APPENDIX XI
A Critical Examination of the Evidence for the Existence of HIV

INTRODUCTION

Following the appearance of AIDS in 1981 many aetiological factors were proposed. In May 1983 Montagnier announced the discovery of a retrovirus, now known as HIV, from lymphatic tissue of a gay man with lymphadenopathy. One year later Gallo reported data which "suggests that HTLV-III [HIV] is the primary cause of AIDS". By 1986 the scientific community accepted the Gallo assertion that the same data was "clearcut evidence" that HIV is the causative agent of the clinical syndrome. Even today the five *Science* papers published by these French and American groups are still widely regarded as proving beyond all reasonable doubt that HIV exists and is the cause of AIDS.

However, not all scientists accepted these findings. In 1987 Peter Duesberg published an invited paper in *Cancer Research* on retroviruses and cancer in which he also questioned the role of HIV in AIDS.[1] At the same time one of us (EPE) also challenged the theory[2] including the data claimed to prove the existence of HIV. Papadopulos-Eleopulos also proposed an alternative, non-infectious aetiology and treatments based on this hypothesis. Since then our group has published papers addressing every facet of the HIV theory[2-18] including a detailed examination of HIV isolation and the HIV genome.[9,19] Here we confine ourselves largely to addressing the data published by Montagnier and Gallo in their 1983/84 *Science* papers. Genomic data are only briefly discussed because the existence of HIV and the HIV theory of AIDS were universally accepted before such data were available. For a comprehensive discussion on genomic data the reader is referred to reference 19.

RETROVIRUSES AND THEIR IDENTIFICATION

A virus possesses two characteristic properties. The first is anatomical, that is, being a microscopic particle of individual morphology, the second, the ability to generate identical progeny by synthetic processes obligatorily occurring within living cells. It is the latter attribute which defines a particle with the appearances of a virus, that is, a viral-like particle, as infectious and thus a virus. The three subfamilies (*0ncovirinae*, *Lentivirinae* and *Spumavirinae*) of *Retroviridae* (Retroviruses) are "enveloped viruses with a diameter of 100-120 nm budding at cellular membranes. Cell released virions contain condensed inner bodies (cores) and are studded with projections (spikes, knobs)".[20] The retroviral particles contain RNA and the enzyme reverse transcriptase (RT), an RNA dependent DNA polymerase which catalyses the synthesis of DNA contrary to the central dogma of biology, that is, in a direction "reverse" from DNA to RNA. According to retrovirologists, such DNA is then integrated into existing cellular DNA as a "provirus". Retroviral particles share the property of concentrating (banding) at a density of 1.16 gm/ml when centrifuged at high speeds in sucrose density gradients, a fact long used in their purification.[21,22]

All retrovirologists agree that to prove the existence of a new retrovirus one must isolate it. However, the term "virus isolation" is beset with semantic difficulties and ambiguities. The dictionary meaning of "isolation" derives from the Latin *insulatus* (made into an island) and refers to the act of separating an object from all other matter that is not that object. "Purification" means to obtain something free from impurities. In this context isolation is the same as purification. Because virus particles are small it is not possible to obtain a single, isolated particle. The next best thing is to obtain a mass of particles separate from everything else. Until the early 1980s, for the isolation of animal retroviruses as well as the "first" human retrovirus HL23V, by isolation retrovirologists meant purification. On the other hand, nowadays both basic and specialised texts rarely define "isolation" and when they do such attempts are non-illuminating. For example, Levy defines isolation as a "sample of a virus from a defined source",[23] and White as the ability to "identify a totally unforeseen virus, or even discover an entirely new agent".[24] Encompassed as "virus isolation" are listed methods of culturing specimens in tissue and chick embryo cells, as well as live animals, following by documentation therein of cytopathic and pathological effects, haemoabsorption, immunofluorescence, antigen/antibody reactions and "characterisation of the viral genome".[24-27] HIV experts, including Luc Montagnier and Robin Weiss define "virus isolation" as "propagating them [viruses] in cells in culture".[28,29] However, if "virus isolation" is to "take a sample of a virus from a defined source", or "propagating them in cells in culture", then first one must have prior proof that a virus exists in "a defined source" or "in cells in culture". One cannot know that a virus exists or define its constituents without purification (isolation) of the putative viral particles.

There are several reasons why this is mandatory:

To prove that the retrovirus-like particles are infectious, that is, they are a virus

The finding of particles with the appearances of retroviruses, is not proof that such particles are retroviruses and even less proof a particle is a particular retrovirus. Particles bearing the morphological characteristics of retroviruses are ubiquitous. In the 1970s such particles were frequently observed in human leukaemia tissues,[30] cultures of embryonic tissues and "in the majority if not all, human placentas".[31] Type-C retroviral particles are present in "fish, snakes, worms, pheasant, quail, partridge, turkey, tree-mouse and agouti"[32] as well as in "tapeworms, insects...and mammals".[33] Gallo was well aware of this problem as far back as 1976 when he wrote: "Release of virus-like particles morphologically and biochemically resembling type-C virus but apparently lacking the ability to replicate have been frequently observed from leukaemic tissue".[30] In other words, it is not possible to claim a particle is a retrovirus merely by appearances. To prove that retrovirus-like particles observed in a culture are a virus one must isolate the particles, characterise their proteins and RNA and introduce the particles into a secondary culture. If any particles are released in the secondary culture they too must be isolated and proven that their proteins and RNA are the same as those from the primary culture. In such experiments one must not ignore the use of legitimate controls and in doing so take in consideration an important difference between retroviral and other infectious agents.

When one finds an infectious agent, for example a virus or a bacterium, either *in vitro* or *in vivo*, one may be assured that the agent has been introduced into the culture or animal from outside. Retroviruses are the exception. This is because normal human and animal genomes contain information which, under the appropriate conditions, leads to the synthesis of retroviral RNA and proteins, or even to the assembly of retroviral particles, that is, to the expression of endogenous retroviruses. And although as late as 1994 both Gallo and Fauci taught "there are no known human endogenous retroviruses",[34] it is known that at least 1% of the human DNA is retroviral DNA and that endogenous retroviruses are present "in all of us".[35,36] Furthermore, new endogenous retroviral genomes may arise from rearrangements of existing retroviral genomes, cellular DNA or both, caused by many factors, including pathogenic processes.[37,38] The expression of endogenous retroviral genomes may arise spontaneously[39] and may be significantly accelerated and the yield increased by conditions which induce cellular activation.[40-42] According to the eminent retrovirologist George Todaro, "the failure to isolate endogenous viruses from certain species may reflect the limitations of in-vitro cocultivation techniques".[43] Endogenously produced retroviruses are morphologically and biochemically indistinguishable from exogenous retroviruses. Because of this, the finding of identical retrovirus in serially "infected" cultures/cocultures is not proof that the cells are infected with exogenous retrovirus. One method which may assist resolve but will not prove whether cells acquire virus from the outside (exogenously acquired retrovirus, infectious retrovirus) and have not assembled a retrovirus from information already existing in normal cells (endogenous retrovirus), is to conduct control cultures/cocultures in parallel with test cultures/cocultures. The only difference between test and control cultures should be the introduction of tissue assumed infected into the test cultures. In every other respect control cultures must be dealt with identically. For example:

(a) because detection of RT and retroviral genetic sequences, and release of retroviral particles depends on the metabolic state of the cells, the physiological state of the cells used in the control cultures should be as close as possible to the test culture;

(b) because the mere act of co-cocultivation may lead to release of endogenous retroviral particles, if test cells are cocultured, so should the controls;

(c) extracts even from normal, unstimulated cells when added to the cultures may increase endogenous retroviral expression. Because of this, when host cells are cultured with supernatant or material which bands at 1.16 gm/ml from cultures thought to be infected, the controls must be cultured with similar material from noninfected cultures;

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

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

To determine their biological effects

Without recourse to pure particles it is impossible to determine whether effects are due to virus particles or contaminants including “chemical stimulants”, a fact stressed as far back as 1911 by the Peyton Rous, the father of retrovirology.[44]

In 1911 Rous induced malignancy in chickens by injections of cell-free filtrates obtained from a muscle tumour. Similar experiments were repeated by many researchers and the tumour inducing filtrates became known as filterable agents, filterable viruses, Rous agents, Rous virus and ultimately retroviruses. However, Rous himself expressed doubts that the agents which caused the tumours were infectious in nature. Indeed he warned, "The first tendency will be to regard the self-perpetuating agent active in this sarcoma of the fowl as a minute parasitic organism. Analogy with several infectious diseases of man and the lower animals, caused by ultramicroscopic organisms, gives support to this view of the findings, and at present work is being directed to its experimental verification. But an agency of another sort is not out of the question. It is conceivable that a chemical stimulant, elaborated by the neoplastic cells, might cause the tumour in another host and bring about in consequence a further production of the same stimulant".[44]

To characterise the viral proteins

The only way to prove that a protein is a constituent of an object is to obtain it from that object, or when the object is very small as is the case of viruses, from material consisting of purified virus particles. If the material contains impurities which are proteins or contain proteins, it is not possible to determine which are viral and which are not. Yet only after the viral proteins are characterised is it possible to employ them as antigens in antibody tests.

To characterise the viral genome

As for viral proteins the only way to prove that a stretch of RNA is viral it is to obtain it from material which contains nothing else but virus particles. If the material contains impurities the impurities must not include RNA. Then and only then can the RNA and its complementary DNA (cDNA) be used as probes and primers for genomic hybridisation and PCR studies.

To act as a gold standard for the antibody tests

The reaction of a virus or viral protein with an antibody present in a patient's serum does not prove that the antibody is induced by or directed against the virus or a viral protein. That is, that the reaction is specific. This is because there are significant obstacles which hinder the interpretation of antibody/antigen reactivity including non-specific stimulation,[45-47] cross-reactivity[48-55] or both. Cross reactivity results from antibody molecules, even monoclonal antibodies, interacting not only with the inducing antigen but also with other antigens. Indeed, there are instances where "cross-reactive antibodies may have higher affinity with antigens other than the inducing antigen. Even antigens that differ for most of their structure can share one determinant, and a monoclonal antibody recognising this site would then give a 100% cross-reaction. An example is the reaction of autoantibodies in lupus with both DNA and cardiolipin...It should be emphasised that sharing a "determinant" does not mean that the antigens contain identical chemical structures, but rather that they bear a chemical resemblance that may not be well understood, for example, a distribution of surface charges".[55] Since polyclonal antibodies are composites of monoclonal antibodies these facts apply equally, if not more so, to polyclonal antibodies. These facts have been extensively exploited in clinical medicine for the diagnosis of diseases such as syphilis and infectious mononucleosis. In these diseases, *T. pallidum* and Epstein-Barr virus cause the appearance of antibodies reactive with ox-heart proteins and sheep and horse red blood cells. However, this does not mean that patients are "infected" with ox-heart, or horse red blood cells and the diseases are induced by these agents. The only way to determine the specificity of an antibody/antigen reaction is to use an independent method, a gold standard to prove the presence or absence of the antigen. The only possible gold standard for a test to prove a virus infection is the virus in question. That is, virus isolation/purification.

METHODS FOR THE ISOLATION/PURIFICATION OF RETROVIRUS-LIKE PARTICLES

Up till the 1950s retroviruses were isolated/purified by filtration although this method was less than satisfactory. With the development of the electron microscope, apparently, for some retrovirologists, the detection of retrovirus-like particles was deemed sufficient to prove the existence of a retrovirus. However, other scientists including the well-known retrovirologist, JW Beard, recognised that cells, including uninfected cells, under various conditions, were responsible for the generation of a heterogenous array of particles some with the appearances of retroviruses. Beard stressed: "identification, characterisation, and analysis are subject to well-known disciplines established by intensive investigations, and the possibilities have by no means been exhausted. Strangely enough, it is in this field that the most frequent shortcomings are seen. These are related at times to evasion of disciplines or to their application to unsuitable materials. As was foreseen, much of the interest in the more tedious aspects of particle isolation and analysis has been diverted by the simpler and undoubtedly informative processes of electron microscopy. While much can be learned quickly with the instrument, *it is nevertheless clear that the results obtained with it can never replace, and all too often may*

obscure, the need for the critical fundamental analyses that are dependent on access to homogenous materials"[56] (italics ours).

