

COURT OF CRIMINAL APPEAL 1  
SULAN J 2  
NO.65/2006 3  
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R V ANDRE CHAD PARENZEE 5  
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MONDAY, 12 FEBRUARY 2007 7  
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RESUMING 10.04 A.M. 9  
VIDEO LINK COMMENCING 10.04 A.M. 10  
WITNESS PRESENT IN BETHESDA, MASSACHUSETTS, UNITED STATES. 11  
OF AMERICA: 12  
13  
MS McDONALD CALLS 14  
+ROBERT CHARLES GALLO SWORN 15  
+EXAMINATION BY MS McDONALD 16  
Q. Dr Gallo, I have to deal with a few formalities first. 17  
Have you had your personal assistant provide for the 18  
court a current curriculum vitae. 19  
A. I believe so. It was done when I was away but I'm sure 20  
it has. I told her to do it. 21  
Q. And also a shorter version of career highlights, if you 22  
like. 23  
A. Yes, I asked her to send both: I don't know if she sent 24  
a list of the publications unless you asked for it. 25  
Q. I think we have a full list of the publications. 26  
EXHIBIT #P80 CURRICULUM VITAE OF DR GALLO TENDERED BY 27  
MS McDONALD. ADMITTED. 28  
29  
EXHIBIT #P81 RESEARCH CAREER NARRATIVE OF DR GALLO TENDERED 30  
BY MS McDONALD. ADMITTED. 31  
32  
Q. Dr Gallo, have you also provided for the court a 33  
three-page response really to what you understand some 34  
of the issues to have been. 35  
A. Yes, that was before I had, really, details of the case, 36  
only the generalities, and I had some correspondence, 37  
and I just wanted to say some general things that were 38

on my mind so I did provide that and I thought they	1
would be things of relevance.	2
	3
EXHIBIT #P82 STATEMENT OF ROBERT GALLO TENDERED BY	4
MS McDONALD. ADMITTED.	5
	6
Q. Dr Gallo, I don't want to take you right throughout your	7
curriculum vitae, we have limited time. I just want to	8
take you to a couple of aspects.	9
A. Sure.	10
Q. You are the Director of the Institute of Human Virology	11
at the University of Maryland School of Medicine; is	12
that right.	13
A. Yes.	14
Q. How big is that institute.	15
A. The institute is right now approximately 300, 310	16
people, although a substantial number - it must be close	17
to 100 now - are physicians or PHD's and almost all are	18
doing virological studies, clinical, epidemiological,	19
public health and in the laboratory.	20
Q. Can you explain what that institute actually does.	21
A. Sure; I was at the National Cancer Institute of the	22
National Institutes of Health, which is government, for	23
30 years. Upon retirement I believed that the country	24
and actually the world needs centres of excellence in	25
virology. I kind of recommended that in a book I wrote,	26
that maybe about a dozen would cover all classes of	27
viruses, and I recommended they should have close	28
cooperation with developing nations as well. Our	29
institute focuses on chronic persisting viral	30
infections, particularly retroviruses, such as HIV and	31
the leukaemia viruses HDLD1 and HDLD2, but also include,	32
to some extent, papilloma viruses that cause cervical	33
cancers; herpes virus 8 Kaposi's sarcoma, it's a cancer	34
named after a Hungarian physician over 100 years ago.	35
It's caused by herpes virus number 8. And we also have	36
studies on hepatitis C virus, particularly because of	37
its association with liver cancer and again with methods	38

by HIV, so chronic persisting viral infections, 1  
retroviruses, in particular HIV of the retroviruses 2  
known to exist in man, but also including scientists who 3  
study those other chronic persisting viruses. Contrast 4  
that, we're not experts in influenza. Although we're 5  
virologists we would not call ourselves experts in the 6  
acute viral diseases such as influenza or adenovirus 7  
that causes respiratory illnesses and go away; only 8  
viruses that stay with you. The institute is divided 9  
into five divisions, a division of basic science which I 10  
also head; a division of epidemiology and public health, 11  
and our head of that division is also the Baltimore 12  
Commissioner for AIDS; the clinical division headed by 13  
Robert Redfield, who is one of the clinical pioneers in 14  
this disease; animal model division; and a vaccine 15  
division. We have enormous involvement with developing 16  
nations, especially Nigeria, but including six other 17  
African countries, Guiana in South America and Haiti. 18  
Institute funding is approximately \$65 million per year. 19  
The institute has a general broad commission to try to 20  
go from laboratory to the clinic within one building. 21  
We have an outpatient division on the first floor taking 22  
care of patients on neutral protocols, HIV and virus 23  
HICV. We started with about a small handful of people 24  
in the National Institute of Health that wanted to do 25  
this with me 10 years ago. I would say that's - I am 26  
also a Professor of Medicine and a Professor of 27  
Microbiology and Immunology. 28

Q. How many patients does the institute treat in Maryland. 29

A. We're taking care of at least to 5,000 people, 30  
particularly people without great economic success. To 31  
be more precise it's probably about 4,700. And we're 32  
involved in treating people in Africa; last year was our 33  
first real full year and from virtually nothing, to give 34  
you an example of the country, in Nigeria we treated 35  
38,000 people, and I think by next year in Africa we'll 36  
be treating over 100,000 people. 37

Q. Have you and your institute been involved in developing 38

a candidate vaccine.

- A. Yes. I should also say, I forgot to add before I answer that question, if I may, that our institute has established sister institutes; there is one now in Nigeria, in Bejaia and one in Monterrey, Mexico that will be ready this coming year, one plant for Guaitara, Mexico, ideas for one in Brazil and ideas being discussed for one in Beijing, and one in Jakarta, Indonesia. And you asked me about a vaccine. One of my two closest or greatest current interests is HIV preventative vaccine development, and also I'm interested in the mechanism of how the virus causes the disease, particularly in direct mechanisms, that is the virus infects this cell, this cell may be in trouble, but many other cells that are not infected are impaired. This is not unique in HIV; you see it in certain other human diseases, but what is the mechanism for that is just starting to become unravelled at this point in time, the last few years, and that's a major interest of mine. We hope understanding that will lead to further new notions for therapy. This is a disease that science is keeping up with but you have to keep fighting to keep up with it because treatment is life-long and in combination. Life-long treatment is, for most diseases of man, as you know, almost invariably associated with problems of side effects or problems with drug resistance by the microbe in question, so we have a major interest in that as well. The vaccine candidate is now in the hands of the spin-off company; that company is involved deeply with a major American pharmaceutical company and we will have an extremely important milestone by the end of this year's springtime, which will tell us whether we should be - I don't like to use the word 'excited' with HIV but whether we'll be really, really interested in this candidate. If you want more on that I'll be happy to add to it.
- Q. With this particular vaccine how is it, and if you could

deal with this question in very simplistic lay terms, 1  
how is it that the vaccine is designed to work. 2

A. I am of the school with HIV - not everybody is in this 3  
school - but because it's a virus that we call a 4  
retrovirus, they integrate their genes in the target 5  
cell DNA in our chromosomes, so the cell that gets 6  
infected is infected forever, and quite rapidly. 7  
Moreover, because it's in the DNA of the cell, when the 8  
cell divides and becomes two cells, those genes are 9  
transmitted to the daughter cell as well so the 10  
individual is infected forever. Thus, I believe we have 11  
to block at the gate where the virus comes into the 12  
cells, and I believe we have to come close to 13  
sterilising immunity which means no cell gets infected - 14  
maybe not absolute, but darn close to it. To my 15  
knowledge, no-one has ever achieved any microbe 16  
sterilising immunity. Let me explain better with an 17  
example. We are all vaccinated against polio but, if we 18  
all have a phial of polio within fluid to drink, we'll 19  
all get infected. You're not protected against 20  
infection, but our immune system will have some weeks 21  
for recall, and it will clear the virus and the problem 22  
is over. With HIV it is too late; within a few days 23  
it's integrated its genes so, if we make a vaccine after 24  
two or three weeks, too bad, you've got viral genes in 25  
your body. When that vaccine response goes down that 26  
virus is going to take off again, so I'm of the school 27  
that says it's all or nothing at all. We have to go for 28  
broke. We have to develop a vaccine that develops 29  
antibodies that blocks at the entry of the virus into 30  
the cell, and that is really difficult. We knew to do 31  
that right at the beginning of the field in 1984 when we 32  
really understood this virus; we knew we would probably 33  
have to achieve that so how do you do that? How do you 34  
make antibodies that block? We call those neutralizing 35  
antibodies. You have to do it with the envelope 36  
proteins. If my hand is the virus, my fingers are the 37  
envelope - with some reports they are called knobs - but 38

the envelope protein is the first thing the cell sees. 1  
The virus will attach to receptors on the molecules of 2  
the cell. This is the cell and my knuckles are 3  
molecules on the cell surface; the virus fingers have to 4  
attach to one specific or two specific knuckles, and 5  
antibodies interfere with that. The problem was that 6  
anybody who tried to make a vaccine against the 7  
envelope, the finger, the antibodies were only specific 8  
against the virus you used to make the vaccine, but HIV 9  
is highly variable; another horrendous problem for 10  
vaccine development. So although the vaccine would work 11  
on this virus it wouldn't work on a brother of this 12  
virus, let alone an HIV of a completely different 13  
strain. You probably know there are - we call them 14  
clays, I don't even keep track of them. We're now 15  
getting combinations; they are so many forms. There 16  
used to be A,B,C,D the E. We have B in America; Europe 17  
has D dominating but now we're seeing other clays and 18  
sometimes what we call recombinant forms. Where a 19  
person gets infected by two different strains of the 20  
virus they can have genetic material combining so you 21  
get a brand new strain. And finally, even within an 22  
individual with one virus strain there are micro 23  
variants ad nauseam; just endless micro variants. These 24  
are the challenges of the vaccine. What our vaccine 25  
candidate we believe has done is to help get over the 26  
problem of the variability. That is to say we have 27  
learned out how to make antibodies that are broad across 28  
the different strains of HIV. It doesn't mean we have 29  
the answer because this immune response will have to 30  
last. As I told you we don't have time for recall and 31  
we haven't achieved that yet. We have a number of 32  
collaborators working on it and that is why I was closer 33  
to your part of the world until yesterday. I was in 34  
Japan at a meeting precisely over this kind of 35  
collaboration. I know that's too much I think. I'm 36  
telling you too much. 37

Q. That's fine. In light of that what do you say to the 38

proposition that's been advanced by the defence in this 1  
case, 'Well, if you know so much about HIV and if it 2  
exists why haven't you found a cure for it'. 3

A. Well, if the person wasn't a layperson I would speak 4  
more harshly. You know, I would say that that's silly. 5  
But let me explain with an analogy. The best analogy I 6  
can give is what I said to laypersons 20 years ago. If 7  
I know everything about Mt Everest there is to know; 8  
every cape, every rock, every strata, every bush or every 9  
tree, I can't climb it until you develop the helicopter 10  
for me. That analogy is apropos to HIV. Obviously this 11  
is full of tricks and there are many microbes that have 12  
been around a long, long time that we desperately need 13  
vaccines for that are much longer around than HIV. 14  
Malaria being one obvious example; a bacterium that is a 15  
parasite - a bacterium tuberculosis is another one. How 16  
many examples would one want. The question has no 17  
meaning. 18

Q. Just to go back to your - 19

A. I would be embarrassed if I asked the question. 20

Q. Just to go back to your own expertise, you've been 21  
working now for 40 years in infectious diseases. 22

A. Cancer and infection diseases, yes. 23

Q. Were you the most referenced scientist in the world 24  
between 1980 and 1995. 25

A. That's what the Institute of Information in Philadelphia 26  
published and that's what the United States National 27  
Academy of Science reported. They also did an impact 28  
study based on how you open up various fields and I was 29  
third in the world in impact factor for I think it's the 30  
last 25 years. 31

Q. Can you describe for us what you mean by impact factor. 32

A. Impact factor is a mathematical - the National Academy 33  
of Sciences worked on a mathematical formula to 34  
determine how many references you have, but also put 35  
into the equation was not only within your field but 36  
outside your field, and how many references in journals 37  
that are impact journals which are called, you know, 38

major journals like Science Magazine etc.. I can't 1  
remember all the parameters, but the answer to your 2  
first question simply is yes. 3

Q. Do you also have 27 honorary degrees from universities 4  
around the world. 5

A. By the first week of July I think it will be 27, yes, 6  
from 11 or 12 different countries. 7

Q. You've been awarded numerous prizes; I won't take you 8  
through all of them, but are there any in particular 9  
that are particularly prestigious. 10

A. If we go by countries I guess the United States' most 11  
prestigious prize, which the Nobel prize winner is on 12  
the committee, it's called the Albert Lasker Award. I 13  
believe I've won it twice. Germany's prestigious award 14  
is Frederick Stohlmann Memorial Award; Spain's is the 15  
Principe de Asturias Award, I received it with 16  
Montagnier; and Japan's is the Japan Prize in the field 17  
of Science and Technology and I received it with Luc 18  
Montagnier; and Canada's most prestigious award I 19  
received with Luc Montagnier. There are some awards 20  
from the cancer society; the General Motors Award for 21  
cancer research which is probably America's second or 22  
third biggest prize. 23

CONTINUED 24



I don't think - you have all of this there. You know, I think this is enough.

Q. I want to move on now to some materials that you have been provided with. For the purpose of giving evidence, have you been provided with some excerpts of the transcript from the witnesses in this case.

A. Yes.

Q. And also with the print-out -

A. Can I say something about that though? I have to reply that there was one that I received over the weekend that I really only glanced through and didn't finish part of, and that was Dr Turner's evidence.

Q. Was that the affidavit.

A. Yes. I did get a chance to read part of that today but I didn't read it all, I have to acknowledge, but I did read all of the other materials that were sent to me, including this one by - I cannot pronounce the name properly - the other one you sent to me. I don't know the person or the scientist in question.

Q. Amongst the material, were you also provided with a print-out of a PowerPoint presentation.

A. Yes, I was.

HIS HONOUR

Q. I think her name is Ms Papadopoulos-Eleopoulos.

A. That's it.

XN

Q. You have indicated that you don't know these particular scientists but do you know generally of AIDS denialist groups.

