

# The Yin & Yang of HIV

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*A theory is a good theory if it satisfies two requirements: It must accurately describe a large class of observations on the basis of a model that contains only a few arbitrary elements, and it must make definite predictions about the results of future observations.- Dr Stephen Hawking*

## Part 1

**Supporters of the 'HIV causes AIDS' hypothesis cannot back up their claims with scientific evidence, yet they continue to reject alternative explanations and promote life-threatening drug treatments.**

**T**he notion that HIV/AIDS is infectious and sexually transmitted is based on a relationship between antibodies claimed specifically induced by a retrovirus, HIV, and particular diseases in certain risk groups. However, the

HIV theory has been challenged for well over a decade in many scientific publications, principally by Peter Duesberg from the USA and Eleni Papadopulos-Eleopulos and her colleagues in Perth, Western Australia.

Failure of HIV/AIDS to spread beyond the original risk groups and particularly to Western heterosexuals, especially non-drug-using prostitutes, signals that the HIV theory of AIDS is in need of urgent reappraisal. This has serious implications for both the way science has been conducted and for public health policy and planning. The HIV theory has cost billions of dollars and locked in enormous amount of energy in research by thousands of scientists worldwide. So far, it has yet to save a single life.

There is an urgent need to establish a truly independent and distinguished international committee to review the current theories and those that challenge them. There needs to be a co-operative but urgent reassessment of AIDS.

## A NOBEL LAUREATE STIRS THE WATERS

In 1988, Dr Kary Mullis, the 1993 Nobel Prize winner for Chemistry, was employed by the US National Institutes for Health (NIH) to set up analyses for HIV testing. When preparing his report, he asked a virologist colleague for a reference that HIV is "the probable cause of AIDS". He was told he did not need one. Mullis was surprised.<sup>[1](#)</sup>

"I disagreed. It was totally remarkable to me that the individual who had discovered the cause of a deadly and as-yet-uncured disease would not be continually referenced in the scientific papers until that disease was cured and forgotten... There had to be a published paper, or perhaps several of them, which taken together indicated that HIV was the probable cause of AIDS." Otherwise, as Mullis was forced to conclude: "The entire campaign against a disease increasingly regarded as the twentieth-century Black Death was based on a hypothesis whose origins no one could recall. That defied both scientific and common sense."

A decade later, Mullis was to write: "I finally understood why I was having so much trouble finding the references that linked HIV to AIDS. There weren't any."<sup>[2](#)</sup>

Indeed, an interested non-specialist observer, armed with a few contacts and a good library, merely has to scratch the surface to realise that the HIV theory of AIDS begs many more questions than it answers.<sup>[1-63\\*](#)</sup>

## THE BEGINNINGS OF AIDS

The few years leading up to the AIDS era and the discovery of HIV are illuminating. It was a time when a promiscuous minority of young, "liberated" gay men in a few large American cities were increasingly developing previously uncommon diseases such as fatal forms of the malignancy Kaposi's sarcoma and a fungal pneumonia known as PCP.

At the time, whilst it was reasonable to implicate an infectious microbe transmitted by rampant, indiscriminant sexual practices interspersed with needle sharing and drug taking, the fact that immune suppression had multiple causes was also known in 1981. Some considered the diseases resulted from multiple assaults to bodily functions caused by the many and varied diseases, toxins and treatments that accompanied the gay and drug-taking lifestyle that had evolved during the late 1970s.

Just how extensive these multiple assaults were was indicated by the English journalist Neville Hodgkinson, documenting the range of infections of just one homosexual, the late Michael Callen, in his book, AIDS - The failure of contemporary science: How a virus that never was deceived the world.<sup>[29](#)</sup> Hodgkinson writes: "From 1973, when he came out as a homosexual, to 1975, he only got mononucleosis and non-specific urethritis (NSU). In 1975, he had his first bout of gonorrhoea... But from there, it all began to snowball. 'First came hepatitis A in 1976 [said Callen]. Then more NSU and gonorrhoea. In 1977, amoebas [intestinal parasites] and hepatitis B...NSU and gonorrhoea. 1978: more amoebas...my first case of shigella [and] more VD. Then in 1979,

hepatitis a third time...non-A, non-B...amoebas...giardias...a fissure [and] my first case of syphilis. And of course, more gonorrhoea [penile, anal and oral]. In 1980: the usual gonorrhoea, shigella twice, and more amoebas...' Added to that list were herpes simplex types I and II; venereal warts; salmonella; chlamydia; cytomegalovirus (CMV); Epstein-Barr virus (EBV); mononucleosis; and finally cryptosporidiosis ('a disease of cattle!')." Indeed, an early US Centers for Disease Control (CDC) study confirmed that the first 100 men with AIDS had a median lifetime number of 1,120 sexual partners.<sup>30</sup> As Callen himself put it: "By 1981, I got some combination of venereal diseases each and every time I had sex."

Not surprisingly, given the widespread belief of a causal relationship between immunity and the maintenance of health, in 1981 the "new" disease became known as Gay Related Immune Deficiency (GRID). In fact, none of the diseases was new. Some were known to occur in drug addicts and haemophiliacs long before the AIDS era.<sup>64, 65</sup> What was "new" was their exponentially escalating prevalence in gay men.

## TECHNOLOGY & VIROLOGY

Coincidental with the beginning of the AIDS era, a technique was developed to classify and count the different types of lymphocyte white blood cells. It was noticed that some AIDS patients had diminished numbers of the so-called T4 "helper" cell subtype and, despite lack of proof, the cells were assumed to be dying at the behest of an agent selectively targeting them. This became the "hallmark" of AIDS as well, forming a measure of the amount of immune deficiency. In turn, this "immune deficiency" (the "AID" in AIDS) caused the diseases (the "S" in AIDS) that constitute the clinical syndrome. The perceptions that T4 cells were dying and AIDS was infectious led to the theory that AIDS is caused by a microbial organism.

Five years prior to the AIDS era, a few laboratories around the world were drawing towards the end of a fruitless search to prove a viral cause for human cancers. During the 1970s, Dr Robert Gallo, the central figure as "co-discoverer" of the AIDS virus, and his colleagues claimed to have discovered three human retroviruses. (The name "retroviruses" arises because of the copying of the RNA which forms the viral "genes" [the genome] "backwards" into DNA - a direction contrary to that long considered universal, that is, from DNA into RNA.)

In 1975, the first human retrovirus, HL23V, was proposed to cause human leukaemia, but by 1980 was considered an embarrassing mistake - in fact, not to have ever existed. Of the remaining two, one was postulated to cause a specific, though rare, form of adult leukaemia, and the second remains orphaned without a disease. What is significant is that the latter two retroviruses are said to exhibit a liking for T4 lymphocytes.

This led Donald Francis, [Gallo](#) and others to propose that an existing or closely related retrovirus was the agent responsible for killing the T4 cells in AIDS patients. When researchers actively sought and then discovered the same diseases in individuals who were not gay, retroviruses, as well as retrovirologists, received renewed interest and GRID became AIDS.

## FIRST PROCLAMATIONS

In May 1983, Professor Luc Montagnier and his colleagues at the Pasteur Institute in Paris published a paper in Science, entitled "Isolation of a T-Lymphotropic Retrovirus from a Patient at Risk for Acquired Immune Deficiency Syndrome (AIDS)".<sup>66</sup> It is important to note that the first word in this paper, "Isolation", serves as a signal that the researchers are claiming proof for the existence of a new virus.

In the interests of science, on several occasions Montagnier sent samples of his tissue cultures to the Gallo laboratory in America, with the express understanding that these "could be used for biomedical, biological and molecular biological studies".<sup>67</sup> However, Montagnier did not claim to have proven his virus was the cause of AIDS, and the French discovery lay on the table until May 1984 when Gallo and Popovic and their colleagues published four papers, also in Science.<sup>68-71</sup>

On 23 April 1984, at a Washington press conference held two weeks before the papers were published, Margaret Heckler, Secretary for Health and Human Services (HSS), announced that Gallo and his co-workers had discovered the "probable" cause of AIDS and had developed a sensitive blood test to detect the virus in the body. A curative vaccine was predicted within two years. Inexplicably, causation was proclaimed merely by association and despite "isolation" of HIV in only 26 (i.e., 36 per cent) of Gallo's 72 AIDS patients - barely a third. (The frequency of "isolation" is no better today.<sup>72</sup>)

In 1985, the Pasteur Institute alleged that Gallo had misappropriated their virus. The ensuing conflict, which eventually reached the US courts, was settled by a negotiated agreement signed in 1987 by Gallo and Montagnier as "co-discoverers", and US President Reagan and French Premier Chirac. Nevertheless, the matter drew the attention of John Crewdson, an investigative journalist, and US Senator John Dingell. In November 1989, Crewdson published a lengthy article in the Chicago Tribune newspaper, which provoked an internal NIH enquiry into suspect data from Gallo's laboratory.

A draft report of the formal investigation, written by the NIH Office of Scientific Integrity (OSI), was published in September 1991, in which the principal author, Mikulas Popovic, was accused "of misconduct for misstatements and inaccuracies" that appeared in the first Science paper, and suggesting that Gallo, as laboratory chief, "created and fostered conditions that give rise to falsified/fabricated data and falsified reports".

The OSI's final draft report, completed in January 1992, was immediately criticised, and was followed by a review of the OSI report by the Office of Research Integrity (ORI) which found Gallo guilty of scientific misconduct.

Nonetheless, even after this long investigation and its conclusion, the US Government withdrew its findings following Gallo's announcement of an appeal. Despite this, in 1994, US officials credited Montagnier and his colleagues as the discoverers of HIV and yielded the French a greater share of royalties from the HIV antibody tests. In taking these unprecedented steps, Dr Harold Varmus, the Director of the NIH, acknowledged that "scientists at the NIH used a virus provided to them by Institut Pasteur to invent the American test kit". This action scarcely vindicated the Dingell report which had concluded that the settlement "barely managed to paper-over the glaring, unresolved issues". Rather, it was the culmination of a cover-up where "political and international reputational imperatives" at HHS "assumed pre-eminence over scientific integrity", while defending Gallo's claim became "tantamount to defending the US Government itself".<sup>73</sup>

According to Eleopulos and her colleagues, regardless of the material uncovered by the OSI, Gallo's data, which still remains the best of its kind, does not prove the existence of HIV and, even if it did, nowhere in the papers is there proof that HIV causes AIDS.<sup>16, 21</sup>

## ENTER PETER DUESBERG

In December 1987, three and a half years after the Washington press conference, Professor Peter Duesberg, virologist and molecular biologist at the University of California, Berkeley, published an invited paper entitled "Retroviruses as Pathogens: Expectations and Reality".<sup>3</sup> Duesberg was a much fêted scientist, considered to be "the golden boy of virology" and "the greatest living retrovirologist". He had developed many of the laboratory techniques for studying retroviruses and their genetic make-up, had discovered cancer-causing genes, and was recipient of a \$350,000 "outstanding investigator" award from the NIH.

But Duesberg dropped a bombshell. He asserted that, apart from the relatively few cancer-causing retroviruses, the majority are virtually harmless. Duesberg argued that HIV is neutralised by antibodies shortly after infection and thus antibodies signal its containment. He also pointed to data proving that well, sick or dying-from-AIDS, HIV-positive individuals contain insufficient amounts of HIV to do harm. Even if HIV were to kill all the T4 cells it had infected every 1 to 2 days, the number of T4 cells needing replacement approximated the amount of blood shed by a man cutting himself shaving.

For the protagonists, the low "viral burden" - that is, the amount of "HIV DNA" in cells - was a fact that no one, not even Gallo, could satisfactorily reconcile with an immunity-destroying pathogen killing gay men within a year or two of diagnosis. However, rather than addressing this as a scientific problem warranting dialogue with someone known to have considerable knowledge of the subject, Duesberg's questions antagonised Gallo to the point where he refused to discuss the matter. Meetings convened to deal with the uncomfortable implications of Duesberg's paper were suddenly cancelled at the highest level.

In 1989, Duesberg presented further argument.<sup>4</sup> HIV does not fulfil the postulates that 19th century bacteriologist Robert Koch had developed to prove a microbe causes a disease. These four postulates are: (a) the organism must be present in all cases of the disease; (b) it must be grown and then isolated in pure culture from the cells of individuals with the disease; (c) it must reproduce the disease when introduced into a susceptible host or experimental animals, (d) from where it must once again be recovered.

According to Duesberg: "From every angle, HIV fails Koch's first postulate." The second postulate was fulfilled but only by subjecting cells to drastic chemical manipulation that did not approach conditions in vivo. (Eleopulos has argued how basic retrovirology has long shown that oxidation which prevails in HIV/AIDS patients and their cell cultures creates internal [endogenous] retroviruses in cells whose DNA was not previously infected from the outside.<sup>12, 14, 15, 74, 75</sup> One per- cent of human DNA, that is, an amount 3,000 times larger than "HIV" DNA, is made up of endogenous retroviral DNA.<sup>76</sup>)

The third postulate failed because, as Duesberg points out: "During the past decade, more than four hundred thousand AIDS patients have been treated and investigated by a system of five million medical workers and AIDS researchers, none of whom [has] been vaccinated against HIV... But ten years later there is not even one case in the scientific literature of a health worker who ever contracted presumably infectious AIDS from a patient... AIDS is not infectious." Similarly, "nine years after the NIH first started infecting chimpanzees with HIV - over 150 so far at a cost of \$40,000-50,000 apiece", all "are still healthy".<sup>5\*\*</sup>

In 1992, Duesberg shifted focus from HIV to argue that "AIDS [is] acquired by drug consumption and other noncontagious risk factors".<sup>5</sup> Apart from illicit and recreational drugs, Duesberg's list included the first "anti-retroviral" drug, zidovudine (AZT). In other words, a specific treatment for HIV infection was postulated to be a cause of AIDS.

