study from any country proving sexual transmission of HIV based upon evidence of HIV in genital secretions. Studies where attempts are made to prove a correlation between HIV in the blood and in genital secretions indicate that approximately 85% of male and 80% of female genital secretion samples obtained from HIV-positive subjects do not harbour HIV.[128]

The only evidence said to prove heterosexual transmission is epidemiological, meaning the correlation of seropositivity and sexual behaviour.

The male homosexual group accounts for 2/3 of all the male AIDS cases in the USA and Europe (90% of all AIDS cases are men). The heterosexual group in the AIDS statistic is made up of the remaining 1/3 of the male cases who are not homosexual, and by women (10% of all AIDS cases are women). Almost all the non-homosexual men in the group are intravenous drug-users, alongside a small number of haemophiliacs. Almost all the women are intravenous drug-users. Yet all these cases have been classified for statistical purposes as heterosexually transmitted for the simple reason that they happened among heterosexual people.

The largest study of female sexual partners of HIV-positive haemophiliacs was conducted between August 1985 and February 1989 by the U.S. Transfusion Safety Study Group. The researchers followed up 151 females HIV negative at the beginning of the study. None became HIV positive despite the fact that 13 of the 151 women became pregnant.[140]

The infective capacity of AIDS and its transmission are more an assumption than a proven fact. Doctors are at risk of contracting AIDS from patients, HIV researchers from virus preparations, wives of HIV-positive haemophiliacs from husbands, and prostitutes from clients. But in the peer-reviewed literature there is not one doctor or nurse who has ever contracted AIDS (nor HIV) from over 816,000 AIDS patients (deaths) recorded in the USA in 22 years (Centres for Disease Control and Prevention 2001). Not one of over ten thousand HIV researchers has contracted AIDS. Wives of haemophiliacs do not get AIDS. And there is no AIDS epidemic among prostitutes, except among intravenous drug-users.[106]

The longest and best study to prove that HIV is heterosexually transmitted, was a 10-year study in northern California completed in 1997.[112] Their findings showed:

The risk factors for seroconversion (becoming positive) were:

- Anal intercourse;
- Having partners who acquired this infection through intravenous-drug use;
- The presence in the females of STDs;
- They estimated that the likelihood of female-to-male transmission was 8 times lower than male-to-female;
- They estimated that the risk to a non-infected male of acquiring HIV infection from his infected female partner per contact is 0.00011 (1/9000).

This means that on average, males having sexual intercourse daily with an infected partner for 16 years (that is 6000 contacts at 365 per year), would score a 50% probability of becoming infected. But if sexual intercourse takes place once a week it would take 115 years to reach the same probability.

Under such circumstances, one must question how HIV could spread to epidemic proportions as the result of bi-directional heterosexual transmission. And yet the claimed mechanism of heterosexual transmission is said to be at the core of the present AIDS epidemic that is ravaging Africa.

## E) MOTHER-TO-CHILD TRANSMISSION

At the end of 1997 the World Health Organisation reported an *estimate* of 1.1 million children living with human immunodeficiency virus (HIV) infection world-wide. Of these, the great majority lived in sub-Saharan Africa and were infected by their mothers during pregnancy, delivery or breast-feeding.

The CDC 1994 Revised Classification System for HIV Infection in Children under 13 years of age, explains that "the diagnosis of HIV infection in children born to HIV-infected mothers is complicated by the presence of the maternal anti-HIV IgG antibody, which crosses the placenta to the foetus. Virtually all these children are HIV-antibody positive at birth, although only 15% to 30% are actually infected. In uninfected children, this antibody usually becomes undetectable by 9 months of age but occasionally remains detectable until 18 months of age. Therefore, standard anti-HIV IgG antibody tests cannot be used to indicate reliably a child's infection status before 18 months of age. Polymerase chain reaction (PCR) and virus culture are probably the most sensitive and specific assays for detecting HIV infection in children born to infected mothers."

To prove that mother-to-child transmission of HIV takes place, one must first have proof that HIV exists. At present, infection of the mother is determined by antibody tests and that of the child by an antibody test, 'HIV isolation' and measurements of HIV-RNA or DNA utilising the polymerase chain reaction (PCR). However, due to the poor infrastructure of many African Health Services and the high cost of laboratory techniques, the Bangui AIDS Definition for children in Africa was drawn. This definition gives clinical criteria to be applied in order to diagnose children infected with HIV by transmission from their mothers without any laboratory tests. In much of the African epidemiological data on children infected with HIV, the infection is solely diagnosed by the satisfaction of clinical criteria.

According to the WHO, a minimum of 330,000 and a maximum of 670,000 children in the world died from AIDS in 1999. The WHO *estimates* as follows:

- Australia and New Zealand had under 100 cases in 1999. By September 2000, 32 deaths from AIDS in children had been recorded in Australia.
- Canada, under 100 deaths in 1999.
- United States, minimum 250 and maximum 380 AIDS deaths in children during 1999. The cumulative total of child deaths from AIDS in the USA up until June 2000 is 8804.
- No estimated deaths from AIDS in children are reported from Western Europe, Eastern Europe or Central Asia, nor from North Africa or the Middle East.
- Ethiopia, *estimated* 35,000 to 91,000 deaths in 1999.
- Nigeria, *estimated* 41,000 to 64,000 deaths in 1999.
- South Africa, *estimated* 36,000 to 74,000 deaths in 1999.
- Uganda, *estimated* 18,000 to 32,000 deaths in 1999.

Although the estimated deaths in Africa, especially in sub-Saharan Africa, are high, they are only estimates.

According to the WHO there are 300-500 million clinical cases of malaria each year and more than 90% of all cases occur in sub-Saharan Africa. Most of the 1-3 million who die from malaria are children, mainly in Africa, in which malaria is hyper-endemic. The mortality rate is highest during the first two years of life.

In the WHO 2000 report we read: "Diarrhoea is the leading cause of illness and death among children in developing countries, where an estimated 1.3 thousand million episodes and 4 million deaths occur each year in under-fives. Worldwide, these children experience an average of 3.3 episodes a year, but in some areas the average exceeds nine episodes each year. Where episodes are frequent, young children may spend more than

15% of their days with diarrhoea. About 80% of deaths due to diarrhoea occur in the first two years of life."

The diseases from which HIV-infected children die are the same diseases from which non-infected children die. In particular, African HIV-infected children die from the common causes in that continent: tuberculosis, diarrhoea, pneumonia and malnutrition.

A paper entitled 'Severe malnutrition and paediatric AIDS: a diagnostic problem in rural Africa' published in 1988[130] reported the Ivory Coast's morbidity among HIV-positive and HIV-negative malnourished children. The study was about 94 children, of which 30 were HIV-positive and 64 HIV-negative. 90% of them suffered from severe malnutrition (weight less than 60% of the expected weight for their age).

| **Disease** | **HIV-positive 30 of 94** | **HIV-negative 64 of 94** |
|---|---|---|
| Chronic diarrhoea | 26 (87%) | 43 (67%) |
| Generalised lymphadenopathy | 22 (73%) | 34 (53%) |
| Oropharingeal candidiasis | 20 (67%) | 34 (53%) |
| Prolonged fever | 19 (63%) | 21 (33%) |
| Persistent cough | 12 (40%) | 13 (20%) |
| Generalised dermatitis | 4 (13%) | 6 (9%) |

The signs and symptoms which are considered to signify death from AIDS in the Bangui definition, or the latest CDC definition, are those of tuberculosis, malaria, gastrointestinal parasitic infections, and malnutrition. With clinical criteria only, it is not possible to distinguish between death from AIDS or other common African diseases.

The following data is from the Survey of Race Relations in South Africa. It illustrates the distribution of the African AIDS-defining disease of tuberculosis (50% of all AIDS cases are TB), and its increase in certain groups and decrease in others according to race, long before the AIDS era:

**Tuberculosis: reported cases 1951 (whole country)**

| Africans | Whites | Coloured | Asian |
|---|---|---|---|
| 19,392 | 1477 | 4586 | 1084 |

**Tuberculosis: reported cases 1968**

| Africans | Whites | Coloured | Asian |
|---|---|---|---|
| 61,292 | 921 | 7,481 | 990 |

**Infant mortality per 1000 live births, in 1953/54**

| Africans | Whites | Coloured | Asian |
|---|---|---|---|
| 210 | 33 | 134 | 66 |

The term 'protein-energy malnutrition' covers the spectrum of clinical conditions seen in children and adults due to under-nourishment. Severe starvation leads to a clinical feature called Kwashiorkor which occurs typically in a young child displaced from breast-feeding by a new baby, and fed a diet with a very low protein content. Apathy, anorexia, generalised oedema, with skin pigmentation and thickening, distended abdomen due to hepatomegalia and/or ascites—reminiscent of the frequent images seen on television of starving children with big bellies. Protein-energy malnutrition leads to a depression of the immunological defence mechanism, resulting in a decreased resistance to infection.[131]

From the same Survey by the Institute of Race Relations, a distribution of reported Kwashiorkor cases among races from 1964/65 shows:

| Africans | Whites | Coloured | Asian |
|---|---|---|---|
| 13,358 | Zero | 410 | 40 |

One last piece of data from the same Survey shows the percentage of children under 12 years of age with stunted growth (including Kwashiorkor) from 1988:

| **South Africa** | | **Neighbouring countries** | |
|---|---|---|---|
| Eastern Cape | 58% | Mauritius | 21% |
| Northern Cape | 80% | Swaziland | 10% |
| Transvaal | 49% | Zambia | 19% |

> "It is important to appreciate that even if the highest [30%] current prevalences of HIV-1 in Africa were found among all women of childbearing

> age, HIV would still only account for a minority of child deaths and rank some way behind mortality associated with respiratory tract infections and diarrhoeal diseases. Similarly, HIV is not the only prevalent lethal congenital infection. Syphilis is a massive source of foetal wastage and infant death in Africa. Our calculations suggest that the HIV-1 epidemic is unlikely to overwhelm most existing differentials between African countries in the level of child mortality. Countries with relatively low child mortality in the 1980s are likely to remain so in the future."[139]

The biggest epidemic in Africa is poverty. Malnutrition is the principal factor responsible for immune deficiency among the African people. The epidemiological race distribution of the diseases is none other than poverty distribution, as the South African figures of TB in the 50s and 60s show. Poverty is also the common denominator among the mothers that supposedly transmit HIV to their children in the U.S./Europe AIDS epidemic.

