IN THE SUPREME COURT

CRIMINAL JURISDICTION

ADELAIDE

APPLICATION FOR LEAVE TO APPEAL AGAINST CONVICTION

BEFORE THE HONOURABLE JUSTICE SULAN

NO.65/2006

R V ANDRE CHAD PARENZEE

TRANSCRIPT OF PROCEEDINGS

TUESDAY, 24 OCTOBER 2006 AT 10.37 A.M.

MR K. BORICK QC, WITH HIM MR HEGARTY, FOR APPLICANT

MS S. MCDONALD FOR RESPONDENT

HIS HONOUR: Mr McKenney, I gather you want to put something to me.

MR MCKENNEY: Yes, very briefly. I have two applications. My first one is that my name be removed from the file, and provided that is granted, I seek leave to withdraw.

HIS HONOUR: I understand that Mr Hegarty has taken over as the solicitor.

MR MCKENNEY: I understand that is the case.

HIS HONOUR: Insofar as it is necessary, I will give you leave to withdraw. I don't think it is necessary but I will do so. It is noted that Mr Hegarty is now the solicitor acting for Mr Parenzee.

MR BORICK: I will be appearing with Mr Hegarty in the matter.

Just to outline what we are doing here, the defence will advance three basic propositions in this hearing: firstly, that viruses are proven to exhibit by a procedure virologists refer to as virus isolation. The presently available evidence does not prove a virus known as HIV has been isolated.

The test routinely used to diagnose HIV is not virus isolation. Infection is diagnosed indirectly by using antibody tests. At present, there are two major antibody tests used, the ELISA and Western blot. The

Western blot test is used as a supplemental confirmatory test because the ELISA is not specific. However, neither of these tests have been scientifically proven capable of determining HIV infection or transmission. In fact, the manufacturers of these tests repeatedly state 'At present there is no standard for determining the presence or absence of HIV in human blood'. Hence, in the absence of such a standard, it is impossible to say how many, if any, people who are said to be HIV positive are, in fact, infected with a retrovirus HIV. Nonetheless, the Western blot test is considered to be nearly 100% specific and is used as a confirmatory test.

However, according to Dr Elizabeth Dax, head of the Australian National Reference Laboratory, 'Confirmatory tests for HIV are sometimes called supplemental tests because they really don't confirm infection'.

It is also a fact that whether or not an individual is regarded HIV positive depends on the laboratory in which that person is tested. This is because around the world there are so many different criteria that define what a positive test actually is so that a person may test positive in Australia, for example, but the same test result may not be positive in Africa.

The third basic proposition is no evidence for sexual transmission of HIV can be found even in the best conducted studies published from the United Kingdom, Europe, United States of America and Africa.

HIS HONOUR: What is the second proposition, Mr Borick? I got the first one but I'm not sure where the second one arises or what the second proposition is.

MR BORICK: The second is that the tests used to in effect diagnose HIV do not do that. What they do is that they measure not the virus itself but antibodies. I will come to that in a little more detail later.

The experts to be called by the prosecution claim the fact that the antiretroviral drugs known as HAART dramatically reduce the mortality rate from AIDS proves HIV is its cause. However, in a major study of 22,000

patients published this year it was reported that improved reductions in 'viral load' which is said to measure the amount of HIV in the body does not decrease AIDS mortality. In fact, in the year that they reported the lowest viral loads, AIDS appeared sooner than in the previous years when the viral loads were higher.

In a second major study also published this year, the authors concluded that not HIV but 'other factors as yet unidentified likely drive CD4 cell losses', cause immune cell depletion that is said to lead to AIDS. This will be explained more on the evidence by the prosecution and, in fact, it has been long accepted that

mortality rates decreased.

The defence has not introduced and nor are we concerned with the issue of whether or not HIV causes AIDS. HIV and AIDS, although generally linked in the public mind, are two separate and distinct issues. In this case, what is important is whether there is any scientific evidence whether Mr Parenzee is infected with the unique virus HIV. The evidence and the arguments we will advance in support of the basic propositions are not new. In fact, they first surfaced shortly after the claim that HIV was discovered in 1983. The reaction from the relevant scientific community and the medical community is one of disbelieve. So far as we are aware, this is the first time the Supreme Court has been required to consider the evidence on this issue and to deliver judgment. The task of the court, as in any other case, is to evaluate the evidence and to utilise the experience of the law to arrive at a judgment.

The two witnesses to be called by the defence are Eleni Papadopulos-Eleopulos - we will refer to her as Mrs Eleopulos - and Dr Valendar Turner.

Mrs Eleopulos is a physicist. She is trained in the most basic of physical sciences. In round terms, that is physics, science, and the most important of all, mathematics. That science underpins biology. In turn, biology underpins virology. It follows that manner and

way in which the prosecution witnesses claim expertise is the same manner and way in which the defence witnesses claim expertise - that is, an understanding of the basic science involved and an understanding of the basic principles, research and experience.

Both the defence witnesses have been involved in the study of this issue since 1983, virtually 25 years. Your Honour has seen the fact that they have had a number of papers published but also the fact that a number of their papers were not published for reasons which will be explained to you.

Neither witness is an experienced expert witness in

court. In fact, for Mrs Eleopulos, this is the first time that she has given evidence. Because of in part some of the complex nature of the evidence which will be provided, a case will be presented more by way of a presentation with a minimum of involvement by me as counsel in the matter. Frankly, I will just get in the way of the flow of our evidence.

The evidence you will hear is that HIV is I think generally accepted to be not a virus but a retrovirus, but a virus is an intact fully assembled infectious particle and viruses are replicated inside specific cells. In this case, the claim is that the virus replicates inside human white blood cells called lymphocytes. A cell has the machinery to gather raw materials from the environment and from this construct a new cell in its own image. Cells and viruses are made up of the same biochemical constituents, the main constituents being proteins RNA and DNA.

When a virus particle leaves a cell and then enters a new cell in which it again replicates, it is said to propagate. This proves the particle is infectious and thus confirms it is a virus. The virus is too small to be seen under an ordinary light microscope but it can be seen and photographed using the power of an electron microscope. HIV is said to be a retrovirus which means it contains RNA and an enzyme which enables it to

transcribe its RNA into DNA. This process is known as reverse transcription. It was always thought that the process was DNA and then RNA and then on from there, but it has now been accepted that it can go the other way, RNA to DNA, which is where the reverse process comes in.

The first step in identifying any virus particle is to examine its appearance and size but what might appear to be a virus-like particle or retroviral particle does not mean it is a retrovirus. For example, cellular fragments may look like retroviral particles. In order to prove that what you are looking at is a new retrovirus, it is necessary to establish that the

particle is infectious and that it has unique proteins and RNA. To achieve this, one must isolate the particles from everything else which also contains proteins and RNA; that is, one must purify the virus particles. Unless that process is undertaken, it is impossible for anyone to claim that a new retrovirus, in this case HIV, exists.

The critical issue in this case is whether or not the presently available evidence proves that HIV has ever been isolated. If the answer is no, then HIV has not been successfully proven to exist. At this point, it would appear to us the only way for the prosecution to escape from that dilemma is to argue that isolation does not matter.

As explained earlier, there were the two test kits, ELISA and Western blot. The only way to have proof for the existence of such proteins is to isolate or purify HIV. The anti-bodies that are formed in our bodies that react with these proteins are assumed to be HIV antibodies. The problem, however, is that antibodies are well-known to react with many different proteins apart from those which led to their production in the first place. Immunologists have described the behaviour of antibodies as promiscuous, which means there can never be any guarantee that a reaction in an antibody test is specific. This fact means that non-HIV

antibodies may also react in these tests.

The tests that are carried out, you can get a reaction which you can see that does not prove that that reaction is specific to HIV. So unless the virus which is said to produce the test kit proteins is isolated and used as a gold standard for comparison with the tests, it is impossible to relate an antibody response specifically to HIV infection. Without the establishment of a gold standard, there is no proof that the antibody tests prove HIV infection of humans.

According to Dr Elizabeth Dax, Western blot tests are to be reported as positive, negative or

indeterminate. She also states that 'at the completion of each run, reactions should be read immediately and then the strip can be dried and stored; that is a written record of reactions, including intensity, or a photocopy of the strips immediately after completion of the assay must also be included'.

In the case of Mr Parenzee, that did not happen and, in fact, all we have is a test result which is said to be reactive and then confirmed positive by an unknown person and the record of the test was not kept.

One prosecution expert who specifically addressed the issue of HIV isolation is Professor French. He accepts that the findings of Montagnier and his colleagues in 1983 'may have had deficiencies' but we are not told what they are. Luc Montagnier is regarded as being -

HIS HONOUR: Mr Borick, you can assume I have read a lot of the material. I'm not saying that I understand it all but I have read it. I know who Professor Montagnier was and I have read the interview which was provided to me, so you can assume some knowledge on my part. It does not necessarily mean full understanding, but some knowledge.

MR BORICK: Just briefly, Montagnier, in 1983, discovered HIV. Our witnesses will be viewing evidence, in this case through Mrs Eleopulos, explaining to you

the experiments that he conducted and then to tell you what is wrong with it or the problems with it.

Our case will be that Montagnier probably conducted the best experiments that have yet taken place and we will challenge the type of testing which now takes place, but in the end result, it is obviously necessary for the court to understand what Montagnier did before we can move forward to the issue of isolation. You will see from the 1997 interview that Montagnier himself said 'We did not purify', meaning 'We did not isolate the virus'.

From the beginning to end, our case is that unless

the scientist can establish that isolation has occurred, then it is impossible to say that HIV has been scientifically proven to exist.

My first witness will be Mrs Eleopulos. Your Honour has read her qualifications and I won't go through them now. She will expand upon that a little in her evidence and in particular she will tell you of how her interest first started, which is when she was doing work in cancer research in about the time of Montagnier's discovery.

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She has done a huge amount of work, as your Honour has seen, since then on this issue. She will tell you of a meeting that she had with Luc Montagnier in Amsterdam in - I think it was the 1980s, late 1980s, and her description of that interview is a little important because it encapsulates what is the central issue in this case. I have explained to your Honour she will be dealing with the question of proof of existence of a retrovirus and isolation with the Montagnier test and some other technical matters which your Honour has seen referred to.

Dr Valender Turner will then deal with the antibody test, and his evidence will conclude with the proposition that the tests have not been successfully proven to be capable of determining HIV infection or transmission, and it is impossible to say how many of any people who are said to be HIV-positive are infected with and HIV retrovirus.

Mrs Eleopulos will then deal with the question of sexual transmission and she will review the various studies, the studies of a group of prostitutes in 1985, an Australian study known as Philpot over in Sydney, three European study groups, and from 1989 to 1994 one of which involved a large number of United States servicemen who had been serving in Germany and arrived back and then testing occurred with their partners in

the United States, and that is a significant one in understanding the way in which these tests have been done.

Most important is the University of California study of Nancy Padiue of 1987 to 1997 - the study took over ten years - and the study from the Rakai District of Uganda in 2001, and we have done a comparison of the Padiue results, I will call them, with the results of the Rakai, and you see a very close correlation that they come to the same fact: that there is no proof that HIV can be sexually transmitted. In fact, the studies reveal it is highly unlikely that HIV is sexually

transmitted.

