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A critical analysis of the Anders Vahlne paper: "A historical reflection on the discovery of human retroviruses"

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We agree with Dr. Vahlne that when an RNA directed DNA polymerase (reverse transcriptase) was discovered in 1970 in "oncogenic RNA viruses (then called oncorna viruses [oncoviruses] and now called retroviruses)", some scientists were of the opinion that reverse transcription is a property specific to retroviruses. But by 1980 there was ample evidence this was not the case. In fact, during the 1970s Robert Gallo and his associates were among the first to show that reverse transcription is not only catalysed by reverse transcriptases but also by the cellular polymerases β and γ, especially when synthetic primer-templates such as $(dT)_{15}\cdot(A)n$ are used. From this Gallo concluded the only way to prove reverse transcription is catalysed by a retroviral enzyme is to extract the enzyme from retroviral particles purified by banding in density gradients.

In regard to "DNA-polymerase γ", Vahlne makes contradictory statements. On the one hand he claims that "DNA-polymerase γ is a normal cellular DNA polymerase which uses RNA as a primer but not as a template" while on the other the "preferred primer-template for DNA-polymerase γ is $(dT)\cong15\cdot(A)n$ over $(dT)\cong15\cdot(dA)n$!".

Indeed, in 1975, an International Conference on Eukaryotic DNA polymerases defined DNA polymerase γ as the cellular enzyme which "copies $(dT)_{15}\cdot(A)n$ with high efficiency but does not copy DNA well".[1]

Montagnier's 1983/1984 evidence for the isolation of HIV

Montagnier's 1983 *Science* paper is accepted by most scientists, including the Nobel committee, as proof of the existence of a new and unique retrovirus, HIV. In his detailed description of this evidence Vahlne, like Montagnier and his colleagues, considers detection of reverse transcriptase (RT) activity in the cell culture containing lymphocytes from the lymph nodes of patient BRU, as proof for "Virus production". In regard to virus transmission Vahlne wrote, "to show virus transmission, cells from patient B.R.U. after three days in culture were also co-cultured with lymphocytes from a healthy donor of the Blood Transfusion Center at the Pasteur Institute. Also with these co-cultures, RT could be detected after 15 days of culture (not before) and amounts of RT remained stable for 15 to 20 days. Transmission of cell-free supernatants from the original culture of B.R.U. cells [it was from the co-culture, not the original BRU culture] was successfully obtained using 3-day-old cultures of T lymphocytes from two umbilical cords".

The only evidence for reverse transcriptase activity in the 1983 Montagnier paper, and thus for what Montagnier defined as HIV isolation and transmission, is transcription of the template-primer $An.dT_{15}$. Since this template-primer is not specific to the retroviral reverse transcriptase, or any reverse transcriptase (it can be transcribed by cellular DNA polymerases β and γ), its transcription cannot be considered proof for virus infection,

propagation and transmission, which is the definition of virus isolation provided by Vahlne.

Like Montagnier and his colleagues, Vahlne states, "The virus isolate had a density of 1.16 (same as HTLV-I) in a sucrose gradient". (Banding at the density of 1.16 g/ml in sucrose gradients is a characteristic of all retroviral particles). Montagnier and his colleagues banded the supernatant from the umbilical cord lymphocytes and claimed the 1.16 g/ml band was "purified" virus. However, they did not publish an electron micrograph (EM) to prove that the "virus isolate" contained virus particles, although they were fully aware that viruses are particles and particulate matter other than retroviral particles may band at the same density. These include membranous vesicles and cellular fragments which may contain RT and nucleic acids. The only evidence they had for the existence of a retrovirus in this band was transcription of the template-primer $An.dT_{15}$. In an *en camera* interview he gave to the French journalist Djamel Tahi in 1997, Montagnier gave the reason for not publishing such an EM: in the 1.16 g/ml band they were unable to find any particles with the "morphology typical of retroviruses", much less particles of a unique retrovirus.[2]

Being aware that HIV is said to be a lentivirus but the particles which Montagnier had identified in the umbilical cord lymphocytes culture were a "typical C-type virus", and after analysing Montagnier's immunological and molecular evidence, Vahlne concluded, "In reality, in my view there is no evidence whatsoever in this paper that a new human retrovirus has been isolated! With the data presented, the virus they isolated could well have been HTLV-I or in particular HTLV-II".