A. Yes, sure I know. If I may, I can tell you that I have seen four or five forms of unusual things in the field. I think the field has some unique aspects to it but one wonders why, but that's not the purpose of the report. One, is that AIDS didn't exist. One of the leaders of this group, Duesberg, denied the existence of the disease itself. This was all about drug addiction and malnutrition and at one point the argument really comes down to that nothing causes AIDS but everything causes

AIDS because I read where it was written by him that 1  
anaesthesia surgery causes AIDS; blood itself without 2  
HIV - blood transfusions - caused AIDS; drugs of every 3  
kind cause AIDS; antibiotics caused AIDS; the drugs you 4  
used to treat AIDS caused AIDS, but there really isn't 5  
AIDS. It is a muffling set of motions. So, that one 6  
group said that AIDS didn't exist but then it evolved 7  
into HIV didn't exist. I saw that only once and that 8  
was at the press conference for the Paul Ehrlich Prize 9  
when a young man interrupted it by saying 'I had to take 10  
a picture of the virus in the body'. I frankly did not 11  
know what world I was in. I mean, it was that weird. 12  
So then, what we have heard is that HIV exists, it is a 13  
virus but it doesn't cause AIDS; and then I have heard 14  
HIV exists and causes AIDS and it was made by the US 15  
government where I worked for 30 years and it was done 16  
to kill blacks. So I think I have heard it all. One 17  
wants not to pay too much attention to any of it. 18

Q. In your observations of these various denialist groups, 19  
including those who say that HIV does not exist, is 20  
there any particular strategy that they employ in terms 21  
of conveying that message and providing information. 22

A. Yes, I don't want to pretend I'm an expert on them. 23  
I'll only tell you what I have seen and what I have seen 24  
in the document given to me by colleagues who kind of 25  
follow it, and I don't know if it is a strategy, or if 26  
it is just obsessiveness or a combination of things, or 27  
just frank ignorance, I don't know, but one thing that I 28  
notice is they use names of people in order to support 29  
themselves and the document you sent to me by 30  
Papadopoulos-Eleopoulos - 31

HIS HONOUR 32

Q. Just call refer to her as 'the witness' rather than 33  
trying to pronounce her name. 34

A. Okay, thank you. I follow that. The witness's document 35  
that you sent to me, for example, quotes Howard Temin. 36  
Howard Temin was a great Nobel Prize winner. He was 37  
also a great personal friend of mine and he would be 38

rotating in his grave if he knew that he was 1  
unsupported. Howard Temin, the witness doesn't mention, 2  
was also the first person to attack this absurdity in 3  
the science magazine with me. I think it was five years 4  
ago, I think it was 1996, in the science magazine. The 5  
reference to another Nobel Prize winner, Varmus, said 6  
reverse transcriptase is frequented and there is a lot 7  
of symptoms of where it says. He would be the first to 8  
say that it is unequivocal that HIV causes AIDS. We 9  
will come back later to reverse transcriptase if you ask 10  
me how misguided, refusing, inappropriate and delusional 11  
the comments on reverse transcriptase are in my honest 12  
opinion, and we need to come back to that, but 13  
therefore, what I see is there are also people who work 14  
with me - the witness uses him with the description of 15  
viral proteins to support them being cellular. I mean, 16  
it is amazing. Also, Constantine is mention. Neil 17  
Constantine works with me now. I mean, this is one 18  
ploy. Another is a lot of material is out of context or 19  
material that is stopped in time. A good example is 20  
Padian, a woman who has done epidemiology who is quoted 21  
as refuting heterosexual transmission because she found 22  
heterosexuals not with AIDS and heterosexuals who have 23  
got AIDS. What is not said is she never looked for HIV. 24  
Of course, when we were doing the antibody studies and 25  
the virus isolation studies in the United States, people 26  
who were healthy people were HIV positive but the 27  
percentage groups were much higher. Therefore, you 28  
could say only anal sex but you have to determine who is 29  
HIV infected. One population doesn't have any HIV at 30  
the time, another population had plenty. Of course it 31  
is going to be anal sex. There are things like that 32  
ad nauseam that for me were frankly - I can't believe 33  
that it occupied the time of the court. I mean, it is 34  
that absurd. It has over and over and over and over 35  
been demonstrated and by the way, the courts of law, 36  
there are precedents in courts of law in the United 37  
States and in Sweden and elsewhere in Europe on this 38

very issue. I am jumping ahead. Today, we know from  
heterosexual transmission - we can study concordant  
couples and show that the strain of the virus  
molecularly analysed and cloned and sequenced are the  
same. There is no chance that it is acquired elsewhere  
except a remote theoretical possibility of one in a  
billion. So you know where it came from. This is no  
longer a matter of guesswork, it is no longer child's  
play, and we quickly passed 1983 and 1984 with far more  
evidence than is given to the court. So I don't know if  
it is a strategy but the mentality stops when they want  
it to stop, apparently in a given period of time. So it  
may be Montagnier's paper in '83 with some things which  
were long ago corrected, or they will take some of  
Padian's paper, call Padian on the telephone and ask her  
whether she believes HIV is sexually transmitted or not  
or heterosexually transmitted. You will get a different  
answer. I don't know if it is absurdly ignorant. If it  
is out of ignorance, how do you hold yourself up as an  
expert? It would be like me arguing with Einstein about  
relativity.

Q. I want to just go back in time for a moment to 23 April  
1984. Was that a significant day.

A. It became significant because the Secretary of Health of  
the United States announced that the cause of AIDS was  
known, that we could mass produce the virus for ever and  
cell cultures and that a blood test was developed which  
would preserve the blood supply of hopefully the world  
in as short a period as possible. In the industrialised  
world, that certainly occurred by early 1985 and by a  
few years later there was no more - essentially no more  
blood transfusion associated AIDS as we took away HIV  
contaminated blood. The blood test detection also  
allowed the epidemic to be followed to allow educational  
programs to begin and ultimately to allow us to know who  
to treat and no longer in America and in Europe, and I'm  
certain in Australia as well, do we have significant  
paediatrics AIDS. Why? Because you can identify an

infected mother; treat her and treat the newborn in the industrialised world. So pediatricians who were experts in HIV are going out of business and blood transfusion is no longer a problem. Those arguments alone, alone I think, should convince the rational world that this is the cause of AIDS and there are far, far more other compelling if not conclusive arguments.

Q. On that date, 23 April 1984, were you involved in a press conference.

A. I was called from Europe to come back to the press conference that I didn't know was going to be called because the Secretary of Health got hold of our papers that were impressed in science and in Lancet. The witness, he referred to as if there is one paper at that time. There were five rapidly and about another half a dozen coming soon thereafter. They were published in May 1984, June and July '84 and throughout '84. So, when she got wind of those papers in the press, she felt compelled to release it. I suppose it wouldn't look good if it wasn't released but we had concern about the press conference for more reasons than one. We were worried that the papers might not be published by the science magazine, for example.

Q. We have heard a lot about -

A. I didn't give the press conference. I answered questions at the press conference.

Q. We have heard a lot in the defence witness's evidence about the early Montagnier work. Your laboratory was also doing work at that time in relation to determining what was the cause of this illness; is that right.

A. Yes, that's correct.

Q. And it was as a result of that work that the five papers that you have referred to in your statement were published.

A. Yes, and rapidly many more.

Q. You have read the criticisms on Montagnier's first publication. Were you involved in that publication in any way.

A. Yes. 1

Q. Please explain to his Honour to what extent and in what 2  
way. 3

A. I guess it is important I provided what we call 4  
reagents. What does that mean? We gave them 5  
antibodies, highly specific antibodies to proteins of 6  
the leukaemia viruses that we had discovered earlier 7  
called HDLV-1 and HDLV-2. Those were the first human 8  
retroviruses and for them to publish they had to be sure 9  
that this was new, so we sent them the antibodies to 10  
distinguish what they had which makes the 1 and 2. We 11  
also sent them molecules that we had discovered known as 12  
Interlucine-2. Interlucine-2 is the protein that allows 13  
us in the laboratory to grow human blood T cells for the 14  
first time. To put it into perspective, it was an 15  
important part of the first time that I received the 16  
Lasker Award. It involved that discovery. Today, that 17  
molecule is also used ironically in the treatment of 18  
AIDS and in cancer to some extent but it allows us to 19  
grow in laboratory culture dishes T cells from humans of 20  
the blood or lymph nodes in order to be able to find any 21  
new virus or any new agent which was in the T cell. You 22  
have to be able to grow it a little bit. Finally and 23  
importantly, I was the reviewer of this paper and it was 24  
accepted into publication. That's it. Okay? 25

Q. Yes. Are there some valid criticisms that can be made 26  
of that very first publication. 27

A. My closest colleagues said that I didn't apply criteria 28  
to them that I would have for the US colleagues and my 29  
view was I was convinced enough, it was nullable, that 30  
it had a good chance of being important and that if they 31  
didn't have the technology at the time to do things to 32  
perfection or as well as possible, they soon would and 33  
the paper merited this ability. So yes, you could make 34  
some critique. The main critique to be made is because 35  
of a technical difficulty of growing the virus in large 36  
quantities. We succeeded in growing HIV in large 37  
quantities by adapting it to permanently growing cell 38

vines. That allows the virus to be produced in mass. 1  
They were growing the virus only with the Interlucin-2 2  
protocols we sent them by taking the blood sample from a 3  
normal person and adding what they called, in this 4  
patient, the fluid containing the virus particles to a 5  
new source of blood cells. Well, blood cells don't grow 6  
very long, so they can't get very much virus. Not 7  
getting very much virus gives a lot of cellular debris 8  
relative to the amount of virus particles. As a 9  
consequence, they couldn't get the viral envelope, what 10  
is referred to by the witness as 'knobs'. So-called 11  
knobs or the viral envelope readily fall off the virus. 12  
If you don't have a lot of virus, how can it be 13  
produced? You lose it. Of course, that's what happened 14  
to them in '83. That didn't happen to us in early '84 15  
and it didn't happen to them thereafter when they learnt 16  
how to properly grow the virus. It was a modest, 17  
technical problem. Yes, it is an imperfect paper but it 18  
was a beginning. 19

Q. It is where the seed was planted. 20

A. Well, you know, I don't like my colleagues and myself 21  
taking third fill or second fill. We were planting the 22  
seed with ideas in technology and it is the first 23  
publication of the right virus, that is a fact, and when 24  
we published we published a lot of viruses, 48, not one; 25  
48 from 48 different patients. Six of them we were able 26  
to grow permanently continuous culture to mass produce. 27  
This gets to an important issue for the judge, I 28  
believe. One of the other things I read by the witness 29  
was a misunderstanding, or if not a misunderstanding a 30  
misrepresentation - I hope it is the former - and that 31  
is this business of purification. You have to purify. 32  
The witness shows a complete lack of understanding 33  
because a sucrose gradient barely purifies. She is 34  
always talking about purifying it into a gradient and 35  
then you have to do that to co-purify. The court should 36  
know that a retrovirus comes out of chromosome membrane. 37  
In so doing, it incorporates some cellular proteins in 38

the virus. You could do it until it freezes over and  
you get viral proteins. What about proteins outside the  
virus? Montagnier's early paper had a lot of that too,  
too much, because by putting it through a sucrose  
gradient it would do hardly anything when you have very  
little virus. So the ratio of cellular material to  
virus, I don't want to say this is an accurate number  
but I will give an example. Let's say it would be a  
thousand to one but when we succeeded in mass producing  
the virus in a continuous culture, you have got an  
enormous purification far beyond the sucrose gradient  
alone because you are now producing loads of virus with  
little amounts of cell. I hope that is clear. And you  
know, I mean, all this purification, it is an extreme  
wild goose chase. The genes of the virus are cloned  
now. All the proteins are purified. We know these  
proteins are encoded by the genes of the virus, we know  
these genes are not in your DNA, nor mine, nor anybody's  
in this courtroom who is not infected by HIV.

CONTINUED



And even a person infected with HIV we know it's not in 1  
his heart cells or kidney cells but specifically only in 2  
the cells that get infected. The DNA is not in the 3  
normal uninfected cell. So this hogwash that the genes 4  
have not been cloned or sequenced in 1985, we published 5  
the complete sequence of HIV, it's not done for many 6  
viruses to this day. Montagnier's group told us within 7  
a few weeks of our paper, it was then done by another 8  
group, by now there are hundreds and hundreds of HIVs 9  
that have been fully sequenced and cloned. Those genes 10  
do not exist in normal cells. I was going out of my 11  
mind reading that. The stupidity of it is to the 12  
extreme. 13

Q. When in your mind was it proved to you that HIV existed. 14  
So when did you become convinced. 15

A. The evidence for us began in the latter part of 1982, in 16  
1983 more evidence accumulated, but it was based only on 17  
reverse transcriptase and we were only detecting it 18  
periodically, we hadn't refined the culture well enough 19  
because HIV gets to come out in a pulse, in a burst, 20  
unlike some of the other retroviruses we were used to 21  
dealing with where you would culture for a longer period 22  
of time before doing the analysis. Little by little we 23  
would learn on how to find it with regularity, starting 24  
with just reverse transcriptase and that was about the 25  
spring of '83, about the time Montagnier's paper came 26  
out, so we have obviously known that something is 27  
cooking, he has a retrovirus, has got a picture of it - 28  
lithium at that time - we were finding reverse 29  
transcriptase now frequently. By the later part of '83 30  
and certainly by January of '84, it was over, for us. 31  
We had enough evidence to make me convinced and if you 32  
say could I convince anybody else? Let me put it this 33  
way, in '83 people were looking for the cause of AIDS, 34  
in '84 the scientific community no longer looked at the 35  
cause of AIDS. So I give you a clear example. National 36  
Institute of Heart and Lung Disease of the National 37  
Institute Development had a \$40 million contract in 1983 38

to find the cause of AIDS. When a press conference came  
out they didn't stop, they continued to contract to find  
the cause of AIDS but when our papers were published in  
Lancet June or July '84 and the four Science papers in  
May '84, that contract was terminated. Overnight the  
scientific community, I think, enormously, was  
convinced. The data that we had accumulated when we  
published was as follows: we didn't find HIV as a virus  
in normal cultures, normal lymphocytes. The witness  
likes to say particles are found from endogenous  
retroviruses, genes exist from ancient infections of man  
in our DNA, of ancient retrovirus infections, part of us  
come out rarely. Yes they can come out occasionally in  
human placenta. Nobody has ever demonstrated, even one  
time, their production from a normal human lymphocyte,  
human blood lymphocyte, not even one. They make it that  
this is just there all the time. That is utter, frank,  
nonsense and if it were true, molecularly it is simple  
to distinguish HIV from endogenous retroviral sequences,  
they are night and day. It is like a giraffe to a  
gorilla. So we have 48 isolates of HIV, from people  
with AIDS or high risk groups, we have none from healthy  
heterosexuals. That was item 1. Item 2, we have  
evidence that the virus was new, and AIDS was a disease.  
No.3, we had evidence that where the virus was there was  
AIDS, but where the virus wasn't there wasn't AIDS until  
the virus came. We had evidence, No.5, that serum  
stored long ago when there wasn't AIDS in the United  
States was uniformly negative for the presence of  
antibodies specific to this virus. No.6, we developed a  
blood test called the ELISA test, which was a technology  
that existed for detecting other things in blood banks.  
We also added the Western Blot, that was the first time  
it was used in clinical medicine as a confirmatory assay  
of specificity. We blindly, that is with coded serum  
samples, tested hundreds and hundreds, ultimately  
thousands of sera from normal healthy heterosexuals in  
the United States. The amount infected were .1%. The

amount of AIDS patients we reported in blinded studies 1  
in Science was 98%, in Lancet it was 100%. That took us 2  
a little bit by surprise, to quote Montagnier 17%, that 3  
is because he didn't have enough virus to do the assays 4  
with proper sensitivity and specificity in that first 5  
'83 paper. We also had the following evidence that 6  
wasn't published then but later but we knew it when we 7  
published and when the announcement was made. In a 8  
collaboration with CBC, and I think I alluded to this 9  
already, we received coded samples of blood from people 10  
who developed AIDS by blood transfusion, along with many 11  
controls. We picked out those that were antibody 12  
positive, they were the ones who had developed AIDS. 13  
Better, they asked us to look at the donors who gave the 14  
blood, the volunteers. We always found one or more 15  
donors who were antibody positive and then through 16  
courtesy CBC went back and followed the history of those 17  
people, all of them got AIDS. This, of itself, is an 18  
enormously obvious evidence that this is the cause of 19  
AIDS. Then the blood is excluded that's HIV positive, 20  
there is no more blood transfusion AIDS. The latter we 21  
didn't know in '84 but it would come, we knew it would 22  
come but the latter wasn't available when we made those 23  
announcements. We knew it infected CD4 T-cells. 24  
Inclinations told us this seems to be a disease 25  
primarily of CD4 cells. I mean what was left, what was 26  
really left? Today, shortly after I should say, you 27  
have the monkey model of a virus that is virtually 28  
identical to HIV2 which causes AIDS routinely in monkeys 29  
that receive it who are not adapted to it, for example 30  
monkeys from India, also Asian monkeys who haven't seen 31  
the relative of AIDS before known as SIV, will give 32  
AIDS, whereas many African monkeys do not because they 33  
are naturally infected with it and evolved with it. A 34  
common phenomenon in epidemiology, virology, biology. 35  
Shortly after that we have therapy. The therapy in and 36  
of itself is an overwhelming argument. The other thing 37  
we knew that babies who were born of mothers that are 38