Duesberg continued to regard HIV as bona fide, but as an inert, harmless "passenger" virus linked to AIDS only through the kinds of activity associated with drug taking (including taking of prescribed drugs). Duesberg, like others before him, pointed to the epidemiological data revealing a 50-fold difference in the AIDS "attack rate" between various groups of HIV-positive individuals, as well as the proclivity of certain AIDS diseases for particular risk groups. Thus, 50 per cent of HIV-positive blood transfusion recipients develop AIDS within one year (but so do 50 per cent of HIV negatives) compared to 1 per cent of haemophiliacs. Kaposi's sarcoma was, for all intents and purposes, confined to gay men.<sup>5,13,77</sup> Thus, even if HIV were necessary to cause AIDS, it could not be the only factor. However, accretion of "co-factors" to the HIV theory rendered the significance of any particular factor problematic. It was possible to argue that HIV may be only a minor factor or, at least in the minds of Eleopulos and Duesberg, not a factor.

Apparently, the role of HIV was also a problem for Montagnier. Although he wrote in Nature in December 1984 that "all available data are consistent with the virus being the causative agent of AIDS",<sup>78</sup> in 1985 he expressed an opinion impossible to reconcile with the HIV theory. "This 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",<sup>79</sup> that is, cause after effect (italics ours). One must surmise that, within a year, the discoverer of HIV was already hedging his bets. His recent interview with the investigative journalist Djamel Tahi<sup>61</sup> (see below) fuels such speculation.

## ELEOPULOS AND THE PERTH GROUP



Eleni Papadopulos-Eleopulos' AIDS research began in 1981. In May 1986, she submitted for publication a paper which refuted every step in the HIV theory, including HIV itself. She also proposed an alternative, non-viral theory (of which "Duesberg's" "Drugs/ AIDS hypothesis" is a subset), and predicated non-toxic and relatively inexpensive treatments.

Her theory was based on a general theory of cellular functioning, which she had formulated in the 1970s as a basis for unravelling the genesis and improving the treatment of cancer and to offer fresh insights into the pathogenesis of cardiovascular diseases and ageing. Eleopulos postulates that normal cellular functioning is determined by the level and oscillations of cellular redox<sup>23</sup> (oxidation and its chemical opposite, reduction). In her view, when oxidation is prolonged or excessive, cells become abnormal, injured and susceptible to diseases.

Eleopulos had noticed a link between the risk groups. Gay men, drug users and haemophiliacs are exposed to chemical stressors in the form of semen, nitrites, illicit drugs and factor VIII (the blood-clotting protein missing from and administered to haemophiliacs). There is abundant evidence that these substances are potent cellular oxidants.<sup>12</sup> In Eleopulos' view, oxidative stress produces low numbers of T4 cells and AIDS, as well as the phenomena inferred as proof for the existence of HIV.

The ready acceptance of the Montagnier/Gallo 1983/84 Science papers posed enormous difficulties for Eleopulos in having her work published. Thus, "Reappraisal of AIDS: Is the oxidation caused by the risk factors the primary cause?" was twice rejected by Nature, eventually finding light of day in Medical Hypotheses, twelve months after Duesberg.<sup>12</sup> However, the editor of this journal had also rejected the paper, only recanting after Eleopulos worked for several months to convince him that equatorial Africa was not in the grip of an epidemic of sexually transmitted immunodeficiency and thus not in breach of her theory.<sup>11, 24, 63, 80</sup>

To paraphrase the theoretical physicist Stephen Hawking: wrong predictions affirm bad theories; correct predictions make them powerful. The HIV theory requires that HIV causes all the AIDS-defining diseases and predicts that HIV/AIDS will become a global epidemic via the oldest and most unstoppable of all human activities. However, Kaposi's sarcoma, one of the two diseases for which the HIV theory was proposed, is no longer attributed either directly or indirectly (via AID) to HIV.<sup>12, 13, 54, 77, 81\*\*\*</sup>

In the OECD countries, the prediction of a sexual pandemic has failed completely. For example, as of the beginning of 1998, 93 per cent of the cumulative deaths from AIDS in Australia occurred in the original risk groups, that is, gay/bisexual men, drug addicts and haemophiliacs. This observation fits the classic demographic profile of non-infectious diseases such as pellagra, beri-beri and scurvy which characteristically remain confined to their risk groups. All are caused by vitamin deficiencies, but in the past were regarded as infectious and sufferers were shunned and quarantined.

The HIV protagonists also predicted a curative vaccine by the end of 1986 and an animal model to prove the HIV theory beyond all doubt. Neither prediction has been fulfilled. A vaccine is not envisaged until well into the next century, possibly around 2010, and animals given "HIV" do not develop AIDS.

On the other hand, the Eleopulos oxidative stress theory predicts the current demographic data, an apparent loss of T4 cells, the risk from passive anal intercourse in both sexes, HIV positive and AIDS patients being oxidised relative to normal individuals, the amelioration of HIV/AIDS by the use of antioxidants, and a non-infectious animal model. Every one of these predictions has materialised. Oxidative stress is well established by hundreds of papers,<sup>14, 62, 82-84</sup> so much so that in the early 1990s the Pasteur Institute was advertising international scholarships for study into the phenomenon. In fact, last year Luc Montagnier became the principal editor of a 558-page book devoted to oxidative stress in cancer, ageing and AIDS.<sup>85</sup>

The Eleopulos theory predicts that a decline in T4 cells can occur without cellular death. In fact, according to the Perth group, there is no evidence to support the notion that T4 cells are dead or that "HIV" kills such cells. In T4 cell cultures, the same number of T4 cells "disappear", regardless of whether one adds "HIV" or merely the chemical stimulants obligatory to "grow" the "HIV".<sup>86</sup> Neither is there proof that low numbers of T4 cells are

either necessary or sufficient to produce the clinical syndrome.<sup>9, 12, 14</sup> This is a view recently expressed by leading HIV/AIDS scientists such as Dr Arthur Anderson from the US Army Medical Research Institute of Infectious Disease<sup>87</sup> and Dr Zvi Grossman at the University of Tel Aviv.<sup>88</sup>

In other words, the central tenet of the HIV theory - virus-induced killing of immune cells leading to AIDS - is now being questioned by HIV/AIDS experts themselves. Nonetheless, and despite so much evidence to the contrary, the orthodox view remains entrenched. In fact, since 1993, the low number of T4 cells has been enshrined in the 1993 CDC AIDS definition whereby AIDS can be diagnosed without a disease. Just as "co-factors" were proposed to rescue the HIV theory in the mid 1980s, in July 1998 Chen and colleagues from the UCLA AIDS Institute, School of Medicine, Los Angeles, reported evidence that "naturally non-infectious virus" or virus "rendered defective" by "anti-HIV" drugs could still contribute to the loss of T4 cells throughout the course of HIV disease.<sup>89</sup> In other words, "alive" or "dead", HIV causes immune deficiency. Such a proposal does not augur well for the use or continued development of "anti-HIV" drugs.

Consistent also with the Eleopulos oxidative stress theory is the direct relationship between high frequencies of passive anal intercourse and the development of AIDS, as well as the fact that the only animal model of AIDS is non-infectious. Mice repeatedly injected with foreign cellular proteins develop a dramatic depletion of T4 cells and Kaposi's sarcoma-like tumours, and "abundant" retrovirus-like particles appear in their spleens.<sup>90</sup> Thus AIDS diseases are followed by the production of retrovirus-like particles, and not the other way around.

To the uninitiated this may seem perplexing, but it is well recognised that retroviral particles appear de novo in cell cultures not previously infected, because all cells contain retroviral information carried in the germ line DNA.<sup>76</sup> Indeed, according to distinguished retrovirologists such as Weiss and Temin, new retroviral DNA arises by rearrangement of cellular DNA, caused by many factors including pathological processes - a view that concedes retroviruses an effect and not the cause of diseases.<sup>74, 75</sup>

## THE RISE AND FALL OF "ANTI-HIV" DRUGS

It would take a second article to discuss AZT and the many other "anti-HIV" drugs. Suffice it to say there is no scientific proof that such drugs kill "HIV" or cure AIDS, but there is ample evidence they are harmful.<sup>1, 53, 56, 91</sup>

In 1994, a double-blind randomised comparison of two policies of AZT treatment (immediate and deferred) was reported (the Concorde trial). This involved 1,749 symptom-free, HIV-infected individuals from centres in the UK, Ireland and France. The 347 clinical endpoints (AIDS and death) outnumbered the total of those in all other published trials in symptom-free and early symptomatic infection. The results showed "there was no statistically significant difference in clinical outcome between the two therapeutic policies".<sup>92</sup> In 1995, extended results of Concorde showed a significant increased risk of death among the patients treated early.

However, despite these data, despite disclaimers that patients treated with AZT may continue to develop the AIDS diseases, that the side effects of AZT may mimic AIDS, and that AZT given to non-HIV-infected babies causes the AIDS-defining pneumonia, PCP,<sup>93</sup> AZT continues to be the most commonly prescribed anti-HIV drug.

Dr Donald Abrams, Professor of Medicine and Director of the AIDS program at San Francisco General Hospital, said: "I have a large population of people who have chosen not to take any anti-retrovirals... I've been following them since the very beginning... They've watched all of their friends go on the antiviral bandwagon and die."<sup>94</sup>

Indeed, even an elementary study of the pharmacological literature reveals that AZT cannot be an anti-HIV drug; it is toxic to all cells.<sup>91</sup> In fact, what unites long-term survivors of AIDS is their resolve not to take "anti-retrovirals".<sup>95-97</sup>

In mid-1996, the latest drugs, the "protease inhibitors" (PIs), were introduced. These are prescribed as one of up to 250 possible combinations of "cocktails" with AZT or similar drugs as "highly active anti-retroviral therapy" (HAART). Detailed data on these drugs, of the kind usually reserved for medical practitioners, appear regularly in glossy, multi-page advertisements in gay men's magazines.

At the July 1996 11th International AIDS Conference, Time magazine Man of the Year David Ho predicted that scientists would "find new drugs to wipe HIV out of the body within three years, possibly within just one".<sup>98</sup> At the July 1998 XIIth AIDS conference, Ho stated it will take at least 10 years of intense combination drug therapy to kill off all the HIV in an infected person's body, but that a sizeable percentage of HIV patients will never get close. Many patients cannot tolerate the untoward effects of these "cocktails", and measurements show that the DNA "viral" burden does not decrease significantly.<sup>99-102</sup> According to Kaufmann et al.: "49% of people taking HAART, who were followed by the Swiss HIV Cohort Study, did not have viral suppression, and similar data are emerging from other centres."<sup>103</sup>

In the May 1998 Proceedings of the National Academy of Sciences, Dr William Paul, former Director of the National Institutes of Health's Office of AIDS Research, wrote: "...no matter how long a person is treated with anti-HIV drugs, there will always be new viruses...you will have to be treated forever... No one is getting cured... This bodes extremely poorly for combination therapy as something curative..."<sup>88</sup>

Dr Michael Saag, at the University of Alabama, Birmingham, USA, is responsible for the treatment of over 1,000 AIDS patients. His treatment is state-of-the-art and his clinic is sought- after by pharmaceutical companies to conduct trials on their newest compounds. In a recent interview, Dr Saag said: "Perhaps the biggest difference between the cure paradigm and whatever paradigm we're in now is, we now should expect failure with whatever [HAART cocktails] we first use. We should plan on it. We should prepare for it. Clinicians should expect failure." Saag warns the HAART "'dam' is already leaking and there's high danger of it collapsing altogether. Failures are occurring right and left." Speaking about his dying patients: "They aren't dying of traditionally defined AIDS illness... I don't know what they're dying of, but they are dying. They're just wasting and dying... It is sobering...while we are making good guesses, they are just guesses. We don't know what we are doing."<sup>104</sup>

Given the toxicity of these drugs, it is unlikely anyone can tolerate taking them for more than a few years. If this outlook is gloomy for HIV/AIDS sufferers, it is even worse considering there is no substantial, alternative therapeutic strategy anywhere on the horizon.

The futility of all "anti-HIV" drugs, past, present and future, is best highlighted in a June 1998 interview by Dr Harold Varmus, Nobel Laureate retrovirologist and Director of the National Institutes of Health: "Trying to rid the body of a virus whose genome is incorporated into the host genome may be impossible."<sup>105</sup>

## THE DEMISE OF SCIENTIFIC DEMOCRACY

The longevity of the HIV theory has been considerably boosted by the virtual refusal of editors of leading medical journals to publish any material which takes HIV to task. Without these data, and the stamp of approval engendered by such publication, it is almost impossible for the debate to reach the ears of those who matter the most: clinicians and their patients. Like generals directing wars, the remoteness of editors begets an objectivity which, while essential to clear thinking, militates against an appreciation of the profound responsibilities editors hold at the bedside.

Ultimately, although the HIV theory is manifoldly problematic, physicians, patients, relatives, politicians, journalists and the tax-paying public are systematically denied knowledge of its existence and substance. Not only is there a total absence anywhere of a disinterested, adjudicated debate, but individuals, whose only motivation is to contribute to solving a disease claimed to afflict millions of people, find themselves censored. For example, Sir John Maddox, former editor of the world's most prestigious science journal, Nature, denied Duesberg the right of reply on issues he raised because his views give "many infected people the belief that HIV



infection is not in itself the calamity it is likely to prove".<sup>29</sup> Yet, in a recent edition of the same journal, but in another context, there is a claim that "the voice of sceptics may grow tiresome, but the mainstream is in trouble if it cannot win a public debate with them".

Officials at the Berlin 10th International AIDS Conference confiscated Dutch AIDS analyst Robert Laarhoven's press pass and threatened him with expulsion from Germany for "criminal trespass" because he placed copies of the dissident journal Rethinking AIDS on an "unauthorised" table.