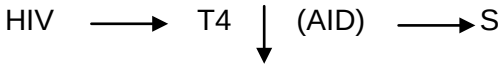
Vahlne claims that Montagnier proved the existence of HIV four months later, in September 1983, at a meeting held at Cold Spring Harbor. "The oral presentation by Luc Montagnier at this meeting is to my mind the first report on a new third human retrovirus, in that electron micrographs on the isolate LAV from patient BRU clearly showed virus with conical cores. A selective tropism of LAV to CD4 positive T-cells (as is the case for HTLV-I) was also demonstrated. The first publication in a peer reviewed journal indicating the isolation of a new retrovirus, distinct from HTLV-I and HTLV-II, isolated from two siblings with hemophilia B of whom one had AIDS, appeared in *Lancet* in April of 1984 and was written by the French group [23]. Again the immunological and molecular characterization of the isolated virus does not convincingly separate the isolated virus from HTLV-I. However, an electron micrograph clearly depicts a virus with a lenti retrovirus type morphology having a cylindrical or conical core, distinctly different from the larger spherical core of HTLV-I, and HTLV-II". In Vahlne's reference 23 Montagnier describes the isolation of HIV from two siblings with haemophilia B.[3] Contrary to what Vahlne states, the morphology of the retrovirus was described by Montagnier as follows: "…ultrathin sections of patient 1's lymphocytes and of in-vitro-infected lymphocytes from a normal donor showed immature particles with a dense crescent budding at the cell surface and mature particles with a small dense eccentric core in the extracellular spaces (fig2). The morphology of these particles was similar to that seen in preparations of T lymphocytes infected with LAV[5]". Reference 5 cited by Montagnier is his 1983 *Science* paper where the particles, as Vahlne pointed out, are described as "typical C-type virus, i.e. with a spherical core (same as HTLV-I)". We agree with Vahlne, seeing is believing. However, since (a) according to Vahlne the only significant difference between the evidence in the 1983 paper and in the April 1984 is "an electron micrograph [which] clearly depicts a virus with a lenti retrovirus type morphology"; (b) according to Montagnier the particles in his April 1984 paper were

typical type-C particles; it follows that Montagnier did not discover a unique retrovirus (HIV), neither in 1983 nor 1984.

According to Hans Gelderblom, arguably the leading expert on the electron microscopy of HIV, "Retroviruses are enveloped viruses with a diameter of 100 to 120 nm budding at cellular membranes. Cell released virions contain condensed inner bodies (cores) and are studded with projections (spikes, knobs)" The HIV particles "can be identified by the relatively homogeneous diameter of about 110 nm, the dense cone-shaped core, and the "lateral bodies"".[4, 5] Neither Montagnier in 1983/1984 nor Gallo in 1984, published electron micrographs showing particles with such morphology. It is significant that in his Nobel lecture, the 16th slide Montagnier showed was an EM of the particles he addressed as HIV. Yet none of these particles satisfy the morphological criteria for a lentivirus.

Gallo's May 1984 evidence for the existence of HIV and its causative role in AIDS
Vahlne wrote, "In conclusion, by April of 1984 the Pasteur group headed by Luc Montagnier had reported on a new human T-lymphotropic retrovirus distinct from HTLV-I and HTLV-II as judged by morphology and which was present in a few patients with AIDS and lymphadenopathy, as well as, in people at risk of acquiring of AIDS. The virus-infected CD4-positiveT-lymphocytes, the very cells affected in AIDS. Although clearly associated with AIDS, they had not yet shown that the new virus was an etiological agent, and the only at that, of this new disease".