Your Honour will have seen from the papers already provided to you, and what we are putting, that very important issues are raised, but from the point of view of this court, evidence will be given on those three topics. Your Honour will decide them within the context of this application and it is going to be a question of understanding the science, and see where the issues lie and see where we go from there.

I anticipate that the presentation of the defence case in-chief will take in the order of seven hours. We are able to be reasonably precise because of the way in which we are putting forward a presentation. It should be about that length of time. We have got a little bit of a problem with the lighting because Mrs Eleopulos is finding it hard to see on this wall. Has your Honour got in mind that we do a little bit of an experiment with the lighting while she sits there?

HIS HONOUR: No. The first thing that you need to identify to me is exactly which affidavits that you rely upon so that we can admit those in.

MR BORICK: Only two: the one from Valender Turner -

HER HONOUR: Valender Turner's is an affidavit of 6 April 2006 which includes a number of annexures, and the affidavit of Eleni Eleopulos of 27 April 2006, which is a short affidavit of ten paragraphs?

MR BORICK: Yes.

HIS HONOUR: Those two affidavits will be admitted.

MR BORICK: With the presentation of the slides and the photos, can we number those as we go along the way, or at some convenient time can we do them?

HIS HONOUR: Yes, we can number them as we go.

EXHIBIT #A1 AFFIDAVIT OF ELENI ELEOPULOS DATED 27/4/2006 TENDERED BY MR BORICK. ADMITTED.

EXHIBIT #A2 AFFIDAVIT OF VALENDER FRANCIS TURNER DATED 6/4/2006 TENDERED BY MR BORICK. ADMITTED.

MR BORICK: Would your Honour number the Luc Montagnier article and with it the response from Mrs Eleopulos?

HIS HONOUR: Do you have any objection to that?

MS MCDONALD: No.

EXHIBIT #A3 INTERVIEW BETWEEN LUC MOTAGNIER AND DJAMEL TAHI DATED 00/00/1997 TENDERED BY MR BORICK. ADMITTED.

EXHIBIT #A4 CRITICAL ANALYSIS OF INTERVIEW BY ELENI ELEOPULOS AND COLLEAGUES PUBLISHED IN A CONTINUUM, VOLUME 5 NO.2 TENDERED BY MR BORICK. ADMITTED.

MR BORICK: Would your Honour mind adjourning for five minutes - it will be no more than that - while we set up the new system?

HIS HONOUR: Yes. You let me know when you are ready.

MR BORICK: I will be five minutes.

ADJOURNED 11.04 A.M.

RESUMING 11.09 A.M.

MR BORICK: I have been accused of lots of tricks, but this is the first time I have been capable of keeping the prosecution in the dark, and I am sorry about that, but we will just do the best we can as we proceed.

MS MCDONALD: Can I be heard on that? I have raised it with my learned friend. It is very difficult to

actually see at the bar table at the moment. Your Honour still has your spotlights on overheard. If I am going to be sitting here for seven hours, I would like to be able to read my notes and cross-reference as we proceed. As I understand it, the witness is saying she still can't see what is on the screen anyway.

MR BORICK: Can we just get started with the first part of it and then we will get this fixed up as soon as we can?

HIS HONOUR: Mr Borick, I think it is important that we get the situation sorted out. Ms McDonald, firstly, do you say you would like some more light?

MS MCDONALD: Yes.

HIS HONOUR: I am just wondering if there is some way in which we can provide a desk light which will enable you to read.

MS MCDONALD: I am content with that.

HIS HONOUR: Is that possible?

SHERIFF'S OFFICER: I will see what I can do. I will have a look.

HIS HONOUR: There are probably desk lights about. In fact, there is one in my chambers which we could get, if necessary, so all we need is an extension cord so Ms McDonald can actually have a light so she can read her notes.

MS MCDONALD: Before we actually go to that length, as I understand it, the reason the lights are off is because the witness couldn't see what was on the screen. I must say I was in here when the lights were on and there was no obvious difficulty to me. As I understand it, the witness still can't see the screen with the lights off.

WITNESS: I can see it but I cannot see it well. I would like to be able to see it better in the court.

MS MCDONALD: I am happy with the latter.

HIS HONOUR: I think we will try and organise a desk light for you so that you can have a light for yourself.

MS MCDONALD: Thank you.

HIS HONOUR: So that is the first thing. Madam Sheriff's Officer, do you need me to leave the bench while you do that?

SHERIFF'S OFFICER: No, I can probably look after that while we are still sitting.

HIS HONOUR: Ms McDonald, can you manage for the moment?

MS MCDONALD: Yes, I can.

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MR BORICK CALLS

+ELENI PAPADOPULOS-ELEOPULOS SWORN

+EXAMINATION BY MR BORICK

Q. You have a degree in nuclear physics from the University of Buchuresti in Romania.

A. Yes.

Q. Were you born in Romania.

A. No, I was born in Greece.

Q. And what took you to the Bucharest.

A. I went to study there because in Greece there was no faculty of nuclear physics.

Q. Could you outline what is involved in obtaining a degree in nuclear physics. What is nuclear physics.

A. Nuclear physics is studying the most basic composition of matter and it involves the then explanation of how matter is not only the composition but what is the fraction of matter. And it is the most basic of sciences. It tries to explain physics and 'physician' originates from the same Greek word and they are really - they are the scientists who study nature. 'Physics' in Greek is 'nature'. So, that is what physics does, it studies nature.

Q. What year did you obtain that degree.

A. I obtained the degree in 1960.

Q. And following graduation, you migrated to Australia.

A. Yes.

Q. And in 1996 or thereabouts, you worked as a laboratory attendant in the Department of Public Health and during this time you studied English.

A. Yes, I didn't know any English. I studied other languages in Romania but not English. So when I came to Australia I studied English.

Q. So since the early 1970s you have engaged in early biological research.

A. Yes.

Q. Would you in your own words explain to his Honour what research projects you were involved in.

A. Really when I start working, I initially as I said I was

working as a laboratory attendant and then after a few years after I learn English I was in the position of as a physicist and initially it involved to do a lot of routine work in the Royal Perth Hospital, then, the department of medical physics where we were studying and treating patients with cancer and other diseases. So, I was coming in contact with patients and I was doing a number of routine works of routine tests with patients. In about mid 1970s, a Dr Holt in Perth with the then premier, they bought a machine which was made by a physicist in Germany to treat cancer and I was asked to evaluate the physics part of the machine. But since the machine involved treating cancer patients and I knew nothing about cancer at that stage or biology for that matter, I thought if I studied two system and I know nothing about one and no matter how much I know about the other I wouldn't be able to come to any conclusion. So then I taught myself biology and that's how my interest in biology started and by the end of 1970 I put forward a theory of cancer and which was published in a small journal, an abstract of it, and then in 1982 was published in one of the most prestigious journals in biological research called the Journal of Theoretical Biology with good reviews.

Q. This is the theory of oxidisation.

A. No, this is the - not the theory only of cancer. To say

what cancer is you have first to find out what a normal cell is. That was my view. I have to know what a normal cell is and how I started to know that, because the most important - or one of the most important properties of a cancer cell is rapid division. So, then I said 'Let's see why do we have this rapid division' but to answer why this normal cell suddenly start to divide so rapidly, I thought I have to find out what division is, why a cell divides. So, I was looking at totally basic principle of cell division and to study that I want to study fertilisation to see what is - what property the ova has, you know, property the sperm has

which make the two when how first of all why they come, why the sperm combines with ova and again, from basic principle. And then I come with a theory when doing this, I came with a theory of normal biological function. So, it was cell - a theory of cellar function but the course was - it was not cancer it involved the theory, make prediction about not only about cancer but other basic or other diseases, chronic diseases for example like cardio vascular diseases, diabetes and made prediction about it. The prediction about cardiovascular diseases was proven in other departments with the help with the professor of neurosurgery and these papers were published, again, in the journal but at that time AIDS appeared and so because the AIDS -

Q. What time was this.

A. That was at the beginning of 1980s. Our experiments were done at the beginning of the 1980s and published in the mid 1980s.

Q. Montagnier and Gallo themselves were involved in a similar sort of work at that stage were they not.

A. All the age - the main age of the experts, that is the discoverer of what is now accepted to be discoverer of HIV, which was Montagnier and the person to have proven Montagnier, what we have discovered we have proven the second time the existence of HIV, Robert Gallo, they are all doing cancer research in that time. In 1970

President Nixon declared war on cancer and some of the researchers were trying to prove that cancer is caused by a virus and this included Luke Montagnier and Robert Gallo but unfortunately they are - their efforts were not successful and by 1980 when, 1981, I say when the first cases of AIDS were diagnosed in gay men, there were two main diseases. One of the diseases was pneumocystis carinii, that is a special lung disease, and the other was kaposis sarcoma which is a malignancy and many times infests on the skin and you can see. So, because both sides, that is Montagnier and Gallo and I were involved in cancer research, they came out, I mean

both groups with bias view, I came with my view on cancer which had nothing to do with viruss and Montagnier and Gallo said these patients have cancer of malignancy so maybe it is a virus and why they came to the conclusion it was a virus was because the gay men who develop kaposis sarcoma were very promiscuous so logically they saw that this may be a cancer which is caused by a virus. On the other hand, with my view on cancer I came out with a totally known infectious theory. So, you have the two theories were put in parallel from two different points of view. They made prediction about AIDS on the virus theory of AIDS and I made prediction on the non-virus theory of AIDS. So, the two theories had been going from the very beginning in paradigm.

Q. We will come back to that a bit later in the evidence. But now, in 1983 when Montagnier said he discovered HIV, what happened after that. From your point of view and from his point of view, where did it all lead.

A. When Montagnier discovered he publish his discovery of HIV in '93 there was not much movement, shall I say, nobody - there was not accepted straightaway but then in 1984 Gallo claimed to have discovered HIV as well and then even Gallo published four papers in the Journal of Science and even before the paper was published there was a meeting where they declared that they discovered

the cause of AIDS and that was a virus called HTLV-3 which now is known as HIV and since in press conference more or less immediately everybody accepted that HIV was the cause of AIDS and it was very hard for anyone else to publish anything which contradicted this theory. Nonetheless, I did manage to publish a paper which was rejected twice by the Journal NAJA, once by the journal Medical Hypothesis and ultimately after I responded to Medical Hypothesis they publish it. That was in 1988.

Q. Now, have you ever met Montagnier.

A. Yes, I met Montagnier in 1992. Before even I met him, I wrote to him and in 1991 I send my papers to him when I

have publish on AIDS then and he responded immediately and he thanked me for sending my papers and he said 'I will come back to you as soon as I study them carefully'. Well, I still don't have a response from him.

Q. But in 1992 you met him.

A. In 1992 there was a few individuals from Amsterdam organised a meeting between the two sides. That is, the people who believed that or who expressed view that AIDS is not caused by an infectious agent, that included me, and the people who believe that AIDS is caused by an infectious agent and that included Montagnier and we have about a week of discussion and -

Q. This was in Amsterdam.

A. In Amsterdam. But Montagnier came only one morning there directly from the airport and that morning the meeting was chaired from the HIV experts from Amsterdam and Montagnier and once the morning session finished Montagnier returned back to Paris straightaway but in the meantime everybody, once the meeting finish, everybody wanted to talk to Montagnier. Of course I wanted to talk to Montagnier, in fact that was my reason for going there but when I see him he didn't want to talk to anyone, I just stood there and he came to me and he said 'I want to talk to you'. I was very surprised but he said 'I haven't got time, you come when I wait

for the taxi, we are talk'. So, I said 'Well, let me introduce myself' he said 'There is no need, I know who you are, let's talk' and I said 'Let me start, I want to know what you mean by HIV - what you mean by the detection of particles in the culture'. He said 'Yes, but as a professor this is not specific, it doesn't prove HIV' and I said 'Yes, you're right and he said 'Then your description of activity' and I said that, important proven HIV'. He said 'Your right'. He said 'The only evidence we have for the existence of HIV is the detention of a protein in the culture, the P24 protein'. And I said 'Well, professor P24 is not

specific to HIV'.