Looking at epidemiological data from the U.S. AIDS epidemic in children, the first feature to appear is again racial distribution. With negligible exceptions, the children who are said to have been infected by their mothers are Black and to a lesser extent Hispanics, that is, these are children born to poor women. According to Dr. H Gayle, Director of the National Center for HIV, STD and TB Prevention (CDC), in 2000: "In the United States, of all the AIDS cases reported in children, 17% are white, 58.6% black, 22.9% Hispanic and the rest other minorities."[129] The race factor of the epidemic is none other than that of poverty.

In all of the U.S. studies reporting evidence of mother-to-child transmission, over 2/3 of the mothers are intravenous drug-users. Maternal drug abuse is one of the main factors responsible for pre-term delivery and low birth weight. The studies from the USA show that children of drug-using or economically disadvantaged mothers are of low birth weight, and develop immune deficiency and a range of diseases.[133] Immune deficiency and illness in Black and Hispanic children are related to parenteral drug use by the mothers, neglect, and malnutrition.[134] In the studies of positive PCR in children with or without AIDS, all the children are born to socio-economically disadvantaged, drug-addicted mothers.[135]

In the USA, the evidence on which the conclusion of what is called vertical transmission, mother-to-child transmission, is based, is from studies of drug-addicted mothers and prostitutes whose immune-suppressed infants suffered from recurring viral and fungal infections and died from pneumocystis carinii pneumonia.[136]

In the European studies, "most of the children were born to mothers who abused intravenous drugs. As well as exposure to intra-uterine HIV infection, these infants are at increased risk of other congenital infections, low birth weight, neurological disorders resulting from drug withdrawal, and other perinatal problems. The observed excess perinatal mortality is probably due to the background of drug abuse as demonstrated by the association between low birth weight and drug abuse during pregnancy, rather than being an effect of HIV infection alone. In many cases social deprivation is encountered after birth, which can adversely affect the child's development and health."[132]

All of these infants from Africa, the USA and Europe whom we see classified in AIDS statistics as infected by HIV, have in common immune deficiency. The African children develop the immune deficiency from malnutrition; in the USA and Europe it is caused by immune-suppression from intravenous drug abuse (heroine, cocaine, amphetamines) by the mothers during pregnancy, and malnutrition.

All of the children in all of the studies are from disadvantaged mothers.

**Breast-feeding and transmission of HIV**

There is unquestionable evidence that breast-feeding protects babies against morbidity and mortality from infectious diseases. It provides ideal nutrition to the infant at no cost and gives an immunological protection against agents responsible for diarrhoeal and respiratory diseases, as well as other infections.

Regarding the isolation of HIV in breast milk, from 1985 until the present, only two publications can be found. After scrutiny, neither of them shows any direct proof of the isolation of HIV in milk. Indirect reverse transcriptase activity, reaction with rabbit hyper-immune serum against HIV, but no direct isolation of HIV. In neither paper did the author have any experimental controls, nor any comparisons with non-HIV mothers.[137]

Posterior studies have been done with PCR, amplifying what is thought to be HIV-RNA. But again this is an indirect method of identification since there is no real proof of the identity of what is being amplified.[138]

In the case of children with AIDS, the positive HIV test is completely irrelevant because the antibodies can come from the mother and not from the child, as the above 1994 CDC classification advises. So an HIV-positive test is no evidence of mother-to-child transmission before 18 months of age. Therefore, if the diagnosis has to be drawn from clinical criteria, and immune deficiency in children can be caused by factors other than

HIV—immune-suppression by intravenous drug abuse and immune-depression by poor protein-calorie intake—there is no way that we can distinguish, on clinical grounds, between an immune deficiency produced by immune-suppression, from one produced by malnutrition, and one produced by HIV. One is forced to wonder just how misleading all the statistical data about HIV-infected children might be.

The real meaning of the diagnosis of mother-to-child transmission is the therapeutic indication for the anti-retroviral drugs AZT and Nevirapine.

A particularly interesting and thorough study on the evidence regarding AZT and Nevirapine and mother-to-child transmission has been conducted by a brilliant group of scientists from Perth, Western Australia.[146] One of them, E. Papadopulos-Eleopulos, has also been responsible for one of the most scientifically elegant explanations of immune deficiency by the oxidative effect of immune suppressants.[23]

## F) AIDS IN AFRICA

AIDS in Africa is almost unrecognisably different from AIDS in the USA and Europe.

As mentioned before, 90% of AIDS victims in Europe and America to this day are males between 20 and 50 years of age. A third of these males are intravenous drug-users, and use those drugs for years at a time, and two-thirds, approximately, are male homosexuals. The remaining approximately 10% identified as AIDS risk groups are haemophiliacs, transfusion recipients, and females, almost all of whom are intravenous drug-users.

Regarding the heterosexual transmission hypothesis, the definition that in Africa the HIV virus is transmitted by heterosexual relationships is based simply on the striking fact that the syndrome in Africa exhibits a gender ratio between men and women of 1:1, whereas in the West it is 15:1.[64, 66] While this hypothesis is awkward in the USA and Europe, due to the distribution of 90% men and 10% women, in Africa the viral theory fits the pattern of an infectious transmitted disease by striking at random, affecting equally both sexes. It is, undeniably, very difficult to scientifically explain how a virus can behave so differently depending on geographical location, so much so that a person becoming infected in a Western country has a 1 in 15 chance of being a woman, while in Africa it is 50-50%.

The other striking feature about the sexual dimension of HIV is that in non-African countries, the only sexual practice leading to an increased risk of becoming HIV-antibody-positive is anal intercourse[68, 69, 70]—which is almost a non-practice among

native Africans. So HIV, like pregnancy, can only be acquired by the passive sexual partner and cannot be transmitted to the active partner. In the whole history of medicine there has never been an example of a sexually transmitted disease that is spread uni-directionally, and certainly not one that is spread uni-directionally in one country and bi-directionally in another.

AIDS researchers in Africa, including those from the CDC and WHO, admit that clinical symptoms similar to AIDS and wide-spread immune deficiency has existed in Africa for a considerable time and that this has not been due to HIV.

**Clinical symptoms of the African AIDS**

Unlike the AIDS definition in the West, the World Health Organisation's Bangui Definition for Africa does not require immunological (T4 count or antibody+) tests or a specific disease diagnosis, but is defined mostly on clinical grounds. African AIDS is not a specific clinical disease, but a battery of previously known and thus totally un-specific diseases:[63, 104]

- loss of weight (> 10% of normal body weight), and
- chronic diarrhoea (lasting at least two months), or
- chronic fever and asthenia, plus
- persistent cough,
- generalised pruritic dermatitis,
- recurrent herpes zoster,
- candidiasis oral and pharyngeal,
- chronic or persistent herpes,
- cryptococcal meningitis,
- Kaposi's sarcoma.

Yet these symptoms are common, non-specific manifestations of many diseases that are endemic in Africa and have been there long before the AIDS era:

- Kaposi's Sarcoma, classified in Western countries as one of the 'definitive diagnoses' or a 'presumptive diagnosis' for AIDS-indicator diseases, has been present in Africa since antiquity. Its characteristic clinical features are described in the Ebers papyrus that dates from 1600 BC.

- "Recognition of paediatric AIDS is particularly difficult in Kinshasa [Zaire], since many children have severe infant and childhood diseases with similar manifestations (e.g. weight loss, chronic diarrhoea)."[64]

- 'Slim disease' is a wasting syndrome seen in Africa since the first colonisation, and although it would not fall under any categorisation of AIDS by the standard empirical definition, it is nevertheless being considered as AIDS in Africa.

**Immune deficiency in Africa**

- "Tuberculosis, protein calorie malnutrition, and various parasitic diseases can all be associated with depression of cellular immunity."[59]

- "A wide range of prevalent [in Africa] protozoal and helminthic infections have been reported to induce immune deficiency."[60]

- "Among healthy Africans resident in an area with no AIDS diseases, the number of helper [T4] and suppresser [T8] lymphocytes were the same in HTLV-III/LAV [HIV] seropositive and seronegative subjects."[61]

- "Africans are frequently exposed, due to hygienic conditions and other factors, to a wide variety of viruses, including the Cytomegalovirus (CMV), Epstein-Barr virus (EBV), and Herpes Simplex Virus (HSV), all of which are known to modulate the immune system. Furthermore, the Africans in the present study are at an additional risk for immunological alterations since they are frequently afflicted by a wide variety of diseases such as malaria, trypanosomiasis, and filariasis, that are also known to have a major effect on the immune system."[62]

**Antibody test for HIV in Africa**

The data presented by the WHO and UNAIDS announcing in 1998 that Africa had gained 23 million living with HIV/AIDS refers not to people who have developed AIDS diseases, but to people who are considered, largely by projected estimates, to be HIV-positive. The figure is not the result of 23 million individual positive tests.[105]

Moreover, the fact that the HIV test is a *non-specific* antibody reaction has become more obvious than ever in African patients tested for AIDS. The cross-reactivity to antigens presented endemically in the blood of African people is forcing scientists to re-evaluate the specificity of the test. At the same time it produces very false statistics on the true extension of the 'AIDS epidemic' in the African continent.