Vahlne claims it was Gallo who proved "that the new virus was an etiological agent, and the only one at that, of this new disease", in four papers published in *Science* in May 1984. The HIV theory of AIDS states: HIV infection, by means which are not fully known, causes the death of T4 cells (acquired immune deficiency = AID); AID leads to the clinical syndrome (S), that is, to the AIDS indicator diseases which are the immediate cause of morbidity and mortality from AIDS.



For Vahlne's claim to be accepted Gallo's papers must contain evidence that proves:

1. The existence of a new retrovirus.
2. The retrovirus is present in all AIDS patients.
3. The retrovirus kills the T4 cells.
4. The decrease in the T4 cells leads to the appearance of diseases which lead to the patients' deaths.

Vahlne states: "On May 4th of 1984 four papers by Robert C. Gallo's group were published in *Science* describing a new human retrovirus virus". The evidence for a new retrovirus is presented in the first and third paper in the series. The principal author of the first paper is Mikulas Popovic and is entitled "Detection, Isolation and Continuous Production of Cytopathic Retroviruses (HTLV-III) from patients with AIDS and Pre-AIDS". The principal author of the third paper is Jorg Schubach and is entitled "Serological Analysis of a Subgroup of Human T-Lymphotropic Retroviruses (HTLV-III) Associated with AIDS". As evidence for the isolation and characterisation of the virus, Gallo, like Montagnier, relied on transcription of the template-primer An.dT_{15}, electron microscopy and immunological data. As Gallo repeatedly pointed out, the two main differences between his and Montagnier's study were:

(i) Gallo used a neoplastic cell line “derived from an adult with lymphoid leukaemia”;
(ii) Unlike Montagnier, who had only one patient, BRU, Gallo had many.

As Vahlne states, a total of 51 single cell clones were obtained from the leukaemic cell line termed HT. One of the clones, H4, was cultured with patients’ T-cells. The cells were first kept for 60 days in culture to which IL-2 was added. To another clone, H9, Gallo added cell-free fluid harvested from T-cell cultures of a patient with lymphadenopathy. They reported virus isolation from the H9 culture and four co-cultures.

Popovic, Gallo and their associates also stated, “The yield of virus from H4/HTLV-III cells was assessed by purification of concentrated culture fluids through a sucrose density gradient and assays of particulate RT activity in each fraction collected from the gradient. As shown in Fig. 2b, the highest RT activity was found at a density of 1.16 g/ml, which is similar to other retroviruses. The highest RT activity was found in the fractions with the largest amount of virus, as determined by electron microscopy”.

However, like Montagnier, Gallo did not publish electron micrographs of the 1.16 g/ml band to prove purification or to justify the RT activity as “particulate”. In his density gradient material Gallo claimed there were purified retroviral particles but without electron micrographic proof it is not possible to claim the existence of particles of any kind, let alone purified particles of retroviral morphology. The published electron micrograph is from the cell culture and has the following caption: “Electron micrograph of the cells showing the presence of extracellular viral particles”.

In regard to the cytopathic effect, in the text of this paper one reads: “The virus positive cultures consistently showed a high proportion of round giant cells containing numerous nuclei (Fig. 1a). These cells resemble those induced by HTLV-I and -II (9) except that the nuclei exhibit a characteristic ring formation”. Yet in “Table 1” Gallo says that “Cloned cells from uninfected cultures also contained some multinucleated giant cells; however, the arrangement of the multiple nuclei in a characteristic ring formation was lacking (see Fig. 1a) and the number of those cells was much less”. In other words, multinucleated giant cell formation was not specific to the virus. (Multinucleated cells are non-specific. The difference in their number and arrangement of all their nuclei may have nothing to do with a virus and be a result of the differences in the history and mode of preparation of the different clones).