HIV infected, just like the virus HDL D1, can be born 1  
infected one third of the time, two-thirds of the time 2  
the virus doesn't transmit to the baby. Who gets AIDS? 3  
Only the baby with HIV. Now you treat the mother and 4  
you treat the baby, they don't get AIDS any more. I 5  
mean we could go on and on and on but we knew that in 6  
that period of time, that early period of time, as well, 7  
So we didn't know the therapy, the therapy came later. 8  
So those are the things we published and those are the 9  
things we knew, not in any one paper but the evidence of 10  
the five papers, in the spring of '84 to the early 11  
summer of '84, convinced the scientific world, and that 12  
can't be denied. 13

Q. Again I want to put a suggestion to you that's been made 14  
in this court and that is that in effect the whole 15  
argument that HIV exists rises and falls on the first 16  
experiments conducted by Montagnier. 17

A. That's silly of course. You know that, I mean everybody 18  
knows that that's sitting in the courtroom. 19

HIS HONOUR 20

Q. Not everybody, Dr Gallo. 21

A. That's sad commentary. Was it your Honour who made it? 22

Q. I made that comment. 23

A. Well I would regard that as a sad commentary. Courts 24  
don't stop in science, as I would have thought everybody 25  
knew, and that the day Fleming - to give you an example 26  
of something that should be close to the hearts of 27  
everyone in Australia - Fleming discovered penicillin. 28  
Many people like to say they too have a fungus, they saw 29  
the same fungus but Fleming did something with it. 30  
Surely that was not going to save the world, that came 31  
from Australia, when it was chemically purified and 32  
identified. Science builds on the backs of each other. 33  
Montagnier built on my back with Interlingua 2, with 34  
reagent and some ideas, I built somewhat on his back 35  
with his first publication and we both built on each 36  
others backs and on many people that preceded us in 37  
virology and in retro-virology. So, I mean you can't 38

stop with Montagnier's paper. Well, Montagnier didn't  
claim in his '83 paper that HIV caused AIDS, he could  
not, he didn't have the data for it, he did not and  
could not. So if they stopped with Montagnier's paper  
they stopped with someone who didn't say HIV is the  
cause of AIDS. I'm saying it and he sure said it after,  
and I said it in the spring of '84 for the first time.

+CROSS-EXAMINATION BY MR BORICK

Q. I'm going to approach this in two ways, firstly, as I  
have with the other witnesses -

A. May I know who's speaking?

HIS HONOUR

Q. Defence counsel.

A. This is Mr Borick?

XXN

Q. Yes, I represent the other side. I'm going to proceed  
in two ways, two parts, as I have with the other  
witnesses. I'm going to put to you extracts from  
Dr Turner's affidavit, which you haven't had a chance to  
look at properly, and I'm going to ask you to comment  
without any cross-examination about it 'Here's what  
Turner says, here's what you say'. All right, that's  
will be an exercise I will undertake.

A. I'm willing.

Q. Then I'm going to spend a half an hour asking you some  
questions about some matters which I think you will be  
familiar with. One thing puzzled me, just before his  
Honour came onto the bench you were asked by his  
Honour's assistant whether you were going to swear on  
the Bible, or you were going to affirm. Do you recall  
that.

A. I certainly do.

Q. And your response was - this is before his Honour came  
on, and his Honour didn't hear this - you said 'I don't  
care, I'll do whatever one you like, if you want me to  
swear I will, if you want me to affirm I will'.  
Remember saying that.

A. I certainly did, because I didn't know the difference in

the requirements of Australian law, which one was better 1  
and I didn't know if there were people who objected to 2  
the Bible in the court. Therefore I said I'll do it 3  
either way, whatever makes it valid I'll tell the truth. 4  
Q. To a man with your qualifications, swearing on the Bible 5  
means once thing and making an affirmation means another 6  
thing doesn't it, one you believe in God and the other 7  
you don't. But anyway let me come to my question. 8  
A. That's none of your business. 9  
Q. Let me come to my question then. 10  
HIS HONOUR: Mr Borick might I say I don't see that 11  
that follows actually. But you can have that discussion 12  
with me later. 13  
MR BORICK: All right, your Honour. 14  
A. All I wanted to say is whatever Australian court demands 15  
of a witness, this witness is willing to say that leads 16  
you to accept it as his truthful statement, and I don't 17  
know the customs of Australian courts, therefore I said 18  
'whatever way you prefer'. That was the meaning and the 19  
tone of what I said. 20  
XXN 21  
Q. All right, his Honour has dealt with that. Again just a 22  
preliminary matter. You mentioned this press conference 23  
which Margaret Heckler, the then Health & Human Services 24  
Secretary of the United States of America. Were you 25  
present at the press conference that she called. 26  
A. Yes, I was. 27  
Q. You heard her tell the world so to speak, that you and 28  
your co-workers had found the cause of AIDS and had 29  
developed a test to show whether the AIDS virus is 30  
present in the blood. 31  
A. I didn't - she called a press conference to do that and 32  
she also gave substantial credit to French colleagues 33  
and said as science goes it sometimes goes in parallel 34  
and I only answered questions. Those questions related 35  
to vaccine and the nomenclature of the virus, my comment 36  
was to call it by the name that the French group used 37  
and that I used - used a hyphenated double name because 38

I believed that the viruses would come out to be of the same specific type but that remained to be proved by molecular sequencing and cloning.

Q. First of all, you had not published any of your papers at that point in time, had you.

A. The papers were in press in Lancet and four of them in Science, they came out a few weeks later - two weeks later, and the contents of those papers were by then given out to a great number of people and of course the contents were reviewed by a substantial number of scientists. And it was two weeks later when the world sought papers.

Q. But there had been no peer-reviewed publication before that announcement.

A. No, two weeks later there would be five. If I may, what is your point? Would you make it.

Q. Had you already applied for patent protection before that.

A. No the United States government applied for a patent and I - just, if we are going to go in that direction I can get there fast. I never patented anything in my life prior to the blood test patent and I never patented HTLV1, which is a required blood test in Japan and America, interleukin 2, etc., but on that day the National Institute of Health changed. I used to think patents were like vice holds in medicine, but government told me that there were two purposes of having to patent. One was to make a semi-exclusive licence to large companies, which turned out to be American and European, at the beginning, in order to bring big companies in so that the screening of the blood of the world could take place as soon as possible. Secondly to prevent against fraudulent blood test which nonetheless did come out a bit. And they asked me and my colleagues to write up the technology, we did, we handed it in and we were not involved again until there was a struggle for VAT rights, Europe and the United States awarded the United States government two patents and did not award

the French anything. The French felt they deserved 1  
something. I was asked if that wasn't true, I agreed 2  
but I said 'What power do I have?'. They said 'You are 3  
the visible one' but the government did see it that way 4  
in the United States and that created patent arguments 5  
which ended with Chirac and Reagan signed the agreement 6  
in '88 to share the royalty of those patents. 7  
Incidentally, some of the media I resent from Australia, 8  
and some of the inferences by your clients were that 9  
money was involved, that we did it for money, there was 10  
no money to be given to a governmental scientist 11  
whatsoever. Ronald Reagan changed the law a few years 12  
later allowing government scientists to make some of the 13  
royalty moneys, which have gone to the pastoral 14  
institute, The National Institute of Health allowing 15  
them to build buildings, in fact, and also went to the 16  
Franco-American Foundation which funded the world's 17  
first educational programs for people in the developing 18  
world that came out of this blood test money. 19

CONTINUED 20



I'd like to cut to the chase.

Q. In 1985 the Pasteur Institute alleged that you had misappropriated LAV in developing the blood test; is that correct. They have made that allegation against you.

A. Yes. I can also cut to the chase there. I suppose, as an unpaid volunteer here, I'm allowed to expand on it. Yes, I was told in advance that might happen because the scientists and the director of the Pasteur got out of the picture and said it would be turned over to businessmen, lawyers and public relations firms. The answer, to come to modern times, is that after several years of investigations, we were totally vindicated, as you must know, because you're a man, I heard; that does his homework, in which the conclusion of the final panellists of all this, after all the sound and fury, one would have expected some culpable evidence of wrongdoing - period. This is not the case - period. I would be glad to have you have that for your records or for whatever else you want to do with it. Scientifically, by 1991, Luke Montagnier acknowledged that what he had sent us was the wrong virus, that is, it was a contaminant in his lab. That occurred with an accidental mix up of his original culture, which is a strain of HIV that can't grow. What we grew was his contaminant, by an accident, in our lab. That also happened in his lab. What you forget and what people want you to forget is we published 48 isolates and not one was a contaminant of his and it was because it was a contaminant in his lab first, which we couldn't know, the properties were very different in what he sent us before, when he said he was sending the same thing. Accidental contaminant in the same strain has occurred in lots of laboratories. It is a virus that grows well in cell line culture. Of the 48 isolates - I told you before we grew six in continuous culture - any one could have been used for the blood test, suffice to say, I'll send you for your records a mountain of material on

vindication, as it has been called.

- Q. After the Pasteur Institute made that allegation, did the National Institute of Health in the United States set up an internal inquiry headed by Yale biochemist Frederic Richards.
- A. No. The National Institute of Health used the Richards committee in the final stages after there was found to be no wrongdoing by the original team of scientists which lasted almost two years, then no guilt was found by anybody. A second group came and again found no guilt, this is when a congressman was obsessively going after American scientists, eight to be inclusive, including a Noble Prize winner David Baltimore. In the end we were found guilty of nothing. The Richards committee was advisory to one of Dingell's persons - the congressman going after the scientist. She was head of that committee. She used an outside group, carefully selected, non-of whom were retrovirologists. This one person didn't know any retrovirology, and he was advising and taking the information from a woman who was working for this congressman, a woman whose background was in sociology, not science. That was their role. They never met with us and that is the one committee that wanted to be critical but did not know the facts of the other side. When it was all reviewed collectively, just know this: no scientific committee ever found me guilty of a single thing, ever. There was political pressure in an office in Washington by a powerful congressman that was paralleling some of the worse stages of American history in the past, in some respects. That congressman went after some scientists, nothing happened. His office, not him, apparently put some pressure on people that were lawyers, such as yourself, in an Office of Research Integrity in Washington DC having nothing to do with science and they acknowledged that in Science Magazine after all this was over. They said they were damned if they found something wrong with me and they were damned if they

1 didn't. That's the quote from the Science Magazine. No 1  
2 scientific review body found me guilty of anything. 2  
3 Lawyers, for a few weeks, did, then dropped it all when 3  
4 my colleague, who did the work that was being contested, 4  
5 went forward and it was reviewed by objective people, by 5  
6 scientists, retrovirologists, molecular biologists - a 6  
7 host of people brought into a room like you're in and, 7  
8 over a considerable period of time, evaluated the whole 8  
9 thing and found him totally innocent as well and they 9  
10 dropped anything with me. I repeat - the early 10  
11 inquiries and investigations of me found nothing by any 11  
12 scientific group that ever evaluated me. 12

13 Q. You referred to a person then by the name of Dingell, 13  
14 that is the United States Senator John Dingell. 14

15 A. He's not a Senator, he's a congressman. 15

16 Q. Following an inquiry by the National Institute of 16  
17 Health, there was then an investigation by an 17  
18 organisation known as NIH, Office of Scientific 18  
19 Integrity or OSI; is that correct. 19

20 A. Yes, that is what I alluded to earlier. There were 20  
21 three or four virologists and the head of the committee 21  
22 was a woman, non-scientist, who was in fact later found 22  
23 to be working for Congressman Dingell, who in fact was 23  
24 let go by NIH when all this was learned, as you must 24  
25 know. She lost her job. The answer is yes and we were 25  
26 found not guilty of anything and having proven, I have 26  
27 documented every single one of the 48 isolates we 27  
28 claimed we had. Everyone was documented by 28  
29 investigations - that must be as much as Galileo went 29  
30 through. All 48 were documented on the day and date we 30  
31 claimed - 48, not 1. I want to keep emphasising that - 31  
32 48 isolates of the virus, not 1, were all documented. 32

33 Q. In September of 1991, did OSI publish a draft report in 33  
34 which you and a Mr - 34

35 A. No, OSI did not publish a drafted report, Congressman 35  
36 Dingell disavowed it. You should know that - it was 36  
37 disavowed by Dingell and done by the same lady. I 37  
38 cannot comment on that lady's overall capacities, only 38

that she had to be released. The lady who released that 1  
wrote it herself. She was released from NIH. She was 2  
not a scientist. She's a sociologist. She had other 3  
problems also that I will not go into but if I were on 4  
trial, I would let you know about them and for you to 5  
use her and quote that release document suggests that 6  
your clients have led you down a really fuzzy path that 7  
they don't understand. That was circulated around by 8  
people that wanted to attack HIV. That document doesn't 9  
exist as any accepted document by anyone. It was not 10  
only thrown out of the appeals court, it was disavowed 11  
in Science Magazine by Congressman Dingell. The woman 12  
who released it used to work for Congressman Dingell, 13  
had a job at NIH, until they found out what she was 14  
doing. She was discredited. That is her document. 15

Q. I just want to put to you the history of the inquiry. 16  
Was the review - 17

A. That is not in the history because that is not a 18  
document that is a formal document, it is her fantasies. 19

Q. Her fantasies then, were they then reviewed by the 20  
Office of Research Integrity, known as - 21

A. I already told you the final conclusion of that. If 22  
you're trying to agitate, you could be succeeding a 23  
little but you'll never get past this point. The final 24  
appeals court - I went through an inquiry and 25  
investigation by scientists, nobody found me guilty of 26  
anything. Amen. That lady set up her own side review 27  
which selected people - none of whom were 28  
retrovirologists, some of whom were pure chemists - who 29  
were not allowed to meet with me, who were not allowed 30  
to meet with my colleagues. We were not allowed to give 31  
any comfort to anything of what she said. She then made 32  
up her own release which was disavowed by Congressman 33  
Dingell, who she was working for. That is in Science 34  
Magazine and then my co-worker, Popovic, who's paper was 35  
being attacked for what I would call very slight things. 36  
Nobody ever in that group charged anyone with 37  
misappropriating a virus, that was innuendo. That was 38

the French patent lawyers but it was never part of the governmental statement. Popovic, my co-worker, was reviewed for whether his paper acknowledges a classic for succeeding in the mass production in HIV - that was making what was important even more golden and that fell down and didn't stand under appeal. When that didn't stand, this group that wanted to do damage to me dropped everything.