> "Leprosy patients and their contacts show unexpectedly high rates of false-positive reactivity to HIV-1 proteins on both Western Blot and ELISA." The cross-reactivity was found to be caused by antibodies directed against two major carbohydrate-containing M. leprae antigens, phenolic glycolipid-I and, in particular, lipoarabinomannan (an arabinose-containing lipopolysacharide) which is also present in M. tuberculosis and other mycobacterium. So "ELISA and WB may not be sufficient for HIV diagnosis in AIDS-endemic areas of Central Africa where the prevalence of mycobacterial diseases is quite high."[65]

The cause of tuberculosis is a mycobacterium. Tuberculosis has been endemic in Africa for generations. Of the 661 million people living in sub-Saharan Africa, 2-3 million have active TB with an annual mortality of 790,000. Since the AIDS era, tuberculosis has become one of the AIDS-indicator diseases, indeed 30-50% of African 'AIDS deaths' are from TB. A 1986 study in a tuberculosis sanatorium in Kinshasa (Zaire) shows that half of the suspected pulmonary cases, one third of the confirmed cases and two thirds of the confirmed extra-pulmonary cases had a positive Western Blot antibody test.[67] Having seen the capacity of cross-reaction between 'HIV proteins' and mycobacteria,[65] what the test is showing is precisely a non-specific antibody reaction that indicates an abnormal immunilogical state in TB patients.

The annual mortality rate in the sub-Saharan region is 12,300,000 deaths, 75,000 of which are considered to be HIV-related. Between 30 and 50% of the HIV deaths are from TB, but every year, as I have said, another 790,000 people die from TB in the same region.

During the African AIDS epidemic the sub-Saharan population has grown at an annual rate of about 2-6% per year—from 378 million in 1980 to 652 million in 2000 (U.S. Bureau of the Census International Data Base 2001). Thus Africa had gained 274 million people since 1980. According to the report of the WHO (World Health Organisation 2001b), during the same period of time, the African AIDS epidemic has produced a cumulative total of 1,093,522 deaths. Therefore a theoretical above-normal loss, due to AIDS, of one million Africans over a period in which over 200 million were gained is statistically hard, if not impossible, to verify, unless again the African AIDS diseases were highly distinctive. But the list of African AIDS diseases cannot be clinically distinguished from their conventional counterparts, only the test can make the distinction, while the WHO decided in Bangui, Africa, in 1985 (the Bangui definition of African AIDS), to accept African AIDS diagnoses without the HIV-Test. This was done because these tests were unaffordable to most African countries (WHO, 1986).

The question then arises whether the mortality claimed for AIDS (0.6% of the annual death rate) is in fact new mortality that can be distinguished from conventional mortality, or whether it is a minor fraction of conventional mortality under a new name. The conclusion is that both acquired immune deficiency and the symptoms and diseases which constitute the clinical syndrome are long-standing in Africa.

Indeed, all the available data is compatible with an old African epidemic, that of malnutrition and poverty-associated diseases. Only the name is new.

However, the naming of the disease is crucial because it defines the treatment.

If we look again at epidemiological data on the HIV 'estimates' we come to another astonishing revelation. In Africa the 23 million estimated pool of HIV-positives (carriers) generate, as seen before, 75,000 cases of AIDS per year, which means one AIDS case per 300 HIV-positives. But in the USA, the pool of 900,000 HIV-positives generates 45,000 AIDS cases per year (Centres for Disease Control, 1999), which means one AIDS case per 20 HIV-positives. Thus the risk of developing AIDS for an American HIV-positive is about 15 times higher than for an African. Now, since over 150,000 healthy (!) HIV-positive Americans are currently treated with DNA chain-terminators and other anti-retroviral drugs,[113] as a measure to pre-empt or delay the development of AIDS, but since the American HIV-positives develop 15 times more cases of AIDS than their African untreated counterparts, to look at the AIDS treatments now becomes imperative.

## G) ANTI-RETROVIRAL DRUGS: THE TREATMENT FOR HIV-AIDS

The therapeutic agents for AIDS are another astonishing aspect of this phenomenon. All the AIDS drugs in the market are extremely toxic and work by producing immune suppression. The use of immune-suppressive drugs against an immune-deficient patient is an enormous therapeutic contradiction.

The rationale behind it is purely theoretical. So far, in 2004, it is still an assumption based on *in vitro* models, that the virus produces immune deficiency by killing T4 lymphocytes. The correlation of T4-cell depletion in AIDS patients is not enough to assume that the loss of T4-cells is due to the action of the virus. An even greater assumption is that by killing the supposed virus, the depletion of T4-cells will stop and health will be restored.

Cancer research is in fact the arena of this phenomenon. The therapeutic arsenal for cancerous malignancies of white cells (leukaemias) are drugs designed to block the excessive cellular proliferations that these malignancies produce. Their chemical action is

directed at stopping these fast-growing numbers of cells by toxically suppressing the bone marrow. These chemotherapeutics are highly poisonous drugs. They are very sophisticated immune suppressants that, by their toxic and suppressive effect, reproduce a toxicological immune deficiency, parallel to the one that the patient already has. Anti-HIV drugs cause AIDS-defining and other specific diseases, regardless of the presence of antibodies to HIV.

The fundamental problem of any chemical anti-virus 'therapy' is that the cell carries out all viral biochemical functions. Thus all anti-viral treatments are inevitably anti-cell treatments. All of the anti-retroviral drugs come from cancer research:

**Azidothymidine (AZT)**

AZT has been the first drug marketed for the treatment of HIV-AIDS. But AZT was developed in 1964, twenty years before the discovery of HIV. Jerome Horwitz of the Detroit Cancer Foundation, financed by the NIH, created a chemically modified form of a DNA building block. When a cell is about to divide it makes a copy of its genetic material by growing a new chain of DNA. Horwitz's altered DNA building block enters that new chain, blocking further DNA growth by stopping further DNA blocks from being added. The cell cannot copy its DNA sequence and dies trying. AZT was a perfect killer of dividing cells.

Experiments conducted on mice with cancer showed no result of stopping cancer, but an extreme toxicity that resulted in the death of the mice. The drug was shelved. But later the anticipation paid off.

After the 1984 announcement of HIV as the cause of AIDS there was a real rush to find a therapeutic agent against the virus. Many pharmaceutical companies joined the race.

David Barry, the head researcher at the U.S. branch of Burroughs Wellcome, selected a handful of previous rejected substances, among them AZT, and sent them to Wellcome's former collaborators. If one of these could be approved, the company would save vast sums of money on research and development. The political pressure of the time for a quick solution played in his favour.

Winning the approval of a drug by the FDA (American Food and Drug Administration) implies that the agency bans most potential drugs, automatically suppressing the competition and granting treatment monopolies for approved drugs. This monopoly alone can be worth hundreds of millions of dollars to the pharmaceutical company holding the patent.

D. Bolignesi, a veteran retrovirus researcher, found that AZT was the most potent compound in stopping the multiplication of cells infected with HIV in the test tube. Barry contacted Sam Broder, in charge of Gallo's laboratory at the National Cancer Institute. The three of them subsequently published the scientific paper.[114] The therapeutic dose of AZT able to destroy the virus was, according to the paper, 1000 times smaller than the one that could kill the T-cells, where the virus allegedly is. This theoretically meant that small doses of the drug could stop HIV without seriously damaging the immune system of the patients. The compound was classified as a reverse transcriptase terminator.

However, AZT does not attack reverse transcriptase directly. It only stops DNA synthesis, as it was designed to do in the first place. Since retroviruses can make viral DNA only in cells making their own DNA, and the T-cell has to copy one hundred times more DNA than the small virus, AZT is 100,000 times more likely to block cell DNA and kill the cell than to block the viral DNA. Moreover, like any other chemotherapeutic drug, AZT is unable to distinguish between an HIV-infected T-cell and the other non-infected T-cells, and since only 1 in 500 T-cells is ever infected even in patients dying from AIDS,[108] AZT needs to kill 499 good cells for every cell that is infected. This is not a good therapeutic index.

Nevertheless, AZT was rushed through Phase I trials, the tests that determine toxicity for humans. The Phase II study is to see whether the drug could stop the development of AIDS.

Double-blind placebo-controlled studies are the cornerstone of Phase II. This rigorous gold standard is the fundamental control for any promising drug. A study was published in 1987.[115] Burroughs Wellcome not only co-authored the study (Drucker, Nusinoff-Lehrman, Segreti, Rogers, Barry) but also paid for the licensing study of what was already its own product. The study had to be aborted early for a variety of reasons. But it concluded that AZT had a good response with a quick increase in the depleted T4-cells. The short duration of the trial (less than 4 months) did not allow anyone to see that the apparent increase of T4-cells was not a curative action of AZT, but only a homeostatic temporary reaction by which the body was increasing the production of T4-cells that were being depleted by AZT. The increase would later wear off as the progressive depletion caused by the toxicity of AZT eventually overcame the body's self-repairing capacity.

But what the study did already show were signs of high toxicity developing in patients that took the drug in comparison with the placebo group. These were somehow largely overlooked. The rumour of an effective drug against AIDS being on trial spread like

wild-fire among the increasingly terrified population of HIV-positive homosexuals. David Barry demanded special permission from the FDA for Burroughs Wellcome to sell AZT while waiting for the official approval, so the FDA granted permission to use an Investigational New Drug (IND). In February 1987 the FDA finally approved AZT. Burroughs Wellcome quickly patented the drug.