By the 1970s there was general agreement that "Virions of RTV [retroviruses] have a characteristic buoyant density, and centrifugation to equilibrium in density gradients is the preferred technique for purification of RTV".[57] The method of banding in density gradients is not ideal either. Substances other than retroviruses may band at the same density. This is why at a meeting held at the Pasteur Institute in 1972, Francoise Barre-Sinoussi and Jean-Claude Chermann stressed that to claim purification of retrovirus-like particles using sucrose density gradients it is absolutely necessary to prove, using the electron microscope, that the 1.16 gm/ml band contains nothing else but particles with "no apparent differences in physical appearances".[21,22]

THE PHENOMENA CLAIMED TO PROVE THE EXISTENCE OF HIV

In May 1983 Luc Montagnier, Francoise Barre-Sinoussi, Jean-Claude Chermann and colleagues published a paper in *Science* entitled, "Isolation of a T-Lymphotropic retrovirus from a patient at risk for Acquired Immune Deficiency Syndrome (AIDS)".[58] This is the paper which, since the resolution of the polemics between Montagnier and Gallo concerning allegations of misappropriation by the latter of the French virus sent to the US by the Pasteur Institute, is accepted as being the study which proved the existence of HIV. There it was shown that mitogen stimulated lymph node cell cultures from a gay man (BRU) with lymphadenopathy were able to transcribe the synthetic RNA primer-template An.dT_{15}. From this data Montagnier and his colleagues concluded that BRU's lymph node cells were infected with a retrovirus. The finding of the same activity in the supernatant of a coculture consisting of the same cells with stimulated lymphocytes from a healthy individual was considered proof for virus transmission as well as isolation. In another experiment supernatants from the cocultures were added to two, three day old, stimulated umbilical cord lymphocytes cultures. "Electron microscopy of the infected umbilical cord lymphocytes showed characteristic immature particles with dense crescent (C-type) budding at the plasma membrane". Supernatant from the culture was banded in sucrose density gradient and the 1.16 gm/ml band was shown to transcribe An.dT_{15}. The proteins in the 1.16 gm/ml band as well as the proteins of a cellular extract were separated according to their molecular weight using "denaturing buffer and electrophoresed on 12.5 percent polyacrylamide-SDS slab gel". When the strips were incubated with human sera many proteins from the cellular extracts were found to react with serum from BRU, another gay man as well as a "healthy donor". In the strips containing the proteins from the 1.16 gm/ml band three proteins including a p25 ('p' for protein, 25 for its molecular weight in thousands) were found to react. They also reported that the p25 did not react with antibodies to HTLV-I. The material banding at 1.16 gm/ml was claimed to be "purified, labelled, virus" although no electron microscopic data were presented. The authors concluded: "A retrovirus belonging to the family of recently discovered human T-cell leukaemia viruses (HTLV), but clearly distinct from each previous isolate, has been isolated from a Caucasian patient with signs and symptoms that often precede the acquired immune deficiency syndrome (AIDS). This virus is a typical type-C RNA tumor virus, buds from the cell membrane, prefers magnesium for reverse transcriptase activity, and has an internal antigen (p25) similar to HTLV p24".[58]

Robert Gallo and his associates did not consider the Montagnier group data as proving "true isolation".[59] As late as 1997, in a book published by one of the best known HIV experts, Jaap Goudsmit, one reads: "The BRU lymph node was first cultured in early January 1983 and, on January 15, it shed an enzyme absolutely unique to the lentivirus group. [The enzyme is not even specific to retroviruses much less to *Lentiviruses* (see below)]...The BRU virus grew slowly and with difficulty, but its identity and activity were reported in the May 20, 1983 issue of *Science*...The Pasteur Group was widely acclaimed but very worried. In the world of virology, finding a new virus is not enough: You must propagate and isolate the organism for analysis by other virologists. The French had not yet isolated their new lentivirus".[60]

Why then did (a) Gallo, who reviewed the Montagnier manuscript, recommend its publication? (b) all the HIV experts including Gallo and Goudsmit (on page 24 one reads: "BRU was the first strain to be isolated") accept that the first isolation of HIV and thus of its existence was proven in the May 1983 *Science* paper?

A year later, in May 1984, Gallo, Popovic and their colleagues published four papers in *Science* in which they claimed "isolation" of another retrovirus from AIDS patients.[59,61-63] However, in addition to the use of a leukaemic cell line, the only difference between the Montagnier and Gallo groups' data were quantitative. In the first paper entitled "Detection, Isolation and Continuous Production of Cytopathic Retroviruses (HTLV-III) from Patients with AIDS and pre-AIDS", (HTLV-III=HIV), experiments were described in which "concentrated culture fluids harvested from short-term [mitogenically stimulated] cultures of T-cells" from patients with AIDS or pre-AIDS were cultured with a mitogenically stimulated leukaemic cell line HT and highly selected clones obtained by culturing HT with irradiated cells of a healthy donor. The data presented as proof of isolation of

HIV were (a) RT activity in cell free supernatants and the 1.16 gm/ml band; (b) reaction in the cultures with "Rabbit antiserum to HTLV-III" and "Patient serum (E.T.)"; (c) EM showing the presence of retroviral-like particles in the cultures.
An enquiry conducted by the National Institutes of Health Office of Scientific Integrity found that the HT cell line was cultured not with concentrated fluids (supernatant) originating from individual AIDS patients, but with concentrated fluids pooled initially from individual cultures of three patients and ultimately from the individual cultures of ten patients.[64] In evidence given to this enquiry the reason given was because none of the supernatants "individually was producing high concentrations of reverse transcriptase". In other words, Gallo and his colleagues did not regard the levels of RT from individual cultures as proof that individual specimens contained a retrovirus. The Gallo investigation found the pooling of specimens to be "of dubious scientific rigor". One scientist described the procedure as "really crazy".[65] Most importantly how could Gallo use the reaction with rabbit antiserum to prove HIV isolation (purification) when, to obtain rabbit antiserum rabbits must first be injected with pure HIV?[66] It is inexplicable how Gallo and his colleagues could already possess "Rabbit serum to HTLV-III" before they had proved the existence of a novel virus. However, if the antiserum was manufactured "from rabbits infected repeatedly with disrupted HTLV-III", that is, the material banding at 1.16 gm/ml, one would expect to obtain antibodies to all the proteins constituting this material even if the proteins were cellular and not viral.

In the second paper entitled "Frequent Detection and Isolation of Cytopathic Retroviruses (HTLV-III) from patients with AIDS and at risk of AIDS", Gallo and his colleagues claimed to have "isolated" HTLV-III (HIV) from 26/72 (36%) of AIDS patients. In this paper isolation was defined as "more than one of the following": "repeated detection of a Mg^{2+}-dependent reverse transcriptase activity in supernatant fluids; virus observed by electron microscopy (EM) [retrovirus-like particles in the culture]; intracellular expression of virus-related antigens detected with antibodies from seropositive donors or with rabbit antiserum to HTLV-III; or transmission of particles". Transmission of particles was defined as "detected by RT assays or by electron microscopic observation, to fresh human [umbilical] cord blood, bone marrow, or peripheral blood T lymphocytes", cultured with supernatants from the "infected" cultures.[61] (It can be seen that the Gallo group method permitted "isolation" of a retrovirus without evidence for either particles or RT).

In the third paper, proteins from the 1.16 gm/ml band which they claimed was "purified" HIV, as well as the proteins from the "infected" cells, were "lysed and fractionated by electrophoresis on a 12% polyacrylamide slab gel in the presence of SDS. The protein bands were electrophoretically transferred to a nitrocellulose sheet" and reacted with different sera. In other words Gallo used a technique which is known as Western blot (WB) antibody test. Many proteins from the cellular extract were found to react with sera from both patients and healthy individuals. They also reported that two proteins from the 1.16 gm/ml band, p24 and p41, reacted with patient sera. For this and no other reason it was claimed that "these molecules are the major components of the virus preparation. p24 and p41 may therefore be considered the viral structural proteins". As far as morphology is concerned, the Gallo group reported that the HIV particle "is produced in high numbers from infected cells by budding from the plasma membrane. A possible unique feature of this virus is the cylindrical shaped core observed in many mature virions...HTLV-III is a true member of the HTLV family". (HTLVs are type C retroviral particles and not *Lentiviruses* as HIV is claimed to be).

In the fourth paper, instead of separating the proteins which banded at 1.16 gm/ml and then incubating them with patient sera, the mixture of all proteins was used, that is, they performed a test known as enzyme-linked immunosorbent assay (ELISA). "To understand the molecular nature of the antigens recognised by ELISA", they also performed WB, with some of the sera. They reported that: "Serum samples from 88 percent of patients with AIDS and from 79 percent of homosexual men with signs and symptoms that frequently precede AIDS, but from less than 1 percent of heterosexual subjects, have antibodies reactive against antigens of HTLV-III. The major immune reactivity appears to be directed against p41, the presumed envelope antigen of the virus…The data presented here and in the accompanying reports suggest that HTLV-III is the primary cause of AIDS". Two years later Gallo wrote that "The results presented in our four papers provided *clearcut evidence that the aetiology of AIDS and ARC was the new lymphotropic retrovirus, HTLV-III*"[67] (italics ours).

COMMENTS
As mentioned, apart from the quantitative difference, the Gallo group experiments are no different from those performed by Montagnier and his colleagues. It follows then, that if Montagnier's group data do not prove "true isolation" neither did Gallo's nor anybody else's because to date everybody has repeated (for the vast majority only part therof) the same experiments as these two groups. It is of pivotal significance that neither group reported the use of valid controls (see above) nor did they prove they had obtained purified retrovirus-like particles. The question then is, do the data obtained by the Montagnier and Gallo group, that is RT, particles in culture and antigen/antibody reactions prove the isolation of a unique, or even a retrovirus, any retrovirus?

Without doubt, if by isolation one expects proof of purification, then the detection of an enzyme, or retrovirus-like particles in a culture, or proteins either in the cells or the 1.16 gm/ml band which react with antibodies present in human or animal sera, do not comply. To argue otherwise one must define the detection of cardiac or hepatic enzymes in the blood of patients suffering chest pain or jaundice as proof for isolation of the human heart or liver. Likewise, if an antibody/antigen reaction is proof for isolation of a virus then antibody reactivity to the protein _HCG is proof for isolation of the human placenta. The assertion that the detection of a retrovirus-like particle in a culture proves isolation is no different from asserting that the detection of a fish-like creature in the ocean is the same as having a definite fish in your frying pan. The detection of RT, retrovirus-like particles and antibody/antigen reactivity can only be considered proof for the detection of a retrovirus, and then if, and only if, there is prior knowledge that the three phenomena are specific to retroviruses.

To claim that the detection of this phenomena proves the existence of a new retrovirus one must have proof that at least one of the three phenomena is different from that observed in all other known retroviruses.

