

AIDS: Scientific or Viral Catastrophe?

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Abstract—Scientists have not been able to ascertain how HIV causes AIDS, despite more than \$100 billion spent on AIDS research and treatment by US taxpayers alone. Predictions about the course of the epidemic have proved inaccurate. While millions are said to be infected and dying in Africa, AIDS deaths have fallen in Europe and the USA and now total fewer than 250 a year in the UK, which has a population of nearly 60 million. Claims that cocktails of antiviral drugs are responsible for a decline in Western AIDS are unsupported by clear evidence. On the contrary, the US Government has reversed a policy of “hit hard, hit early” in HIV-positive people, citing “unexpected toxicities” from the drugs. The HIV theory of AIDS causation has fulfilled certain social and public health needs, but the scientific community has not acknowledged or addressed serious flaws in AIDS theory and medical practice, in particular a failure to validate “HIV” diagnostic tests against isolation of virus. Genetic and chemical signals produced by disordered immune cells may have been misinterpreted as evidence of the presence of a lethal virus. There is vast over-diagnosis of AIDS and “HIV disease” in Africa and other countries where malnutrition and grossly impoverished living circumstances, with associated infections, are the real killers. The harmful consequences of these mistakes and omissions are increasing now that the World Health Organization and the Joint United Nations Program on HIV/AIDS (UNAIDS), convinced of an African pandemic, are urging finance ministers of African countries to devote more domestic funds to HIV/AIDS activities. On the other hand, if debt relief and other emergency aid for which UNAIDS is also campaigning are used appropriately, enormous relief of human suffering will be possible. A reasoned response from the scientific community to the full range of evidence challenging the HIV theory is overdue.

Keywords: HIV—AIDS—HIV test—poverty—Africa—virus isolation

Introduction

An African girl stands beneath a tall, makeshift wooden cross planted in a freshly dug grave. Her sad face, eyes accusingly upturned, dominates the black-and-white cover of a special issue of the *British Medical Journal (BMJ)*, “Global Voices on the AIDS Catastrophe”.¹ Inside, we read that without access to retroviral drugs, “most of the 40 million people currently living with HIV will die”; that more than 600,000 infants are infected with HIV from their mothers every year; that the epidemic will kill 55 million people by 2010; that HIV drugs should be made available free to poor countries; and that our generation will be

judged by its success or failure in developing an HIV vaccine and ensuring equitable access to it. Such declarations have become a kind of litany, recited regularly in news media as well as professional journals. The intention is to sustain awareness of the suffering HIV is held to cause and of the need to remain vigilant against its further spread, and to encourage provision of remedial help.

This article is about a world-wide body of informed opinion that dissents from the beliefs, assumptions and interpretations of evidence underlying and arising from the HIV theory of AIDS. For those who subscribe to this dissenting view, the statements in the above paragraph have a very different meaning. They signify a tragedy of errors. The “dissidents” do not dispute that suffering caused by immune deficiencies exists on a large scale in poor countries, and that the need for help is real and urgent. They have varying ideas about what actually does cause AIDS. But they are united in questioning the bleak picture painted by mainstream AIDS scientists, considering it an unfounded assault on the minds and hearts of millions. The dissidents are also agreed in challenging the belief that AIDS is caused by a single virus, in opposing use of the HIV test to diagnose “HIV disease”, and in arguing that there are more appropriate and compassionate ways to counter AIDS than use of antiviral drugs and the search for a vaccine.

To many scientists, especially those steeped in AIDS work, it is no longer a theory but a fact that HIV is the cause of AIDS. My own view, after studying the issue now for more than 10 years, is that this is not because of an overwhelming weight of evidence in favour of the HIV hypothesis, as is often believed and claimed. On the contrary, there is powerful evidence that the science underlying the oft-quoted statistics and the paradigm that gives rise to them has missed the mark in several crucial aspects. There is even a strong question mark over the very existence of the virus as a unique infectious entity. The signals that have been interpreted as indicating its presence may instead arise as a result of heightened cell decay in a compromised immune system. Most people do not know about this evidence, because HIV became an article of faith for modern medicine almost as soon as the theory was proposed, and questioning it a heresy. Feelings around AIDS ran so high, and the drive to promote the idea that all were at risk was so strong, that contrary views were marginalized and suppressed from the beginning and remain for the most part unheard. Dissidents who challenge the theory have often been ridiculed as “flat-earthers” by colleagues privileged to enjoy the mainstream of AIDS beliefs. The result has been a persistent failure to acknowledge or explore shortcomings in the science surrounding the virus explanation.

When AIDS was first medically recognised in the early 1980s, the drive to defeat it brought out qualities and aspirations among many of those involved that went beyond the call of professional duty. These efforts have brought profound social and political benefits. Sympathy for homosexual men, hardest hit by Western AIDS, grew steadily and the social status of the gay community has been transformed. In more recent years, awareness of the millions who die prematurely in Africa has increased the sense that AIDS is one of the most

urgent challenges facing humanity and has triggered a substantial response in human and financial resources. In the USA alone, where taxpayers have spent more than \$100 billion on HIV/AIDS research, treatment, and other programs over the past two decades,² the Bush administration budgeted \$780 million in 2002 to help foreign nations grapple with the disease. To the surprise of the *New York Times*, both Republicans and Democrats pressed for more. “With Convert’s Zeal, Congress Awakens to AIDS” was the headline on a *Times* report that the eventual US contribution to the global fight would probably approach \$1.3 billion. The recently-formed Global Fund to Fight AIDS, Tuberculosis and Malaria quickly obtained pledges of more than \$2 billion, with \$700 million available for immediate disbursement (though the *BMJ* argued that these figures are still hugely disproportionate to what is needed). The World Bank, which has earmarked more than \$2 billion for HIV/AIDS since 1986, including loans, is also intensifying its efforts.

Paradoxically, however, the scale of these endeavours, along with the HIV theory’s value as a catalyst for aid, has made it increasingly difficult for dissenting voices to be heard. Most people, now firmly believing that the world faces an “AIDS catastrophe”, respond to escalating claims about the extent of the epidemic not just with concern, but with gratitude that despite the immensity of the problem, science, medicine and politics have the virus in their sights and that huge resources have been mobilised in support of their efforts. It then seems churlish, irresponsible and even dangerous for anyone to write or say anything that could be perceived as weakening resolve to fight the spread of HIV.

Scientists and non-scientists alike question the hypothesis at their peril. President Thabo Mbeki of South Africa is still struggling to cope with the political fall-out over his suggestion that poverty, not HIV, is responsible for much of African AIDS. When Mbeki questioned the value of antiviral drugs in preventing mother-to-child transmission of AIDS, he was portrayed by the UK media as a monster (e.g. “Mbeki ‘lets Aids babies die in pain’ ”, *The Observer*, 20 August 2000; Mbeki “Enemy of the people”, *Sunday Times*, 27 August 2000). Across the world, newspapers and broadcast media, doctors and scientists, charities, UN agencies, financial institutions and politicians, even up to the level of the White House, joined in the criticism. “Under pressure to spend millions to prescribe AZT, President Mbeki indulges AIDS flat-earthers”, said *Time* magazine in April 2000, in response to news that Mbeki was defending his right to include about a dozen “dissident” scientists on a 40-strong advisory panel on AIDS. The disease was threatening to wipe out a quarter of South Africa’s population by the year 2010, said *Time*’s medical correspondent, yet the government was backing away from its treatment responsibilities by refusing to make available the antiviral drugs AZT or nevirapine to rape victims and pregnant women. Hundreds of thousands, if not millions, of people would suffer because of Mbeki’s “misplaced distrust of medical authority”. The latest (August 2002) attack on Mbeki is a CD remake of famous songs of the anti-

apartheid era in which he and his health minister Manto Tshabalala-Msimang are portrayed as the new oppressors.

This state of affairs is indeed dangerous, but not because Mbeki is wrong. For if one thing is certain it is that both AZT and nevirapine are very dangerous drugs, and that neither of them has been demonstrated to benefit babies. As we shall see, the benefit is entirely speculative, based on an effect on certain surrogate markers believed to indicate HIV infection. Studies in terms of actual outcome on the babies' health show that those exposed to the drugs do worse than those who remain drug-free. This is contrary to expectations based on AIDS orthodoxy and may prove to be the spur for a long-overdue re-examination of many of the claims of AIDS experts.

How the Theory Took Hold

Deep social, psychological and political currents were involved in the construction and almost immediate acceptance of the HIV hypothesis, but a convenient place to begin the story is April 23, 1984. That was the day when Margaret Heckler, the then US Health Secretary, announced at a press conference that the "probable" cause of AIDS had been found. It was a virus, later to become known as the Human Immunodeficiency Virus. A process had been developed to mass-produce this virus, Heckler said, resulting in a "blood test for AIDS which we hope can be widely available within about six months . . . we have applied for the patent on this process today".