The medical manufacturer Burroughs Wellcome sold the drug under the commercial name of RETROVIR (AZT), in the form of capsules of 100mg each. The manufacturer does not warn about the toxic effects of AZT.

The biochemical manufacturer Sigma Chemical Co., on the other hand, marketed the drug under the commercial name ZIDOVUDINE (AZT). Their 100mg capsules come in a bottle with a label that has the skull-and-crossbones symbol for unusual chemical hazards. The warning reads: "TOXIC. Toxic by inhalation, in contact with skin and if swallowed. Target organ(s): Blood bone marrow. If you feel unwell, seek medical advice (show the label where possible). Wear suitable clothing."

The daily recommended dose at the time was 500mg for asymptomatic HIV-positives, and 100mg prescribed to pregnant, HIV-positive mothers.

The toxicity of AZT is due to its targeting of bone marrow. AZT does what it was designed for, to destroy DNA replication, and with it the cell. AZT is cytotoxic. It suppresses the bone marrow, depleting the organism of the same T-cells that HIV is supposed to attack, so it is immunotoxic. The immune-suppressive action of AZT ends up by producing an acquired immune deficiency. Asymptomatic HIV-positive patients taking the prescribed doses of AZT develop:[106]

- Lymphoma (in 46% of patients after 36 months of treatment)
- Dementia
- Weight loss
- Yeast infections
- Pneumocystis pneumonia

The problem is that all of the above diseases are AIDS indicator diseases. So the progressive deterioration of health and the eventual death are seen as the unfortunate development of AIDS in previously asymptomatic HIV-positive men. The cruel truth is that the chemical compound that they are taking is replicating the supposed mechanism of action of HIV: it destroys T-cells, but at a faster rate.

Considering their mechanism of action, all the DNA chain-terminators are inevitably cytotoxic and immunotoxic, like most other chemotherapies.

**DDI: another DNA chain-terminator produced by Bristol Myers Squibb**

This new drug with a similar action to AZT added two more toxic effects not observed with AZT: fatal pancreatic damage and neural peripheral destruction (Merck Index).

**HIV protease inhibitors**

The HIV protease inhibitors were designed to inhibit specifically auto-proteolytic processing of HIV proteins, which is necessary for HIV assembly.

The doses at which these inhibitors 'block HIV replication' in the test tube (we must not forget that the proof of blocking is indirect, by looking for RT), did not produce any evident therapeutic effects.

The doses were increased to 4-5 orders of magnitude above what is needed to render HIV non-infectious *in vitro*, or to 1-2 grams per day.[115]

Saquinavir by Roche and Ritonivar by Abbot are two of these new retrovirals. The toxicity of these drugs is enhanced by the amounts prescribed. The doses currently administered to patients are at least 50 times higher that what is needed to completely inhibit the cellular intestinal aspartyl protease cathepsin D. The destruction of cathepsin D accounts for anorexia (weight loss) and diarrhoea. Affecting probably, as it does in mice, the thymus and spleen, a massive loss of T-cells and B-cells leads to death.

We see the same pattern that we saw with DNA chain-terminators. The toxic effects of the protease inhibitors can cause at least three AIDS defining diseases.

**Drug cocktails**

AZT and other DNA chain-terminators are now typically supplemented by inhibitors of proteases to form drug 'cocktails'. A daily dose of these includes about 1g of one or more DNA chain-terminators per clinically ill person, and 0.5 g per asymptomatic HIV-positive per day, which is equivalent of 1.5 to $3 \times 10^6$ molecules of DNA chain-terminator per body cell.

**Nevirapine**

Nevirapine is an anti-retroviral drug manufactured by Boehringer Ingelheim/Roxane and commercialised under the name of Viramune. Nevirapine is one of the anti-retroviral drugs that has been promoted for stopping mother-to-child transmission of HIV. Especially in Africa, Nevirapine has been pushed hard for use in pregnant women. That use of Nevirapine comes from a single study, the 1999 Uganda study. In the study, "a single 200mg tablet was given to the mother at the onset of labour, and a single dose of nevirapine suspension 2mg/kg for the neonate administered at 72 hours after birth or at discharge from hospital, whichever occurred first."[143]

The study claims to have reduced the percentage of HIV transmission (15-30%) further than AZT, and it campaigns for the use of Nevirapine instead. But since Nevirapine, like AZT, due to its pharmacological mode of action, is capable only of preventing infection of cells not already infected and is unable to inhibit the expression of HIV within already infected cells or eradicate the virus, when the drug is given to neonates, especially 3 days post partum, it will have no effect on mother-to-child transmission *in utero* or during labour and delivery. Nor will the single dose of 200mg of Nevirapine have any effect on the mother's milk, because it does not reach the concentration necessary to have an anti-retroviral effect. The same happens with the single dose given to the infant.

The European Agency for the Evaluation of Medicinal Products (2000) recommends the use of Nevirapine only for combination therapy and only for "infected patients with advanced or progressive immunodeficiency."

Nevirapine monotherapy does not have a significant effect on the increase of T4-cells and does not inhibit progression to AIDS.[144, 145]

The following information is quoted from the Physicians Desk Reference (PDR), 2001 edition:[38]

- Nevirapine is a non-nucleoside reverse transcriptase inhibitor that belongs to the dipyridodiazepinone chemical class of compounds.

- Nevirapine binds directly to reverse transcriptase and blocks the RNA-dependent and DNA-dependent DNA polymerase activities by causing disruption of the enzyme's catalytic site.

- Animal studies have shown that nevirapine is widely distributed to nearly all tissues and readily crosses the blood-brain barrier. It is also widely distributed in humans, readily crosses the placenta, and is found in breast milk.

- Nevirapine is known to be active in peripheral blood mononuclear cells, monocyte derived macrophages, and lymphoblastoid cell lines.

- Nevirapine is known to increment oxidative metabolism in humans. *In vivo* studies in humans and *in vitro* studies with human liver microsomes have shown that nevirapine is extensively biotransformed via cytocrome P450 metabolism to several hydroxylated metabolites. Nevirapine is extensively metabolised by the liver and nevirapine metabolites are extensively eliminated by the kidneys. However, the pharmacokinetics of nevirapine have not been evaluated with either hepatic or renal dysfunction.

- The relationship between *in vitro* susceptibility of HIV-1 to Nevirapine and the inhibition of HIV-1 replication in humans has not been established. At the present, there are no results from controlled clinical trials evaluating the effect of Viramune (nevirapine) in combination with other anti-retroviral agents on the clinical progression of HIV-1 infection, such as the incidence of opportunistic infections or survival.

- Patients should be informed that Viramune therapy has not been shown to reduce the risk of transmission of HIV-1 to others through sexual contact or blood contamination.

**Side Effects**

The long-term effects of Nevirapine are unknown at this time [year 2001].

There are no adequate and well-controlled studies in pregnant women. Nevirapine should be used during pregnancy only if the potential benefit justifies the potential risk to the foetus.

Nevirapine produces significant decrease in foetal body weight. The evaluation of the pharmacokinetics of nevirapine in neonates is ongoing and the safety profile in neonates has not been established. Three clinical trials with nevirapine in paediatric patients reported an overall incidence of related adverse events in 46% of children.

Nevirapine causes anaemia, thrombocytopenia and granulocytopenia, especially in paediatric patients.

WARNINGS: *Severe life-threatening skin reactions, including fatal cases, have occurred in patients treated with Viramune. These have included cases of Steven-Johnson Syndrome, Toxic Epidermal Necrolysis, and hypersensitivity reactions characterised by rash, constitutional findings and organ dysfunction. Patients developing signs or symptoms of severe skin reactions or hypersensitivity reactions must discontinue Viramune as soon as possible.*

*Severe and life-threatening hepatotoxicity, including fatal Hepatic Necrosis, has occurred in patients treated with Viramune.*

*Resistant virus emerges rapidly and uniformly when Viramune is administered as monotherapy, therefore, Viramune should always be administered in combination with anti-retroviral agents.*

There is a more sinister aspect to these drugs besides their toxicity. By producing immune-suppression, they are toxically inducing an immune deficiency that replicates the one from which the patient is already suffering. What the anti-retroviral drugs in fact do is to further increase the immune deficiency by selectively destroying T4-cells, and the patient ends up dying and fulfilling at the same time the expectation that the virus is cytophatic for T4-cells, and kills the patient by depleting the immune system of these T4-cells. The drugs do this even more effectively and quickly than the virus. The virus can take up to 20 years to develop into AIDS. With sustained treatment using anti-retroviral drugs, you can be dead in 3 or 4 years.

## V. DECONSTRUCTION OF THE APPARENT PARADOX

Initially we have looked at the epidemiological data on AIDS and encountered paradoxical behaviour in the epidemic. Furthermore, we have realised that the paradox was in fact created artificially by judging the cause of the epidemic to be a single infectious agent, the retrovirus HIV.

More doubts arose while looking at the above evidence on HIV. These doubts can be summarised as follows:

### Reasons to doubt that HIV is the cause of AIDS

#### a) Because of its non-randomness

The most striking characteristic of the U.S. and European epidemic has been, and still is, the specific distribution of AIDS—what we called at the beginning the geography of AIDS.

AIDS in the USA and Europe continues to affect the same group of people: male homosexuals (2/3), intravenous drug-users (1/3), 1% are haemophiliacs and other transfusion recipients, and 1% are children born to drug-addicted mothers.