Reverse transcriptase

At present some of the leading HIV researchers consider RT as being the "*sine qua non*" of retroviruses and regard the detection of reverse transcription in lymphocyte cultures from AIDS patients not only as proof of the presence of such viruses but of HIV itself including HIV isolation[68-70] and quantification.[71] However, according to some of the best known retrovirologists including its discoverer, as well as the Nobel Laureate and former Director of the US National Institute of Health Harold Varmus, reverse transcriptases are present in all cells as well as bacteria and viruses.[72-74] "Reverse transcriptase (RT) was first discovered as an essential catalyst in the biological cycle of retroviruses. However, in the past years, evidence has accumulated showing that RTs are involved in a surprisingly large number of RNA-mediated transcriptional events that include both viral and nonviral genetic entities".[75] Even if RT were a property only of viruses it is not specific to retroviruses. According to Varmus, "Reverse transcription was assigned a central role in the replication of other viruses [hepatitis B and cauliflower mosaic viruses] and in the transposition and generation of other kinds of eukaryotic DNA".[76] "The hepatitis B viruses (HBVs) are small DNA viruses that produce persistent hepatic infections in a variety of animal hosts and replicate their DNA genomes via reverse transcription of an RNA intermediate. All members of this family contain an open reading frame (ORF), "P" (for *pol*), which is homologous to retroviral *pol* genes"[77] [*pol*=polymerase]. "Hepatitis B virus (HBV) resembles retroviruses, including HIV, in several respects. In particular, both viruses contain reverse transcriptase, and replicate through an RNA intermediate". Because of this, it has been suggested that hepatitis B infection should be treated with the same antiretroviral agents as HIV infection.[78,79]

At present, evidence exists which shows that although the major target organ for hepatitis B virus is the liver, cells other than hepatocytes "including peripheral blood lymphocytes and monocytes, may become infected with HBV".[80] Lymphocyte stimulation in general and PHA (an agent employed in the majority of cultures with tissues from AIDS patients), is associated with the production of hepatitis B virus from peripheral blood lymphocytes in patients infected with HBV including "viral replication in chronic hepatitis B infection of childhood".[81,82] Hepatitis B virus infection is widespread in the AIDS risk groups.

In the early 1970s Gallo proved that cultures of leukaemic cells transcribe the $An.dT_{15}$ template-primer as does material banding at 1.16 gm/ml originating from "PHA stimulated (but not unstimulated normal human blood lymphocytes".[83] Reading the Gallo 1984 *Science* papers the impression is gained that the leukaemic HT cell line and thus its clones, including H9 which Gallo used, was a new cell line developed in Gallo's laboratory. However it is now known that the HT cell line is the HUT78 cell line which originated from a patient with adult T cell leukaemia, a disease which Gallo claims is caused by the retrovirus, HTLV-I. A year earlier Gallo claimed to have proven that the HUT78 cells are infected with HTLV-I.[84] If this is the case, the Gallo group would have detected RT in their cultures even if the enzyme is specific to retroviruses and the cultures were not infected with HIV. Other researchers reported RT activity in normal non-infected spermatozoa.[85]

It must also be pointed out that the presence of HIV reverse transcriptase was detected by both Montagnier and Gallo, (and all HIV experts since) indirectly, that is, by detecting the transcription of the template-primer $An.dT_{15}$. However, at least in 1984 if not 1983, Montagnier and his colleagues knew that in the 1970s there was proof that “Among a number of template primers, $(rA)_n.(dT)_{12-18}$ has been most frequently employed since RT shows high activity with this template primer. However, the problem is that the cellular DNA polymerases (pol β and pol γ) also effectively utilize the same template primer”.[86] In fact, in 1975, an International Conference on eukaryotic DNA polymerases defined DNA polymerase _ as the cellular enzyme which "copies $An.dT_{15}$ with

high efficiency but does not copy DNA well".[87] One year earlier Gallo wrote: "Under appropriate reaction conditions DNA polymerase _ can efficiently transcribe poly (A) primed by oligo (dT)".[88] Thus it is possible to detect transcription of An.dT_{15} even when no RT, either viral or cellular, is present, especially under the conditions used to prove the existence of HIV. (Both Montagnier and Gallo accept that the phenomena detected in cultures which are said to prove HIV isolation cannot be detected unless the cells are chemically stimulated[89,90]). Nowadays the non-specificity of RT is broadcast even in the popular press to readers contemplating the purchase of shares in biotechnology companies.[91]

In conclusion, even if one accepts Montagnier and Gallo's groups' definition of isolation, given that RTs and reverse transcription are nonspecific to retroviruses, their detection even in an unlimited number of consecutive cultures/co-cultures cannot be considered proof for isolation and propagation of a retrovirus.

"HIV" particles

Montagnier and his colleagues reported HIV initially as a type C particle, then as a type D particle and then as a *Lentivirus*. In 1984 Gallo and his colleagues reported HIV as a type C particle. However, in 1985 Gallo wrote: "A possible unique feature of the virions is the cylindrical core observed in many presumably mature virions. Virions having this type of core have been frequently reported for certain type D retroviruses, and in some instances, for type C retroviruses". Jay Levy reported HIV as a type D particle.[92] Others at the University of California wrote that "AIDS virus isolated show morphologic characteristics of type C, type D and Lentiviruses".[93] According to Anthony Fauci and others" "T-cells and macrophages handle the virus very differently. In the T-cell, virus buds out of the external plasma membrane of the cell. In the monocyte/macrophage cultures it buds into membrane-bound vesicles inside the cells".[94] The latter is a description of a type A, retroviral particle.[33] Thus the leading HIV experts have described HIV as a member of two subfamilies and three genera of *Retroviridae*. These taxonomical differences imply that if HIV was a newly discovered mammal, it could have been either human, a gorilla or an orang-utan. By consensus at present HIV is regarded as a *Lentivirus*. This agreement was reached when it was realised that in "HIV" positive individuals AIDS did not appear soon after "infection", although the designation *Lentivirus* is a morphological description.[33]

"HIV infected cultures" contain in addition to the particles with the morphology attributed to HIV many other "viral particles". For example: Hockley and his colleagues from the Electron Microscopy and Photography Section and Division of Virology at the National Institute for Biological Standards and Control in the United Kingdom describe a profusion of particles which they divide broadly into three groups, mature, ring-like and small with spikes. The mature particles "were approximately spherical in shape and 100 to 150nm in diameter. The outer lipid membrane was frequently broken or absent in places, and there was no evidence of surface spikes...A few mature particles were found that were larger than average and appeared to contain a double nucleoid...in the preparation of HIV there were always many vesicles with granular contents in which it was not possible to recognise a distinct nucleoid". Also, "The ring-like particles had a more consistently spherical shape and were larger (140nm in diameter)" and the small particles "were unusually spherical but sometimes slightly angular in shape and 65 to 90nm in diameter" and had spike-like projections on their surface.[95] Hans Gelderblom who has done most of the EM studies in HIV/AIDS research reported that although *Lentiviruses* and thus HIV is considered to have a cone shaped core, he and his colleagues found centrosymmetric and tubular cores as well. The caption to one photograph reads: "Virions can be seen having either elongated, 'baton-like' tubular cores 30-35nm in diameter or containing more than one core. Tubular and regular cone-shaped cores can coexist within one virion". The text states: "Rarely, tubular core structures reminiscent of batons with a diameter of 30-35nm and a length of 150-250nm are observed".[20] Lekatsas and other virologists from Pretoria and Johannesburg reported: "We used the characteristic cylindrical structure in the core as an identifying characteristic for the virus to distinguish it from cellular debris and also noted that it may vary considerably in its dimensions and morphological features. We have found two basic virus particle sizes, 90nm and 120nm, both present in large numbers. The larger particle bears no surface projections while the smaller particle is rarely 'naked' and usually bears projections".[96] The US CDC reported: HIV particles are "usually round and have a diameter of about 85-95nm...Virus with bar-shaped nucleoids and particles with a tear-drop shape are commonly seen in HTLV-III/LAV infected lymphocytes, sometimes ring-shaped particles without dense nucleoids are also seen".[97] The question then arises if the particles with the "unique" morphology considered to be HIV represent an exogenous retrovirus originating from tissues of AIDS patients or those at risk, then what is the origin and role of the many non-HIV particles and which, if any, of the "HIV" or non-HIV particles band at 1.16 gm/ml? That is, which have the density characteristic of retroviruses? Retrovirus-like particles have been found in non-HIV-infected cord blood lymphocytes cell cultures and in cells used for HIV "isolation" such as and H9 (HUT-78), CEM, C8166 and EBV transformed B-cells.[98] Retrovirus-like particles antigenically related to HIV have been found in cultures of salivary gland extracts from patients with Sjorgen's syndrome.[99] In the only EM study,

either *in vivo* or *in vitro* in which suitable controls were used and in which extensive blind examination of controls and test material was performed, particles indistinguishable from "HIV" were found in 18/20 (90%) of AIDS as well as in 13/15 (87%) of non-AIDS related lymph node enlargements. This led the authors to conclude: "The presence of such particles do not, by themselves indicate infection with HIV".[100]
It is of pivotal significance to note that:

1. As Hans Gelderblom and his colleagues pointed out in 1998, to date nobody has reported the presence of "infectious plasma HIV".[101]

2. In no "HIV-infected" cultures are there particles which display both principle morphological characteristics of retroviruses, that is, "a diameter of 100-120nm" AND surfaces which "are studded with projections (spikes, knobs)". The electron microscopist agree that the "HIV" particles are devoid of knobs[95-98] and thus of the "HIV" protein gp120 (see below) said to be the constituent protein of the knobs.

Hans Gelderblom and his colleagues have estimated that immediately after being released from the cell membrane "HIV particles" possess an average of 0.5 knobs per particle which are rapidly lost, but also pointed out that "it was possible that structures resembling knobs might be observed even when there was no gp120 [knobs] present, i.e. false positives".[102] Yet all HIV experts agree that the infectivity of the HIV particles is determined by the gp120 (knobs). Thus, according to Montagnier *et al*, "The gp120 is responsible for binding the CD4 receptor"[103] while for Matthews and Bolognesi, "First gp120 binds to the CD4 receptor on an uninfected cell; then gp41 becomes anchored in the adjoining membrane; next the two membranes begin to fuse, and the virus spills its contents into the cell".[104] Callebaut *et al* state, "The human immunodeficiency virus (HIV) infects lymphocytes, monocytes, and macrophages by binding to its principal receptor, the CD4 molecule, through the viral envelope glycoprotein gp120. The V3 loop of gp120 is critical for HIV infection".[105] Others are in complete agreement.[106-111]

The general agreement that gp120 (knobs) is absolutely necessary for HIV particles to be infectious and the fact that this protein (knobs) is not present in the cell free particles leads to one unavoidable conclusion, that is, the "HIV" particles are not infectious, they are not viral particles. Furthermore,

1. To infect umbilical cord lymphocytes and the HUT-78 cells Montagnier and Gallo used cell-free fluids (supernatants). Montagnier cultured umbilical cord lymphocytes with supernatant from the cocultures of lymphocytes from BRU cultured with lymphocytes from a healthy individual. Gallo cultured the HUT-78 cell line with supernatants from the cultures of ten "infected" individuals. Even if the supernatants contained particles, being cell free the particles would be devoid of knobs (gp120), that is, they would not have been infectious. This means that even if Montagnier and Gallo had proof that their umbilical and HUT-78 cloned cultures contained particles with all the morphological characteristics of retroviruses, that in sucrose density gradients the particles banded at the 1.16 gm/ml band and the band contained nothing else but particles, the particles could not have originated from their patients. Furthermore, since the only samples which the Pasteur Institute gave to the Gallo laboratory[67] were cell free, one must question the basis upon which Gallo was accused of misappropriation of the French virus.

2. It is generally accepted that haemophiliacs are infected with HIV by contaminated factor VIII. However, to date nobody has reported the presence of "HIV" particles in plasma. Even if such particles were present the particles would be devoid of gp120. Since gp120 is "crucial to HIV's ability to infect new cells" it is not possible for haemophiliacs to be infected with HIV through factor VIII administration. (There is another fundamental reason why it is impossible for haemophiliacs to be infected with "HIV" from "contaminated" factor VIII. According to a publication from the CDC,[213] "In order to obtain data on the survival of HIV, laboratory studies have required the use of artificially high concentrations of laboratory grown virus...the amount of virus studied is not found in human specimens or any place else in nature,...it does not spread or maintain infectiousness outside its host. Although these unnatural concentrations of HIV can be kept alive under precisely controlled and limited laboratory conditions, CDC studies have shown that drying of even these high concentrations of HIV reduces the number of infectious viruses by 90 to 99 percent within several hours. Since the HIV concentrations used in laboratory studies are much higher than those actually found in blood or other body specimens, drying of HIV-infected human blood or other body fluids reduces the theoretical risk of environmental transmission to that which has been observed--essentially zero". Given that factor VIII is dispensed as a freeze-dried powder which spends many weeks or months waiting use, it is incomprehensible that the CDC and others continue to regard patients with haemophilia at risk for HIV infection via contaminated factor VIII concentrates and enigmatic that another explanation for "HIV" and AIDS in haemophiliacs has not been sought.[4]

The only conclusion one can draw from the electron microscopy data is that the reported particles are non-HIV or even retroviral specific which means that the detection of such particles, be it in an unlimited number of consecutive cultures/cocultures, is not proof for isolation, no matter how isolation is defined.

