IS HIV THE CAUSE OF AIDS?

An interview with Eleni Papadopulos-Eleopulos

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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: Eleni, many thanks for agreeing to this interview.

EPE: My pleasure.

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 Montagnier and 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)

CJ: Perhaps you should explain what leads you to this rather radical view.

EPE: I think the easiest way to begin is to ask the question, "What is a virus?". The answer is quite simple. A virus is microscopic particle that reproduces itself inside a cell...

CJ: Don't bacteria do that?

EPE: They may but there's a very important difference. Bacteria are not obliged to replicate inside a cell. Viruses must. You see, what bacteria take from the cell, or from an inanimate source of food and energy, is all turned into the next generation of bacteria inside the bacterial cell itself. That's also how our own cells replicate. But viruses can't do that. The virus particle is really no more than a few proteins strung around a piece of RNA or DNA but without the machinery needed to replicate.

CJ: So whereas a cell is a factory, a virus is a blueprint that must hijack a factory?

EPE: I can't better that analogy.

CJ: How does a virus replicate?

EPE: It has to get inside the cell. To do this the protective envelope of the viral particle fuses with the cell membrane and then the particle passes inside. Once inside, using the cellular metabolic machinery, the virus particle is disassembled. Then, using the same machinery, separate pieces of new virus are synthesised. Finally, all the viral components are put together and out come the new virus particles.

CJ: Out of where?

EPE: The virus either destroys the cell or in the case of retroviruses the virus particles have a more orderly exit by budding out of the cell membrane. But that's not what happens with HIV. Unlike retroviruses, HIV is said to destroy the cells.

CJ: Well, what about HIV particles? Are you suggesting they're not a virus?

EPE: 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 analyse 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. That's the whole point of putting the virus question. Not all particles that look like viruses are viruses. You have to prove that whatever particle you nominate can actually make copies of itself. No replication, no virus. I'm sorry but this is an extremely important point. No one, especially virologists, can afford to ignore it.

CJ: That seems to make sense. I guess it would be hard to get sick catching a particle that could not make more of itself.

EPE: Exactly.

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.

CJ: You better explain retroviruses before you go on.

EPE: I should. As you probably know, HIV is claimed to be a retrovirus. Retroviruses are incredibly tiny, almost spherical particles that...

CJ: How tiny are they?

EPE: One hundred nanometres in diameter.

CJ: How tiny is that?

EPE: One ten thousandth of a millimetre. Millions would fit comfortably on the head of a pin.

CJ: How do you actually 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 they have an outer envelope covered with knobs and an inner core consisting of some proteins and RNA.

CJ: So, if it exists, HIV is an RNA virus?

EPE: Yes. 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 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".

CJ: Understood.

EPE: There's one more thing. One of the proteins inside a retrovirus particle is an enzyme which catalyses this process. Not surprisingly, it's called reverse transcriptase.

CJ: And that's it?

EPE: Well, 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 tumours 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 things began to be sorted out.

CJ: What was actually sorted out?

EPE: It was these that led to the method of identifying and purifying retroviral particles.

CJ: That's the same as isolating them?

EPE: Yes. To purify particles of any kind a scientist has to develop a method of separating out the particles he wishes to study from everything else.

CJ: How did electron microscopes and high speed centrifuges make purification of retroviruses possible?

EPE: The electron microscope enabled particles this small to be seen. The other part was played by the high speed centrifuge and was extremely important. 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 and this was utilised to purify the particles by a process called density gradient centrifugation.

CJ: Sounds complicated.

EPE: The technology is complicated but the concept is extremely simple. You prepare a test tube containing a solution of sucrose, ordinary table sugar. But it's 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 and 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 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, or to use virological jargon, that's where they band. That band can then be selectively extracted and photographed with an electron microscope.

CJ: And do retroviral particles band at a characteristic point?

EPE: Yes. In the sucrose solutions they band at a point where the density is 1.16 gm/ml.

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: True. Did Montagnier and Gallo not 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 a few particles which they claimed are a retrovirus and are HIV. But photographs don't prove particles are a virus and the existence of HIV was not proven 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 published up until March this year are of unpurified cell cultures. Not the gradient. Before March this year, no one had ever published a picture of a density gradient.

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 retroviral particles?

EPE: Yes. That's another reason why you need a photograph. To see everything that's going on. 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 recognisable 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 and take on the appearances of retrovirus particles.

CJ: What are nucleic acids?

EPE: DNA and RNA.

CJ: Surely though, if retroviral particles are released from cells without disrupting the cells, it must be possible to guard against cellular contamination?

EPE: Well it is and it isn't. Certainly the animal retrovirologists were well aware of this problem and strongly advised handling the cultures gently and regularly topping them up with nutrients to keep the cells alive. So they don't disintegrate. But in the case of HIV there are additional problems. We are told that HIV is cytopathic meaning it kills cells. So one could hardly claim that putative virus

particles are the only things likely to be floating around in culture fluids or at 1.16 gm/ml. The other confounding fact is that in many HIV experiments the cells are deliberately broken up by the experimenter as part of the experiment. 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 specialised and expensive?