Robert Gallo, the US Government researcher who led the team responsible for the apparent breakthrough, confirmed at the press conference that in his mind the cause of AIDS was unequivocally a new retrovirus,³ that it was probably the same as one found by Luc Montagnier's group at the Pasteur Institute in Paris, and that a reliable blood test "that could quickly save lives" had been developed.⁴ The blood test had been made possible because "we have the problem of mass production solved", Gallo told reporters. "That's one of the significances of what we're telling you today".⁵

In staking his claim to have been the first to truly characterise "the AIDS virus", Gallo had previously sought to play down the significance of the Pasteur group's work. "No one has been able to work with their particles", he wrote to the editor of *The Lancet* earlier that year. "Because of the lack of permanent production and characterisation it is hard to say they are really 'isolated' in the sense that virologists use this term".⁶ Gallo's "initial disbelief of Montagnier's claim to have isolated a virus from AIDS patients, which he has since acknowledged to have been unfortunate", as *Nature* put it,⁷ included doubts over electron micrographs published by the French. Gallo also originally dismissed as "ridiculous" the French team's claims that they had identified a retrovirus specific to AIDS on the grounds that their culture reacted with antibodies in blood samples from AIDS patients. "That's bad virology", Gallo had said.

“Patient sera, especially in AIDS patients, has antibodies to a lot of different things”.⁸

Gallo’s scepticism gave way to a different attitude after his own earlier candidate as the AIDS virus, HTLV-1, failed to convince, not least because it had been linked with uncontrolled white blood cell growth, rather than the loss of cells seen in AIDS. The April 1984 press conference concerned his second candidate, a retrovirus purportedly related to HTLV-1, that he called HTLV-3. The following month, Gallo’s group published four articles in *Science* in which he sought to demonstrate that HTLV-3 was the primary cause of AIDS.⁹

These *Science* papers, along with Montagnier’s claims, soon became the almost unchallenged basis of the scientific community’s belief in the theory that AIDS was indeed caused by a new virus. Between 1984 and 1987 it was accepted that between them, Gallo and Montagnier had succeeded in isolating the virus and producing a diagnostic test to detect its presence in patients and in blood supplies. Screening surveys using these new tests gave rise to the idea that HIV was spreading rapidly via sexual intercourse, mother-to-baby transmission, blood transfusions, and needles shared by drug addicts. The antiviral drug AZT soon followed, initially developed and promoted by US Government scientists, although with a drug company, Burroughs Wellcome (now subsumed in the giant GlaxoSmithKline group) reaping most of the rewards. The world was assured that a vaccine would not be far behind.

Three core propositions soon became established as a firm belief system, essentially unchanged to this day. These hold that:

1. HIV is a lethal viral infection, probably originating in Africa, that gradually and inexorably destroys cells of the immune system, so that the victim eventually dies from an inability to resist a variety of previously known disease conditions.
2. The virus’s presence can be reliably detected with the HIV test.
3. AZT and similar drugs can save lives by quelling the virus, blocking its growth and transmission. Consequently, the best way to fight the epidemic is with antiviral drugs and the hunt for a vaccine, alongside prevention work including condom distribution and discouragement of breast-feeding by HIV-positive mothers.

The world was ready to hear this story. It was as if a huge, collective sigh of relief went up, that the complex and frightening collapse of the immune system seen in AIDS could be attributed to a single microbe. Leaders of the gay community were particularly relieved. They had fought for years through the Gay Liberation movement for more humane attitudes towards homosexuality. Those advances had come under threat during the first years of AIDS, when the “gay plague” stigma had been used by a right-wing administration as an excuse for inaction. Doctors and scientists who had seen the devastation the new illness was causing to young lives were also relieved. A deadly new virus meant an enemy that could be fought cleanly, without prejudice, using scientific tools that

doctors and scientists were familiar with. The media, too, love killer virus stories. As the idea developed that the virus itself was not prejudiced either, and would in time prove a threat to just about everyone, big money started to roll for AIDS research and treatment.

These and other social, political and even religious factors gathered behind the HIV hypothesis and swiftly turned it, in most people's minds, into a creed. Gay men who suggested there could be a link between AIDS and the drug-driven, multiple-partner promiscuity of the early Gay Lib years—not with feelings of blame or guilt, but rather, of trying to understand and prevent the disease—were quickly denied a voice. One of these, the late Michael Callen, whose “conservative” estimate was that he had had sex with more than 3,000 partners by the age of 27, once commented: “HIV breeds a form of scientific nationalism: you're either for it or against it. And like America, one must apparently love it or leave the AIDS debate”.¹⁰

Not long after the launch of Gallo's virus as the cause of AIDS, a fierce scientific dispute arose surrounding it which, paradoxically, also had the effect of fixing the viral theory all the more securely in most people's minds. HTLV-3 was found to be identical to the particles obtained by the Pasteur team, which they had named LAV; and a sample of LAV had been sent to Gallo's laboratory. Had there been a laboratory mix-up? Did Gallo “steal” the French group's virus? A prolonged and bitter argument began over who should be credited with its discovery.

Gallo claimed that even if it was the same virus, his team had made a significant advance on the French work by getting it to grow (in a highly abnormal, leukemic cell line) in sufficient quantity to do the laboratory work from which the first antibody test kits could be manufactured.

Years later, an investigation by the US National Institutes of Health Office of Scientific Integrity led to a report listing 20 instances of “knowledgeable misreporting or errors” in the first and main *Science* paper. Eight of these errors, the report said, were serious enough to constitute scientific misconduct.¹¹ Gallo, whilst maintaining innocence of deliberate misconduct, has acknowledged that the four papers were written during what he called the “passionate” stage of his group's work, when they were under a variety of pressures to publish quickly, including political pressure from Heckler's department.¹²

Robin Weiss, the leading British AIDS scientist, also initially discounted the French group's claims and rejected a key Montagnier paper in 1983.¹³ In 1985 Weiss also independently claimed isolation of an AIDS virus, from which he patented the British blood test, after Montagnier had sent him, too, samples of “LAV”. An investigation revealed in early 1991 that his virus also appeared to be identical to the French virus, and Weiss publicly agreed that he might have accidentally contaminated his cultures with LAV.¹⁴

With Gallo and Montagnier fighting each other from the start over who should receive the credit for discovering the virus, the possibility that *neither* might have done so was overlooked by the world scientific community. Acceptance of

the term “Human Immunodeficiency Virus” as a supposed compromise between HTLV-3 and LAV set in stone the assumption that a new virus was the cause of AIDS. Yet in retrospect, it was certainly remarkable that not just Montagnier and Gallo but Weiss too, the three prime movers of the HIV story, all seemed to have based their claims on work with identical particles from a single source.

Anger and Disbelief Greet the Early Challenges

During the second half of the 1980s, while working as medical correspondent of the London *Sunday Times*, I shared and reported on the rapidly-established belief that HIV was a contagious, sexually transmitted microbe, silently imperilling the world because of a time lag of years between infection and immune system breakdown. There was a contagious element to this belief itself, to which I remember being first fully exposed at the international AIDS congress in Washington in 1987. A lot of emotion was present. There was anger, as gay men, already stricken by terrible losses, lobbied for faster release of anti-HIV drugs; but there was also a shared sense of excitement, as speaker after speaker emphasised the peril that HIV presented, while also offering the assurance that science and medicine were mobilising against this microbe and that given the right social and financial support, would sooner or later defeat it.

After living and working with this idea over the next few years, I was incredulous when in June 1990 a British television documentary questioned this belief. Made by Meditel, a film-making company in London, and transmitted as part of Channel 4’s *Dispatches* series, it highlighted a challenge to the HIV theory by Professor Peter Duesberg, a US molecular biologist. Previously considered at the forefront of his profession, Duesberg had become ostracised after arguing that HIV was a harmless bystander in AIDS.¹⁵ The real causes, he believed, were drug abuse, heavy exposure to blood and blood products, and, as panic over HIV took hold, toxic medical treatments directed against the virus.¹⁶

The main plank in Duesberg’s argument against HIV was (and is) that there is so little active virus in patients, even those with full-blown AIDS, that it cannot be doing the damage attributed to it. At one time it was thought AIDS resulted from the virus running over the immune system “like a truck” (in Gallo’s words), destroying a particular class of cell, known for short as T4 cells, crucial in coordinating the body’s response against infections. That theory has not stood up. According to a recent review in *Nature*, “much remains left to the imagination” as to how HIV causes immune deficiency.¹⁷ After nearly two decades of work, AIDS scientists still do not know how or why HIV is pathogenic. This fact in itself lends strong support to Duesberg’s position.

About 18 months after the Meditel film was shown, I met its director, Joan Shenton, who urged me to look more deeply into Duesberg’s critique. By this time, he had the backing of about 40 scientists and other AIDS analysts, called the Group for the Reappraisal of the HIV/AIDS Hypothesis¹⁸ (the group’s membership later ran into the hundreds). In May 1992 an “alternative” AIDS

conference featuring Duesberg and other “AIDS dissidents” took place in Amsterdam, Holland, providing me with an opportunity to describe their arguments for the first time to a national newspaper audience anywhere in the world.¹⁹

The article brought a furious response from AIDS scientists, who said it would endanger lives by weakening the public health response to the epidemic. Robin Weiss invited me to his laboratory to see the “harmless” virus I had written about. He never actually showed it to me, but berated me for two hours over my work.