Furthermore:

1. Even if reverse transcriptase activity and retrovirus-like particles are specific to retroviruses, they are not specific for a unique retrovirus. The only evidence both groups presented for the existence of a unique retrovirus, HIV, is the antigen/antibody reaction, as acknowledged by both Montagnier and Gallo.

2. It is accepted that the finding of a retrovirus in culture, especially under the conditions used by Montagnier and Gallo is not proof that the retrovirus is present *in vivo*. The only evidence which both Gallo and Montagnier's groups presented for the existence of HIV *in vivo* was the antigen/antibody reaction.

3. Even today the only evidence given as proof that HIV is the cause of AIDS is a "correlation" between the antibody test and the appearance of AIDS.

   It is obvious that:

   (a) the Montagnier and Gallo interpretation of the antigen/antibody reaction is crucial for the HIV hypothesis of AIDS and in fact for the existence of a unique retrovirus, HIV;

   (b) if the interpretations are not correct, one would have no choice but to question not only the HIV hypothesis of AIDS but also the existence of HIV.

It has already been mentioned that the mere interaction between an antigen such as a protein or a virus and an antibody does not prove any more than a chemical relationship. In all other aspects the reaction may be totally nonspecific. To prove the specificity of the reaction one must employ an alternative, independent method of proving the existence of the antigen, that is, one must use what is generally known as a gold standard. The only possible gold standard for the HIV antibody tests (the antigen/antibody reaction) is HIV itself, that is, HIV isolation. No matter how one defines isolation, obviously the definition cannot include the antibody/antigen reaction (the antibody test, neither the WB nor the ELISA) because, by doing so, not only does one not have an independent analysis, the test becomes its own gold standard and is thus rendered meaningless. Reading the 1984 Gallo *Science* papers it appears that Gallo always defined isolation as "more than one of the following: reverse transcriptase activity either in the supernatant or the 1.16 gm/ml band; retrovirus-like particles in the culture or reaction between proteins (either in the cultured cells or the 1.16 gm/ml band) with antibodies present in the patients sera or "antiserum to HTLV-III". However, in an interview Gallo gave to Huw Christie, the editor of the British magazine *Continuum*, at the 1998 Geneva AIDS Conference, Gallo said, "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 because we could isolate the virus when we did it".[112] In other words:

1. In 1984 Gallo knew that to prove the specificity of the antibody test one must use a gold standard.

2. The gold standard had to be HIV isolation.

3. Although it is not known how he and his colleagues initially defined isolation, the definition did not include the antibody/antigen reaction (the Western Blot). This means that Gallo and his colleagues were aware that an antibody/antigen reaction cannot be used as proof for isolation. Yet, by the time their *Science* papers were published, and in order to reconcile the low correlation between what they initially called isolation and the antibody/antigen reaction, arbitrarily, and against all scientific reasoning, they "added the Western Blot" to their definition of isolation. (It is interesting that even with their novel definition the correlation between "isolation" and antibody tests was still less than perfect. They "isolated" HIV from 26/72 (36%) of patients with AIDS while 88% of patients with AIDS were seropositive using an ELISA test Gallo considered highly specific[62]).

In an interview which Montagnier gave to the French Journalist Djamel Tahi in 1997, he stated: "analysis of the proteins of the virus demands mass production and purification. It is necessary to do that". Indeed, the only way to prove that a protein is a viral constituent is to obtain it from material which contains nothing else but viral particles. Instead Montagnier's and Gallo's groups incubated the proteins which banded at 1.16 gm/ml with sera from AIDS patients. The proteins which were found to react more often with the sera were said to be HIV proteins (although neither group published any evidence for the existence of retrovirus-like particles, pure or

impure, at the 1.16 gm/ml band); and the antibodies to be HIV antibodies. However, even if the antigen/antibody reaction is 100% specific, that is, antibodies react only with inducing antigens and with no other, from such a reaction it is impossible to determine the origin even of one reactant much less the origin of both the antigen and the antibody. Let us consider differing scenarios:

1. Both Gallo and Montagnier had proof that the 1.16 gm/ml band contained nothing else but retrovirus particles and that their antibodies reacted specifically with their proteins. According to Montagnier, in the cultures which contain cells originating from patients with adult T4 cell leukaemia, as did Gallo's HUT-78 cell line, "it is a real soup" of retroviruses. Indeed, even if the cultures do not harbour HIV they will harbour HTLV-I and, given the condition used by Gallo and the fact that the cells were leukaemic, they may also contain endogenous retroviruses.[35,113-115] According to one well known HIV expert, Myron Essex, 35% of AIDS patients possess antibodies to HTLV-I.[116] (In the same issue of *Science* in which Montagnier published his isolation of "HIV", Gallo published three papers claiming isolation of HTLV-I from AIDS patients suggesting this retrovirus was the cause of AIDS). According to another equally as well known HIV expert, Reinhard Kurth, from the Paul-Ehrich Institute in Germany, 70% of "HIV-positive patients" have antibodies which react with the retrovirus HTDV/HERV-K, an endogenous retrovirus, or, as Kurth put it, a retrovirus present "in all of us".[35] Thirty seven per cent of HIV positive individuals were also found to have antibodies to type D retroviruses whereas HIV is claimed to be a *Lentivirus*.[117] Although Montagnier's cultures may not have contained HTLV-I, the result may still have been "a real soup" of endogenous retroviruses especially if one considers their culture conditions and the cells used, umbilical cord lymphocytes, which have been shown to release retrovirus-like particles even when not infected with HIV.[98] Since we cannot obtain the proteins from each particle and characterise them, the next best thing is to take the mass of particles, disrupt their proteins and position them in an electrophoretic strip according to their molecular weights. Although we know that the proteins in the strip are retroviral, we have no way of determining which protein belongs to which retrovirus if more than one retrovirus is present at the 1.16 gm/ml band. When the proteins are incubated with sera, we may find some of the proteins react. From such a reaction we will be able to say that the antibodies and obviously the proteins are viral but we cannot determine which protein belongs to which virus and by which virus the antibodies were induced. We will be definitely wrong if we consider such a reaction proof that the 1.16 gm/ml band (a) contains only one retrovirus; (b) the retrovirus is a new exogenous retrovirus, HIV; (c) the proteins are the HIV proteins; (d) the antibodies are HIV antibodies; (e) these data are proof that the patient is infected with HIV even if the antibody antigen reactions are specific.

2. Both Montagnier and Gallo had proof that the vast majority of the 1.16 gm/ml band was composed of retrovirus particles but that the band also contained non-retroviral material. This material could have been of cellular origin (cellular constituents also band at 1.16 gm/ml[38,57,72]) and may be of bacterial, fungal and viral origin (constituents of the many infectious agents other than retroviruses, known to be present in the cultures and the patients). It is a fact that in the USA, Europe and Australia individuals with AIDS as well as those at risk have antibodies to many infectious agents including viruses such as EBV, CMV and hepatitis B virus. Evidence also exists which shows that individuals with AIDS and those at risk have circulatory immune complexes, rheumatoid factor, anti-nuclear, anti-cellular, anti-platelet, anti-red cells, anti-actin, anti-tubulin, and anti-myosin antibodies.[118,119] Montagnier himself demonstrated that individuals with AIDS and those at risk have high levels of antibodies to the ubiquitous cellular protein actin whose molecular weight is 41,000, as well as to another ubiquitous protein, myosin, which has two sub-units of molecular weights, 18,000 and 25,000.[118] Anti-lymphocyte antibodies have been found in 87% of patients who have a positive "HIV" antibody test and their levels correlate with clinical status.[120] It is also acknowledged that Africans with AIDS and those at risk are infected with many agents other than HIV.

We know that it is not possible to take a protein from the 1.16 gm/ml mass and know from which component it originates. Let us then follow the same steps as Gallo and Montagnier. Take the mass of material banding at the 1.16 gm/ml, disrupt the proteins and electrophoretically position the proteins in a strip according to their molecular weights. Using this technique we are unable to state from which component of the 1.16 gm/ml band mass a protein on the strip derives. Next we incubate the proteins in the strip with patient sera and discover that some of the proteins react with antibodies present in the sera. Even if the antibody/antigen interaction is 100% specific such a reaction does permit us to define the origin of the proteins. Yet from such a reaction and without any proof that the antibodies specifically reacted, Montagnier and Gallo defined the origin of both the proteins and the antibodies as “HIV”.

As mentioned, Montagnier found three proteins in the 1.16 gm/ml band which reacted with antibodies present in his patients’ sera. These were p25, p80 and p45. Gallo found two proteins, p24 and p41. Montagnier concluded

that his patients were infected with a retrovirus which "contains a major p25 protein, similar in size to that of HTLV-I", but made no comment in regard to the p80 protein. Regarding p45 he wrote: "The 45K [p45] protein may be due to contamination of the virus by cellular actin".[58]

In 1997 Montagnier said that the protein he detected in 1983 had a molecular weight of 43,000 and was actin.[29] (The molecular weight of actin is neither 45,000 nor 43,000 but 41,000. However since Montagnier and Gallo determined the molecular weights of the proteins by their migration in an electrophoretic strip, and because the migration may be influenced by other factors, for example, by the protein's charge, it is possible that these slight differences in the molecular weight are simply the result of experimental variation).

When Gallo and his colleagues used the cellular proteins as antigens in the WB they reported: "The most prominent reactions were the antigens of the following molecular weights: 65,000, 60,000, 55,000, 41,000 and 24,000. Antigens with molecular weights of approximately 88,000, 80,000, 39,000, 32,000, 28,000 and 21,000 gave less prominent reactions…A large protein with a molecular weight of approximately 130,000 and a protein of 48,000 were also detected". In another experiment the "antigens from virus purified from the culture fluids", that is, the 1.16 gm/ml band, were incubated with different sera. They found an "Extensive" reaction of the AIDS patients sera with p24 and p41 and concluded: "these molecules are the major component of the virus-preparation. P24 and p41 may therefore be considered viral structural proteins…Furthermore, an antigen with a molecular weight of approximately 110,000 was detected in the virus preparation but was below limit of detection in the cells. Also, p39 was present in the virus preparation…Occasionally an additional set of antigens was recognised by a serum but their relation to the antigens described above is unclear".[63]

Between 1983-87 the detection of antibodies in patient sera which reacted with p24 or p41 (Montagnier considered reaction with p24 and Gallo with p41 HIV specific) was considered proof that the patient was infected with HIV. In the same period of time it became obvious that a significant number of individuals at no risk of AIDS had antibodies which reacted with these proteins. Since 1987 most of the proteins which Gallo found to react either in the cell extracts or the 1.16 gm/ml band are now considered HIV proteins[121] and laboratories require the presence of antibodies which react with more than one protein before the patient is considered infected with HIV. The number and identity of antibody/protein (Western blot) bands required vary from continent to continent, from country to country and even between and within laboratories in the same country. Thus it is possible for the same patient to be HIV seropositive in New York for example, but not in Africa or Australia (Figure 1.1 in Part I). However at present evidence exists which shows that the "HIV" proteins which react with antibodies present in patient sera are in fact cellular proteins.

The inescapable confusion is that the antibody/antigen reaction is not HIV specific and thus cannot be used to prove HIV isolation, no matter how isolation is defined.

