Between the lines

a critical analysis of Luc Montagnier's interview answers to Djamel Tahi

by Eleni Eleopulos and colleagues

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1. If "culture, purification of the material by Ultracentrifugation, Electron Microscopic (EM) photographs of the material which bands at the retrovirus density, characterisation of these particles, proof of the infectivity of the particles" is not isolation, then why did Montagnier and his colleagues claim in 1983 to have isolated "HIV" by either performing or claiming to have performed all but one (no EM photographs of the banded material) of these procedures? Why in the 1984 paper where they claimed the first isolation of "HIV" from haemophiliacs, as well as in their other studies that year in which they also claim "HIV" isolation, have they either performed or claimed to have performed all but one of these steps?[20-21] Why in their study entitled "Characterisation of the RNA dependent DNA Polymerase of a new human T lymphotropic retrovirus (lymphadenopathy associated virus)"[22] did they state that the virus was "purified on sucrose gradient using isopycnic centrifugation (8)"? Reference 8 is the paper presented by Sinoussi and Chermann at the 1972 Pasteur Symposium where they stressed the importance of showing that the banded material contained nothing else but particles with "no apparent differences in physical appearance".[14]

2. The finding of some or all of the phenomena Montagnier outlines are not proof of isolation. These phenomena can be considered only proof for viral detection and then, if and only if, they are specific to retroviruses. The word "isolation" is derived from Latin "insulatus" meaning "made into an island". It refers to the act of separating an object from all the extraneous matter that is not that object. Here the object of interest is a retroviral particle. The words 'isolation' and 'passing' have different and distinct meanings. 'Isolation' means to obtain an object, a retrovirus particle for example, separate from everything else. 'Passing' means to transfer an object (which may or may not be isolated) from one place to another, for example, from one culture to another. Therefore, even if one assumes that the "something" which Montagnier and his colleagues passed from one culture to another by means of transferring cells or culture supernatants was a retrovirus, and that it was passed to an infinite number of successive cultures, it still is not evidence for isolation. For example, if one has a series of bottles containing water in which the first has a dye added, then takes part of the first and puts it in the second, and from the second passes a sample into the third *et cetera*, clearly this procedure has not isolated the dye from the water. A culture contains a myriad of things and thus by definition is not evidence for isolation of an object. The only way possible to claim that one has "made a culture of the virus", is to have had proof for the existence of the virus before making a culture. The only thing which Montagnier and his colleagues have proven is the emergence in the co-culture with "lymphocytes from a blood donor" of RT activity. Detection of an enzyme in a culture, even if specific to retroviruses is not evidence for isolation. For example, the measurement of cardiac or liver enzymes in cases of myocardial infarction or hepatitis respectively cannot be construed as "isolation" of the heart or liver. The finding in the culture of particles with the morphological characteristics of a retrovirus and of reverse transcriptase activity either in the culture or the 1.16g/ml band, even if "truly specific of retroviruses" is not evidence for retroviral isolation. Even if Montagnier and his colleagues knew beforehand that some of the proteins present in the culture or the 1.16g/ml band were retroviral, and the patients had retroviral antibodies which reacted with these proteins, such a reaction is not evidence for isolation. Argument based on analogies, or even on knowledge of other retroviruses, cannot be construed evidence for isolation. For example, observing something in the ocean which looks like a fish (even if it is a fish), is not equivalent to having the fish in your frypan separate from everything else that occurs in the ocean.

3. We agree with Gallo that Montagnier et al did not present proof for "true isolation" of a retrovirus, any retrovirus, either old or new, exogenous or endogenous.

4. The "knowledge of other retroviruses" shows that not all particles with RT activity and "visual properties of retrovirus" are viruses. This is a fact acknowledged even by Gallo well before the AIDS era.[23] It also shows that RT is not "truly specific of retroviruses". Non-infected cells as well as bacteria or viruses other than retroviruses have RT. According to some of the best known retrovirologists including its discoverers, as well as Nobel Laureate and Director of the US National Institutes of Health, Harold Varmus, reverse transcriptases are present in all cells including bacteria.[13,24-25] Indeed RT activity has been reported in many of the cell lines from which "HIV" is "isolated", including H9 and CEM as well as normal lymphocytes even when they are not infected with "HIV".[26-27] Montagnier, Barre-Sinoussi and Chermann themselves have shown that RT activity is not specific to retroviruses. In their 1972 paper Barré-Sinoussi and Chermann wrote: "There was significant activity in the sample zone and the fastest sedimenting peak, consisting mainly of cell debris. This enzymatic activity can be explained by the presence of some virus particles in these regions, and, since similar polymerase activity has been found in normal cells, may be mainly ascribed to the cellular enzyme". In this interview, Luc Montagnier answering question 14 says: "For example, one day I had a very fine peak of RT, which F Barre-Sinoussi gave me, with a density a little bit higher, 1.19 and I checked! It was a mycoplasma, not a retrovirus". How is it then possible for Montagnier to say that RT is specific to retroviruses? We agree that RT activity is characteristic of retrovirus. However, 'specificity' does not have the same meaning as 'characteristic'. Hair is characteristic of human beings but not every animal with hair is human.

5. Isolation means to obtain an object separate from everything else. Retroviruses are particles and no amount of "analogy" can

prove that one has isolated a retroviral particle. "Knowledge of other retroviruses" can be of assistance in choosing the best method to obtain isolation. The "knowledge of other retroviruses" shows that the best, but by no means perfect method to isolate and prove the existence of retrovirus, is to perform isopycnic (identical density of particle and portion of the gradient) banding and to perform all the assays specified at the 1972 Pasteur symposium. The "knowledge of other retrovirus" also shows that there is nothing specific about the morphology of retroviral particles, protein-antibody reactions or even banding at the density of 1.16g/ml in sucrose density gradients. Retroviral particles band at the density of 1.16g/ml but not everything at that density, including particles with the morphology of retroviral particles, is a retrovirus.[11-13,28] To remind ourselves this is the case, one needs go no further than to consider the "first" human retrovirus, "HL23V".

In the mid-1970's Gallo and his colleagues reported the isolation of the first human retrovirus. In fact the evidence for the isolation of "HL23V" surpassed Montagnier's et al and everybody else's evidence for "HIV" in at least three important aspects [see p. 29]. Unlike "HIV", in the case of "HL23V" Gallo's group

(a) reported the detection of RT activity in fresh, uncultured leucocytes;

(b) did not need to stimulate their cell cultures with various agents. (Both Montagnier and Gallo concede that none of the phenomena which they say prove the existence of "HIV" can be detected unless the cultures are stimulated with several agents);

(c) published an electron micrograph of virus-like particles banding at a sucrose density of 1.16g/ml.[23-29] However, today nobody, not even Gallo, considers "HL23V" as being the first human retrovirus or even a retrovirus. (For a more detailed discussion see Papadopulos-Eleopulos et al [30-32]).

One also must not forget the following additional knowledge in relation to retroviruses:

(a) the lesson of the enzyme adenosine triphosphatase. Like RT, this enzyme was considered to be specific to retroviruses and at least in the 1950s was used not only for their detection and characterisation but also for their quantification.[8-11] Yet at present it is accepted that this is one of the most widely spread enzymes.

(b) a much higher percentage of sera from AIDS patients and those at risk reacts with proteins of endogenous retroviruses than the sera of healthy people, 70% versus 3%.[33]

2

1. It is true that Montagnier and his colleagues found a peak of RT activity at the density of 1.16g/ml. However, finding this peak is not proof that the band was made up of retrovirus particles either pure or impure. Therefore this evidence cannot be considered that "one has fulfiled this criterion for purification".

2. In the same issue of *Science* where Montagnier and his colleagues published their study Gallo pointed out that "the viral envelope which is required for infectivity is very fragile, it tends to come off when the virus buds from infected cells, thus rendering the particles incapable of infecting new cells". Because of this Gallo claimed that "cell-to-cell contact may be required for retroviral infection".[34] At present all "HIV" experts agree that for "HIV" infectivity gp120 is absolutely necessary.

