To control AIDS, we must 'get more momentum into the research'

We are honored to present to our readership on five continents—including thousands of scientists, government and military officials, policymakers, and the general public—the following exclusive interview with Prof. Jean-Claude Chermann, laboratory chief and head of a research team at the Pasteur Institute of Paris. The interview was conducted in French by our correspondent Garance Upham Phau in Paris in early October. Professor Chermann is part of the Pasteur Institute's department of Oncogene Virology, under Prof. Luc Montagnier, which first discovered the AIDS virus in 1983, then called LAV and today rechristened Human Immunodeficiency Virus (HIV).

Professor Chermann rocked the scientific and medical community in August, at the Fourteenth International Congress on Cancer held in Budapest, Hungary, when he announced that his team had found that cells of various insects (mosquitoes, black beetles, lion-ants, tse-tse flies, as well as ticks), all coming from Africa, were infected with the HIV virus in their genome.

Dr. Chermann explains precisely what has been shown by his experiments and what science has yet to discover. Despite the extraordinary scientific progress made to date in our understanding of this deadly pandemic, there yet remain vast amounts of scientific work to be done. We therefore fully endorse Professor Chermann's call to "develop more research teams." *We need more momentum in research.*

We hope that by publishing Dr. Chermann's interview in such an extensive and unedited form, including all the nuances and subtleties of formulation in distinguishing between what science has already fully established and what remain only hypotheses and areas of speculation, we shall help to encourage more young scientists and established researchers to put on their laboratory coats, and get on with productive experiments in this area to advance our scientific frontiers.

We hope, as well, that by printing this interview in a journal with wide global circulation, we shall help foster the climate in which populations generally give their governments a full mandate to embark upon Apollo Moon-shot-scale crash biomedical research programs. There is much we still don't know about the nature of this disease, yet in the spirit of Louis Pasteur himself, we shall always remain optimistic so long as science is fully backed to find those solutions which mankind so desperately requires.

Warren J. Hamerman, Director
EIR Biological Holocaust Task Force

EIR: What led you to study the role of insects in the context of AIDS?

Chermann: I would put the question differently, because it didn't happen in that way. We were interested in finding out why a sub-group of T4 lymphocytes was infected. So we immediately investigated the possibilities of there being a receptor other than the T4 molecule on the outside of the lymphocyte, to identify these cells.

We developed a technique of fluorescent marking of the virus to locate the sensitive cells. When we saw that the techniques we had developed were specific, we asked ourselves what other kind of cells could fix the virus. We looked at many different types of human cells, but as we were working at the same time here on insect cells in culture, we also looked at them and found that certain fly and mosquito cells fixed the virus in culture. That's how we came to insects.

In the second phase, we said, okay, cells in culture can fix the virus, but is it the same in nature? So we looked around the Paris area and in Africa, and in Africa we found that some insects were infected, that is, they had integrated the virus genotype into their own genotype, that is to say they incorporated viral DNA into their DNA. We did not prove that there were free viruses, but that the genotype was present.

EIR: How do you evaluate the importance of the discoveries made by the Johannesburg team, which were published in the *Lancet* and quoted in your article on the report of the Paris Academy of Sciences?

Chermann: When you find infected insects, there are two possibilities. The first is that the insect got contaminated from man, as the MacLeod group showed: Bedbugs sucking in blood could keep the virus in their blood for one hour. The other possibility is that these insects were infected by another animal. At this point, I absolutely cannot say whether man infected the insect or if it was infected some other way. But we will find out. We are studying that now.

EIR: What is your hypothesis on the way insects captured in Africa, in which you detected a viral DNA homologeous to the HIV virus, were contaminated?
Chermann: We are first going to investigate whether there are contaminated insects only in infected zones or whether there are also contaminated insects in non-infected zones. We also have to find out whether male mosquitoes are infected, since only females bite: If both males and females are infected, then it is a case of transovarian contamination.

EIR: Are you working on that now?
Chermann: Yes, we are, but we have as yet no answer. For the moment, I believe that in our areas, in Europe and America, there are no examples of transmission other than by blood or sex.

EIR: What do you think of the possibilities of HIV being replicated in insect tissues?
Chermann: I don't know. All we did in our cell cultures was to show very, very clearly that first of all the virus fixes on the surface of the cell and secondly enters into the cell and integrates into the genotype. We didn't find any replication, that is, no RNA, no viral protein formed and no virus on the outside. Secondly, there was a blocking of replication of this virus, and this may perhaps help to slow down the cell adhesion and to eliminate the cell in culture. So in fact the insect cell seems to protect itself against the invasion of an extra germ. That is very clear. We have the insect being infected without replication, which is true for the 6 to 10 days during which the virus is traced, but after that we don't find it anymore. So there is a negative selection. That is what we have shown in culture.

EIR: Could there possibly exist an animal reservoir (mammals, insects, or others) for the HIV virus, other than monkeys? Rats, for example . . . ?
Chermann: No. We do not even know whether the monkey is a reservoir, whether the monkey virus can be transmitted to man or not . . . at least not for the moment. As for the origin of the virus, there are two possibilities: Either it is a human virus which came from some tribe somewhere and was not dangerous then, but became so in spreading out; or else it came from an animal. But nobody knows. If it originated in insects, French insects would also be infected.