Further anger greeted a *Sunday Times* article²⁰ heralding the appearance on Channel 4 in March 1993 of another Meditel documentary, this one challenging the idea that Africa was in the grip of an AIDS epidemic. Under the headline “Epidemic of AIDS in Africa ‘a tragic myth’”, I wrote that the film would outrage much Western medical opinion, because of the belief that “heterosexual AIDS” in Africa was a warning of what could happen elsewhere. Nevertheless, a growing body of expert opinion believed that false claims of devastation by HIV were leading to a tragic diversion of resources from areas of genuine medical need such as malaria, tuberculosis and malnutrition. Some of the “heretics” were even saying there was no evidence of a new sexually transmitted disease in Africa, but that instead, death rates had increased in some countries because of civil war, and because of poverty and malnutrition linked to economic decline. Predictions by the World Health Organisation (WHO) and other agencies that millions would die because of HIV were based not on scientific evidence, but on unfounded assumptions about the extent of HIV infection in Africa and its links with AIDS.

The documentary was based on a two-month investigation in Uganda and the Ivory Coast, thought to be epicentres of what agencies were calling a “pandemic” of AIDS. It argued that because international funds were available for AIDS and HIV work, politicians and health workers had an incentive to classify people as AIDS sufferers who previously would have been diagnosed as having other illnesses. The Ugandan government could afford to spend less than \$1 a head on health care from its funds, but the previous year it received \$6 million for AIDS research and prevention from foreign agencies.

Part of the problem was that HIV testing was frequently misleading in Africa, as the tests reacted to antibodies to other diseases, producing high rates of false positives. Furthermore, most AIDS diagnoses in Africa did not involve an HIV test, but were based on a WHO definition that relied on clinical signs including weight loss, chronic diarrhoea and prolonged fever. The scope for misclassification was enormous. According to Dr. Harvey Bialy, an American scientist who worked as a tropical disease expert in Africa for many years and who accompanied the television crew, there was “absolutely no believable, persuasive evidence that Africa is in the midst of a new epidemic of infectious immunodeficiency”.

Bialy, whom I interviewed for the article, told me that the only “utterly new”

phenomenon he had seen was in drug-abusing prostitutes in Abidjan in the Ivory Coast. The girls were being destroyed by viciously adulterated smokable heroin and cocaine. Otherwise, he had seen malaria, tuberculosis, and diarrhoeal diseases, which arguably had become more severe, but reason told him that this was because of general economic decline, a decline in health care, and the development of drug-resistant strains. Those factors, he felt, could explain what was going on much more efficiently and persuasively, and to much greater good for the public health, than saying the diseases were being made worse by HIV.

HIV Test Never Validated Against Isolation of Virus

Bialy was working as scientific editor for *Bio/Technology* magazine, which includes among its specialities the detailed examination of diagnostic tests. He had in press a paper on HIV that did more than highlight a problem with false positives: it challenged the very basis of the test as indicating the presence of a specific virus, HIV, arguing that it had never been validated against the accepted gold standard for such a test, isolation of the virus itself. The article concluded that positive test results, whether using the Elisa or Western blot testing methods, might represent nothing more than cross-reactivity with non-HIV antibodies present in AIDS patients and those at risk, and that use of the test as a diagnostic and epidemiological tool for HIV infection should be reappraised.

Published in June 1993,²¹ the review article, which carried 161 references, showed that the data presented by Gallo and Montagnier did not prove that a retrovirus had been isolated from the tissues of AIDS patients.

Traditionally, in determining whether a virus is the specific cause of an illness, microbiologists first purify it from a patient with the disease so that they know what it looks like under the electron microscope and precisely what they are working with. They then grow the purified virus in the laboratory; show that it is present in all cases of the disease, that there is a lot of it, and that it is active in the body in a way that accounts for the disease; and demonstrate that it reproduces the original disease when introduced into a susceptible animal.

In the case of "HIV", none of these requirements has been met, according to Eleni Papadopulos-Eleopulos, a medical physicist and cell biology expert at the Royal Perth Hospital, Western Australia, and the main author of the *Bio/Technology* paper. She and consultant physician Val Turner, her prime collaborator in what has come to be known as the Perth group of AIDS scientists, have been working tirelessly for nearly 20 years to demonstrate their conviction that HIV has not even been proved to exist.

They acknowledge that particles presumed to be the virus can appear after intensive co-culturing procedures, using abnormal (leukemic and fetal cord) cell lines, but those particles might be endogenous products of the stimulated cells. Furthermore, it has never proved possible to obtain a concentration of HIV particles, through centrifugation, at the sucrose density gradient considered

characteristic for retroviruses, 1.16 gm/ml.²² Thus, HIV has never been properly isolated, in the sense of being separated from other constituents of disrupted cells, including nucleic acids, and characterised as a unique set of retrovirus particles. Because of this, it has also proved impossible to photograph purified virus with the electron microscope. Claims of “virus isolation” in the AIDS literature usually refer to a variety of indirect signals presumed to indicate HIV activity, but such presumptions may be false; the signals have not been proved to relate to a specific, invasive virus.

This interpretation is strongly supported by another remarkable fact about “HIV”: no two of its genomes are the same, even from the same person, a phenomenon that has caused some commentators to consider it a “quasispecies” of virus.²³ In any one patient, there are more than 100 million genetically distinct variants, according to one estimate.²⁴ The variations led another researcher to conclude, “The data imply that there is no such thing as an [AIDS virus] isolate”.²⁵ Howard Temin, who shared the 1975 Nobel Prize for Medicine for his discovery of an enzyme characteristic of retroviruses, makes a similar point in a chapter contributed to *Emerging Viruses* (ed. Stephen Morse, Oxford University Press, 1993, p. 221): “The data indicate that in any one AIDS patient, at any one time, there are many different virus genomes”. These observations do not support the concept of a unique, invasive viral entity. They are more consistent with the idea that we are looking at chaotic genetic activity from within disordered cells.

The genetic material that Gallo, Montagnier and Weiss obtained from their cell cultures—all probably from the same source, as it turned out—and now called the HIV genome has never been purified directly from patient tissues and properly characterised.²⁶ Particles containing active genetic material are released after some weeks of the laborious co-culturing procedures, and this material can be passed from one cell to another and its genetic composition determined. But it has never been shown to have the properties of a unique, self-replicating, disease-inducing virus.

None of 150 chimpanzees inoculated with “HIV” has developed AIDS. According to HIV theory, the “virus” crossed into humans from chimpanzees and sooty mangabeys; but these animals do not get AIDS naturally, despite carrying “essentially the same virus”.²⁷ In an attempt to explain these findings, Dutch researchers, working with University of California statisticians, recently postulated that an AIDS-like epidemic wiped out huge numbers of chimpanzees two million years ago, leaving modern chimps—who share more than 98% of their DNA with humans—largely resistant to HIV.²⁸ Such theorising is seen by the “dissidents” as indicating the desperate lengths to which HIV protagonists will go to defend the virus construct.

The Perth group maintain that the failure to purify meant none of the originators of the HIV hypothesis knew what they were working with, and that this problem continues to this day. They have shown that the antibodies the HIV test detects can all be put into the circulation because of a variety of other, non-

HIV challenges to the immune system. This is a particularly significant addition to the Duesberg critique, because it offers a non-HIV explanation for the close correlation between raised levels of “HIV” antibodies and risk of illness—a correlation that has been the main plank in the case that HIV causes AIDS.

Furthermore, the Perth group accept that some of these non-HIV immune challenges are transmissible through blood and other body fluid abnormalities, and that the “HIV” blood test screens for these abnormalities. “From the public health point of view we are in total agreement with HIV experts”, Eleopulos says. “If anything, we would go further. Certainly it is good to test all blood, not only blood from risk groups, because the test shows when blood is abnormal and should not be given. We also advocate safe sex, especially in passive anal intercourse, irrespective of whether the active partner is or is not HIV-positive, though there is even more risk if they are positive. Semen itself is oxidising, and if it comes from a person who is diseased it can be even more toxic. Clean needles are obviously better than dirty needles for drug users but we also say no needles at all, because the contents of the syringe cause the problem too”.²⁹

It is the use of the test to diagnose “HIV disease” with which the Perth group take issue. There are two main categories of the test, using methods known as Western blot (WB) and Elisa. The WB is held to be the more specific, because it detects activity by individual protein antibodies rather than looking for their presence as a group, as with the Elisa. However, the *Bio/Technology* paper showed that none of the proteins used in the WB test had been demonstrated to be specific to a unique retrovirus. There were other potential sources for all of them. It also cited studies showing false-positive results with the “HIV” test in people with many different sources of immune system activation, including tuberculosis and malaria.

Patients with AIDS, and promiscuous homosexual men or drug addicts leading lives likely to expose them to multiple immunological challenges, were certainly much more likely to test positive than healthy Americans, a finding that was used as the basis for claiming the test did have diagnostic validity. But another reason for this association could be that antibodies looked for by the test were to normal cellular proteins such as actin, released under conditions of immune system stress.

Other studies have confirmed that the “HIV” test does indeed detect such antibodies. Patients with the autoimmune condition lupus erythematosus, for example, test positive for “HIV” because they have antibodies with anti-actin activity.³⁰ Chronically recurrent disease due to hepatitis viruses also often causes autoimmune reactions, in which antibodies to actin and other cell proteins predominate. Hepatitis viruses are extremely common in the main AIDS risk groups (with hepatitis C almost universal in them), and this has led researchers to suggest that the autoantibodies frequently seen in patients with hepatitis could be responsible for positive “HIV” test results.³¹

The *Bio/Technology* paper demonstrated that as well as being non-specific, the various “HIV” tests were non-standardised. When stringent criteria for

a positive result were imposed by the US Food and Drug Administration (FDA) in 1987, for example, it was found that fewer than 50% of AIDS patients tested positive. That compared with 80% according to criteria required by the Consortium for Retrovirus Serology Standardisation.