OBJECTION: MS MCDONALD OBJECTS.

MS MCDONALD: I have to object. If it is going to be the basis for some further evidence about this witness and she has gone and done some further work so be it but if it is going to be suggested that in some way the defence can rely on something another witness has purportedly said to this witness, it is rank hearsay. I have let it go for a while but where it is going just escapes me for the moment.

HIS HONOUR: I am not sure either but Mr Borick, I am not sure what you intend to rely upon from this evidence because clearly anything that's purportedly said by Professor Montagnier to this witness is not evidence, it could never be evidence.

MR BORICK: No, I am still going through the basic qualification process which I am just about finished and the fact that she has a peer relationship with a man is all I am saying. But as I said -

HIS HONOUR: It is going to purportedly to the qualifications of the witness.

MS MCDONALD: Conversation at a taxi stand, I won't -

HIS HONOUR: Let's let it go and we will see where he goes.

MR BORICK: As I said in the opening address, it is a conversation that only took a few minutes but it

encapsulates what this case is about, and both your Honour and my learned friend will hear a lot more about a protein called P24 before this is all over and it is important to see right from the outset when you look at Montagnier experiments and Gallo experiments, this protein P24 is of great significance. Anyway, I think we just finish the conversation.

HIS HONOUR: I am not sure we had.

XN

Q. Did we get it finished, did we get a response from Montagnier.

A. I said it 'It is not' and he said 'I don't know that'.

And I said 'I will send you the evidence' and I came back to Perth and I send to him the evidence but Montagnier never responded.

Q. I want to turn to the presentation of your evidence and it is necessary first for you to give the court a few definitions, is that correct.

A. Yes, that is true because I am going to use some terms which may be not familiar to everybody so maybe it would be better if I -

MS MCDONALD: Can I indicate I don't accept the expertise of this witness to give the evidence she is purporting to now give. I also don't want to slow this process down.

HIS HONOUR: I was going to say to you, might it not be better for me to take the evidence if necessary de bene esse and you can then cross-examine the witness both as to her evidence as her expertise in due course and if there is an objection to her expertise I will deal with that question at the conclusion rather than try and split the whole thing up.

MS MCDONALD: That was going to be my suggestion to your Honour but I thought I should just flag it at the outset so there is no misapprehension.

HIS HONOUR: Yes, I recall from something said on a prior occasion that expertise may be an issue. Mr Borick, I think that's the best way.

MR BORICK: I was going to suggest that as being the sensible way out of it.

HIS HONOUR: We will proceed that way.

MR BORICK: The law is pretty clear, you have got to judge the issue, not what the prosecution experts said in their reports, it is your job.

HIS HONOUR: No, I understand that.

MR BORICK: So it is better you hear it. It is a very sensible way.

HIS HONOUR: There is a threshold question but I can decide that when I am deciding on the merits of the matter.

MR BORICK: With respect, a sensible approach to it.

XN

Q. Now, just to get us started, the definition of a virus.

A. Now, first of all the main property of virus is that they are particles but they are very small particles which cannot be seen by the light microscope. So, to study them you need the electron microscope. Now, under the microscope.

MR BORICK: Just pause for a minute. I am making an assumption that you are familiar with the concept of an electron microscope from other cases.

HIS HONOUR: Yes.

A. So with the electron microscope some viruses look like the one we see in this light.

HIS HONOUR: I think we probably need to identify these slides so that if this matter goes to another court they will at least know what we are talking about. Now, I have got a number of sheets numbered 1 to 15 with slides on them. Do you have the same document that I have got?

MR BORICK: Yes, I do.

HIS HONOUR: I am just wondering whether we could mark the document which is a series of slides. We will mark that A5.

EXHIBIT #A5 TWO SERIES OF SLIDES, ONE CONSISTING OF 89 INDIVIDUAL SLIDES AND ONE CONSISTING OF TEN INDIVIDUAL

SLIDES TENDERED BY MR BORICK. ADMITTED.

MR BORICK: A5 and then definitions.

HIS HONOUR: And could I just work out the methodology. Do the slides work from the left to the right as you move down?

MR BORICK: Yes.

HIS HONOUR: So we will mark each slide so the definitions are marked A5(1) and then now the slide that is now on the screen is slide 2. All right.

MR BORICK: We will keep our own record.

HIS HONOUR: I will just check that my associate is

doing the same as I am doing. So, my associate will mark them as they come up Mr Borick.

A. That is, as I said, some virus look at they appear in that slide. Now, cells. Cells are also particles but much larger than the virus and -

HIS HONOUR: I am sorry, I have to stop you but I have to get this on the record. Witness is now referring to a slide marked 3.

A. Now, this is what a cell looks like. As I said, it is a particle and you can see it is a particle but it is much larger and can be seen with the light microscope.

HIS HONOUR

Q. In order for me not to keep stopping you, when you change from one slide to the next, can you just say 'I am now looking at slide No.4', this one is 3 so the next one will be 4. So, if you identify the slide by number then we can then keep this on the record. Thank you.

A. The next slide which is slide 4 is still the cell and I define what a cell is. A cell is the smallest unit of heredity. The cell has the machinery to gather raw materials from its environment and to make an identical copy of itself. Cells have organelles and sometimes they can look even like viruses and this is like 4. I go to slide 5. Slide 5, it shows that unlike cells which can get the raw material from the environment and make an identical copy of themselves, viruses cannot do

that. The virus haven't got that metabolic machinery. So for a virus to multiply a virus has to work in the cell. There it is, a virus come. Because of the light you cannot see there are some viral particles which they assume to go into the cell, they are in the cell. They multiply. Some virus destroy the cell and they come out. Other viruses like a retrovirus they don't destroy the cell, they butt the surface of the cell and then come out.

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So for these particles to be viruses, they have to be able to propagate, that is to be transmitted from person to person or from cell to cell. So the virus particle comes out from one cell, they go into another cell and in the other cell they happen the same thing and they come out from the other cell.

Q. When you say a retrovirus -

A. I will -

Q. You will explain that later, will you.

A. Yes, sorry.

Q. You just explain it as you wish.

A. The next line is line 6. Now, if you want to see that if a cell is infected with a virus, then you put the cells into a tube and you put everything - a test tube - and you put everything which is necessary for the cell to survive and then after a while you will look in that culture fluid around the cell which is not supernatant, and if the cell is infected the particles will come out in the supernatant.

HIS HONOUR: That is in the bottom right-hand corner of p.1.

MS MCDONALD: It is not on my copy. I think there appear to be other versions.

HIS HONOUR: I will just hand down what I have got, Ms McDonald, so you can have a look. If need be, we will have to get you an extra copy.

MR BORICK: I have got a correct copy for my friend.

HIS HONOUR: We are on p.2 of A5, slide number 7.

A. Slide No.7, it shows that cells and viruses are made of the same biochemical constituents. The main components of viruses and cells are proteins, RNA and DNA. All cells contain both RNA and DNA. Some viruses contain only RNA. Now, the building blocks of proteins are amino acids. Slight 8 shows proteins which make the building blocks are joined together by some bonds called peptides. Slide 9, some proteins have many functions in the cell. Some proteins are enzymes. An enzyme is a catalyst. In a catalyst is the stuff that accelerates

or initiates a reaction, a chemical reaction, without itself being changed at the end of the chemical reaction. So the enzyme acts on the reactants A, B and C and we end up with a different product X and Y. Measuring the product, it gives you the activity of the enzyme. The building blocks of DNA are nucleotides, and the nucleotides contain phosphates and sugars. The DNA is the mechanics and the sugars in the DNA are the deoxyribose. Apart from that, the DNA contains four bases, thymine, adenine, guanine and cytosine, the basis of the protein to pair, and it is always the specific pairing. G pairs all the time with C and A pairs all the time with T.

HIS HONOUR

Q. That is slide 10.

A. Now, slide 9 shows -

Q. No, this will be slide 11.

A. Slide 11 shows the RNA. Now, RNA has more or less the same composition as DNA with some changes. There is only one polymer. One of the bases in DNA, the thymidine in RNA is uracil, and the sugar, instead of being deoxyribose, in RNA it is ribose. Now, this is slide 12. Soon after the DNA was discovered in 1953 a theory was put forward which states, and which is known as, biological dogma. The theory states that the information in cells goes always one way from DNA to RNA

and from the RNA to the protein, that is, you use DNA to see if there is RNA, and that is called transcription. The RNA then is used as a template to synthesize proteins and that process is called translation. But, in the 1970s, some researchers discovered an enzyme and that enzyme can make the flow to go backwards. Instead of from DNA to RNA, it goes from RNA to DNA. You can use RNA as a template to synthesize DNA. It is called reverse transcription and the enzyme is called reverse transcriptors. This enzyme was found in some viruses which, until then, were known as ONCO viruses, and these viruses have only RNA and because the enzyme was found

in these viruses, the viruses since then which became known as retroviruses was discovered. The enzyme, researchers objected to this because he said that it seems that the enzyme is present only in this biological entity which he did not believe in. He was even then predicting that the enzyme will be found in another biological system. So, what reverse transcription does, slide 13, you have RNA and you put all the building blocks for DNA and if you have the enzyme there you end up with the DNA, and the amount of DNA measures the reverse transcription activity. Slide 14, what everyone has to do to prove the existence of the retrovirus, first of all, you have to culture the cells which you think are infected with the retrovirus and then you have to demonstrate that in the culture fluid, in the culture sugar, sooner or later particles are released which have the morphology of retroviruses. The particles have to have the reverse transcription. These particles, when put into another test tube, would produce the same particles, that is particles which have the same morphology, and the same proteins; that is, you prove that the particles are transmitted, can replicate that is the particles that are infectious. To prove that it is a new retrovirus, then you have to show that the particles which are released in the culture have proteins and RNA which are unique to them and they are

not found in any other retrovirus particles. Slide 15, now, what is the evidence for the existence of HIV? Now, as I said today, everybody accepts that Montagnier, in his thing - in fact, there are 12 authors, I think - the principal author was a lady called Barre-Sinoussi, the second author is Jean Claude Chermann, the principal and the second author, and the last author Luc Montagnier was the coordinator of the team. So now it is asserted that Luc Montagnier and his team have proven the existence of the HIV. The paper was published and was entitled 'Isolation of activity for topic retrovirus if a patient at risk for acquiring immune deficiency

syndrome. That is AIDS. The gentlemen is known today as Bru. Now, when isolation -

Q. You have just referred to slide 15 and now you are referring to slide 16, are you; isolation, the next slide.

A. Now, this paper, I must point here that this paper, from 1983 until now, has been cited by at least another 4,000 publications and everybody cited this as being the paper of where the existence of AIDS has been reported. Now, this is slide 17.

