

Christine Johnson - Immunity Resource Foundation

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Does HIV Exist?

An interview with Eleni Papadopulos-Eleopulos

Christine Johnson

Dr. Eleni Papadopulos is a biophysicist and leader of a group of HIV/AIDS scientists from Perth in Western Australia. Over the past decade and more she and her colleagues have published many scientific papers questioning the HIV/AIDS hypothesis. This interview by Christine Johnson looks at this work and especially her group's views on the AIDS virus itself.

CJ: Does HIV cause AIDS?

EPE: There is no proof that HIV causes AIDS.

CJ: Why not?

EPE: For many reasons, but most importantly, because there is no proof that HIV exists.

CJ: That seems a rather bold and incredible statement to make.

EPE: I suppose it is, but, nevertheless, that's where my research takes me.

CJ: Didn't Luc Montagnier and Robert Gallo isolate HIV back in the early eighties?

EPE: No. In the papers published in Science by those two research groups, there is no proof of the isolation of a retrovirus from AIDS patients [1, 2].

CJ: They say they did isolate a virus.

EPE: Our interpretation of the data differs [3-5]. To prove the existence of a virus you need to do three things. First, culture cells and find a particle you think might be a virus. Obviously, at the very least, that particle should look like a virus. Second, you have to devise a method to get that particle on its own so you can take it to pieces and analyze precisely what makes it up. Then you need to prove the particle can make faithful copies of itself. In other words, that it can replicate.

CJ: Can't you just look down a microscope and say there's a virus in the cultures?

EPE: No, you can't. Not all particles that look like viruses are viruses.

CJ: So where did AIDS research go wrong?

EPE: It's not so much a question of where the research went wrong. It's more a question of what was left out. For some unknown reason the decades-old method of retroviral isolation [6,7] developed to study animal retroviruses was not followed. Retroviruses are incredibly tiny, almost spherical particles with diameters of about one hundred nanometers (one ten-thousandth of a millimeter). Millions would fit comfortably on the head of a pin.

CJ: How can you see something that tiny?

EPE: You need an electron microscope. That's how we know the size and shape of retroviral

particles, that they're almost round and have an outer envelope covered with knobs and an inner core consisting of some proteins and RNA. Another important point is that retroviruses do not directly use their RNA blueprint to make more virus. According to retrovirologists, what sets them apart from nearly all other viruses is that retroviruses first make a DNA copy of their RNA. This DNA then moves into the cell nucleus where it becomes part of the cellular DNA. This stretch of DNA is called a provirus, and there it sits, hibernating, perhaps for years, until something activates the cell.

CJ: What happens then?

EPE: The proviral DNA is copied back into RNA, and it is this RNA, not the original RNA, that instructs the production of the necessary proteins to make new virus particles.

CJ: Why are they called retroviruses?

EPE: Because for a long time biologists believed that the only direction of information flow in the cells of all living things was from DNA to RNA, and thence to the proteins whose synthesis the RNA instructs. If we say this direction is "forwards" then what retroviruses do first is copy their information "backwards." One of the proteins inside a retrovirus particle is an enzyme which catalyses this [backwards] process [of turning RNA into DNA]. That enzyme is called reverse transcriptase. That's why they're called retroviruses.

CJ: You mentioned the decades-old method of isolating retroviruses. How many decades are we talking about?

EPE: From the 1940s until the late 1970s. You see, retroviruses were among the first viruses discovered. Dr. Peyton Rous at the Rockefeller Center in New York originally encountered them when he was doing experiments on malignant muscle tumors in chickens [8]. Not that he could actually see them. That was back in 1911. It wasn't until the invention of the electron microscope and the high speed centrifuge that the method of isolating and identifying retroviral particles was worked out.

CJ: My understanding is that high-speed centrifugation is used to produce samples consisting exclusively of objects having the same density, a so-called "density-purified sample." Electron microscopy is used to see if these density-purified samples consist of objects which all have the same appearance — in which case the sample is an isolate — and if this appearance matches that of a retrovirus, in terms of size, shape, and so forth. If all this is true, then you are three steps into the procedure for obtaining a retroviral isolate. (1) You have an isolate, and the isolate consists of objects with the same (2) density and (3) appearance of a retrovirus. Then you have to examine this isolate further, to see if the objects in it contain reverse transcriptase and will replicate when placed in new cultures. Only then can you rightfully declare that you have obtained a retroviral isolate.

EPE: Exactly. It was discovered that retroviral particles have a physical property which enables them to be separated from other material in cell cultures. That property is their buoyancy, or density, and this was utilized to purify the particles by a process called density gradient centrifugation.

The technology is complicated, but the concept is extremely simple. You prepare a test tube containing a solution of sucrose, ordinary table sugar, made so the solution is light at the top but gradually becomes heavier, or more dense, towards the bottom. Meanwhile, you grow whatever cells you think may contain your retrovirus. If you're right, retroviral particles will be released from the cells and pass into the culture fluids. When you think everything is ready, you decant a specimen of culture fluids and gently place a drop on top of the sugar solution. Then you spin the test tube at extremely high speeds. This generates tremendous forces, and particles present in that drop of fluid are forced through the sugar solution until they reach a point where their buoyancy prevents them from penetrating any further. In other words, they drift down the density gradient until they reach a spot where their own density is the same as that region of the sugar solution. When they get there they stop, all together. To use virological jargon, that's where they band.