Dr. Roberto Giraldo, an infectious diseases specialist working at a laboratory of clinical immunology in New York City, has expressed surprise at finding that to run the Elisa test, an individual's serum has to be diluted to a ratio of 1:400 with a special specimen diluent. He says this dilution ratio is at least 20 times greater than those used in most other serologic tests that look for the presence of microbial antibodies, suggesting that normal blood samples contain a lot of material reactive with the "HIV" test.³² Other reviews of the scientific literature have documented as many as 70 different reasons for getting a positive reaction unrelated to HIV infection.³³ These conditions, Giraldo says, have in common a history of polyantigenic stimulation, evidence that leads him to suggest that a reactive Elisa test at any serum concentration means no more than the presence of nonspecific or polyspecific antibodies, which could be present in all blood samples, but at different levels. "They are most likely a result of the stress response, having no relation to any retrovirus, let alone HIV. . . a reactive test could be a measure of the degree of one's exposure to stressor or oxidising agents".

Abbott Laboratories, one of the main producers of Elisa "HIV" kits, is well aware of the specificity problems with the test, Giraldo adds. The company's literature states that there is no recognised standard for establishing the presence and absence of HIV antibody in blood, and therefore Elisa testing alone cannot be used to diagnose AIDS.

Regulatory authorities have known about these problems from the beginning but like Pontius Pilate, they washed their hands of the problem. As far back as 1986, an FDA official told participants at a WHO meeting that the primary use of the test was for screening blood donations, and that "it is inappropriate to use this test as a screen for AIDS or as a screen for members of groups at increased risk for AIDS in the general population". He added, however, that enforcing this intention "would be analogous to enforcing the Volstead Act, which prohibited alcoholic beverages sales in the United States in the 1920s—simply not practical".³⁴

In correspondence, Robin Weiss has told me that there were early problems of cross-reactivity with the test, but that these were overcome in later versions. He has presented no evidence for that claim. In contrast, Eleopulos *et al* say the test is intrinsically defective as a diagnostic tool, because of the inability to validate it by showing the unequivocal presence of the virus in any patients.

Instead, the test kits are calibrated—with the enormous dilution factors—to ensure that most healthy people test negative, whereas many AIDS patients, and people at risk for AIDS, test positive. Giraldo drives this point home by quoting the Abbott Laboratories' literature (emphasis is Giraldo's):³⁵

The Abbott studies show that: Sensitivity based on an *assumed* 100% prevalence of HIV-1 antibody in AIDS patients is *estimated* to be 100% (144 patients tested).

Specificity based on an *assumed* zero prevalence of HIV-1 in random donors is *estimated* to be 99.9% (477 random donors tested).

At present *there is no recognised standard* for establishing the presence and absence of HIV-1 antibody in human blood. Therefore sensitivity was computed based on the clinical diagnosis of AIDS and specificity based on random donors.

There is much evidence that the tests are as beset with problems today as ever.³⁶ In the USA, an “HIV” diagnosis will not be given on the basis of the Elisa test alone; “confirmation” with WB is required. In the UK, by contrast, diagnosis relies on repeat tests with various types of Elisa. The WB test is regarded by British experts as too unreliable to be used other than as a research tool. This is a tragic state of affairs, considering the life-and-death consequences of a positive test result.

Use of recombinant and peptide antigens has overcome an earlier problem with the Elisa of not knowing precisely what antigens are present in it, but it is not much use knowing what has gone into the test kits if you still do not know whether or not those antigens are specific to a new virus. This criticism applies as much to the WB as to Elisa. If Elisa and WB are not sufficient for “HIV” diagnosis, then what is? According to the Perth group—nothing. Eleopulos says: “We have to question all types of the antibody test, especially in AIDS patients, who have all types of infectious agents in them. . . . If the test is no good, you can repeat it a thousand times and it still won’t be any good. When the principle of the test, the basis of it, has not been established, it doesn’t matter how many times you repeat it, you still won’t prove anything”.

The same criticism applies to so-called viral loads, in which small genetic segments attributed to HIV are amplified millions of times using the polymerase chain reaction (PCR) technique in order to reach detectable levels. These tests have found an extensive market in supposedly monitoring “HIV disease”. Like the antibody test, they probably do indicate immune system disturbance, but the segments of genetic material these tests detect have not been shown to be specific to HIV. Kary Mullis, who won the Nobel Prize for Chemistry in 1993 for inventing PCR, says inappropriate conclusions are being drawn from PCR’s use in these tests. In a foreword to Peter Duesberg’s 1996 book *Inventing the AIDS Virus* (Regnery Publishing, Washington, DC), Mullis goes further, writing that he does not think Duesberg “knows necessarily what causes AIDS; we have disagreements about that. But we’re both certain about what *doesn’t* cause AIDS. We have not been able to discover any good reasons why most of the people on earth believe that AIDS is a disease caused by a virus called HIV. There is simply no scientific evidence demonstrating that this is true”.

The root of the problem with testing “viral load” is the same as with the antibodies: the research community’s inability to purify and unequivocally demonstrate the existence of HIV directly from patients. Thus, when experts claim to see a rise in drug-resistant strains of HIV, what they are actually reporting is a decrease in the ability of the drugs to suppress production of certain genetic segments believed to belong to HIV, but never proved to be such.

The resistance is not necessarily microbial at all. It may be an immune cell response to the drugs, and the heightened genetic activity a consequence of immune disorder rather than a cause.³⁷ Similarly, claims that different subtypes or “clades” of HIV have been identified across the world are not based on isolation of virus. They are based on analysis of segments of HIV’s purported genome. The segments usually looked at are the so-called viral envelope sequences, but we do not know that these sequences belong to a virus. The broad differences between them may simply reflect genetic variability of different population groups.

“They have not proven that they have actually detected a unique, exogenous retrovirus”, says Perth group member John Papadimitriou, of the University of Western Australia, a professor of pathology renowned for his work on electron microscopy. “The critical data to support that idea have not been presented. You have to be absolutely certain that what you have detected is unique and exogenous, and a single molecular species. They haven’t got conclusively to that first step. Just to see particles in the tissues, and fail to look for evidence that it is an infective virus, is wrong. Are these particles that cause disease? The proper controls have never been done”.³⁸ Val Turner goes even further. “HIV is a metaphor for a lot of quasi-related phenomena”, he says. “No one has ever proved its existence as a virus. We don’t believe it exists”.³⁹

A similar view is offered by another experienced pathologist, Etienne de Harven, emeritus professor of the University of Toronto. De Harven worked for 25 years at the Sloan-Kettering Institute in New York, where he pioneered a method of purifying viruses. In 1960 he coined the now familiar word “budding” to describe steps of virus assembly on cell surfaces. “I am very familiar with the many reports and electron microscope pictures of ‘HIV particles’”, he says. “Indeed, they show particles which could very well be taken as retroviruses on the basis of their ultrastructure alone”.⁴⁰ But all those particles had been found in complex cell cultures, the result of intensive laboratory stimulation. Recent attempts to purify and demonstrate the presence of such particles directly from the serum of AIDS patients—with studies that “should have been done years ago”—produced results disastrous for the HIV theory, de Harven says, suggesting “billions of research dollars gone up in smoke”.⁴¹

A further demonstration of the non-specificity of phenomena interpreted as indicating the presence of HIV surrounds a finding of “virus-like” particles in the lymph nodes of AIDS patients with lymph node enlargement.⁴² Such particles have often been assumed to be HIV. However, a control study using electron microscopy—the only one in which suitable comparisons and procedures were used, according to the Perth group—showed particles that looked just the same in non-AIDS patients who had swollen lymph glands for other reasons, leading the authors to conclude that “such particles do not, by themselves, indicate infection with HIV”.⁴³

The Perth scientists declare that whatever the condition, AIDS or otherwise, a positive test result does not indicate HIV infection but is a nonspecific marker

for a variety of conditions. "Consequently the general belief that almost all individuals, healthy or otherwise, who are HIV antibody positive are infected with a lethal retrovirus, has not been scientifically substantiated".⁴⁴

Why "HIV" Positivity is Correlated With Risk of Illness

The Perth group believes that in Western AIDS, the close correlation seen between testing positive and risk of illness arises because of heavy burdens on the immune system present in all the main risk groups, with oxidative stress on the immune cells the common mechanism of disease. A similar interpretation is offered by a Swiss-based organisation, the Study Group for AIDS Therapy, which draws particularly on the work of two German scientists, Heinrich Kremer, a physician and clinical researcher, and Stefan Lanka, a virologist.⁴⁵

The Gay Liberation years of the 1970s brought unprecedented opportunities for men to have sex with one another, and all the early gay victims of AIDS were leading the fast-track sex-and-drugs lifestyle. Exposure to sperm and seminal fluid from many different partners, as well as repeated bouts of sexually transmitted diseases,⁴⁶ chronic use of antibiotics,⁴⁷ and the debilitating effects of heavy exposure to recreational drugs^{48,49} may have combined to put such men at risk.