In the third paper “Lysates of two immortalized infected human T-cell clones, H4/HTLVIII and H17/HTLV-III, were tested with samples of human serum in a strip radioimmunoassay (RIA) based on the Western blot technique...Sera from patients with AIDS or pre-AIDS, and from some homosexuals and heroin-addicts, recognized a number of specific antigens not detected by sera from heterosexual subjects. The most prominent reactions were with 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”. Interpreting their findings Gallo and his colleagues wrote, “These results show clearly that the antigens detected after virus infection are either virus-coded proteins or cellular antigens specifically induced by the infection”. To find out which of these antigens are viral, “The antigens of H4/HTLV-III were also compared with antigens from virus purified from the culture fluids of H4/HTLV-III”. To obtain purified virus, the culture fluids were

banded in sucrose density gradients. The proteins in the band which were said to be "purified virus" were incubated with sera from a few patients. Two proteins, p24 and p41 were found to react. They concluded "P24 and p41 may therefore be considered viral structural proteins". However, Gallo in 1984, like Montagnier in 1983, did not publish electron micrographs to prove that the material which he called purified virus contained purified retroviral particles or unpurified retroviral particles or indeed particles of any kind, purified or not. In 1997, when Montagnier was asked if Gallo purified HIV he replied "I don't know if he really purified. I don't believe so".[2]

Although Gallo did not have proof that the antigens in what he called purified virus belonged to retrovirus particles, the reaction of some of the antigens with antibodies present in the patient sera was considered proof that both antigens and antibodies were HIV. However, given the nature of antibody reactivities, from such a reaction one cannot define the origin even of one reactant, much less both.

In the same paper, Gallo describes many similarities between the HLV-III proteins with those of HTLV-I and –II as well as immunological cross-reactivity. The caption to an electron micrograph published in this paper reads: "Note the dense cylindrical core region of HTLV-III".

Gallo is the principal author of the second Gallo group *Science* paper entitled: "Frequent Detection and Isolation of Cytopathic Retroviruses (HTLV-III) from Patients with AIDS and at Risk for AIDS". Summarising their findings in regard to their virus the authors wrote, "That the viruses we have named HTLV-III belong to the HTLV family is indicated by their T cell tropism, Mg^{2+}- dependent RT of high molecular weight, antigenic cross-reactivity with HTLV-I and -II, cytopathic effects on T lymphocytes, and **their morphological appearance in the electron micrograph**. HTLV-III also contains some structural proteins similar in size to those of other members of the HTLV family" (emphasis ours).

Note:
(i) The title of all his papers contains the word retroviruses, not retrovirus.
(ii) HTLVs are not lentiviruses. They are type-C particles.

Obviously, given Vahlne's criteria, there are as many reasons in the Gallo papers as there are in the Montagnier papers for one to conclude, "there is no evidence whatsoever in this paper[s] that a new human retrovirus has been isolated. With the data presented, the virus they isolated could well have been HTLV-I or in particular, HTLV-II".

There is also the question of the cell line which Gallo used to characterise his virus. Reading Gallo's first *Science* paper one gets the impression that the cell line which they called HT was established in Gallo's laboratory. In fact the HT cell line was none other than the HUT-78 cell line. This cell line, as well as the cell line HUT-102, "originally cultured [in 1977] by Dr. Carney were maintained in culture under the direction of Dr. Adi Gazdar. HUT-78 was derived from peripheral blood of a 53-year-old white male with Sezary syndrome".[6] According to Gallo "The patients from whom we first isolated HTLV-I had malignancies of mature T4 cells accompanied by skin abnormalities, which result from infiltration of the skin by malignant blood cells…Such a clinical picture has been called mycosis fungoides or Sezary T-cell leukemia". In 1983 Gallo and his colleagues published a Letter to *Nature* in which they reported that HUT 78 "contained HTLV

proviral sequences".[7] In 1993 evidence was published that non-infected H9 cells (HUT-78 cells) express retrovirus-like particles.[8]