EPE: It may have been in the early days but not anymore. 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 this year, for some unknown reason, there haven't been any of the density gradient.

CJ: All right. Let's talk about the pictures of the density gradient published this year. What do we see there?

EPE: Two groups, one Franco/German (9) and one from the US National Cancer Institute (10), published pictures of density gradients. 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 the vast majority of the material in the density gradient is cellular. The authors describe all this material as "non-viral", or as "mock" virus or "microvesicles".

CJ: What are microvesicles?

EPE: 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. The band should contain billions and when you take an electron micrograph they should fill the entire picture.

CJ: So the banded material contains only a few HIV particles and from the HIV particles' point of view is rather impure?

EPE: Yes.

CJ: Do the experts comment on this?

EPE: They say the cellular material "co-purifies" with the HIV particles.

CJ: Tell me, the few particles they say are HIV, do they look like a retrovirus?

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 a retrovirus. 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: All right, but that aside, what's the difference between these particles and a real retroviral particle?

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 nanometres and are covered with knobs.(12,13) The particles the two groups claim are HIV are not spherical, no diameter is less than 120nM, 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's 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. They're aren't too many twelve foot humans. In fact, the tallest human ever recorded was eight feet eleven inches. But there's more than size involved here.

CJ: What else?

EPE: If we assume both the Franco/German and US groups sought particles at the correct retroviral density then the particles found by both groups must have the same density, 1.16 gm/ml. If you measure the major and minor diameters of the particles in the EMs they claim are HIV and take the average diameters and for argument's sake, assume they're all spherical, then the Franco/German particles are 1.14 times larger than genuine retroviral particles and the US particles are 1.96 times larger. Now, to translate this into volumes, we have to cube the ratios of the diameters. So, if we take 120nM as the upper limit for the diameter of a retroviral particle and do the sums, the Franco/German particles have 50% more volume than a retroviral particle and the US particles have 750% more volume. And the US particles are five times more voluminous than the Franco/German.

CJ: Which tells us what?

EPE: It tells us that the Franco/German and US particles must contain 50% or 750% more mass than genuine retroviral particles.

CJ: Why is that?

EPE: Because density is the ratio of mass to volume. If the volume goes up by a certain amount, to keep the same density, the mass has to go up by the same amount.

CJ: OK but what's your point?

EPE: The point is that any genuine retroviral particle contains a fixed amount of RNA and protein. No more and no less. If that's the case then these particles are made up of much more material than a genuine retrovirus. Which means that if these different sized particles are truly HIV then HIV cannot be a retrovirus. The only other explanation is that the electron micrographs are not from the 1.16 gm/ml band. If that's the case then we have no choice but to redefine retroviruses and more importantly, not to consider the 1.16 band as HIV. But if we do that then all the research done on HIV

using this band cannot be used because this is what everyone uses as purified HIV. That would mean for example that this band cannot be used to obtain proteins and RNA for use as diagnostic agents to prove HIV infection.

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) If HIV particles do not have knobs how is HIV able to replicate?

CJ: You mean it can't get hold of the cell to get inside?

EPE: Precisely. And if it can't replicate, HIV is not an infectious particle.

CJ: That sounds like a serious problem to me. 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 so many ramifications. For example, three quarters of all haemophiliacs tested are HIV antibody positive. And the claim is that haemophiliacs acquired these as a result of becoming HIV infected from infusions of contaminated factor VIII which they need to treat their clotting deficiency. The problem is that factor VIII is made from plasma. That's blood with all the cells removed which 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 those HIVs have no way of getting into fresh cells to infect them.

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

EPE: My colleagues and I have published several papers discussing alternative explanations including a detailed analysis of haemophilia in an invited paper in the 1995 special issue of Genetica (17) devoted to the HIV/AIDS controversy.

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

EPE: Unfortunately that is true but perhaps I can persuade you with one quick and simple explanation. Tell me this. If someone HIV positive is cut and bleeds how long does the blood remain infectious? Outside the body?

CJ: According to what I've read, for only a few hours at the most.

EPE: And why is that?

CJ: Because HIV dries out and dies. Certainly that's what the CDC says.(18)

EPE: OK. Let me ask you this. How is factor VIII made?

CJ: From donated blood.

EPE: Right. Have you ever seen a vial of factor VIII?

CJ: No.

EPE: All right I'll tell you. It comes as a dry, flaky, yellowish powder and by the time it's used it's at least a couple of months old. Do you see the problem?

CJ: I do. If it's dry and that old any HIV in it should be long dead.

EPE: Exactly. So how does factor VIII cause HIV infection and AIDS in haemophiliacs?

CJ: I don't know but I think I'm beginning to see why your group is not the toast of the town. Perhaps we'd better not get diverted into a discussion about haemophila. Why do you think until now most HIV experts have been happy enough to regard the material at the 1.16 density as pure HIV?