Drug addicts, another group at risk of AIDS, suffer immune deficiencies because of directly damaging effects of opiates on T-cells, for which they have an enormous affinity, as well as because of malnutrition and infections caused by sharing needles. This group's risk of developing AIDS is much higher when they continue to inject drugs than it is when they stop.⁵⁰

People with the blood-clotting disorder hemophilia, also at risk, were known to suffer immune disorders, signalled by a decline in their blood T4 cell count, resulting directly from their treatment. During the 1970s and 1980s, such treatment involved repeated intravenous infusion of concentrates made from the blood of thousands of people. It was estimated that a typical patient receiving 40 to 60 treatments a year could be exposed to blood from up to two million donors.⁵¹ The greater the amount of clotting factor they received, and the longer they received it, the greater their risk of immune deficiency.

In the late 1980s, when HIV-positive hemophiliacs were switched to an extremely pure version of the clotting factor (made using genetic engineering techniques), their T4 cell counts ceased to decline and in some instances did a U-turn.⁵² All too conveniently, a 1995 British study showing a big increase in death rates in HIV-positive hemophiliacs as compared with those who remained HIV-negative, only covered deaths to 1991, stopping short of the point (1992) where use of pure Factor VIII became widespread.⁵³ The study was hailed as proving the validity of the theory that HIV causes AIDS.⁵⁴ It did no such thing. It gave no evidence that the increased deaths were from AIDS, merely describing a proportion of them as from "AIDS, HIV etc.", which as Eleopulos pointed out⁵⁵ was meaningless. It also took no account of the fact that patients

diagnosed as HIV-positive were in most cases receiving high doses of the toxic antiviral drug AZT. In addition, several previous studies had shown that the patients who became “HIV-positive” were older and had received Factor VIII for longer and in larger doses than those who did not.

Another contribution to the increased death rate may have been the terrifying and debilitating “HIV” diagnosis itself. The contribution of mental and emotional stress to the physiological phenomena surrounding AIDS was demonstrated recently with a finding that intensive grief therapy significantly reduces “HIV viral load”, as well as fostering the maintenance of a healthy immune cell profile, in gay men who have lost a partner or close friend to AIDS.⁵⁶

Duesberg has argued that blood transfusion recipients were also a very high-risk group and did not need HIV to become sick. In one US study, about half the recipients of non-infected blood transfusions died within one year after receiving the transfusion.⁵⁷

The biggest confusion of all has arisen in Africa. When the “HIV test” was first marketed in the mid-1980s, Western scientists looking for an origin for the virus went to several central African countries with their diagnostic kits and found high percentages of people testing positive—more than 50% in some areas. As the Meditel documentary found, and as I later also reported after a six-week investigation in Africa for *The Sunday Times*, this created a climate of doom about HIV/AIDS, in which those suffering from traditional diseases of poverty and malnutrition, including tuberculosis, pneumonia, chronic intestinal infections, and malaria, were liable to be diagnosed as AIDS patients by virtue of their HIV antibody status.

Convinced that a terrible epidemic was unfolding, the WHO added to the confusion by allowing doctors to diagnose AIDS in Africa even without the use of the HIV test, on the basis of a combination of persistent symptoms such as fever, cough, diarrhea, or weight loss—the so-called Bangui clinical case definition. “Dressed up as HIV/AIDS, a variety of old sicknesses have been reclassified”, says Charles Gesheker, professor of African history at California State University, Chico. After a recent trip to Africa—his fifteenth—Gesheker concluded that it was impossible to distinguish these common symptoms from those of malaria, tuberculosis, or other indigenous diseases of impoverished lands. He adds that it is “well understood that many endemic infections will trigger the same antibodies that cause positive reactions on the HIV antibody tests. . . . The problem is that dysentery and malaria do not inspire headlines or fatten public health budgets. Infectious ‘plagues’ do”.⁵⁸

Millions Wrongly Diagnosed as Victims of “HIV” Disease

There is strong evidence that the nonspecific nature of the HIV test is causing millions to be wrongly diagnosed as victims of “HIV disease”. Sufferers and carriers of the microbes responsible for leprosy and tuberculosis are particularly

at risk. A 1994 study from Zaire,⁵⁹ in which 65% of leprosy patients and 23% of their contacts tested positive with Elisa, and even higher percentages were reactive with WB analysis, concluded after more detailed testing that in all but two of the patients, antibodies induced by *Mycobacterium leprae* were causing misleading results (on the basis of Eleopulos's work, those two could not be said to be HIV-infected either). Cross-reactivity occurred with all the supposed "HIV" antibodies. *M. leprae* might have this potential "since the disease it causes is associated with an immunodeficiency that resembles HIV-1 in several respects", the researchers said. "In addition, the immune dysregulation induced by *M. leprae* is often accompanied by the production of autoantibodies to numerous cellular proteins".

The authors, who included Harvard retrovirologist Max Essex, concluded that leprosy patients and their contacts "show an unexpectedly high rate of false-positive reactivity of HIV-1 proteins on both WB and Elisa". Since *M. leprae* shared several antigens with other members of the mycobacterial family, including *M. tuberculosis*, "our observations of cross-reactivity... suggest that HIV-1 Elisa and WB results should be interpreted with caution when screening individuals infected with *M. tuberculosis* or other mycobacterial species. 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".

"Quite high" is an understatement. According to the WHO, *M. tuberculosis* infects a third of the world's population and has an estimated annual death toll of three million people, of which about a third reside in Africa.⁶⁰ Malnutrition, drug resistance, and bad medical practice are likely causes of a spiralling epidemic. As far back as September 1992, a *WorldAIDS* briefing paper published by the Panos Institute stated that at any one time between 9 and 11 million people are suffering from the active infection—95% of them in Asia, Africa and Latin America. "In Africa TB has already become the prime cause of death in adults with HIV", the paper said. According to Panos, "the established epidemic of TB and the new epidemic of HIV have shown a disturbing tendency to coalesce and to co-infect individuals. It is a dangerous liaison both for those who are co-infected and for those communities in the developing world at risk of TB". Yet it seems clear from the Zaire study that this "epidemic of TB/HIV co-infection", as the WHO calls it, is a tragic error created by the non-specificity of the "HIV" test. People with active TB infection are at greatly increased risk of testing positive because of *M. tuberculosis*, not HIV.

Claims that "HIV infection" increases susceptibility to TB are not supported by evidence that TB responds to treatment just as well in "HIV-infected" people as in those who test negative for "HIV" antibodies. Studies conducted in Nairobi, Kenya and Kinshasa, Zaire, cited in 1992 by Dr. Paul Nunn of the London School of Hygiene and Tropical Medicine, measured the concentration of TB bacilli before and after drug treatment. Nunn reported that "surprisingly, the rate of decline of the concentration is faster in HIV-positive than negative patients. So the early bactericidal effect of anti-tuberculous therapy is not

adversely affected by HIV and possibly the reverse. Nor is the rate of persistently positive cultures at six months of therapy increased by HIV". Deaths were clearly greater among the HIV-positive group, but the research suggested this was "partly due to tuberculosis itself, but more important are non-tuberculous, non-AIDS-defining, bacterial infections . . . the main contribution to this excess mortality is from curable infections".⁶¹

The study most frequently quoted in the UK as offering support for the idea that HIV is devastating parts of Africa was conducted in rural Masaka, southern Uganda, funded by Britain's Medical Research Council (MRC). It involved 15 villages—about 10,000 people in all, mainly subsistence farmers and their families. Over a two-year period, five deaths were diagnosed as AIDS. However, 23% of HIV-positive adults died. This was a much higher death rate than that found among non-HIV-positive adults, and it was concluded that the excess, which resulted in a doubling of the overall death rate, was attributable to HIV. Deaths in the 13–44 age group totalled 51 among those who were HIV-positive and 18 among those who were HIV-negative. On the basis of those figures (and because there were far more HIV-negative than HIV-positive villagers), young HIV-positive adults were calculated to have a 60-fold greater risk of dying than the "non-infected" (96/1,000 against 1.4/1,000 man-years). The position looked even worse for the 13–24 age group, among whom 14 people died who tested HIV-positive, and only three out of a much larger group who tested HIV-negative died, producing a relative mortality ratio of 87.

This study, which eventually appeared in *The Lancet*,⁶² was repeatedly publicised beforehand by medical authorities in Britain and elsewhere, attracting newspaper headlines such as "HIV is Africa's big killer"⁶³ and "Africa study shows HIV victims 60 times likelier to die in two years".⁶⁴ Readers were told that this "latest and most comprehensive study of AIDS in Africa" provided "conclusive evidence that HIV has become a major killer on the continent", and that it showed "young adults with HIV were 87 times more likely to die prematurely than their uninfected contemporaries".⁶⁵ The newspapers did not mention that this horrific-sounding statistic was based on 14 deaths. Nor were they told that in the entire study, the number of AIDS diagnoses was *five*.

More importantly, the study authors did not consider the non-specificity problems with the HIV test. Their interpretation of the findings rested entirely on an assumption of "unequivocal HIV-1 serology", which in view of the evidence cited above is a contradiction in terms. They gave no details of the actual causes of death, nor of treatments offered. They acknowledged, however, that with a substantial proportion of the patients progressing to death within six months, on average, from having had either no symptoms or only mild illness, it was plausible to consider that lack of medical care was a contributory factor.