A recent report from the WHO (September 2003, HIV/AIDS in the European Region) confirms the same pattern:

> "The European Region is experiencing the fastest growing HIV epidemic in the world, and significant further growth is likely. Between 1995 and 2003, the number of newly reported HIV-positives in western Europe doubled to almost 170,000, and in central and eastern Europe grew from 27,000 to 320,000. It is now *estimated* that at least 1.7 million people in Europe are already HIV-positive.
>
> An epidemic of injecting drug use is fuelling the HIV epidemic. In the former Soviet Union, where 2/3 of all Europeans infected with HIV live, 84% of all HIV cases with a known transmission route are attributed to <u>injecting drug use</u>.
>
> In western Europe sexual transmission is the dominant route, with the largest number of infections among <u>men who have sex with men</u>."

**b) Because other factors present in all of the AIDS cases can account for the same AIDS-specific diseases, with the same hallmark of T-cell depletion.**

The homosexual group

The major sources of Acquired Immune Deficiency in this group are immune suppression caused by:

*I. Toxicity of prescription drugs*

Ia. Anti-retroviral drugs

About 2/3 of all AIDS cases (deaths) in the USA and Europe are among male homosexuals. Of the group of AIDS-specific diseases, this group typically develops Kaposi's sarcoma, lymphoma, dementia, weight loss, yeast infections and pneumocystis pneumonia.

Apart from Kaposi's sarcoma, these diseases are developed by all patients under long-term prescription of AZT. The immune suppressive effect of the retroviral chemotherapy efficiently kills the patient in the same way that the virus is supposed to. Since about 450,000 U.S. citizens are currently on DNA chain-terminator and protease inhibitors as a prophylaxis against or a therapy for AIDS, these drugs alone could have been sufficient to cause all of the 43,158 AIDS deaths reported in the USA in 2001.

Ib. Other prescription drugs

Gonorrhoea, syphilis, hepatitis B, herpes and amoebiasis are much more common in homosexual males than among heterosexuals. Also, homosexual males develop various bowel infections that cause persistent and recurrent diarrhoea.[121] Most of the agents used for the treatment of these diseases are oxidising, mitogenic and immune-suppressive agents.[122]

Steroids and antibiotics are probably among the most abused medical substances in this group.

Ic. Other viruses in homosexuals

Cytomegalovirus (CMV) and Epstein-Barr virus (EBV) are present among homosexual men as well as in the rest of AIDS patients. Both viruses produce clinical and immunological abnormalities similar to those seen in AIDS patients. These viruses induce immune suppression *in vitro* and *in vivo*, including abnormalities in the T4/T8 ratio both in humans and animals. Both viruses have been isolated from many sites, including KS, from almost all AIDS patients.[121] Unlike the above viruses, HIV has never been isolated in fresh AIDS tissues.[123]

*II. Toxicity of recreational drugs*

Kaposi's Sarcoma only develops among male homosexuals using Nitrite inhalants. These drugs are immune-suppressive, mitogenic, and carcinogenic.[120]

Studies post-1984 show categorically that male homosexuals with AIDS or at risk from AIDS have continued to use nitrite inhalants, amphetamines, cocaine, heroin, and steroids.

*III. Immune-suppression by anal sex*

"Receptive anal intercourse accounted for nearly all new HIV infections among homosexual men enrolled in this study." (Multicenter AIDS Cohort Study of 4995 male homosexuals).[119] What that means is that the rectal absorption of sperm was responsible for the immune-suppression. Sperm is a strong immune-suppressive agent. Mature sperm is much more effective in producing immune-suppression than immature sperm. Mature sperm is a highly oxidising agent. Oxidation of the host immune system leads to immune-suppression.

The critical structural difference between the epithelium of the rectum and vagina reveals how biologically unnatural anal intercourse is. The vagina is lined with a thick stratified squamous epithelium that makes ulceration and penetration of the semen into the vascular lamina unlikely. In contrast, the semen in the rectum is separated from blood vessels and lymphatics by a single layer of cells that is easily penetrated and ulcerated during anal intercourse. "In addition to lymphoma and Kaposi's sarcoma, the male homosexuals develop another two malignancies: cancer of the tongue and rectum."[121]

The Intravenous drug-user group

About 1/3 of all AIDS cases in the USA and Europe are male and female intravenous drug-users, and of those, 75% are male.

The AIDS-defining diseases that this group commonly share are: tuberculosis++, dementia+, weight loss+, yeast infections+, pneumocystis pneumonia+.

Heroine, cocaine and amphetamines are oxidising agents, just like amyl nitrite.

Immunological and clinical abnormalities similar to those seen in AIDS have been reported in drug abusers as far back as 1973.

- Immunological abnormalities include: absolute lymphopenia (low count of lymphocytes), decreased concentration of IgM and IgG antibodies, and false-positive results to many serological tests in as many as 40% of intravenous drug-users.

- Clinical abnormalities include: lymphadenopathy ranging from benign hyperplasia to malignant lymphoma, other malignancies, fever, night sweats, chills, weight loss and increased susceptibility to opportunistic infections.

Transfusion recipients group

1% are haemophiliacs and other transfusion recipients.

"The abnormal T-lymphocyte subset is a result of the intravenous infusion of Factor VIII concentrates *per se*, not HTLV-III (HIV) infection."[118]

Factor VIII has been found to be immune-suppressive both *in vitro* and *in vivo*, the T4/T8 ratio being inversely correlated with the quantity of factor VIII concentrate administered. Factor VIII is a high molecular weight glycoprotein complex, whose subunits are linked by a large number of SS bonds. The oxidant activity comes from the SS bonds needed for agglutination activity. Evidence exists that all clotting agents are oxidising agents. Randomly selected patients from the haemophiliac group and from the male homosexual group show that 70% of the haemophilic group was reported to be HIV-positive compared to 45% in the homosexual group, but only 0.06% of haemophiliacs develop AIDS.[124] As in all other AIDS patients, the virus in these groups has been isolated only *in vitro*.[125]

The artificially sustained life of the T-cell in a test tube has revealed the proteins used for the test. This means that the HIV test, at best, is an indirect test that correlates immune-suppression with the appearance of certain proteins.

Children group

1% are children born to drug-addicted mothers.

Most babies with AIDS in the USA and Europe are born to mothers who have used recreational drugs (heroine, cocaine, amphetamines) intravenously during pregnancy, but have also used anti-viral drugs.

All HIV-positive, pregnant mothers are now treated with AZT during the last 6 months of their pregnancy to reduce the possibility of mother-to-child transmission of HIV, which stands at 25 to 50%. Therefore, the HIV-free babies born to these mothers—that is, more than 50% of them—have all been treated with AZT.[126] Yet because of the toxic effects of AZT, these HIV-free babies suffer from diseases such as fever, pneumonia, anaemia and mitochondrial dysfunction.

There is no mother-to-child transmission of the disease. What the mother passes on to the child is polluted blood, an immune-deficient state produced by immune-suppressive drugs, recreational and medical.

**c) Because of the difference in the epidemic's features between countries with very different economies**

The geography of AIDS, we have seen, is even more specific. There is an AIDS of Western developed countries—countries with thriving capitalist economies and decadent behaviour patterns stemming from social collapse—and there is an AIDS of poor underdeveloped countries, with endemic low protein-calorie intake, poor water resources and sanitation, and endemic intestinal parasitosis. And in these poor countries AIDS, like hunger, attacks at random: 50% men, 50% women.

Scientists have allocated a different virus to each type of AIDS geography, HIV-1 for the rich and HIV-2 for the poor.

The prevalent U.S.-European AIDS-specific diseases:

| | |
|---|---|
| Pneumocystis pneumonia ++ | (homosexuals and iv-drug-users) |
| Kaposi's sarcoma ++ | (homosexuals—nitrite inhalants) |
| Lymphoma + | (AZT) |
| Weight loss + | (AZT, retrovirals, iv-drugs) |
| Yeast infections + | (AZT, retrovirals, antibiotic overuse, iv-drugs) |
| Dementia + | (AZT) |
| Tuberculosis ++ | (characteristic of iv-drug-users) |
| Bacterial pneumonia ++ | (children in USA and Europe) |

The prevalent African AIDS-specific diseases:

| | |
|---|---|
| Tuberculosis ++ | (endemic: almost 3 million per year in sub-Saharan region) |
| Weight Loss ++ | (slim diseases; endemic under-nourisment) |
| Bacterial pneumonia + | (children and adults antibiotic resistant) |
| Diarrhoea + | (endemic intestinal parasitosis) |

If we put together both epidemics, USA-Europe and Africa, the promoting factors we identify for the development of AIDS-defining diseases are: homosexuality, iv-drug use, retroviral medications and other chemical pathogens, and malnutrition. Distinct chemical pathogens cause distinct AIDS-defining diseases. Since chemicals are not self-replicating like viruses, pathogenicity is dose- and time-dependent (i.e. it takes 20 or more years of smoking to develop lung cancer). And since there is no immunity against drugs or malnutrition, nor against drug- or malnutrition-induced diseases, the corresponding epidemics are not self-limiting, as an infectious epidemic would be once natural immunity has developed (that being the rationale behind vaccinations).

Therefore people who are not subject to drugs or malnutrition, or who discontinue drug use or malnutrition, before irreversible damage has occurred, do not develop AIDS, regardless of having antibodies to HIV.

What we can see is that the Acquisition of the Immune Deficiency Syndrome in the USA and Europe is done willingly. The immune-suppression is self-inflicted by unhealthy behaviour, or inflicted by medical prescriptions, or medical products. It is nothing other than blood pollution, the disease of plenty.

The Acquisition of the Immune Deficiency Syndrome in Africa, on the other hand, is done unwillingly. The immune deficiency is determined by factors beyond the control of the sufferers. The new forms of economic colonialism are keeping the sub-Saharan

region under deprived poverty. It is nothing other than undernourished blood, the disease of the poor.