EPE: I think it's premature to assume these pictures have changed anyone's minds about the 1.16 gm/ml portion of the density gradient being anything but pure HIV.

CJ: Well how does your group respond to these pictures?

EPE: On the evidence provided by these pictures there is no reason to claim that this material is pure or that it contains retroviral-like particles let alone a retrovirus or more importantly, a specific retrovirus, HIV. And this vindicates the position we have held ever since the beginning. And a position we long ago put into print That there is no evidence proving the isolation of a retrovirus from AIDS patients or those at risk of AIDS.

CJ: OK. Let's set aside the March pictures and talk about what we could deduce from what was known beforehand. How solid is the evidence prior to March 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. In these cultures several research groups including Hans Gelderblom and his associates from the Koch Institute in Berlin who specialise in this area have reported not just one type of particle but a stunning array of particles.(13,19,20) This raises several questions. If one of these particles really is a retrovirus experts call HIV, what are all the others? If the HIV particles originate from the tissues of AIDS patients, where do all the others come from? 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 particles also cause AIDS? Why don't all the particles cause AIDS? Or why doesn't AIDS or the cultures cause the appearance of the particles? And when it comes to HIV, the HIV experts can't even agree what is the HIV particle. 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: All right. Let's suppose that we do have a picture of a density gradient and it contains nothing but thousands of particles all the right size and shape, and with knobs, to be called a retroviral particle. Let's go over what should be done next.

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 which turns RNA into DNA 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, but perhaps I can explain things more clearly by talking about what has been done. Some of 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. Whether an HIV particle has been isolated and therefore any claim that it exists. The HIV proteins used in the antibody tests. The RNA used especially to diagnose children infected with HIV and now used to measure the so called viral load. And more. But the question is are they good enough?

CJ: Good enough?

EPE: Good enough to claim the existence of a unique retrovirus called HIV and that it causes AIDS.

CJ: OK. Tell us about Gallo's experiments. Why was he 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 leukaemia. 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 used antibody reactions to "prove" which proteins in the cultures were viral proteins. And not long afterwards others claimed to have found the same antibodies in many people who did not have leukaemia. However, a few years after that these same antibodies were shown to occur naturally and be directed against many substances that had nothing to do with retroviruses.(22,23) Then it was realised that HL23V was a big mistake. There was no HL23V retrovirus. So the Gallo data turned out to be an embarrassment and HL23V is now extinct. What's interesting for us though 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 was better than HIV.

CJ: Better in what way?

EPE: Well, unlike HIV, Gallo found reverse transcriptase in fresh tissue. Without having to do cultures. And he published an EM of density gradient material present at 1.16 gm/ml.

CJ: But it still turned out to be a false alarm?

EPE: Not even Gallo talks about HL23V anymore. But in 1980 he said he'd discovered another retrovirus. It was yet more of the same kind of data from leukaemia patients and this time he called it HTLV-I and claimed it caused a particular rare form of leukaemia which Gallo now calls adult T4 cell leukaemia, ATL. In fact, there are some very interesting parallels and paradoxes between HIV and HTLV-I.

CJ: What are they?

EPE: They're said to infect the same cells and to be spread the same way. Yet unlike HIV, HTLV-I has not gone beyond where it was discovered. The greatest prevalence of HTLV-I was reported from Africa and Southern Japan and that's where it's remained. That's longer than we've had AIDS and don't forget that although this virus is said to cause leukaemia, less than 1% of persons who test positive ever develop leukaemia. Even after forty years. But I digress. What I was about to say was that many of the first AIDS patients had a cancer known as Kaposi's sarcoma, as well as low numbers of the same T4 cells which are present in excessive amounts in ATL. This was known because the technology to count the different classes of lymphocytes came along about the same time that AIDS appeared.

CJ: HIV was hypothesised to be killing the T4 cells?

EPE: Well, this was too early for HIV but it was hypothesised that something was killing them. Later Gallo actually went through a stage of thinking that HTLV-I might be the culprit but that theory was a problem because HTLV-I allegedly causes leukaemia which is far too many T4 cells. Also, despite the high prevalence of antibodies to HTLV-I in Southern Japan, there were no AIDS cases. However, because gay men with AIDS had such a high incidence of the cancer Kaposi's sarcoma, and because something seemed to be affecting their T4 lymphocytes, Gallo persisted in trying to find a retrovirus to explain it all.

CJ: What happened next?

EPE: Gallo and his colleagues did a lot of experiments which culminated in four consecutive papers published in Science in May 1984. That was a year after the French published their paper also in Science. Gallo's group began by culturing lymphocytes from AIDS patients but apparently, none of the cultures produced enough reverse transcriptase to convince Gallo that a retrovirus was present. At that time Gallo had a Czech researcher called Mikulas Popovic working for him and so Popovic and Gallo agreed to mix up culture fluids from ten AIDS patients and add that to a culture of leukaemia cells. The leukaemia cells they used in this culture had been obtained years earlier from a patient with ATL. When they did this enough reverse transcriptase was produced to convince Gallo and Popovic they now did have a retrovirus.