Nature has repeatedly rejected every paper and letter submitted by Eleopulos and her colleagues since 1986, without providing a single scientific reason and invariably citing space constraints in the journal. Not even the profound implications of the Tahi/Montagnier interview are of any apparent concern to Nature. Professor John Kaldor, one of Australia's foremost "established experts" on AIDS, admits that dissidents "intersperse their cases with grains of fact".<sup>106</sup> However, because of Kaldor and colleagues' "strong instinct not to dignify the sceptics' arguments by attempting to refute them", arguments based on these "grains of fact" and many other data remain unanswered and unresolved.

## Part 2

**When put to the test, conventional HIV/AIDS theory is at odds with the clinical evidence. Is "purified HIV" no more than a tangle of cellular debris?**

*The real purpose of scientific method is to make sure Nature hasn't misled you into thinking something you don't actually know... One logical slip and an entire scientific edifice comes tumbling down. One false deduction about the machine and you can get hung up indefinitely.*

- Robert Pirsig, *Zen and the Art of Motorcycle Maintenance*

## SOME SCIENTIFIC PROBLEMS WITH THE HIV THEORY

### The theory vs the definition

The central premise of the HIV theory of AIDS is that there exists a unique retrovirus, transmissible via blood and sexual secretions, which induces specific antibodies and kills T4 cells whose relative absence then causes the appearance of approximately 30 diseases which constitute the clinical syndrome. The theory, however, is rendered completely contradictory by the official AIDS definition used clinically.

In Australia, an individual is diagnosed as having AIDS if he or she fulfills the criteria set out in the latest (1993) revision of the US "CDC surveillance case definition for AIDS".<sup>107</sup> (Other definitions in use around the world make scientific comparisons almost impossible. In Africa, AIDS is diagnosed on symptoms and without blood tests.<sup>108</sup>) Since from 1985 the Centers for Disease Control "accepts" HIV as the cause of AIDS, it should not be possible to diagnose AIDS by any means inconsistent with the HIV theory. However, even a cursory reading of the 1993 definition reveals AIDS can be diagnosed - with the imprimatur of the CDC - with Kaposi's sarcoma (which even Gallo<sup>54</sup> accepts is not caused by HIV), in the absence of immune deficiency, "without laboratory evidence of HIV infection" and, extraordinarily, "*in the presence of negative results for HIV infection*"<sup>109</sup> (italics ours).

### Sexual transmission

HIV/AIDS is claimed to be bidirectionally sexually transmitted. Data to support this claim are based not upon microbial isolation and contact tracing, as is the orthodox practice for proving diseases are infectious and sexually transmitted, but on mostly retrospective studies of highly selected groups of individuals - including

homosexual and bisexual men, heterosexual men and women including prostitutes - for antibodies in blood which react with certain proteins deemed "HIV-specific". Included in these studies are estimations of risk factors for the specific sexual practices of penile-insertive, vaginal, anal-receptive and oral-receptive intercourse.

## Homosexual men

In 1984, Gallo and his colleagues showed that "Of eight different sexual acts, a positive HIV antibody test correlated only with receptive anal intercourse".<sup>[110](#)</sup> They also found that the more often a homosexual man had insertive anal intercourse, the less likely he was to become HIV-positive. This is incompatible with an infectious cause. In 1986, Gallo and his colleagues reported they "found no evidence that other forms of sexual activity contribute to the risk" of HIV seroconversion in homosexual men.<sup>[111](#)</sup>

In an extensive review of 25 studies of homosexual men reported in 1994 by Caceres and van Griensven, the authors concluded that "no or no consistent risk of the acquisition of HIV-1 infection has been reported regarding insertive intercourse".<sup>[112](#)</sup>

In the West, the largest and most judiciously conducted prospective epidemiological studies, such as the Multicenter AIDS Cohort Study (MACS) of 4,954 gay men,<sup>[113](#)</sup> have proven beyond all reasonable doubt that in homosexual men the only significant sexual act related to becoming HIV-antibody-positive is receptive anal intercourse. Thus, in gay men, AIDS may be likened to the non-infectious condition, pregnancy. It is acquired by the passive partner but is not transmitted to the active partner.

Significantly, the MACS also showed that once a homosexual man becomes HIV-positive, progression to AIDS is further determined by the amount of passive anal intercourse sustained after "infection". This is contrary to all that is known about infectious diseases. Infection, not repeated infections, causes disease. Indeed, the Royal Australasian College of Surgeons (RACS) considers HIV-positive surgeons to be "infectious" and that they "should not perform invasive procedures or operations", but "they may provide these services to patients who have the same infection".<sup>[114](#)</sup>

## Heterosexuals

The largest and best-conducted studies in heterosexuals, including the European Study Group,<sup>[115](#)</sup> showed that, for women, the only sexual practice leading to an increased risk of becoming HIV-antibody-positive is anal intercourse. The unidirectional transmission of "HIV" observed in OECD countries is supported by Nancy Padian's 10-year study of heterosexual couples (1986-1996). There were two parts to this study: one cross-sectional, the other prospective.

In the cross-sectional study, "The constant per-contact infectivity for male-to-female transmission was estimated to be 0.0009 [1 in 1,111]". The risk factors for the women were: (i) anal intercourse; (ii) having partners who acquired this infection through drug use (Padian says this means the women may also be IV drug users); (iii) the presence of STDs (antibodies to their causative agents may react in an "HIV" antibody test).<sup>[15](#), [20](#)</sup> Of the HIV-negative male partners of 82 HIV-positive female cases, only two became HIV-positive - but under circumstances that Padian considered ambiguous.

In the prospective study, starting in 1990, 175 HIV-discordant couples were followed for approximately 282 couple-years. At entry to the study, one third used condoms consistently and, in the six months prior to their last follow-up visit, 26 per cent of couples consistently failed to use condoms. There were no seroconversions after entry, including the 47 couples not using condoms consistently. Based on the 2 in 86 men who became HIV-positive in the early study, the risk to a non-infected male from his HIV-positive female partner was reported to be in the order of 1 in 9,000 per contact. From this statistic one can calculate that, on average, a male would need to have 6,000 sexual contacts with an infected female to achieve a 50 per cent chance of becoming HIV-

positive. If sexual intercourse were to commence at age 20 and average three times weekly, this would occupy a lifetime.[57](#), [116](#)

## Female Prostitutes

The notion that HIV is a virus which "does not discriminate" is also markedly inconsistent with the data obtained from studies of female prostitutes. Even if by some unknown means a sexually transmitted infectious agent found its way into the promiscuous portion of the gay male population in certain large cities in the United States in the late 1970s (as is widely accepted), and given the facts that prostitutes are frequented by bisexual men and that, at the very earliest, "safe" sexual practices date from 1985, one would have expected HIV/AIDS to have spread rapidly through prostitutes and thence to the general community. However, the prevalence of "HIV" antibodies amongst prostitutes is almost entirely confined to those who are drug users. Virtually all other prostitutes have not been, and are not becoming, HIV-positive.

In September 1985, 56 non-intravenous drug using (IVDU) prostitutes were tested "...in the rue Saint-Denis, the most notorious street in Paris for prostitution. More than a thousand prostitutes work in this area... These women, aged 18-60, have sexual intercourse 15-25 times daily and do not routinely use protection." None was positive.[118](#)

In Copenhagen, 101 non-IVDU prostitutes, a quarter of whom "suspected that up to one fifth of their clients were homosexual or bisexual", were tested during August-October 1985. The median numbers of sexual encounters per week was twenty. None was positive.[118](#)

In 1985, 132 prostitutes (and 55 non-prostitutes) who attended a Sydney STD clinic were tested for HIV antibodies. The average number of sexual partners (clients and lovers) in the previous month was 24.5. When an estimate was made to separate clients and lovers, the median number of sexual contacts per year rose from 175 to 450. The partners of only 14 prostitutes (11%) used condoms at all, and 49% of their partners used condoms in fewer than 20% of encounters. No women were HIV-positive.[119](#)

The same Australian clinic repeatedly tested an additional 491 prostitutes who attended between 1986 and 1988. Of 231 out of the 491 prostitutes surveyed, 19% "had bisexual non-paying partners and 21% had partners who injected drugs. Sixty-nine per cent always used condoms for vaginal intercourse with paying clients, but they were rarely used with non-paying partners. Condoms were rarely used by those clients and/or partners for the 18% of prostitutes practising anal intercourse." No women were HIV-positive.

At the time of this report, a decade into the AIDS era, the authors commented that *"there has been no documented case of a female prostitute in Australia becoming infected with HIV through sexual intercourse"* (italics ours). Yet, these investigators from the Sydney Sexual Health Centre concluded that "there are still many women working as prostitutes in Sydney who remain seriously at risk of HIV infection".<sup>120</sup>

In Spain, of 519 non-IVDU prostitutes tested between May 1989 and December 1990, only 12 (2.3%) had a positive test, which was "only slightly higher than that reported five years ago in similar surveys". Some prostitutes had as many as 600 partners a month, and the development of a positive antibody test was directly related to the practice of anal intercourse. The authors also noted that "a more striking and disappointing finding was the low proportion of prostitutes who used condoms at all times, despite the several mass-media AIDS prevention campaigns that have been carried out in Spain".[121](#)

Similar data from two Scottish studies,<sup>122</sup> the 1993 European Working Group on HIV Infection in Female Prostitutes study,<sup>123</sup> and a 1994 report on 53,903 prostitutes working in the Philippines and tested between 1985 to 1992, confirm that non-IVDU prostitutes remain virtually devoid of HIV infection. For example, in the latter study, only 72 women (0.01%) were found to be HIV-positive.

In studies where there appears to be a high incidence of HIV amongst prostitutes, there are uncertainties that defy explanation. For example, although "HIV has been present in the commercial sex work networks in the Philippines and Indonesia for almost as long as it has been in Thailand and Cambodia", the prevalence of HIV in the former is 0.13% and 0.02% respectively and 18.8% and 40% in the latter.<sup>124</sup>

If these are accurate data, the discrepancy defies epidemiological explanation and has indeed baffled the experts, although the latter postulate "behavioural factors", such as one country's prostitutes and clients being considerably more or less sexually active than another. However, one could also pose another question. What are the "HIV" antibody tests actually measuring? Be that as it may, since 5,674 (44%) and 4,360 (34%) of the 12,785 Cambodian "HIV and AIDS Case Reports" till 31 December 1997 are listed as "Unknown" in gender and age respectively,<sup>125</sup> data collection, at least by the World Health Organization in Cambodia, must be regarded as problematic.


## Contradictions

Why should HIV avoid non-drug-using prostitutes? If female prostitutes who do not use drugs do not become HIV-infected despite being "seriously at risk of HIV infection", what is the risk of infection to the majority of Australian women who are neither drug users nor prostitutes? According to data from the National Centre in HIV Epidemiology and Clinical Research, vanishingly little. A 1989 study testing 10,217 blood samples of newborn babies (unambiguous evidence of unprotected heterosexual intercourse) found no babies and thus, presumably, no mothers HIV-positive.<sup>126</sup> If such women remain non-infected, how do their non-drug-using, male heterosexual partners become infected with HIV?

According to Simon Wain-Hobson, a leading HIV expert from the Pasteur Institute, "a virus's job" is to spread. "If you don't spread, you're dead". The "overwhelming" evidence from studies both in homosexual men and heterosexuals is that HIV/AIDS is not bidirectionally sexually transmitted. In the whole history of medicine there has never been such a phenomenon. Since microbes rely on person-to-person spread for their survival, it is impossible to claim from epidemiological data that HIV/AIDS is an infectious, sexually transmitted disease. Indeed, Professor Stuart Brody, from the University of Tübingen, has argued that physicians ignore the actual heterosexual data and instead promote the politically correct idea that everyone is at risk. "Ideological knowledge about AIDS is far more likely to filter through society than scientific knowledge."<sup>37</sup>

### THE HIV WESTERN BLOT TEST

The HIV Western blot test consists of a thin nitrocellulose strip in which are embedded proteins claimed to be unique to HIV. Each protein is labelled with a "p" followed by its molecular weight in thousands. Serum is added to the strip and, if there are antibodies to a particular protein, this band will "light up". The HIV Western blot is not standardised, and thus, around the world, different combinations of bands are considered positive. Hence a positive test in one country is not positive in another. An African would not be positive in Australia. A person from the MACS would not be positive anywhere in the world, including Africa. Yet the HIV Western blot is considered to be highly specific and is considered synonymous with HIV infection.



|      | AFR | AUS | FDA | RCX | CDC 1 | CDC 2 | CON  | GER | UK  | FRA | MAC |
|------|-----|-----|-----|-----|-------|-------|------|-----|-----|-----|-----|
| p160 |     |     |     |     | p160  | p160  | p160 |     |     |     |     |
| p120 | ANY | ANY | ANY | ANY | p120  | p120  | p120 | ANY | ANY | ANY | ANY |
| p41  |     |     |     |     | p41   | p41   | p41  |     |     |     |     |
| p68  |     |     |     |     |       |       |      |     |     |     |     |
| p53  |     |     | p53 | ANY |       |       | p53  |     | p53 | ANY |     |
| p32  |     |     | ANY | ANY | ANY   | OR    | ANY  | OR  | ANY | OR  |     |
| p55  |     |     |     |     |       |       |      |     |     |     |     |
| p40  |     |     |     |     |       |       |      |     |     |     |     |
| p24  |     |     | p24 | ANY | p24   | p24   | ANY  |     | p24 | ANY |     |
| p18  |     |     |     |     |       |       |      |     |     |     |     |

**Key:** AFR = Africa;<sup>1</sup> AUS = Australia;<sup>2</sup> FDA = US Food and Drug Administration;<sup>3</sup> RCX = US Red Cross;<sup>3</sup> CDC = US Centers for Disease Control;<sup>3</sup> CON = US Consortium for Retrovirus Serology Standardization;<sup>3</sup> GER = Germany; UK = United Kingdom; FRA = France; MAC = US Multicenter AIDS Cohort Study 1983-1992

According to data presented in Lundberg et al.,<sup>3</sup> when the US FDA criteria are used to interpret the HIV Western blot, less than 50% of US AIDS patients are HIV-positive, whereas 10% of persons not at risk of AIDS are also HIV-positive by the same criteria.