Q. No, it is 16.

A. Now, isolation, as I said, the first word on the title is 'isolation'. Now, by isolation, according to the Oxford dictionary, it is meant to make into an island. It comes from the Latin and it means as a place apart or alone, separate, a substance from everything else, from a mixture, but this is not apparently what Luc Montagnier and his team meant by isolation. Now, slide 17. Luc Montagnier and his team published three main experiments so let us take each separate. In the first experiment, he took the lymphocyte from the limp nodes of his patient, that is normal lymphocytes, and he took two lymphocytes from Blu's lymph nodes, put them in culture - put everything which is necessary for these cells to grow and many other chemicals, including a chemical pha, which is an extra protein. After about 15

days in the fluid, in the culture fluid, he would take the reverse transcription. Now, as I said before, the main characteristic of viruses is particles and you can see them with the electro microscope. Montagnier did not publish any picture of what he had in the culture but just by the taking the reverse transcription activity, he said the culture was producing virus and he said the retention of the reverse transcription activity proves detection of retrovirus. Now, that is, we can see the detection of the reverse transcription activity proves detection of the retrovirus if, and only if, reverse transcription cannot be found anywhere else.

That is only specific to these viruses, to retroviruses, but this is not the case. This is slide 18. Now, let me quote from Harold Varmus who is a retrovirologist. That is a biologist who studied, who investigated, the relationship between viruses, who is doing cancer research. He got a Nobel for his research on cancer and viruses; in fact, oncogenes. Now, let me quote, as I say, from Varmus. He says 'Reverse transcription is hardly unique to retroviruses; it is now recognised as a widespread phenomenon in eukaryotic cells. That is in our cells, human cells. 'Evidence has made it clear that reverse transcription takes place in the uninfected cells in yeast, insects and mammals'. So, according to Varmus, reverse transcription is not specific to.

A. Retroviruses and that means detecting reverse transcriptase activity. Just detecting reverse transcription, the culture you cannot say is proof for the existence in the culture of a virus or a retrovirus. Now, slide 19, the people who studied the origin of life, according to them, or lately, they say that what was first was RNA, then DNA, came. So the DNA was made using RNA as a template. In fact, today many molecular biologists consider that about 40% of our DNA was obtained by reverse transcription of RNA. It is a known fact that many viruses contain reverse transcription activity apart from retroviruses including the hepatitis

B virus which a very high per cent of gay men and which intravenous drug users are infected with. So, too, bacteria. As far back as 1972 Gallo, who is, as I said, the second researcher considered to have proven the existence of HIV - as far back as 1972 he has shown that normal cells, if you put PHA in the culture and you put normal cell uninfected, they will start to reverse transcribe, that is, you take reverse transcription in your cells. At present even people who deal with shares and read their magazines will find out that reverse transcriptase activity is not specific to retroviruses. So, we can conclude RNA, reverse transcriptase, is not

specific to retroviruses. In detecting it you cannot say that you have a retrovirus there. In fact, Montagnier - this is slide 20 - Barre-Sinoussi, as I say, was the first author of Montagnier's 1983 paper and John Claude Chermann was the second. In 1973 they organised a meeting of the institute and the proceedings were published and even there they wrote - they found a reverse attributor at that time. As they say 'This enzymic activity can be explained by the presence of some virus particles in these regions, and since similar polymerase activity has been found in normal cells, may be mainly ascribed to the cellular enzyme'. So, in 1973 they knew that the enzyme can be found in cells and not only in retroviruses. In 1997 Montagnier gave an interview to the French investigative journalist, Djamel Thai, so he gave an interview to the French investigative journalist, Djamel Tahi. When initially Tahi asked him about the specificity of the reverse transcriptase activity, he said it is very specific to retroviruses tryptase, but later on when at the end of the day Tahi insisted Montagnier said it is being a characteristic of viruses, but there is a big difference between being characteristic and specific. Slide 21, for example, hair is a characteristic of human beings, but is not specific because there are many other animals which have also have that. So finding a hair somewhere

is not proof that a human being was there. Slide 22, we now have Montagnier's second experiment. In the second experiment Montagnier took Bru cells, so he took Bru cells and to them he added lymphocytes from a healthy blood donor and also added all these other things including PHA, growth factors, which is necessary for the cell to survive. Again, after an amount of time, he detected reverse transcriptase activity in this culture and he interpreted this as proving propagation of HIV from the Bru lymphocytes to the healthy blood donor

HIS HONOUR

Q. Just to make it clear for the transcript, Bru is a

person.

A. He is a person. He was a gay man, a French gay man. In fact, I think it was -

Q. You understood that Bru was a gay French man and it was his cells that were being used.

A. I don't know exactly what he -

XN

Q. Was he a gay French fashion designer.

HIS HONOUR

Q. You understand he was a fashion designer.

A. I did not know exactly what he was doing. He interpreted that the first experiment showed that Bru was infected with a retrovirus with HIV. The second experiment he said proved that the HIV from Bru was transmitted to the healthy blood donor cells, and that, he said, proved propagation of HIV in that HIV isolation, but again he did not publish any pictures. As I said, the main characteristic of viruses is they are particles. He still did not come up with any evidence for particles in either the first or the second experiment. This is slide 23. In conclusion, we can conclude from the first and the second experiment the finding of RT activity in Bru's cell culture was considered as being equal to the detection of HIV. Again, I am repeating. The second culture, the Bru cells, plus the healthy donor cells, it was proved for

isolation and propagation of HIV. This is slide 24. Maybe some people will object, given that Montagnier and Chermann were aware that - because, as I said, everybody knew that reverse transcriptase activity is not specific to retroviruses, including Baiid Ccsinousr and Chermann. They are fully aware that reverse transcription is not specific to retroviruses. Some people will think that we have misinterpreted their findings. Now, this is not the case as is obvious from a paper co-authored by Gallo and Montagnier and it was published in Scientific American in 1988. This has the history of - they discovered HIV by Montagnier. They wrote 'The specimen,

that is, the lymph node, the cells from the lymph nodes of Bru, was missed, put into tissue culture and analysed for reverse transcriptase. After two weeks of culture, reverse transcriptase activity was detected in the culture medium. A retrovirus was present but which one?'. So they definitely considered the transcriptase activity as proving that Bru was infected with the retrovirus and the virus was propagated or transmitted to the healthy blood donor cells. The question: as to which one? Montagnier tried to respond to show that what he found there was a new retrovirus; not only that he had that retrovirus, but was a new retrovirus, because at that time by 1983 there were two other human retroviruses known and they were HTLV1, HTLV2. What Montagnier wanted to show was that these viruses - HIV is a different virus, is a new virus, is not one of the old human retroviruses, no. So, how did they prove? Slide 25: maybe I should again interrupt and define a few things. First of all, the animal kingdom is divided into different categories. For example, humans belong to the same family who is gorillas and chimpanzees, but we are in different, genesis and different species. Slide 26, the viruses are also divided in families, genera and species. By definition particles belonging to the family of retroviruses are: 'Enveloped viruses with a diameter of 100 to 120 nm budding at cellular

membranes. Cell released virions, that is, individual virus particles, contain condensed inner bodies known as cores and are studded with projections which are known as spikes or knobs' and that from a paper by Hans Gelderblom, one of the best known Micron Microscopica in general and in HIV in particular. This is a diagram of what a retrovirus looks like. The main thing to see here is the diameter 100 to 120 nm and the particles have all these knobs on.

Q. Referring to slide 27.

A. Slide 28.

Q. This one is 27.

A. Yes.

Q. 28 is a slide headed 'Retroviral taxonomy'.

A. Yes, so the family is called Retroviridae. That is the family to which HIV belongs, or is said to belong, and the family is divided in sub-families which are oncoviruses, lentiviruses and spumaviruses. In turn, they are divided in geneses which is oncovirus type B, type C, type D and lentivirus belong to a different genus and they are called lentivirus.

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Now, let's now go to Montagnier's third experiment. In this experiment Montagnier took, from his second experiment, the supernatant from the second experiment and he also added it in the culture in these supernatant umbilical cord lymphocytes and after a while he detected again reverse activity. Now, this time he looked at the culture for his search for virus particles. And he found in this third experiment some particles to which he had some characteristics of retroviruses and they were both on the culture released and on the cells. Now, he did not have any controls. That is, he did not have umbilical cord lymphocytes, that is done in research. You always have a control culture. So, what he should have there is to have umbilical cord lymphocytes in another tube and to which he added everything else apart from the supernatant from the second experiment but he did not have that.

So, let's see what he found. This is slide 30 and as you see, this is Montagnier's picture and it shows this whole thing there, that is the cell and on the cell you can see some buds. And these, he said, are the HIV particles budding from the cell surface and these are HIV particles 3 release into the area. Now, he called these particles, he said these particles belong to the sub family of oncovirus and in fact there are specific type C particles. Typical type C particles. This is

taken from his paper and he had these in both. In the abstract and the text that they were type C particles. So did Gallo in 1984. Gallo also said that the names of particles are type-C particles.

Slide 31. However, in the joint paper which Montagnier and Gallo published in 1988 on the history of the discovery of HIV, they say that what Montagnier is seeing with the electron microscope, let me quote 'Electron micrographs of the new virus were different from those of HTLV-1. HTLV-1 is a type-C particle and resembled those of a retrovirus of horses'. The horse retrovirus belong to a difference family. It is a

lentivirus. So, suddenly, with no evidence in 1988, despite what Montagnier said in 1983, in 1988 they said that Montagnier has seen lentivirus, not type-C virus.

Now, slide 32. Let us see now where HIV is. Now, still even today there is no agreement as to what sub family or genous or even sub family HIV belongs. As I said, in 1983 Montagnier, in 1984 Gallo considered HIV, reported HIV as a type-C particle. In 1984 Montagnier and in the same year Levy, another HIV expert, said that HIV is a type-D particle. Since then, Montagnier and many others say that actually HIV is a lentivirus but there is still no agreement. In 2003 Kuznetsov reported HIV as a type-C particle and Elizabeth Dax in 2005 in her book says that HIV is a type-D particle. So, there is still no agreement as to which genous or even some family HIV belongs.

So, 33. This is no different by seeing one and the same thing and one is saying that it is a human then a chimpanzee and then a gorilla and then vice versa.

34. Now, so the first problem with what is called HIV particles is that even today there is no agreement as to what these particles look like. The second problem is that type-C particles are ubiquitous in biological systems or so they remain a mystery. They are found, as you can see there, in the main biological system and in fact in 1970 there were many reports in

many of these particles were found in human Leukaemia patients and cultured in embryonic cells and in the majority of human placenta. Now umbilical lymphocytes, that is the lymphocytes in Montagnier's experiment originates from placentas. Here it is, a retrovirus, a type-C particle from placentas. This is slide 35.

Now, the next slide, 36, shows the two, the placenta in Montagnier particles, one next to the other.

Slide 37.

Q. Just stopping there for a minute. If you can just go back for a moment. Just explain what we are looking at there please. A bit more detail.

A. Here it is. Let me find my way there. There it is, the placenta. This line shows the placenta-type-C particles and these are Montagnier type-C particles. So they don't look any different. Both are recorded as type-C particles. They are reported as type-C particles, the people who did the placenta studies and Montagnier reported them as a typical type-C particles.