Q. One final question on this topic, what was the finding of the inquiry by the Office of Research Integrity.

A. The Office of Research Integrity said the following - you have to be able to understand the sentence: - following their conclusion, in an interview in Science Magazine, I will repeat again: they claimed they were damned if they found anything wrong with me and damned if they didn't. They were trying to say - what they did say for two weeks in duration was I was asked 'Do I take responsibility for everything my name appears on, a publication as a senior author, being the last or the first author, with the introduction and of discussion of the world?', and I said 'Yes'. In an effort to get down and find something, it wasn't misappropriation of the virus that you are misleading the courtroom, it was for this sentence - listen to the sentences. I took full responsibility - not Popovic - I said me - the sentence says: the viruses we described in these papers - viruses plural - 48 - are likely to be the same type as the virus described in the paper by Luc Montagnier and colleagues. That is sentence one. There will just be three. No problem. Sentence two: But there are apparent differences. For example, Montagnier thought gp41 was a self-protein called actin. Montagnier didn't have what you call knobs - the viral envelope as we clearly described it. That is sentence two. Sentence three: differences may be due to the fact that the virus wasn't produced in adequate quantity because it wasn't grown in a cell permanent culture. That sentence is the problem. Why? Because we had succeeded in growing the

virus but obviously we're referring to the only paper in the literature, we're referring to the French. But because it was technically not true, they said that therefore it is false, therefore that could be falsification. That is how far madam Hadley went with the lawyers downtown - they were lawyers, not scientists - to try and find guilt. That was hung over my head for two weeks. It was then entirely dropped, vindication in the newspapers came out and the court of appeals looked at the Popovic thing and they dropped everything and said there was no evidence - one iota of evidence of wrongdoing. You have got the summary of the final legal conclusions.

Q. Turning to Dr Turner's affidavit, he starts by saying it is his understanding of the point of view that you take.

A. I just want to say, I understand the approach - what you just tried to do. It is not fruitful and it wasn't needed and anybody who read the conclusions knows the story. My integrity is not on trial.

Q. Turning to Dr Turner's affidavit, he starts by putting forward his view of the stance you take. He described it in this way 'According to the HIV AIDS experts, the HIV theory of AIDS is as follows: hence, a person has AIDS when he or she has HIV and develops one or more of these diseases. HIV does not directly cause the approximately 30 different AIDS indicator diseases but indirectly by its effect on the immune system'. Do you agree that he's explained your position correctly.

A. Part of it. Part of it is wrong. By the way, can I ask you who is Dr Turner? I didn't have a chance to really study who he is. I have never heard of him before.

Q. He's a doctor who works at the Royal Perth Hospital and he's worked in collaboration with the witness over the last 25 years and has published quite a number of papers, in conjunction with her, on this issue. That is who he is.

A. Is he a virologist? Does he do experiments on AIDS?

HIS HONOUR	1
Q. No, he's qualified in emergency medicine.	2
A. I see. I'm not. Don't ever come to me if you're hurt.	3
XXN	4
Q. Has he correctly described your view.	5
A. I said, yes and no. Yes, in parts, and, no, in other	6
parts.	7
Q. Perhaps we'll deal with the nos. What does he have	8
wrong.	9
A. HIV itself impairs the immune system and I thought you	10
said that it was only other things that came after. HIV	11
leads to -	12
Q. I'll read it again so you can listen to it. 'HIV does	13
not directly cause the approximately 30 different AIDS	14
indicator diseases but indirectly by its effect on the	15
immune system'. Do you agree with that or not.	16
A. I agree with that but I certainly never said that	17
because I wouldn't know there was 30. I have no idea	18
how many things there are. There's a lot. When a	19
person is immune deficient, as you know from reading and	20
studying this case, you can have a lot of things happen.	21
You can have an increase in select types of human	22
cancer, particularly those of which that are viral	23
caused. Your witness says that the viral cancer program	24
where I worked failed, there are no viruses which cause	25
human cancer, which is equally as nonsensical that HIV	26
doesn't exist as a virus and doesn't cause AIDS. That's	27
another story. There's statements all over the place.	28
There are a lot of things that can cause disease in an	29
immune-impaired person that cannot cause that disease in	30
a healthy person, that is the case. Cryptococcus	31
infections, all kinds of infections that are mild	32
transit and some people can be serious and	33
life-threatening in an immune person and frequent,	34
rather than rare.	35
Q. Turner goes to another topic 'A virus is a microscopic	36
particle (a minute piece of matter) made up of a nucleic	37
acid genetic blueprint (RNA or DNA) and some proteins.	38

Viruses are so small they lack the space necessary to  
contain the raw materials from which to produce the  
substances and energy required for their replication  
(reproduction). Hence, in order to replicate, viruses,  
unlike bacteria for example, are obligate parasites of  
living cells'. Do you agree with that.

CONTINUED



A. Yes, textbook. He's textbook, that's a classic statement about a virus. You teach it in first year biology. 1 2 3

HIS HONOUR 4

Q. That is what I was going to ask; is that what they would teach medical students. 5 6

A. Exactly. That's probably the first statement you'd make. It's a textbook statement. 7 8

XXN 9

Q. He then says 'retrovirus particles can only be visualized and their morphology studied using the electron microscope.' Do you accept that. 10 11 12

A. Fundamentally that is true. When you want to study structure it's by electron microscopy, yes. There are variations of that technology now. Certainly at that time it was - 13 14 15 16

Q. He says 'Controls are an essential component of the retrovirus isolation experiments because 'retrovirological phenomena' may arise, even spontaneously, in material known not to be infected with a retrovirus'. 17 18 19 20 21

A. Yes; that's a classic example of a nanogram of knowledge is dangerous. So the answer is a yes, no. There are endogenous virus like particles encoded in our genome. What does that mean? Long ago we were infected by retroviruses from one species or another of animals. Those retroviral genes integrated in our DNA and we evolved with it and, eventually, they became infected genomes so you couldn't make fully infectious viral particles. A considerable amount of our genome contains such sequences that may encode nothing or they may give us a few kind of retroviral endogenous proteins, or they may actually give rise to a particle which so far is not infectious. However, your client or I should say your witnesses before, and this one, make it appear these things are jumping beans that come out all over the place. This is nonsense. In normal human lymphocytes they've never been seen, they've never been identified 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38

and, if they were, it's as easy as eating a piece of  
apple pie to distinguish one retrovirus from another.  
Morphologically it's not so good, but epidemiologically  
protein characteristics and especially by molecular  
biology, which is never in your witness's accounts, with  
electron microscopy since 1984 it's simple to  
distinguish. Retrovirus endogenous particles of man,  
which were seen for the first time probably in the  
1980's, have never infected another cell. You can't  
transmit it. To this day no-one has taken such  
particles and put it in another cell, unlike HIV, which  
obviously, Mr Borick, no-one could have misappropriated  
any virus if it can't grow. If you can't put it in a  
culture and it can't grow, none of these endogenous  
particles can grow. The witnesses also fight with  
themselves throughout the witnesses' testimonials. They  
say one thing here which is incompatible with what they  
say later. This would be a beautiful example. We could  
transmit HIV into normal cells; you can't do that with  
endogenous retroviral particles. Where they've been  
seen occasionally is in normal human placenta. I've  
never seen a report to this day of any verified  
endogenous human retrovirus particle coming out of  
normal blood so it is misleading, I assume not  
deliberately; I assume it's because of lack of  
knowledge, to say they're popping out of cells all the  
time. That is utter nonsense. One wishes so; there  
would have been a lot of publications early in one's  
career when we were looking for such things. It's like  
looking for needles in a hay stack. It took us 10 years  
to find the first human retrovirus HDL1. In that time  
we cultured thousands of tumorous tissues, thousands of  
normal human blood cells. We never cultured an  
endogenous human retrovirus particle. We also never  
could transmit today endogenous human retrovirus  
particles. No one has done that to this day in culture  
or in man.

Q. Okay; I move on to the next topic. Dr Turner says

'However, what Montagnier reported as isolation was  
detection of an enzyme activity, that is, reverse  
transcription - not purification of virus-like particles  
proven to be infectious'. Do you have any comment to  
make on that.

A. Yes, sure; there was originally the paper. If you say  
Montagnier didn't say it was infectious we have to be  
careful here - you and me. We have to distinguish  
between invitro and infoetal. Invitro, in order to  
propagate it, he had to take normal blood from donors  
weekly and add the soup around the culture that  
contained virus particles to the new normal human  
lymphocytes, and he finally did - he had viral  
particles. In short in my mind he certainly transmitted  
it invitro. He satisfied me, but let me tell you where  
your people are confused about purification. The  
witness, not Dr Turner, but the witness that I read -

Q. We know what you mean, the witness -

A. Yes - keeps coming to this point of purification. I'll  
make the statement that it is utterly absurd. Let me  
tell you why. I said it before; I'm going to say it in  
this context now. All retrovirus particles that form,  
form from lifting off the cell membrane, pulling out of  
the cell. We call it the phenomenon of budding. All  
enveloped viruses, they have a lipid fatty substance  
around them formed by budding off of cell membrane. All  
such viruses carry within them, right within the virus,  
if you purify you see it is all over, cellular proteins  
that are not virus encoded. In addition, around the  
virus you'll still have some cellular protein. You  
can't purify just by putting it through sucrose  
gradient. Montagnier's early problem was inadequate  
growth of the virus. I'm saying this repeatedly and I  
don't want to say it as a criticism of Montagnier's  
paper. He reported a new retrovirus particle. He could  
transmit it invitro. He didn't say it was the cause of  
virus in the baby. He couldn't characterise it well.  
We cannot fault him for that because he couldn't grow it

properly. Once we could mass-produce this virus, that's purification. If you have a tonne of something and you contaminate it by a drop of water, didn't you purify it? It's the ratio of cell protein to viral protein. Sucrose gradient gives you a little bit of help but you could do that five times and it's not going to purify as much as we did by mass-producing it. To use the extreme hyperbole, if you have a tonne of some something and a drop of water, you've purified it. That's what we did. Stop focussing on the Montagnier paper. The world doesn't end with the Montagnier paper. The beginning began before it - it's the beginning of the Wright virus which is the cause of AIDS which comes out subsequently.

Q. I'll refer to his next comment which I think you've already answered in that. But he says 'Subsequent researchers have not performed experiments substantially differently from those reported by Montagnier and his colleagues. Hence, based on the currently available data it is not possible to claim that a unique retrovirus has been isolated -' that is in purified ' - from the tissues of AIDS patients'. You disagree with that don't you.

A. I'd disagree with it. It's sad. He doesn't even read the papers. This was in one of our five first papers in spring of '84, the first one in fact. We succeeded in putting six of the 48 isolates into permanent culture, meaning in a cell line, in a leukaemic cell line that, itself, doesn't have virus particles, and the virus comes out in great quantity and forever, thus making purification already accomplished. But, of course, we also use banded virus by sucrose gradient which they make a case out of we never did. You don't publish that. Of course we did, but it isn't needed and wouldn't be needed if you could mass-purify it. But, for other purposes, we did it. For example, in a paper we published in '85 we showed for the first time in literature that the Wright self-classification was not type C but type D, but that paper used banded

retroviruses. Apparently they don't know these papers 1  
were ever published and they don't know molecular 2  
biology was ever published because they ignore that the 3  
full genome of HIV, it's over 9,000 - it's full genome 4  
has been completely sequenced in 1985 by us and by the 5  
Pasteur Institute group to which are these publications, 6  
in very visible journals, ours in Nature and I think 7  
there was in Cell. Another one from us from another 8  
isolation of HIV from another patient in Cell the year 9  
after and many, many hundreds of HIV isolates have now 10  
been sequenced, and all the testimony of the witness is 11  
in denial of this. It avoids it. It says HIV is 12  
unique; it's sequences are not in normal DNA and 13  
therefore cannot be endogenous viral particle. It's 14  
proof. 15

Q. Turner makes the point that 'virus isolation is not the 16  
routine method of diagnosing HIV infection because it is 17  
technically demanding, time consuming and expensive and 18  
unnecessary'. Would you agree with that. 19

A. Yes. I believe that the surrogate test has been more 20  
than validated that came from our laboratory in the 21  
science papers. That test has been verified world-wide 22  
except in the minds of the witness, not Dr Turner - I 23  
guess Dr Turner too - but everyone else in the world 24  
that an ELISA test followed by a Western blot will score 25  
with enormous sensitivity and enormous precision. Let 26  
me tell you something that we didn't publish but we gave 27  
to the Heart Institute in that period of time. We 28  
received blinded, that is coded, samples to do the 29  
antibody testing on and we tested and those we got 30  
positive they sent out the blood cells along with 31  
controls from normal donors that were not infected. We 32  
isolated HIV from every case that was antibody positive. 33  
Antibody positivity means virus when done properly. 34

Q. Turner says 'Individuals who fulfil criteria deemed a 35  
positive test result, which vary considerably, are 36  
referred to as being HIV antibody positive. This term 37  
is synonymous with HIV positive and neither term means 38

- HIV particles have been isolated from a person'. 1
- A. Yes; of course, you have to go through the expense and 2  
time and labour and astronomical costs; if you did that 3  
your Australian blood supply would not be preserved 4  
today as it is. That is to say, if you were going into 5  
the hospital and received five blood transfusions for, 6  
God forbid, some serious problem, you had a good chance 7  
if you were travelling abroad, even in Australia, but 8  
particularly in some parts of the world of getting HIV 9  
by blood and dying in the early 1980's or late 70's, but 10  
now the blood supply in the industrial world is 11  
completely protected. You won't get AIDS any more. 12  
That's proof. That's the antibody testing but, if you 13  
had to do virus isolation, it would take a week and it 14  
would cost many many thousands of dollars, and you made 15  
need the blood in an emergency. You'd be dead. 16
- Q. Going on with Turner, he says 'Antibodies are not 17  
viruses. Antibodies and hence a positive antibody test 18  
may be indirect evidence of a viral infection but if and 19  
only if the antibodies are proven specific'. Do you 20  
agree with that. 21
- A. I certainly agree that if you do the test improperly and 22  
if you're incompetent you could get false 23  
interpretations and think you're positive when it's 24  
negative or vice versa. The test - no test in medicine 25  
is perfect. To the best of my knowledge this is as 26  
close to being as good as it gets. I repeat again, in 27  
my hands or my group's hands, we found the virus every 28  
time we found antibody positivity in that study designed 29  
to verify the foolproofness of the blood test done 30  
properly. Having said that, there are rare cases where 31  
you can get fooled. That is true with any test and, 32  
frankly, as far as I know it's true in almost anything 33  
in science. We rely on science very very commonly, if 34  
not always, on surrogate test results as you must know. 35  
You don't measure directly an electron and proton. We 36  
know they exist. 37
- Q. Turner says 'To perform a test to determine whether 38

there are antibodies that react with HIV two things are 1  
required; (a) the HIV proteins, (b) a serum specimen 2  
from the person being tested'. Do you agree with that. 3