**Note:** In February 1993, the US FDA relaxed its stringent criteria in order to "reduce the number of HIV-1 seroindeterminate Western blot interpretations"; that is, to increase the number of HIV-positive individuals.<sup>4</sup>

### Endnotes

1. WHO (1990), "Acquired Immunodeficiency Syndrome (AIDS). Proposed criteria for interpreting results from Western blot assays for HIV-1, HIV-2 and HTLV-I/HTLV-II", Weekly Epidem. Rec 65:281-298.
2. Healy, D.S., Maskill, W.J., Howard, T.S. et al. (1992), "HIV-1 Western blot: development and assessment of testing to resolve indeterminate reactivity", AIDS 6:629-633.
3. Lundberg, G.D. (1988), "Serological Diagnosis of Human Immunodeficiency Virus Infection by Western Blot Testing", JAMA 260:674-679.
4. Kleinman, S., Busch, M.P., Hall, L. et al. (1998), "False-positive HIV-1 test results in a low-risk screening setting of voluntary blood donation", JAMA 280:1080-1083.

## THE DIAGNOSIS OF "HIV" INFECTION

### The HIV antibody tests

There are two "HIV" antibody tests in common use: the ELISA and Western blot (WB). The ELISA causes a colour change when a mixture of "HIV" proteins reacts with antibodies in serum from a patient. In the WB, the "HIV" proteins are first separated along the length of a nitrocellulose strip. This enables individual reactions to the 10 or so "HIV" proteins to be visualised as a series of darkened "bands". The Western blot test is used to "confirm" repeatedly positive ELISAs because experts agree that the ELISA "overreacts"; that is, it is insufficiently specific.<sup>x</sup>

Prior to 1987, one "HIV-specific" WB band was considered proof of HIV infection. However, since 15%-25% of healthy, no-risk individuals have "HIV-specific" WB bands,<sup>127, 128</sup> it became necessary to redefine a positive WB by adding extra and selecting particular bands, otherwise at least one in every seven people would be



diagnosed as infected with HIV. (Notwithstanding, in the MACS, one band remained proof of HIV infection in homosexual men until 1990.<sup>129</sup>) On the other hand, although AIDS in Europe and the US began to decline in 1987,<sup>130, 131</sup> this trend was countered by the addition of more and more diseases and, most recently, mere laboratory abnormalities<sup>132</sup> to each revision (1985, 1987, 1993) of the first, 1982 CDC definition.

The net effect of these changes was to maintain a correlation between "HIV" antibodies and "AIDS" amongst the "risk" groups, while the risk of an HIV/AIDS diagnosis outside these groups remained slight. This was further accentuated by avoiding testing outside the risk groups. However, when such studies were performed amongst 89,547 anonymously tested blood specimens from 26 US hospital patients meticulously chosen to be at no risk of AIDS, 0.7%-21.7% of men and 0.0%-7.8% of women aged 25 to 44 years were found to be HIV-WB-positive.<sup>133</sup> (It is estimated that approximately 1% of men are homosexual. Also, at the five hospitals with the highest rates of HIV antibodies, one third of positive tests were in women. Yet men vastly outnumber women as AIDS patients.)

In addition, the US Consortium for Retrovirus Serology Standardization reported that 127 (10%) of 1,306 individuals at "low risk" for AIDS, including "specimens from blood donor centers", had a positive HIV antibody test by the "most stringent" US WB criteria.<sup>127</sup>

Thus the correlation of "HIV" antibodies with AIDS - which experts accept as the only in vivo proof that HIV causes AIDS - is not a statistic related to the natural, unbridled activity of a virus, but is instead a contrivance generated by mankind. Not only does correlation never prove causation, the artificiality of this particular "correlation" severely compromises its scientific analysis.

One of the most bizarre aspects of the HIV/AIDS theory is that different laboratories, institutions and countries define different sets of WB bands as a positive test (see chart on previous page). The global variation in interpretive criteria means that in Australia, for example, a positive test requires particular sets of four bands. In the USA, different sets of two or three suffice, which may or may not include the bands required in Australia. In Africa, only one designated set of two is required. Put simply, this means that the same person tested in three cities on the same day may or may not be HIV-infected.

If the diagnosis of HIV infection were a game of poker, a flush would require five cards the same suit in one country, but only one or two elsewhere. A virus cannot behave in this manner, but according to the HIV test, which is claimed to have a specificity of 99.999%,<sup>134</sup> it does. As incomprehensible as this appears, further difficulties remain. For example, an Australian tested in Australia with one or two "HIV-specific" bands would not be reported as HIV-infected.<sup>109</sup> Clearly, however, there must be a reason why an uninfected individual, such as a healthy blood donor or military recruit, can possess any, even one, "HIV-specific" band. According to the experts, these bands are caused by cross-reacting, that is, "false", "non-HIV" antibodies which react with the "HIV" proteins. Thus it is axiomatic that an antibody which reacts with a particular protein is not necessarily an antibody which the immune system has generated specifically in response to that protein.

The Australian National HIV Reference Laboratory (NRL) concedes that "False reactivity may be to one or more [HIV] protein bands and is common (20%-25% of anti-HIV-negative blood donors [will] exhibit one or more bands on a WB)".<sup>128</sup> However, Eleopoulos argues that if "non-HIV" antibodies cause "one or more protein bands", then why are they not able to cause four or five? Or all ten? On what basis do experts assert which antibodies are "false" and which are "true"? Or, how do the same three bands, caused by "false", non-"HIV" antibodies, become "true" when accompanied by one extra? On what basis do experts assert there are any "true" HIV antibodies? If the Australian traveller were to be tested in the USA, where two or three bands are sufficient to diagnose HIV infection, are his antibodies "false" in Australia but "true" as his aeroplane touches down in Los Angeles?

In 1994, one of us (VFT) wrote to the *Medical Journal of Australia*, seeking justification of both the Australian criteria for a positive Western blot test and the global variability.<sup>28</sup> The response by Dr Elizabeth Dax of the NRL<sup>135</sup> did not answer either question, and subsequent correspondence failed to pass the editorial staff at the

same journal. When the same questions were later put via the offices of Senator Chris Ellison, the first question was again unanswered, and the widely different criteria between Australia and Africa were justified on the basis that, in Africa, "comparatively false reactivity is far less common [than in Australia] so that interpretation criteria to define [true] positivity may be less strict".<sup>128</sup> However, no scientist can make such a claim without data.

All antibody tests are subject to the vagaries of cross-reactions, and the only way to calculate the incidences of "true" and "false" antibodies is to scrutinise reactions against what the test is purportedly meant to measure, that is, against HIV itself.

HIV isolation is the only "gold standard" by which the specificity of the antibodies can be determined, and this must be evaluated before the test is introduced into clinical practice.

However, despite the WB test being in widespread use and "a stalwart"<sup>135</sup> of HIV testing, these data have never been reported by the NRL or any other laboratory. Even without such evidence - since (a) the NRL concedes that cross-reacting antibodies cause misleading reactions in the WB in one quarter of healthy Australians, and (b) unlike Australians, Africans (similar to the AIDS risk groups) are exposed to a multitude of infectious agents producing myriad antibodies, each capable of cross-reactions - "false reactivity" will be much higher in Africa where the WB criteria should be the most stringent. If "HIV" antibodies indeed prove that one third of heterosexual adults in certain central and eastern African countries are infected with HIV, "life in these countries must be one endless orgy".<sup>39</sup>

If the proteins used in the HIV ELISA and WB tests are unique constituents of an exogenous retrovirus, and if such a virus induces specific antibodies, we would never expect to find such antibodies in the absence of HIV. Yet, in addition to the circumstances above, there are numerous others where antibodies which react with the "HIV-specific" proteins arise where HIV/AIDS experts concede there is no HIV. These include healthy mice injected with lymphocytes of similar mice<sup>136</sup> or bacterial extracts (V. Colizzi et al., personal communication); following the transfusions of HIV-free blood<sup>137</sup> or a person's own irradiated blood<sup>138</sup>; and 72 out of 144 dogs tested at a veterinary clinic in Davis, California, USA.<sup>139</sup> In addition, antibodies to the microbes which cause the fungal and mycobacterial diseases affecting 90% of AIDS patients react with the "HIV-specific" proteins.<sup>20, 140</sup>

This year it was reported that 35% of patients with primary biliary cirrhosis, 39% of patients with other biliary disorders, 29% of those with lupus, 60% of patients with hepatitis B, 35% with hepatitis C - all non-HIV, non-AIDS diseases - have antibodies to the "HIV" p24 "core" protein.<sup>141</sup> Until 1990, an unknown number of the 4,954 homosexual men in the MACS were diagnosed HIV-infected on the basis of an antibody to the "HIV-specific" p24 protein, that is, with one WB band. Why do not all similar tests prove infection with HIV? Why are gay men with a single p24 band infected with a deadly virus, while biliary and liver disease patients with the same band are not? Why were the criteria for diagnosing HIV infection set less rigorously for homosexual men and Africans? And if HIV antibodies are specific and HIV infection is "for life", why do reformed drug addicts, leading healthy lives, lose their HIV anti-bodies?<sup>142</sup>

Although all HIV experts accept cross-reactivity in HIV antibody testing, in 1993 the New South Wales Department of Health interpreted the discovery of "HIV" antibodies in four women as "compelling evidence" for transmission of HIV from a homosexual man during the course of minor, office surgery in 1989.<sup>143</sup> However, there was no proof that the man was HIV-infected at the time of surgery, or that any of the four women were operated on after the man.

This report remains the only one of its kind in the world, and it immediately led to the establishment of a special committee of the Royal Australasian College of Surgeons which wrote to all College Fellows, inviting submissions upon the matter. But, rather than seizing upon the rarity of the event and following advice urging a formal scientific enquiry into whether "HIV" antibodies are caused by infection with a retrovirus,<sup>144</sup> the College accepted these data as proof of cross-infection but concluded, "The mode of transmission is unknown".<sup>114 xx</sup>

Unlike HIV/AIDS experts, who claim the specificity of the HIV antibody test is 99.999%, one manufacturer of HIV antibody tests states in the package insert: "At present there is no recognized standard for establishing the presence or absence of HIV-1 antibody in human blood. Therefore sensitivity was computed on the clinical diagnosis of AIDS, and specificity based on random [healthy blood] donors..."<sup>145</sup> The latter were chosen as de facto non-HIV-infected for the purposes of determining how many tests are false positives. However, by this "reasoning", since the majority of HIV-positive individuals are healthy, they cannot be infected. Thus the WHO<sup>146</sup> predictions of a global pandemic are patently untrue.

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## Endnotes:

× In most countries, including Australia, individuals with two positive ELISAs have HIV infection "confirmed" by performing a Western blot test. However, this testing algorithm selects individuals who have a higher rate of cross-reacting antibodies and are therefore more likely to react in the Western blot test. (This is analogous to determining the number of heart attacks in the community by performing ECGs only on patients with chest pain - an experiment which grossly underestimates the real number because many heart attacks are "silent".) On the other hand, England and Wales do not use the HIV WB to "confirm" reactive HIV ELISAs because Dr Philip Mortimer, Director of the UK Public Health Laboratory Service, claims that "truly positive" antibodies are "easily" detected "because these are reactive in all ["methodologically different machine-read" ELISA] assays".<sup>181</sup> (This reasoning is analogous to performing a chest X-ray with several machines and claiming a suspicious abnormality is lung cancer because the appearances are repeatedly the same.) Asked at the 1998 Geneva AIDS Conference to comment on the UK dropping the Western blot test, Gallo remarked: "Well, the bulk of the world uses it. If some technology comes across better, I'd be the first to say 'do it'. I mean, obviously, the Western blot's a valuable test as defining the proteins that you have antibodies to. Everybody uses it experimentally and most people use it around the world... Britain doesn't use it. Maybe there are two countries that have found a better way. God bless them. Okay?"

×× In 1997, the Perth group attempted a second time to engage the Royal Australasian College of Surgeons (RACS) in debating the HIV/AIDS controversy by submitting a paper entitled "A critical analysis of the evidence for the isolation of HIV" (see website <[www.virusmyth.com/aids/data/epappraisal.htm](http://www.virusmyth.com/aids/data/epappraisal.htm)>). It is RACS editorial policy to "welcome personal views of surgeons on a variety of topics" and to publish papers on "current and controversial issues". Although both reviewers accepted the bulk of the scientific arguments and found the paper "interesting reading", they advised against publication because, in their view, an analysis of evidence for the isolation of HIV was of "no real relevance...to a surgical audience" or "would be of little interest or use to the majority of readers of the Australian and New Zealand Journal of Surgery".

××× Of the cumulative 7,766 Australian AIDS cases to date, 387 (5%) are reported in the "Heterosexual contact" exposure category. However, 22 of these qualify on the basis of "Sex with injecting drug user", "Sex with bisexual male", "From high prevalence country" (where heterosexual spread is deemed dominant), "Sex with HIV-infected person, exposure not specified", or "Not further specified".<sup>177</sup> Thus, injecting drug use, anal intercourse in women, the presumption of any form of sexual intercourse, and lack of sufficient data, question the mode of acquiring HIV infection in at least 330 (85% of) individuals listed in this exposure category.