ORIGIN OF THE "HIV" PROTEINS

The p41 protein

Although Montagnier and his colleagues found a protein p45 (p41) in their "purified" virus[58,122] and the protein reacted with antibodies present in the patients' sera, they concluded that the protein was not viral but the cellular protein actin. Since then many researchers reported the presence of actin in "HIV"[123-127] Indeed some of the best known HIV experts acknowledge that the proteins with molecular weight of approximately 41,000 present in "HIV" are in fact actin.[128]

The p24/p25 protein

At present there is ample evidence that antibodies which react with p24 are common in both human and animal sera, which can only be interpreted as that either p24, the antibodies, or both, are non-HIV-specific or a significant proportion of both humans and animals are infected with HIV. For example, if the interaction between p24 and the antibodies is considered proof for HIV infection then about 30% of individuals who are transfused with HIV negative blood become infected as a result.[129] Since, according to the AIDS vaccine Clinical Trials Group[130] "The presence of p24 was common among low-risk, uninfected volunteers and complicated the interpretation of the Western blot results", HIV infection should be common among healthy, no risk individuals. In fact, because of such evidence, since 1987, with perhaps only two exceptions, Montagnier and researchers conducting the Multicenter AIDS Cohort Study in the United States, no laboratory anywhere in the world considers a reaction between the p24 in the WB and antibodies present in sera proof of HIV infection. Yet, when the same reaction takes place between an antibody to p24 and a patient's serum, it is considered proof of viraemia, and when between an antibody and material present in a cell culture, the same reaction is considered proof of HIV isolation. In fact since 1987 this reaction has been the method of choice for "HIV" isolation by the vast majority of laboratories. However, the non-specificity of the p24 antigen test is obvious and is accepted by no

less an authority on HIV testing than Philip Mortimer and his colleagues from the UK Public Health Laboratory Service, "Experience has shown that neither HIV culture nor tests for p24 antigen are of much value in diagnostic testing. They may be insensitive and/or non-specific".[131]

The reaction of a protein, even if known to be HIV protein with antibodies is not proof for viral isolation, "viraemia" or "viral load". That such a finding is also non-specific can be best illustrated by a few examples. In 1992, Jorg Shupbach, the principle author of one of the first four 1984 *Science* papers published by Gallo's group on HIV isolation, reported that the whole blood cultures of 49/60 (82%) of "presumably uninfected but serologically indeterminate individuals and 5/5 seronegative blood donors were found positive for p24".[132] If p24 is an HIV protein then it must be present in all AIDS patients if not all seropositive patients and not in persons not at risk of developing AIDS. Yet Jackson *et al*, who claim an overall 98.3% "HIV isolation" rate, are unable to detect p24 in serum of 58% of AIDS patients, 63% of ARC patients and 83% of asymptomatic seropositive individuals.[133] This rate of detection is much lower than in non-HIV-infected organ transplant recipients. "In one kidney recipient (the donor was negative for p24 antigen) who, three days following transplantation developed fever, weakness, myalgias, cough and diarrhoea, all bacterial, parasitological and virological samples remained negative [including HIV PCR]. The only positive result was antigenaemia p24, positive with Abbot antigen kits in very high titers of 1000pg/ml for polyclonal and 41pg/ml for monoclonal assays. This antigenaemia was totally neutralised with Abbot antiserum anti-p24…2 months after transplantation, all assays for p24-antigen became negative, without appearance of antibodies against HIV. Five months after transplantation our patient remains asymptomatic, renal function is excellent, p24 antigenaemia still negative and HIV antibodies still negative.[134] In one study, p24 was detected transiently in 12/14 kidney recipients. Peak titres ranged from 850 to 200 000 pg/ml 7-27 days post-transplantation. Two heart and 5/7 bone marrow recipients were also positive, although the titres were lower and ranged from 140-750 pg/ml. Disappearance of p24 took longer in kidney (approximately 6 months) than in bone-marrow (approximately 4-6 weeks) recipients. According to the authors: "This may be related to differences in immunosuppression therapy". Discussing their findings they wrote: "The observation of a 25-30kD protein binding to polyclonal anti-HIV human sera after immunoblots with reactive sera raises several questions. This protein could be related to a host immune response to grafts or transplants…Its early detection after transplantation might indicate the implications of immunosuppression therapy…the 25-30kD protein could therefore be compared with the p28 antigen recently described with human T-cell-related virus lymphotropic-endogenous sequence…The characterisation of this 25-30kD protein may represent an important contribution to the detection of HIV-1-related endogenous retroviruses".[135]

Ninety-seven percent of sera from homosexuals with immune thrombocytopenia (ITP) and 94% of sera from homosexuals with lymphadenopathy or AIDS contain an antibody that reacts with a 25kD membrane antigen found in platelets from healthy donors and AIDS patients, as well as a 25kD antigen found in green-monkey kidney cells, human skin fibroblasts, and herpes simplex cultured in monkey kidney cells. This reaction was absent in sera obtained from non-homosexual patients with ITP or non-immune thrombocytopenic purpura".[136] Using monoclonal antibody p24 has also been found a constituent of the normal human placenta.[137]

As far as Montagnier is concerned, p24 is the crucial HIV protein. However, if p45 (p41) which also bands at 1.16 gm/ml and reacts with antibodies present in patient sera is the cellular protein actin, just because actin is ubiquitous, and has the same molecular weight, why should not p24 be the equally as ubiquitous protein, myosin, which is known to have a sub-unit of the same molecular weight? Especially if one considers the presently available evidence which shows that like actin, myosin is present in the "HIV particles"[123-127] and that Montagnier himself demonstrated that individuals with AIDS and those at risk have high levels of antibodies to this protein.[118]

The p32 protein

In 1987 Henderson isolated the p30-32 and p34-36 of "HIV purified by double banding" in sucrose density gradients. By comparing the amino-acid sequences of these proteins with Class II histocompatability DR proteins, they concluded that "the DR alpha and beta chains appeared to be identical to the p34-36 and p30-32 proteins respectively".[138] That these proteins are cellular is acknowledged by other HIV experts.[128]

The p17/p18 protein

Sera from AIDS patients bind to a p18 protein mitogenically stimulated "HIV" infected T-cells but not to uninfected, unstimulated lymphocytes. However, when the lymphocytes are mitogenically stimulated but uninfected, the AIDS sera bind to a p18 protein in these uninfected lymphocytes.[139] A monoclonal antibody to "HIV" p18 reacts with dendritic cells in the lymphatic tissues of a variety of patients with a number of non-AIDS related diseases[140] and the "same pattern reactivity was present in normal tissue taken from uninfected

individuals as in those taken from HIV positive subjects".[53] It is of interest that one of myosin's two sub-units has a molecular weight of 18,000. The p17/18 protein is also present in the normal human placenta.[137]

The p160 and p120 proteins

The general agreement amongst the HIV experts is that p120 and p41 are cleavage products of p160, and that p160 is found only in the "infected" cells but not the "purified" virus, that is, the 1.16 gm/ml band. As mentioned, p120 (which in 1984 Gallo reported as p110), is said to be present only in the "HIV" particles knobs, spikes, and to date no electron microscopist could prove the existence of such knobs on cell-free "HIV" particles. Yet under certain experimental conditions, p120 and p160 are found in the electrophoretic strips prepared from proteins of the "purified" virus. According to Burke, "Most Western blot strips prepared in the United States before 1987 lacked appreciable bands corresponding to the high molecular weight viral envelope glycoproteins (gp120 and its precursor gp160). Underrepresentaion of these proteins was probably owing to several factors, especially by ultracentrifugation, and by denaturing of antigenic activity during electrophoresis and transblotting. Since early 1987 blot preparation methods have been modified to ensure that the high molecular weight envelope bands can be clearly identified with most patient sera".[121] However, no amount of "blot preparation" modification are able to create what is not present. The explanation for the presence of the "high molecular weight envelope bands" was discovered in 1989 by researchers from New York who showed that in the Western blot strip, "the components visualised in the 120-160kDa region do not correspond to gp120 or its precursor but rather represent oligomers of gp41".[141] It was also shown that the WB pattern obtained is dependent on many factors including temperature and the concentration of sodium dodecyl sulphate used to disrupt the "pure virus". "Confusion over the identification of these bands has resulted in incorrect conclusions in experimental studies. Similarly, some clinical specimens may have been identified erroneously as seropositive, on the assumption that these bands reflected specific reactivity against two distinct viral components and fulfilled a criterion for true or probable positivity. The correct identification of these bands will affect the standards to be established for Western blot positivity: it may necessitate the reinterpretation of published results".[142] Little, if any notice was taken of these findings and recommendations. In fact, in Africa, the finding of antibody reactivity in an individual's serum with any two of p160, p120, p41, that is, antibodies to actin and its polymers, is considered proof that Africa is in the grip of an epidemic of HIV induced immunodeficiency. Also, as is the case of both p24 and p18, p120 is present in the normal human placenta.[137]

Even if proof exists that the "HIV" proteins do indeed belong to a unique exogenous retrovirus, it cannot be assumed that antibodies that react with them are diagnostic of HIV infection. To prove the specificity of the antibody tests it is mandatory to:

(a) test a large number of subjects with and without AIDS. The subjects without AIDS must not exclusively be healthy individuals (since they do not have high levels of antibodies, one would expect few if any reactions) but include the sick, such as patients with infectious diseases (other than those which are said to result from HIV infection), those receiving chemotherapy and those with auto-immune disorders;

(b) simultaneously (preferably on the same blood sample) perform tests for HIV isolation;

(c) compare the antibody test results with the results of HIV isolation, that is, use HIV as a gold standard for the antibody test.

To date nobody has published such studies using any definition of HIV isolation. However, the fact that such antibodies are present in sera obtained from humans accepted not be infected with HIV demonstrates that the antibodies are non-specific:

1. Although they did not comment (Montagnier commented only on p41), both Montagnier and Gallo found many proteins in the "infected" cells which reacted with antibodies present in both AIDS patients and "healthy donors". If the antibodies in the AIDS patients were HIV antibodies what were the antibodies in the healthy donors?

2. The first antibody tests in Africans were performed in 1984 by Montagnier and nineteen of his associates including researchers from the CDC.[143] The sera were tested by ELISA and then by a radioimmunoprecipitation assay (a procedure similar to the Western blot). The latter was considered positive if a p24 band was present. The p41 band and also an 84-kDa band were not considered diagnostic because "The 43-kD [p41] band and the 84-kDa band are cellular contaminants that are immunoprecipitated in all the tested sera", from both patients and controls. (Yet today, in Africa, the p41 and its polymers, on its own is considered to represent a positive WB and thus proof of HIV infection). Thirty-two patients (88%) were positive by both tests. So were six out of 26 (23%) healthy controls.