Q. And the placenta photograph is from healthy individuals, healthy females.

A. The majority of healthy pregnant woman. The placentas of the majority of healthy pregnant woman has type-C particles, the same type of particles as Montagnier and Gallo reported in 1983 and 1984 reported respectively and Kuznetsov to have reported the HIV particles 2003.

Now, I think this is -

HIS HONOUR: Slide 37.

A. The third problem with the particles is that they are nonspecific. You can see particles that have all the characteristics of retroviruses but they are not retroviruses and they are not viruses. Cellular fragments can look like retrovirus particles. This was accepted by Tamin, the researcher who discovered reverse transcriptase activity and he got a Nobel for it and he was drawing the attention that you can have particles which look like retroviruses but they are not viruses. So did Montagnier sorry, so did Gallo in 1976 and he

said even particles which have reverse transcriptase can look like retroviruses but may not be viruses.

Now, there is a first problem with the particles. Cells may reproduce retroviruses spontaneously. That is -

HIS HONOUR: Slide 38. That's all right.

A. I feel embarrassed.

HIS HONOUR: You are a scientist, I am a lawyer.

A. Now, as I said, you put normal cells in culture and sooner or later they start to produce retrovirus and the cell can't be accelerated by the cancer condition up to a million fold. They are called endogenous

retroviruses. Now, why this is the case. If you have another virus you know if somebody detects a virus, and actually a virus in a person, then you know that that virus can say 'If I was infected with them, I should have come in contact with somebody else who had measles or who had Epstein-Barr virus infection and was transmitted to me'. Because, we have not got any information in our cells to synthesise viruses. This is not the case with retroviruses. Our cells up to about 10% in some people think even more than that, of our DNA is constituent information to synthesise retroviruses. So, if you have the right condition, if you put my cell in a test tube and you have the right condition, my cells will start synthesising retroviruses. Even if I never come in contact with anyone with a retrovirus.

HIS HONOUR

Q. What do you mean by synthesis.

A. Make.

XN

Q. And I am not sure you described or defined what you meant by 'endogenous'.

A. Endogenous.

Q. I am not sure, I might have missed that.

A. That's what I meant by endogenous. They are called endogenous because they are inside our cells. Exogenous means 'exo', from outside. 'Endo' means from inside.

So we have measles virus, Epstein-Barr virus they are exogenous. If you find in a person they came from outside. But if you find a retrovirus now, it may come from outside but may have come from endogenous. It was in-house it is always in our endo, it is in our DNA to synthesise these type of viruses so they are called endogenous viruses.

Q. Just explain what you mean by 'outside' in this context. Outside of our bodies.

A. Outside of our bodies. Yes, I mean as I said if I have Epstein-Barr virus or measles, then somebody else from -

HIS HONOUR

Q. Measles.

A. Measles. Now, according to one of the best neurologists and cancer researcher George Todaro, the failure to isolate endogenous viruses from certain species may reflect that an - of invito cocultivation techniques, that means in a test tube you don't have the right condition in a test tube and that is why you did not detect them. If you have the right condition no matter where the cells originated from, you will find retroviruses reflect the limitation of.

This is slide 39. Now, Professor Martyn French in his statement said that 'The first demonstration of a virus with retrovirus features that was subsequently shown to be HIV was reported by Dr John Armstrong and colleagues from Royal Perth Hospital in 1984'.

Now, 1984 when Dr Armstrong published his paper I suggested to him that what he seen there, to be fair to him, he did not say is a retrovirus. He say it is a retrovirus-like particle. So, I suggested to him to what he has seen there is not - may not be a retrovirus but may be a cell constituent which appear as a consequence of the disease because he has seen these retrovirus-like particle in the lymph nodes from AIDS patients.

XN

Q. Just get the timing of this right. Now, this statement

as we look at now by Martyn French, that is in his report he has given to the court just recently; is that right.

A. Yes.

Q. Now, he refers to some work done by John Armstrong at the Royal Perth Hospital in 1984.

A. Yes.

Q. And you and John Armstrong were colleagues at the Royal Perth in that year; is that right.

A. Yes. Our offices at that time were very close in fact.

Q. So you are now talking about a conversation you had with John Armstrong shortly after he had published his work

in 1984.

A. Yes.

Q. I am not sure whether that was clear. Would you mind just going back over that again.

A. So, yes, I went and saw him and I said 'Look, maybe what you are seeing there is not a retrovirus and maybe the result of the abnormality of the lymph nodes, some cellular product due to the disease's cells'. I said 'Unfortunately you did not have a test control. That is, that you did not study lymph nodes from patients who did not have AIDS but their lymph nodes were abnormal' and he said 'It is a pity but we could not do it because it takes a long time'.

OBJECTION: MS MCDONALD OBJECTS

MS MCDONALD: I make the same objection I made before.

HIS HONOUR: I will hear the evidence and how we deal with it can be a matter for further discussion.

HIS HONOUR.

Q. You go on.

A. Thank you. So, unfortunately this experiment was not done but as I predicted, four years later in 1984 - in 1988 the paper was published by the researchers from Harvard University and they have examined in fact this is the only study in HIV research which was blind and was controlled and they studied lymph nodes from AIDS patients, normal lymph nodes from AIDS patients and a

normal lymph node from non-AIDS patients.

XN

Q. Can would just go back and explain what 'blind' means in this context.

A. That is, the people who are looking with the electron microscope, they did not know the origin of the specimen, they did not know if they were from AIDS patients or they were from non-AIDS patients. That's what means 'blind'. The people who were examining did not know the origin and they report exactly the same particle with the same frequency in both. Patients with AIDS and patients with non-AIDS normal lymph node.

Q. What are we looking at there. Just explain, what is on the left and what is on the right.

HIS HONOUR

Q. This is slide No.40. Just take the left. What is that.

A. That is non-AIDS related.

Q. That is the cell looked under an electron microscope.

A. Yes, they are a specimen from the notes, and as seen in both of them, this is from an AIDS patient on the right and on the left from a non-AIDS patient.

Q. You are saying if you look at those slides, the two slides are the same particles.

A. The same particles, right, and they concluded - slide 41 - the presence of such particles do not, by themselves, indicate infection by HIV. Slide 42 -

HIS HONOUR: Did you want to say something, Mr Borick?

MR BORICK: No, I just got a bit distracted.

HIS HONOUR: We have just done slide 41.

MR BORICK: Perhaps I will just interrupt for a second. The lady sitting next to the witness is just assisting with the pushing of the buttons.

HIS HONOUR: I understood that.

MR BORICK: And because of her background experience in putting together presentations of this nature, she is not coaching the witness in any way at all or assisting in the giving of the evidence. She is simply there as -

ASSISTANT: She couldn't read what was on the screen.

A. I had a problem reading it.

MR BORICK: I just wanted to clarify that for my friend's sake.

MS MCDONALD: Can I indicate, I have no issue with the young lady sitting next to the witness box. My concern was there seemed to be a couple of private exchanges and it should be made clear what those are, if they are occurring.

HIS HONOUR: I understood the last private exchange was that the lady assisting the witness was reading what was on the screen. So we had 41 and now this is slide 42. Do you want to go back to 41?

MR BORICK: Would you mind going back to 41 for me please.

XN

A. The researchers from Harvard concluded the presence of such particles do not by themselves indicate infection by HIV.

Q. What I want you to just take a bit more time over is what is meant by 'such particles'. Perhaps you might have to relate that back to slide 40.

A. Well, the particles which they think - the particles, the same particles which they think which Dr Armstrong thinks, in the leaf notes from the patients, and Professor Martin considered them to be HIV, the researcher from Harvard says that these particles, such particles do not prove HIV infection and certainly cannot be considered as proof that Dr Armstrong isolated HIV in 1984.

Q. You are going to move on now to -

A. No, we have another problem now with the HIV particle and this is extremely significant more than any other, in my view. They all are but this is. Now, this is a diagram again taken from Gelderblom of HIV.

HIS HONOUR

Q. This is slide 42.

A. Slide 42. Now, the characteristics of the HIV as presented in this diagram are first of all the diameter.

I will start from the bottom. The diameter is 100 to 120 nM. Now, they have a cone shaped core. Notice there this core which is cone shaped. They have what they call lateral bodies, that one and this here (INDICATES), and most importantly, all the retroviral particles, to be infectious, they have to have these knobs on their surface. Slide 43, when you culture tissue, when you put in cultures tissue from AIDS patient, you don't see only this type of particles, you see many kind of particles. For example, you can see particles with a diameter from 65-250 nM. The one which has a diametre of 65-90 nM with knobs, the one with

100-120 nM have no knobs. The one with diameter higher than 120 nM again do not have knobs. You can have cores which are conic and you have cores which are tubular. You can have more than one core in one of the same particle. You can have different cores in one and the same particle and you can have particles which have no cores. Now, if there was core in particles of 100-120 nM HIV, and I say these originated from the AIDS patients which are infected with the virus, now what are all these other particles and what is their role and where is their origin? So, this is another problem. Slide 44, a sixth problem with the virus particles. In AIDS research, in HIV research, there are - by now, they don't use - HIV experts do not use cells originating from AIDS patients. They usually use cells which can survive for ever in their test tube and then they add some tissue which originated from AIDS patients. However, this cell lines confine virus particles, retrovirus-like particles, even when you don't add any tissue from AIDS patients. That is when they are not infected. So this is again a big problem. In fact, Montagnier, in his interview with Djamel Tahi, said in this kind of cultures you retroviruses, retrovirus particles.

XN

Q. You are now I think moving to the topic of knobs which

you spoke about.

HIS HONOUR: No, there is a seventh problem of particles.

XN

Q. You were going to deal with knobs there and the next five slides have to be considered together, don't they, as a whole, because you are dealing with the topic of knobs.

A. Yes. They are all on the topic of knobs.

Q. We will start with slide 45.

A. I think - it is up to you. It's been given a number to each of them.

HIS HONOUR: They have to number each of them.

A. So this is now the seventh and the most serious problem with the HIV particles. First of all, how many knobs on HIV because they do have to have knobs? Now, according to Montagnier, in his book published in 2002, Montagnier says: 'Particles of HIV are shaped like little spheres, each with roughly 80 rounded projections shaped like pegs', not as spikes as they are also known. In her book published in 2005, Elizabeth Dax and her collegues state there are '72 knobs or spikes of the external envelope of HIV', on the external envelope of HIV as shown there.

XN

Q. Just stopping you there, the significant feature of this is that Montagnier is talking about roughly 80 whereas HIV experts Constantine and Dax are quite specific, 72 knobs, is that correct.

A. Yes, that is correct but that is not the biggest problem. There are more problems with that.

Q. You continue on.

A. The next one, that is 46, slide 46. Now, the knobs, according to all the HIV experts, no knobs - if the particle has no knobs, it cannot be infection. The knobs are critical. There is no exception. They all state the same thing. The knobs are critical, are crucial for the particles to be infectious, otherwise

you can't - infection cannot take place. Now, the knobs are made up of a protein which is said to be HIV and the protein is called gp120. 'G' stands for sugar and 'p' stands for protein. 120 is the molecular weight in 1,000. In a paper published in 1998 by Gelderblom, as I said, the best expert on HIV particles, his collegues estimated that immediately after being released from the cell membrane HIV particles possess on average .5 knobs per particle which are rapidly lost.

HIS HONOUR

Q. That should be 0.5.

A. 0.5 knobs per particle which are rapidly lost, but also,

they pointed out that 'It was possible that structures resembling knobs might be observed even when there was no gp120', that is when there were no knobs present 'ie false positives.