In 1993 Montagnier himself said that for the "HIV" particles to be infectious they must first bind to the cellular CD4 receptor and that "The gp120 is responsible for binding the CD4 receptor".[35-36] However, to date nobody has published EM of cell-free particles having the dimension of retroviral particles and also knobs, spikes, that is gp120, not even Hans Gelderblom and his colleagues from the Koch Institute in Berlin who have conducted the most detailed electron microscopy studies of the particles present in culture/co-cultures containing tissues derived from AIDS patients. In one of their latest publications where this matter is discussed they estimate that immediately after being released, "HIV particles" possess an average of 0.5 knobs per particle but also pointed out that "it was possible that structures resembling knobs might be observed even when there was no gp120 present, i.e., false positives".[37] This means that neither Montagnier and his colleagues nor anybody else subsequently could infect the cultures with cells from healthy donors, umbilical cord lymphocytes or any other cultures with the "purified HIV" or, even the cell-free fluids (the culture supernatant) even if the "purified" virus contained nothing else but particles. In other words, it is impossible for Montagnier and his colleagues to have had any infectivity even "a little" with either the culture supernatant or the "purified labelled virus". For the same reason the "second strain" could not be contaminated by "the first". Furthermore, since Montagnier et al provided Gallo with cell-free supernatants, it would have been impossible for the Gallo cultures to be contaminated with BRU, LAI or a mixture.

3. Montagnier's "virus" did not come "from an asymptomatic patient" but a patient with "lymphadenopathy and asthenia". Neither in their study nor even today, after nearly fifteen years of "HIV", is there proof for the existence of a human retrovirus which has the ability to "kill cells". The study which at present is most often quoted as proving "HIV" kills T4 cells, considered to be the "hallmark" of AIDS, was published in 1984 by Montagnier and his colleagues. They cultured CD4+ (T4) cells from a haemophilic patient who was "an asymptomatic virus carrier", "in the presence of phytohemagglutinin (PHA) followed by IL-2". In the culture they detected RT activity and "virus particles characterised by a small eccentric core". The number of T4 (CD4+) cells in the culture were measured by counting the number of cells able to bind a monoclonal antibody claimed specific for the CD4 protein. The number of cells which were able to do so decreased with time. Discussing their finding they wrote, "This intriguing phenomenon may be due to virus-induced modulation at the cell membrane, or by steric hindrance of the antibody binding site", that is, the decrease is not due to cell killing.[38-39] Given their data, the conclusion that the decrease in T4 cells is not due to cell killing is not surprising. However, their conclusion that the effect may be induced by the "virus", is surprising. Montagnier and his colleagues were aware of the experimental evidence which showed that under certain conditions, (including exposure to PHA, IL-2 and other oxidising agents) decrease in T4 cells appears in the absence of "HIV". In this type of culture, T-cells lose their CD4 marker and acquire other markers, including CD8, while the total number of T-cells remains constant.[40-43] Furthermore, they had evidence that in "infected cells, this phenomenon cannot be detected unless the culture is stimulated by substances such as PHA or antigens. (Proteins such as the "non-HIV" proteins present in the "infected" cultures.[39]) Given the above facts it is even more surprising that Montagnier and his colleagues did not have controls, that is, cultures of T4 cells originating from patients who were not at risk of AIDS but who nonetheless were sick and to which they added PHA and IL-2. Such experiments were reported in 1986 by Gallo and his colleagues. They presented data on three cell cultures which contained 34% CD4 cells to begin with: One culture was "infected" and stimulated with PHA, the other was not infected but was stimulated with PHA and the third was neither infected nor stimulated. After two days of culture, the proportion of CD4+ cells in the stimulated-uninfected and stimulated-infected culture was 30% and 28% respectively, while at 6 days the number was 10% and 3%. The number of CD4+ cells did not change significantly in the non-infected non-stimulated culture.[44] By 1991 Montagnier and his colleagues had performed experiments with uninfected, unstimulated cells when they studied "HIV" induced apoptosis, which was said (and is still said by many), to be the principle mechanism by which "HIV" kills cells. They showed that in acutely "HIV infected" CEM cell cultures in the presence of mycoplasma removal agent, cell death (apoptosis) is maximum at 6-7 days post infection, "whereas maximal virus production occurred at Days 10-17", that is, maximum effect preceded the maximum cause. In chronically "infected" CEM cells and the monocytic cell line U937, no apoptosis was detected although "these cells produced continuously infectious virus". In CD4 lymphocytes isolated from a normal donor, stimulated with PHA and "infected with HIV" in the presence of IL-2, apoptosis becomes detectable 3 days post infection and clearly apparent at 4 days. "Intriguingly, on the 5th day" apoptosis became detectable in "uninfected", PHA stimulated cells. They concluded: "These

results demonstrate that HIV infection of peripheral blood mononuclear cells leads to apoptosis, a mechanism which might occur also in the absence of infection due to mitogen treatment of these cells".[45]
In conclusion, all the presently available data shows that "HIV infection" in the absence of stimulating agents neither decrease the T4 cell number, nor induce apoptosis, while stimulating agents (similar to those to which patients at risk of developing AIDS are exposed) do so in the absence of "HIV". That is, neither the "HIV", which Montagnier and his colleagues "stumbled" at the beginning, nor any other "HIV" since then has been shown to "kill cells".

3

Retroviruses are not esoteric, nuclear or cosmological notions whose postulated existence can only be inferred by indirect observations. They are particles which can be seen, albeit not with the naked eye. Since Montagnier and his colleagues admit to not seeing particles at the 1.16g/ml band having the morphology of retrovirus, to claim the presence of a retrovirus much less a "purified virus" is totally unsubstantiated and defies belief. The 1.16g/ml band can be likened to a fishing net. The difference is that the band traps objects according to their density, not their size. Imagine a fisherman who sees in the ocean many different objects some of which may be fish. He throws the net, waits, and upon retrieval of the net performs a thorough examination of its contents and shows that it contains many sea creatures but nothing that looks like a fish. Yet strange as it may seem, he claims to have caught fish. In fact, he claims that the net has nothing else but pure fish.

4

Although budding from the cell membrane is the manner in which retroviral particles appear, this process is not virus specific. In other words, just because a particle buds and has the morphological characteristics of retroviral particles does not prove it is a retrovirus. That this is the case can be illustrated by two facts and by quoting two of the best known retrovirologists: "Budding virus-like particles" have been found in non-infected "T-cell lines CEM, H9 and C8166; In 2 lines of EBV transformed B-cell lines; and in cultures of primary human lymphoid cells from cord blood, which were either PHA stimulated or not and grown with or without serum and in cord lymphocytes directly after Ficol separation"[46] (italics ours). Following an extensive, in vivo study conducted by O'Hara and colleagues from Harvard, "HIV particles" were found in 18/20 (90%) of patients with enlarged lymph nodes attributed to AIDS. However, identical particles were also found in 13/15 (87%) of patients with enlarged lymph nodes not attributed to AIDS and at no risk for developing AIDS. These data led the authors to conclude, "The presence of such particles does not, by themselves indicate infection with HIV".[47]
In 1986 Gallo and his colleagues discussing the "First isolation of HTLV-III" wrote: "At the time we obtained LAV it was the contention of several experts in virus morphology that the particles shown in the electron micrograph published in Science by Barre-Sinoussi et al was an arena virus...Since we considered the mere detection of virus particles in cultures from AIDS and ARC patients to be insufficient to confirm scientifically our hypothesis that such particles were implicated in the aetiology of the disease, we decided first to obtain specific reagents against the new virus in order to publish definite results concerning AIDS aetiology".[48]
According to Peter Duesberg the "HIV" "particles and proteins could reflect non-viral material altogether".[49]

5

In their study Montagnier and his colleagues wrote: "Electron microscopy of the infected umbilical cord lymphocytes showed characteristic immature particles with dense crescent (C-type) budding at the plasma membrane...This virus is a typical type-C RNA tumor virus". In 1984 Montagnier, Barre-Sinoussi and Chermann reported that their virus was "morphologically similar to D particles such as those found in Mason-Pfizer virus or the virus recently isolated from simian AIDS".[38] (By 1984 researchers from the primate research centres in the United States claimed the existence of AIDS in monkeys and that the cause of AIDS was a type-D retrovirus similar to the Mason-Pfizer virus, a typical type-D retrovirus and suggested that the monkey AIDS and these retroviruses could be helpful in the study of human AIDS and "HIV"). In the same year, in yet another publication, Montagnier et al claimed that the "HIV" particles had "morphology similar to that of equine infectious anaemia virus (EIAV), and D type particles". The EIAV and the visna virus are neither type C nor type D retroviruses but lentiviruses, that is, viruses which have totally different morphology and said to induce diseases long after infection. (By the time this paper was published it was realised that patients who had a positive "HIV" antibody test did not develop AIDS immediately, that is, there was a delay between the positive test and the appearance of AIDS.) It is most astonishing that the morphology of one and the same virus is able to change genus from a typical type-C to a typical type-D particle and then to a completely different subfamily, namely a typical lentivirus, apparently at will. (The family Retroviridae is divided in three subfamilies, Oncovirinae, Lentivirinae and Spumavirinae. Oncovirinae are in turn divided into genus type-B,-C and -D particles. These findings are analogous to describing a new species of mammal as human, a gorilla and an orang-utan).