3. Biggar and his colleagues were among the first to raise the possibility that, at least in Africans, the antibody tests may not be 100% specific, as was generally believed. They found that in healthy Africans the probability of finding a positive HIV antibody test increased significantly with increasing immune-complex levels. They concluded that "reactivity in both ELISA and Western blot analysis may be non-specific in Africans...the cause of the non-specificity needs to be clarified in order to determine how they might affect the seroepidemiology of retroviruses in areas other than Africa, such as the Caribbean and Japan...Serological studies from Africans would need to be re-evaluated with a more specific test before conclusions can be drawn".[144] In the same year, 1985, one of the best known HIV experts, Robin Weiss, and his colleagues, accepted that African sera "may give a false-positive result on direct binding assay systems, or on Western blots".[145] One year later some of the best known experts on HIV/AIDS in Africa expressed the view that in Africa "...serodiagnosis is complicated by the need for confirmatory tests because of the presence of possible cross-reacting antibodies".[146] However, experiments to determine the specificity of the "HIV" antibodies in Africans or anywhere else have never been reported. In fact, AIDS reporting in Africans is based on clinical grounds without the requirement for antibody testing or immunological function.[147]

4. In a study conducted in Africa[148] 83% of patients with suspected AIDS were HIV positive, but so were 44% with malaria, 97% with herpes zoster, 43% with pneumonia, 67% with amoebic dysentery and 41% with carcinoma. In another study 42% of women with recurrent abortions, 67% with vaginal ulcerations and 33% with haemorrhoids had a positive HIV antibody test.[149] In 2001 Ghosh reported that of 2/33 (6%) of patients with "Severe *P. falciparum* malaria" had a false positive HIV ELISA, considered to be caused by "intense, non-specific immune stimulation".[150]

5. In 1985 Gallo and his colleagues reported testing a number of sera collected in 1972/73 from the West Nile district of Uganda. These were obtained from healthy children randomly selected as controls for a study of Burkitt's lymphoma. Their mean age was 6.4 years and both ELISA and WB were performed . Fifty of the 75 children (67%) were found to be positive. According to HIV experts these positive results are explicable by virtue of mothers infecting their children. Thus Gallo and his colleagues expected to find at least an equal percentage of infected adults. Mortimer *et al* assert that "Very few HIV-infected children are surviving into adulthood in good health" and, given the fact that neither these children nor adults had treatment for HIV or AIDS, and the incubation period of AIDS in Africa is claimed to be four years and HIV heterosexually transmitted, then if the tests are HIV specific and if HIV causes AIDS, by now, few, if any Ugandans should be alive.[151]

6. One of the principal major signs of the Bangui definition of AIDS in Africa is loss of body weight. However, in a study of Rwandan women, over a 24 month period beginning in 1988, it was reported that nutritional status assessed by loss of body weight "was a significant predictor of eventual HIV seroconversion. Subsequent seroconvertors lost an average of 1.5 kg during the six months of the study compared with 1.0 kg gain ($p = 0.001$) for non-converters. Nine of 27 (33%) seroconvertors., compared with one of 44 (2%) controls, lost at least 5 kg in the 6 month period beginning one year before seroconversion...In addition to those findings for measured weight loss during follow-up, reported weight loss before enrolment was also a risk factor for subsequent seroconversion".[152] In other words, the effect (weight loss) has preceded the cause (HIV) by many months or even years.

7. In 1986, Jaffe *et al* tested 1129 serum samples from IV drug users and 89 controls from non-users. All samples were collected during 1971-1972 and tested by two commercial ELISAs and WB. Seventeen of the samples from the IV drug users, but not one of the controls was found positive. They concluded: "On the basis of our positive Western blot data, it appears that parenteral drug users may have been exposed to HTLV-III or a related virus as early as 1971. An alternative but equally viable explanation is that the HTLV-III seropositivity detected in these specimens represented false positive or non-specific reactions".[153]

8. In 1991, Elizabeth Dax and associates from the US National Institute on Drug Abuse HIV reanalysed 1985 Western blot strips of sera originally obtained from intravenous drug addicts in 1971-72. (Twenty years later the actual sera themselves were not available for retesting). Ten persons "with potentially positive WB patterns, when the more specific 1985 criteria were used", were traced. One patient had died from a motor vehicle accident and there were "no lymphoreticular changes at autopsy, and a thorough retrospective analysis provided no evidence of either current substance abuse of HIV infection". Of the nine living addicts, two could not be assessed clinically, seven were not chronically ill, (one was in prison but in good health, one had been successfully discharged from a methadone program, one was enrolled in a methadone

program, another sporadically consumed illicit drugs). "The two former patients whose 1971-72 WB results were most strongly reactive had current ELISA and WB assays that were negative. The immune function parameters were inconsistent with immune suppression". Their data led the authors to conclude, "it is possible that antibodies to a nonpathogenic virus would have disappeared during the 17 to 18 years...follow-up. Although this potential cannot be ruled out, it is more likely that the earlier results were false positives...definitive evidence of HIV infection in the United States' addict population as early as 1972 is still lacking".[154]

9. HIV is said to be transmitted by infected needles, yet a higher percentage of prostitutes who use oral drugs (84%) than intravenous (46%) have positive "HIV" antibody tests.[155]

10. According to the HIV experts, once infected with HIV, unless treated, always infected. Yet, in healthy individuals, partners of HIV positive individuals, organ transplant recipients and patients with systemic lupus erythematosus, a positive WB may revert to negative when exposure to semen, immunosuppressive therapy or clinical improvement occurs.[156,157]

11. Amazonian Indians who have no contact with individuals outside their tribes and have no AIDS have a 3.3-13.3% HIV WB seropositivity rate depending on the tribe studied.[158] In another study they found that 25%-41% of Venezuelan malaria patients had a positive WB, but no AIDS.[159] The above data means either that HIV is not causing AIDS "even in the presence of the severe immunoregulatory disturbances characteristic of acute malaria", as concluded, or the HIV antibody tests are non-specific.

12. Lundberg and his colleagues from the US Consortium for Retrovirus Serology Standardisation reported that 127/1306 (10%) of individuals at "low risk" for AIDS including "specimens from blood donor centres" had a positive HIV Western blot by the "most stringent" of the US criteria, that is, the presence of antibodies to p24, p32 and gp41 or gp120/160.[160]

13. p24 seroreactivity "was found in 27 (35%) of 77 patients with primary biliary cirrhosis, 14 (29%) of 48 patients with systemic lupus erythematosus, 14 (50%) of 28 patients with chronic viral hepatitis, and nine (39%) of 23 patients with either primary sclerosing cholangitis or biliary atresia, compared with only one (4%) of 24 patients with alcohol-related liver disease or alpha 1 - antitrypsin-deficiency liver disease, and only one (4%) of 25 healthy volunteers ($p = 0.003$)".[161]

14. An individual was given six 5 ml injections of donated Rh positive serum, administered at 4 day intervals. His "wife and child were seronegative on HIV ELISA". The donor serum "was shown to be negative on HIV antibody and antigen ELISA". "Blood taken after the first immunisation was shown to be negative on HIV antibody ELISA and immunoblot assay. After the second immunisation a weak signal on ELISA, slightly above the cut-off level, was monitored. After the third immunisation the signal was strong and immunoblot revealed distinct interaction with p17 and p55 proteins. An even stronger signal was monitored after the fifth immunisation. Interaction with p17, p31, gp41, p55 and some other proteins was evident.[162]

15. 11/208 (5%) of healthy blood donors and 10/50 (20%) of patients with measles, mumps, herpes simplex, dengue and other viral illnesses had either a p24 or p18 band on the HIV Western blot test.[163]

16. The "HIV proteins (p17, p24)" appear in the blood of patients (previously negative for all HIV markers) following "transfusions of HIV-negative blood and UV-irradiation of the autoblood".[164]

17. In 1991 Kion and Hoffman injected non-HIV -infected mice with T-lymphocytes from another strain of non-HIV-infected mice. The recipient mice developed antibodies to the HIV gp120 and p24 proteins.[165]

18. In 1991, Strandstrom and colleagues reported that 72/144 (50%) of dog blood samples "obtained from the Veterinary Medical Teaching Hospital, University of California, Davis" tested in commercial Western blot assays, "reacted with one or more HIV recombinant proteins [gp120--21.5%, gp41--23%, p31--22%, p24--43%].[166]

19. The Australian National HIV Reference Laboratory 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".[167]

20. Amongst 89,547 anonymously tested blood specimens from 26 US hospital patients at no risk of AIDS, between 0.7% to 21.7% of men and 0-7.8% of women aged 25-44 years were found to be HIV WB positive. It is important to note that this study not only excluded patients from the known AIDS risk groups but also those with even meagre HIV/AIDS risks including "gunshot and knife wounds, conditions which have been reported to be associated with a higher than expected rate of HIV-1 seroprevalence".[168]

21. In the USA, in a larger study of 1.2 million healthy military applicants, approximately 1% of all initial ELISAs were positive of which 50% were subsequently negative; 30-40% of first WB were positive and 96% of second WB were positive. In other words there were 6,000 individuals with an initially positive but subsequently negative ELISA, 4,000 individuals with two positive ELISAs followed by a negative WB, and 80 individuals with two positive ELISAs, an initially positive WB and a negative repeat WB. Thus several thousand healthy individuals were found to have antibodies that reacted with "HIV proteins" but who were ultimately deemed not to be HIV infected. Even in the best laboratories 80 of the healthy applicants would be diagnosed as HIV infected since only one WB is performed and it is considered 100% specific. The situation in Africa would be even worse since in Africans two positive ELISAs are considered 100% specific for HIV infection.

Anyone drawing a conclusion regarding the existence of HIV from an antibody/antigen reaction must not forget a lesson from history. In the mid-1970s, Gallo and his colleagues reported the isolation of the “first” human retrovirus, HL23V. In fact, the evidence for the “isolation” of HL23V surpassed that of HTLV-I and HIV in at least two aspects. Unlike HIV, Gallo's group: (a) reported the detection of reverse transcriptase activity in fresh, uncultured leucocytes, and (b) published an electron micrograph of virus-like particles banding at a sucrose density of 1.16 gm/ml, the density which defines retroviral particles. Following the discovery of HL23V, some researchers attempted to determine its prevalence utilising antibody tests while others were interested in determining the specificity of the antibody reactions. The latter included one group from the Laboratory of Cellular and Molecular Biology, National Cancer Institute, and another from the Laboratory of Viral Oncology, Memorial Sloan-Kettering Cancer Center. Using the “viral glycoproteins”, these groups found that the antibodies present in human sera which reacted with these proteins were “directed against carbohydrate structures” and concluded that “The results are consistent with the idea that the antibodies in question are elicited as a result of exposure to many natural substances possessing widely cross-reacting antigens and are not a result of widespread infection of man with replication competent oncoviruses” (retroviruses).[169,170] In 1981 Gallo accepted the evidence that the antibodies which reacted with the presumed viral proteins of HL23V were not so directed “but against the carbohydrate moieties on the molecule that are introduced by the host cell as a post-transcriptional event, and which are therefore cell-specific and not virus-specific”.[171] This discovery was of such significance that today nobody, not even Gallo, considers HL23V as being the first human retrovirus, or even a retrovirus.

There is compelling evidence that “HIV” antibodies also arise from exposure to “widely cross-reacting antigens” and are not a result of infection with a novel retrovirus.

1. “One half of the molecular weight of gp120 is represented by oligomannosidic oligosaccharides...Polyclonal antibodies to mannan from yeast also recognise the carbohydrate structure of gp120 of the AIDS virus.[172]

2. “The immunochemical determinants of the antigenic factors of *Candida albicans* display a high identity with the glycoprotein (gp) 120 of HIV-1: they contain _(1_2) and _(1_3)-linked mannose terminal residues”.[173]

3. Antibodies to the mannans of *Candida albicans* “block infection of H9 cells by HIV-1” as well as the binding of lectins to gp120.[173]

4. Recognition of gp120 by antibodies to a synthetic peptide of the same antigen was “partially abolished if it was absorbed with the total polysaccharide fraction of *C. albicans*” while the antigen recognition by antibodies to “gp120 from human T-cell lymphotropic virus type IIIB...was totally blocked”. From these data the authors concluded: "These results indicate that mannan residues of *C. albicans* can serve as antigens to raise neutralising antibodies against HIV infection".[173]

5. “Normal human serum contains antibodies capable of recognising the carbohydrate moiety of HIV envelope glycoproteins...from 100 ml of human serum approximately 200µg of MBIgG was recovered [MBIgG = mannan-binding IgG antibodies]...MBIgG bound to HIV envelope glycoproteins gp160, gp120 and gp41”.[174]

6. Kashala, Essex and their colleagues have shown that antibodies to carbohydrate-containing antigens such as lipoarabinomannan and phenolic glycolipid that constitute the cell wall of *Mycobacterium leprae*, a bacterium which "shares several antigenic determinants with other mycobacterial species" cause "significant cross-reactivities with HIV-1 pol and gag [p32, p55, p68, p24, p18] proteins". This led the authors to warn that among leprosy patients and their contacts there is a "very high rate of HIV-1 false-positive ELISA and WB results", that "ELISA and WB results should be interpreted with caution when screening individuals infected with *M. tuberculosis* or other mycobacterial species", and furthermore that "ELISA and WB may not be sufficient for HIV diagnosis in AIDS-endemic areas of central Africa where the prevalence of mycobacterial diseases is quite high".[175]

7. Not only mycobacteria (*M. leprae, M. tuberculosis, M. avium-intracellulare*) but also the walls of all fungi (*Candida albicans, Cryptococcus neoformans, Coccidioides immitis, Histoplasma capsulatum,* including *Pneumocystis carinii*), contain carbohydrate (mannans). One hundred per cent of AIDS patients (even those with "no *Candida* clinically") have *C. albicans* antibodies, leading researchers from St. Bartholomew's and St. Stephen's Hospitals to state: "It is possible that Candida may act as a cofactor in the development of overt AIDS in HIV-infected individuals".[176] It is significant that in gay men the only sexual act which is a risk factor for seroconversion is passive anal intercourse[177] (exposure to semen) and that mannose is present in both sperm and seminal plasma.[178]

8. Researchers from the University of Rome injected healthy mice with an *E. coli* lipopolysaccharide (LPS) and reacted their sera with two synthetic peptides, one encompassing gp120 V3 loop of "HIV-1 MN" and the other "representing a gp41 immunodominant epitope". The LPS-treated mice showed a "significant antibody reactivity" with the two peptides. (V. Colizzi *et al*, personal communication).