Retroviruses band at a characteristic point. In sucrose solutions they band at a point where the density is 1.16 gm/ml.

That band can then be selectively extracted and photographed with an electron microscope. The picture is called an electron micrograph, or EM. The electron microscope enables particles the size of retroviruses to be seen, and to be characterized by their appearance.

CJ: So, examination with the electron microscope tells you what fish you've caught?

EPE: Not only that. It's the only way to know if you've caught a fish. Or anything at all.

CJ: Did Montagnier and Gallo do this?

EPE: This is one of the many problems. Montagnier and Gallo did use density gradient banding, but for some unknown reason they did not publish any EMs of the material at 1.16 gm/ml which they and everyone afterwards call "pure HIV." This is quite puzzling because in 1973 the Pasteur Institute hosted a meeting attended by scientists, some of whom are now amongst the leading HIV experts. At that meeting the method of retroviral isolation was thoroughly discussed, and photographing the 1.16 band of the density gradient was considered absolutely essential.

CJ: But Montagnier and Gallo did publish photographs of virus particles.

EPE: No. Montagnier and Gallo published electron micrographs of culture fluids that had not been centrifuged, or even separated from the culture cells, for that matter. These EMs contained, in addition to many other things, including the culture cells and other things that clearly are not retroviruses, a few particles which Montagnier and Gallo claimed are retroviruses, and which all belonged to the same retroviral species, now called HIV. But photographs of unpurified particles don't prove that those particles are viruses. The existence of HIV was not established by Montagnier and Gallo — or anyone since — using the method presented at the 1973 meeting.

CJ: And what was that method?

EPE: All the steps I have just told you. The only scientific method that exists. Culture cells, find a particle, isolate the particle, take it to pieces, find out what's inside, and then prove those particles are able to make more of the same with the same constituents when they're added to a culture of uninfected cells.

CJ: So before AIDS came along there was a well-tried method for proving the existence of a retrovirus, but Montagnier and Gallo did not follow this method?

EPE: They used some of the techniques, but they did not undertake every step including proving what particles, if any, are in the 1.16 gm/ml band of the density gradient, the density that defines retroviral particles.

CJ: But what about their pictures?

EPE: Montagnier's and Gallo's electron micrographs — and every other electron microscope picture supposedly featuring images of HIV published up until March 1997 — are of entire cell cultures, or of unpurified fluids from cultures. Before March of 1997, no one had ever published a picture of a density gradient band in the name of HIV science.

CJ: Which is what we need to do to prove isolation of retroviral particles?

EPE: Yes.

CJ: Can the 1.16 band contain material other than retroviruses?

EPE: Yes. That's another reason why you need a photograph, to see everything that's going on. Although density-purified samples contain only particles that have the same density, many different particles share the same density, including the 1.16 gm/ml density that characterizes

retroviruses. All retroviruses have this density, but not everything that has this density is a retrovirus. It was known long before the AIDS era that retroviral-like particles aren't the only material that may find their way into this part of the density gradient. Tiny cellular pieces, some recognizable as internal structures of cells, or just cellular debris, can band at 1.16 gm/ml. And some of this material can enclose nucleic acids — DNA and RNA — and take on the appearances of retrovirus particles. Knowing all this, it's a complete mystery why any HIV researcher could have omitted the crucial step of taking an EM of a density gradient [5].

CJ: Could it be because electron microscopy is highly specialized and expensive?

EPE: For the past twenty years at least electron microscopy has been used daily in most hospitals to diagnose all kinds of diseases. Besides, there are plenty of EMs of HIV cultures. It's just that until 1997, for some unknown reason, there weren't any of density-purified specimens.

CJ: What do we see in those pictures of bands from a density gradient published in 1997?

EPE: These photographs vindicate the position we have held ever since the beginning. Two groups, one Franco/German [9] and one from the US National Cancer Institute [10], published pictures of density gradient bands. In the Franco/German study the pictures are from the 1.16 gm/ml band. It is impossible to tell from which density the pictures in the American study are taken, but let's assume it's the correct 1.16 density for retroviral particles. The first thing to say is that the authors of these studies concede that their pictures reveal that the vast majority of the material in their density gradient bands is cellular. The authors describe all this material as "non-viral", or as "mock" virus or "microvesicles," which are encapsulated cell fragments.

CJ: Are there any viral particles in these pictures?

EPE: There are a few particles which the researchers claim are retroviral particles. In fact, they claim these are the HIV particles, but give no evidence why.

CJ: Are there lots of these HIV particles?

EPE: No. For these particles to form an isolate, the band should contain billions of these particles, and when you take an electron micrograph they should fill the entire picture. Instead, these candidate retroviruses are minority constituents of the published electron micrographs. Thus, molecules extracted from these samples can not be assumed to come from those retroviral-like particles.

CJ: Do the experts comment on this?

EPE: They say the cellular material "co-purifies" with the HIV particles. These bands constitute density-purified samples, meaning that all of their constituents do have the same density. But they are not truly pure, in terms of their constituents all belonging to the same species, which would include having, in addition to the same density, the same appearance as well.

CJ: Do they look like retroviruses, those few particles they say are HIV?

EPE: They bear only the vaguest resemblance to retroviral particles. For sure they look more like retroviral particles than all the other particles and material, but even if they looked identical to retroviral particles, you cannot say they are retroviruses. Even Gallo admits to the existence of particles which band at 1.16 gm/ml and which have the appearances and biochemical properties of retroviruses but which are not retroviruses because they are incapable of replicating [11].