A. Yes, sure. 4

Q. He goes on to say 'To obtain the HIV proteins first it 5  
is necessary to purify the virus particles. This is 6  
because viruses replicate only in cells and cells 7  
themselves, like viruses and living matter in general, 8  
are also made up of RNA and proteins. Luc Montagnier, 9  
the discoverer of HIV, agrees with this commonsense 10  
requirement. Added to that is a reasonable link between 11  
the current test and the original Montagnier virus. 12

A. You know, if a person has the antibody and you have a 13  
viral prep that is contaminated by some cell debris, the 14  
antibodies pull down three or four proteins that are 15  
found in no normal cell, and you have another prep that 16  
does the same thing without the virus, in one case you 17  
get bands on Western blot that signifies something 18  
unique that is not present in the normal. That would be 19  
a great beginning, but the situation is far beyond that 20  
after Montagnier's paper. Yes; people use great 21  
quantities of mass-produced virus which by itself is 22  
purified virus. You are not being told that either 23  
because they don't understand it or they don't know; I 24  
don't know. It has gone through a far greater 25  
purification than any banding can produce. The blood 26  
test that became available to people who knew how to do 27  
it came from mass-produced virus, originally from in 28  
Frederick, Maryland. The genes now have been cloned. 29  
We started in publications in 1984 throughout 1985 which 30  
showed that each of the proteins you pick up with 31  
Western blot is coded by one of the genes of HIV or 32  
another. Therefore we know those proteins come from HIV 33  
and, when a patient's serum reacts with them, we know 34  
that patient, untreated, will almost always get AIDS. 35  
You can't get science any better or anything more 36  
definitive. As a generalisation, I've said before and 37  
it's in the record, I sincerely believe - well, put it a 38

different way, to my knowledge there is no disease of  
microbial origin that there is more evidence for than  
there is HIV being the cause of AIDS. I've said that  
for the past 15 years.

CONTINUED



A. Tuberculosis, you would be looking for the cause of TB 1  
for the rest of your days. 2

Q. I am just keeping an eye on the clock and I have got a 3  
bit of a way to go. I don't want to cut you off but if 4  
you could be a bit more shorter in your answers. 5

A. Okay. 6

Q. Turner says 'During an interview in 1997, in response to 7  
a question about what was necessary to characterise the 8  
HIV proteins', he answered 'Analysis of the proteins of 9  
the virus demands mass production and purification. It 10  
is necessary to do that'. What do you say to that. 11

A. I said we did long ago, in '80. We, my lab, our lab, 12  
did it. It is done. That is short and sweet. It is 13  
done. And I think Montagnier wouldn't say that today 14  
because of gene cloning technology. Then, yes. Now, 15  
no. 16

Q. Turner goes on 'Yet in the 1983 science paper in which 17  
Montagnier and his colleagues claimed to have first 18  
isolated and purified HIV, they did not publish any 19  
electron micrographs to prove that the material which 20  
they called the "purified virus" contained particles 21  
bearing the morphology of retrovirus'. 22

A. I would have to review the paper. I wouldn't have 23  
accepted it if I thought it didn't show retrovirus 24  
particles. However, the pictures were not perfect 25  
quality because they couldn't produce much virus in the 26  
presence of - you will get virus particles that are 27  
partly degrading. That's what happened to them, but 28  
there was sufficient evidence to convince me. Heck, if 29  
you are not convinced, just go to my papers. I will be 30  
sure they are convincing there. 31

HIS HONOUR 32

Q. Have they published pictures since. 33

A. Montagnier - 34

Q. No, anywhere. Have pictures been published. 35

A. Anywhere? Your Honour, yes. I don't want to say of 36  
course but, you know, hundreds and hundreds and hundreds 37  
of pictures are published. My lab published many 38

pictures of it. You don't need electron micrography any  
more. You are wasting time and money. Indeed, it is  
hard to find electron micrography around when you know  
where it is already and you want to study the structure.  
Experts are throughout the world. He certainly  
published a lot of electron micrography. I collaborated  
them. He has used, by witness 1, names. He doesn't  
believe in HIV. This guy would be flabbergasted. He  
has published many papers on it.

XXN

Q. I will continue on with Turner. I will come back to  
that topic in a little time. He says 'Research  
published since 1983/84 shows that the proteins  
considered unique to HIV may be found in non-HIV  
infected cells'.

A. That's extremely wrong, Mr Borick. We are talking about  
the proteins we know are encoded by HIV. Of course -  
let me try to make sure we are not talking past each  
other. There, when you do an Eliza today, let alone  
today, and western blot, yes, yes, you will get proteins  
that are from normal cells, and sometimes some people  
will have an antibody that will cross-react with some  
normal protein but the pattern of proteins that are  
unique, gp120, gp141, gp44, gp17, p6, these proteins are  
nominal to HIV. I'm not saying there is no proteins in  
normal cells of that size but when you get antibodies  
from a person that react and give a pattern, almost  
always you will isolate HIV. Almost always that person  
is going to get AIDS and those proteins that are proven,  
as we did in our papers, with specific antibodies,  
including monoclonal antibodies which don't cross-react  
with anything, we know are encoded by HIV and are  
certainly not normal cells. If you know what you are  
doing, you can point out an HIV infected person and  
isolate a virus from that person today and prove your  
point, and we did it.

Q. Turner goes on 'Nonetheless, HIV experts apparently  
believe there are proteins belonging to a retrovirus HIV

and claim to use them to detect "HIV antibodies" and 1  
thus prove HIV infection. Even if there was proof these 2  
proteins are those of a purified infectious particle 3  
proven to be a retrovirus, the fact that patients have 4  
antibodies that react with these proteins is not proof 5  
the antibodies are caused by infection with HIV. This 6  
is because antibodies induced by a particular antigen 7  
react not only with that antigen but they also react 8  
with other antigens. This is a critically significant 9  
issue'. Could you comment on that. 10

A. Yes, I will have to repeat again, that's why we added 11  
the western blot to the Eliza. The Eliza is very 12  
sensitive, it gives too much false positives, but if you 13  
rely on - 14

HIS HONOUR 15

Q. Can you go back professor. The reporter is trying to 16  
take you down. 17

A. Let me make it brief and go back. Look, we in our 18  
papers told the scientific world screen with the Eliza 19  
but confirm with the western blot. New technology not 20  
discovered by us, discovered by other people in the 21  
basic science lab who brought the medicine to us because 22  
there would be too many false positives with Eliza 23  
alone. Very sensitive. So yes, you get some cellular 24  
degree and you make it from antibodies reacting and you 25  
think that person is positive when the person won't be 26  
positive. Having said that, nonetheless, Eliza alone 27  
isn't bad, it is it just gives too many false positives. 28  
In the first early years, because the major companies 29  
that were protecting blood supplies in the world for 30  
other diseases had in hospitals the Eliza equipment 31  
already but not the technology for the western blot, 32  
they didn't wait, they just applied the Eliza. At that 33  
time I was pulling my hair out. They were right and I 34  
was wrong. They had to act because they were saving 35  
lives. Even if some false positives would go in, they 36  
were saving lives in the blood bank, but eventually 37  
everybody put the western blot in and the western blot 38

gives the specificity coupled with the Eliza because the 1  
western blot looks at a whole series of proteins that we 2  
know are encoded by HIV. They have specific patterns, 3  
okay. When we showed this pattern, we did - you don't 4  
do it today but in order to be sure of what we were 5  
saying, we isolated the virus and samples like I told 6  
you before any time we got an antibody positivity. They 7  
sent us samples blind. We say, Harry, Jane and Tom have 8  
antibodies positive. They sent us a whole lot of blood 9  
samples that included Harry, Jane - what did I say - 10  
yes, Jane. We would find the virus only in those 11  
people. Jesus, I don't know what else we could do. I 12  
mean, that's it and that's all virology, by the way. 13  
This is not unique for HIV. If you apply this HIV, 14  
you've got to start going back to tuberculosis and every 15  
virus that ever existed that you had a diagnosis for. 16  
It is done by serology, not by virus isolation, and I 17  
wonder if they did it as well as we did to get blind 18  
samples of the virus every time you have an antibody. 19  
With the retrovirus by the way, you know 'antibody' 20  
means infection because once the retrovirus infects, we 21  
told you before, it integrates antigens. Infection is 22  
for ever. You don't get an antibody and then it goes 23  
away. You get an antibody when you become infected and 24  
that antibody stays with you because you will always 25  
have the virus genes making some viral proteins. Almost 26  
always that is the case. So the antibody is just, in 27  
fact, marvellous. But just for the sake - for the 28  
court, people have gone beyond that today, not that it 29  
is needed, but some people have very low level of virus 30  
that is beyond the sensitivity of the best test I ever 31  
think it was developed. So it has employed nucleic acid 32  
technology to find the viral genes today and that is 33  
also better because if you or I were to get infected 34  
tomorrow, it takes several weeks to make antibodies. So 35  
for those several weeks I could be donating blood and I 36  
would be donating virus positive blood. So today you 37  
want to look for the virus itself and they do so by 38

using sensitive techniques that pick up the virus in the blood; not by isolation but by finding the genes of the virus in the blood. That is clear to you? I'm not sure if I'm making anything clear.

HIS HONOUR

Q. Yes, I have heard a lot of evidence and I hope I understand some of it. I may not understand all the technical science but I certainly understand what you are saying.

A. I am very aware of what this must be for people to go through. It is amazing that you can go through it but let me repeat that. This is very important. Today and for the last decade or so, five years anyway, we don't just look at antibodies. You do that in the bloodstream, yes, but when people are being treated, we also want to know earlier - it takes several weeks to make antibodies but if I got infected today, you could tell in two or three days I have a virus not by antibody detecting but by looking at the blood through the virus itself. How do you look in blood for the virus itself? There are tools of molecular biology today that allow you to find the specific genes as however, the nucleic acid in the blood, you know, shortly after infection. So making the test now a nucleic acid test, which, if you want to pay for that test, you can find out if you are infected within a short period of time. You don't have to wait three weeks, and you are finding virus, not antibodies.

XXN

Q. I'm going to have to leave out some parts of what I would want to put to you because time is running out on us.

A. If you want to, I will move fast.

Q. You have got the whole thing there and I'm sure his Honour wouldn't mind if you wanted to make comments about other things he says.

A. Okay, I will try to be as fast as I can but some of the things are complex to explain properly, especially when

there is so much misleading information in my mind. 1

Q. He says 'According to the HIV AIDS experts, HIV is a 2  
retrovirus with the unique RNA genome. The term 3  
"genome" is defined as the full compliment of genes and 4  
the genome is necessary for the HIV particles to 5  
reproduce the virus particles'. Would you agree with 6  
that. 7

A. Yes, of course. Absolutely true. 8

Q. 'To identify RNA as that of a retrovirus a scientist 9  
must first purify the viral particles. This is because 10  
the cells in which the virus is replicated also contains 11  
RNA. Since the particles said to be HIV have not been 12  
purified and it is not possible to claim a particular 13  
RNA as that of HIV -' I think you have probably already 14  
answered that in your other answers. 15

A. Yes. Briefly, I totally disagree with that statement. 16  
This statement is from someone who doesn't understand 17  
molecular virology and what you can do. If you want to 18  
have - virus produced in mass is already cloned ribo. 19  
You don't need that today. When we clone HIV, you can 20  
make a DNA copy of the viral RNA through reverse 21  
transcription. You can show that those DNA called CDNA 22  
have representation to the normal cell. They don't bind 23  
to anything in a normal cell, only in a virus infected 24  
cell. In a virus infected cell, you can clone the genes 25  
right out of my GNA. If I am infected, you can take my 26  
blood stems and you can clone if my cell is HIV. You 27  
don't need to purify HIV. There are many ways of 28  
skinning the cat. He doesn't understand. 29

Q. I want to deal briefly with PCR techniques. Turner says 30  
'However, there are no published correlations between 31  
the viral load (Number of RNA molecules) and the number 32  
of particles considered to be HIV in blood. This is 33  
because to date no HIV researcher has published even one 34  
electron micrograph demonstrating the existence of even 35  
one such particle in the blood of even one AIDS patient; 36  
B, RNA molecules are not viral particles and viral 37  
particles are needed for infection to take place. 38

Hence, the term "viral load" is both unfounded and misleading'. I think you have already answered -

A. Briefly, viral load measured by PCR looks at viral RNA. It looks at the RNA of what is called HIV. That is never found in a person who is not HIV infected. That is never found except in association with HIV proteins if you accompany it with virus isolation. Where does Dr Turner think the RNA which came from that is not from HIV? There are two places it comes from. It is present in HIV particles there in the blood. Yes, to equate it with just a number of particles is not completely accurate because there are cells that are degrading that release some HIV RNA that can float around in the blood as well, so it doesn't give a direct one to one correlation, but all of it is derived from HIV presence in the human body. Those sequences are unequivocally not found in normal uninfected cells. If I take blood from all the uninfected people in the court, I cannot find those sequences in you no matter where I look no matter what sensitivity. They are virtually specific for this virus. But to equate, I will agree that you can't equate viral load with absolute number of virus particles because there will be dying cells that will release some viral RNA from the cytoplasm of the cell. I wonder where Dr Turner thinks that RNA is coming from? It is not in anywhere else except associated with HIV particles.

Q. Turner says 'HIV experts acknowledge that there are problems measuring the actual viral load. Different laboratories and different PCR tests obtain markedly different results for the same viral load of the identical specimens'. Do you agree with that.

A. Yes, I can't testify to the court that it is markedly. There are also variations depending on how they purchase the assay and exactly what you look for and what your primers for the PCR are. We are getting technical but you can look for this part of the genome or you can look for that part of the genome and sometimes these don't

exactly correlate one to one. That is true.  
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And certainly no AIDS expert. And when you say - like I didn't work in AIDS all my life, I worked in virology, so I have a background that's before HIV and I worked in retroviruses since 1970, that's getting to be a long time. But I don't know if Dr Turner worked as long or as hard as I did in retro-virology but I got it. I'm just telling you that when you use PCR, properly done you are picking up HIV, the exact - HIV, I would agree with Dr Turner, is sometimes debatable precise differences in techniques, differences in detecting something.

Q. Dr Turner says 'In order to count RNA molecules, a scientist must have a test able to distinguish between HIV RNA and all other RNA's'; do you accept that.

A. What was the premiss?

Q. 'In order to count RNA molecules a scientist must have a test able to distinguish between HIV RNA and all other RNAs'.