**Doubts regarding the cytopathic action of HIV and its route of transmission**

The inference that the low count of T4 lymphocytes is the result of the cytophatic action of HIV is based on the belief that HIV is inside the cell that is killed. The proof of the pathological effect of HIV is based on evidence from *in vitro* models and epidemiological data of the concomitance of low counts of T4-cells in patients suffering from AIDS-defining diseases.

The heterosexual transmission of AIDS is, again, epidemiological data under the wrong heading. Heterosexual AIDS patients in the USA and Europe are either intravenous drug-users or haemophiliacs.

The African positive HIV-test is very important to the West. Indeed, the randomness of Africa's epidemic is the very proof needed to support the theory that AIDS is heterosexually transmitted, which supports the hypothesis of a sexually transmitted disease. Quite apart from the imperative suggestion that Africans must be very promiscuous in order to develop an epidemic of such proportions, this is also a smoke screen to vindicate the unnatural practice of homosexuality. The fact is that the outcome of homosexual promiscuity is an immune deficiency, and therefore an extraordinary unveiling of the sickness of the practice. It is *contra-natura*: it makes you ill. But by blaming a virus, everyone is made to seem at risk.

**Doubts regarding the diagnosis of AIDS by a positive HIV test**

The test is the only common ground between these two otherwise completely different phenomena. The core support for the HIV hypothesis is that people with AIDS have a positive antibody reaction to the proteins of HIV. The fact that many people with AIDS test positive shows *per se* a correlation but not a causation.

As explained before, the 1984 identification of HIV is scientifically unreliable, and there is no scientific evidence that can substantiate the claim that the proteins obtained from that experiment are viral proteins.

Strictly speaking, according to the evidence, what we have is proteins (p24 and p41), which we know to have come from diseased lymphocytes, to which people with acquired

immune deficiency (low T-cell counts and AIDS-defining diseases) and people with no immune deficiency (normal T-cell counts and good health) react positively.

This means that in both groups their blood has the ability to recognise these proteins by means of antibodies. Having antibodies against these proteins gives no indication of, indeed bears no relation to the state of health of the person.

Because the testing for antibodies against these proteins was never done before 1984, we do not even know since when the human organism has had the capacity for immune recognition of them. Furthermore, if these proteins belong to ill lymphocytes it means that, if they are new, what they express is a phenotypic change that the disease is causing in the lymphocytes. The proteins that trigger the antibody reaction are normal cellular proteins, cellular proteins with new antigenic epitopes, or newly induced cellular proteins. The immune system's ability to spot a new protein on the surface of an ill lymphocyte is precisely one of its inherent capacities. As we have seen in the evidence so far, activated lymphocytes will express new molecules on their cell surfaces. The recognition of these new molecules in somebody's immune system only indicates that that person has a *sensitive marker* for activated lymphocytes. These antibody markers may even be hereditarily transmitted in the same way that we inherit other immunoglobulins or endogenous retroviruses. No-one has systematically tested parents or grandparents of adult HIV-positive patients.

> "The cancer protocol of immune-vigilance (checking for specific proteins as specific indicators of cancerous diseases) is based on a clear concept that tumour cells express antigens that are not present in their normal homologous tissues. The malign transformation can be accompanied by cellular phenotype transformation, with the loss of normal antigenic components of the cell surface and the acquisition of neo-antigens. Some of these neo-antigens are capable of evoking an adaptive immune response."(117)

The 1983/84 HIV test is at best a non-specific indicator of altered lymphocytic homeostasis. The proteins represent no more evidence than that.

The test is the export from the West to Africa. With it comes the renaming of well-known, long-established diseases, and its unique medical value is that it defines the treatment.

## VI. THE LONG-RUN ECONOMIC COST OF AIDS-BUDGETS

In an article in TIME Magazine, 23 April 2001/Vol. 157, No. 16, a plan that "could save the lives of 5 million people" was budgeted as follows:

| | |
|---|---|
| HIV tests for 10 million people | $43 million/year |
| AIDS drugs for 1 million patients | $650 million/year |
| Supervision of therapy | $200 million/year |
| Clinical treatment | $230 million/year |
| Research costs | $25 million/year |
| | |
| Total initial funding required | $1.15 billion/year |

If we take the annual GDP of the African countries, and then calculate, using the figures given by the WHO of HIV-carriers in Africa, what the cost per country of 'saving' its infected people would be, then we realise the kind of predicament the African continent is facing.

## VII. CONCLUSIONS

Now that we have looked at the evidence, we can start to draw conclusions.

We will begin by looking at what the Acquired Immune Deficiency Syndrome epidemic is **not**:

- AIDS is **not** an infectious disease, is not contagious and is not transmitted sexually or otherwise.

- AIDS is **not** caused by HIV—neither in the USA and Europe, nor in Africa. Therefore, in order to be precise, we can no longer call the phenomenon HIV-AIDS.

- AIDS, Acquired Immune Deficiency Syndrome, is **not** a syndrome in every place in the world. In accordance with the proper meaning of the term, we have to say that AIDS only exists in the USA and Europe, and that is because:

    - **Acquired**: only in the USA and Europe is the immune deficiency acquired—added to which, the acquisition is voluntary.

    - **Immune Deficiency**: only in the USA and Europe is the immune deficiency produced by immune suppression. This immune suppression is <u>self-induced</u> by unnatural sexual practices, prescription and recreational drug abuse, intravenous toxicomania. Or it is <u>medically induced</u> by the prescription of anti-retroviral chemotherapy. So as we said before, it is a voluntary acquisition of immune deficiency.

    - **Syndrome**: only in the USA and Europe can we accept that the Acquired Immune Deficiency is a Syndrome, in the sense that immune suppression is the common cause of the different AIDS-defining diseases, and therefore all these diseases can be classified as part of the same syndrome.

    - **Epidemic**: after 1981 the AIDS epidemics of the USA and Europe increased steadily for a decade and, after reaching peaks in the 1990s, they decreased to about what is currently half of their peak levels (WHO, 2001b). The new Eastern Europe epidemic is an intravenous-drug abuse epidemic, and the ‘counting’ (HIV testing) only begun recently. The figures that

make the syndrome look like an epidemic are not the numbers of people dying of AIDS, but the recorded figures of people who have tested HIV-positive. The only epidemic, if there is one, is the one created by the test: the epidemic of tested HIV-positives together with the estimates.

- AIDS in Africa, according to the proper meaning of the term, does not exist at all, and that is because:

  - **Acquired**: the African immune deficiency is not acquired, but developed by endemic poor protein-calorie intake. There is no voluntary acquisition, rather it is imposed.

  - **Immune Deficiency**: in Africa the immune deficiency is not produced by the use of immune-suppressants, but is the consequence of underdeveloped immunity, caused by endemic malnutrition. Therefore, as we have said, it is not acquired but developed, not voluntary but imposed.

  - **Syndrome**: the African AIDS-defining diseases do not have immune deficiency as their sole common cause, therefore they cannot be associated in the same group to form a syndrome. The different diseases of African AIDS have been endemic to the region for a very long time and each one has its own well-known, varying causes. In some cases immune deficiency is the cause, in some cases it is concomitant with the cause, in some cases it is the result of the cause, and in other cases it is not there at all.

  - **Epidemic**: in Africa, AIDS is not epidemic but **endemic**: and has always been here. The only epidemic is the epidemic of HIV-positive estimates, by the statisticians working for the World Bank, IMF, WHO, CDC, and other organisations. After applying their algorithmic projections to the 'estimated' numbers of HIV-positive people, or 'carriers' as they prefer to call them, they arrive at terrifyingly apocalyptic numbers of future generations wiped out.

    The most frightening aspect of all is that all the economic packages of the financial institutions (IMF, WB) come together with these statistical projections, which of course are presented as a liability to the nation-borrower. As a collateral the nation has

> to undertake to 'tackle the problem' by giving to every AIDS patient the expensive anti-retroviral drugs. These institutions are on hand to lend these sums, perhaps even at a discount for charitable reasons. But by giving the toxic anti-retrovirals to everyone who is HIV-positive, their projections will become true, only with one slight correction: the HIV-positive patient will not die of AIDS, but of AIDS-treatment. Statistically it will be the same. And aside from the loss of human life, the country will be bankrupt in footing the bill.

After what we have said, if there is indeed any AIDS in Africa it must be from the U.S.-European type of AIDS risk disease group, meaning: young male homosexuals, male intravenous drug-users, young female intravenous drug-users.

Again, in order to be precise with nomenclature—the naming—we definitely would need to have new names for the phenomenon of immune deficiency, all according again to its geographical features. According to that the name could be:

- For the disease seen in the male homosexual group: Acquired T4 lymphopenia from immune-suppression as a result of anal sex.
- For the disease seen in the iv-drug-user group: Acquired T4 lymphopenia from immune-suppression by iv-drug use.
- For the diseases seen in Africa: T4 lymphopenia related diseases, seen in some cases or stages of TB, chronic diarrhoea, slim syndrome, etc.

It is also time to see what HIV is **not**:

- HIV is **not** the cause of AIDS. HIV does not produce immune deficiency.
- HIV is **not** a transmissible virus.
- HIV proteins, the ones that make the HIV-test, are **not** from a virus. The HIV proteins come from diseased lymphocytes, stimulated and grown in a laboratory test tube. The proteins are the phenotypic changes produced by an ill lymphocyte. At the most, a positive antibody reaction to these proteins (a positive HIV test) means an indirect indication of a degree of homeostatic lymphocyte failure, most probably caused by whatever disease, past or present, the patient providing the lymphocytes is suffering from. Therefore:

- The HIV test does **not** prove the presence of any virus.

- HIV does **not** exist as an individual entity. As part of a phenomenon, HIV appears only in the confinement of the test tubes of cancer research laboratories, where leukaemic lymphocytes are artificially being stimulated and grown.