A reanalysis of the MRC study has shown that far from demonstrating that "HIV is Africa's big killer", the data seriously conflict with that view.⁶⁶ Instead, the data support the argument that "HIV" positivity is a consequence of deteriorated health, rather than a cause. The proof was offered by Vladimir

Koliadin, of the Kharkov Aviation Institute, Ukraine, in correspondence with the Royal Statistical Society. His letter was not published.

Koliadin complained that “the basic tenet of inductive statistical inference—that correlation cannot prove causation—seems to have been completely ignored”. He reasoned that if HIV was a new pathogen, causing deaths independently of other illnesses typical to the region, then deaths in the group who tested HIV-negative would stay the same as usual. On the other hand, “if HIV-positivity is only a marker of infectious diseases (the main causes of deaths among young adults in that region), mortality in HIV-negatives would be lower than normal”. That was simply because a big proportion of “normal” deaths would be linked with HIV positivity, and thus would be eliminated from the HIV-negative group.

So, the crucial question was whether the annual death rate of 1.4/1,000 seen in the HIV-negative group of young adults was “normal” for the region. The answer was, definitely not. A death rate of 1.4/1,000 was even lower than mortality in the US population of the same age range (1.5/1,000). Yet, mortality in Africa is notoriously high, compared with developed countries. High proportions of the population die from infectious diseases relatively young. It was reasonable to assume that the usual mortality rate in young adults in Uganda would be at least several times higher than in the USA. Assuming a rate of between 5/1,000 and 9.3/1,000 person-years (the overall death rate observed in this study), the actual distribution of deaths between the HIV-positive and HIV-negative subjects was 30–70 times higher than that predicted by the HIV-causes-AIDS theory.

Predicted Heterosexual Epidemics Never Happened

Long-term trends in Uganda’s population numbers are consistent with Koliadin’s analysis, as well as with the Perth group’s insistence on the non-specificity of the HIV test. In 1985 Robert Gallo and his colleagues reported testing stored sera collected in 1972/1973 from the West Nile district of Uganda. The samples had come from healthy children, mean age 6.4 years, randomly selected as controls for a study of Burkitt’s lymphoma. Both Elisa and WB tests were used. Fifty of the 75 children were found to be HIV-positive (67%).⁶⁷ As the Perth group comment, “According to HIV experts these positive results are explicable by virtue of mothers infecting their children. Thus, Gallo and his colleagues expected to find at least an equal percentage of infected adults. Mortimer *et al* assert that ‘Very few HIV-infected children are surviving into adulthood in good health’ and, given the fact that neither these children nor adults had treatment for HIV or AIDS, and the incubation period for AIDS in Africa is claimed to be four years and HIV heterosexually transmitted, then if the tests are HIV specific and HIV causes AIDS, by now few, if any, Ugandans should be alive”.⁶⁸ In fact, Uganda’s population is currently growing by

a healthy 2.5% per annum. This phenomenon is explained by protagonists of the HIV theory as demonstrating the effectiveness of condom campaigns!

In prosperous countries, the predictions of spread of the virus that was said not to discriminate have proved wildly wrong. Wherever AIDS deaths can be properly tracked, they remain linked to the original risk groups. In cases where none of those risks are apparent, the ill effects of long-term use of antibiotics⁶⁹ as well as antiviral drugs and the intensely damaging effect of an HIV diagnosis may have been to blame.⁷⁰

In 1992, when AIDS cases were already falling in the USA and Europe, experts agreed on an arbitrary widening of the range of disorders eligible for registration as AIDS, including, for the first time, HIV-positive people with no illness but with T4 cell counts below 200, as well as women with cervical cancer. In the USA, this produced an artificial doubling in the number of AIDS cases reported, but despite further expansions in classification, registrations have been declining ever since. About 650,000 cases of AIDS were registered in the USA from 1982 to mid-1998, and three quarters of those were clearly identified as occurring within high-risk groups.

More significantly, of 1,789 babies registered cumulatively as AIDS cases over the same period, 1,774 (99%) were born to mothers in high-risk groups.⁷¹ An analysis of data from the AIDS epicentres of New York City and California by Gordon Stewart, emeritus professor of public health, University of Glasgow, Scotland, a former WHO adviser on AIDS, shows that “perinatal and neonatal AIDS are minimal except where mothers and infants are exposed to risks in ethnic, drug-using and bisexual situations. After 20 years of intensive surveillance in a country where AIDS is as prevalent as in some third world countries, this in itself excludes any appreciable spread of AIDS by heterosexual transmission of HIV in the huge majority of the general population”.⁷² This is a far cry from the heady days of the Washington AIDS conference in 1987, when a computer model prepared at the Los Alamos National Laboratory contemplated the possibility of one adult in 10 becoming infected by 1994, and when Oprah Winfrey reflected the current perception by opening her show with the words: “Hello everybody. AIDS has both sexes running scared. Research studies now project that one in five—listen to me, hard to believe—one in five heterosexuals could be dead of AIDS in the next three years”.

In Europe, despite continuing efforts by public health officials to talk up AIDS so as to prevent complacency over unsafe sex, time has killed the idea that millions could be affected. Whereas in 1985 the UK’s Royal College of Nursing had predicted that one million people in Britain “will have AIDS in six years unless the killer disease is checked”, 15 years later (in 2000) AIDS deaths totalled 263—“less than the number of people who died from falling down stairs”.⁷³ The disease has remained almost exclusively confined to the original risk groups. Around 25,000 people are currently diagnosed as HIV-positive in the UK—half to a quarter of the estimated totals made in the mid- to late-1980s. The picture is similar across the European continent, with deaths now at double

and single figures in many countries. Cases have increased in some eastern European countries but mainly among drug users, and where poverty has increased vulnerability to TB.

Professor Stewart comments that “disastrous epidemics due to heterosexual transmission of HIV were confidently predicted in general populations of developed countries but they never happened. AIDS has diminished in incidence and severity though it is continuing in female partners of bisexual men and some other communities engaging in or subjected to behaviours which carry high risks of infections, various assaults and misuse of drugs”.⁷⁴ He has been trying for years to persuade scientific and medical colleagues that the statistics do not support the theory that AIDS is caused by an unselectively infectious agent. Despite a lifetime’s work in epidemiology and preventive medicine, and despite his predictions for the development of the epidemic having proved to be much closer to reality than those based on the orthodox view, his carefully argued papers have been consistently rejected by leading journals. He says that by 1987 there was no evidence whatsoever that AIDS was being transmitted heterosexually in general populations. When he submitted the relevant data and interpretations in a report to the WHO, they received attention internally, but were barred from publication. “Meanwhile, medical literature exploded, with world-wide coverage in all media, to accommodate the consensus view that AIDS was becoming a global pandemic. Alarming figures accepted at face value by WHO from some third-world countries were used to support this assertion”.⁷⁵ Stewart adds that since 1990, *Nature*, *Science*, the *New England Journal of Medicine*, the *British Medical Journal* and other mainline, peer-reviewed journals “have preferred to reject papers by others besides my colleagues and myself containing verifiable data that throw doubt on the claim that AIDS is capable of causing epidemics in general populations of developed countries by heterosexual transmission of HIV, and also falsify the hypothesis that HIV is the sole cause of AIDS”.

Disillusionment Over Antiviral Drug Treatments

To escape embarrassment over the failed predictions, AIDS experts have argued that antiviral drug treatments are responsible for the decline in AIDS. This is hard to reconcile with the fact that the decline started well before the more recent drug treatments were introduced or with the unsatisfactory record of these treatments.

AZT, the early “gold standard” of treatment, is now widely understood to have killed more patients than it helped (that is putting it kindly—there has been minimal evidence of help, beyond a broad, temporary, anti-microbial effect). The longest and most thorough trial of the drug, the Anglo-French Concorde trial, found 25% more deaths among those treated early than in those for whom treatment was deferred. The difference would almost certainly have been larger if the deferred treatment group had been a genuine control and had been kept

AZT-free. The drug made no difference in terms of progression to AIDS or AIDS-related illnesses. In a separate analysis of data from the first year there was a slight advantage to being in the immediate-treatment group; this lost statistical significance by 18 months.⁷⁶ Despite intense efforts by the drug's manufacturers to minimise the significance of these results, AZT is now known to have caused much harm, and possibly many thousands of deaths.

Similar high hopes, followed by disillusionment, accompanied a "hit hard, hit early" policy introduced in 1996—a policy of attacking the virus with cocktails of several antiviral drugs, including a group called protease inhibitors. Stories abounded of AIDS patients rising from their sickbeds like Lazarus, and there were proud boasts that HIV was on the run at last. But as with AZT, this was more wishful thinking than sound science. People with AIDS suffer many viral and other infections, and the drug cocktails gave relief to some of these, but giving the drugs to people simply on the basis of their "HIV" positivity was to prove another disaster. For several years it was left to the dissident network to report unexpected deaths on the drugs, but eventually the "hit hard, hit early" policy was reversed in February 2001 US Government guidelines acknowledging "unanticipated toxicities".⁷⁷ Drug companies were also ordered to stop advertising their antiviral drugs with images that imply they cure AIDS (such as photographs of "robust individuals engaged in strenuous physical activity") or reduce its transmission. This reversal came a year after an article by American journalist Celia Farber that began, "In 1996 a scientist claimed he'd found a way to defeat AIDS. In the wave of euphoria that followed, a batch of new drugs flooded the market. Four years later, those drugs are wreaking unimaginable horror on the patients who dared to hope. What went wrong?"⁷⁸ The article was reluctantly accepted as accurate by veteran AIDS activist Larry Kramer, previously a strong advocate of the antiviral drug approach as a means of tackling AIDS.