CJ: You mean a retrovirus would not grow in individual cultures from AIDS patients but did when the specimens were mixed up and cultured?

EPE: Yes.

CJ: Isn't that a little puzzling? How can a germ do that? Surely if it's present in one of the specimens, as long as the cultures are done the same way, it should grow no matter what?

EPE: You would think so.

CJ: And if you mix up all the specimens, how would you know who had the virus in the first place? It might have come from just one patient. Was Gallo ever questioned about this?

EPE: He was and in a 1993 television documentary said he didn't care whether the virus came from a single patient or whether it came from a pool of patients.

CJ: Did you not say that the leukaemic cells used in the cultures were originally obtained from a patient with adult T4 cell leukaemia?

EPE: Yes.

CJ: Then surely the cultures must have contained many T4 cells?

EPE: That's true.

CJ: If those cultures were made up from T4 cells and if HIV kills these cells, how could a cell killing virus be expected to grow?

EPE: That's another of the problems with the HIV theory of AIDS. Even though HIV is said to kill T4 cells and make people immune deficient, that's what the "AID" in AIDS actually refers to, the leukaemic cell line as well as its H9 clone which Popovic eventually produced, 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. The H9 clone is widely used in both research and commercially for producing what are regarded as the HIV proteins for use in the antibody tests kits.

CJ: OK. 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 electron microscopic photographs of a few particles in the cultures, not the gradient, finding reverse transcriptase and observing that some antibodies present in a haemophilia patient as well as rabbits reacted with some of the proteins in the cells of the cultures.

CJ: That was reported as isolation of a virus?

EPE: Yes.

CJ: Is that really isolation?

EPE: No. Isolation means separation from everything else. Not just detection of some phenomena. The only way to prove the existence of an infectious agent is to isolate it. That's what this debate is all about.

CJ: Yes, but isolated or not, how do you respond to Gallo's claim that his cultures grew a retrovirus?

EPE: Let me repeat, there is no question of isolation. Gallo did not isolate a virus. There were no electron microscope pictures of a banded specimen that one would expect to show nothing but

retroviral particles. How could there be? There were no EMs at all of a banded specimen. Just pictures of cells with a dozen or so particles lying nearby but no extraction and analysis and proof that these particles could replicate into identical particles. But what we must ask is whether Gallo had the proof to say he had even detected a retrovirus. In our view he did not. And it's vitally important at this point to state that finding particles and reverse transcriptase is not proof that a retrovirus is present.

CJ: You said retrovirus particles contain reverse transcriptase.

EPE: They do, in fact reverse transcriptase was discovered in retroviruses but there's a catch. The catch is two things. The way the presence of RT is proven and the fact that RT is not unique to retroviruses.

CJ: RT?

EPE: Reverse transcriptase. 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. Other enzymes, 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 (24) and to claim their cultures contain HIV RT and thus HIV. And what's more, when you read the AIDS literature, it becomes apparent that some researchers who publish claims to have isolated HIV have done no more than detect RT.

CJ: That's quite disconcerting.

EPE: There's much more to RT. 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 bacteria have RTs. And it's known that some of the chemicals that are an obligatory component of these cultures cause normal lymphocytes to reverse transcribe. And leukaemic cells can also do the same trick unaided when not cultured with such chemicals or cells from AIDS patients.

CJ: That's many possible reasons for RT then?

EPE: Yes and there's yet another. Remember that Gallo and Popovic used H9 cells to demonstrate the existence of what they claimed was a new retrovirus. But as I said before, if you trace the lineage of the H9 cell line it comes from the HUT78 cell line, a cell line which began life in a patient whom Gallo says had a form of malignancy caused by HTLV-I. If that malignancy is caused by HTLV-I then HTLV-I and its RT will be in the very cells Gallo used to prove the presence of HIV.

CJ: But surely no one would search for a new retrovirus using cells that already contained another retrovirus?

EPE: You would think not especially since a year earlier Gallo published a paper in Nature reporting HTLV-I genetic sequences in the cell line from which the H9 cells ultimately originated.(25)

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: But even so Eleni, how can you dismiss particles? They're so 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: I appreciate your point but I think 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 leukaemia 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 leukaemic cells and also because Montagnier obtained his EMs from cultures done with umbilical cord blood lymphocytes. There's also a large group of retroviral particles classified as type-C particles that are found in fish, snakes, worms, pheasant, quail, partridge, turkey, tree mice, agouti, tapeworms, insects as well as mammals. And amongst its many official guises HIV has been described as a type-C particle, by both Montagnier and Gallo.(26) 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: All right. Let's leave particles. 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.

CJ: Could you explain that a little more please?