## Part 3

**When put to the test, conventional HIV/AIDS theory is at odds with the clinical evidence. Is "purified HIV" no more than a tangle of cellular debris?**

*Discovery consists of seeing what everybody has seen and thinking what nobody has thought.*

- Albert Szent-Györgyi, Physician and Nobel Laureate

## THE DIAGNOSIS OF "HIV" INFECTION

### What proof is there for the existence of HIV?

Scientific evidence for the existence of a retrovirus must be consistent with the definition of a retrovirus as a particular kind of replicating, microscopic particle. Thus researchers must demonstrate the correct size, shape and construction of particles; that these particles have been purified and analysed and contain RNA as well as an enzyme (reverse transcriptase) that makes DNA from RNA; and that the particles are infectious - that is, when pure particles are introduced into fresh cell cultures, identical progeny appear. The latter necessitates a second round of purification and analysis. Indeed, although this method is entirely logical and was deemed essential at a meeting held at the Pasteur Institute in 1973,<sup>[147](#),[148](#)</sup> it has been ignored by all HIV researchers.

Although there are electron microscope (EM) photographs from unpurified cell cultures of particles purported to be HIV particles, it was not until March 1997 that EMs of "purified HIV" were published.<sup>[149](#),[150](#)</sup> Yet such data is the first, most essential step in attempts to prove particles are a virus, and for subsequent extraction of constituents for analysis and use as diagnostic reagents. These long-awaited micrographs reveal "purified HIV" to be a tangle of cellular debris. Scattered amongst this are scant particles which, without evidence, the authors claim are the HIV particles and which "copurify" (sic) with the cellular material. Close examination of these particles as well as other evidence in the papers shows they are too large, wrongly shaped, have too high a mass, and are devoid of knobs that HIV experts unanimously assert are absolutely essential for the "HIV" particle to cause infection. It is from this material that HIV/AIDS experts and biotechnology companies obtain proteins and RNA to use in tests to pronounce humans infected with a unique, exogenous, AIDS-causing microbe.

On 17 July 1997, the French investigative television journalist Djamel Tahi interviewed Professor Luc Montagnier in camera at the Pasteur Institute in Paris. Montagnier was asked: "Why do the EM photographs published by you [in 1983] come from the culture and not the purification?" His reply was: "There was so little production of virus it was impossible to see what might be in a concentrate of the virus from the gradient ["pure virus"]. There was not enough virus to do that. Of course one looked for it, one looked for it in the tissues at the start; likewise the biopsy. We saw some particles but they did not have the morphology typical of retroviruses" [italics ours].<sup>[61](#)</sup> Questioned about the Gallo group, Montagnier replied: "Gallo? I don't know if he really purified. I don't believe so." This should have been both the beginning and the end of HIV.

Retrovirus-like particles are virtually ubiquitous in biological material,<sup>[151](#),[152](#)</sup> including, for example, cell cultures and "the majority if not all human placentas".<sup>153</sup> (Note that Montagnier refers to EMs obtained from umbilical-cord blood lymphocytes.) However, as Gallo confirms, the majority of retrovirus-like particles are not retroviruses because they do not replicate.<sup>[151](#),[154](#)</sup> The "HIV" particle has been "classified" into two subfamilies and three genera of retroviruses. This is analogous to describing a new species of mammal as a human, a gorilla and an orang-utan.

Besides the "HIV" particle, cell cultures contain other particles of numerous morphologies whose origin and role are unknown.<sup>[18](#) [155](#),[156](#)</sup> A long and detailed study from Harvard<sup>157</sup> revealed the identical "HIV" particle in 18 out of 20 (90% of) AIDS-related lymph node enlargements but also in 13 out of 15 (88% of) non-AIDS-related enlargements.

HIV experts claim to detect and even "isolate" HIV merely by demonstrating "reverse transcription" in cultures. However, although a property of retroviruses, reverse transcription is not, as many HIV/AIDS experts claim, unique to retroviruses or even viruses.<sup>[158](#),[159](#)</sup> Well before the AIDS era, Gallo himself showed that chemically stimulated (a technique absolutely essential to "isolate HIV" from cultures) normal lymphocytes possess this function.<sup>[160](#),[161](#)</sup>

### The "HIV" proteins and antibodies

Although both Montagnier and Gallo have never published EMs to prove the presence of retrovirus-like particles in their "pure virus", and Montagnier now concedes there weren't any, both groups, and all others since, claim



such material is "pure HIV". This claim is based on the fact that such material contains proteins which react with antibodies present in AIDS patients. However, such reasoning is untenable.

Imagine a scientist who mixes two solutions together, obtains a precipitate and then proclaims the identity and source of several reactants. One does not need a degree in chemistry to realise this is an impossibility. Nonetheless, because cultures and antibodies derived from AIDS patients react together, the proteins are declared to belong to "HIV" and the antibodies - the "HIV-specific" antibodies.

In fact, Gallo admits that, for him, an antibody test is the quintessence of "HIV isolation". During an interview at the 1998 Geneva AIDS Conference he admitted: "Sometimes we had Western blot positive but we couldn't isolate the virus. So we got worried and felt we were getting false positives sometimes, so we added the Western blot. That's all I can tell you. It was an experimental tool when we added it, and for us it worked well 'cos we could isolate the virus when we did it."<sup>162</sup> Actually, in 1984, Gallo's "false positives sometimes" were antibodies in 88% of AIDS patients but "virus-isolated" in 36% of AIDS patients. Gallo solved the twin dilemmas of the "missing virus" and gross non-specificity of the "HIV" antibodies by making the Western blot antibody test an integral part of virus isolation.

However, an antibody test is not isolation of a virus. HIV proteins can only be defined by extracting them from particles purified and proven to be a unique retrovirus. Such material has never been shown to exist, and such extraction never reported. Notwithstanding, since the mid-1980s, HIV researchers claim that a culture which reacts with a monoclonal antibody to one of the "HIV" proteins, the p24 protein, is proof of isolation of HIV. Since "to isolate a virus" is to obtain infectious particles separate from everything else, it is particularly difficult to see how so many scientists persevere in referring to a chemical reaction in this manner.

### **The origin of the "HIV" proteins**

According to Eleopulos and her colleagues, all data presented to date are consistent with the "HIV" proteins being cellular. Using "HIV" antibodies as probes, "HIV" proteins have been identified in the tissues of persistently HIV-negative, healthy individuals, including in blood platelet and skin cells, thymus, tonsil and brain.<sup>15</sup> As a mark of the bewildering status of the HIV theory, while HIV proteins could not be found in the placentas of 75 HIV-positive, pregnant women,<sup>163</sup> they could be found in the placentas of 25 healthy, HIV-negative women.<sup>164</sup>

That the HIV proteins are cellular is further strengthened by a recent two-part experiment. Human lymphocytes, cultured in the absence of material from AIDS patients, were "purified" as they would be to obtain the "HIV" proteins. This "uninfected" material served as a "mock virus" in experiments involving both "HIV" and "SIV" (simian [monkey] immunodeficiency virus, claimed similar to "HIV"). Analysis of "mock virus" reveals qualitatively a series of proteins bearing the same molecular weights as the proteins of "real" virus, strongly suggesting that the "HIV" proteins are cellular because the existence of HIV proteins demands they appear exclusively in cultures derived from AIDS patients.<sup>149</sup>

In the second experiment, monkeys were immunised on several occasions with "mock virus" - a procedure which subsequently protected them from a "challenge" with "real" SIV.<sup>165, 166</sup> However, immunisation is specific. Immunisation with hepatitis vaccine does not protect against poliomyelitis. It relies on exposure of the animal's immune system to material specific to the organism against which protection is sought. Since proteins from the cells in which "SIV" is "grown" ("mock" virus) protect against "real" SIV, these must be exceedingly similar if not identical. That is, the "SIV" and, by inference, the "HIV" proteins are all cellular.

### **The "HIV genome"**

As is the case with the "HIV" proteins, the RNA purported to be the "HIV" genome has not been obtained from particles purified and proven infectious, but from the conglomerate material described above. Molecular biologists have produced possibly more information about the "HIV" genome than any other object in the Universe. Nonetheless, there are no reports of even one individual possessing a complete, full-length "HIV" genome, and there is no agreement as to how many genes HIV possesses. Opinions have varied from four through to eight, nine or ten. Human DNA and chimpanzee DNA differ by less than 2%, but variation in the



composition of the "HIV genome" (derived from analysis of "pieces" measuring 2%-30% of the presumed total) measures between 3%-40%. For comparison, two RNA-containing viruses (polio and influenza, the latter after 27 years of dormancy) vary by less than 1%, as do RNA molecules self-assembled in test tubes, denied the organising influence of living cells.[167](#), [168](#)

Given that the DNA sequence determines the composition of a virus's proteins, and the latter the physical, biochemical and biological properties of a virus, how is it possible for such variation to represent one and the same agent? For example, how is it possible that HIV can induce the same antibodies which can be recognised in a universal antibody test containing the identical proteins? Since, as the molecular biologist Duesberg reminds us, "there is a range, a small range, in which you can mutate around without too much penalty, but as soon as you exceed it you are gone, and you are not HIV any longer, or a human any longer...then you are either dead or you are a monkey, or what have you",[8](#) it is evident that whatever the "HIV DNA genome" represents, it cannot be a virus.

If there exists certain RNA which is unique to a retrovirus HIV, then finding such RNA should prove infection. However, the concordance between antibody and genetic tests varies between 40%-100%,[169](#) and "HIV" RNA can be found in individuals not infected with HIV. In responding to this scientific dilemma, the HIV experts have proclaimed that "Plasma viral load ["HIV" RNA] assays are designed for monitoring the effectiveness of antiretroviral therapies and for measuring the quantity of virus in patients with confirmed HIV infection, not for the diagnosis of HIV infection. Their performance in patients who are not infected with HIV is unknown" and their use leads to "Misdiagnosis of HIV infection".[170](#) One manufacturer of PCR states that "The Amplicor HIV-1 Monitor test is not intended to be used as a screening test for HIV or as a diagnostic test to confirm the presence of HIV infection" (Roche Diagnostic Systems, 06/96, 13-08088-001).

These being the case, the specificity of "plasma viral load" is unknown and it is difficult if not impossible to claim that "HIV" RNA is the unique constituent of a specific retrovirus. How can one even consider using such tests to monitor or diagnose a supposedly deadly virus when the "viral load" obtained varies between zero and a million copies on the same sample, depending on which technique or strain of HIV is involved?

### **Lessons from the past?**

The evidence for the existence of Gallo's "first human" retrovirus (HL23V) was much stronger than that for HIV (see Part 1).[20](#), [25](#), [172](#) However, in 1980, the antibodies to the HL23V proteins were shown to occur following a large variety of common, non-infectious factors and in far more humans than could ever have developed leukaemia.[173](#), [174](#) Thus, from signifying that an "infectious mode of transmission [of leukaemia] remains a real possibility in humans" and "infection with an oncovirus [retrovirus] may be extremely widespread",[175](#) the "first" human retrovirus abruptly disappeared from the annals of science. At present no one, not even Gallo, believes it existed.

However, had it not been for the efforts of the two research groups at the National Cancer Institute and Sloan-Kettering, there was the distinct possibility that by now the world would be facing a pandemic of "HL23V disease" as well as a pandemic of "HIV disease".

In the AIDS era, experts recognise that antibodies to the "HIV-specific" proteins occur where there is no HIV and in many more individuals than will ever develop AIDS. On what basis, then, does "HIV" still exist?

## **THE DISSIDENT CASE: POLITICS AND PUBLIC HEALTH POLICY**

The failures of the past 15 years are fairly and squarely affixed to the five Montagnier and Gallo 1983-84 Science papers. That the titles of three of these papers contain the word "isolation", and yet no such evidence was presented, must stand as a memorial to the demise of editorial integrity. The dissident cases - that HIV does not exist (Eleopulos), or, if it does exist, it does not cause AIDS (Eleopulos/Duesberg) - ultimately imply there will be devastating outcomes in terms of scientific credibility, including the failure of peer review, the demise of reputations of many experts and non-experts, the challenge to citizens' trust in governmental, scientific and

medical leaders, as well as an uncertain period of ignominy for the medical profession as a whole. Weaving a just resolution through this maze of socio-medico-legal bedlam will require the utmost perspicacity and tenacity from political leaders.

Perhaps there are already signs of quiet beginnings with the Americans' 1994 return of the discovery of HIV to the French, followed by Montagnier's most recent admissions in his 1997 interview. Perhaps it is also written in the faces of the Nobel Committee and the stubborn absence of a Nobel Prize awarded for any of the 100,000 scientific papers representing HIV/AIDS research.

### **Exceptionalism**

Over and above all the uncertainties surrounding the HIV/AIDS debate, AIDS science/medicine must stand as the most remarkable case of "exceptionalism" in history. The funding it attracts far outstrips that justified by its prevalence and economic impact.<sup>176</sup> For example, over the past 17 years, Australia has a cumulative total of 7,766 AIDS cases including 5,575 deaths.<sup>177, xxx</sup>

The big spenders are (in order) the United States, France, the United Kingdom, Germany and Italy. Their combined annual HIV/AIDS research budget amounts to US\$1.8 billion for a cumulative total of 761,572 AIDS patients (many of whom are dead). Of an additional \$US20 million spent by the European Union in 1994-98, most "money goes to support travel and meeting costs rather than laboratory research".<sup>178</sup>

While thousands of dollars per patient are spent on HIV/AIDS research, only a few dollars are spent on heart disease, cancer, mental illness, suicide prevention or road trauma.

The funding paradox reaches epidemic, almost farcical proportions in developing countries where Western AIDS workers spend their days dispensing advice and condoms to a population dying for want of potable water, adequate sanitation and nutrition, and antibacterial, antitubercular and antimalarial medicines - in a word, dying of poverty.