In my opinion, it is a secondary contamination in Africa. It is not something that has always existed in the philogeny of insects. It is more recent. Ants or cockroaches from Africa are contaminated, but French ones are not, so it does not happen during philogeny, it is a secondary contamination.

EIR: Dr. Whiteside from the Tropical Medicine Institute in Miami has considered the possible role of viruses other than HIV in contracting AIDS. What is your opinion?
Chermann: We think there are a number of co-factors in AIDS, which would cause an asymptomatic carrier to get AIDS. Of course. You have cytomegaloviruses which cause immuno-deficiencies; you have the Hepatitis B virus which has been shown to be present in lymphocytes and which can modify the function of lymphocytes or create immuno-deficiencies; you have the Epstein Barr virus which can also cause all kinds of transient immuno-deficiencies and, combined with the HIV virus, accelerate the catching of AIDS. That is definitely true. But I think these co-factors, as we have studied them, only come into play after HIV.

I am not really familiar with the Belle Glade case, I have not discussed it with Dr. Whiteside, but I think that what may be happening is that another insect-transmitted virus can create a favorable terrain for the spreading of HIV. But I don't know the situation there very well.

EIR: The possibility of recombinant viruses among insects in tropical region has also been discussed. What do you think of this?
Chermann: There is no evidence. We here are working on virus receptors. What I am really interested in is finding a cure for AIDS as quickly as possible. For me, insects are an instrument for purifying the receptor. I think what we have shown may be only an epiphenomenon, that insects are in fact infected but do not transmit.

You know, we went to Africa. [My colleague] Françoise Barre-Sinoussi went to Africa, and me too. And I can assure you that we are sero-negative. You see what I mean? There is a threshold effect, a kind of critical mass effect, of the virus. A certain quantity of the virus is needed to get infected.

EIR: In your document to the Paris Academy of Sciences, you say there is a possibility of insect transmission. . . .
Chermann: There are not enough viruses. . . . If viruses were as infectious, for example, as Hepatitis B, all the nurses who accidentally stabbed themselves with the needle would be positive, but there are only a few of them. Secondly, if the virus were as infectious as is said, saliva would also be contagious. But saliva is not contagious, except in one reported case, but this is one out of many. You know, if it were a virus like the hemorraghic fever virus, everyone would be dead by now. I think this virus is not very contagious. Its incubation time is of course very impressive because people remain infected for so long, but I think it's very little contagious, if it stays the way it is. We have been working with quantities of this virus since 1983, and in those three years, none of us in the laboratory has been infected.

EIR: Is it possible that new mutants of the virus appear, having different epidemiological or clinical characteristics?
Chermann: For the moment, I don't know. For the moment, we don't know if these are mutants which can come up in different clinical conditions; so far, we do not completely know what AIDS is. It's a new disease and every year, we

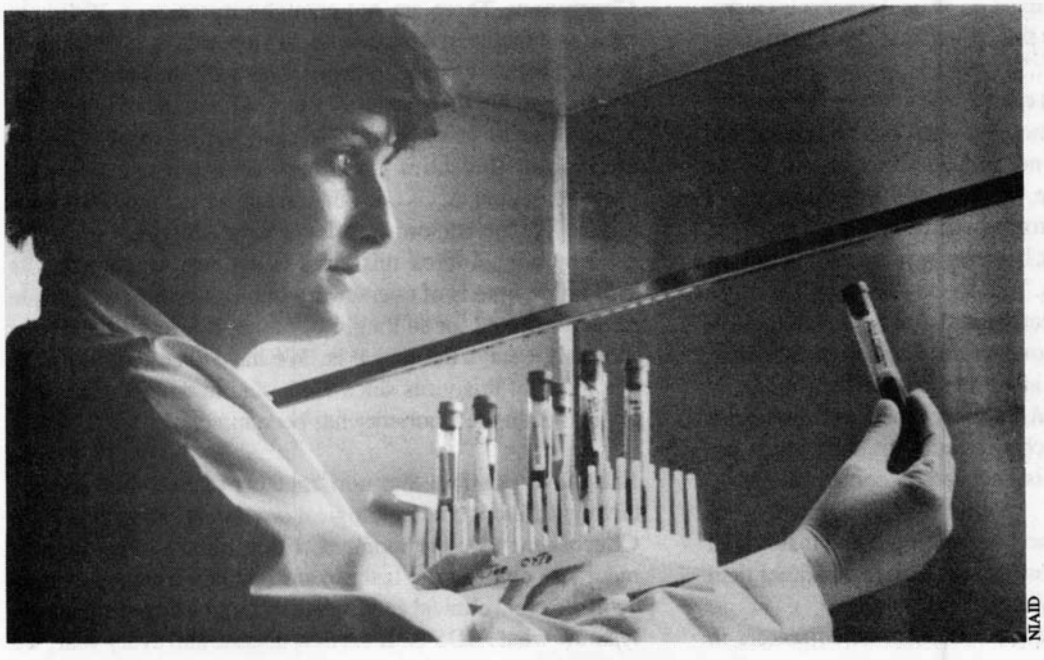
see symptoms we did not know about previously. Anyone would have appeared to be mad who would have said, two years ago, that we were going to see the neurological symptoms that are seen today. We don't know what manifestations will appear in other people infected, for example, after six years, next year, etc. I don't believe we can attribute it to a mutation of the virus. It is a new disease, it started with pneumocystis, followed by a Kaposi sarcoma, then a neuromeningic toxoplasmosis, and now manifestations of dementia. These new nervous manifestations show up but are due to the same virus, to the immuno-deficiency. But for the time being, nobody knows or even can predict whether a new virus will create new symptoms.