Treatment guidelines published in the *Journal of the American Medical Association* in July 2002⁷⁹ acknowledge that "The future of antiretroviral therapy rests with the development of new drugs that will result in simpler, more effective, and less toxic regimens along with development of an improved understanding of innate immune system responses". The authors assert in the first paragraph of this document that "potent antiretroviral therapy has resulted in dramatic reductions in morbidity and mortality, and health care utilization", and they offer three references to this claim. But according to Dr. David Rasnick, an organic chemist who worked in the US pharmaceutical industry for more than 20 years, all three references are to observational studies and not to actual clinical trials. "This is crucial", he writes. "Only clinical trials can show whether or not drugs actually work. To date, there are no drug clinical trials that show people taking the anti-HIV drugs live longer or at least better lives than a similar group of HIV-positive people not taking the drugs".⁸⁰

Some of the most experienced mainstream AIDS researcher/clinicians, as well as dissidents such as Rasnick, had long predicted that "hoopla" over antiviral

drugs could lead to disappointment and danger. Jay Levy, M.D., a professor in the department of medicine at the University of California, San Francisco, commented in 1996: "... get any virologist aside and they'll say this is not how we are going to win, it's high time we look at the immune system".⁸¹ Two years later he wrote: "These drugs can be toxic and can be directly detrimental to a natural immune response to HIV. This effective antiviral immune response is characteristic of long-term survivors who have not been on any therapy".⁸² Donald Abrams, professor of medicine at San Francisco General Hospital, revealed in a 1996 interview: "In contrast with many of my colleagues, I am not necessarily a cheer-leader for anti-retroviral therapy. I have been one of the people who's questioned, from the beginning, whether or not we're really making an impact with HIV drugs and, if we are making an impact, if it's going in the right direction. ... I have a large population of people who have chosen not to take any antiretrovirals. They've watched all of their friends go on the antiviral bandwagon and die, so they've chosen to remain naïve [to therapy]. More and more, however, are now succumbing to pressure that protease inhibitors are 'it'. We are in the middle of the honeymoon period, and whether or not this is going to be an enduring marriage is unclear to me at this time".⁸³ The marriage should by now have been annulled, but it is immensely hard for physicians to acknowledge that they could have been harming their patients, and it is also difficult for "HIV" experts to lose such an important plank in their defence of the beleaguered virus theory of AIDS.

Alive & Well AIDS Alternatives is a support and research organisation founded in the USA by a group of people diagnosed HIV-positive "who live in health without AIDS drugs and without fear of illness".⁸⁴ Christine Maggiore, the founder, a former awareness educator for prominent AIDS groups, began to scrutinize AIDS science after a series of tests she took fluctuated between HIV-positive, -negative, and indeterminate. In line with Abrams's observation, she had also noticed that her ill and dying colleagues were the ones following doctor's orders. She says that carefully considered choices "keep me and hundreds of other unmedicated HIV positives defiantly alive and well".⁸⁵ The organisation supports a growing network of groups and affiliates in America, Brazil, Canada, Kenya, Namibia, Nigeria, Mexico, South Africa and Zambia.

A face-saving shred of benefit for the HIV belief system seemed to have been found when it was shown that use of AZT in pregnancy could cause fewer children to be born testing positive. However, since we do not know the meaning of "HIV" antibodies, we do not know what this means in terms of the babies' health. Rasnick, who for several years has been the most active of the US AIDS "dissidents", told President Mbeki's inquiry into AIDS science in South Africa in July 2000 that he had "scoured the literature" for evidence of tangible benefit, with zero results.⁸⁶ Several studies have shown harm, including a major Italian survey which found that children born to mothers treated with AZT in pregnancy were more likely to get severely sick and die by the age of three than those whose mothers were left untreated.⁸⁷ AZT's proven toxicities include severe

muscle pain, weakness, and atrophy; heart muscle changes and malfunction; bone marrow suppression, with consequent anemia and loss of all types of blood cells; liver failure; and broad-ranging and sometimes irreversible loss and poisoning of mitochondria, the energy factories within our cells. The drug also leads to permanent DNA damage, and studies in mice and monkeys have raised concerns that babies exposed to AZT will face an increased risk of cancer later in life.⁸⁸

Nevirapine, the other antiviral drug heavily promoted by AIDS activists in Africa as essential in curbing mother-to-child transmission of HIV (and by others who sought to batter President Mbeki when he questioned orthodox thinking on HIV and AIDS) has similarly not been shown to have any clinical benefits and has been shown to carry a high risk of toxicity.⁸⁹

Triumph or Tragedy? Scientifically, the HIV Theory Has Failed to Deliver

In scientific terms, the HIV hypothesis has failed to deliver. The predictions of spread to which it gave rise have not materialized, and the drug treatments it spawned have disappointed, despite billions spent on research. It is not known how HIV harms the immune system, and there is uncertainty over its very existence. The blood test is non-specific (although, serendipitously, its very non-specificity has helped protect blood supplies against the broad range of pathogens that can cause “HIV” antibodies to become elevated), as are the “viral load” tests. The search for a vaccine is never-ending despite (or possibly because of) a commitment of hundreds of millions of dollars in US federal monies. Over the past 15 years, world-wide, more than 30 candidate vaccines have been tested in early-phase trials involving about 10,000 people. Out of these, only two are proceeding to phase III trials, and these are beset with difficulties. According to the WHO, the main stumbling blocks are lack of information about how best to measure protective immunity, the variability of HIV strains and lack of a good animal model.⁹⁰ According to Elepulos, “a vaccine is never going to happen. It can’t, because without HIV isolation, you do not know what you are dealing with”.⁹¹

In social terms, the HIV theory has produced some real benefits. The democratising of the threat of AIDS brought the world together in a way that has been profoundly beneficial for gay men, now considerably more accepted and valued in society than they were 20 years ago. Along with the red ribbon, “HIV/AIDS” has also become a symbol of unity and compassion. Perhaps it even served the West by providing a diffuse “enemy” against which to focus hostile energies released following the fall of the Soviet Union.

As Elepulos acknowledges, the condom and clean needle campaigns will also have had value. Lifestyle changes implemented within a certain section of the gay community, previously at great risk, probably lie behind the huge

diminution in AIDS in most of Europe, along with greatly reduced dosages and increased awareness of the toxicity of AZT. Whatever the cause of AIDS, many studies have demonstrated clear risks attached to anal intercourse and needle sharing. Animal studies show that transmissible AIDS-like diseases can be induced—without any exogenous infection—when the immune system is thrown into confusion through certain immunisation procedures (these have involved injecting female mice, previously mated with genetically distinct males, with lymphocytes from those males).⁹² There may be a genetic mechanism in AIDS akin to the “jumping genes” phenomenon, but involving transfer of genetic information out of the cell and in exceptional circumstances, from person to person.

To Rudolf Werner, professor of biochemistry at the University of Miami Medical School, such studies support the idea that AIDS is essentially an autoimmune disease. “We still know very little about autoimmunity and how it works”, he says. “Introduction of foreign protein into someone else’s system quite clearly upsets that person’s immune system. We need to learn much more about immunological tolerance and autoimmunity”.⁹³ Anti-lymphocyte auto-antibodies are present in 87% of HIV-positive patients and their levels correlate with clinical status.⁹⁴ Werner agrees that although AIDS drugs have been credited for the reduction in AIDS deaths, “there is no scientific evidence that these toxic drugs prolong life”. In a letter published by *The Miami Herald* (July 18, 2002) headed “Does the HIV virus really cause AIDS?”, he points to a study showing that the time between becoming HIV-positive and the time of death was identical in a Uganda group who received no AIDS drugs and a US group who did. “Since most people in the Uganda study were malnourished and multiply infected, doesn’t that suggest that antiretroviral drugs reduce life expectancy? . . . Unfortunately, the government suppresses alternative explanations of AIDS. This dogmatic approach certainly will lead to a medical disaster”.