Q. That is all set out in slide 47.

A. Slide 47.

Q. You are going to slide 48.

A. Slide 48. Now, in a paper published in 2003 by researchers using one of the most modern method, as I said before, gp120 is the constituents of the knobs, what is said to be the constituents of the knobs. They say the clusters of gp120 do not form spikes on the surface of the HIV as is commonly described in the literature. 'We suggest that spikes, knobs, observed by negative-tainting electron microscopy may be an artifact of the penetration of heavy metal stain between envelope proteins'; that is, they said that, like Gelderblom, they conclude that there are no knobs on the HIV particles. In a paper published -

XN

Q. This has got to be contrasted, if we remember back in 45 where Montagnier said there were roughly 80 but Constantine and Dax is specific at 72.

A. Yes. Now, in a paper published this year -

Q. This is 49.

A. Slide 49. This, the top part, is a slide of SIV immuno

deficiency virus particles, and as you can see, the particles have knobs on their surface. The lower slide presents HIV particles. We could not see any knobs on these particles apart from there but then down there there is something similar where there are no particles. So, I could never say they may be just - as Gelderblom would say, they may be just artifacts.

HIS HONOUR

Q. Go back to 48. You'll see Kuznetsov at the bottom, slide 48. As Kuznetsov said -

A. There may be artifacts. Indeed, the authors of this 2006 paper, they said they don't call what is seen there

on HIV as being proven knobs, they say they are putative knobs. 'Putative' means supposedly. So they haven't got any proof that these are actually knobs.

XN

Q. So far as you are aware, who called in the expression putative, what seems to be knobs that are not there.

A. Sorry?

Q. Whose expression is it, 'putative'

A. Putative is the authors, Zhu P. The authors, if you look at the describes where they describe these knobs, they say they are putative knobs.

HIS HONOUR

Q. The authors are professor Zhu and others.

A. Yes, they themselves do not consider this. What they have seen there in one particle, they do not consider it as proof for being knobs. They say 'putative'; that is, they may be, they may be, but they are not sure.

XN

Q. Their word was putative, was it.

A. It is their word, yes. That is how they described it.

Q. Who has defined it as 'supposedly'.

A. Sorry?

Q. Who defined 'putative' in this context as 'supposedly'. Is that your definition.

A. That is our interpretation.

Q. I just want to make that clear.

A. That is what is in the dictionary, 'putative' means 'supposedly'.

Q. What we are looking at here, we are looking at the monkey retrovirus and the knobs -

A. They are on the monkey virus. They are obvious.

CONTINUED

Q. The bottom line is there's an electron photograph of an HIV -

A. The bottom, yes.

Q. - virus, allegedly, and -

A. Yes.

Q. - are there any knobs on there at all.

A. No, we can't see any. As I said, we couldn't see any. In fact, we wrote a paper to Nature where this was published. Unfortunately they rejected it, and even the author said that, you know, they can see only in a few parts there, they see something which will, some not, but they don't say that they're definitely not. They called them, repeatedly, putative.

HIS HONOUR

Q. Just to get it clear, you and some others wrote a paper to Nature.

A. Yes, discussing this.

Q. Discussing this, in which you said that you didn't observe any knobs.

A. No, we put much more than that.

Q. Yes, I know, but that's part of what you said.

A. Yes.

Q. You've said that paper was rejected.

A. Was rejected, which was not surprising because they would give to their - we were criticising.

Q. Yes, I understand.

A. So there are so many problems and, as I said so today, nobody has proven the existence of knobs in this particle. So summary of RT in particles.

Q. This is slide 50.

A. That is evidence -

XN

Q. We've done 50, have we.

HIS HONOUR: Yes, this is slide 50.

XN

Q. I'm sorry, I just then got distracted with something else. I want to take you back to 49 please. When we started on knobs I said to you we were going to look at

this as a whole. When we were looking at slide 46, that says knobs are critical for infection to take place. In other words, knobs were critical to the appearance of the HIV virus particle.

A. Yes, if you don't have - if you have - if the particles do not have knobs, they're not infection, and if they are not infection, they cannot be a virus.

Q. Montagnier says you should have around about 80, Constantine and Faulk say 72.

A. Yes, but the evidence contradicts that.

Q. Yes, the evidence - by the 'evidence', now you're talking about slide 49 which we're looking at and that's a study done in 2006.

A. Yes, incidentally one of the most recent papers.

Q. Contradicting basically a fundamental proposition advanced by the founders of, or the finders of HIV.

A. As I say, in every single property of this particle is important, but this is crucial because if you don't have knobs, and this is a general agreement, it's not only one or two HIV expert who say it, they all say the same thing, that we have that in our scientific publications.

Q. In a practical way, what's the purpose of the knobs.

A. They have to attach to the cell. If you don't - if the particles haven't got the knobs they cannot attach to the cell, and they have to attach to the cell to go inside the cell. If they don't go inside the cell they

can't multiply.

Q. Is it, knowing what happens to the knobs after they attach themselves to a cell and have gone inside, they go about their business.

A. Once they go inside the cell, the whole particle, not only knobs, once they are inside the cell they become disruptive, there, the way the viruses they are there now, they're very organised and they become like crossed, it is crossed bonds between proteins and between the DNA and the RNA, but once they are in the cell they are reduced and all the thing comes apart. For them to multiply they have to come apart.

Q. What we're looking at, in the bottom photograph there, means that these are cells sort of on the outside.

A. No, these are not cells, these are particles.

Q. Yes, sorry, they're particles and they're not inside the cell.

A. Yes, they're outside the cell.

Q. They're waiting to hang on to something.

A. Yes.

Q. But they haven't got the knobs to do it.

A. Yes.

Q. Okay, could you move on please.

A. Now, let's summarise the evidence, continue evidence so far. We have a problem that the RT, which is detection of which is that proof of infection but RT - that is, RT means reverse transcriptors activity - is not specific. There is no agreement as to the toxonomy of the age of the particle. Particle even with RT activity are not proof that they are infectious, that is they are viruses and this is accepted, most accepted by Gallo as far back as 1976, particles may appear in culture even if the culture is not infected with HIV. Knobs are fundamental to the definition of retrovirus and so far nobody has proven they existed or not, the particles which are said to represent the HIV virus, and as I said, they are absolutely necessary for infectivity. If they have no knobs, there can't be infection and they cannot be

transmitted.

HIS HONOUR: We're going to have lunch now. I was going to discuss -

MR BORICK: I can tell your Honour we've got about an hour of this presentation to go.

HIS HONOUR

Q. Ms Eleopulos, you don't need to sit in the witness box. We're going to break for lunch now, so if you could come back at 2.15 but you can sit down in the body of the court. You don't have to stay there. I'm just going to discuss the timetable with counsel, that's all.

HIS HONOUR: You think you've got about an hour to go?

MR BORICK: I will be one hour to go - just from experience with matters of this presentation, about an hour, and then Dr Turner for the rest of the afternoon.

HIS HONOUR: I don't know, I presume that, Ms McDonald, you want to cross-examine the witness.

MR BORICK: We've reached agreement that we'll go through our presentation and -

HIS HONOUR: So you're going to have the two witnesses give evidence-in-chief first; is that the position?

MR BORICK: That's right, because they are a connected whole.

HIS HONOUR: Ms McDonald, are you happy with that?

MS MCDONALD: It's a little more complicated than that; this witness goes and the other one comes and this witness goes back. In light of all the toing and froing I agree with my friend, if all the evidence is finished and then we had cross-examination.

HIS HONOUR: How long are you going to be cross-examining?

MS MCDONALD: I have no idea.

HIS HONOUR: This is going to throw the timetable out a bit in respect of your evidence, I presume.

MS MCDONALD: Yes, it again throws the timetable out. I don't have the schedule in front of me. Professor Cooper I believe is booked in from Wednesday morning - Thursday morning, I think I might have lost a day now.

HIS HONOUR: You've got Professor Cooper for Thursday morning but I can't see you're going to reach him.

MS MCDONALD: The difficulty we have with Professor Cooper, your Honour will see from the CV, he is someone who's very busy to say the least in the international arena and we really were lucky to get that time with him. If we get to that point, it will be my application his evidence be interposed.

MR BORICK: I think we'd fit in with that.

HIS HONOUR: That may -

MR BORICK: At the moment I do.

HIS HONOUR: I think we'll just have to test it as we

go, Ms McDonald, but there's another person in this equation who happens to be sitting up here who's got to try and understand all of this material. If it's going to come in piecemeal, it makes it terribly difficult. I mean, I've read the reports and I've read what's been presented to me, but I really need some explanation of some of it. If you're halfway through your cross-examination and then we get to Professor Cooper, it's does make it very difficult.

MS MCDONALD: It does. We spent some time reshuffling witnesses.

HIS HONOUR: I know. And I apologise that I've caused some of these problems but that's life, I'm afraid.

MS MCDONALD: It can't be helped. I think we'll have to take it as it comes to some extent. Can I say this: given the nature of the reports that have been exchanged and the fact that already some of the applicant's witnesses are dealing with some of the points that have been raised by some of the respondent's witnesses, and this is a leave application, I don't propose to cross-examine these witnesses in chapter and verse at every detail based on their conclusions.

HIS HONOUR: I know, but the problem is if I - let's deal with some hypotheticals. If I were to grant leave, I don't imagine that the Court of Criminal Appeal is really going to want to rehear all this evidence.

MS MCDONALD: I don't know.

HIS HONOUR: Well I don't either because I'm not a member of it, but what I've been trying to avoid is the potential that the Court of Criminal Appeal may have to reinvent the wheel. So I wanted to hear as much of the evidence as possible at this stage so that, at least if the matter goes any further, the court will be in a position to actually make up its mind what and if any evidence it wants to hear or how it wants to proceed. But that's one of the reasons why I've proceeded the way I have. Anyway, the question as to how much you cross-examine is a matter for you.

MS MCDONALD: Yes. I appreciate all of that and I realise that's why we've set aside two weeks for this. But can I also add, at the risk of sounding like I'm whingeing about things, I asked for full disclosure of the reports of these two experts. As your Honour is aware, we were given a half a page affidavit from this witness. Had we been given this material before close of business last Friday, which was when we received the PowerPoint, it might be the reports could have been much clearer. I'm in a very difficult situation in that our experts haven't had the chance to address these issues in the reports.

HIS HONOUR: I know. I was going to say to you, having read the reports, it seemed to me that a lot of the material was like two ships passing in the night. I must say I'm not surprised that you may need to take some further instructions about some of this material. Anyway, I just raise that and I'll leave it to counsel to work out how you're going to go about this. I would have thought that there might be some difficulty in dealing with Professor Cooper in a couple of hours. I don't know how long Mr Borick intends to cross-examine.

MS MCDONALD: All of those have been on line until this point, until the detail of his evidence has come out.

HIS HONOUR: The difficulty about Thursday is that as you know I've got another arrangement. So I've got to

adjourn at 12.30. So perhaps it's something you need to think about, whether you're going to be able to complete Professor Cooper even if you were to start him on Thursday.

MS MCDONALD: I'm concerned now, having heard the evidence, really where we're going. In the next two weeks we have seven witnesses to get through.

HIS HONOUR: That's one of the difficulties.

MS MCDONALD: All I can do is have some discussions with my friend as we proceed.