CJ: What's the difference between these particles and real retroviruses?

EPE: Gallo and all other retrovirologists — as well as Hans Gelderblom, who has done most of the electron microscopy studies of HIV — agree that retrovirus particles are almost spherical in shape, have a diameter of 100-120 nanometers (nm) and are covered with knobs [12,13]. The particles the two groups claim are HIV are not spherical, no diameter is less than 120nm, and in fact many of them have major diameters exceeding twice that permitted for a retrovirus. And none of them

appear to have knobs.

CJ: Surely size can't be that critical. Many things in biology have a range of sizes. What about humans? There are plenty of humans twice the size of other humans. They're all still humans.

EPE: What's true for humans is not true for retroviruses. For a start, retroviruses don't have to grow up. They're born adults. So the correct comparison is between adult humans. There aren't too many twelve foot humans. In fact, the tallest human ever recorded was eight feet eleven inches. If you measure the major and minor diameters of the particles in the EMs they claim are HIV and calculate the average diameters, then the Franco/German particles are 1.14 times larger than genuine retroviruses, and the US particles are 1.96 times larger.

Now, to translate this into volumes, we have to cube the diameters. If we take 120nm as the upper limit for the diameter of a retrovirus, the Franco/German particles have 50% more volume than a retrovirus, and the US particles have 750% more volume, with the US particles being five times more voluminous than the Franco/German. The point is that any genuine retrovirus contains a fixed amount of RNA and protein. No more and no less. More volume means more RNA and protein.

CJ: You mentioned the particles lacked knobs. How serious a deficiency is that?

EPE: All the AIDS experts agree that the knobs are absolutely essential for the HIV particle to lock on to a cell, as the first step in infecting that cell. So, no locking on, no infection. The experts all claim that the knobs contain a protein called gp120, which is the hook in the knobs that grabs hold of the surface of the cell it's about to infect [14]. Without knobs, HIV cannot enter a new cell, and thus it is not infectious.

CJ: That sounds like a serious flaw in the idea that these particles are transmitted from one person to another. How do the experts respond?

EPE: They avoid it. And the knobs problem is not something new. The German group drew attention to it in the late 1980s and again in 1992 [15,16]. As soon as an HIV particle is released from a cell, all the knobs disappear. This single fact has many ramifications. For example, three quarters of all hemophiliacs tested are HIV-antibody positive. The claim is that these hemophiliacs have HIV infections acquired from infusions of contaminated factor VIII, which they need to treat their clotting deficiency. The problem is that factor VIII is made from plasma, which is blood with all the cells removed. That means if there are any HIV particles present in factor VIII, they must be floating free in solution. But if cell-free HIV has no knobs, then those HIVs have no way of getting into fresh cells to infect them.

CJ: Then how do you explain HIV antibodies and AIDS in hemophiliacs?

EPE: My colleagues and I have published several papers discussing alternative explanations for AIDS in hemophiliacs, including one wholly devoted to the subject [17].

[Editor's note: Even if there is no HIV, the presumed HIV proteins are still present in the plasma from which HIV is claimed to be isolated. Thus, Factor VIII made from such plasma would contain those proteins, and it would therefore cause patients treated with it to produce antibodies against those proteins. So you don't need HIV to become "HIV-positive." As for "AIDS," Papadopoulos-Eleopoulos and her colleagues in their papers explain that clotting factor therapy itself is immune suppressive.]

CJ: I find it very hard to accept that hemophiliacs have not been infected through contaminated clotting concentrates. And I bet hemophiliacs do, too.

EPE: Let me explain another way. According to the CDC, if an HIV-infected person sheds blood due to a cut, that blood is only infectious for a few hours, because drying destroys HIV [18]. Have you ever seen a vial of factor VIII? It comes as a dry, flaky, yellowish powder. By the time it's used, it's at least a couple of months old. So how can factor VIII cause HIV infection?

CJ: How solid is the evidence prior to March 1997 that HIV exists?

EPE: Sticking to particles, all the evidence comes from electron micrographs of whole cell cultures. Not density gradients. From this evidence it can be said that cell cultures contain a large variety of particles, some of which are claimed to look like retroviral particles. That's all. None of the particle data has been taken further. No purification, no analysis, and no proof of replication.

Several research groups, including Hans Gelderblom and his associates from the Koch Institute in Berlin who specialize in this area, have examined these cultures and found not just one type of virus-like particle, but a stunning array of them [13, 19, 20].

This raises several questions. If one of these particle species really does represent a retrovirus experts call HIV, what are all the others? Which of these particles band at 1.16 gm/ml? If the HIV particles cause AIDS, why doesn't one or several of the other retroviral-looking species also cause AIDS? Why don't all the particles cause AIDS? Or, rather than causing AIDS, is it possible that AIDS causes these objects, including the ones called HIV, to appear? Is it possible that the culturing process causes these objects to appear?

And when it comes to HIV, the HIV experts can't even agree upon which of these various objects to call HIV. There are three subfamilies of retroviruses, and HIV has been classified by different research groups under two of these subfamilies, as well as three different species.

CJ: Where does this leave us?

EPE: We still don't know what any of the particles are. We don't have a definite particle proven to be a retrovirus from which to take proteins and RNA to use in tests for infection in people, or to do experiments to try and understand what is happening if there truly is a virus causing AIDS.

CJ: Let's suppose that we do have a picture of a band at the right density gradient, and it contains nothing but thousands of particles all the right size and shape, and with knobs. What should be done next to prove these objects are retroviruses?