Currently, the annual cost of anti-HIV drugs for one person is about \$US15,000 (greater than the entire health budget for many a Third World village). With 650,000 to 900,000 HIV-positive patients in the USA as of July 1996, it would take US\$10 billion to pay for drugs alone. This must be viewed against the World Health Organization's estimate that by the year 2000 there will be 30 to 40 million HIV-infected people.

Without HIV, AIDS patients and specialist AIDS units and their employees can rationally be absorbed into the existing infrastructure of clinics and hospitals. The pursuit of expensive drugs designed to kill HIV will be irrelevant, as will be the travail of the legions of HIV researchers. The same applies to AIDS councils, the armies of AIDS educators, fundraisers, volunteers and AIDS organisations. In the US alone, there are 93,000 of the latter - one for every four persons ever diagnosed with AIDS.<sup>34</sup>

### **Clear thinking**

Homo sapiens (thinking man) was not named in vain. An honourable society provides unfettered information and encourages its members to make rational choices. Epidemiology shows that the development of a positive "HIV" antibody test and AIDS is not so much related to a given sexual practice, but rather to the frequency of passive anal intercourse in both men and women.

It follows that AIDS is not a disease of sexual orientation, and as far as women are concerned it is prudent to note that, in absolute terms, innumerable more women than men engage in anal intercourse. Thus AIDS is not unlike the case of the recently appended AIDS-defining disease, cervical cancer, which long before the AIDS era was known to be related to the frequency of vaginal intercourse. Even so, it is not the act itself, but the very high frequency of the act, which is pathogenic.

As serious as public reaction to an ill-conceived retrovirus may prove, it will not be anywhere as serious as the legal backlash. There are countless individuals alive who believe they are infected with a deadly microbe, and many of them are currently treated with potentially toxic drugs with no proven benefit. They avoid intimacy, avoid having children, and sometimes avoid even casual contact with others. It would take a flotilla of poet

laureates to voice the collective pain and suffering engendered by such a mistake. It would take an army of mathematically gifted lawyers to quantify, and the nation's coffers to compensate, those whose lives have been ruined by what Neville Hodgkinson has called "the greatest scientific blunder of the 20th century".<sup>29</sup>

This is not to forget patients and relatives who have died at their own hands. In 1987, former US Senator Lawton Chiles of Florida told an AIDS conference of a tragic case where 22 blood donors were informed they were HIV-infected on the basis of an ELISA test. Seven donors then committed suicide.<sup>179</sup>

In June this year, the Swiss AIDS analyst Michael Baumgartner persuaded United Nations officials to include a dissident session at the XIIth International AIDS Conference held in Geneva. Speakers included: Huw Christie, editor of Continuum magazine; AIDS analyst and documentary film-maker Joan Shenton; epidemiologist Professor Gordon Stewart; retrovirologist and electron microscopist Professor Etienne de Harven; virologist Dr Stefan Lanka; and, by satellite, Eleni Eleopulos and her group from the Royal Perth Hospital. In the audience were observers from the Pasteur Institute and the US National Institutes for Health. The topic of the session was a scientific critique of the HIV antibody tests and the evidence for the existence of HIV.

At the official press conference held after the meeting, Professor Bernhard Hirschel, chairman of the organising committee, accused the speakers of "using outdated and untrustworthy scientific data". However, it was this "outdated" data, that of Montagnier and Gallo, that led to the 1984 proclamation that HIV is the cause of AIDS. That considered "untrustworthy" is the HIV experts' own data.

Notwithstanding these and many other challenges to the current dogma, HIV/AIDS experts are not in the least disquieted by sceptical patients, relatives or scientists, and inveigh heavily against inquisitive journalists alleging great harm to public health. Thus it appears that the only hope for an immediate resolution of this troubled issue is to have lawyers appearing for plaintiffs who desire judgements that they are, or are not, infected with an AIDS-causing virus. However, even if an examination of "HIV science" is destined to be scrutinised by courts of law, at present one must be realistic that in the short term the status quo is extremely unlikely to change.

### **A real debate?**

Nonetheless, it is inexorably drawing nearer to the time when world governments will convene an international, adjudicated debate on this subject. In contrast to the 13,775 participants from 177 countries who attended the June 1998 Geneva AIDS Conference, this should be a small gathering where a dozen or so experts from each side put their respective cases to a disinterested group of scientists of the utmost stature - for example, another dozen made up largely of Nobel laureates. There is a precedent for such a "consensus conference" or *conférence de citoyens* in common sense and "along the lines of a model invented in Scandinavia and since applied in the United Kingdom and elsewhere". A "jury" of 14 people "screened for independence from interested parties" would have issues "debated in front of them by scientists, non-governmental organizations, industrialists and other bodies", as "The power of public research bodies is probably the best guarantee of independence with respect to private sector research and the influence of multinationals".<sup>180</sup> By AIDS standards, funding for such a meeting would be trivial. Indeed, such would be its significance that it would make money for the organisers.

Perhaps a disinterested observer could be forgiven for concluding that, although we are now well into the 18th year of the AIDS era and have spent many billions of dollars on treatments and research, the words of Dr Peter Duesberg continue to taunt us: "By any measure, the war on AIDS has been a colossal failure...our leading scientists and policymakers cannot demonstrate that their efforts have saved a single life."<sup>1</sup>

Perhaps the words of Eleopulos's group are of even greater portent: "The single most important obstacle in finding the explanation for AIDS is the belief in HIV."<sup>19, 26</sup>

In his recent book, *Dancing Naked in the Mind Field*, Dr Kary Mullis writes: "Years from now, people will find our acceptance of the HIV theory of AIDS as silly as we find those who excommunicated Galileo."<sup>2</sup>

Indeed, it was Galileo who counselled: "In Science, the authority embodied in the opinion of thousands is not worth a spark of reason on one man."

Perhaps, seventeen years in, we should all pause, look around, and then take a long look back.

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## Endnotes:

\* US journalist Christine Johnson's interview (now available in six languages) with the leader of the Perth group was reviewed by scholar and international gay media personality Professor Camille Paglia in her column in the US Salon magazine (28 October 1997): "For a superb critique of the scandalously overpoliticized scientific research on AIDS, see Christine Johnson's long interview with Australian biophysicist Eleni Papadopoulos-Eleopoulos in the new issue of the British AIDS magazine, Continuum [vol. 5, no. 1, autumn 1997]. The American major media have effectively suppressed longstanding questions about whether the AIDS test is reliable or whether an HIV virus in fact exists at all."

\*\* On 5 May 1998, two US Republicans said they were exploring ways to give a comfortable retirement to 1,500 chimpanzees that were bred for AIDS research. Accompanied by primate expert Jane Goodall, House Speaker Newt Gingrich and Rep. Jim Greenwood (R-Penn.) said they were working on a bill to set up sanctuaries for the chimps. The chimps, bred in the United States specifically for AIDS research, did not turn out to be the effective models that scientists had anticipated. With no research use, the primates that are man's closest cousins are languishing in cages at an annual cost of US\$7.3 million.

\*\*\* In 1988, Eleopoulos' paper that HIV does not cause Kaposi's sarcoma was thrice rejected by the Medical Journal of Australia on the advice of an "established expert". The reviewer stated: "The author tries to argue that Kaposi's sarcoma cannot be caused by HIV infection, and that therefore AIDS is not due to HIV infection. The arguments put forward by the author are quite unsatisfactory, and are not supported by even a desultory reading of the literature quoted. In addition, the author fails to examine the body of epidemiological, immunological and cellular literature concerning the pathology, pathogenesis and clinical associations of this fascinating manifestation of HIV infection." Yet this is the very "epidemiological, immunological and cellular literature" which eventually led the "established experts" to accept that "this fascinating manifestation of HIV infection" is not caused by HIV infection.

xxx Of the cumulative 7,766 Australian AIDS cases to date, 387 (5%) are reported in the "Heterosexual contact" exposure category. However, 22 of these qualify on the basis of "Sex with injecting drug user", "Sex with bisexual male", "From high prevalence country" (where heterosexual spread is deemed dominant), "Sex with HIV-infected person, exposure not specified", or "Not further specified".<sup>177</sup> Thus, injecting drug use, anal intercourse in women, the presumption of any form of sexual intercourse, and lack of sufficient data question the mode of acquiring HIV infection in at least 330 (85% of) individuals listed in this exposure category.

1. Duesberg, P.H., *Inventing the AIDS Virus*, Regnery Publishing, Inc., Washington, USA, 1996.
2. Mullis, K.B., *Dancing Naked in the Mind Field*, Pantheon, 1998.
3. Duesberg, P.H. (1987), "Retroviruses as carcinogens and pathogens: Expectations and reality", *Cancer Res.* 47:1199-1220.
4. Duesberg, P.H. (1989), Human immunodeficiency virus and acquired immunodeficiency syndrome: correlation but not causation", *Proc. Natl Acad. Sci.* 86:755-764, USA.
5. Duesberg, P.H. (1992), "AIDS acquired by drug consumption and other noncontagious risk factors", *Pharmacol. Ther.* 55:201-277.
6. Duesberg, P.H. (1995), "Foreign-protein-mediated immunodeficiency in hemophiliacs with and without HIV infection", *Genetica* 95:51-70.
7. Duesberg, P. and Rasnick, D. (1997), "The drugs-AIDS hypothesis", *Continuum* 4:1s-24s.
8. Duesberg, P.H. (1996), "Peter Duesberg responds", *Continuum* 4:8-9.
9. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Hedland-Thomas, B., Causer, D., Page, B. (1995), "A critical analysis of the HIV-T4-cell-AIDS hypothesis", *Genetica* 95:5-24.
10. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Causer, D. (1995), "Factor VIII, HIV and AIDS in haemophiliacs: an analysis of their relationship", *Genetica* 95:25-50.



11. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Bialy, H. (1995), "AIDS in Africa: Distinguishing fact and fiction", *World J. Microbiol. Biotechnol.* 11:135-143.
12. Papadopoulos-Eleopoulos, E. (1988), "Reappraisal of AIDS: Is the oxidation caused by the risk factors the primary cause?", *Med. Hypotheses* 25:151-162.
13. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M. (1992), "Kaposi's sarcoma and HIV", *Med. Hypotheses* 39:22-9.
14. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M. (1992), "Oxidative stress, HIV and AIDS", *Res. Immunol.* 143:145-8.
15. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M. (1993), "Is a positive Western blot proof of HIV infection?", *Bio/Technology* 11:696-707.
16. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M. (1993), "Has Gallo proven the role of HIV in AIDS?", *Emerg. Med. [Australia]* 5:113-123.
17. Papadopoulos-Eleopoulos, E., Turner, V.F., Causer, D.S., Papadimitriou, J.M. (1996), "HIV transmission by donor semen", *Lancet* 347:190-1.
18. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M. (1996), "Virus Challenge", *Continuum* 4:24-27.
19. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Causer, D. (1996), "The Isolation of HIV: Has it really been achieved?", *Continuum* 4:1s-24s.
20. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Causer, D. (1997), "HIV antibodies: Further questions and a plea for clarification", *Curr. Med. Res. Opinion* 13:627-634.
21. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Causer, D. (1997), "A critical analysis of the evidence for the isolation of HIV"; at website <[www.virusmyth.com/aids/data/epappraisal.htm](http://www.virusmyth.com/aids/data/epappraisal.htm)>.
22. Papadopoulos-Eleopoulos E., Turner, V.F., Papadimitriou, J.M., Causer, D. (1995), "A reply to Wei and Ho"; at website <[www.virusmyth.com/aids/perthgroup/](http://www.virusmyth.com/aids/perthgroup/)>.
23. Papadopoulos-Eleopoulos, E. (1982), "A Mitotic Theory", *J. Theor. Biol.* 96:741-758.
24. Papadopoulos-Eleopoulos, E., Turner, V.F. (1994), "Deconstructing AIDS in Africa", *The Independent Monthly* 50-51.
25. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Causer, D., Page, B. (1998), "HIV Antibody Tests and Viral Load: More Unanswered Questions and a Further Plea for Clarification", *Curr. Med. Res. Opinion* 14:185-186.
26. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Causer, D. (1997), "Why no whole virus?", *Continuum* 4:27-30.
27. de Harven, E. (1997), "Pioneer deplores 'HIV'", *Continuum* 5:24.
28. Turner, V.F. (1994), "The HIV Western blot", *Med. J. Aust.* 160:807-808.
29. Hodgkinson, N., *AIDS: The Failure of Contemporary Science*, Fourth Estate, London, 1996.
30. Callen, M., *Surviving AIDS*, HarperCollins Publishers, 1991.
31. Johnson C. (1997), "Is HIV the cause of AIDS?" *Continuum* 5:8-19.
32. De Marchi, L. and Franchi, F., *AIDS: la grande truffa*, Edizioni SEAM, Rome, 1996.
33. Shenton, J., *Positively False*, I.B. Tauris, London, 1998.
34. Maggiore C., *What if everything you thought you knew about AIDS was wrong?*, HEAL, Los Angeles, 1997.
35. McDonald, J.F. (ed.), *Genetica*, Kluwer Academic Publishers, London, 1995.
36. Anonymous (1995), "Missing Virus: The Jody Wells Memorial Prize", *Continuum* 3:4.
37. Brody, S., *Sex at Risk: Lifetime Number of Partners, Frequency of Intercourse, and the Low AIDS Risk of Vaginal Intercourse*, Transaction Publishers, New Brunswick, NJ, 1997.
38. Caton, H., *The AIDS Mirage*, The University of New South Wales Press Ltd, Sydney, 1994.
39. Chirimuuta, R.C., Chirimuuta, R.J., *Aids, Africa and Racism*, R. Chirimuuta, Bretby House, Stanhope Bretby, Burton-on-Trent, UK, 1987, 1st ed.
40. Christie, H. (1995), "HIV Positive? It depends where you live", *Continuum* 3:21.
41. Christie, H. (1996), "Counterculture", *Continuum* 4:18-21.
42. Christie, H. (1997), "From Hype to Hesitation", *Continuum* 4:11-12.
43. Christie, H. (1998), "Wake the Law", *Continuum* 5:28-29.
44. Christie, H. (1998), "Do antibody tests prove HIV infection?", *Continuum* 5:10-19.
45. Cohen, J. (1994), "The Duesberg Phenomenon", *Science* 266:1642-1649.