- HIV-AIDS is **not** a viral, transmissible disease that causes the immune deficiency, which in turn causes an array of 30-odd diseases. HIV-AIDS is a failed hypothesis, a case of bad science. In itself it is merely an artificial and flawed correlation, and as such does not exist.

After having seen what AIDS and HIV are not, and what HIV-AIDS was supposed to be, it is time to find out what HIV-AIDS really is.

To do this we will have to turn the whole affair upside down and look at it from a phenomenological viewpoint. The phenomenon will appear clearly in front of us as soon as we are able to identify where it takes place—the existential arena of the phenomenon—and what the indicators of the phenomenon are—that by which it acquires its meaning, the meaningful references, the references that give reality to the phenomenon. So, let us look at the biographical aspect of HIV-AIDS, at the chronological coming-into-being of the phenomenon, to see where it begins and where it ends.

In so doing we will establish the conditions to allow the real phenomenon behind the apparent HIV-AIDS to show itself to us, from itself, from its own reality, as itself, as what it is.

- **1964.** The first element to appear in the existential arena is the drug that destroys the DNA of viruses that can supposedly cause cancer, that is the DNA chain-terminator, AZT. This chemotherapy drug was designed for a virus that would produce cancerous malignancies. The virus and the cancerous diseases that the virus supposedly produces are yet to be found. So the drug ‘needs’, as it were, a disease, a cancerous malignancy, and a virus that causes it, to fulfil its destiny intended by the manufacturer: to be a cancer therapeutic drug prescribed everywhere.

- The **second** element to come into being is the set of specific laboratory biological tools.

Analytical laboratory tools are in one way or another a counting mechanism. Counting requires that you look for something that can be counted. But because they did not have the disease yet, nor the virus either, the specific laboratory diagnostic parameters that would allow them to measure the phenomena (how to diagnose the presence of the virus and the action of the virus) were the outcome of the counting elements, the tools. In fact, the tools ended up determining the parameters, not the other way around as should be the case. 'Techne' sets the enframing, the way in which things need to be looked at in order to be countable. All of these analytical tools produced indirect evidences to sustain the apparent phenomenon of HIV-AIDS. These tools were:

- **1970. Reverse transcriptase (RT)**. An exclusive viral-replication enzyme from a leukaemia cell (lymphocyte) appeared. It was a particular DNA polymerase. This enzyme was believed to be exclusive to retroviruses. So finding reverse transcriptase became the indirect method of identifying the presence of a retrovirus. Science now knows that RT also occurs naturally, and is produced in especially large amounts in fast-dividing cells such as lymphocytes in the bone-marrow. Finding RT is therefore no longer specific proof of retroviruses.

- **1975. Interleukins 2 (IL2)**. Interleukins are growth factors (IL-1 and IL-2) that allow the growth of leukocytes in a test tube. Confirming the hypothesis of the retrovirus required the cultivation of cells for long periods in order for the virus to reveal its proteins. The need for cellular growth in a test tube brought about interleukin-2. In fact, R. Gallo himself was involved in the discovery of T-cell growth factor, as he called it, which is now know as interleukin-2.

- **1975. HT23V.** The first human retrovirus, found by R. Gallo in lymphocytes from a rare form of leukaemia. It was a mistake, and is no longer mentioned in text books.

- **1977. HTLV-I.** Now the first known leukaemia virus. Found by R. Gallo.

- **1979. HTLV-II.** Second human retrovirus from another type of rare leukaemia. Also found by R. Gallo.

- **1980.** Analytical technology with monoclonal antibodies allows scientists to **count CD4+** subsets of lymphocytes for the first time. Selective immune deficiency of T-cell lymphocyte appears in the medical arena.

- **1984. HTLV-III or HIV** is officially declared to be the cause of AIDS. Third human retrovirus discovered, also by R. Gallo. Now we can see why the other two medically irrelevant retroviruses are so important. They give reality to HIV by giving it a historical background: a family, a 'belonging'. HTLV-I and II are oncoviruses because they produce cancer, HTLV-III or HIV is a lentivirus because it is slow, and all of them 'belong' to the family of retroviruses. The imagination of Gallo went so far as to suggest that the slave-trade and African monkeys were the route by which the virus might have reached the West.[127]

- **1984.** The **HIV test** appears. It was made from viral proteins that appeared in a mixture of blood from different AIDS patients, grown in a clone of a lymphocyte cell line from a patient with adult T-cell leukaemia, kept alive for more than ten years in a test tube lab. These proteins are the antigens for the HIV test. The HIV test is patented. The diagnostic tool is finalised. HIV-AIDS is marketed world-wide. The test spawns a new epidemic of HIV-positive people.

- **1987.** The **first anti-retroviral AZT** appears on the pharmaceuticals market. R. Gallo was also indirectly involved in the trials for licensing the drug. AZT, after 20 years of existence on a laboratory shelf, became the saving drug for AIDS. Patented by Burroughs Wellcome, it was commercialised under the name Retrovir. Every HIV-positive person in the USA and Europe, with or without AIDS symptoms, was advised by the medical establishment to begin to use it.

As we can see, the phenomenon of HIV-AIDS begins with anti-cancer drugs (AZT) and ends up with anti-retroviral drugs (AZT and others).

A thing comes into being by the driving force that surges out of its need to fulfil its purpose of existence, that for which the thing was created. The core of 'techne' unravels creation (nature) as 'a standing reserve' for future profit. Profit, in our days more than ever, defines the direction of research. Routes of investigation are pursued to the end, only when a good profitable outcome can be foreseen. In pharmaceuticals, that means developing a new drug that could be licensed for a disease that has no previous successful treatment, or no treatment at all. The markets of the pharmaceutical conglomerates are the human diseases themselves, and for the last sixty years cancer has been at the top of the research agenda for most of the big companies. Since the 1950s, pharmaceutical drug development has been shaped to a great degree by cancer research.

Cancer research has long been pursuing the idea that cancer can be transmitted by a virus, since this can bring good profits. If an oncovirus is the cause of cancer, this reduces the target of the treatment to a single cause. Therefore, targeting the virus means treating cancer specifically. If the cause of cancer were your life-style, then the cure would be to change that life-style, but that hypothesis is not that profitable, at least not to the pharmaceuticals. A single agent that can be targeted by a sophisticated drug, on the other hand, can be a winner.

The phenomenological approach allows us to conclude as follows:

The phenomenon in itself is the development of specific drugs for cancer treatment.

The meaningful references are the analytic laboratory tools that cancer research, in its quest for the virus, has itself developed.

The existential arena is cancer research grants, cancer research laboratories and their retrovirologists, looking for viruses among the pathological output of the decadent, post-industrial, capitalist American society and its European counterpart.

The goal of the phenomenon is the ultimate existential purpose of that specific drug, which is to find the disease for which it has been developed, in order to fulfil the ultimate purpose of its creation: the use of the drug by the sufferers of the diseases. And if it is the only drug on the market for that disease, all the better. Since the drug has achieved its goal in a sustained manner for the last 20 years, after reflecting deeply, we can only conclude that what appears in the HIV-AIDS phenomenon is the unfolding of a successful marketing scheme.

That marketing scheme is done almost exclusively by the test. The test defines the new market: the new disease of HIV-AIDS. Phenomenologically speaking, we can say that HIV-AIDS is only a new disease as long as HIV is attached to AIDS as a definition. HIV has made AIDS a new disease, the disease for anti-retroviral drugs, and has lent it its epidemic proportions of millions of HIV positives.

Hence, the HIV test is the most pivotal reference of meaning, because medically it is the foundational parameter that confirms the disease, the treatment and the prognosis.

Without HIV, AIDS would still exist, but no anti-retroviral drug would be used for it. Therefore HIV is there only to sustain the production and prescription of anti-retroviral drugs. A legal drug cartel.

After twenty years this marketing scheme is still in place and thriving. New markets have been opening in Africa, India, China and now, with the new Russia and the new EU members, the market of Eastern Europe is emerging.

When a new drug is licensed by the FDA and patented, the pharmaceuticals company practically acquires a monopoly over the market. So, 'anti-retroviral-drug-against-HIV-AIDS' being the main phenomenon—the real phenomenon—means that the phenomenon itself is also patented.

The way to patent a medical hypothesis is by dogma. Once it acquires the status of a dogma, the hypothesis becomes immune to any review, critique, or opposition. Nothing can harm it, because no-one can question it. That is the only way in which a flawed hypothesis can withstand the passage of time, which otherwise proves it wrong in its predictions, wrong in its therapeutic approach and success, and wrong in its prophylactic measures.

With all the available funds for research in prophylaxis, besides the ancient condom, no-one has come up with a vaccine after 20 years of constant research, whereas in a matter of months we get a flu vaccine for every new flu virus (with much greater amount of DNA) that emerges in China. Of course, the dogma will immediately say that these retroviruses are far more complicated, that they mutate, and so on. Any explanation will do, since every honest scientist is working within the enframing of the hypothesis that by definition is unquestionable.

And if anyone dares to question or shed doubt about the fundamentals of the hypothesis, they will be cast out and their scientific findings will no longer reach the pages of those prestigious scientific journals (*Lancet, Nature, Science*, etc.) which set the guidelines of the medical trade by feeding the medical practitioners with the latest output of the research community. Dissidents will also encounter the opposition of the in-the-frame health professionals, whatever their field might be. Scientists like P. Duesberg, E. Papadopulos and The Group of Perth University, Australia, along with hundreds of other clear minds, have been progressively silenced.

The dogma cannot be questioned by anyone. Not by scientist, nor indeed by Head of State.