EPE: The next steps are to disrupt the particles, find out what proteins and RNA are in them, prove one of the proteins is an enzyme, reverse transcriptase, which turns RNA into DNA, and that the RNA codes for all the proteins. And finally, take more of the density gradient and prove that when pure particles are put into a virgin cell culture, exactly the same particles made up of the same constituents come out.

CJ: And has this been done?

EPE: No. To understand what has been done, we really must examine Gallo's experiments from 1984.

CJ: Isn't 1984 a bit ancient?

EPE: No, because that's when the best research on HIV isolation was done. Those experiments are vitally important because everything believed and taught about HIV is founded on what happened back then.

CJ: Everything?

EPE: Yes, every single solitary thing. The criteria used to isolate HIV particles, and claim that it exists and causes AIDS, were established in Gallo's 1984 papers, and have been the only ones used since. The HIV proteins used in the antibody tests come out of Gallo's 1984 experiments. Ultimately, so did the RNA used especially to diagnose children as HIV-infected, and now used to measure the so-called viral load. Nobody since has done as thorough a job as did Gallo in 1984 of establishing HIV's existence, and its causal role in AIDS. But the question is, was Gallo's job good enough?

CJ: Why was Gallo interested in AIDS anyway?

EPE: By 1984 Gallo had already spent more than a decade researching retroviruses and cancer. He was one of the many virologists caught up in President Nixon's decade of war against cancer. In the mid-1970s Gallo claimed to have discovered the first human retrovirus, in patients with leukemia. He claimed his data proved the existence of a retrovirus which he called HL23V [11, 21].

Now, just like he would later do for HIV, Gallo "proved" which proteins in the cultures were viral not by abstracting them from a viral isolate, but by identifying which ones reacted with antibodies from the blood of patients. This required, among other things, the assumption that not just these antibodies, but all antibodies, be specific. That is, they react only with the proteins that triggered their production, and nothing else triggers their production. But there's no such thing as specific antibodies, and, not surprisingly, the same antibodies a few years later were shown to occur naturally and be directed against many proteins that had nothing to do with retroviruses [22, 23].

That was the end of HL23V. Gallo's claim for its existence was recognized as a big mistake. There was no HL23V retrovirus. The Gallo data turned out to be an embarrassment, and HL23V is now extinct. Not even Gallo talks about HL23V anymore.

What's interesting for us is that the evidence used to claim proof of the existence of HL23V is the same kind of evidence said to prove the existence of HIV. In fact, the evidence for HL23V is better than the evidence for HIV.

CJ: Better in what way?

EPE: Well, for HIV, Gallo found reverse transcriptase only in stimulated cultures. But for HL23V, he observed reverse transcription in fresh tissue, tissue that has not been added to cultures. And he published an EM of density gradient material present at 1.16 gm/ml.

CJ: Does whatever HIV is kill T4 cells?

EPE: That's another problem for the HIV theory of AIDS. HIV cultures consist of lymphocytes — including T4 cells — or plasma (cell-free blood) from patients, added to stock cultures of leukemia cells. Leukemia cells are lymphocytes, including T4 cells, which are cancerous, and do well in culture because they tend to stay alive, and not die naturally like non-cancerous lymphocytes, including healthy lymphocytes, when placed in culture.

One of the requirements for showing that a virus causes a disease is to observe how it affects culture cells. Even though HIV is said to make people immune-deficient by killing T4 cells, the HUT78 leukemic cell line — as well as its H9 clone which Gallo eventually produced from it — are both immortal, even when infected with HIV. That means rather than being killed by HIV, the cells permit what is regarded as HIV to grow indefinitely.

Also, observations of the phenomena which are said to prove the existence of HIV only occur when these cultures are stimulated with special chemicals. So there's no evidence that HIV phenomena occur in vivo. That could only be demonstrated by observations of HIV in fresh — uncultured — plasma, which has never been reported.

CJ: What did Gallo actually do to prove he had isolated a new retrovirus from AIDS patients?

EPE: If you read the first paper, what was called isolation consisted of (1) observation of reverse transcription in those stimulated cultures, and (2) electron microscopic photographs of a few particles in those stimulated cultures, but not in gradient bands or fresh plasma; and (3) observation that blood from a hemophilia patient — as well as from rabbits that had injected into them the material from those cultures which banded at 1.16 — contained antibodies that reacted with some of the proteins from those bands.

CJ: That was reported as isolation of a virus?

EPE: Yes, but this is not isolation. And, since this is the best evidence for HIV's existence, scientists must question the existence of HIV for exactly the same reason scientists at the Sloan Kettering and National Cancer Institute questioned the existence of HL23V.

CJ: Tell me about the reverse transcriptase observations. Why don't they prove the presence of a retrovirus?

EPE: Reverse transcriptase was discovered in retroviruses, but observation of it doesn't mean you've got a retrovirus, much less one particular retrovirus, because reverse transcriptase is not the only enzyme that can reverse transcribe, and reverse transcriptase is not unique to retroviruses.

The existence of RT is proven indirectly. By putting some RNA into a culture and seeing if DNA bearing the corresponding sequence appears.

CJ: You mean the presence of RT is implied by the ability of the culture to do this particular trick?