46. Current, S. (1995), "HIV and AIDS: Causation or coercion?", *Provincetown Positive* 19-22.
47. Gildemeister, V. (1996), "Is Maddox mad or is he just pretending?", *Continuum* 4:6-7.
48. Hodgkinson, N., "Research disputes epidemic of Aids", *The Sunday Times*, London, 22 May 1994, p. 24.
49. Hodgkinson, N., "World AIDS Conference", *The European*, 22-28 June 1998, pp. 30-31.
50. Konotey-Ahulu (1987), "AIDS in Africa: Misinformation and Disinformation", *Lancet* ii:206-207.
51. Koliadin, V.L. (1995), "Critical analysis of the current views on the nature of AIDS", *Genetica* 95:71-90.
52. Koliadin, V.L., "Some facts behind the expansion of the definition of AIDS in 1993", personal communication, 1997.
53. Lauritsen, J., *The AIDS War*, Asklepois Press, New York City, 1993.
54. Lauritsen, J. (1994), "Gallo admits...we have never found HIV DNA in Kaposi's sarcoma", *Continuum* 2:4.
55. Lauritsen, J. (1994), "NIDA Meeting Calls for Research into the Poppers-Kaposi's Sarcoma Connection", *The New York Native*, at website <[www.virusmyth.com/aids/data/jlpoppers.htm](http://www.virusmyth.com/aids/data/jlpoppers.htm)>.
56. Lauritsen, J., *Poison by Prescription: The AZT Story*, Asklepois Press, New York, 1990.
57. Padian, N. and Pickering, J., "Female-to-male transmission of AIDS: a re-examination of the African sex ratio of cases", *JAMA* 256:590.
58. Root-Bernstein, R.S., *Rethinking AIDS: The Tragic Cost of Premature Consensus*, Macmillan, New York, 1993.
59. Root-Bernstein, R.S. (1995), "Five myths about AIDS that have miscredited research and treatment", *Genetica* 95:111-132.
60. Mullis, K.B. (1995), "A hypothetical disease of the immune system that may bear some relation to the Acquired Immune Deficiency Syndrome", *Genetica* 95:195-197.
61. Tahi, D. (1998), "Did Luc Montagnier discover HIV?", text of video interview with Professor Luc Montagnier at the Pasteur Institute, 18 July 1997, *Continuum* 5:30-34.
62. Turner, V.F. (1990), "Reducing agents and AIDS: Why are we waiting?", *Med. J. Aust.* 153:502.
63. Turner, V.F. (1998), "Where we have gone wrong?", *Continuum* 5:38-44.
64. Rizza, C.R., Spooner, R.J.D. (1983), "Treatment of haemophilia and related disorders in Britain and Northern Ireland during 1976-80: Report on behalf of the directors of haemophilia centres in the United Kingdom", *Br. Med. J.* 286:929-933.
65. Johnson, R.E., Lawrence, D.N., Evatt, B.L., et al. (1985), "Acquired immunodeficiency syndrome among patients attending hemophilia treatment centers and mortality experience of hemophiliacs in the United States", *Am. J. Epidemiol.* 121:797-810.
66. Barré-Sinoussi, F., Chermann, J.C., Rey, F. et al. (1983), "Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS)", *Science* 220:868-71.
67. Gallo, R.C., Sarin, P.S., Kramarsky, B., Salahuddin, Z., Markham, P., Popovic, M. (1986), "First isolation of HTLV-III", *Nature* 321:119.
68. Gallo, R.C., Sarin, P.S., Gelmann, E.P. (1983), "Isolation of Human T-Cell Leukemia Virus in Acquired Immune Deficiency Syndrome (AIDS)", *Science* 220:865-867.
69. Popovic, M., Sarngadharan, M.G., Read, E., Gallo, R.C. (1984), "Detection, Isolation, and Continuous Production of Cytopathic Retroviruses (HTLV-III) from Patients with AIDS and Pre-AIDS", *Science* 224:497-500.
70. Sarngadharan, M.G., Popovic, M., Bruch, L. (1984), "Antibodies Reactive to Human T-Lymphotropic Retroviruses (HTLV-III) in the Serum of Patients with AIDS", *Science* 224:506-508.
71. Schupbach, J., Popovic, M., Gilden, R.V., Gonda, M.A., Sarngadharan, M.G., Gallo, R.C. (1984), "Serological analysis of a Subgroup of Human T-Lymphotropic Retroviruses (HTLV-III) Associated with AIDS", *Science* 224:503-505.
72. World Health Organization (1994), "HIV type 1 variation in World Health Organization-sponsored vaccine evaluation sites: genetic screening, sequence analysis, and preliminary biological characterization of selected viral strains", *AIDS Res. Hum. Retroviruses* 10:1327-1343.
73. Butler, D. (1995), "US accused of 'cover up' in defence of Gallo claims", *Nature* 373:372.
74. Temin, H.M. (1974), "On the origin of RNA tumor viruses", *Harvey Lectures* 69:173-197.
75. Weiss, R.A., Friis, R.R., Katz, E., Vogt, P.K. (1971), "Induction of avian tumor viruses in normal cells by physical and chemical carcinogens", *Virology* 46:920-938.
76. Lower, R., Lower, J., Kurth, R. (1996), "The viruses in all of us: Characteristics and biological significance of human endogenous retrovirus sequences", *Proc. Natl Acad. Sci. USA* 93:5177-5184.

77. Beral, V.D., Bull, R., Darby, S. (1990), "Kaposi's sarcoma and sexual practices associated with faecal contact in homosexual or bisexual men with AIDS", *Lancet* 339:632-636.
78. Alizon, M., Sonigo, P., Barré-Sinoussi, P. et al. (1984), "Molecular cloning of lymphadenopathy-associated virus", *Nature* 312:757-760.
79. Montagnier, L. (1985), "Lymphadenopathy-Associated Virus: From Molecular Biology to Pathogenicity", *Ann. Int. Med.* 103:689-693.
80. Papadopoulos-Eleopoulos, E., Turner, V.F., "Reconstructing AIDS in Africa: Reply to Kaldor and Ashton", *The Independent Monthly*, February 1995, pp. 23-24.
81. Beral, V., Peterman, T.A., Berkelman, R.L., Jaffe, H.W. (1990), "Kaposi's sarcoma among persons with AIDS: a sexually transmitted infection?", *Lancet* 335:123-128.
82. Buhl, R., Jaffe, H.A., Holroyd, K.J. et al. (1989), "Systemic glutathione deficiency in symptom-free HIV-seropositive individuals", *Lancet* 2:1294-8.
83. Eck, H.P., Gmunder, H., Hartmann, M., Petzoldt, D., Daniel, V., Droge, W. (1989), "Low concentrations of acid-soluble thiol (cysteine) in the blood plasma of HIV-1-infected patients", *Biol. Chem.* 370:101-8, Hoppe-Seyler.
84. Herzenberg, L.A., De Rosa, S.C., Dubs, J.G. et al. (1997), "Glutathione deficiency is associated with impaired survival in HIV disease", *Proc. Natl Acad. Sci. USA* 94:1967-72.
85. Montagnier, L., Olivier, R., Pasquier, C. (eds), *Oxidative Stress in Cancer, AIDS and Neurodegenerative Diseases*, Marcel Dekker, Inc., New York, 1998.
86. Zagury, D., Bernard, J., Leonard, R. et al. (1986), "Long-Term Cultures of HTLV-III-Infected T Cells: A Model of Cytopathology of T-Cell Depletion in AIDS", *Science* 231:850-853.
87. Rosenberg, Y.J., Anderson, A.O., Pabst, R. (1998), "HIV-induced decline in blood CD4/CD8 ratios: viral killing or altered lymphocyte trafficking?" *Immunol. Today* 19:10-7.
88. Grossman, Z., Feinberg, M.B., Paul, W.E. (1998), "Multiple modes of cellular activation and virus transmission in HIV infection: A role for chronically and latently infected cells in sustaining viral replication", *Proc. Natl. Acad. Sci. USA* 95:6314-6319.
89. Poon, B., Grovit-Ferbas, K., Stewart, S.A., Chen, I.S.Y. (1998), "Cell cycle arrest by vpr in HIV-1 virions and insensitivity to antiretroviral agents", *Science* 281:266-269.
90. Ter-Grigoryan, V.S., Krifuks, O., Liubashevsky, E., Nyska, A., Trainin, Z., Toder, V. (1997), "A new transmissible AIDS-like disease in mice induced by alloimmune stimuli", *Nat. Med.* 3:37-41.
91. Papadopoulos-Eleopoulos, E., Turner, V.F., Papadimitriou, J.M., Causer, D., Alphonso, H., Miller, T. (1999), "A critical analysis of the pharmacology of AZT and its use in AIDS", *Curr. Med. Res. Opinion* 15; in press.
92. Concorde Coordinating Committee (1994), "Concorde: MRC/ANRS randomised double-blind controlled trial of immediate and deferred zidovudine in symptom-free HIV infection", *Lancet* 343:871-81.
93. Heresi, G.P., Caceres, E., Atkins, J.T., Reuben, J., Doyle, M. (1997), "Pneumocystis carinii pneumonia in infants who were exposed to human immunodeficiency virus but were not infected: an exception to the AIDS surveillance case definition", *Clin. Infect. Dis.* 25:739-40.
94. Abrams, D., "Lecture to Medical Students", *Synapse*, 1996.
95. Buchbinder, S.P., Katz, M.H., Hessol, N.A., O'Malley, O.M., Holmberg, S.D. (1994), "Long-term HIV-1 infection without immunologic progression", *AIDS* 8:1123-8.
96. Munoz, A., Kirby, A.J., He, Y.D. et al. (1995), "Long-term survivors with HIV-1 infection: incubation period and longitudinal patterns of CD4+ lymphocytes", *Journal of Acquired Immune Deficiency Syndromes & Human Retrovirology* 8:496-505.
97. Pantaleo, G., Menzo, S., Vaccarezza, M. et al. (1995), "Studies in subjects with long-term nonprogressive human immunodeficiency virus infection", *NEJM* 332:209-16; see comments.
98. Kilzer, L., "Optimistic AIDS reports beguiling at-risk men", *The Rocky Mountain News*, 3 May 1998, p. 3A.
99. O'Brien, W., Grovit-Ferbas, K., Namazi, A. et al. (1995), "Human immunodeficiency virus type 1 replication can be increased in peripheral blood of seropositive patients after influenza vaccination", *Blood* 86:1082-9.
100. Lee, T.H., Sheppard, H.W., Reis, M., Dondero, D., Osmond, D., Busch, M.P. (1994), "Circulating HIV-1-infected cell burden from seroconversion to AIDS: importance of postseroconversion viral load on disease course", *J. Acquir. Immun. Defic. Syndr.* 7:381-388.
101. Holodniy, M., Mole, L., Winters, M., Merigan, T.C. (1994), "Diurnal and short-term stability of HIV virus load as measured by gene amplification", *J. Acquir. Immun. Defic. Syndr.* 7:363-8.
102. Bruisten, S.M., Reiss, P., Loeliger, A.E. et al. (1998), "Cellular proviral HIV type 1 DNA load persists after