EPE: Again, let's not take the data any further than good science allows. The experiments reported in the first Gallo paper tell us that some antibodies present in a patient with haemophilia, as well as in rabbits, reacted with some proteins in H9 cells cultured with lymphocytes from AIDS patients.(1)

CJ: That's the data?

EPE: That's the data we have to work on. What's important is how we interpret the data. Now, for what he called isolation of HIV Gallo regarded the antibodies as the crucial evidence. How do we know this? For two reasons. First, what we have already said. Gallo knew there are particles which look exactly like retroviruses, which band at 1.16 gm/ml and which contain RT but which do not replicate. So, whatever they are, no matter how they arise, they can't be viruses. Second, we know

because in one of Gallo's papers he actually talks about the need to have specific agents to identify a particle as a virus. And by that he means specific antibodies or proteins. The Gallo hypothesis is that there is a virus causing AIDS, it's foreign so when it infects a patient the patient develops antibodies to the virus.

CJ: So it works backwards as well as forwards? Virus produces antibodies and antibodies can be used to point to the virus?

EPE: No. That's the problem. Antibodies do not work backwards. We'll get to why in a minute. The important thing here is not to forget what question we're trying to answer. We're trying to define which proteins are unique constituents of a retroviral particle. For me, there's only one way to do that. And it's easy. We define viral proteins exactly the same way we define our arms and legs. Or our kidneys.

CJ: Meaning what?

EPE: My bits and pieces of anatomy are mine because they're part of me. Either inside or outside. If one of my kidneys is diseased and has to be removed the first thing the surgeon must do before I'm put on the operating table is to check and make sure it's me. It's no different with viruses. Viral proteins are the proteins that come out of particles proven to be a virus. It's that simple. If you want to define the proteins of a retroviral particle first you must prove you HAVE a retroviral particle.

CJ: Antibodies are too imprecise?

EPE: Antibodies are imprecise but that's not the issue here. Antibodies are irrelevant. You prove proteins come from a virus particle by isolating the particle and then doing a dissection. You don't prove proteins are constituents of a viral particle by performing chemical reactions on what is essentially a culture soup. It has nothing to do with it. So what if some proteins and antibodies react? There's many reasons why these reactions might take place.

CJ: Such as?

EPE: There are many antibodies and antibodies to one thing can and do react with other things.(28,29) Immunologists call these cross-reactions. This is a fact of Nature and it causes problems because an antibody reacting with a protein in a culture could just as well be an antibody made to something totally unrelated. Quite possibly something not even in the culture. To put it into plain language, antibodies adopt other partners. My colleague Val Turner adopted the term "promiscuous" to explain this behaviour. The only way to prove a reaction you see is caused by the one antibody reacting with the one protein is to see how the reactions compare with what you think they signify. What we have to do is correlate the reactions against HIV itself. Antibodies are specific to HIV if and only if they are present only when HIV is present.

CJ: Not if HIV is absent?

EPE: One hundred percent specific means no antibodies reacting when HIV is absent. 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: I'm all ears.

EPE: Think about what's happened so far. There's an old, logical, reliable, commonsense method of proving the existence of a retrovirus. It's based on nothing more than the definition of a retrovirus as a particle having a particular size, shape, appearance and constituents and the ability to replicate. But for some unknown reason this method has been abandoned in the HIV era. Don't ask me why but it has. In its place we have a disparate collection of data including particles not photographed in density gradients and some evidence for reverse transcription either in the culture or the material which bands at 1.16 gm/ml. Neither of these are proof that a retrovirus exists in the cultures. Gallo says so himself.

CJ: I'm following. Go on.

EPE: Then along comes the idea with antibodies. If there really is a virus then being foreign, it should induce antibodies in people it infects. Perhaps these antibodies are indeed specific meaning they are made solely in response to HIV and react with viral proteins and nothing else. OK. Let's assume this unlikely specificity is a fact and let's make an even less probable assumption.

CJ: Yes?

EPE: Let's say what's considered true of the so called HIV antibodies is true for all antibodies. Every single antibody ever made only reacts with what stimulated its production and with nothing else. Antibodies to the tuberculosis germ only react with the tuberculosis germ. Antibodies to hepatitis virus only react with hepatitis virus et cetera. OK. We have some cultures of tissues derived from AIDS patients which react with antibodies present in the serums of AIDS patients. What next? We know that AIDS patients are infected with many different agents. So if these agents, or bits of them, are present in AIDS patients, they're also likely to be in their cell cultures. Isn't this why laboratory workers are believed to be at risk from handling these specimens? And we also know that despite being labeled immune deficient, everyone agrees that AIDS patients have myriads of antibodies to all manner of things. Including antibodies to human T-cells, the cells that make up the cultures. If you add some antibodies from the same kind of patients to these cultures, even if each antibody only reacts with its mate, wouldn't you expect to see lots of reactions between lots of different things?

CJ: I see your point. Since all you see is reactions you can't tell what is reacting with what.