6

1. Apart from retroviruses other particles may possess "the assemblage of properties" (the density, RT, budding and the analogy with the visna virus). It follows that the detection of particles having this "assemblage of properties" is not proof that the detected particles are retroviruses. In fact, Montagnier and his colleagues did not report the detection of "HIV" particles having this "assembly of properties". Since Montagnier and his colleagues could not find particles with the morphological characteristics of retrovirus at the "density" of 1.16 gm/ml, even after "a Roman effort", it follows that the evidence for the existence of "HIV" from the density gradient was not only non-specific but was non-existent. (This fact alone is sufficient to dismiss any claim of proof for the existence of a retrovirus, no matter what else they found anywhere including budding particles from the cell surface, retrovirus-like particles in the culture, RT at the "density" or proteins at the same density which react with patient sera).
2. It is true that Montagnier et al reported RT activity at the density of 1.16g/ml but since:
(a) Barre-Sinoussi and Chermann accept that cells and cellular fragments also have RT activity;
(b) at the 1.16g/ml band no particles with the morphological characteristics of retrovirus were seen;
(c) at that density Montagnier et al found cellular fragments, it follows that the evidence for the existence of "HIV" by detecting RT activity at that density was not only not specific but non-existent.
Given the facts that:
(a) there are significant differences in the nature of the budding processes between type-C, type-D particles and lentiviruses[50] and that in 1983 Montagnier et al reported their retrovirus as type-C and in 1984 as either type-C or type-D, and even later that year as EIAV;
(b) visna virus and EIAV are lentiviruses, it follows that at least up mid 1984 Montagnier's et al evidence for the existence of "HIV" (if "HIV" is a lentivirus) from "pictures of budding" and the analogy with EIAV and visna virus was not only non specific but non-existent.

7

We agree there are endogenous retroviruses [51]. These endogenous retroviruses cannot be distinguished from exogenous retroviruses either morphologically or chemically. Furthermore, evidence exists which shows that 70% of AIDS patients and those at risk compared with 3% of people not at risk have antibodies to endogenous retroviruses.[33]
Given these facts and the culture conditions which Montagnier and his colleagues and all other "HIV" researchers use to detect

"HIV" together with the presently available data on "HIV" and AIDS, it is more probable that "HIV" (if proven to exist) is an endogenous retrovirus rather than an exogenous retrovirus.
Part of the data related to the culture conditions can be summarised as follows: In culture, cells sooner or later start to release endogenous retrovirus. The appearance of endogenous retrovirus can be accelerated and the yield increased up to a million fold by stimulating the culture with mitogens, co-cultivation or by adding to the culture supernatant from normal, unstimulated cell cultures. Indeed, as far back as 1976 retrovirologists recognised that "the failure to isolate endogenous viruses from certain species may reflect the limitation of in vitro co-cultivation techniques".[52] To detect the "assemblage" of the "four characteristics" of "HIV", Montagnier et al (as well as everybody else) employed at least two of the above techniques. In fact, both Montagnier and Gallo admit that not one of the four "characteristics" can be detected unless the cultures are stimulated. Similarly, part of the data related to "HIV" and AIDS can be summarised as follows:

(a) It is true that endogenous retroviruses may have no pathological role in AIDS, but it is also true that to date neither is there such proof for "HIV".[53] According to Montagnier and Gallo the "hallmark" of immunodeficiency in AIDS is the decrease in T4 cells, said to be the result of killing of T4 by "HIV". However Montagnier and his colleagues admitted as far back as 1984 that at least in vitro the observed decrease in T4 cells after "HIV" infection is not due to cell killing but decreased binding of the T4 (CD4) antibody to the cells. Two years later the Gallo team's experiments proved beyond doubt that the decrease in T4 cells (of the CD4 antibody binding) was not due to "HIV" infection but to the PHA which was present in the "HIV" preparation. As mentioned, at the beginning of the AIDS era there was ample evidence that treatment of cell cultures with PHA and other oxidising agents leads to decreased binding of the CD4 antibody and to increase binding of the CD8 antibody, that is, a decrease in T4 cells was accompanied by increase in T8 cells, while the total cell number remained constant. AIDS patients and individuals belonging to the AIDS risk groups are continuously exposed to strong oxidising agents. At present it is accepted that in both AIDS patients and those at risk, the decrease in T4 cells is accompanied by an increase in T8, while the T4 + T8 cell number remains constant.[53] Also, it is of interest to note that as far back as 1985 Montagnier wrote: "This syndrome [AIDS] occurs in a minority of infected persons, who generally have in common a past of antigenic stimulation and of immune depression before LAV infection"[54], that is, Montagnier recognised that in the AIDS risk group, immune deficiency precedes "HIV" infection. In 1984 Montagnier and his colleagues including Barre-Sinoussi and Chermann stated that "Definite evidence will require an animal model in which such viruses [LAV, HTLV-III=HIV] could induce a disease similar to AIDS." Up to today, no such model exists. When pursued by the Nobel Laureate Kary Mullis for even one scientific paper proving the HIV theory of AIDS, Montagnier advised him "Why don't you quote the work on SIV" (Simian immunodeficiency virus);[55]