- long-term RT-inhibitor therapy in HIV type 1 infected persons", *AIDS Res. Hum. Retroviruses* 14:1053-1058.
103. Kaufmann, D., Pantaleo, G., Sudre, P., Telenti, A. (1998), "CD4-cell count in HIV-1-infected individuals remaining viraemic with highly active antiretroviral therapy (HAART)", *Lancet* 351:723-724.
104. Garrett, L. (1999), "The virus at the end of the world", *Esquire* 103-172.
105. Garrett, L., "AIDS, After The 'Cure': Amid setbacks, search for new hope", *Newsday*, Sunday 14 June 1998, p. A07.
106. Kaldor, J. and Ashton, L., "The AIDS Debate: Reconstructing AIDS in Africa", *The Independent Monthly*, February 1995, pp. 23-24.
107. CDC (1992), "1993 Revised Classification System for HIV Infection and Expanded Surveillance Case Definition for AIDS Among Adolescents and Adults", *MMWR* 41:1-19.
108. WHO (1986), "Acquired Immunodeficiency Syndrome (AIDS): WHO/CDC case definition for AIDS", *Wkly Epidem. Rec.* 61:69-76.
109. Fauci, A.S., Lane, H.C., "Human Immunodeficiency Virus (HIV) Disease: AIDS and Related Disorders", in *Harrison's Principles of Internal Medicine* (Isselbacher, K.J., Braunwald, E., Wilson, J.D., Martin, J.B., Fauci, A.S., Kasper, D.L., eds), McGraw-Hill, Inc., New York, 1994, 13th ed., pp. 1566-1618.
110. Goedert, J.J., Sarngadharan, M.G., Biggar, R.J. et al. (1984), "Determinants of retrovirus (HTLV-III) antibody and immunodeficiency conditions in homosexual men", *Lancet* 2:711-6.
111. Stevens, C.E., Taylor, P.E., Zang, E.A. et al. (1986), "Human T-cell lymphotropic virus type III infection in a cohort of homosexual men in New York City", *JAMA* 255:2167-2172.
112. Caceres, C.F., van Griensven, G.J.P. (1994), "Male homosexual transmission of HIV-1", *AIDS* 8:1051-1061.
113. Kingsley, L.A., Kaslow, R., Rinaldo, C.R. et al. (1987), "Risk factors for seroconversion to human immunodeficiency virus among male homosexuals", *Lancet* i:345-348.
114. West, R.H., O'Connor, T.W., Penny, R. et al., "Policy Document: Infection Control in Surgery", Royal Australasian College of Surgeons, Melbourne, 1998.
115. European Study Group (1989), "Risk factors for male-to-female transmission of HIV", *Brit. Med. J.* 298:411-414.
116. Padian, N.S., Shiboski, S.C., Glass, S.O., Vittinghoff, E. (1997), "Heterosexual transmission of human immunodeficiency virus (HIV) in northern California: Results from a ten-year study", *Am. J. Epidemiol.* 146:350-357.
117. Anonymous (1985), "HTLV-III antibody in prostitutes", *Lancet* ii:1424.
118. Krogsgaard, K., Gluud, C., Pederson, C. et al. (1986), "Widespread use of condoms and low prevalence of sexually transmitted diseases in Danish non-drug-addict prostitutes", *Brit. Med. J.* 293:1473-1474.
119. Philpot, C.R., Harcourt, C., Edwards, J., Grealis, A. (1988), "Human immunodeficiency virus and female prostitutes, Sydney 1985", *Genitourinary Med.* 64:193-7.
120. Philpot, C.R., Harcourt, C.L., Edwards, J.M. (1991), "A survey of female prostitutes at risk of HIV infection and other sexually transmissible diseases", *Genitourinary Med.* 67:384-8.
121. Pineda, J.A., Aguado, I., Rivero, A. et al. (1992), "HIV-1 infection among non-intravenous drug user female prostitutes in Spain: No evidence of evolution to Pattern II", *AIDS* 6:1365-1369.
122. McKeagney, N., Barnard, M., Leyland, A., Coote, I., Follet, E. (1992), "Female streetworking prostitution and HIV infection in Glasgow", *Brit. Med. J.* 305:801-804.
123. Anonymous (1993), "HIV infection in European female sex workers: epidemiological link with use of petroleum-based lubricants", *European Working Group on HIV Infection in Female Prostitutes*, *AIDS* 7:401-8.
124. Anonymous (1998), "The HIV/AIDS/STD epidemics in Asia and the Pacific", *Australian HIV Surveillance Report* 14:1-8.
125. Samrith, C., "Official HIV and AIDS Case Report", World Health Organization, Phnom Penh, Cambodia, 1997.
126. McLaws, M.L., Brown, A.R.D., Cunningham, P.H., Imrie, A.A., Wilcken, B., Cooper, D.A. (1989), "Prevalence of maternal HIV infection based on anonymous testing of neonates, Sydney 1989", *Med. J. Aust.* 153:383-386.
127. Lundberg, G.D. (1988), "Serological diagnosis of human immunodeficiency virus infection by Western blot testing", *JAMA* 260:674-679.
128. Wooldridge, Dr M., Australian Federal Minister for Health and Human Services, Letter to Senator C. Ellison, 1997.

129. Phair, J., Jacobson, L., Detels, R. et al. (1992), "Acquired Immune Deficiency Syndrome Occurring Within 5 Years of Infection with Human Immunodeficiency Virus Type-1: The Multicenter AIDS Cohort Study", *J. Acquir. Immun. Defic. Syndr.* 5:490-496.
130. Melbye, M., Begtrup, K., Rosenberg, P.S. et al. (1998), "Differences in susceptibility to AIDS development: A cohort study of Danish and American homosexual-bisexual men, 1981-1995", *J. Acq. Immune Def. Syndr. Hum. Retrovirol.* 18:270-276.
131. Lemp, G.F., Porco, T.C., Hirozawa, A.M. et al. (1997), "Projected incidence of AIDS in San Francisco: The peak and decline of the epidemic", *J. Acq. Immune Def. Syndr. Hum. Retrovirol.* 16:182-189.
132. CDC (1993), "1993 Revised Classification System for HIV Infection and Expanded Surveillance Case Definition for AIDS Among Adolescents and Adults", *MMWR* 41:1-19.
133. St Louis, M.E., Rauch, K.J., Peterson, L.R., Anderson, J.E., Schable, C.A., Dondero, T.J. (1990), "Seroprevalence rates of human immunodeficiency virus infection at sentinel hospitals in the United States", *NEJM* 323:213-218.
134. Burke, D.S., Brundage, J.F., Redfield, R.R. et al. (1988), "Measurement of the false positive rate in a screening program for human immunodeficiency virus infections", *NEJM* 319:961-964.
135. Dax, E. (1994), "The HIV Western blot: Reply to letter", *Med. J. Aust.* 160:808.
136. Kion, T.A., Hoffmann, G.W. (1991), "Anti-HIV and anti-anti-MHC antibodies in alloimmune and autoimmune mice", *Science* 253:1138-1140.
137. Genesca, J., Jett, B.W., Epstein, J.S., Shih, J.W.K., Hewlett, I.K., Alter, H.J. (1989), "What do Western Blot indeterminate patterns for Human Immunodeficiency Virus mean in EIA-negative blood donors?", *Lancet* ii:1023-1025.
138. Kozhemiakin, L.A., Bondarenko, I.G. (1992), "Genomic instability and AIDS", *Biochimii* 57:1417-1426.
139. Strandstrom, H.V., Higgins, J.R., Mossie, K., Theilen, G.H. (1990), "Studies with canine sera that contain antibodies which recognize human immunodeficiency virus structural proteins", *Cancer Res.* 50:5628s-5630s.
140. Kashala, O., Marlink, R., Ilunga, M. et al. (1994), "Infection with human immunodeficiency virus type-1 (HIV-1) and human T-cell lymphotropic viruses among leprosy patients and contacts: correlation between HIV-1 cross-reactivity and antibodies to lipoarabinomannan", *J. Infect. Dis.* 169:296-304.
141. Mason, A.L., Xu, L., Guo, L. et al. (1998), "Detection of retroviral antibodies in primary biliary cirrhosis and other idiopathic biliary disorders", *Lancet* 351:1620-1624.
142. Lange, W.R., Ball, J.C., Adler, W.H. et al. (1991), "Followup study of possible HIV seropositivity among abusers of parenteral drugs in 1971-72", *Pub. Health Rep.* 106:451-455.
143. Chant, K., Lowe, D., Rubin, G. et al. (1993), "Patient-to-patient transmission of HIV in private surgical consulting rooms", *Lancet* 342:1548-1549.
144. Turner, V.F., Papadopoulos-Eleopoulos, E. (1994), "Patient-to-patient transmission of HIV in a surgeon's private rooms: Invited deposition to the Royal Australasian College of Surgeons", at [www.virusmyth.com/aids/perthgroup/](http://www.virusmyth.com/aids/perthgroup/).
145. "Human Immunodeficiency Virus Type-1. Qualitative Enzyme Immunoassay for the Detection of Antibody to Human Immunodeficiency Virus Type-1 (HIV-1) in Human Serum or Plasma", Abbott Laboratories, Diagnostics Division, Abbott Park, Illinois, USA, 1988.
146. WHO, "The Current Global Situation of the HIV/AIDS Pandemic", Geneva, 1995.
147. Sinoussi, F., Mendiola, L., Chermann, J.C. (1973), "Purification and partial differentiation of the particles of murine sarcoma virus (M. MSV) according to their sedimentation rates in sucrose density gradients", *Spectra* 4:237-243.
148. Toplin, I. (1973), "Tumor Virus Purification using Zonal Rotors", *Spectra* 225-235.
149. Bess, J.W., Gorelick, R.J., Bosche, W.J., Henderson, L.E., Arthur, L.O. (1997), "Microvesicles are a source of contaminating cellular proteins found in purified HIV-1 preparations", *Virol.* 230:134-144.
150. Gluschkof, P., Mondor, I., Gelderblom, H.R., Sattentau, Q.J. (1997), "Cell membrane vesicles are a major contaminant of gradient-enriched human immunodeficiency virus type-1 preparations", *Virol.* 230:125-133.
151. Beard, J.W. (1957), "Physical methods for the analysis of cells", *Ann. New York Acad. Sci.* 69:530-544.
152. Grafe, A., A History of Experimental Virology, Springer-Verlag, Heidelberg, 1991.
153. Panem, S. (1979), "C-Type Virus Expression in the Placenta", *Curr. Top. Pathol.* 66:175-189.
154. Gallo, R.C., Wong-Staal, F., Reitz, M., Gallagher, R.E., Miller, N., Gillespie, D.H., "Some evidence for infectious type-C virus in humans", in *Animal Virology* (Balimore, D., Huang, A.S., Fox, C.F., eds.), Academic Press, Inc., New York, 1976, pp. 385-405.

155. Hockley, D.J., Wood, R.D., Jacobs, J.P. (1988), "Electron Microscopy of Human Immunodeficiency Virus", *J. Gen. Virol.* 69:2455-2469.
156. Lecatsas, G., Taylor, M.B. (1986), "Pleomorphism in HTLV-III, the AIDS virus", *S. Afr. Med. J.* 69:793-794.
157. O'Hara, C.J., Groopman, J.E., Federman, M. (1988), "The Ultrastructural and Immunohistochemical Demonstration of Viral Particles in Lymph Nodes from Human Immunodeficiency Virus-Related Lymphadenopathy Syndromes", *Hum. Pathol.* 19:545-549.
158. Varmus, H. (1987), "Reverse Transcription", *Sci. Am.* 257:48-54.
159. Varmus, H.E. (1989), "Reverse transcription in bacteria", *Cell* 56:721-724.
160. Gallo, R.C., Sarin, P.S., Wu, A.M., "On the Nature of the Nucleic Acids and RNA-Dependent DNA Polymerase from RNA Tumor Viruses and Human Cells", in *Possible Episomes in Eukaryotes* (Silvestri, L.G., ed.), North Holland Publishing Company, Amsterdam, 1973, pp. 13-34.
161. Tomley, F.M., Armstrong, S.J., Mahy, B.W.J., Owen, L.N. (1983), "Reverse transcriptase activity and particles of retroviral density in cultured canine lymphosarcoma supernatants", *Br. J. Cancer* 47:277-284.
162. Christie, H., Betacam interview with Dr Robert Gallo at Palexpo Conference Centre, Geneva, 1 July 1998.
163. Peuchmaur, M., Delfraissy, J.F., Pons, J.C. et al. (1991), "HIV proteins absent from placentas of 75 HIV-1-positive women studied by immunohistochemistry", *AIDS* 5:741-5.
164. Faulk, W.P., Labarrere, C.A. (1991), "HIV proteins in normal human placentae", *Am. J. Reproductive Immunology* 25:99-104.
165. Stott, E.M. (1991), "Anti-cell antibody in macaques", *Nature* 353:393.
166. Arthur, L.O., Bess, J.W. Jr, Urban, R.G. et al. (1995), "Macaques immunized with HLA-DR are protected from challenge with simian immunodeficiency virus", *J. Virol.* 69:3117-24.
167. Eigen, M., Schuster, P. (1977), "The hypercycle", *Die Naturwissenschaften* 64:541-565.
168. Eigen, M., Gardiner, W., Schuster, P., Winkler-Oswatitsch, R. (1981), "The origin of genetic information", *Sci. Am.* 224:78-94.
169. Defer, C., Agut, H., Garbarg-Chenon, A. et al. (1992), "Multicentre quality control of polymerase chain reaction for detection of HIV DNA", *AIDS* 6:659-663.
170. Rich, J.D., Merriman, N.A., Mylonakis, E. et al. (1999), "Misdiagnosis of HIV infection by HIV-1 plasma viral load testing: A case series", *Ann. Int. Med.* 130:37-39.
171. Coste, J., Montes, B., Reynes, J. et al. (1997), "Effect of HIV-1 genetic diversity on HIV-1 RNA quantification in plasma: comparative evaluation of three commercial assays", *J. Acq. Immune Def. Syndr. Hum. Retrovirol.* 15:174.
172. Gallagher, R.E., Gallo, R.C. (1975), "Type-C RNA Tumor Virus Isolated from Cultured Human Acute Myelogenous Leukemia Cells", *Science* 187:350-353.
173. Barbacid, M., Bolognesi, D., Aaronson, S.A. (1980), "Humans have antibodies capable of recognizing oncoviral glycoproteins: Demonstration that these antibodies are formed in response to cellular modification of glycoproteins rather than as consequence of exposure to virus", *Proc. Natl. Acad. Sci. USA* 77:1617-1621.
174. Snyder, H.W., Fleissne, E. (1980), "Specificity of human antibodies to oncovirus glycoproteins: Recognition of antigen by natural antibodies directed against carbohydrate structures", *Proc. Nat. Acad. Sci. USA* 77:1622-1626.
175. Kurth, R., Teich, N.M., Weiss, R., Oliver, R.T.D. (1977), "Natural human antibodies reactive with primate type-C antigens", *Proc. Nat. Acad. Sci. USA* 74:1237-1241.
176. Casarett, D.J., Lantos, J.D. (1998), "Have we treated AIDS too well? Rationing and the future of AIDS exceptionalism", *Ann. Int. Med.* 128:756-759.
177. Anonymous (1998), *The National AIDS Registry. Australian HIV Surveillance Report 14:14*, National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst, NSW 2010, Australia.
178. Balter, M. (1998), "Europe: AIDS research on a budget", *Science* 280:1856-1858.
179. Stine, G.J., "Testing for Human Immunodeficiency Virus", in *AIDS Update 1994-1995*, Prentice Hall, New Jersey, 1995, p. 231.
180. Glover, E. (1998), "French panel calls for closer monitoring of genetic modification", *Nature* 394:4.
181. Mortimer, P.P. (1988), "The AIDS virus and the AIDS test", *Med. Internat.* 56:2334-2339.

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