EPE: Yes. It's measured by demonstrating the process of reverse transcription. Like many enzyme tests, the test for reverse transcriptase measures what the enzyme does, not the actual enzyme itself. So in the case of RT it measures the production of DNA copied from a synthetic piece of RNA introduced into the cultures. The problem is that RT is not the only thing capable of doing this trick, as you call it. Normal cellular enzymes can also do this trick. In fact, they do it very well with the same synthetic RNA that all HIV researchers introduce into their cultures to copy into DNA in order to claim that their cultures contain HIV RT, and thus HIV [24].

What's more, when you read the AIDS literature, it becomes apparent that some authors who claim to have isolated HIV have done no more than detect reverse transcription.

But even if you actually have RT molecules, and not just reverse transcription, that does not mean presence of a retrovirus. For instance, according to Harold Varmus, Nobel Laureate and head of the National Institutes of Health, RTs themselves are also present in normal cells. And it's known that some of the chemical stimulants that are required to observe all HIV phenomena in cultures will cause normal lymphocytes to reverse transcribe.

Bacteria also have RTs, and so do some viruses not classified as retroviruses. Strange as it may seem, hepatitis B virus (HBV) has a reverse transcriptase enzyme. Most AIDS patients are infected with HBV, which doesn't just infect liver cells. It also infects T-lymphocytes.

There's yet another explanation for why these stimulated cultures should contain RT even if there is no HIV. A year earlier Gallo published a paper in Nature reporting that he had found in the HUT78 cell line another retrovirus, HTLV-I [25]. And remember, the other cell line used for HIV cultures, H9, is derived from HUT78.

CJ: So the evidence using RT does not look good?

EPE: The problem with RT is the same problem with all the evidence. It's just like the particles Gallo photographed. They might be the particles of a retrovirus. The reverse transcription might be caused by the RT of a retrovirus. But "might" is not scientific proof. You don't construct scientific theories from what "might" be going on.

CJ: How can you dismiss particles? They're very convincing. How can you escape the fact that no matter how widely Gallo and everybody else deviated from the traditional method of isolating a retrovirus, there are particles in these cultures, and a lot of very important people regard them as particles of a retrovirus.

EPE: Particles have to be viewed with a considerable amount of perspective. Retroviral-like particles are practically ubiquitous. In the 1970s such particles were frequently observed in human leukemia tissues, in cultures of embryonic tissues, and in the majority of animal and human placentas. This is of significance given that the H9 cell line is made up of leukemic cells and also because Montagnier obtained his EMs of "HIV" from cultures done with umbilical cord blood lymphocytes.

As already noted, some of these objects that look just like retroviruses are not viruses of any sort. Or they may be endogenous retroviruses. Only isolation can sort all this out.

CJ: What's an endogenous retrovirus?

EPE: Normal human DNA contains retroviral information which did not get there following a retroviral infection. The cell was born with it. So amongst all our DNA there are stretches made up of some retroviral information, and that may sit there all your life until something happens. The DNA starts to direct the production of endogenous retroviral particles. They're called endogenous because they're not something that got in from the outside, like HIV is supposed to. Something that gets in from the outside is called exogenous.

Long before the AIDS era everyone knew that in animal cells endogenous retrovirus production could occur spontaneously. Up until 1993, neither Gallo nor Anthony Fauci, [26] accepted that humans contain the DNA to make endogenous retroviruses. But now it's accepted that endogenous retroviral DNA forms about 1% of human DNA. For the record, that's about 3,000 times larger than what the experts claim is the size of the HIV genome. And what's more, new retroviral genomes can arise by rearrangements and recombination of existing retroviral genomes.

CJ: Can you tell endogenous and exogenous apart?

EPE: No. Endogenously produced retroviruses are morphologically and biochemically indistinguishable from exogenous retroviruses.

CJ: If HIV is an endogenous virus, why would AIDS patients produce such viruses and other people not?

EPE: Because the patients are sick. In fact, they are sick before they ever develop AIDS. So their cells are sick, and their sick cells find themselves in the right condition in cultures to be activated. That's what's needed to produce endogenous retroviruses, and that's been known for decades.

Also, there's an electron microscope study reported in 1988 by O'Hara and colleagues from Harvard [27]. They examined enlarged lymph nodes from both AIDS and non-AIDS patients and found "HIV" particles in 90% of both groups. They had to concede that particles alone do not prove infection with HIV.

CJ: What about the antibodies that reacted with the cells in the cultures? Surely that must signify something that ordinarily isn't present? Wouldn't this fit with a retroviral infectious agent?

EPE: It might fit, but there's that word again. It's simply not possible to prove proteins belong to a retrovirus or antibodies are caused by a retrovirus, or to claim proof of the isolation of a retrovirus just because some things react together in a test-tube. Again, let's not take the data any further than good science allows.

The experiments reported in the first Gallo paper tell us that Gallo had some material that banded in density gradients at the correct 1.16 gm/ml location. That material came from H9 cells cultured with lymphocytes from AIDS patients [1]. We now know from the Franco/German and US National Cancer Institute pictures of March, 1997, that these banded specimens contained all sorts of things, including a few objects that look like retroviruses, and which are said to be HIV. Gallo took the single 1.16 gm/ml density bands with their various proteins (including those attached to retrovirus-looking objects) and separated them based on molecular weight into various new bands containing single protein species, creating what is called a Western blot.

Since the density bands consisted not of isolates, but rather of many different things, Gallo had to determine which of the resulting protein bands belonged to the objects that looked like retroviruses. To do this he used the Western blots to see which proteins reacted to antibodies present in the blood taken from two sources.