A. Well, you do do that but the answer is really no, but you do do it and you wouldn't want it very much but of course the answer is no. You can do it by showing differences in proteins, by showing differences in the immune reaction with proteins using normal antibodies, you can do it by using different biology, by showing different cell type that these viruses affect that cell, this cell, you can do it all sorts of ways but certainly at the genomic level, molecular biology level that has been done. It has been done.

Q. 'According to the United States Centres For Disease Control "In adults, adolescents and children infected by other than perinatal exposure, plasma viral RNA nucleic acids tests should not be used in lieu of licensed HIV screening tests"'. Is that the position.

A. I don't know. I don't know that. I don't know if that's the position anywhere.

Q. I will do it quickly: CD4 cells Turner says 'Physicians treating HIV positive and AIDS patients monitor the number of CD4 cells in the peripheral blood. A decline

in their numbers is interpreted as proof. The cells are being killed as a consequence of infection ... the fact that CD4 cells are administered into the bloodstream does not prove the cells are being killed'; do you accept that.

A. I would accept that, yes, I do accept that but I want to make a general statement to the court, that never in the history of any microbe has anyone had to understand the mechanism of disease or what we call pathogenesis, before you can conclude aetiology, causation. We still don't know exactly how polio works, we are still working on new leads on how influenza causes influenza. There are many ways HIV leads to impairment of the immune system and some of them are still mysterious and some of them may involve - factors when you are sufficiently immune suppressed. There is no doubt that HIV affects CD4T cells as its primary target, there is no doubt that that is the cell that is chiefly declining in HIV infected people. Let me just say there are many ways HIV leads to immune impairment. Some may not be direct from HIV but from some things that act as factors that promote aggression. For instance - let's go in the other direction - there are people who progress very slowly to AIDS, in contrast to most of us who would progress at a certain rate, by year 7 or 8 you would be in trouble. We know the mechanism in some cases. An example, if I am born with one of my knuckles missing, the receptor in my CD4T cells for HIV, if I am born with a low number in this particular knuckle here that HIV needs to infect the cell, then if I'm infected I progress to AIDS much slower. That is proven time and again, so it's a mechanism and it obviously is totally comparable with our knowledge of HIV. So there are people who progress slower, given reasons, but HIV, the statement as Mr Borick read it I have no objection to.

XXN

Q. We are moving away from the affidavit now. In Nature 1986 you wrote 'The results presented in our four papers

provide clear-cut evidence that the aetiology of AIDS and ARC was the new lymphotropic retrovirus HTLV3 (HIV)', is that right.

A. Well I first came to that conclusion two years earlier in Science. So if you want to say I said it again in Nature, okay, I don't remember - say it all the time.

Q. In your report to the court, which the judge has, you gave a number of examples of evidence that HIV is the cause of AIDS, and I won't read them in full because we have them in front of us but it's 1 to 4 - 'Initial 48 isolates' you understand.

A. Yes.

Q. Taking those first four in combination, doesn't it mean the only evidence -

A. You said 'Nature 1986', I don't know what paper you referred to in Nature '86.

Q. It doesn't matter when you said it, it's what you believe, isn't it.

A. Well it does tell me the lack of care of whoever's writing it is. It tells me that people don't even read - I'm pretty convinced that nobody reads any of the papers or understands what they read in time, other than Montagnier's early paper - and reverse transcriptase -

Q. It's a paper -

A. I don't think they are reading the papers.

Q. - a paper written by you, HTLV Nature 321 (1986) 119. I don't care whether you said -

A. It's an '86 it was probably looking back on something but go ahead, tell us the point.

Q. Take the first four points that you made in your report to the court, in combination, doesn't that mean the only evidence you had that HIV causes AIDS was two things. First the isolation of HIV from 48 out of 119 patients, that is only 40% -

A. Whoa, whoa.

Q. Let me finish my question and you can respond.

A. Well you are quoting the wrong information to the court, it wasn't 119 patients that we -

Q. How many was it. 1

A. Well I think we isolated in almost every patient with 2  
AIDS that we tried. I can't remember exactly unless I 3  
look at the paper but certainly we didn't get negative 4  
results in 70 other people. You may be including the 5  
normal controls where it wasn't isolated. You have to 6  
look at that. We certainly didn't fail to isolate in 7  
the others. 8

Q. I got it from Nature 1986 May 4 paper and a number of 9  
other people - 10

A. Assume that, assume you are correct but it isn't 11  
correct. 12

Q. What we are putting to you is that the only evidence you 13  
had that HIV causes AIDS was two things, firstly 14  
isolation of HIV from 48 out of 119 patients, that is, 15  
40%. Second, the finding of positive antibody tests in 16  
88% of the patients in the Science papers and 10% in the 17  
Lancet papers. Do you agree with that proposition. 18

A. I agree but I think the denominator is wrong in the 19  
virus isolation but that's part of the argument, yes. 20  
There are more. 21

Q. Do you agree that the isolation of HIV from only 40% of 22  
patients is not proof that HIV causes AIDS. 23

HIS HONOUR: He doesn't accept that proposition 24  
Mr Borick. 25

MR BORICK: I am just asking him to put it. 26

A. You are right, Judge, I don't accept the denominator and 27  
if Mr Borick is surprised my memory has failed me but I 28  
would say of course, in and of itself 40% isolation of a 29  
new virus I wouldn't say is the cause. But Mr Borick 30  
let me stop you and say, I would be phenomenally 31  
stimulated by it, especially a virus that targeted CB4T 32  
cells, especially CB4T cells and was new and unknown in 33  
man before and was being found in risk groups but not in 34  
healthy heterosexual populations at that time. We also 35  
reported isolation of the virus from Haitians, the 36  
absence AIDS, not frequently but much more than the 37  
healthy US population. In gay men acknowledging a 38

history of some contacts, and in IV drug addicts. These  
were non-risk groups at the time. That's where we  
isolated the virus, we failed to isolate in any healthy  
heterosexual. That was argument one, taken together,  
not isolating the argument with one piece, the  
denominator of which I strongly suspect is not accurate.

Q. We are both at a disadvantage with the time problem.  
I'll put the proposition to you, you respond yes if you  
agree, or no if you disagree.

A. I just go through what I said, it's faster than you can  
put the questions. Maybe I forgot something when I  
wrote the letter. The evidence was this, what we just  
said on virus isolation. The fact that the virus was  
new, the disease was new. The antibody tests of 88% was  
done again blinded 100%, with finding the virus in only  
1 in over 1,000 healthy heterosexuals, which was a .1%  
in healthy heterosexuals. The fact that we knew that  
all serum was negative when there was no AIDS in  
different countries that we were receiving. The next  
argument - I said all this at the beginning - was that  
we had knowledge of, though not yet published, the  
results in donor recipient matched people, which I said  
to you in and of itself is more powerful than anything I  
know about a lot of other diseases. Let me - the Gallo  
laboratory, let's say 10 people with AIDS, 35 people  
without AIDS and who had received blood transfusions, we  
score all the ones with AIDS positive, the ones that  
didn't get AIDS are negative, and then we go to the  
donors and we pick out the donor who has AIDS, who gave  
the blood transfusion, and of itself that's a mighty  
powerful thing, I knew that when that press conference  
occurred and when we told Secretary Heckler we were  
convinced that the cause of AIDS was in our hands. That  
and the fact that it also targeted CB4T cells and  
impaired them - invitro - was what I was aware of on  
that day and very very rapidly - oh, and I was aware  
that babies that were HIV positive had AIDS, babies born  
of the same HIV positive mother who were not HIV

positive didn't get AIDS. Well, you know, they love to  
quote Robert Pope - which were made from bacteria and  
not viruses because it is impossible to fulfil the way  
Pope wrote them - as you have Turner pointed out - of  
its own and can't survive outside a cell very long. But  
Robert Pope, thank goodness, was smart enough not to be  
so obsessive with his own postulous that he didn't  
fulfill very many when he acted on column, he never  
fulfilled anything but one or two of his four or five  
postulous when he acted to take care of people by trying  
to make power of that genome therapy. In short, he had  
brains.

Q. I get you about postulous. Do you accept that 87% of  
AIDS patients have antilymphocyte antibodies.

A. Antilymphocyte antibodies?

Q. Yes.

A. No, I never said that.

Q. You don't accept that.

A. I don't understand. You mean anti-HIV antibodies?

Q. The question I put to you is -

A. Something's wrong.

Q. Do you accept the proposition that 87% of AIDS patients  
have antilymphocyte antibodies.

HIS HONOUR

Q. Do you understand the question.

XXN.

Q. If you don't understand the question that will be it.

A. No, I understand the question, I don't know what he  
means antilymphocyte antibodies, HIV -

Q. Antilymphocyte antibodies, that's the question.

A. I don't know what that means.

Q. If you don't understand the question I can't take it any  
further with you.

HIS HONOUR: What is the source for that statement?  
That's what the witness is asking you. Where did you  
get it from?

XXN

A. You can't find that in my - 1  
Q. I'm not suggesting that it's in your paper. 2  
MR BORICK: Unfortunately I haven't got the source of 3  
that. I can't take that any further. 4  
A. I don't know what it means, I just don't know what you 5  
mean, what the science of that means. I don't know who 6  
said it, why they said it and what the heck it's 7  
supposed to mean. 8  
XXN 9  
Q. In '83 a person you will know well, Essex, showed that 10  
about a third of AIDS patients have - you agree with 11  
that - Essex showed that about a third of AIDS patients 12  
have antibodies - 13  
A. Yes, I don't know that he said a third but I will accept 14  
it, yes, I will accept it. 15  
Q. In that case why isn't HTLV the cause of AIDS. 16  
A. Simple. Because first of all Essex was using an immune 17  
fluorescence reactivity which he acknowledged he was 18  
picking up - immune fluorescence reacts with anything on 19  
the membrane and it fluoresces by a technique that you 20  
can do for labelling, then you call it positive, it's a 21  
notoriously inaccurate test. Moreover, 10% of people 22  
with HIV in the United States at that time were doubly 23  
infected with HTLV1 and HIV, so his per cent comes down 24  
now from 20 to 30. So it's 20% with HTLV, doubly 25  
infected. When you prove this - because the test 26  
immunofluorescence is a very notorious test for picking 27  
up cross-reactive antigens, you look at the whole cell, 28  
you look at the antibodies - you take serum from a 29  
patient, add it to the cell, the cell infected with 30  
HTLV2 lit up a third of the time. It's not sufficiently 31  
scientific - 32  
Q. How is that then borne out - 33  
A. We know because we have been told by lots of witnesses. 34  
Time moves on. 35  
Q. If the tests were improved how do you know that you 36  
wouldn't find HTLV1 in all AIDS patients. 37  
A. Because I looked, I discovered the virus and no-one 38

knows how to test better than I do. In Essex - they are  
wrong. You may realize that I would have loved the  
disease to be caused by a variant of HTLV1, then I would  
have discovered - no matter what you are driving to, I  
had all this expertise in HTLV1, I discovered it, used  
it since '80. They use it to test for it in Japan, the  
United States uses immunofluorescence - artefacts, we  
know that, Essex relied on it, unfortunately. He is a  
respected colleague and my friend and that was a  
mistake, and he knows it.

Q. Do you agree that 70% of AIDS patients have antibodies  
to endogenous retroviruses.

A. I never heard such a figure and I don't know what it  
means because endogenous retroviruses aren't viruses as  
your first witness properly said, they are particles,  
they have never been transmitted. A virus is something  
that infects, that you prove goes from person A to B.  
Short of that they are particles. Where a virus at  
least has to be transmitted invitro in the laboratory,  
it goes from one cell to another, it's never been  
demonstrated for endogenous retrovirus. Hence it is  
meaningless and I don't know where you get that and if  
somebody reported that they are doing crappy assays in  
immunology.

CONTINUED



The genome of endogenous retrovirus particles is already 1  
known in sequence. You have to be aware of where the 2  
science is. We already know the full genome of the 3  
endogenous retrovirus sequences in men. Nothing is the 4  
same as HIV - nothing. 5

Q. In your evidence quite early on you refer to Harold 6  
Varmus, a noble warrior. 7

A. Yes. 8

Q. Do you accept, in the 1987 paper 'Reverse Transcription 9  
at Scientific American' he said 'Reverse transcription 10  
is hardly unique to retrovirus. It is now recognised as 11  
a widespread phenomenon in eukaryotic cells. Evidence 12  
has made it clear -' 13

A. I went through this - 14

Q. '- evidence has made it clear that reverse transcription 15  
takes place in the uninfected cells of yeast, insects 16  
and mammals'. Do you agree with him. 17

A. Absolutely, absolutely. I actually worked on that a 18  
bit, but you have to understand that Harold Varmus would 19  
rather go into blazes than to agree with your 20  
insinuations. You're saying that you think it could be 21  
confused. These are reverse transcriptases that are 22  
coming in these particles, but, no, the reverse 23  
transcriptions Dr Varmus is talking about exist within 24  
the cell are transient, are not outside the cell in a 25  
particle form and are never transmissible from human 26  
cell A to human cell B and nobody but the dumbest of the 27  
dumb could confuse a retrovirus reverse transcription by 28  
innumerable parameters from the reverse transcription 29  
process that Dr Varmus has talked about. He certainly 30  
didn't talk about that as a confusion for HIV. 31

Q. Do you agree that the p24 protein which Montagnier 32  
discovered is one of the most significant HIV proteins 33  
and is the core protein. Is that your position. 34

A. Significant, in terms of quantity, certainly, yes. I 35  
would say yes to both points, though he labelled it p25, 36  
it is 24,000 closer in its molecular science, than 37  
25,000, as we published. 38

Q. In his 1983 science paper he referred to the 1.16 band as the purified virus, is that right. 1 2

A. I suppose. He did a 116 cross-gradient in that paper, yes. I don't know if he said it was purified. If you do that you don't have much virus. 3 4 5

Q. Is it also true that in this material he found three proteins - p24, p45 and p80 - which reacted with the serum from his patient but concluded that only one of these proteins - p24 - was an HIV protein and the antibodies that reacted with it were the HIV antibodies. 6 7 8 9 10

A. I don't know about 80 and something else but that is probably true. Certainly p24 is a protein, the heat detected, it is the same p24 that we found. 80 we never found. 11 12 13 14

Q. In a paper published by you in 1984, May, 'Frequent Detection and Isolation of Cytopathic Retroviruses (HTLV3) from patients with AIDS and at risk of AIDS', you noted that virus was observed by electron microscopy. Do you recall that. 15 16 17 18 19

A. Yes, I recall it and we published it, which Dr Turner and the witness seem not to know. There is at least three electron microscopic pictures of HIV in those first four papers. 20 21 22 23

Q. In that same paper, as I understand it, you say 'For each of the following categories for AIDS, the number positive from HTLV3, the number tested and percent positive are listed. For juvenile AIDS the percentage positive was 37.5%, for adult AIDS with Kaposi's sarcoma, 30.2%, and for adult AIDS with opportunistic infections 47.6%'. Would you accept those figures, that that's what you reported. 24 25 26 27 28 29 30 31