9. In the same study, the authors reported data from the sera of HIV-negative subjects with autoimmune disorders. Recombinant gp120 and a panel of synthetic peptides derived from the amino acid consensus sequences of the HIV gp120, gp41, p24 or several unrelated proteins were tested by specific ELISA. "The first set of experiments performed on four patients with Sjorgern syndrome (SjS) and four patients with systemic lupus erythematosus (SLE) revealed a significant anti-gp120 reactivity compared to healthy HIV-negative controls. Moreover, such binding could be almost completely inhibited by preincubation with free gp120. A significant anti-p24 reactivity was observed in 18 out of 29 [62%] sera from SjS patients and in 13 out of 25 [52%] from SLE patients, while anti-gp41 was observed only in 3 out of 14 [21%] SjS and in 2 out of 20 [10%] SLE affected patients. Similar analyses were performed in the murine model of autoimmunity, showing that sera from MRL/lpr mice were able to bind all HIV related peptides in age-dependent manner. The analysis of a panel of HIV unrelated peptides showed that SLE as MRL/lpr sera bind both HIV related and unrelated peptides while SjS sera failed to do so". In other words, sera which contain autoantibodies react with the principal "HIV" proteins gp120, gp41 and p24.[179]

10. The same authors also reported similar results from (i) experiments where "Two month old male CBA mice were immunised for 6 weeks with 50 x 10^6 allogenic lymphoid cells obtained from either BALB/c or B6 male mice"; (ii) "Sera from 62 polytransfused (at least 10 transfusions/year) patients with thalassemia".

Since antibodies to mannans react with the "HIV" proteins then, as Essex and his colleagues pointed out, for mycobacterial infection in Africa, one would expect the sera of all people infected with fungi and mycobacteria to cross-react with the "HIV-1 glycoproteins" as well as to cause "significant cross-reactivities with HIV-1 *pol* and *gag* proteins". Since humans and animals subjected to non-infected blood and blood products develop antibodies which react with one or more of the "HIV" proteins, one would expect that gay men, haemophiliacs, IV drug users, blood transfusion recipients who are repeatedly exposed to foreign blood and blood products will have a positive "HIV" antibody test even if not infected.

Given that:

1. Individuals with fungal and mycobacterial infections have antibodies which may react with "HIV proteins" in the absence of "HIV" and that *E. coli* is an intestinal commensal and a potential bacterium in all of us, how can one assert that:

    (a) reactions between antibodies in the sera of AIDS patients and proteins present in cultures derived from the tissues of AIDS patients is proof that the reacting proteins are constituents of a unique retrovirus HIV and the antibodies are specific to these proteins?

(b) PCP, candidiasis, cryptococcosis, coccidioidomycosis, histoplamosis, tuberculosis or *Mycobacterium avium-intracellular* disease, that is, the vast majority of the opportunistic infections (88% of AIDS cases diagnosed between 1988 and 1992 had one or more fungal or mycobacterial infections) which signify AIDS are caused by HIV on the basis of a positive antibody test?

(c) a positive antibody test in individuals with fungal and mycobacterial infections proves HIV infection?

2. Since:

(a) mice and patients with autoimmune diseases (SjS and SLE) and AIDS patients share many clinical and immunological (autoantibodies) manifestations;

(b) patients polytransfused with allogenic blood and mice injected with foreign cells and foreign proteins develop “HIV” antibodies but are not infected with HIV;

why should gay men, IV drug users and haemophiliacs, who are all exposed to foreign cells and/or foreign proteins, may not also develop “HIV” antibodies and even if not infected with HIV? The inevitable interpretation of the above data is that HIV antibodies have not been proven to exist and thus all seropositivity is the result of cross-reactivities or non-specific immune stimulation or both.

Indeed, given the pivotal importance of specific “HIV” antibodies not only in defining HIV infection but also in proving the existence of HIV proteins and HIV, it would seem crucial to test this interpretation in the same manner as twenty years ago when researchers questioned antibodies to the now defunct “first” human retrovirus HL23V. These two groups analysed the specificity of HL23V antibodies “in absorption tests with normal human serum proteins, assays of viral gp70 antigenicity after exposure to exo- and endoglycosidases or trypsin, and carbohydrate hapten inhibition studies”. These data proved that antibodies to the HL23V glycoprotein were elicited non-specifically by “exposure to many natural substances possessing widely crossreacting antigens” and not by “widespread infection of man with replication-competent oncoviruses” (retroviruses).[169,170] We predict that in the AIDS era a significant number if not all of the “HIV positive” sera will have reactivity to the “HIV” proteins reduced or eliminated by absorbtion with fungal, mycobacterial and auto antigens.

RECENT DEVELOPMENTS

By 1997 some of the best known HIV experts[180,181] accepted that:

1. "Purified" HIV contains cellular proteins, "some are over-represented in comparison to the relative amount in the cell membrane, whereas others appear to be absent", and that these proteins "serve as protective immunogens in vaccination experiments".

2. HIV “used for biochemical and serological analyses or as immunogens is frequently prepared by centrifugation through sucrose gradients", but in none of the studies "the purity of the virus preparation has been verified". In other words, up till 1997 nobody has published electron micrographs of the 1.16 gm/ml band to prove that the "purified virus" contained nothing else but virus particles.

In that year, in *Virology*, two studies were published, one by a USA team, principal author Julian Bess, and the other by a Franco-German group, principal author Pablo Gluschankof, with the first electron micrographs of "purified HIV". While in the Gluschankof *et al* studies the EMs were from the 1.16 gm/ml band, in the Bess *et al* band they were from pooled bands. The authors of both studies claimed that their "purified" material contained some particles with the appearances of retroviruses and in fact were HIV particles. But they admitted that their material predominantly contained particles which were not retroviruses but "budding membrane particles frequently called microvesicles" or “mock virus”. Indeed the caption to the Gluschankof *et al* electron micrograph reads, "Purified vesicles from infected H9 cells (a) and activated PBMC (b) supernatants", not purified HIV. In further experiments the supernatant from non-infected cultures was also banded in sucrose gradients. Both groups claimed that the banded material from these cultures contained only microvesicles, "mock virus" particles, but no HIV. Both the "HIV" particles and the mock-virus particles possessed membranes. In the USA study the "HIV-1 particles" were differentiated from the microvesicles "by the electron dense cores", whereas in the other study the "HIV" particles were "identified by the relatively homogenous diameter of about 110 nm, the dense cone-shaped core, and the "lateral bodies". However, in the arrowed particles which are said to be HIV it is difficult if not impossible to locate any which have cone-shaped cores or bilateral, "lateral bodies". In fact no particle in any study has the principle morphological characteristic of

retrovirus, a diameter of 100-120 nM and surface spikes, knobs. In the Franco-German study the average "HIV" particle diameter is 136 nM and no particle had a diameter less than 120 nM. In the USA study the corresponding dimensions are 236 nM and 160 nM. In other words, the American “HIV” is twice the diameter of the European “HIV”, and all other “HIV” particles. The US authors did not note or explain this discrepancy and “were much more focused on showing the mixture of particles in the preparations as opposed to their actual diameters”. The diameter of the microvesicles "range in size from about 50 to 500 nm".[180] Both the "HIV" and the "mock" virus particles contained RNA. The RNA of the latter had contained mRNA which is known to be rich in adenine and which, according to Gallo is specific to retroviral RNA.[182,183] According to Gluschankof *et al*, "The vesicles contain large amounts of protein and nucleic acids which are unstructured and thus are transparent by electron microscopy", that is why many, but not all, appear "empty" by electron microscopy, while the nucleic acids in the "HIV" particles are structured and for this reason they appear to have an "electron dense cores". However, according to a leading retrovirologist, John Bader, the core density can be changed by external conditions, that is, the culture conditions.[57] It is well known that a structural virus particle or virus-like particle can become "unstructured" in the presence of reducing agents. The possibility cannot be excluded then, that the apparent morphological differences between the two types of particles may be due to nothing more than the difference in the redox of the microenvironment in which they are assembled, released or both. It is significant there is evidence that actin polymerisation (or actin/myosin interaction) "mediates HIV budding" and release.[123,124,126,127] Evidence also exists that:

1. As shown by Bess *et al*, uninfected cells exhibit buds which are no different from those in infected cells.

2. There is an association between the redistribution of polymerised actin, myosin and other cellular proteins (glycoproteins) and many cellular processes including budding unrelated to release of retrovirus.[184-188]

3. Polymerisation of actin, actin-myosin interaction and cross-linking of polymers in general is regulated by the redox state, oxidation leading to interaction.[189-191]

4. Both AIDS patients and cultures derived from AIDS patients are subjected to oxidising agents. In fact, for the detection of "HIV" proteins and particles the cell cultures must be stimulated (treated with oxidising agents).

5. In the presence of antioxidants no "HIV" phenomena can be observed.[2,3,7,192]

The minimum absolutely necessary but not sufficient condition to claim that what are called "HIV-1 particles" are a retrovirus and not cellular microvesicles is to show that the sucrose density fractions obtained from the "infected" cells contain proteins which are not present in the same fractions obtained from non-infected cells. However, Bess *et al* have shown this is not the case. The only difference one can see in their SDS-polyacrylamide gel electrophoresis strips of "purified virus" and "mock virus" is quantitative, not qualitative. This quantitative difference may be due to many reasons including the fact that there were significant differences in the history and the mode of preparation of the non-infected and "infected" H9 cell cultures, in addition to the “infection".

A similar finding was reported by the same authors a few years earlier. However, while in both studies the proteins of molecular weight "near 42 kDa" (42,000) are labelled as "Actin" and "in the 30- to 40-kDa range" as "HLA DR", all the proteins with molecular weight higher than approximately 42,000 and lower than approximately 30,000 are left unlabelled in the earlier paper. In the 1997 study, three proteins of molecular weight lower than 30,000 are labelled as $p24^{CA}$, $p17^{MA}$, and $p6/p7^{NC}$ and are said to represent "major bands of viral proteins”. However, also according to the authors, “these labels were added when one of the reviewers asked for them. He felt it would help orient readers when looking at the figure - the reviewer is correct. We did not determine the identities of the bands in this particular gel”.

In their earlier study the researchers from the USA presented further evidence that the "viral proteins" were nothing more than cellular proteins. In their efforts to make an HIV vaccine they immunised macaques with, amongst other antigens, "mock virus", that is, sucrose density banded material from the supernatants of non-infected H9 cell cultures. After the initial immunisation the monkeys were given boosters at 4, 8 and 12 weeks. The animals were then challenged with "SIV" propagated either in H9 cells or macaque cells. When the WBs obtained after immunisation but before "SIV" challenge were compared with the WBs post-challenge, it was found that challenge with "SIV" propagated in macaque cells had some additional bands. However, the WBs obtained after the challenge with SIV propagated in H9 cells were identical with the WBs obtained after immunisation but before challenge. In other words, the protein immunogens in the "virus" were identical with the immunogens in the "mock virus". Since both the "mock virus" and "purified" virus contain the same

proteins, then all the particles seen in the banded materials including what the authors of the 1997 *Virology* papers call "HIV" particles must be cellular vesicles. Since there is no proof that the banded, "purified virus", material contains retrovirus proteins and thus retrovirus particles then there can be no proof that any of the banded RNA is retroviral RNA. When such RNA (or its cDNA) is used as probes and primers for hybridisation and PCR studies, no matter what results are obtained, they cannot be considered proof for infection with a retrovirus, any retrovirus.