(b) Unlike endogenous retroviruses which are transmitted vertically, "HIV" is said to be transmitted horizontally especially by sexual intercourse. Indeed at present it is generally accepted that the vast majority of individuals have been infected via heterosexual contact. According to Montagnier and Gallo the first study to have proven beyond doubt that "HIV" is a bidirectionally heterosexually transmitted virus was published in 1985 by Redfield et al. However, in a book published in 1990 entitled AIDS and SEX, its editors, Bruce Voeller, June Machover Reinisch and Michael Gottlieb, discussing this cross-sectional study, as well as other similar studies, wrote: "government researchers published data indicating that United States armed forces personnel infected with HIV-1 had caught the virus from prostitutes, triggering calls for increasing campaigns against prostitution. When infected soldiers were interviewed by nonmilitary researchers whom they trusted, it became clear that nearly all had been infected through intravenous drug use or homosexual contact, acts for which they could be expelled from the armed services, which prevented them from being candid with the original military researchers. In each of these flawed published studies, researchers, journal editors, and peer reviewers failed to correct mistakes that should have been recognised". Nancy Padian from the Department of Epidemiology and Biostatistics, University of California, and her colleagues, who to date have conducted the most thorough studies on heterosexual transmission discussing Redfield's et al study as well as other studies who claimed proof of such transmissions, wrote in 1991: These "studies may not have adequately controlled for other confounding nonsexual routes of transmission such as risks associated with intravenous drug use. At first blush, cases that appear attributed to heterosexual transmission may, after in-depth interviewing, actually be linked to other sources of risk...because partner studies are by definition not random samples, and most reported results are based on retrospective or cross-sectional analyses, some studies may overselect couples in which both partners in a couple are infected because such couples may be more easily identified, thus biasing transmission rates. Furthermore, it is often difficult to establish the source of infection in such couples. When few prospective data are available, enrolling monogamous couples in which the serostatus of the partner is unknown, as was the case for most couples in this study, is one of the only ways to control for this bias".[56] Indeed, there is no proof from the prospective studies, few as they are, that "HIV" is sexually transmitted.[57-58] In her ten year study, unquestionably the longest and the best study of its kind, Padian[59] and her colleagues have spared no effort in an attempt to prove that "HIV" is heterosexually transmitted. There were two parts in her study, one cross-sectional, the other prospective. In the former, of 360 female partners of infected male index cases, "The constant per-contact infectivity for male-to-female transmission was estimated to be 0.0009". The risk factors for seroconversion were:
(i) anal intercourse. (Montagnier himself showed that a positive antibody test reverts to negative and a low T4 cell count to normal by stopping anal intercourse, which means that the positive outcome is not due to a retrovirus;[60]
(ii) having partners who acquired this infection through drug use (Padian herself says that this means that the women may also be IV users);
(iii) the presence in the female of STDs. (antibodies to their causative agents may cross-react with the "HIV" proteins;[31] of 82 negative male partners of positive female index cases only two seroconverted. They estimated that the likelihood of female-to-male transmission was 8 times lower than for male-to-female. Padian herself questioned the validity of these two cases. For the first one she gave several reasons in 1991, when this case was reported for the first time. In the second case they mentioned the fact that "chlamydia was transmitted simultaneously or close to transmission of HIV is striking", that is, the positive "HIV" antibody test appeared at the time when he became infected with chlamydia.
In the prospective study, starting in 1990, "We followed 175 HIV-discordant couples over time, for a total of approximately 282 couple-years of follow-up...The longest duration of follow-up was 12 visits (6 years). We observed no seroconversions after entry into the study...At last follow-up, couples were much more likely to be abstinent or to use condoms constantly ...Nevertheless only 75% reported consistent condom use in the 6 months prior to their final follow-up visit".
Note: Not only seroconversion were reported only in the cross-sectional study but all the cases were diagnosed before 1990. However:
(i) All the "HIV" experts agree that the specificity of the test kits used then was inferior to those used at present;
(ii) The WB criteria used to define "infection" then are not sufficient at present.
Even if one accepts Padian et al data from the cross-sectional study, they have estimated the risk to a non-infected male of acquiring "HIV" infection from his infected female partner per contact is 0.00011 (1/9000). This means that on average, males having sexual intercourse daily with an infected female partner for sixteen years (that is, 6000 contacts at 365 per year), would score a 50% probability of becoming infected. If sexual intercourse takes

place on average weekly then it would take one hundred and fifteen years to reach the same probability. Under such circumstances one must question how "HIV" could become epidemic as the result of bi-directional heterosexual transmission.

8

1. In the Montagnier et al 1983 study, the detection of nothing else but RT activity in the stimulated cultures of lymphocytes originating from a gay man was considered proof that he was infected with a retrovirus. The finding of the same activity in the supernatant of a co-culture of the same cells with lymphocytes from a healthy blood donor was considered proof of passing the retrovirus from the gay man's lymphocytes to the donor's lymphocytes and also for virus isolation. However, passing an activity (RT) is not the same as passing an object (retrovirus).
Furthermore, since non-"HIV" infected lymphocytes as well as many bacteria and viruses other than retrovirus possess RT activity (RT activity has been reported in many "non-HIV" infected cell lines used to isolate HIV such as H9 and CEM and as far back as 1972 in normal, PHA stimulated lymphocytes), finding RT activity in successive lymphocyte cultures each of which contains material which originated from the preceding one, is not proof even for passing RT activity. To illustrate what Montagnier and his colleagues have done, let us return to the analogy of the fisherman and his net: Assume the fisherman casts his net and catches some sea creatures. He leaves a few in the net as bait and then throws it out again. This time, in addition to sea creatures he catches some fish as well. He removes the fish, leaves some sea creatures in the net, throws the net again and this time he catches even more fish. He repeats the procedure several times and every time he catches more fish. Like Montagnier et al who remove the cells and re-use the supernatants, the fisherman removes the fish and re-uses the sea creatures ("the bait"). Clearly the fish caught in the net are not offspring of the "bait". The purpose of the "bait" is to create the right conditions for fish to appear in the net. (Indeed, real fisherman spend a lifetime determining the right conditions). All the fisherman is "passing" is the means for catching the fish, not the fish themselves. Similarly, Montagnier et al appear to be "passing" the conditions to generate RT activity thus generating the illusion of "passing" RT activity.

2. Having a peak of RT activity is not proof for having "replication" of a retrovirus. Keeping track of RT is not the same thing as keeping "track of the virus".

3. Let us assume that one has isolated and proven the existence of a retrovirus in cultures with tissues originating from humans. "The first question put" by Nature is: 'Is it an endogenous retrovirus?' Only when one has evidence that it is neither an exogenous nor an endogenous human retrovirus does the question of "laboratory contamination" with animal retroviruses arise.

4. What the patient had was antibodies which reacted with a protein which in sucrose density gradients banded at 1.16g/ml. Since at that density Montagnier and his colleagues could not find particles with the morphological characteristics of a retrovirus, the evidence that this protein was retroviral was non-existent. In fact they had no evidence that the protein was embodied even in non-retroviral particles, any particles whatsoever present at that density.

5. If Montagnier and his colleagues somehow knew beforehand that the protein which banded at 1.16g/ml and reacted with the gay man's serum was the protein of a retrovirus which was present in his lymphocytes (and not the lymphocytes of the healthy donor or the umbilical cord), and at the same time that the antibodies were directed against "his own virus", why was it necessary to have all these experiments to prove its existence?

9

Even though they had RT activity, at the density of 1.16g/ml they had no evidence for the existence of retroviral particles and thus the activity could not be considered proof for the existence of such particles.

10

In 1983, Montagnier, Barré-Sinoussi and Chermann and their colleagues proved the existence of the enzyme reverse transcriptase "using the ionic conditions described for HTLV-I", that is, "5mM Mg^{2+}" and "poly(A).oligo-(dT)12-18 as template primer". These conditions and this template primer may be characteristics for retroviruses but they are not specific for retroviral RT nor indeed any RT. Even before the AIDS era it was known that this template-primer, under the conditions used by Barré-Sinoussi, Montagnier and their colleagues, can be transcribed not only by RT but by cellular DNA polymerases as well. Suffice to mention the study entitled: "Characteristics of the RNA-dependent DNA polymerase [RT] of a new human T lymphotropic retrovirus (lymphadenopathy associated virus)" ("HIV") in which Montagnier, Barré-Sinoussi, Chermann and their colleagues "characterised" the "HIV" RT. There they used the same ionic conditions as in 1983 and three template primers "Activated DNA", poly (A).oligo-(dT)12-18 and poly Cm .oligo-dG 12-18. They reported that while poly Cm .oligo-dG 12-18, "a reverse transcriptase specific template primer" was transcribed only by the "HIV infected" cells, "Activated DNA" and poly (A).oligo-(dT)12-18 were transcribed by both infected and non-infected cells.22 In other words, finding RT activity by using the template primer An.dT12-18 is not even proof for the existence of RT and even less for the existence of a retroviral RT.

11

No comment.

12

No comment.

13

We agree with Montagnier that when using lymphocyte cultures infected with exogenous retroviruses such as MT2, MT4 and H9 (HUT-78), all of which originated from patients with "adult T4-cell leukemia", said to be caused by HTLV-I, it "is a real soup". However, given the existence of endogenous retroviruses, when one uses lymphocytes from normal individuals and umbilical cord lymphocytes, the result is still "a real soup". Maybe a different soup, but nonetheless still "a real soup".

14

We agree that patients with AIDS and those at risk are infected with a "stack of things". Furthermore, the cultures with tissues from these patients in addition to these agents may also be infected in vitro with other agents, such as mycoplasma.