One source was a hemophiliac patient with the initials "E.T." ET had a condition known as pre-AIDS. That's enlargement of lymph nodes in many parts of the body. ET was assumed to be infected by HIV since he was showing symptoms attributed to HIV. But pre-AIDS is not AIDS, and is caused by many infectious agents which are present, for example, in gay men, intravenous drug users, and hemophiliacs even when there is none of what is called HIV present.

The other source was a group of rabbits which had been repeatedly injected with what Gallo called HIV, which was, at best, the heterogeneous material from the density bands. [Editor's note: Presumably, blood taken from the rabbits before the injections did not react with the Western blot proteins, so the post-injection antibodies that did react were considered to identify the HIV proteins.] But since the density band material contained many proteins besides those associated with the objects called HIV, these injections would have produced antibodies to many non-HIV proteins. Only injections with pure HIV [28] could have exclusively produced antibodies against HIV proteins.

There are many problems here. One is that antibodies can identify proteins only if all antibodies are specific, meaning, they only react with the proteins that stimulated them in the first place. For example, if ET had an antibody species elicited in response to some non-HIV protein species, and if that antibody "cross-reacts" with one of those Western blot proteins, then that protein would be mis-identified as an HIV protein.

The only way to avoid the antibody specificity requirement is to obtain an isolate of objects that look like retroviruses and which meet the other requirements of a retrovirus. Then you know whatever proteins emerge in a Western blot belong to those objects.

Without that, you are left depending upon the hypothetical concept of specific antibodies. But there is no such thing as specific antibodies. Antibodies against one protein can and do react with other proteins [29, 30]. And AIDS patients have many different antibodies that react with many different proteins, and thus many chances for cross-reactions. Many investigators have found that antibodies that react with the so-called HIV proteins also react with many other proteins as well. Since the existence of HIV is premised upon specific antibody reactions, rather than on viral isolation, then the non-specificity of so-called HIV antibodies is a critical flaw in the view that HIV exists.

But even if there was such a thing as specific antibodies, and all antibodies were specific, that would not rescue Gallo's analysis, or eliminate the need for isolation. Since the density bands used to construct Gallo's Western blots are heterogeneous, even if HIV-specific antibodies were added, there would be no way to know which of the reacting proteins belong to the objects that look like retroviruses, and which represent the other material. Furthermore, since this material was part of what Gallo injected into the rabbits, the rabbits would have antibodies against proteins in it, as well as proteins associated with the retroviral-looking objects. So there would be no way to tell which antibodies were the ones specific for those objects, the supposed HIV. The same is true for AIDS patients, and even ET, since they have antibodies against many biological entities.

The technique of viral isolation provides a method by which scientists can start with blood and derive from it definite viral proteins, and then identify the species of antibodies that react with each of those proteins. However, there is no reverse method for deriving first the antibodies and then identifying viral proteins. Even if antibody specificity were a reality, there is no theoretical starting point by which antibodies for an unknown virus can be obtained, and used to identify the proteins of that virus.

What Gallo and everyone since has done, essentially, is take blood from patients, culture it with stimulated leukemia cells, obtain from the culture fluids density gradient bands that we now know are highly heterogeneous, separate the proteins therein into a Western blot, then add some of the patient's uncultured blood to see which proteins react with the antibodies in the blood, and then declare those proteins to belong to a unique retrovirus, HIV.

Now, as my colleagues and I see it, using antibodies to prove the existence of a retrovirus is the crux of the problem. This is a very important part of our argument, so I hope to get this very important message across.

CJ: What about proof that HIV causes AIDS? Did Gallo prove that in 1984?

EPE: To be fair, in his 1984 Science papers Gallo did not make such a direct claim. He said HIV was the probable cause of AIDS. But even this conclusion is questionable. Even if Gallo's evidence

was incontrovertible proof he had isolated a retrovirus, he only managed to isolate it from 26 out of 72 AIDS patients. That's just 36%. And only 88% of 49 AIDS patients had antibodies. And that was mostly using ELISA, the antibody test considered the least specific. No one diagnoses HIV infection on a single ELISA. And if the virus was present in only 36% of patients why did 88% have antibodies? I mean, there were more patients with antibodies without virus than there were patients with virus. And there was not even a hint of proof that HIV was killing T4 cells or that having low T4 cells could cause all the diseases diagnosed as AIDS.

Two years later, when Gallo was defending the accusation he had used the French virus to discover his version of HIV, he was much more definite about his 1984 papers. He said they provided "clear cut" evidence that HIV is the cause of AIDS. And his opinion was no different in 1993. Let me read you Gallo's own words from the 1993 TV documentary, *The Plague* : "The compelling evidence that convinced the scientific community that this kind of virus is the cause of AIDS came from us. The proper growth of the virus came from this laboratory principally through Mika Popovic. The development of a sensitive, workable blood test. I don't think that we have to debate. I think the history speaks for itself."

CJ: Is it fair to say that the HIV-antibody tests are useless?

EPE: No, they're not useless. There is no doubt being in a risk group and having these antibodies is not a good thing.

CJ: How can that be?

EPE: Because empirically such people are more likely to develop the illnesses we classify as AIDS. In fact, there is evidence published in *The Lancet* that a positive test also predicts increased mortality from diseases which are not classified as AIDS [31]. But what the tests don't do, or at least there is no proof that they do, is prove HIV infection. Or even less that HIV infection is the reason people develop AIDS. You may not appreciate that the only evidence that HIV causes AIDS is these tests. If the tests are unproven for HIV infection then there is no proof that HIV causes AIDS [3-5, 26, 32-34].