HIS HONOUR: I think you'd better think about it, perhaps have some discussions, because I really want to

try and deal with this matter and complete it as soon as practicable, and if we're not going to complete it in the next two weeks, a question will arise whether I can then go into a third week, and that will depend on availability of counsel and various other things, plus the fact that I'm listed to commence something else on 6 November. It means trying to adjust that too. I don't know what the availability of judges etc. will be.

MS MCDONALD: I can indicate to your Honour I have a two month trial, a trial that will go right up to Christmas starting on 6 November before David J.

HIS HONOUR: These are matters that perhaps you need to give some thought to because at the moment I can't see this matter completing itself in the next two weeks.

MS MCDONALD: Not given today so far.

HIS HONOUR: No. If we recommence at, say, 2.20.

ADJOURNED 1.10 P.M.

RESUMING 2.25 P.M.

XN

Q. Looking at slide 51 -

HIS HONOUR: That's 50 on mine.

MR BORICK: I've got the top right-hand corner, 'Summary of RT particles'.

HIS HONOUR: Maybe I've got it wrong.

MR BORICK: I've got that one as 50.

HIS HONOUR: Yes, that's 50, that's the one up on the

screen.

MR BORICK: Yes, then 51 is the next one, which is antibodies and antigens.

A. Montagnia said that the two principal scientific evidence of the virus which he seen was not HD51 or HD52; first of all that the multicategorical characteristics or the particle. However, as I said, in 1983 the particles he seen, which he said he seen, was exactly the same like the HD1 and HD2 which were type C particles. The other evidence which he said proves that the particle which he seen were not HD1 or HD2 were that these particles had different - they had unique growth

which were not found in the other immune virus. Before I go to describe his actual evidence, let me again make a few definition. Antibodies: first of all, antibodies are antigens. The immune system respond to presence of foreign material such as proteins or bacteria and viruses by producing proteins which are known as antibodies. The proteins, the external proteins or viruses which cause the antibodies are called antigens.

XN

Q. 52.

A. Slide 52, now, it was known, from the time that the proteins and antibodies were defined, it was known that the antibodies react with inducing antigen, and this can be seen as a colour change. When this reaction, when the antibodies, antigen take place, you can see it by the colour changes. Now this reaction for a long time was thought to be specific; that is, the antigen was always - always reacts with the antibody which it induced, and this method was used to prove the existence of antibodies in humans or animals when the antigen was known. 53, now the blood contains red cells, white cells and serum, that is the white stuff, the cream - the cream stuff here in the test tube (INDICATES). Now, the antibodies are always present in the serum, so that's why the antibody test sometimes are called like serological test or seroconversion.

Q. 54.

A. Now, but not - since the 1970s it was known that the antibodies do not always, or they don't only react with inducing antigens, but they are - some immunologists call they are not monogenous. So here, for example, so Nassal in 1971 in his book wrote 'An antibody molecule made following the injection of one antigen frequently can combine also with a second antigen, or of related or similar shape. In other words, the antibody cross-react with the second antigen'. This - 55?

Q. 55.

A. In the paper which was published in 2001, one

immunologist wrote 'The immunological community was shocked to find that antibodies would be polyreactive in binding to multiple antigens that were complex and ostensibly unrelated to one another'. In fact, he used the word, 'The antibody are promiscuous'. This means -

Q. 'Polyreactive', what does that mean.

A. Polyreactive, that means an antibody will react not only - 'poly' means many in Greek so it will react with many antigens, which are not the antigens which induce its appearance.

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.HAC...00109 51 E. PAPADOPULOS-ELEOPULOS XN

A. This means an antigen cannot identify another body just because the antibody - the axis is an antigen you can't say what was the antigen which induced it and conversely antibodies cannot identify an antigen. If you have an antibody just because you find the antigen to the axis doesn't mean that the antibody was caused by that.

Q. 56.

A. Now, Montagnier claimed that the particle he observed in the culture had totally - had distinct products which are not found in other viruses. So, the proteins are retroviro and the retrorivro system.

57. Now, to prove the existence of HIV protein by definition, protein should be found in the viral particle. If you don't take the proteins from the viral particle you cannot say that they are HIV. But it is very hard or impossible to take a protein from one viral particle. So the second best thing is to purify the particles, that is to obtain the particle separate from everything else which also contains proteins. And, Montagnier agrees with that.

58. When Djamel Tahi interviewed in 1997 he ask him, you know, one of the question was how you characterise the HIV protein. He said to do that - Djamel ask - by the cancer point when one must to the characterisation of the virus. This mean what are the proteins of which it is composed. And Montagnier reply

'That's it. So then analysis of the proteins of the virus demands mass production and purification. It is necessary to do'. So, it is, this comes from Montagnier's claim, you have got to purify the virus particles to be able to claim that protein is a HIV protein.

Q. And 'purify' in this context means the same thing as 'isolate'.

A. If we take the definition of 'isolation' from the Oxford dictionary, then purification and isolation mean the same thing. That is, you have to obtain the particle, isolate it, separate it for anything else which has the

same components as the viral particle. That is, proteins and iron and in this case we are interested in the proteins.

Q. Now, we are coming to a very important part of this evidence when we deal with retroviral isolation. Just before we come to this segment, could you explain in the simplest terms you can what is meant by 'mass production'.

A. Mass production, it means that you have to make - when you put the infected cells in the culture, in the culture you must find a lot of particles to be able to get a material which will contain many particles. You cannot get the proteins just from a few particles. You have to have a lot of particles so you have to have mass production of viral particles, of HIV particles.

Q. In giving your evidence in your definitions you have referred to cells and particles. Now, if we can just go back again over that a little bit. When you referred to particles in that answer, what is the relationship with cells.

A. Sorry?

Q. You referred to particles. What is the relationship between the word 'particle' in that answer and 'cells'.

A. The cells are totally different.

Q. I just want to make sure. So just explain that again will you. What do you mean when you are talking about a

particle.

A. What I mean about retroviral particles.

Q. Yes.

A. I am just saying a particle which has the morphology of retroviruses, that is it looks like a retrovirus because the retrovirus has certain characteristics by definition.

Q. Slide 59 simply refers to a laboratory procedure called density gradient centrifugation; is that right.

A. Yes, this is a laboratory procedure which has been used for over 30 years now for the purification of retroviral particles. That is, it is a procedure recognised to

separate virus - large particles from everything else and the procedure is called density gradient centrifugation because it is based on the fact that retroviral particles have unique density and in across density gradients they band or they stop or they appear in the gradient which has the density of 1.16 gm/ml.

Q. And at the beginning of the interview, this Montagnier refers to the density of 1.16, doesn't he.

A. Yes.

Q. The next slide, I think 60, can you explain to us then how this works.

A. Yes. Now, the density gradient measure consists of the following: you have a tube and in that tube you put sucrose, that is sugar with different densities, just bands of sugar with different densities. Then you take that tube and you spin it. It is like, you know, just putting it in the washing machine and spin. You spin it for a long time but before you start spinning you put your sample, which you think that it has the viral particle. That is, you take the supernatant, the fluid, from the culture which you think has your particles, like Montagnier took the supernatant from the third experiment, the third culture, put it there at the top of there tube and then you start spinning it for a long time. Now, if there were any particles, any retroviral particles in this sample in his short experiment then

those particles should have stopped at this band.

Q. That's this band, you say this band or these bands.

A. This band, one band. This band there represents 1.16 gm/ml. The density in this band by design is 1.16 gm/ml. So, if there were any particles in his culture they all should have stopped there. Then what you do after you spin it for a long time, you take each band, you open the bottom of that tube and then you take each band separate. You discharge all the ones which you are not interested in and you take the one you are interested in and you look to see what you have in that band. That band 1.16 gm/ml.

Q. You had better explain how you discharge it in this procedure.

A. As I say, you just take the tube once it is finished spinning. At the bottom you can open it, they are specially made tubes. You open it and then each band starts to come out separate. We have this band come down, then we have the other band come down and you discharge because you are not interested in all these other bands.

Q. I am not sure whether you did, will you explain why in the word 'Sucrose' you have got the small 'S' at the top and the big 'E' down the bottom and the gradient down the line.

A. That is because the heavier the particle is and sooner it will come down the gradients and it will stop at the higher density so that is why we have the different densities. So the bottom has to be the higher density than the top, the lower density.

Q. Thank you.

A. So, what is important here is that they - in the recent matter which you can't separate the particles and that method consists of them banding by definition. Montagnier gives a lot of credit to these properties because he says the density is the most important thing to tell us the particles are retrovirus. So you take that band, the 1.16 gm/ml, and that's what Montagnier

said he did. He took that band and he called it 'Pure HIV'. Purified virus. This is in his paper. The 1.16 gm/ml is called purified virus. All right. Now, the problem was, he left it to us to believe him that what he had there was a purified virus.

Q. You are looking at 61.

A. 51?

Q. 61.

A. He said that what he had there was a purified virus. But he did not publish a picture to prove that what he had there was purified virus and this is what we have been asking from the very beginning: why they did not

have a picture to show that what they have there was nothing else but particles which have the morphology of retrovirus. Especially when Barre-Sinoussi, that is the first and second author of this paper, in 1973 stress, that the only way to claim that to have a purified virus, that is the 1.16 gm/ml band, is to present pictures which show that you have nothing else but particles with the same physical characteristics. And yet in 1983 they haven't presented such a picture. But they have done something else. What they did, the proteins which were in the 1.16 gm/ml band. Can I please go on one back. Also, the 1.16 gm/ml band, the retroviral particles band there, it is not only the retroviral particles which band there. There are these - banding is characteristic for retroviral particles but it is not specific. There are many other particles including cellular fragments which band at that density. So, that is why it is very important to have a picture to show that you have only particles which look like retroviruses. So, I repeat, they did not publish such a picture. What they did is they took the proteins which are the 1.16 gm/ml there and they separated them, you can do that by retrophoresis and the reaction of these proteins in the BRU serum.

HIS HONOUR

Q. That's the name. And it is serum.

A. Serum. They react to the proteins which band it at 1.16 gm/ml with the BRU serum and they found three proteins which reacted with antibodies which were present in his serum. Now, the proteins were P24. As I said, P stands for protein, 24 stands for molecular weight. P 45, P80. He did not comment as to what P80 was. He said that the P41/45 protein which was in the purified virus and reacted with the patients serum was a similar protein acting. And he said the only proteins which was HIV was P24. He called this 'most specific' and everybody today considers P24 as the more specific HIV protein. And then he took antibodies to the HTLV-1 protein, P24, that

is HTLV-1, that is the other human retrovirus also has a P24 protein and he took antibodies which were directed against the HTLV-1 protein and he said they did not react with the HTLV-1 protein, HIV-1 proteins or with the HIV protein. He said this means that P24 is a protein to a different virus. Now, this raises several problems. First of all, if P24 is HIV and - if P41 is HIV and P80 is again a non-HIV protein then the person has antibodies which react with it, now why then, why this P24 cannot also be a cellular protein. How does he know that that was an HIV protein? He didn't have any other evidence apart from the reaction. And how did he know that the antibodies which reacted, which are present in the BRU serum, were actually antibodies which are caused by infection with HIV. They could have existent antibodies, promiscuous or cross-reactants, they could be antibodies with something else and which react, even if people had HIV. So, another thing is that no virus has only one protein. If the 1.16 gm/ml band was purified to HIV, then he should have found many more proteins there to react with the BRU serum.