The exclusion of research into other possible causes of AIDS that accompanied the establishment of the HIV paradigm may already have cost many lives, through failure to provide more effective advice on prevention and treatment. The efforts of those calling for a scientific reappraisal of the “HIV” hypothesis have usually been met with indifference and on occasions, abuse. In common with Duesberg, I have been called a “pariah of my profession” for broadcasting flaws in AIDS science to the public, bypassing the silence on this subject maintained by most mainstream scientific and medical journals and their supporters in the mainstream media. When Duesberg persisted in challenging the HIV theory he was derided by former colleagues, refused renewal of a \$350,000 “outstanding investigator” award from the National Institutes of Health and “all but exiled from American science”, as Rasnick puts it. Rasnick, who is perhaps the most persistent as well as articulate of the US dissidents, wrote in 1997: “As a scientist who has studied AIDS for 16 years, I have determined that AIDS has little to do with science and is not even primarily

a medical issue. AIDS is a sociological phenomenon held together by fear, creating a kind of medical McCarthyism that has transgressed and collapsed all the rules of science, and imposed a brew of belief and pseudoscience on a vulnerable public".⁹⁵

The Perth group has also suffered pervasive censorship, in which the AIDS mainstream has simply refused to enter into any discussion of their work. They were given satellite symposium time to present their case at the 1998 International AIDS Conference, in Geneva, as a result of intense lobbying by patient advocates, and against the wishes of the scientific committee; out of about 12,000 delegates, some 15 attended. That was at least an advance on the behaviour of organisers of the Berlin conference four years previously. "Dissidents" who persisted in setting out their literature on an unused table were ejected from the conference and told that they would be arrested and deported from Germany if they returned.

However, the biggest tragedy arising from the HIV paradigm has been the marketing and acceptance world-wide of an unvalidated diagnostic test, represented as demonstrating infection with a lethal virus. Millions are suffering the stigma and fear associated with this "HIV disease" diagnosis. Continents and subcontinents are being encouraged to funnel scarce resources into fighting what may be a mythical enemy. As Papadimitriou remarked to me, of AIDS in Africa, "Why condemn a continent to death because of HIV when you have other explanations for why people are falling sick?"

WHO experts are so convinced of a pandemic that they multiply the AIDS cases registered with them many times over to reach an estimate of the "actual" level. Furthermore, the multiplication factor has been regularly increased, as discovered by Christian Fiala, an Austrian physician who has spent years researching AIDS epidemiology, including a fact-finding mission to Uganda and Tanzania. In 1996, reported cases in Africa were multiplied by WHO statisticians by 12 to reach estimated totals; in 1997, by 17; and over an 18-month period in 1997/1998, by 47.⁹⁶

UNAIDS, which brings together seven United Nations agencies, including WHO, in a joint programme on AIDS, is doing work with huge potential for helping Africa by campaigning for debt relief and other forms of emergency aid. But it risks destroying the value of its efforts by tying them exclusively to the HIV/AIDS paradigm, increasingly questioned within Africa itself. By urging African finance ministers to devote more domestic funds to AIDS activities, "notwithstanding the weak fiscal situations in many of the worst affected countries in Africa",⁹⁷ it may exacerbate the real problems, which, as South Africa's Thabo Mbeki has indicated, are mostly related to poverty. UNAIDS has actually spelled out that it wants resources programmed for welfare, education, rural development and other health purposes to be redirected into HIV/AIDS care and prevention.

In the South African context, this would be particularly disastrous. Dr. Sam Mhlongo, professor of primary health care and family medicine and chief family

practitioner at the Medical University of Southern Africa, Pretoria, a member of Mbeki's Advisory Panel on AIDS, points out that 50 years of apartheid have left half the population of South Africa with no access to sanitation and clean drinking water. Sub-standard housing, shacks and overcrowding favour the risk of massive infection and re-infection with tuberculosis (added to AIDS-defining criteria in 1993). Starving and malnourished children are particularly susceptible to respiratory and gastrointestinal infections and septicæmia. "Long before Luc Montagnier's HIV/AIDS 'discovery', Professor John Reid of the Durban Medical School noted that 50% of black children in rural areas of South Africa died before the age of five", Mhlongo writes.⁹⁸ "The commonest causes of death amongst these black infants were recorded as bronchopneumonia, dehydration and diarrhoea".

"Apartheid conditioned people not to see; when it comes to AIDS many still will not open their eyes", Mhlongo says.⁹⁹ What Mhlongo sees, in eastern and southern Africa, is chronic protein deficiency, a breakdown in civilian services, rising incidence of TB and malaria, declining prices for agricultural output, high inflation and unemployment, displacement by civil violence, and cutbacks in government services due to economic adjustments mandated by the International Monetary Fund and the World Bank. "There is no need to conjecture the mysterious antics of some retrovirus from the rainforest that supposedly jumped from monkeys to humans".

In the earlier years of AIDS, after US, British and French scientists successfully marketed the "deadly new virus" concept and the tests and treatments that went with it, the perception that there was a public health emergency made it hard for dissenting views to be expressed. Today, the silence may owe as much to the power of commercial interests, along with embarrassment over the failures of AIDS science, as to any altruistic motives. Perhaps also it is easier on the West's conscience to keep blaming an epidemic of a deadly new virus for an increase in immune deficiency in less-developed countries than it is to acknowledge the effects of worsening poverty consequent on economic restructuring,¹⁰⁰ crippling debt, and the after-effects of decades of socially destructive policies towards black people such as occurred under apartheid.

A reasoned response from the scientific community to the full range of evidence that has mounted against the HIV theory is overdue.

Notes

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³ Retroviruses use an enzyme, reverse transcriptase, to convert their

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- ²¹ Papadopoulos-Eleopoulos, E., Turner, V. F., & Papadimitriou, J. M. (1993). Is a positive Western blot proof of HIV infection? *Bio/Technology*, 11, 696–707.
- ²² Montagnier himself admitted in a 1997 interview with Djamel Tahi, a French TV journalist, that “we did not purify” the virus and added that he did not believe Gallo had done so either.

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- ³⁵ Giraldo (1998). *Ibid.*
- ³⁶ For an extensive review of this evidence, see chapter 9 of my book *AIDS: The Failure of Contemporary Science* (London: Fourth Estate, 1996).
- ³⁷ See Sheppard, H. (1993). Viral burden and HIV disease. *Nature*, 364, 291: “The high level of plasma virus observed by Piatak et al was about 99.9% non-culturable, suggesting that it was either neutralized or defective. Therefore, rather than supporting a cytopathic model, this observation actually may help explain the relatively slow dissemination of the infected cell burden and thus the relative ineffectiveness of therapy with nucleoside analogues which target this process. . . . The results presented are equally consistent with the conclusion that higher viraemia is a consequence of, rather than the proximate cause of, defective immune responses”.
- ³⁸ Personal communication.
- ³⁹ Personal communication.
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- ⁴⁵ This group's treatment recommendations are available at www.virusmyth.net.
- ⁴⁶ See Sonnabend, J. A., & Saadoun, S. (1984). The acquired immunodeficiency syndrome: A discussion of etiologic hypotheses. *AIDS Research*, 1, 107–120. This article pointed out that semen and sperm were well documented as a cause of immune system abnormalities in anal intercourse, when the proteins involved permeate the colon's thin lining and enter the bloodstream. (In vaginal sex, the vagina's thick walls restrict such invasion to its intended target, the womb.) There are antigens expressed on cells in the ejaculate that are shared by cells of the immune system, raising the possibility that repeated exposure could set up a reaction in the body against one's own immune cells. Anal sex has been around a long time, of course, but the Gay Liberation years brought exceptional exposures. A Centers for Disease Control study of the first 100 gay men with AIDS found that their median number of lifetime sexual partners was 1,160; a subsequent group boasted 10,000 or more partners. See also Root-Bernstein, R. (1993). *Rethinking AIDS: The Tragic Cost of Premature Consensus*. New York: The Free Press (pp. 115–120).
- ⁴⁷ Kremer, H. (2001). *Die Stille Revolution der AIDS und Krebsmedizin* (The Silent Revolution in AIDS and Cancer Medicine). See *Raum + Zeit* magazine interview, March 2002, on www.virusmyth.net/aids/index/hkremer.htm. Accessed November, 2002.
- ⁴⁸ Duesberg, P. (1996). *AIDS: Virus- or Drug-Induced?* Dordrecht, The Netherlands: Kluwer Academic Publishers.
- ⁴⁹ See also Hodgkinson, N. (1996). Drugged. In *AIDS: The Failure of Contemporary Science* (pp. 69–99). London: Fourth Estate.
- ⁵⁰ One of the best examples of this phenomenon was a study by Maurizio Luca Moretti of the Florida-based Inter-American Medical and Health Association, who collaborated with colleagues in Italy on a study of 508 former intravenous drug abusers. [Root-Bernstein, R. (1993). *Rethinking AIDS: The Tragic Cost of Premature Consensus*. New York: The Free Press (pp. 359–360).] The men, all HIV-positive, were voluntarily confined to a rehabilitation centre where their lives were under the daily management of staff. Most were found to be severely malnourished on arrival, 397 of them chronically so. Their nutritional status was returned to normal, their drug use ended, and their sex lives were curtailed (the centre is a monastery, where patients sleep in small groups under supervision). Among 139 individuals who had been using heroin daily for an average of more than five years, all were still free of AIDS symptoms after an average of more than four years since they had first tested positive. This is a phenomenal success rate compared with the USA, where a third of HIV-positive addicts develop AIDS within two years and more than half develop AIDS within four years.

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- ⁵⁴ More conviction on HIV and AIDS [Editorial]. (1995). *Nature*, 377, 1. Also Horton, R. (1995). Will Duesberg now concede defeat? *Lancet*, 346, 656.
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- ⁵⁸ The plague that isn't. *Canadian Globe and Mail*, 14 March 2000.
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conducted a massive social experiment in poor African countries. It is called structural adjustment. . . . Africa urgently needs a realistic evaluation of the continuing effects of debt and neo-liberal economic prescriptions on the health of its people”.