When President Thabo Mbeki of the Republic of South Africa had the courageous will to say no to anti-retroviral treatment in order to protect his people from the toxic effects of useless and harmful drugs, and to protect the country from indirect pillage by the vast sums that are required to fulfil the anti-HIV-AIDS protocols, almost everyone rallied against him, from local opposition to the corporate world of the IMF and the World

Bank. During that time CNN was broadcasting for Africa a very friendly and educative World Bank advertisement about HIV-AIDS, informing everyone that HIV is the cause of AIDS and how to treat it. We have not been shown that advertisement for the last two years, ever since President Mbeki, out of intolerable political pressure, had to relinquish his position and accept the introduction of anti-retroviral drugs like Nevirapine, supposedly to protect his people from AIDS. Unfortunately President Mbeki had no media group on his side to support him.

In the recent South African General Elections one of the electoral slogans was the promise of 'Free AIDS Drugs'. Knowing the cost of this, in lives and in resources, the marketing scheme is in fact a cruel marketing scam.

At this point we must emphasise that there is no conspiracy sustaining all of this. Indeed, no-one could ever orchestrate it. The phenomenon happens as a spontaneous emergence of a common interest that coincides in a particular arena. The 1980-83 life-style hypothesis of AIDS was not an adequate framework for any confluence of commercial, political or personal interests. But with the 1984 launch of the viral hypothesis, the perfect arena was created for, as it were, a spontaneous collusion of interests to emerge.

HIV as the cause of AIDS makes everything simple for everyone. Simple for R. Gallo, who in 1984 claimed to have discovered HIV. The first thing he did was to patent the HIV test. Simple for the pharmaceuticals corporations, who were already analysing the business possibilities that the new HIV-AIDS hypothesis could bring by the marketing of anti-retroviral drugs, tests and related pharmaceutical products. Simple for cancer researchers on retroviruses, who faced dismal prospects for future grants in the cancer field, but who suddenly became the new HIV-experts, eager to embrace the discovery of HIV, make their mark, and grab one of the numerous grants now available for research on HIV-AIDS. Simple for politicians, waiting to get their hands on anything that can get them more votes, and 1984 was a re-election year for Ronald Reagan. The discovery of HIV was boasted by the politicians as the discovery of the century, again another proud American contribution to the advancement of science.

No-one has ever proven that HIV causes AIDS. No-one has ever produced definite scientific evidence that HIV really exists. There are no major scientific journals without corporate connections to the companies who make things for scientists to buy, and the companies who make drugs for doctors to sell. If Montagnier and Gallo's original 1983/84 scientific papers that claim to have identified HIV did not really find the evidence, why was their work published? Since then, tens of thousands of scientists and researchers have spent billions of dollars a year working on the idea contained in those papers and endorsed by prestigious medical journals.

HIV as the cause of AIDS also made things easy for those medical practitioners who like things clear and simple, and who were ready to support the reductionist new HIV-AIDS hypothesis. When HIV was announced as the cause of AIDS in 1984, the Centres for Disease Control requested that doctors should report any patients displaying certain symptoms and test them for antibodies to this organism. The CDC's report, intended for physicians, did not identify any of the original work on the basis of which this clinical protocol was being established. Physicians did not need to know the source of the information, since any physician would assume that the CDC had real evidence and proof that HIV was the cause of AIDS. The new, clear parameters to diagnose and treat what was before a puzzling syndrome made everything easier. Now AIDS is an infectious disease, diagnosed by the HIV-test and treated with anti-retrovirals.

Socially, HIV was the perfect scapegoat to disguise the evidence of the pathological consequences of homosexual practices. The American and European homosexual lobby unconditionally supported and promoted HIV, the virus that can attack 'anyone'. Suddenly everyone was at risk, exonerating them publicly from the consequences of their unnatural practice. Also, with the fantasy of a viral disease, homosexual HIV-positives were the first to rush to take AZT even before it was approved by the FDA (of course, rumours of its wonders were spread during the AZT trials for FDA approval). And we should not forget that among the other beneficiaries of the HIV-AIDS hypothesis, the latex industry is booming with the manufacture of condoms.

As one insightful scientist has stated, "HIV is an entity of convenience that meets the needs of powerful groups."

We have to realise that what is happening is in the very nature of the capitalist 'free market'. Capitalism is evolutionism at work, the survival of the greediest and most deceitful by economic selection. As the scorpion said to the frog, "I am sorry, but it is in my nature, I have to sting you!" And that is the sting of capitalism, it is in its nature, one might say a metabolic product of its organic function. As an organism, like the scorpion, it produces venom. One of the toxic compounds in the venom of capitalism is usurious money, which intoxicates every transaction happening in the world and pays for every medical research taking place on the planet.

To take this metaphor further, the toxic effect of that particular compound (paper money) is a progressive depletion of the wealth of the host, of the country of the host, of the planet of the host. So wealth suppression is the destructive mechanism by which the poison works. Like the immune-suppressive anti-retroviral drugs, it interferes with the replication of the genetics of existence in the human transaction: justice, and just transactions.

Its interference is by introducing a new artificial 'gene pattern' that blocks the replication of justice within people's transactions. The gene is the code for interest.

The toxic effect of the new protein, interest, by perpetuating unjust transactions, becomes a norm and replaces the natural knowledge of 'how to live' which, in medical terms, is called physiology, from physis: what is natural, and logos: knowledge, and replaced by one that causes corruption on the earth. Once interest becomes natural, it becomes pathological. When a human being, like any cell, stops knowing 'how to live', it dies.

Thus interest, the chain-terminator of the existential natural pattern, like the DNA chain-terminator AZT, finally destroys man by depleting him of power, of sovereignty, territory, and meaning.

It is therefore no surprise to see that when the usurious gene that holds the code for interest parasitises the market of pharmaceutical research, the result is the production of a drug that poisons by prescription. It kills you, and you pay for the cost.

The phenomenon finally reveals itself (usury), from itself (toxicity), as itself (poisonous drugs).

Categorically, we can say that any HIV-positive person, with AIDS symptoms or without, is better off without anti-retrovirals.

The most recent stage of this business so far has been to export the whole package of HIV-AIDS to Africa, India, China, and other countries where poverty is the common denominator. HIV test, T-cell count and anti-retrovirals are an export of the USA. With the test, old diseases are renamed, they now have a new cause, the new market has been created, and the drugs are ready.

* * * * *

While the clarification of the African AIDS epidemic was the original question that led to this report, scientific accuracy requires further explanation on the aetiological side.

Since we know that the HIV test is not a diagnostic tool for AIDS, its only use can be to engender the world-wide epidemic of HIV-positives, the 'HIV infected' people, recipients of anti-retroviral treatment and HIV-AIDS medical protocols. Medical data becomes political when it is used in statistical projections by the international community of financiers (IMF, World Bank, and others), forcing the African governments to accept AIDS packages.

Besides the HIV-positive estimates, what we are left with in Africa are people with diseases associated with immune deficiency.

It has been said before that tuberculosis, protein-calorie malnutrition and parasitic diseases can all be associated with the depression of cellular immunity.(59)

A wide range of protozoal and helmintic infections prevalent in Africa have been reported to induce immune deficiency.(60)

Africans, in certain areas, are endemically exposed to a wide variety of viruses, including cytomegalovirus, Epstein-Barr virus and herpes simplex virus, all of which modulate the immune system. Furthermore, other areas of Africa have a variety of endemic diseases which have a major effect on the immune system, such as malaria, trypanosomiasis and filariasis.(61)

We have described the African immune deficiency epidemic as being an epidemic of poor, underdeveloped countries, and thus implied that poverty is the major factor responsible for poor protein-calorie intake, poor water resources and sanitation, and endemic intestinal parasitosis. All of the African governments are telling their electorates that they are implementing development plans to reduce poverty.

We are forced to take our scientific inquiry further, because if we stop at poverty, we risk allowing a major flaw in our thesis by misreading the evidence.

The statement made previously that there is an 'AIDS of poor underdeveloped countries' is not phenomenologically true if we move the scope of our focus out from the micro-element representing a poor child dying of malnutrition, and into the broader view of the rich ground in which the dying child is going to be buried. He will be buried with all the other underground natural wealth that still exists beneath the African soil.

The African countries may be underdeveloped in certain respects, and for certain people (under the Apartheid regime, South Africa had developed an atomic bomb), but what they are not is poor countries. That statement would be completely unscientific.

In any given pathological process we can identify primary and secondary causes.

Poverty means a lack of wealth, but the African countries are not poor countries with poor natural resources. On the contrary, African countries have gold, diamonds, platinum, oil, and so on, quite aside from their extensive fertile lands. We cannot consider poverty to be the primary cause of the phenomenon, but rather the lack of wealth of the majority of the individuals that display the poverty-defining diseases. This is something completely different.

If the country has wealth, but a large proportion of its people suffer from poverty-defining diseases, we have to conclude that the wealth of the country does not reach the source of the condition that creates the revealing diseases. A lack of distribution of wealth could account for the impairment of economic life, and therefore the poverty-defining diseases.

In order to distribute wealth you first have to have ownership over it. Here this is the case more than ever, since the wealth is under the feet of the people, but not in their hands.

What this means is that between the extraction of the precious minerals from beneath the ground, and the hands of the person—although this is a very small distance—the wealth disappears.

Ultimately the disappearance of the wealth, by whatever means, and the unavailability of it for the majority of the individuals living in the African continent, is the *prima causa morbo*—that which causes the individual itself to reflect the consequences of wealth impairment. One of the biological markers of this pathological situation [pathos: pain] is the African immune deficiency epidemic of related diseases.

The physician's highest mission—his only mission—is to restore the sick to health, to 'cure' as it is termed. And in order to find the cure, the doctor has to know first what *needs* to be cured.

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