15

It may be true that sometimes it is easier to detect a particle with the morphological characteristics of a retrovirus in the culture than in the plasma. However, since the viral "concentrate" is obtained from the culture supernatant and since by definition a "concentrate" would have more particles per unit volume than the culture supernatant, it follows that it should be much easier to see a particle in the concentrate than in the culture. Since Montagnier and his colleagues "saw nothing major" in the "concentrate", that is, in the 1.16g/ml band, then why in their 1983 paper did they state the "concentrate" not only contained viral particles but "purified" virus? In the electron microscope picture which Montagnier and his associates including Charles Dauget published there are buds on the cell surface, some of which are more pronounced than others. But what is the evidence that they are virus or they are in the process of becoming a virus?

16
We agree it could be anything.

17
We agree that familiarity may sometimes enable one to distinguish between retroviral-like particles and other viral-like particles using morphological features. However, there are particles which are NOT viruses (including retroviruses) that exhibit identical morphological features as retroviruses. Therefore from morphological considerations both the buds and cell-free particles cannot be considered to be retroviruses. Furthermore, cultures of tissues derived from AIDS patients contain a plethora of viral-like particles with diameters ranging from 65-250nM, shapes which are spherical, angular and tear drop, surfaces with and without spikes, and which contain cone shaped, bar shaped, centrosymmetric and tubular cores, as well as double cores and a mixture of cores. Like the several particles of varying taxonomy deemed the "HIV" particle, none of these particles have been purified and characterised and, like "HIV", their origin and role must remain conjecture.[9,61-64]

18
1. If they did not purify the particles why did they claim to have done so and continue with the same claim up to this interview?
2. It is true that they reported the peak of RT activity at the density of 1.16g/ml, that is, at the density in which they claimed to have "purified, labelled virus". However, how is it possible to claim that the RT activity "was soundly that of a retrovirus", when they "didn't take the peak...or it didn't work", that is when at that peak they did not even find retrovirus-like particles, not to mention retroviruses? To pass a retrovirus from one culture to another, one must first have proof for the existence of a retrovirus in the first culture. "Passing" non-specific phenomena is no proof for passing a retrovirus. Furthermore, since all the phenomena which Montagnier and his colleagues considered as proof for the existence of a retrovirus, including RT activity and virus-like particles, could arise de novo in the cultures, especially under the culture conditions they used, they cannot claim proof for passing anything. How did Montagnier and his colleagues know that if they had suitable controls, the same phenomena would not have occurred in the blood donor's culture as well as the umbilical lymphocytes even if they were not "infected" with "HIV"?

19
1. If the stage of purification (isolation) is not necessary, then why did Montagnier and his colleagues claim to have proven the existence of "HIV" because they "isolated" it, "purified" it?

2. Since any piece of DNA can be cloned and amplified, cloning and amplifying a piece of DNA provides no information whatsoever in regard to its origin, that is, if it is retroviral or not. Neither is it possible by sequencing a piece of DNA to say that it is "truly a retrovirus" unless prior proof exists that these sequences are present in a retroviral particle and nowhere else. There is nothing specific about the "structure of retroviruses". If indeed there is a unique "sequence of DNA" indicating "it is truly a retrovirus" and "all the retroviruses have a familiar genomic structure with such and such a gene", then no such proof exists for the "HIV genome".[32]. Suffice to mention that to date no two identical sequences for the "HIV genome" have been published. One and the same patient may have different "HIV DNA" sequences. According to researchers from the Pasteur Institute, "an asymptomatic patient can harbour at least 10^6 genetically distinct variants of HIV, and for an AIDS patient the figure is more than 10^8.[65-66] The genetic differences may reach 40%.[67] (Compare this to the 1-2% difference between human and chimpanzee DNA). The length of the "HIV DNA" has been reported to be between 9-15Kb. In 1985 the Pasteur researchers reported that "The deduced genetic structure is unique; it shows, in addition to the retroviral gag, pol, and env genes, two novel open reading frames we call Q and F".[68] In 1990 the "HIV" genome was said to consist of ten genes,[69] in 1996 Montagnier reported that "HIV" possesses eight genes[70] and, according to Barré-Sinoussi,[71] "HIV" has nine genes.

20
1. For isolation of retroviruses the stage of purification IS obligatory. One CANNOT ISOLATE retroviruses WITHOUT PURIFYING. By definition, isolation means "to place apart or alone" (Concise Oxford Dictionary) and purify means "to clear of foreign elements" (Concise Oxford Dictionary). Thus, unless the contaminants are removed from around the "HIV" particles (that is, to purify the "HIV"), the "HIV" particles are NOT ISOLATED.

2. We agree that to transmit a retrovirus one does not need pure material. However, to transmit something, one first must know what one is transmitting, that is, one must have proof for its existence. For retroviruses such evidence can only be obtained by isolating (purifying) the particles, determining their physical and chemical properties and proving they are infectious.

21
Yes, it is impossible to determine the identity of the proteins including that of RT without isolation.
1. Montagnier and his colleagues, even after a Roman effort could not find even retrovirus-like particles at this density thus, from his experience (experimental evidence), there are zero chances and NOT 999 out of 1000 that RT activity at the density of 1.15, 1.16 represents a retrovirus in their case.

2. We agree that it could be a retrovirus of different origin. The existence of endogenous retroviruses, together with the presence in AIDS patients and those at risk of antibodies which react with their antigens, means that even if Montagnier et al had proven the existence of a retrovirus, it would have been impossible to say that the retrovirus originated in the gay man and not in the donors or umbilical cord lymphocytes.

3. The "molecular biology", the "cloning and sequencing" of the "HIV" genome has been discussed in detail elsewhere.[32-49] Suffice to mention here that:
(a) proof for the existence of "HIV" and indeed for its causative role in AIDS was claimed before any "molecular biology", "cloning and sequencing";
(b) since any piece of nucleic acid can be cloned and sequenced, cloning and sequencing of a piece of nucleic acid cannot be used to prove the existence of a retrovirus or of its genome. To the contrary, proof for the existence of viral nucleic acids (viral RNA and cDNA) can be accepted if and only if it is shown that the RNA is a unique molecular entity belonging to particles with morphological, physical and replicative characteristics of retroviral particles. This can only be done by separating the particles from everything else, by purifying them. Instead, Montagnier and Gallo used "a real soup" of cultures and co-cultures (Montagnier's group even purposely infected the cultures with Epstein-Barr virus). The supernatant from these cultures was banded in sucrose density gradients. From all the RNA (and DNA) which banded at 1.16g/ml they arbitrarily chose some RNA using totally non-retroviral specific criteria and called it "HIV RNA", without any proof that the band contained even retroviral like particles;[32]
(c) the first, absolutely necessary step in proving that the "HIV RNA", retroviral or not, originated from the lymphocytes of "HIV" infected individuals, is to perform hybridisation experiments using fresh, uncultured lymphocytes and the "HIV DNA" (obtained by reverse transcription of the "HIV RNA"), as a probe. It is hard to understand why Montagnier and his colleagues did not report such experiments. Gallo's group did and the results were negative. In 1994 Gallo was quoted in this magazine as saying: "We have never found HIV DNA in the tumour cells of KS...In fact we have never found HIV DNA in T-cells".[72] At present there is no study proving the existence of even one single copy of the "full-length HIV genome" in the fresh T-cells even of

a single AIDS patient or a patient at risk of AIDS;
(d) Currently the number of "HIV" particles in the plasma is quantified by measuring "HIV RNA", the viral load which is reported to be "15 x 10^3 to 554 x 10^3 virions per ml".[73] Many studies claim proof that the "viral load", the "HIV RNA", can be decreased to undetectable levels by the use of both RT and protease inhibitors. However, since:
(i) it is accepted that the "HIV RNA" is a transcript of the "HIV DNA";
(ii) by their nature neither the RT nor the protease inhibitors have any effect on DNA transcription, they only inhibit infection of new cells, that is, the decrease in "HIV RNA" is a consequence of the decrease in 'HIV DNA";
one would expect that the effect of these drugs would be determined by measuring the level of "HIV DNA". Yet hardly any such studies have been published. The very few which exist show that neither RT nor protease inhibitors have any effect on "HIV DNA",[74-76] which means that no relationship exists between "HIV RNA" and "HIV DNA".