EPE: Exactly. Antibodies react and things light up but who's got a finger on the switch? And for this argument we've agreed that every antibody is directed against one agent and only reacts with that agent. What if we bring back real life where antibodies cross-react as well?

CJ: I guess it's a big mess. It's difficult to tell where any proteins or antibodies come from.

EPE: That's absolutely correct. And one must not confuse origins with composition. For sure you can't prove the origin of a protein by an antibody reaction. Why should a reaction tell you that a protein comes from a particle any more than it comes from Mars? But you can't prove identity either. That's because antibodies do not work backwards.

CJ: Are there any germs in AIDS patients that could actually react like you've said?

EPE: Yes. A good example is hepatitis B virus. Many, and in the case of haemophiliacs, virtually all AIDS patients are infected with hepatitis B virus. And HBV doesn't just infect liver cells. It also infects

T-lymphocytes. And strange as it may seem, hepatitis B virus has a reverse transcriptase enzyme. And people make antibodies to this virus...

CJ: OK. I get the drift.

EPE: But there's more to Gallo's experiments. For a start, the serum that Gallo used in this experiment came from a patient with the initials "E.T.". But ET didn't actually have AIDS. He had a condition known as pre-AIDS. That's enlargement of lymph nodes in many parts of the body. But pre-AIDS is caused by many infectious agents which are present for example in gay men, intravenous drug users and haemophiliacs even when there is none of what is called HIV present.

CJ: So ET might not have had HIV antibodies?

EPE: Exactly and the other puzzle is the rabbits.

CJ: Yes. I was going to ask about that.

EPE: Gallo claims he had a serum from rabbits that contained antibodies specific to HIV. Just imagine for a moment the scene in Gallo's laboratory. They've cultured H9 cells with lymphocytes from AIDS patients and when they come to determine which proteins in their cultures originate from a presumed virus they reach up on the shelf and, lo and behold, they pull down a bottle labeled "specific antibodies to HIV". How did they manage to get those antibodies? This was the first paper they wrote but they already had a bottle containing rabbit antibodies specific to a virus they were currently attempting to isolate for the very first time.

CJ: Well how did they do it?

EPE: They say they prepared rabbit antibodies by repeatedly infecting rabbits with HIV. But if they were preparing antibodies to HIV they would have had to inject rabbits with pure HIV (30) which again means they must have already isolated what they were now attempting to do for the first time. It doesn't make sense.

CJ: Well, if they didn't inject pure HIV into the rabbits what did they inject?

EPE: At the very best, if they used a banded specimen which they and everyone else regard as pure HIV, the evidence is that what they injected would have been something akin to what we see in the Franco/German and US National Cancer Institute pictures. Now any immunology book will tell you that proteins are the most potent antibody producing substances available. Even more so if they're introduced directly into the blood stream. So, by injecting their culture material into rabbits, even if they had used a banded specimen, Gallo and Popovic would have exposed their rabbits to a multitude of cellular proteins. The rabbits would have then produced antibodies to all those proteins and when they added these antibodies back with the material they injected of course there would be reactions. That's exactly what you would expect but that doesn't make the material you inject into a virus. And even less into a unique retrovirus.

CJ: OK. I understand what you're saying. Your argument is that, before he had a virus, there was no way Gallo could have known there were antibodies in patient ET or in AIDS patients or rabbits that would specifically recognise HIV proteins.

EPE: Yes. Before he had a virus there was no way of knowing that antibodies to HIV existed at all. Anywhere. To even begin to talk about specific antibodies to specific HIV proteins first you have to prove the proteins are constituents of a retroviral-like particle that is able to replicate. And the only way to do that is to isolate the particles and do everything else I've described. You need the virus BEFORE you go looking for proteins and antibodies.

CJ: Well what on Earth are these antibodies in AIDS patients which everyone calls HIV antibodies?

EPE: What my colleagues and I have been arguing all these years is that there is no evidence they are HIV antibodies. The only way to find out if they're HIV antibodies is to do the experiment comparing antibodies with virus isolation. That is what's meant by having a gold standard. Using virus isolation as a totally independent means of determining whether there truly are specific HIV antibodies. You can think of HIV as being the adjudicator. If antibodies specific to a retrovirus called HIV exist they will reveal themselves by reacting only when a retrovirus called HIV is present. Nothing could be simpler. Now, although you may not realise, there's another problem. There might be specific HIV antibodies but what if there's non-specific HIV antibodies as well?

CJ: I can see people getting confused. Could you please elaborate?