CJ: What about a positive test in people who are apparently healthy and not in any risk group? Should they be worried?

EPE: There is no data to answer that question, and I think it would be impossible to ever obtain that data. There would have to be an experiment comparing matched groups of healthy people with and without these antibodies. In other words, follow people with a positive test over a period of years and see who developed AIDS and who did not.

The trouble is it would be very difficult for most people knowing they are HIV positive, as well as their physicians, not to believe that sooner or later they're going to get very sick and eventually die of AIDS. And that mindset may greatly affect the results of such an experiment. From both sides.

CJ: What do you mean from both sides?

EPE: I mean that patients' health will be affected knowing they are HIV positive, and their physicians will feel compelled to offer treatments with drugs given in the belief they are necessary to kill a virus the patients do not even have.

CJ: The drugs themselves might be harmful?

EPE: Well, AZT, the original and still most widely used anti-HIV drug, is certainly well known for its toxic effects, and in fact some of these effects mimic AIDS.

CJ: What if we did this experiment, and we did it blind, and found that the HIV positives were more likely to develop AIDS than the HIV negatives? What would that tell us?

EPE: On our present data that would mean the same it means in the AIDS risk groups. Gallo and his colleagues serendipitously discovered a test which for some reason predicts a tendency to get

sick from certain diseases, most of which are lumped together as AIDS.

But it doesn't prove that the link to all these diseases is a retrovirus. That can never be proven unless HIV is proven to exist. Even then, you can't say HIV causes AIDS just because it's present in AIDS patients. Association doesn't prove causation. You can be present at a bank robbery but not be the robber. You need other data to prove causation. In fact, according to the CDC AIDS definition, you don't even need to be HIV-infected to be diagnosed as having AIDS. Under some circumstances, the CDC AIDS definition requires a patient to be diagnosed as a case of AIDS even if the patient's antibody tests are negative [35].

CJ: What about the RNA tests. The PCR viral load and the like?

EPE: All these tests rely on matching a piece of RNA or DNA from the patient's blood to a test piece of RNA or DNA deemed to originate from a particle called HIV. But if a retroviral particle hasn't been isolated and purified and shown to be a virus, how does anyone know where this piece of RNA comes from? The HIV experts themselves say that there are about one hundred million distinct HIV RNAs in every AIDS patient [36]. With that much variation one would think that a virus is the most improbable source for such RNA. I mean, how can a virus have that much variation and still be the same agent? Still make the same proteins and induce the antibodies? Still perform all the same tricks?

CJ: If there is no virus, where do all the things Montagnier and Gallo found come from? I assume you do believe they did find something in their cultures?

EPE: Of course they found something. They found many things. All the things we've discussed. In our view, it is possible the RT and retrovirus-like particles could be produced when cells from sick people are stimulated in cultures. We know that both normal and pathological processes can be associated with the appearance of retroviral-like particles. There's absolutely no doubt about that. What exactly are all these particles? Well, some may be no more than pieces of disintegrating cells. Others certainly look more uniform and might conceivably be considered viral-like, or even retroviral-like. But in the context of HIV what really matters is proof that at least one of these varieties of particles is actually a retrovirus, and an exogenous retrovirus at that.

Either the agents to which the patients are exposed induce the right conditions or the culture conditions play a part. Perhaps a major part. I don't know which contribution is the greater, but that might have been sorted out a long time ago if the first HIV researchers had included a few control experiments.

CJ: What do you mean by control experiments?

EPE: When you do a culture of, say, lymphocytes from an AIDS patient with some H9 cells and all the chemicals which are added to make the culture produce "HIV," you really don't know if what you find is the difference that sets AIDS patients apart from everyone else. What if you were to find exactly the same thing in similar patients that don't have AIDS? So, to convince yourself that what you find and call HIV is present only in AIDS patients and therefore might have something to do with AIDS, you must use controls.

Controls are experiments run in parallel with your main experiment, conducted exactly the same way using exactly the same materials. The only difference is the one variable you're chasing.

A control in the case of HIV isolation would be a culture of cells from some patients of the same age and sex and environmental exposures who are sick with diseases like AIDS but not AIDS. Even better: if the cells came from patients who have low T4 cells and who are oxidized [3, 32]. AIDS patients have both these abnormalities — low T4 cell counts and oxidation — but they're not the only patients to have them.

And one must also not forget to add the same chemicals to all cultures. We already know that one of these chemicals causes reverse transcription in normal lymphocytes. Now, if you did all that you might well find that lymphocytes from men in New York who were sick with non-AIDS diseases

also develop particles and RT and antibody reactions when cultured. That would mean that one would have to be very cautious interpreting that data as being something special to AIDS. Remember, O'Hara found "HIV" particles in both AIDS and non-AIDS patients who had enlarged lymph nodes [27].

But hardly anyone uses controls in AIDS research, and when they do, they're often the wrong type.

CJ: Could what is called HIV be an effect of AIDS rather than a potential cause of AIDS?

EPE: Yes. Having AIDS may just be a prescription for developing those abnormalities. Retrovirologists themselves have argued that retroviruses may arise as the result of a disease and not vice versa. Getting cause and effect the wrong way around is not new to medicine. One example is the 1926 Nobel Prize, which went to Av Hill and Otto Myerhoff for proving that lactic acid production causes muscle contraction. But we all know now that it's the other way around: muscle contraction causes lactic acid production.