4. In 1984 Montagnier and his colleagues reported that "preincubation of T4+ lymphocytes with three different monoclonal antibodies directed at the T4 glycoprotein blocked cell infection by LAV", that is, blocked the detection of RT activity in T4 cells "infected" with "HIV". They concluded their "findings strongly suggest that the T4 glycoprotein is at least associated with all or part of the receptor for LAV".[38]. However, blocking a non-specific "HIV" phenomena, namely RT activity, cannot be considered proof of blocking "HIV" infection or association of "HIV" with T4 cells.

22

We agree that "analysis of the proteins of the virus demands mass production and purification. It is necessary to do that". In this respect they have not just partially failed, but TOTALLY FAILED. If the "analysis of the proteins of the virus demands mass production and purification", so does the analysis of "nucleic acids, cloning etc". If one fails to purify the virus then it fails:
(a) to characterise the viral antigens and to obtain a gold standard for the antigen-antibody reaction, that is, one cannot use antibody tests to define infection with the retrovirus;
(b) to obtain and characterise the retroviral nucleic acids, RNA (cDNA) and thus probes and primers for hybridisation and PCR studies, that is, one cannot use molecular tests to define retroviral infection. That this is the case is accepted by Donald Francis, a researcher who with Gallo, played a significant role in developing the theory that AIDS is caused by a retrovirus. In 1983, Francis, then the chief collaborator of the AIDS Laboratory Activities, US Centers for Disease Control and former chief of the WHO smallpox program, speculated on a viral cause for AIDS: "One must rely on more elaborate detection methods through which, by some specific tool, one can "see" a virus. Some specific substances, such as antibody or nucleic acids, will identify viruses even if the cells remain alive. The problem here is that such methods can be developed only if we know what we are looking for. That is, if we are looking for a known virus we can vaccinate a guinea pig, for example, with pure virus...Obviously, though, if we don't know what virus we are searching for and we are thus unable to raise antibodies in guinea pigs, it is difficult to use these methods...we would be looking for something that might or might not be there using techniques that might or might not work"[77] (italics ours).

23

It is impossible to characterise two viral unknowns, namely its proteins and the antibodies directed against them, by the formation of an antibody/antigen complex let alone characterise the "virus". By what means did Montagnier know that somebody had antibodies against the proteins of the virus and that the proteins with which the antibodies react were viral? It is a scientific impossibility to know that somebody has antibodies to a virus and at the same time, the 1.16g/ml band contains proteins of the same virus before one has proven its existence.

24

1. It is true that Montagnier had controls but the controls were not suitable. Montagnier and his colleagues reacted the proteins which banded at 1.16g/ml with the sera from two gay patients with lymphadenopathy. The patients with AIDS and those at risk were already known to have a plethora of antibodies, all with potential for cross-reactivity. Therefore, one would have expected that Montagnier et al to have used as controls sick individuals who did not have AIDS or pre-AIDS and who were not at risk for AIDS but who also had a plethora of antibodies, all with potential for cross-reactivity. Instead their controls consisted of two blood donors whose state of good health is characterised by lower levels of antibodies.

2. Montagnier et al did not obtain proof for "a specific reaction". The sera from the patients and the donors were reacted with both the "purified virus", that is the 1.16g/ml band, and extracts from the "infected" cells. In their published strips, with "purified virus", it is not possible to distinguish any reacting proteins with any of the sera. In the text they state: "When purified, labelled virus [the 1.16g/ml band] from patient 1 was analysed...three major proteins could be seen; the p25 protein and proteins with molecular weight of 80.000 and 45.000". No such reactions were reported with the donors' sera. In the published strips with extracts from the "infected cells", it is obvious that many proteins reacted with both the patients' and the healthy blood donors' sera. One year later Montagnier and his colleagues confirmed that "sera from some AIDS patients bound a lot of cellular proteins...This banding was apparent in the RIPA and only sera which specifically precipitated the p25 were regarded as positive". In other words, for some unknown reason, they concluded that from all the reacting proteins only p24 (their p25) was retroviral and from all the antibodies only the one which reacted with p24 was directed against the retrovirus. Even if one considers the reaction between the p24 which bands at 1.16g/ml and the antibody present in the sera specific, that is, not due to cross-reactivity, from such a reaction it is impossible to draw the conclusion that p24 is retroviral protein and the antibody is elicited as a result of infection with this retrovirus. Indeed given the fact that Montagnier et al could not even detect retrovirus-like particles at 1.16g/ml, their conclusions regarding p24 and the antibody reacting with it completely defies scientific reasoning.

25

1. No antibodies, not even monoclonal antibodies are "very specific" or even specific.[78-84] Indeed, there are instances where "cross-reactive antigen binds with higher affinity than the homologous antigen itself...The most obvious fact about cross-reactions of monoclonal antibodies is that they are characteristic of all molecules and cannot be removed by absorption without removing all reactivity...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".[80] However, "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".[80] It is of importance to note that "HIV" experts concede "cross-reactivity" as the reason for "indeterminate" antibody reactivity seen in the "HIV" Western blot, as well as, for example, reactivity between monoclonal antibodies to the "HIV" p18 protein and dendritic cells in the lymphatic tissues of a variety of patients with a number of non-AIDS related diseases[85] and normal tissues taken from "non-HIV" infected individuals.[86] For one to be convinced that all "antibodies [including monoclonal] are polyspecific, that is, they are able to react with various dissimilar antigens such as: proteins, nucleic acids and haptens", "they are able to react with more than to self or non-self antigens, often without any apparent antigenic similarities", all one has to do is to read the scientific publications of the researchers from the Pasteur Institute such as Stratis Avrameas.[83-87]

2. It cannot be concluded that a protein which bands at 1.16g/ml is viral merely because it reacts with an antibody present in the patient's sera even if somehow one knows that the antibodies present in the sera are monoclonal. Let us assume an ideal situation where:
(a) all the antibodies present in the patients' sera are monoclonal and "very specific";
(b) the 1.16g/ml band contains in addition to the many unembodied and microvesicles, embodied proteins of cellular origin and maybe of bacterial, fungal and viral origin (constituents of the many infectious agents, other than retroviruses, present in the culture and the patients) and, as shown in a 1997 Franco/German study, a number of retrovirus-like particles.
Even in this ideal situation, it is NOT POSSIBLE TO CLAIM that just because a protein such as p24, p41, or others is found in this band and reacts with the sera, the protein is a constituent of the retrovirus-like particles.

3. The reality is that:
(a) all AIDS patients and those at risk have a plethora of antibodies including auto-antibodies. The auto-antibodies include anti-lymphocyte, and as Montagnier and his colleagues have shown[88] anti-actin and anti-myosin antibodies, that is antibodies to the two ubiquitous cellular proteins actin and myosin.
(b) all the antibodies present in the sera have the potential of cross-reactivity.
(c) the proteins from the supernatant of non-infected lymphocytes which in sucrose density gradients band at 1.16g/ml, the mock virus, include proteins having the same molecular weights as the "HIV" proteins;[89]
(d) animals inoculated with the mock virus develop antibodies which react with the "SIV" proteins, a "retrovirus" whose proteins share the same molecular weights as the "HIV" proteins and is said to be the closest relative of "HIV";[90]
(e) AIDS patients and those at risk are repeatedly subjected to allogenic stimuli including allogenic lymphocytes;
(f) up till 1997 no evidence existed showing that the 1.16g/ml band contained even retrovirus-like particles.
Given this reality, to claim that just because a protein bands at 1.16g/ml and reacts with antibodies present in the patients' sera is at best no different than the following:
(i) A researcher has two bowls, one of them contains a mixture of raw eggs, some known and maybe some unknown, and maybe some milk originating from several animals. The other contains several acids. Again some known and maybe some unknown. Once the contents of the two bowls are mixed he gets a precipitate. He claims that the precipitation proves the existence in the bowl of milk from a previously unknown animal and an unknown acid and that the reaction is between the unknown acid and a protein of the previously unknown milk.
(ii) This claim is scientifically impossible since any protein in the eggs could have reacted with any acid to produce the observed precipitate.
Thus, given the reality as outlined in (a) to (f) above, it is completely unscientific to claim that the reaction between proteins which band at 1.16g/ml and react with antibodies present in the patients' sera is proof of the existence of "HIV" proteins. To claim that the reaction between proteins which band at 1.16g/ml (in the absence of evidence that the band contains even retrovirus-like particles) with antibodies present in the sera indicates not only the band contains retroviral proteins, but proteins of a new retrovirus, is no different than the following: A fisherman has sea creatures but no fish in a net. He throws some animals into the net. The fisherman observes that the animals eat some proteins present in the net and claims that the proteins were not just fish proteins but the proteins of a completely new fish, a fish which nobody has seen before, a golden fish.