A. I don't remember but, okay, I accept the figures. 32

Q. The abstract of the paper states 'These results and those reported elsewhere on this issue suggest that HTLV3 may be the primary cause of AIDS'. If that is right, how did you conclude this when fewer than half the people with AIDS were positive, by your criteria. 33 34 35 36 37

A. Let me start with where you're going with this. Let me 38

ask you and your witnesses this question: what 1  
percentage do you think you can isolate of any virus 2  
from any disease, other than in a peak of an acute 3  
viremia with an acute infection virus? Do you know? Do 4  
you know how many times you can isolate HTLV1, the 5  
leukaemia virus, it would probably be in the same range 6  
or a little less. If you went back to molecular 7  
hybridisation, you would find it in the tumor with an 8  
adult that has leukaemia of a certain time that was 9  
described. To isolate the parts, you wouldn't do better 10  
than 30%, surely, maybe less. Because of technical 11  
difficulties in samples - because samples you get are 12  
decayed, they are liced and cells are destroyed. If you 13  
don't have enough of the target cells, CD4 T cells 14  
harbour the virus, CD4 T cells in a person with AIDS are 15  
low. The population of the blood you get, you have 16  
hardly any CD4 T cells to work with. You would be a 17  
liar if you said you isolated it every time. The 18  
percentage is going to fall off because you don't have 19  
many CD4 T cells to deal with and those that are there 20  
are impaired. You put all the data together that we 21  
knew in 1984 - let me put it this way to you, to 22  
Dr Turner and to witness number one and to everyone else 23  
in the court: WHO evaluated this problem, the National 24  
Institute and Health evaluated this problem, the 25  
National Academy of Sciences, USA, evaluated this 26  
problem, the Institute of Medicine evaluated this 27  
problem, UNA evaluated this problem, less so and the 28  
Pasteur Institute. All have come to the same 29  
conclusion, that the evidence is overwhelming. You're 30  
relying on a non-scientist, who works in an emergency 31  
room, that hands you an affidavit that you take to court 32  
to debate with me, who spends, since 1970 full-time, in 33  
retroviruses and showing and studying disease in the 34  
origin of diseases. Be my guest. 35

Q. As I understand, you see an electron microscope, a 36  
photograph of a virus, easy to recognise. You can see 37  
it straightaway; is that right. 38

A. No, of course not. It depends on the ratio of virus to cell, if there's a lot of cellular debris there, it will degrade virus particles and change morphology. If you culture it too long the envelope will fall off. You won't see knobs - as they're called by your witnesses - if you have minimal production it is good to see good virus particles, so, no, I don't agree with that at all. It is highly dependent on the circumstances, the amount of virus you're producing relative to cellular debris and the speed with which you do the electron microscopy.

Q. I thought you said earlier you would easily recognise this virus, HIV in the photograph.

A. Yes, you can. If you do the conditions properly and if you have a good virus producing cells like the one we have, those viruses were put into that clone of cells, that leukaemic cell line, anybody could get good electron micrographs but if you have human blood cells, as Montagnier had to deal with, and you put virus into that, you'll get a great amount of cell debris relative to virus particles and it will be almost impossible to get really good quality electron micrographs. That is the point.

Q. In May 1984 did the group that you are with, in a paper 'Serological Analysis of Subgroup of human T-lymphotropic retroviruses HTLV3 associated with AIDS' published in science p.504 of that paper, was there a set of three electron micrographs labelled HTLV3.

A. There were a series of micrographs that were labelled HTLV1, HTLV2 and HTLV3.

Q. Was it really HTLV1 or was it actually LAV from the Institute Pasteur.

A. We have gone through that before and I will tell you again -

Q. No, we haven't.

A. The answer is this - listen carefully folks: because that was led to the patent lawyers of Pasteur talking about misappropriation. The answer, first of all, by background, we also published electron micrographs of

RF. If you look at the whole and not what you're trying 1  
to prejudice the court with. Moreover I told you that 2  
Montagnier sent us a contaminant of LAV. LAV cannot be 3  
grown and cultured. It was acknowledged in 1991 in 4  
nature that they sent us the wrong virus. They sent us 5  
LAV twice, you can't grow it. They sent us LAV a third 6  
time, they got a contamination in their lab, they have 7  
acknowledged it. On the 20th anniversary of HIV, 8  
Montagnier and I published together, it is Gallo and 9  
Montagnier, December science, in which he acknowledges 10  
it very forthrightly. There is no way we could have 11  
known that was his virus because it is a virus that 12  
grows like a weed, and anybody who knows what they're 13  
doing can cross-contaminate one of our cultures. That 14  
is why we went through all the crap we went through and 15  
documented that we had 48 viruses of our own. Those 16  
papers include electron micrographs of RF as well as 17  
others, if you look at all the papers. The question and 18  
the purpose of the question is meaningless, except to 19  
misconstrue. 20

Q. Does it not mean that in the four papers that you and 21  
your group published in science in May 1984, not one of 22  
them contained an electron micrograph of your punitive 23  
AIDS virus. Is that the fact or not. 24

A. Did you listen to anything I said? Are you listening to 25  
anything I say? I told you there is a picture of RF. 26  
Look at the paper. You'll see RF, that is us. You're 27  
not even obligated to show an electron micrograph. 28  
Nobody shows electron micrographs any more unless 29  
they're doing a structural study. If you're doing the 30  
study and structure of a virus you show an electron 31  
micrograph. We showed electron micrographs of RF and we 32  
showed, unfortunately, the cross-contaminating one that 33  
is now totally understood in which we were completely 34  
vindicated that we had 48 isolates of the virus. We 35  
have electron micrographs on six. We published on two 36  
and two is enough. One was a cross-contamination. 37  
Nobody is obligated to publish any electron micrographs. 38

I dare say, I hardly know a virology paper that goes  
with that. I don't understand something, I have to ask  
the court: how can the lawyer ask me that question when  
I have already gone through it and when he has the  
science papers and there are other electron micrograph  
papers of the virus RF? Why would you ask me if that is  
the only one showed? When you said 'isn't that the only  
one you showed', that is provocative.

HIS HONOUR

Q. I understand what you are saying, counsel from time to  
time can be provocative, professor, it is a matter of  
answering the questions as best you're able to answer  
them, whether they be provocative or not.

A. Even when I have already answered it before? You have  
to listen sometimes.

Q. Even when you say you've answered it before.

A. This is kind of a new experience for me. You'll have to  
understand.

XXN

Q. In the first of the two papers I have referred to you  
have said 'The virus was observed by electron  
microscopy', and then, as I understand it, in the other  
paper you have published three photographs saying 'This  
is the photograph of the virus', and that was a mistake.  
Is that right.

A. Well, it is not a mistake that it is a virus, it is not  
a mistake that is a beautiful electron micrograph. It  
is a mistake that was a laboratory contamination of the  
virus Montagnier sent to us, in part, because he sent us  
a contaminant that occurred in his lab that he didn't  
know occurred in his lab and didn't have the properties  
of anything other than viruses we were dealing with, so  
we thought that was our virus because his doesn't do  
that. That's the point, but it is not true that that is  
the only virus picture we showed and it is not true that  
you need to show a single virus picture and it is not  
true that we didn't prove we had 48 isolates - we did.  
When the first buyers in the committee got through, they

said nobody in the history of medicine had to go through 1  
what we did. Nobody in the world has documented all the 2  
virus isolates like this and thank God they were in the 3  
day and time we said we got them. We didn't make any 4  
mistakes in the 48. They're documented in the notebooks 5  
and the genes are available and they form, by the way, a 6  
great spot for the whole world. They are a part of 7  
Australia's blood supply, some of them, and they were 8  
the world's for the first 10 or 15 years. I don't 9  
expect you to thank me but I don't expect to be provoked 10  
to that degree. 11

Q. In the Scientific American article that you and 12  
Montagnier published in 1988 - in the Scientific 13  
American - as I understand it you say that Montagnier's 14  
EM picture shows a lentivirus, but in his original 1983 15  
paper, Montagnier described his EM as showing a typical 16  
type C particle and then in the abstract of your third 17  
1984 science paper, you state that HTLV3 is just a true 18  
member of the HTLV family, which is a type C retroviral 19  
particle and not a lentivirus. 20

A. Right. 21

Q. Do you agree with that. 22

A. That is absolutely correct with the quoting. This, I 23  
understand and I can understand your line of questioning 24  
for it and its reason. Do you want the explanation? 25

HIS HONOUR 26

Q. I would. 27

A. Retrovirus - quite frankly - 28

XXN 29

Q. Would you just mind if I clarified my question. In 1983 30  
and '84 you were saying these pictures show a typical 31  
type C particle and in 1998 you say the same pictures do 32  
not show a typical type C particle but a virus particle 33  
belonging to a totally different family of retrovirus. 34  
That is the specific question. 35

A. You are right. The subclassification of retroviruses 36  
into different families is subtle and, ultimately, of no 37  
consequence, either to the science or to the human 38

health. It is a subtlety. I, no doubt, was  
overwhelmingly influenced by my prior influence with  
HTLV1 and HTLV2. We knew that HTLV1 and HTLV2 were  
transmitted by blood, by sex and by mother to child,  
mainly and we knew that HTLV1 and HTLV2 were targetting  
CD4 T cells. These characteristics, plus the fact we  
knew they were prevalent in Haiti and in Africa, led us  
to think almost certainly this retroviruses could belong  
to the HTLV family. Moreover, it took us 10 years to  
convince people of one retrovirus that would be a  
different category of retroviruses, certainly it would  
seem to be. Frankly speaking, I never relied on  
electron microscopy. I don't think electron microscopy  
does much, except for the person who's a structural  
biologist and wants to look at real structure. No-one  
uses electron microscopy and virology any more - nobody.  
It is as rare as hens teeth. I have come to the point -  
to me, they look more like type C. I had no experience,  
99.9% of retrovirologists had no experience with  
lenti-retroviruses - indeed, unless you read the  
veterinary literature - I did - but most  
retrovirologists did not. They had never heard of  
lenti. They are associated with non-retroviral disease  
and they were associated with encephalitis and found in  
animals called angulates and they were seen and only  
studied in the veterinary literature and there was  
hardly biochemistry done in the animals. It is not a  
category of virus that was in your head - a subcategory  
of retrovirus. The same is due to Montagnier. I will  
repeat: 99.9% of retrovirologists came out of cancer  
research and were leaning towards expertise in molecular  
biology and many never heard of lenti-retroviruses.

CONTINUED



It was Matt Gomba who was a collaborator of mine who 1  
happened to have his background in veterinary medicine 2  
who said we were sending him a lot of virus in the cell 3  
producing the large amounts of virus, and he was banding 4  
it and doing electron microscopy and it was him - 5  
although I'm a co-author, I contributed really nothing 6  
to that paper. It was Gomba who recognised the 7  
lenti-retrovirus nature, not me. He corrected a mistake 8  
from lack of the structural familiarity by almost all 9  
retrovirologists with this family known by 10  
veterinarians. That is the answer. Montagnier, of 11  
course, didn't know. It wouldn't be almost in our 12  
vocabulary. It was an unknown world. Really, it 13  
doesn't get me anywhere, you anywhere, human health 14  
anywhere, or these questions anywhere. It is a 15  
retrovirus. Who gives a damn if it is lengthy or not. 16  
It doesn't affect anything. It is scientifically 17  
accurate and important to the science that we classify 18  
it right, but for human health, for this case, for 19  
disease causation, for the blood test, for anything we 20  
are talking about, for pathogenesis, it tells us 21  
nothing. 22

Q. What is the correct classification for this retrovirus. 23

A. It is a lenti-retrovirus. It is clear even by its 24  
genomic analysis. The problem was there was no detailed 25  
genomic analysis of the animal lenti-retrovirus. There 26  
was no background to go to. There were just pictures of 27  
them. They weren't studied by vets in great molecular 28  
detail. It is HIV virologists who pushed the molecular 29  
study. We now know their genomes are much closer to HIV 30  
than is HTLV1 or HTLV2. Surprise. 31

Q. In your view, does seeing a particle which looks like a 32  
retrovirus prove that particle is a retrovirus. 33

A. No, it gives strong suspicion only, but it can't be 34  
confused with arenaviruses and it can't be confused with 35  
endogenous particles if you are dealing with early 36  
embryonic life. That would be a retrovirus-like 37  
particle. You wouldn't know one from the other just 38

based on that, especially if the quality of the  
electromicrograph is not really good. The quality  
published and unpublished vary enormously. It depends  
on the microscopist, which I am not one, it depends on  
how quick you got your culture and how much quantity of  
virus you have relative to cell debris.

Q. In a paper published by you and others in 1976, 'Some  
evidence for infectious type C virus in humans', you  
said: 'Virus-like particles morphologically and  
biochemically resembling type C virus, but apparently  
lacking the ability to replicate, have been frequently  
observed'.

A. By us? No. That's not true. You can't show me a paper  
I published in '76 that they aren't able to replicate -  
found frequently in humans did I ever publish, sorry.  
You are misunderstanding or misreading something.

Q. It is a paper published by you and someone called  
Wong-Stall and others, 'Some evidence for infectious  
type C virus in humans', Baltimore, D. Huang, A.S. Fox  
CF, Animal Virology, New York, Academic Press, 1976.

A. You mean particles, not viruses. You said 'viruses'  
before. Particles, yes, you can find particles not  
released from cells in some cases of human leukaemias  
that we were looking at. Those are likely to be  
endogenous retroviral elements not forming full virus  
and certainly not transmitted in culture and certainly  
not infectious.

Q. Does all this mean particles with the morphology of  
retroviruses which have reverse transcriptase activity  
are not necessarily retroviruses because they do not  
replicate.

A. Absolutely. If you had a virus that didn't replicate,  
you couldn't call it a virus, unless you transmitted it.  
Montagnier did succeed in his paper in transmitting it.  
Obviously he did. It has been produced all over the  
world. Certainly in Australia, in every lab, I think,  
it is produced in continuous cell lines. We transmitted  
it readily, not all of them, but most of the HIVs we

transmitted, and the six of them - in the very first  
paper there are six, if I don't miss my count. Maybe it  
is five. I think it is six. Certainly we transmitted  
it all right. I think Montagnier did from getting blood  
from a number of donors. He had to keep the thing going  
for a while. That is relative to the amount of virus.  
That was the problem. I take nothing away from his  
contribution.

Q. Would you agree with the, I think, well-known  
retrovirologist George Todaro that 'All cells in culture  
sooner or later, under the right conditions, will  
produce retroviruses'.

A. No. That hasn't been documented by anyone, including  
Todaro. It is absolutely untrue. What he, I think, is  
referring to is that all creatures have archaic  
remnants, fossils, of ancient infections of retroviruses  
which get into our DNA, so you have DNA, the genes of  
these things, sometimes the genes are very little and so  
you can't make out anything, you just make an occasional  
protein once in a while. We have a few that look to be  
full length that can give rise to virus particles that  
stay with humans and not with a mouse, whether inbred to  
do exactly what Todaro was talking about, and in humans,  
if you - the only endogenous retrovirus particles known  
are HERV-K: human endogenous retroviruses K. They make  
particles. No-one has transmitted it. They have  
morphology; they can't be transmitted. They are  
irrelevant to everything you are talking about. They  
have never been found, as I said three times, in normal  
human lymphocytes, in normal blood cells. If Todaro  
ever found those viruses in normal human lymphocytes, he  
would have published five papers on it. He has  
published none. I grew up with him. He worked at NCI.  
What I mean by that, we were colleagues in NCI for  
almost 20 years. We published together several times.