Vahlne not only claims that, unlike Montagnier's 1983 evidence, Gallo's 1984 evidence proves the existence of a new retrovirus, but the evidence in two of the papers proves, for the first time, that the virus is the cause of AIDS. One of them is the second paper in the series of four with Gallo as a principle author. As the title implies, "Frequent Detection and Isolation of Cytopathic Retroviruses (HTLV-III) from Patients with AIDS and at Risk of AIDS", Gallo reported that, "Retroviruses belonging to the HTLV family and collectively designated HTLV-III were isolated from a total of 48 subjects including 18 of 21 patients with pre-AIDS, three of four clinically normal mothers of juveniles with AIDS, 26 of 72 adult and juvenile patients with AIDS, and from one of 22 normal male homosexual subjects". In other words, HIV could not be isolated from more than half the pre-AIDS or AIDS patients. Or, from only 36% of the AIDS cases.

The other paper is the fourth in the series. The principle author is Sarngadharan and is entitled "Antibodies Reactive with Human T-Lymphotropic Retroviruses (HTLV-III) in the Serum of Patients with AIDS". Discussing the evidence in this paper Vahlne wrote: "antibody reactivity to HTLV-III antigens in patients with pre-AIDS and AIDS was determined by an enzyme-linked immunosorbent assay (ELISA) as well as a Western electrophoretic blotting technique using a lysate of sucrose gradient purified HTLV-III from a cell line continuously producing HTLV-III as antigen". Yet, as already mentioned, nowhere in these papers is there evidence for purification. "The number of sera that gave positive scores in the ELISA were: 43 of 49 (88%) of patients with AIDS (two of whom had developed AIDS after blood transfusion), 11 of 14 patients with pre-AIDS, 3 of 5 intravenous drug users (of which one positive was also homosexual), 6 of 17 homosexual men…Of note, in Western blot the antigen most prominently and commonly detected among all of the sera from AIDS patients had a molecular weight of 41,000 (now designated gp41)…The French group did not detect gp41 in their immune precipitation studies using purified LAV".

First, in his "purified" virus, where Montagnier did not have even retrovirus like particles much less particles of a unique virus, he found three proteins; p80, p25 (now called p24) and p45. The latter protein he said was cellular actin, which contaminated his "purified" virus. The molecular weight of actin is 41,000 not 45,000. Furthermore, according to researchers from AIDS Vaccine Program, National Cancer Institute, the major band of "~ 42-kDa" found in the sucrose purified virus, is actin.[9] (These variations can be accounted for by the conditions applying in the gels in which the MWs were determined). Second, not all the sera which reacted in the ELISA were retested with the WB. Significantly, Gallo considers the WB test to be more sensitive than the ELISA.

Even if one assumes that Montagnier and Gallo had proof that both p24 and p41 were constituents of a viral particle, the antibodies found in patient sera which react with them cannot be assumed to be directed against such a virus. In other words, such a reaction cannot be considered proof that the patients are infected with this virus. There are many reasons for this including the fact that antibodies cross-react and AIDS patients have a plethora of antibodies directed against non-retroviral infectious agents and non-infectious agents, including auto-antibodies. Any of these antibodies may cross-react with p24 and p41 irrespective of their origin. Even if they are antibodies directed against a retrovirus, the retrovirus may be HTLV-I or HTLV-II. Gallo wrote, "Both HTLV-I and HTLV-II have been isolated from cultured T-cells of patients with AIDS…Proviral DNA of

HTLV-I was detected in the cellular DNA of two AIDS patients, and serum samples from some patients were shown to react with antigens of HTLV-I. A larger proportion of the sera reacted with a cell membrane antigen specific to HTLV-I-infected cells...Immunological studies presented in an accompanying report also indicate that HTLV-III is antigenically different from HTLV-I and -II, but that it also shares a variety of antigenic determinants with them, especially with HTLV-II".