26
1. It is not possible for both Montagnier and Gallo to be "reasonably right". Both Gallo and Montagnier reacted the 1.16g/ml band with patient sera. Irrespective of the method used to detect the reaction (RIPA or WB), or the number of reactions performed, they should have found the same reacting proteins.
2. In their 1983 study, Montagnier and his colleagues found three proteins, p25, p45 and p80. Regarding p45 they wrote: "The 45K protein may be due to contamination of the virus by cellular actin which was present in immunoprecipitates of all the cell extracts". In a study published in 1984 they had "a prominent p25, a p18, a low molecular weight protein at the bottom of the gel (p12), and three proteins of high molecular weight (43.000, 53.000, 68.000). The band at 43.000 may include a component of cellular origin, since it was also found in a similar preparation made from the control uninfected cells".
3. Since both patients' and healthy blood donors' sera repeatedly reacted with the p45/p43 protein from both infected and uninfected cells one would have expected Gallo to also detect this protein. However neither Gallo nor anybody else since then reported such a band irrespective of the method used to detect the antigen/antibody reaction. The discrepancy can be resolved if one takes into consideration the fact that the migration of proteins in an electrophoretic strip, in addition to the molecular weight, may be also influenced by other factors, for example the charge carried by the protein. Thus one and the same protein may appear to have slightly different molecular weight when detected by either RIPA or the WB. For example, both p25 detected by Montagnier and the p24 detected by Gallo at present are considered to be both one and the same "HIV" protein p24.
4. The molecular weight of actin is neither 45,000 nor 43,000 but 41,000. At present there is ample evidence that the 1.16g/ml band the "Pure HIV" contains cellular actin[91-94] and as has been already mentioned Montagnier himself showed that the sera of AIDS patients and those at risk contain antibodies which react with actin. In other words when the 1.16g/ml band is reacted with patients' sera, irrespective of the presence of "HIV", a p41 (p45/43) band must be present, and represent cellular actin. (If Montagnier now believes that p41 is an "HIV" protein, why does he persist in excluding this band from his criteria for a positive Western blot?[95])

27
The p24 protein is not sufficient for diagnosing "HIV" infection because it is not specific. Indeed, no other "HIV" protein not even p41 (p45/43) has been reported to react more often with sera from healthy (at no risk of AIDS) individuals. Neither has a monoclonal antibody to any of the other "HIV" proteins been found to react more often with proteins present in non "infected" cultures or sera from individuals at no risk of AIDS. According to Montagnier because:
(a) "these are cellular proteins that one meets everywhere - there is a non-specific background noise";
(b) one such protein, having a molecular weight of 45/43, is actin;
(c) this protein reacted with sera from individuals at no risk of AIDS;
the p45/43 represents a cellular and not a viral protein.
However, since:
(i) myosin is as ubiqitous as actin.
(ii) myosin has a light chain with a molecular weight of 24,000.
(iii) the cytoskeletal proteins (of which actin and myosin are the most abundant) have been reported in "pure HIV".[91-94] Indeed, myosin and actin are said to play a crucial in budding and release of the "HIV" particles.[91]
(iv) Montagnier has shown that patients with AIDS and at risk of AIDS have anti-myosin antibodies.
Why should not one consider the p24 band as representing myosin?

28
We agree that no protein is sufficient to diagnose "HIV" infection. The problem then, as it is today, was not "to know whether it was an HTLV or not", but whether it was retroviral or not. Not everything which is not HTLV is retroviral.

29
1. To date there is no proof that any of the proteins

which band at 1.16g/ml are "HIV" proteins. The only reason that 20% of the proteins which band at 1.16g/ml are said to be "HIV" is that this fraction of proteins is found to react with different AIDS patient's sera at some time or another.

2. We agree that with the technique used by Montagnier's group, one cannot prove which proteins (or nucleic acids) are cellular and which are viral.

3. We agree. The only way one can prove the existence of the viral protein (nucleic acids) is "to purify the virus to the maximum", that is, to obtain density gradients which contain only particles with the morphological characteristics of retrovirus and nothing else. This has never been done to prove the existence of the "HIV" proteins and nucleic acids.

4. If one always "stumbles on the same proteins" in successive gradients, this is no proof that these proteins are viral and the ones which disappear are cellular.

30

1. No matter how many times the banding is repeated, if one starts with no retrovirus-like particles one will end with no such particles. Some times, by successive bandings, one may be able to eliminate non-retroviral components and obtain a band which contains nothing else but particles with morphological characteristics of retroviruses. However, to be able to do so, even after the first banding, one must begin with a relatively high proportion of retrovirus-like particles.

2. Once again, the origin of the proteins cannot be determined by molecular analysis, that is, by sequencing the proteins.

3. We agree that if the proteins of a retrovirus are coded by its genome, as is generally accepted, then it may be possible to characterise the retroviral proteins by its genome. However, to do this one must first prove that the RNA (cDNA) is a constituent of a retroviral particle. This has not been done for the "HIV" genome. In fact even today there is no proof that the "HIV" RNA is a constituent of a particle, any particle viral or non viral.

4. To date there is no proof of a relationship between the sequences in the "HIV" RNA (DNA) and the sequences in the proteins "observed with immunoprecipitation or with gel electrophoresis". In fact there is no relationship even between the size of the proteins coded by the "HIV" genes and the size of the proteins "observed with immunoprecipitation or with gel electrophoresis". For example, in 1987 Gallo and his associates performed a "computer-assisted analysis" of the "amino acid sequences of the envelope protein complexes derived from the nucleic acid sequences of seven AIDS virus isolates", and concluded that "gp41 should be about 52 to 54 daltons by calculation".[96]

5. One of the many puzzling aspects of "HIV" is the following:
(a) "HIV" experts agree that no two "HIVs" have the same genomic sequences and the difference may be as high as 40%;[67]
(b) They also admit that the vast majority (99.9%) of the "HIV" genomes are defective, that is, either part of a gene(s) or whole gene(s) are missing;
How then is it possible:
(i) to measure the viral burden ("HIV DNA") and the viral load ("HIV RNA") by using one and the same hybridisation probes and PCR primers?
(ii) to perform antibody tests for all the different "HIV"s using kits containing the same antigens?

6. Indeed, the history as to how "HIV" researchers have tried to prove the existence of p120 and how they ultimately agreed on its existence is very interesting.[32] However, given the fact that the p120 protein is said to be present only in the knobs, no cell-free "HIV" particles possessing knobs have been reported so far. It follows neither the particles in the culture supernatant nor the "pure" virus will have gp120. In other words, it is impossible for either the RIPA or the WB strips to have a "HIV" protein of molecular weight 120,000.

31

No such proof can be found in the published literature.

32

1. Prior to March 1997 no group of "HIV" researchers had published even a single electron micrograph of material banding at the density of 1.16gm/ml in a sucrose density gradient. The first EMs of material banded in sucrose density gradients appeared in 1997 in two publications, one Franco/German and the other from the US National Cancer Institute (NCI).[89] The Franco/German EMs are from the 1.16 gm/ml sucrose density gradient whereas it is not possible to tell from which density the NCI data originate. The data from both studies reveal that the vast majority of the material is "non-viral", "mock" virus, cellular "microvesicles", that is, the banded material is virtually all cellular. These particles, like the retroviral particles, contain nucleic acids in addition to proteins but they are not as condensed.