In the interview which Montagnier gave to Djamel Tahi he was asked why they did not publish an electron micrograph of the 1.16 gm/ml band to prove that the band represented "purified" virus, as they claimed. He replied that the reason for this was that even after "Roman effort" in their "purified" virus they could not see any particles with the "morphology typical of retroviruses. They were very different. Relatively different". When Montagnier was asked if Gallo isolated HIV he replied: "I do not believe so".[29] If there were no retrovirus-like particles in Montagnier's "purified" virus, then obviously Montagnier and his colleagues could not claim to have isolated a specific exogenous retrovirus, HIV. If HIV does not exist then the cause or causes of AIDS must be urgently reappraised and alternative hypotheses heeded.

THE "HIV GENOME"

To claim that the stretch of RNA (cDNA) is the genome of a unique retroviral particle, HIV, the most basic requirement is proof for the existence of a unique molecular entity "HIV RNA" ("HIV DNA") that is, a unique fragment of RNA (DNA) identical in both composition and length in all infected individuals. The claim that a stretch of RNA (cDNA) is a unique molecular entity which constitutes the genome of a unique retrovirus can be accepted if and only if it is shown that the RNA belongs to particles with the morphological, physical and replicative characteristic of retroviral particles. Proof of this can only be obtained isolating the particles, that is, by obtaining them separate from everything else (purifying them). In 1984 both Gallo's and Montagnier's groups reported finding polyadenylated (adenine rich) RNA (poly(A)-RNA) in the 1.16 gm/ml band material obtained from "infected" cultures. The RNA was claimed to be HIV RNA that is, the HIV genome, and its complementary DNA, the HIV provirus. However,

(a) as mentioned, although Montagnier claimed his 1.16 gm/ml band contained particles, neither his band nor that of Gallo's contained any particles with "morphology typical of retroviruses";

(b) poly(A)-RNA is not specific to retroviruses. It can be found in all cells and even at the 1.16 gm/ml band obtained from "non-infected" cells;[180]

(c) there is no proof for the existence of a unique molecular entity, "HIV-RNA" or "HIV-DNA", while the genomes of the most variable RNA viruses do not differ by more than 1%.[193] The difference between the human and the chimpanzee genomes is no more than 2% while there is up to 40% variation between "HIV" genomes;[194]

(d) in hybridization studies using the "HIV RNA" or cDNA, Gallo and since then many other researchers have been unable to prove the existence of the HIV genome in fresh lymphocytes from AIDS patients.[195] In 1994 Gallo stated "We have never found HIV DNA in the tumour cells of KS…In fact we have never found HIV DNA in T-cells".[196,197]

(e) All the claims of the existence of HIV in humans are based on polymerase chain reaction (PCR) studies using small fragments of the "HIV" genome as primers. However even researchers who believe that there is proof that the HIV primers and probes used in these studies are HIV accept that the specificity of this assay, using the antibody as a gold standard, varies between zero and 100 per cent.[198] Even with the PCR nobody has reported the existence of the full "HIV" genome in the fresh lymphocytes of even a single AIDS patient. Nonetheless it is generally believed:

(i) there is proof for the existence of a unique molecular entity RNA (cDNA) which is the genome of a unique retrovirus, HIV;
(ii) HIV RNA (HIV DNA) can be found only in infected individuals.

Furthermore, despite the ample evidence to the contrary, for most retrovirologists the finding of a novel nucleic acid in a cell can be due to nothing else but an infectious agent. Half a century has passed since the Nobel Laureate Barbara McClintock discovered the phenomenon of transposition which can lead to the appearance of new genotypes and phenotypes. According to McClintock, the genome can be restructured not only by transposition but also by other means as well. In her Nobel lecture of 1983, she said, "rapid reorganisation of genomes may underline some species formation. Our present knowledge would suggest that these reorganizations originate from some "shock" that forced the genome to restructure itself in order to overcome a

threat to its survival...Major genomic restructuring most certainly accompanied formation of new species". The "genomic shock" which leads to the origin of new species may be "either produced by accidents occurring within the cell itself, *or imposed from without such as virus infections, species crosses, poisons of various sorts, or even altered surroundings such as those imposed by tissue culture*. We are aware of some of the mishaps affecting DNA and also of their repair mechanisms, but many others could be difficult to recognize. Homeostatic adjustments to various accidents would be required if these accidents occur frequently. Many such mishaps and their adjustments would not be detected unless some event or observation directed attention to them...Unquestionably, we will emerge from this revolutionary period with modified views of components of cells and how they operate, but only however, to await the emergence of the next revolutionary phase that again will bring startling changes in concepts".[199] (It is worthy adding that although McClintock's ideas concerning the generation of novel nucleic acids and new species represent a milestone in their own right, one cannot fail to commend the prescience of Peyton Rous writing seventy three years earlier).

As we have mentioned elsewhere an exogenous retrovirus is only one possible explanation for the finding of novel nucleic acids in AIDS patients. Other explanations are:

1. The genome of an endogenous retrovirus, that is, a stretch of RNA with a corresponding proviral DNA present in the cellular DNA of uninfected animals and which is passed from generation to generation vertically (from parents to offspring via the germ cell line) and which under certain conditions can be expressed and incorporated into retroviral particles.

2. The genome of a retrovirus *de novo* assembled by genetic recombination and deletion of: (a) endogenous retroviral sequences or (b) retroviral and cellular sequences or (c) non-retroviral cellular genes.

3. An RNA obtained by transposition, that is, by certain replicating DNA sequences (transposons) becoming inserted elsewhere in the genome, or by retroposition, that is, by particular RNA (retrotransposons) first being transcribed into DNA and then similarly being inserted into the genome. Retroposition can "use cellular mechanisms for passive retroposition, as well as retroelements containing reverse transcriptase". The retroelements may be retrovirus-like elements or nonviral elements.[200,201] Not only can retroposition "shape and reshape the eukaryocytic genome in many different ways"[200] but the nonviral retroelements may be similar to the retroviral elements.

A basic principle of molecular biology is that the primary sequence of RNA faithfully reflects the primary sequence of the DNA from which it is transcribed. However, in the 1980s RNA editing, "broadly defined as a process that changes the nucleotide sequences of an RNA molecule from that of the DNA template encoding it", was discovered. In the process a non-functional transcript can be re-tailored, producing a translatable mRNA, or modify an already functioning mRNA so that it generates a protein of altered amino acid sequences. Sometimes editing is so extensive that the majority of sequences in a mRNA are not genomically encoded but are generated post-transcriptionally producing the "paradoxical situation of a transcript that lacks sufficient complementarity to hybridize to its own gene!".[202-204] According to Nancy Maizels and Alan Weiner from the Department of Molecular Biophysics and Biochemistry at Yale University, "the central dogma has survived hard times. The discovery of reverse transcriptase amended but did not violate the central dogma of how genes make proteins; introns qualified the conclusion that genes are necessarily collinear with the proteins they encode; somatic rearrangement of lymphocyte DNA called stability of eukaryotic genomes into doubt...and catalytic RNA challenged the pre-eminence of proteins and breathed new life into the ancient RNA world". However, the discovery of RNA editing "could come close to dealing it a mortal blow".[205]

Thus the finding of novel RNAs in human cells, especially those of AIDS patients and those at risk, can no longer be regarded as incontrovertible proof that the RNA has been exogenously introduced by a putative HIV or any other infectious agents.[9,19] That this may be the case has of late been accepted by Luc Montagnier. In a written testimony dated February 2nd 2000, to the US House of Representatives Committee on Government Reform, Subcommittee on National Security, Veterans Affairs and International Relations, in support of the work of his colleague, Howard B Urnovitz, (Montagnier is on the scientific advisory board of a publically traded biomedical company whose director is Urnovitz), Montagnier wrote: "I have reviewed Dr Urnovitz's published research and the testimony prepared for presentation to this Committee and strongly advise that future research on Gulf War Syndrome should include the study of the detected genetic material".[206]

Urnovitz and his colleagues presented evidence of the existence, in Persian Gulf War veterans, of "novel", "nonviral" RNAs, "possibly induced by exposure to environmental genotoxins". They concluded: "The patterns of the occurrence of RPAs [polyribonucleotides] in the sera of GWVs [Gulf War Veterans] and healthy controls are sufficiently distinct to suggest possible future diagnostic applications…Our studies of patients with active

multiple myeloma suggest that patients with individual chronic multifactorial diseases may have unique RPAs in their sera. Validated tests for such putative surrogate markers may aid in the diagnosis of such diseases or in the evaluation of responses to therapeutic modalities".[207]

It is also highly significant that in his "STATEMENT FOR THE DURBAN AIDS CONFERENCE",[208] which begins "What is 'HIV'?", Urnovitz offers no explanation for his parenthetic use of the terms "'HIV'", "'HIV" genome" and "'HIV' biomarker". In the same document it is also implied that the HIV genome may result from the "reshuffle" of cellular retroelements. That is, Urnovitz agrees with one of several possible explanations summarised above and earlier put forward by our group to account for the presence of novel RNAs in the cells of AIDS patients but which may not be present in the cells of healthy individuals.[9,19] Urnovitz also agrees with us that "Missing from the landmark 1983 analysis of "HIV" was an understanding of the role "poikilogenic" agents play in the laboratory protocol that is used to study human retroviruses. The term "poikilogenic" is derived from the Greek "poikilo" which means *diversity* and "gen" which stands for *generate.* Poikilogenic agents are those entities–chemical, physical, or biological–that create genetic diversity via genetic recombinatorial events. These events may include the inductive expression of retroelements and the resulting byproducts of newly reshuffled genetic material. One such poikilogenic agent was reported in the 1983 discovery of "HIV". The agent is phytohemagglutinin (PHA). "Using an HERV-H LTR probe, 6 and 4.5 kb transcripts were detected by Northern blot analysis which were induced in normal peripheral T cells after treatment with phytohaemagglutin"[209] (HERV≡known endogenous retrovirus). PHA has been and continues to be used as a laboratory agent not only by Montagnier but by virtually every retrovirologist who claims proof for HIV isolation.

Since Montagnier agrees with Urnovitz that novel, nonviral RNAs appear in the Gulf War Veterans, then why should the existence of novel RNAs:

1. In AIDS patients and those at risk be the genome of a retrovirus HIV and not the result of the many toxins including genotoxins to which they are exposed?[2,7,19,191,210]

2. In cultures containing tissues from AIDS patients be interpreted as HIV RNAs rather than the result of the many toxins including genotoxins[2,7,8] to which both the patients and the cultures are exposed? Especially when both Montagnier and Gallo accept that HIV cannot be detected in cultures which are not treated with such toxins (oxidant agents) including PHA?[89,207,208,211,212] When hard pressed all the HIV experts will ultimately accept the non-specificity of retroviral-like particles, reverse transcription and antibody/antigen reactions.

CONCLUSION

In 1983 Luc Montagnier and his colleagues and in 1984 Robert Gallo and his colleagues claimed to have proven the existence of HIV by purifying retroviral particles, that is, by obtaining a mass of particles isolated from everything else and showing that the particles are infectious. A critical analysis of their evidence shows that neither group presented proof of isolation of a novel retrovirus from AIDS patients. The phenomena they interpreted as HIV are all non-specific and were known to be so long before the AIDS era. In fact, given the origin of the cells and the culture conditions, one would expect to find all these phenomena even if the cultures are not infected with a retrovirus. In 1997 Montagnier himself stressed that to prove the existence of a unique retrovirus purification is absolutely necessary and admitted that he had not presented such proof, and in his view, neither had Gallo. Recognition of these facts may prove the first step in solving the problem of AIDS.

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