Most importantly, according to Gallo and others, antibodies which react with the retroviral glycoproteins are widespread and are directed "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".[10] Indeed, this is the discovery that led to the demise of Gallo's "first human retrovirus", HL23V.[11, 12]

Nonetheless:

a. The reported reactivity of antibodies present in the patient sera with p24 and especially with the glycoprotein gp41 in the fourth paper;
b. Virus isolation "from a total of 48 subjects including 18 of 21 patients with pre-AIDS, three of four clinically normal mothers of juveniles with AIDS, 26 of 72 adult and juvenile patients with AIDS, and from one of 22 normal male homosexual subjects", in the second *Science* paper

led Vahlne to conclude, "Taken together, these two papers from Gallo's group for the first time convincingly demonstrated that AIDS was caused by a new human retrovirus distinct from HTLV-I and HTLV-II". However, even if one accepts in the four *Science* papers there is evidence for a new retrovirus, there is no evidence which proves the second absolutely necessary, but not sufficient condition for this claim. In other words, all the AIDS patients are infected with a unique new retrovirus, or any retrovirus. Even if such evidence existed it could only prove a correlation. Correlation does not prove causation.

In regard to immune deficiency (low T4 cells) in their patients, Gallo and his colleagues wrote: "The 48 HTLV-III isolates were obtained from 18 of 21 patients with unexplained lymphadenopathy and leukopenia with an inverted T4/T8 lymphocyte ratio". However, there were no data documenting (i) the absolute numbers of T4 or T8 lymphocytes such that low ratios caused by low T4s, high T8s or both could be distinguished; (ii) that all patients from whom HIV was isolated had an inverted ratio; (iii) the immune status of the patients from whom HIV was not isolated. In addition, on the basis of these four papers, Gallo was never in a position to prove that infection with HIV preceded immune deficiency, even if we ignore the ambiguity of "an inverted T4/T8 ratio". Without proof of a temporal relationship it is impossible to prove HIV causes AID and thus AIDS.

Patients' T4 and T8 cells are only mentioned in the first and second paper. In the second paper one reads, "AIDS is diagnosed as a severe, unexplained, immune deficiency that usually involves a reduction in the number of helper T lymphocytes and is accompanied by multiple opportunistic infections or malignancies. A number of other clinical manifestations, when occurring in members of a group at risk for AIDS, are identified as its prodrome (pre-AIDS). These include unexplained chronic lymphadenopathy or leukopenia involving a reduction in the number of helper T lymphocytes". A similar comment can be found in the first paper. Even if Gallo had

proven that all his patients had low T4 cell counts, the cause could have been other than HTLV-III.

In none of the papers is there evidence that HTLV-III kills T4 cells, a fact absolutely necessary but not sufficient to prove the HIV theory of AIDS. To the contrary, in the second paper, with Gallo as principle author, one reads. “Primary cells from patients usually produce virus for 2 to 3 weeks (Fig. 1). After this time the production of virus declines even though the culture may contain actively replicating cells that can be maintained for long periods in the presence of added T-cell growth factor (TCGF)”.

Nowhere in the four *Science* papers is there evidence which proves the last, absolutely necessary but not sufficient condition, that is, a decrease in T4 cells leads to the clinical syndrome.

Vahlne wrote, “In the July 6th issue of *Science* (submitted April 6th 1984), Donald Francis’ group in collaboration with Montagnier’s group reported on the isolation of a retrovirus from a blood donor-recipient pair with AIDS [31]”. In the cited reference, Donald Francis, Montagnier and their associates concluded, “An etiologic association between HTLV-III and AIDS was reported recently (4). The most likely explanation for the parallel evidence for HTLV-III and LAV being the cause of AIDS is that the two viruses are the same”. Reference 4 is Gallo’s four *Science* papers. Since HTLV-III and LAV (the virus from BRU isolated in May 1983) “are the same” and since according to Vahlne, Montagnier’s LAV is not new but HTLV-I or HTLV-II, then neither is HTLV-III new. This means even if, in the four *Science* papers evidence had existed which proved causation, the virus could not have been HIV.

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