2. The EM micrographs in both studies also contain a small minority of particles which have morphologies more closely resembling retroviral particles than the "mock" particles. Both groups claim the fewer particles are "HIV".

3. In the NCI study no reasons are given for the claim that these particles are "HIV". The authors of the Franco/German study claim that the particles are "HIV" because they have:
(a) "diameters of about 110nm;"
(b) a "dense cone-shaped core";
(c) "lateral bodies";
and because no such particles were seen in the banded material from the "non-infected" control cells.
However, according to well known retroviral researchers such as Bader and Frank, one type of "oncoviral particle" can change to another, and "immature" cores to "mature", merely by changing the extracellular conditions.[11-97] However the culture conditions in the "infected" and non-infected cells were not the same. A diameter of 100-120nm and surface knobs are two morphological characteristics shared by all retroviruses. None of the particles appear to have knobs and none has a diameter of less than 120nm. Averaging the major and minor diameters of the particles indicated and said to represent "HIV" and, assuming all particles are spherical, shows that in the Franco/German study the particles are 1.14 times larger than bona fide retroviral particles and the NCI particles are 1.96 times larger. These data translate into volumes 50% and 750% greater respectively. Since density is the ratio of mass to volume these particles must therefore have correspondingly higher masses. Given the maximum diameter of retroviral particles and the fact that such particles contain a fixed mass of RNA and protein, it appears untenable that the particles which both groups regard as "HIV" are the same particle or are retroviral particles. The only other explanation for these data is that the electron micrographs are not from the 1.16gm/ml band or the banding has not been to equilibrium in which case one must redefine the buoyant density of retroviruses.
The "HIV" particles are said to have a cone shaped viral core, with dense lateral bodies at either side of the core. No such feature can be seen in the EM published in these two studies. Thus, by definition, the particles cannot even be said to be retrovirus-like.
Taking into consideration that in both studies the control "non-infected" cultures were of H9 cells and the fact that Gallo as far back as 1983 claimed that these cells are infected with HTLV-I, the non-reporting of virus-like particles in the banded material from these cultures is an enigma.

33

Pictures of the 1.16g/ml are of profoundly significant interest. How else can it be known that there are retrovirus-like particles there, especially since even Montagnier admits that other things may band there. For any scientist who claims proof for isolation, purification of a retrovirus using sucrose density gradient banding, it is vital and absolutely necessary to obtain electron micrographs of the 1.16g/ml band showing nothing else but retrovirus-like particles.

34

If this is the case why is such data not available in the scientific literature?

35

In one of their 1984 papers[22] Montagnier and his colleagues wrote, "Several characteristics indicate that LAV or LAV related viruses belong to the retroviruses family. Budding particles at the plasma membrane have been observed in electron microscopy. The density of the virus in sucrose gradient is 1.16 and a Mg^{2+} dependent reverse transcriptase activity has been found to be associated with RNA containing virions". However,
in this interview Montagnier admits:
(a) "We published images of budding which are characteristic of retroviruses. Having said that, on the morphology alone one could not say it was truly a retrovirus...With the first budding pictures it could be a type C virus. One cannot distinguish...No .. well, after all, yes...it could be another budding virus".
(b) at the sucrose density of 1.16 gm/ml not only did Montagnier and his colleagues not see a retrovirus particle, they repeatedly said they did not see retroviral-like particles;
(c) although at the sucrose density of 1.16 gm/ml they detected reverse transcription of the template primer An.dT12-18 in the presence of Mg2+, they had no particles and thus no evidence for "reverse transcriptase activity found to be associated with RNA containing virions".

Furthermore, in this study[22,] they showed that DNA polymerases ß and gamma and of non-infected cells reverse transcribe $An.dT^{12-18}$ in the presence of Mg^{2+}. Thus, Montagnier's own conditions and data do not prove his claim that what he has "seen" and "encountered" is a retrovirus. If "HIV" "exists", and it is "clear" to Montagnier that he has "seen it" and "encountered it", where is his proof?

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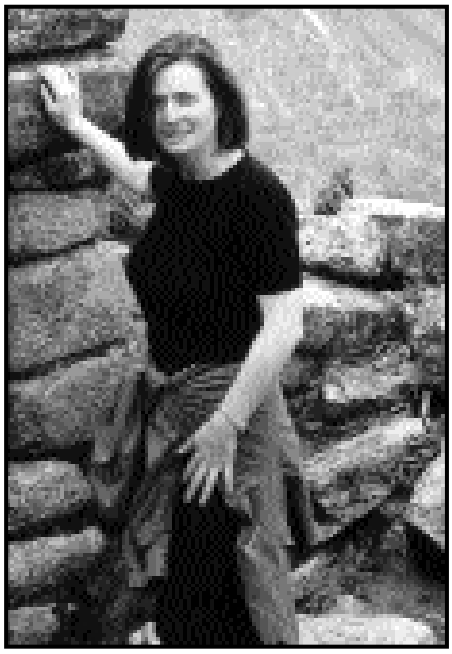
LONG-TERM SURVIVAL STUDY

By Clair Walton

Clair Walton was given an hiv antibody diagnosis in 1987. Eleven years later, at the age of 36, she has become aware of the deep and damaging psychological and social implications of a hiv antibody diagnosis. Through her past involvement with several hiv/aids organisations, including Positive Space for Women/OXAIDS and Positively Women, and her attendance at many conferences, including the Global Network of People Living with hiv/aids Conference in Cape Town, 1995 and the Long-Term Survivors Conference in London, 1996, she believes the concerns of diagnosed people around the issue of long-term survival are often marginalised.



For many challenged by an hiv antibody diagnosis the possibility of long-term survival has become a reality. Many of us are aware that not only are we very much alive and well after many years but have discovered that there are challenges, from within the scientific community to the fundamental hiv/aids science, that may explain our continued existence.
Unfortunately, it is has been left to individuals and organisations such as Continuum to disseminate the information, with many of the aids organisations purporting to represent the interests of the individual either repressing, indifferent to or totally ignorant of that knowledge.
How can someone make decisions affecting their future if not fully aware of the facts? The simple fact that the isolation of the putative.virus has been challenged could play a vital role in an individual's appraisal of their future.
Incredibly, the sixty or so accepted conditions producing cross-reactions to the test are not known to many diagnosed. So even if they accept the view that the virus exists and it causes aids, they may not be aware that they may not be considered positive under the orthodoxy if tested differently, and ignorantly continue to allow themselves to be assaulted by the yet to be proven hypothesis.

The original prognosis of certain death was always an unjustified burden and in itself arguably instrumental in the fulfilment of the prophecy as psychological torment and self destruction came into play. With the notion of the certain death sentence foisted on a gullible public, seemingly unquestioned by the scientific community and accepted by many of the mushrooming aids organisations, we, the diagnosed, were left with the onus to prove otherwise. But why should we provide the counter-evidence? - It wasn't our hypothesis. In any case, it is particularly difficult to prove survival when the goal posts keep moving; as the latent period extends or the death sentence is relaxed.
Whilst there have been several scientific studies of long-term survival published, a comprehensive study embracing some of the factors many survivors themselves believe are responsible has not been forthcoming, nor have most of the previous studies accessed those outside conventional medicine.
A study is currently being prepared to redress that urgent need. Initiated by the group Action positive Switzerland (ApS) the study will be carried out by several organisations, including Gay International Association Trust(GAIA) and Continuum, hopefully accessing as many diagnosed as possible with a questionnaire covering aspects of health history of the individual. The study evaluates health prior to testing as well as the various strategies for survival after. The study has been planned for completion in twelve months with questionnaires being distributed as widely as possible in several countries and analysed by a panel of various professionals; research physicians, clinical psychologists and clinical social workers plus long-term survivors.

Continuum is eager to participate in the study in the hope that it will provide insight into the many factors that effect health but also hopefully awaken the closed-minded diagnosed to the fact that survival is a reality. The timing of this is all the more important as the push, aided by an ignorant media, is towards having us believe that survival is now being achieved through the use of drugs. It is important that those who have chosen to reject the orthodox view, and consequently the drugs, participate in a study so that the facts are registered.

For more information contact Clair at Continuum (see index page)