EIR: It has been proposed that AIDS may have come from a species jump, from animals. . . .
Chermann: No one can say for sure. In each species you have retroviruses, and the human species have some too. The origin of the AIDS virus may very well be a retrovirus which was harmless among some tribe or another, or in one part of the world or another, but which all of a sudden caused something new in our countries. Smallpox, after all, was fatal outside of our countries.

EIR: How do you view casual transmission? Do you know of any unusual but probable modes of transmission? Do you think such transmission is possible, albeit rare?
Chermann: Our organism is able to fight off a small amount of virus. For example, if you look at the sexual partners of AIDS victims, only 70% of them have the virus, the remaining 30% do not, even though their type and quantity of sexual practices are the same. Which means that the latter haven't received enough of the virus to be infected. . . . That would explain that mosquitoes do not transmit the virus, because our organism is capable of resisting a small quantity of the virus. This phenomenon is easily shown in the case of cow leukemia [bovine leukemia virus—ed.]. If you give less than 2,000 infected cells to new-born sheep, cows, etc., none of them will get infected. But if you give more than 2,000, all of them—100%—will be sero-converted and have leukemia. This threshold idea is an important notion for lentiviruses.

EIR: Well, in this light, the issue of insects in Africa becomes even more interesting, because the population already has a very low immunity, due to a much worse nutrition than we have. Moreover, these countries are already infected with many, many diseases, so the resistance level. . . .
Chermann: First, I would remind you that no free viruses have as yet been shown in mosquitoes, although they may be tomorrow. Secondly, remember what you have read in my paper, not only blood-sucking insects are infected, but also cockroaches, lion-ants, and animals which are never in contact with man, who only eat insect larvae. It's a different situation, and we should not jump to conclusions faster than science.

EIR: To come back to what you said about resistance level. A person with a good overall immunology would need a sizeable dose, perhaps an injection, of HIV virus in order to come down with the disease. So the susceptibility of getting infected or not depends on one's immune system prior to contacting the virus, and the same could be said of carriers. . . .


NIAID

A technician at the National Institute of Allergy and Infectious Diseases works under the "hood" with serum from an AIDS patient.

Chermann: It's quite likely, but we have no evidence. I just said what I did because epidemiology has shown that persons injecting themselves with the HIV virus do not become seropositive, whereas if they had injected the hepatitis virus, they would have caught hepatitis. That means that people who get a small quantity of virus eliminate it. This is the reason why you basically don't get infected from saliva, sweat, tears, or, *a fortiori*, mosquitoes.

EIR: Do we know what is the maximum incubation time for the HIV virus in humans?
Chermann: No, we don't. We only know that some people come down with the disease after three years, and some have not had the disease after six or eight years. So I have no idea. We know of some people who have been infected since 1978, and still have not come down with the disease. Although we have not had enough time to be sure of that, it would seem that the incubation time is shorter with new-born children than with adults.

EIR: Can a so-called healthy carrier transmit the virus before developing symptoms?
Chermann: You mean an asymptomatic carrier. The CDC says that 65% of asymptomatic carriers also have the virus, so, since it is in the sperm, they can transmit it sexually. I don't know in how much time. So we try and tell people who are sero-positive to be careful, that they can transmit the virus, not to give blood, not to take drugs.

EIR: What direction would you hope to see research take at this time?
Chermann: You know, we discovered the virus in 1983, that is three years ago. Look at the progress we've made: We know that the virus causes AIDS, we know its genotype, its identity, we know it is a family of lentiviruses, that anti-viral substances are possible, we have developped a blood test, and we know that a vaccination is possible.

EIR: Do we know that?
Chermann: Yes, I repeat, we know a vaccination is possible. Look, we are getting to know the disease, to see the receptor, we know the variability of the envelope of the virus. . . . All this was found out in three years. So I am optimistic, especially if we get more momentum into the research. I think we should develop more research teams to move ahead more quickly, as all that which has been done in the last three years has been done by very few research teams. But with more research, I am optimistic about the possibility of controlling this disease.

EIR: Would this be a vaccine? For those who have not yet been in contact with the virus or those who have it already?
Chermann: A vaccination is always preventive, so it would be for those who have not yet been in contact with the virus.