CJ: How long have you and your colleagues held the view that HIV may not exist?

EPE: Ever since my first publication on HIV, in 1983. I published my first paper on AIDS in 1988, in *Medical Hypotheses*, [3] a well known journal of ideas. There I put forward a non-viral theory of AIDS, and included some of what we've talked about today. The discussion on HIV isolation is not as frank as we've had today, but back then it was virtually impossible to question the existence of HIV. It was important to be subtle in order to get into print.

Even so, it took a few years for that paper to be published. Initially I submitted it to a much more prominent journal but it was rejected. Twice in fact.

CJ: Which journal was that?

EPE: That's not important. Then, in 1988, Val Turner and I wrote a paper which directly spelt out all the problems we've discussed today. We aimed that paper at clinicians and offered it to a journal read by practicing doctors in Australia. But we had no luck getting it published. We are the only people to ever publish data in scientific journals questioning the existence of HIV and arguing that the HIV antibody tests are not proof of HIV infection.

CJ: Tell me about your non-viral model of AIDS.

EPE: We were among the first people in the world to put forward the idea that non-infectious factors explain AIDS in gay men, and the first to propose a non-infectious theory for all risk groups as well as a unifying mechanism. What's more, our theory predicts that the factors which cause the development of the AIDS diseases are also responsible for the phenomena which everyone else infers as the "isolation" of a retrovirus from AIDS patients.

CJ: What has the reaction been to your theory?

EPE: Unfortunately, there has been very little reaction. But some research groups have confirmed some of our predictions, including our prediction that antioxidants may be useful for treating individuals who are at risk for developing AIDS. We haven't had much luck in the scientific press, but some gay men and gay men's organizations have become our greatest allies. If it wasn't for them, I think our task would be almost impossible.

CJ: Why, despite everything you have explained today, do virtually all the world's scientists and physicians appear extremely comfortable with the very evidence you find so hard to accept?

EPE: The problem is not a matter of accepting evidence. It's how evidence is interpreted. Most of the scientists and doctors who believe in HIV and that HIV causes AIDS do so because they accept the interpretation of a relative minority of experts. It's totally unrealistic to expect all the people who work in AIDS to analyze the data to the degree we have. As far as the HIV experts themselves are concerned, I don't know why they interpret the evidence as they do. I can only speculate. Perhaps it's because we are all subjective and look at problems from our own perspective. They're

retrovirologists, so they see retroviruses in the data.

CJ: Doesn't the same apply to your group's interpretation of the literature?

EPE: Certainly it does. But don't lose sight of one very important aspect of all this that is not subjective: The definition of a virus and the method that follows for proving the existence of a virus. The same method that was endorsed by the Pasteur Institute in 1973. Nobody can deny that here is a method which constitutes absolute proof for the existence of a retrovirus. And another thing nobody can deny is that HIV has never been accorded reality according to that method.

CJ: Do you think now that we've seen what's actually in a density gradient, the tide will turn against HIV?

EPE: I would expect that data to be a turning point, especially as more people get to see or know about it. And it confirms what our group has been saying for a very long time. In the introduction to the Franco/German paper, the authors clearly affirm that before their pictures, the 1.16 gm/ml density gradient was "considered to contain a population of relatively pure viral particles." That's our point. HIV has never been isolated and yet for the past fourteen years scientists and biomedical companies have been using this material to obtain proteins and RNA as if it is pure HIV. Pictures are powerful, and that cuts both ways.

CJ: What do you think should happen now to AIDS research?

EPE: I think that the traditional method of virus isolation should be applied as urgently as possible using cultures with cells from AIDS patients as well as suitable controls.

As I said, we must find out once and for all if there is such a thing called HIV. It's taken fourteen years to get a mere handful of electron microscope pictures of a density gradient. And even if these had shown nothing but the right-looking kind of particle, we're still missing all the other steps which are needed to arrive at a retrovirus.

CJ: Which steps are the most important?

EPE: All the steps are important. Establishing the presence of retroviral-like particles in cultures, purification and analysis of those particles, proof the particles can replicate, and proof that the antibodies in patients' blood which react with the proteins taken from the particles are specific.

CJ: And if this is not the case?

EPE: If these phenomena are also seen in control cultures, or if the particles which band at 1.16 gm/ml are of the wrong morphology or are not infectious, or if the antibodies present in AIDS patients are not specific to those articles, then AIDS patients cannot be said to be infected with a unique virus HIV.

CJ: Which means HIV could end up similar to HL23V?

EPE: That is quite possible. The proteins said to belong to HL23V were defined in the same manner as the HIV proteins. By antibody reactions. So, when the antibodies were shown to be non-specific, HL23V disappeared. My group thinks that scientists will eventually accept that the same is true of HIV antibodies. You see, AIDS patients are inundated with antibodies to so many different things that a few of these could easily react with two or three of the ten proteins present in the "HIV" test. That's all that's required to be HIV-positive. In fact, there's now ample evidence that antibodies produced as a result of infection with the two germs that infect ninety percent of AIDS patients react with all the HIV proteins. I mean the germs known as mycobacteria and yeasts, which between them cause two of the most common AIDS defining diseases. We have a recent paper on this, in the British journal *Current Medical Research and Opinion* [37]. If that's the case, how can anyone say these antibodies prove infection with HIV, or that these diseases are caused by HIV?

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