EPE: All right. The problem using antibodies is that there could be two types of antibodies. One type is specific meaning antibodies caused by HIV and nothing else and reacting with HIV and nothing else. The other type is non-specific meaning they're antibodies caused by other agents or stimuli and sure they react with those agents but they also react with HIV. If you add a person's serum to some of the "HIV" proteins in a culture or in a test kit and see a reaction how can you tell which type of antibodies are doing the reacting? In fact there are three possibilities. All the antibodies might be the specific type or none of them might be. Or there might be a mixture. All you see is a reaction. Something changes colour. That's all. So how do you tell? Simple. You test for antibodies in all sorts of patients, some with AIDS, some who are sick but who don't have AIDS and in some healthy people as well. But in the same experiments, at the same time, you use HIV as the adjudicator. To judge what type of antibodies they are. And if antibodies show up when there's no HIV then non-specific antibodies must exist.

CJ: What about the experiment to sort out the antibodies?

EPE: The experiment, which should have been done long before HIV antibody testing was ever introduced into clinical medicine, has never been done. And in fact it could not have been done because to date nobody has isolated HIV. But there's plenty of evidence that people who all the experts accept are NOT infected with HIV do have antibodies which react with what are claimed to be the HIV proteins. So there are non-specific "HIV" antibodies and if some are non-specific how do you know how many? Why not all of them? Even if it's only some how can you tell them apart? The answer is you can't and that means that not one single person can be diagnosed using an antibody test. It also means that 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: So your argument essentially boils down to "HIV" antibodies not arising because of or being directed against HIV in spite of the fact that everyone calls them "HIV" antibodies?

EPE: That's right.

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 only 36 percent. 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.

CJ: The evidence in 1984 was light on?

EPE: There was no evidence. But 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 "clearcut" 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: Do the problems you see with the Gallo papers also apply to the tests used to diagnose patients infected with HIV when cultures are not done?

EPE: You mean the antibody tests?

CJ: Yes.

EPE: It's the same test. Can you see what's happened here? The HIV researchers have used some antibodies in the patients' blood to convince themselves that some proteins in their cultures are unique constituents of a particle which they say is a retrovirus and call HIV. That's the first thing. But having done that they've then turned around and said, "OK, if these proteins are from HIV then the antibodies must be THE HIV antibodies". So they've used the one and same chemical reaction to prove which each reactant is when in fact there's no way an antibody reaction can tell you even what one reactant is even if you know the other to start with. That's why you need a independent gold standard adjudicator. As far as actually doing the test is concerned, the difference from cultures is that the patient's blood is mixed with proteins extracted from H9 or other cell cultures and put either all together in a test tube or separately at discrete spots along a thin paper strip. The first is called the ELISA and the second the Western blot. If these proteins react with the blood, and in the Western blot the number and type of reacting proteins required to produce a positive test vary all over the world and that's yet another huge problem, then the patient is reported HIV positive.

CJ: So the HIV antibody test is really the same procedure that was used to prove the existence of HIV in cultures from AIDS patients in 1984?

EPE: Yes. And also by the French in 1983. And by Gallo and his colleagues to prove the existence of HL23V in the mid seventies. Our group find it intriguing that any scientist could regard antibodies reacting with proteins as proof of viral isolation. Is an antibody joined to a protein a virus? What would you expect to see under the electron microscope? A particle with a core and knobs?

CJ: Then 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.(31) 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. 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 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 effect 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 have.

CJ: The drugs themselves might be harmful?

EPE: Well AZT, the original and still most widely used 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 that 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 by isolating it first and then used to validate the antibodies as HIV antibodies. 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 AIDS.

CJ: That sounds really crazy.

EPE: It's written down in the literature. 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 and viral load and like?

EPE: That's another huge subject but I can say just one thing. All these tests rely on matching a piece of the patient's RNA or DNA to a test piece of RNA or DNA deemed to originate from a particle called HIV. You can think about this like the rabbit antibodies. There's another bottle on the shelf and the label on this one reads "HIV RNA". 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: Tell me Eleni, 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. And your question is fair. In our view it is possible the RT and particles could be some reaction produced when cells from sick people are cultured. Or the result of the chemicals introduced into the 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 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 a retroviral particle. Even if we had that proof, the RT and the particles and proteins could all come from an endogenous retrovirus.

CJ: What's an endogenous retrovirus?

EPE: Unlike the case for all other infectious agents, 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 maybe all your life until something happens. The DNA starts to make RNA and hence proteins, and this may go even further and lead to the assembly 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. You make a cell culture and do nothing else. Just leave it on the bench for a few days or maybe a few weeks and then one day it starts to produce retroviral-like particles. They seemingly come out of nowhere and the process can be significantly accelerated and the yield of particles increased, sometimes millions of times, by conditions which induce cellular activation, the same conditions which are obligatory to obtain what is called HIV from cell cultures. Interestingly, up until 1993, neither Gallo nor Fauci who is another well-known HIV researcher, (37) 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 that 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: So HIV could be an endogenous retrovirus?

EPE: There are many explanations for the laboratory phenomena held up as proof for the existence of HIV. We went into all these in a very long article we wrote for Continuum magazine last October.(38)

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 when we don't?

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 virus and that's been known for decades. 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 are they?

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. They're 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.

CJ: Could you explain that further?