Q. I want to ask you a few questions on reverse  
transcriptase. Isn't it a fact that, in 1988, in the  
scientific American paper you co-authored with

Montagnier, you described how Montagnier took the lymph node of a patient, mixed it up, cultured it for two weeks, at which stage he detected reverse transcriptase activity.

A. I don't remember, but obviously if Montagnier has dated that, yes, I signed it. I believe what he wrote. We wrote together, we wrote it in portions, we both agreed on the parts the other guy wrote and published it together. Basically that is his statement. He is the one who did it.

Q. You said in that paper a retrovirus was present, but which one, and the question I'm asking you: does the detection of reverse transcriptase prove detection of a retrovirus.

A. If it is in a particle - with definitive reverse transcriptase that you distinguished, I spent no less than nine years on distinguishing reverse transcriptase from the normal DNA proliferations of human cells and I distinguished, by purification, the enzyme of about a dozen different retroviruses from about half a dozen different species. Along with David Baltimore, I think we contributed the most to what the current assays for retroviruses by reverse transcriptase are today. Baltimore discovered reverse transcriptase. In other words, I worked hard at making sure we had assays, then Montagnier used the assay from a previous paper from mine. The answer to your question: no. To make it a virus, you have to show it is transmitted. I have said this before, maybe 10 times today - tonight, in my place - if he didn't transmit it, I would not call it a virus, but he transmitted in the paper. I would be glad to take all credit and say he didn't and we are the ones who transmitted it, but he showed transmission through normal blood of another donor. In other words, he had supernatant containing particles from the patient, he added it to blood of a normal donor, blood cells, and he could continue propagating these particles to another donor, to another donor, to keep it going. That is good

news, it is transmissible, it makes it a virus. It is bad news if you have to do it that way, you get a lot of cell debris, hence, pictured by electron microscopy, and the immunology would be difficult to do. That is why they didn't have a good assay. That is why they didn't win the patents and we did; because they couldn't reduce it to practice.

Q. Is reverse transcriptase activity specific to retroviruses.

A. Do I have to answer that? We just talked about it.

Q. I want to make sure I understand you. What I want to find out from you -

A. I want to go fast and we can all get the heck out of here and I can go eat. Let's say it again: the process of reverse transcription occurs in nature widely, even in yeast. The enzyme that does it is different. The enzyme that catalyses our DNA is readily distinguishable from a retrovirus than it is in the things that Varmus is talking about. These are so-called retro transposons, elements that may have something to do with our evolution and from shuffling genes in the course of evolution. That enzyme that does that is not a problem, it is not packaged and released as a virus, No.1; No.2, the characteristics of the enzyme are those things that have nothing to do with the characteristics of the enzyme of HIV. I told you before you have to be really stupid to confuse the two, beyond stupid. So the argument by the witness is because he doesn't understand.

Q. How could Montagnier say he has found a retrovirus in his cultures based upon his detection of reverse transcriptase activity. That is the question we want to put to you.

A. It is in a particle, he took a picture, it had particle form, we acknowledge not a great picture, but enough to say it is a virus, and if it is a virus with reverse transcriptase, it belongs to the retrovirus family, with rare exceptions, rare exceptions. The kind of reverse

transcriptase he described had me convinced it is the  
kind that is only found in retroviruses. That is where  
I completely disagreed with the electron microscopists  
giving him problems at the time and took his side right  
away. We all are in total agreement, including the  
electron microscopists that were critical at the  
beginning, because the characteristics of a retrovirus  
reverse transcriptase are completely different than the  
characteristics of the catalysing reverse transcription  
among retro transposons that Varmus is referring to.  
In addition, Montagnier showed the virus was  
transmissible, it was not a particle that was indeed a  
virus he could put into normal human blood cells and  
keep transferring it. Can you go on to another question  
because it is driving me nuts with this?

Q. In that answer you said that Montagnier described to you  
what he had seen. What did you mean by that. Are you  
basing it on something he told you or are you basing it  
on something else.

A. Basing it on the paper that I reviewed. I'm basing it  
on the paper I reviewed and, of course, on talks and  
communications between us. We don't just make things  
up, I tell him something and he makes something up and  
tells me something.

Q. What did he describe to you.

A. Do you believe what the judge tells you? Do you believe  
it? Do you have colleagues in law, do you believe them?  
We were collaborating for a while in our careers. Yes,  
I believe what he tells me, and I read his paper; both.

Q. What did he describe to you that convinced you this was  
a retrovirus, and apparently a unique retrovirus.

A. There are two matters here. Why you are still focused  
as if the world stood still when he published - I know  
what I published. Let's deal with what I published.  
You find someone who can argue with me about reverse  
transcriptase and its characteristics, find me anyone, I  
would be glad to deal with them. Montagnier's paper had  
reverse transcriptase in a particle that had structural

morphology compatible, in my eyes, with a retrovirus. 1  
 That has obviously been proven to be the case today. I 2  
 was convinced he transmitted it in vitro. It is not his 3  
 best paper, it is obviously not the best of papers. He 4  
 was new into retrovirology, I was old into 5  
 retrovirology. I would have maybe done a few more 6  
 things before releasing it, but release it he did, and I 7  
 think he was right to do so. That is why I accepted it 8  
 for publication. You ask me this question. You know I 9  
 reviewed the paper. If I reviewed the paper, I was 10  
 convinced. If I was unconvinced, do you think I would 11  
 give him a gift ahead of me? I'm supposed to be 12  
 competitive. Would I be tempted to find criticism of 13  
 it? I thought it was important. 14

Q. David Baltimore, who is a Nobel laureate, and is a 15  
 well-known retrovirologist and a discoverer of reverse 16  
 transcriptase, quite clearly says - 17

A. Which I just said one minute ago. 18

Q. - he says that reverse transcriptase activity is not 19  
 specific to retroviruses, then he goes on - this is what 20  
 I want to put to you - he, as I understand it, says 21  
 about 50% of our DNA is obtained by reverse 22  
 transcription of our RNA. 23

A. Yes. We just talked about that, transposal elements - 24  
 irrelevant to the case, irrelevant to the virus. David 25  
 Baltimore is probably the senior most hostile person to 26  
 your position in the world. You quoting him - I won't 27  
 make an allegation - he would be pretty excited. It is 28  
 the same thing you brought up twice before. These are 29  
 retro transposons. They definitely are used in 30  
 amplification of DNA. You don't find it in normal human 31  
 lymphocytes going on. You don't find it in AIDS 32  
 patients going on. It isn't packaged in a particle that 33  
 is released and is infectious. It's activity, for the 34  
 last time. I won't answer the question again. You have 35  
 to give me a break here. I'm not going to answer this 36  
 again. Reverse transcription is a process by which RNA 37  
 is converted to DNA. Yes, it goes on. It is not 38

released as a particle, that is point 1; point 2, the  
enzyme that does it, the enzymatic activity is quite  
distinct that only a fool would mistake it for  
retrovirus reverse transcriptase. It has different  
properties. I said that just before and before that and  
I'm not going to say it again. My answer is on the  
record.

CONTINUED



- Q. I want to put this one question to you. Is it true that  
around March 1984 Francis and Jim Curran of the CDC  
arranged a blinded test of HIV antibody test using tests  
from the Institute Pasteur, your laboratory and their  
own test. Is it true that a number of samples that the  
Institute Pasteur recorded as positive were recorded as  
indeterminate on your test.
- A. I honestly don't remember details but something like  
that is probably true. I thought that the - see they  
weren't doing the Western blot there and they hired  
somebody out of my lab to help them, which didn't create  
friendships very much. Basically in any event they were  
doing ELISA and the Western blot and they were making  
it - we thought that the cut-off for the ELISA - the bar  
was too down so there would be too many false positives,  
so we had agreement on some that were not verifiable by  
Western blot, that is true.
- Q. My instruction is that after you got their positive  
tests back your institution changed the results which  
you had previously had as indeterminate to positive.
- A. I don't - I don't recall that being the case but if we  
did we made the sensitivity better and returned to the  
Western blots that's perfectly plausible, I don't know  
but you have to recall that Montagnier in his pattern  
had only 17% of AIDS patient positive in that period and  
he went up to about 30% by early 1984, that's in all the  
publications. But it was a matter of him being able to  
get virus in cell lines. In May of 1984, or March 1984  
rather we gave him the virus producing cell lines from  
our lab and they then had virus in considerable amounts,  
and clearly their test knowledge would improve. Then an  
English scientist groped one of their isolants for him.  
Eventually they succeeded. If we went back and found  
some indeterminates to be positive, it would be we did  
the Western blot with greater sensitivity and more care.
- Q. At that stage, 1984, is it true that only 48% of your  
test results from people were AIDS were positive using  
your testing methodology.

A. You asked me that before. I don't remember the exact number but it is true that people with AIDS lose antibodies towards the terminal part of the illness so that explains sometimes - did you say antibody testing? No, antibody testing was between 88 and 100%.

Q. The expression I used was your testing methodology at that time in 1984.

A. No, in March 1984 I went to a restaurant in Bethesda with Jim Curran and he handed me an envelope and I handed him an envelope. They were coded samples. When we opened the letters up he looked at me and he said 'It's all over'. When you went to that restaurant I was convinced I knew the cause of AIDS but when I came out I was happy because I knew from Jim Curran that he knew the case of AIDS also. That was always in the blood transfusion patients. According to Jim we had substantially higher than anybody else we hit. According to Curran it was over when we did it. So you know - what I objected to was Francis lowering the bar in areas that I thought were inappropriate and I don't remember that we went back and did what you said. It's plausible but I don't remember, I don't think so. If my colleague Dr Sandra Darren were here she would know that with certainty. There was no question that we had better testing. That's why the patterns went to us and that's why everybody uses the test to this day.

Q. Is it correct that in your first paper in 1984 you said HTLV-3 in this case is usually not detected. The bands are often faint. HTH-3 sequences are found really if at all in peripheral blood mono nucleic cells -

A. Yes, I don't know what paper you are referring to. It's not the first science paper. The first science paper is the continuous production in the viruses, the technical break-through that led to savings of countless lives on earth. That's the first paper. You are speaking of some other paper, I don't know what other paper but it's not the first paper. In fact no paper in the first four or five has to do with gene analysis with molecular

biology. Paper 1 is the continuous production, the  
break-through. Paper 2 is the 48 items. Paper 3 is  
Sandra Darren's where there was 88%. Paper 4 is  
detailed analysis of the viral proteins. Paper 5 is the  
paper in Lancet and that's 100% sero positive. What you  
describe is rational. So therefore I hope I said it  
somewhere. But it isn't in the first science paper.

Q. In 1994 did you say they have not been found in the  
tumour cells of Kaposi sarcoma.

A. Sure that's true. I don't know if I said it in 1984. I  
would have said it much earlier. It must have been in  
1988 or '89. I said that in early 1984. HIV is not the  
sine qua non of Kaposi's sarcoma. HIV markedly  
increases the incidence of it. Or the fungal diseases  
that occur in an immune expressed person. That is also  
misinterpreted and misrepresented and misunderstood by  
your witnesses.

Q. I have to finish that quote. It was 'We have never  
found HIV -

A. I agree with you. I agree with you. The answer is yes.

Q. 'We have never found HIV DNA in the tumour cells of  
Kaposi tumour. In fact we have never found DNA in  
T cells'.

A. T cells. What? In T cells?

Q. Yes.

A. Did you say T cells? I don't know where you are getting  
that from. Certainly in Kaposi's sarcoma DNA - the very  
first paper we published on molecular cells shows the  
T cells. What is difficult is if you get a tissue,  
let's say you get a lymph node. What is a lymph node?  
It's composed of many kinds of cells. Amongst them are  
T cells. Among them are CD4 T cells. It's very  
difficult to see the whole tissue. So you pull out  
those cells. But if you are talking about peripheral  
blood it's hard because it's not sensitive because the  
number of CD4 T cells is low in all of us. It is not  
the substantial percentage of our blood cells. 70% of  
our blood cells are neutrophils. 15% or so are - 30%

are lymphocytes. Sorry, 60% are neutrophils or 1  
granocytes as a whole. About 30% are lymphocytes. 2  
Maybe 15, 20%. Amongst those you are B cells, NK cells 3  
and CD4 cells. So it's a minor fraction of our - when 4  
we take blood cells out of our population. Added to 5  
that the person has - if the person has AIDS many of the 6  
CD4 T cells are low already and that makes it still more 7  
problematic. Added to that in any one moment in time 8  
HIV is infecting only a fraction of those CD 4 T cells 9  
because those cells are often destined to die 10  
prematurely. Of course you are looking for a needle in 11  
the haystack. Western blot was not adequately adequate. 12  
Or you have leukemia where every cell in the tumour has 13  
the viral sequences because the tumour is formed from a 14  
clone of a single transformed cell, so all its progeny 15  
contain the viral sequence. It's not HIV. It's not 16  
directly causing any cancer. 17

Q. Do you agree that the nucleic acid tests, that is the 18  
PCR tests, cannot be used to prove HIV infection. 19

A. I mean of course you can use it as a component of 20  
evidence. You couldn't use it to prove necessarily a 21  
virus. Let me point out to you that Baltimore at 22  
Chiron, I think his first name is David but I'm not 23  
sure, discovered HIV virus simply by doing subtractive 24  
PCR hybridisation. He took a normal liver and a person 25  
with hepatitis that didn't have hepatitis A or B and he 26  
did a subtraction, that which hybridised the normal DNA 27  
was thrown away. He found DNA extra in the hepatitis C 28  
liver. What was extra was found to be the hepatitis C 29  
virus. So PCR can be used to greatly - he did that with 30  
just DNA amplification. He found sequences in one that 31  
he amplified and he found that there was something 32  
beyond what is in normal human DNA liver. That extra 33  
DNA hybridised to the liver of a patient who didn't have 34  
hepatitis A or B. They purified that DNA. They get the 35  
sequences of it. It led to the discovery of the 36  
hepatitis C viruses. It depends what you mean. You 37  
take these things with some simple-minded story that you 38

use PCR to discover a virus, yes. 1

Q. If anyone's HIV, DNA is that HIV specific. 2

A. Is what HIV specific? 3

Q. Your HIV DNA, is that HIV specific. 4

A. There is no DNA in HIV. But you can make a DNA through 5  
reverse transcription. That's DCNA which hybridised 6  
back and, yes, it's specific. It doesn't go back unless 7  
it's been contaminated by something. Then you can clone 8  
it or again get rid of contaminants. You can clone it 9  
from a person's DNA and show that there is no normal 10  
sequences in human DNA. There are a series of a million 11  
publications that your witnesses don't seem to know 12  
anything about. 13

MR BORICK: I better let my friend have some time for 14  
cross-examination. 15

MS McDONALD: I have no re-examination. 16

ADJOURNED 1.20 P.M. 17

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