EPE: A control 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 oxidised.(3,32) AIDS patients have both these abnormalities 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.

CJ: There weren't any controls?

EPE: This is yet another problem with so much AIDS research. Hardly any one uses controls and when they do they're often the wrong type.

CJ: Is it possible we've got AIDS back to front? You hinted at this before. Could the patients or the cultures be responsible for what is called HIV and not the other way around?

EPE: Right. 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. The Nobel Prize has even been awarded under such circumstances.

CJ: It's almost time to finish up. I have three more questions. First, how long have you and your colleagues held the view that HIV may not exist?

EPE: Ever since the first publication on HIV. In 1983.

CJ: So it's not something you recently came to?

EPE: No.

CJ: Have you published these particular arguments? I mean in a scientific journal?

EPE: Yes. In my first paper on AIDS in 1988. There I put forward a non-viral theory of AIDS and I also included some of what we've talked about today.

CJ: Where was that published?

EPE: In Medical Hypotheses.(3)

CJ: Not a well known journal?

EPE: It is a well known journal of ideas. There 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 practising doctors in Australia.

CJ: No luck?

EPE: No luck.

CJ: So only the people who read Medical Hypotheses would have known what you thought ten years ago?

EPE: Yes.

CJ: You mentioned your non-viral theory of AIDS. Tell me a little about that.

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: How much reaction has there been to your theory?

EPE: Unfortunately very little 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.

CJ: Have you managed to overcome the inertia to your ideas?

EPE: We haven't had much luck in the scientific press but some gay men and gay mens' organisations have become our greatest allies. If it wasn't for them I think our task would be almost impossible.

CJ: If you had to nominate a single obstacle hindering the resolution of the scientific problems with AIDS what would that be?

EPE: In our view the greatest single obstacle to understanding and solving AIDS is HIV.

CJ: That would explain why your group has written so many papers against HIV?

EPE: That's quite right. In fact we've written a lot more papers than we've had published. Unfortunately, we've only managed to get about a dozen or so papers into print in the scientific journals. One of the most important was a paper published in Bio/Technology (5) which is now called Nature/Biotechnology There we said straight out there is no proof of HIV isolation. That paper was certainly noticed but again, no one responded to our views.

CJ: So you remained a minority?

EPE: We aren't just a minority. We are still 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: Eleni, 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. The way I see it is this. 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 analyse 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 pictures are so powerful. There are pictures containing particles which look like a virus and there's reverse transcriptase in the same cultures as the particles. It is possible mentally to connect particles, reverse transcription and proteins and the antibodies that react with the proteins and make this into evidence for the existence of a retrovirus. Especially for a retrovirologist. I suppose that is the whole problem. We must not forget we are all subjective and we look at problems from our own perspective.

CJ: Well 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.

CJ: What is that?

EPE: 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 what nobody can also deny is that HIV has never been accorded reality according to this method. In other words, in spite of AIDS being regarded as one of the gravest conditions ever to afflict the human race, no one has deemed it necessary to use a proven method to establish the existence of the putative cause of this dread disease. Instead everybody's opted for a set of non-specific criteria and appear to imagine that if you put all these together they must somehow metamorphose into the right answer.

CJ: Doesn't that have some merit? If they're all clues to a retrovirus surely the more you have the closer you get?

EPE: Certainly not. What if the true cause is something unexpected? Or something of which you have no knowledge or cannot even possibly imagine? In that case the more clues you have to what you are expecting, or what you want it to be, the more likely you will be misled. It all boils down to whether you would rather deal in probabilities rather than facts. That's what I mean about being subjective. It's like a physician seeing a patient with fever, diarrhoea, vomiting, weakness and shock and then declaring the cause is cholera. Sure it might be cholera but what about the dozens of other germs that cause a similar pattern? What if your life depended on it?

CJ: I see your point. Do you think now 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 the 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 having 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: 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 particles, 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. In the case of HL23V it was relatively easy because the antibodies occured in so many people who were never going to get leukaemia they were bound to be something unrelated and that's what was eventually proven at Sloan Kettering and the National Cancer Institute. 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 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 that between them cause two of the commonest AIDS defining diseases. We have a paper on this in press in the British journal Current Medical Research and Opinion.(39) If that's the case how can anyone say these antibodies prove infection with HIV or that these diseases are caused by HIV?

CJ: Eleni Papadopulos-Eleopulos, many thanks you for your time today.

EPE: My pleasure. *

This interview, examining the very roots of the controversial HIV/AIDS paradigm, was reviewed by scholar and international gay media personality **Prof. Camille Paglia**, in her column in the US Salon magazine October 28th: "For a superb critique of the scandalously overpoliticized scientific research on AIDS, see Christine Johnson's long interview with Australian biophysicist Eleni Papadopulos-Eleopulos in the new issue of the British AIDS magazine Continuum. The American major media have effectively suppressed long-standing questions about whether the AIDS test is reliable or whether an HIV virus in fact exists at all."

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