Q. Could you go now to the next slide, 62, and just briefly explain what Gallo's experiment was in 64 then go to 63 and we will come back to 61 again. So, 62, just explain, it is headed 'Gallo 1984'.

A. Now, in 1984 Gallo did similar experiments to

Montagnier. In fact, the main difference between what Montagnier did and Gallo did is instead of Gallo using umbilical cord lymphocytes he used an H9 leukaemic cell line. And although a leukaemic cell line is now known, at that time in 1984 he said that a leukaemic cell line was a leukaemic cell line but this creates equally just using a leukaemic cell line creates many problems because Gallo himself knew that leukaemic cell lines, even not infected with HIV, they will produce virus-like particles. Secondly, it turns out after Gallo was investigated by the congress, that leukaemic cell line, this H9 cell line actually originated from a patient

which had a type of leukaemia which Gallo was saying was caused by HILV-1. In fact, in 1983 he said that this leukaemic cell line was infected with HLTV-1. So, this cell line should have had a retrovirus even if there was no culture with tissue from AIDS patients. The other main difference between Montagnier and Gallo's experiment was that Gallo, unlike Montagnier, used more than one AIDS patient. And, he found, unlike Montagnier, - slide 63 - he found many more proteins to react with AIDS patients' serum. First of all, he had the P24 protein like Montagnier. But unlike Montagnier he said the P24 protein is not characteristic of HIV because this protein cross-reacts or reacts with antibodies to HTLV-1. For him, the P41 protein was the most characteristic HIV protein where, for Gallo, P41/45 was acting. So there is a bit contradiction between Montagnier and Gallo.

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Q. Could you just go back to 61 so that you can just refresh people's memory on that issue.

A. Sorry?

Q. 61.

A. Yes. Montagnier said that his p41/p45 is actin, cellular protein, make a cellular protein. Gallo said this is the most characteristic HIV protein.

Q. This is Montagnier, p24 is HIV and P41 is -

A. Cellular.

Q. Cellular.

A. Nothing to do with the virus.

Q. And you go back to 63.

A. And Gallo says this is the most characteristic HIV protein.

Q. You go back to 63, Gallo discounts p24, but says that P41, which Montagnier calls cellular, is the most specific.

A. Yes.

Q. So it's impossible to reconcile the two opinions.

A. It is as far as we are concerned, it is a contradiction there, a significant contradiction, one may add. But, as I said, unlike Montagnier, who used only one cell from one patient, Gallo used from many patient and some of these cellular reacted with other proteins which he said, like Montagnier, was purified virus. Like Montagnier said, the 1.16 gm/ml was purified virus and

those proteins reacted with many patients serum, and some of the patients react with many other proteins p7, p17/18, p31/32, p39, p51, p55, p66, p120 and p160, so these proteins then, just because some of them reacted with some of the AIDS patients' serum, became known as HIV without ever having evidence that all the so-called purified virus actually even contained particles which looked like a retrovirus.

Q. If you go to 64.

A. Now this is how a purified retroviral band, 1.16 gm/ml band should look like. There it is, a purified one of the first human retroviruses, the leukosarcoma virus,

was published in 1961.

Q. We're looking at 65 now.

A. Yes. As they say, we have been asking for evidence that Gallo, what Gallo called purified virus, or Montagnier called purified virus, and others following them also called purified virus, to come up with evidence. Nobody came. Even up till 1997. Some researcher agree with us and they have - these researchers are two groups; one a German called operation group and another group from the United States, they accepted that up till 1997, let me call virus, could be 'used for biochemical and serological analyses or as an immunogen, that is an antigen, is frequently prepared by centrifugation through sucrose gradients'. They also said that in none of the studies has the purity of the virus preparation been verified, so there are many people after Montagnier who are calling the 1.16 gm/ml band as purified virus, but nobody publish any evidence that that is what they had. These two research groups, the Franco-German and the Americans, they started to try to purify HIV and they published their papers; there were two papers published on virology in 1997. Now this, the first slide, is the slide from the Franco-German studies and the top two parts of the slide -

HIS HONOUR

Q. Slide 66.

A. Yes, slide 66, now this part here (INDICATES) is the 1.16 gm/ml band from infected cells, that is H9 cells infected with HIV. This other part, the middle part represent the 1.16 gm/ml band, again from infected cells, but this time cells from normal individuals. The bottom part represents 1.16 gm/ml band obtained from non-infected cultures. As you can see, whichever these two slides represent, they are not purified HIV. In fact, the author label the first and the second slide as 'Purified vesicles from infected H9 cells (a)', that was the top, 'and activated peripheral blood mononuclear cells (b)'. So they can't or they are not purified. In

fact, even the authors themselves call them purified vesicles, not purified HIV, as vesicles meant cellular fragments -

Q. Sorry, I will let you finish.

A. So what is in there, the majority of the things which they see for the 1.16 gm/ml band is cellular fragments. They did label a few particles as representing HIV, like that one, where the arrows are, the one at the top there and the one there, and here - that one and the one there as being HIV - but they are so few amongst all the cellular debris, but if you look at the bottom, there are some particles even in this part which originated from non-infected cells which have some particles which look similar to the ones which are arrowed and are said to be HIV (INDICATES).

Q. Who put the arrows on there.

A. The authors.

Q. Sorry. The authors.

A. Lysenko and his colleagues, the Franco-German group.

Q. To the outside, it's difficult to see what the criteria were for selecting those particles.

A. Yes. Whatever the criteria are, these particles do not have all the morphological characteristics which a retroviral particle should have.

Q. Who has put the rectangles or squares on the third one.

A. You mean at the bottom?

Q. Yes.

A. We put them so that to make it easier for you to see them, like this (INDICATES).

Q. From a subjective -

A. That is our interpretation, that they look similar to what in the top part is called HIV.

Q. In the print at the bottom, you better explain what the bracketed A and the bracketed B stand for.

A. That is the top part of the slide; (a) is the top part, this one, and (b) is this and, as I say, these two should represent purified HIV, that is they should have nothing else there but particles which look like this at

least but, as you can see, there is nothing. In fact, I repeat: the authors, they did not call them purified HIV, they called them purified vesicles, that is purified cellular families (INDICATES).

Q. The 'PBMC' in (b), what does that mean.

A. Sorry? Peripheral blood mononuclear cells. That is the cells which originated from a healthy person.

Q. What is the connection between the 'and activated' in (b).

A. This 'activated' means there are cells which are - thank you for drawing my attention to this - activated cells means that they put a lot of - in the test tube, they put a lot of chemicals which made the cell to divide, including PHA. That is a very important observation because although they put in the first, in the second of this slide, in the cultures, they put - they said that the cells were HIV infected, they also put many chemicals, many chemicals, which they did not put in the third culture, so if they would have put in the third culture, that is if they had a proper control, then the possibility cannot be excluded that all they had in the first and the second part, they would have had exactly the same appearance in the third.

Q. Thank you. The reference to the HIV diameter.

A. None. As I said, one of the first observations here is the particles which are arrowed as being HIV, the

average diameter is 149 nm, which is not that for the retroviral particles.

Q. The diameter of the retroviral particles is accepted as being 120.

A. 100-120.

Q. 100-120.

A. Yes.

MR BORICH: For your Honour's reference, that takes you back to slide 26, 26 and 27.

A. Yes. Now the main characteristics, one of the main characteristics to lentiviral.

XN

Q. Have we moved to 67.

A. Yes.

Q. Sorry, keep going.

A. If HIV is a lentivirus, one of the main characteristics of lentivirus is, as we can see in that picture - and I do apologise, it's not a very good one - but you can see is they have a cone-shaped core and they have knobs on the surface, and that's how it is represented. As I said this is a graphic representation. Now none of the particles in the Franco-German study -

Q. Sorry, you have now moved to 68.

A. Yes.

Q. You better just go back to 67 for a minute.

A. Which is the same slide.

Q. Just go back for a second. You see on the left-hand side there is what looked like pointers or arrows.

A. Yes.

Q. What are they.

A. They are knobs.

Q. No, do you see in the bottom left-hand corner, there is a couple of things that look like arrows, and then -

A. They are the knobs. Yes, they are the knobs. The arrows point to the knobs.

Q. They are arrows pointing to the knobs.

A. Yes.

Q. Thank you. Sorry. Going back to 68.

A. Yes. Now repeating the same slide, just to draw their attention, that these particles which are arrowed are HIV, they have - they don't have a cone-shaped core, they don't have antibodies and they don't have knobs, so they cannot be viral particles, retroviral particles.

Q. How do you get to see cones. Knobs I can understand, but cones.

A. It is easy to see them if they are there but, as you can see, these particles which I arrowed as representing HIV, they have just some dots inside there (INDICATES) but they don't look like a cone at all.

Q. It takes a trained eye to appreciate that, would you agree with that.

A. Yes. Of course.

Q. You want to move on to 69.

A. Right. Now these are the American experiments or the American researchers' effort to purify HIV. At the top they are H9 infected cells. In the middle is a clone of the HIV9 cells, and this middle picture originated from a culture which was drastically manipulated, including being core cultured with cells which are heavily radiated and more or less dead, and the bottom represents again the band from a non-infected culture. Again, as you can see here, it is very hard to see any difference between all of them. Also the middle seem to have a little bit more particles. As I said, they are two totally different maps, and so there are hardly any proper controls but still, the difference is very hard to - it's very hard to find particles which have the morphology of retroviruses in the first and second picture. Now 'MV' there, this 'MV' represent microvesicles, where the arrows which 'MV' represent microvesicles.

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Q. We are on 70 now.

A. Yes. Again, the particles which the arrow is representing HIV, first of all, the average diameter is 234. So, it is impossible for them to be HIV. In fact, we corresponded with Bess. Bess is the principal author of this study. We corresponded with him and he said he doesn't know why these particles was so large; he cannot give an explanation. He will ask them but they never come back to us. We don't know how could this particle be called HIV when they had such a large diameter. Again, these particles do not have a cone shape at core, they don't have lateral bodies and they don't have knobs, so they don't have the morphology of the retroviruses.

Q. If you go to 71 -

A. Now, this is 71.

Q. We are still dealing with Bess at the moment, aren't we.

A. We are still dealing with Bess and Bess -

Q. Can I interrupt you there. With 69, 70 and 71, what is Bess trying to achieve with this. What is his purpose.

A. He tried to obtain purified HIV and obviously he did not manage.

Q. Can you explain 71 now.

A. Now, they have done something which nobody until then has done it. They took the proteins from all this tests, from the first the second and the third. Like,

from the infected cultures and from the non-infected cultures, they took the protein and separated them by using eletrophosphuresis and they obtained the -

Q. We have gone to 72.

A. Slide 72. Now, the first - here are the proteins from the band which was non-HIV infected and these are all the proteins in that band. The second and the third are from the infected bands. That is what the proteins which were in the infected bands, the HIV infected bands, was there for. Now, as far as we are concerned, the only difference between all these bands is qualitative, it is not quantitative. That is all the

bands are present, all the proteins are present in all the bands. The difference is one of quantity. In the one from the non-infected cells, all the proteins are there but the bands are weaker; that is, there is less protein than there are in the so-called HIV infected bands.

Q. To the layman's eye, when you look at that, in that area between 31 and 21.5, the two on the right are quite distinctive, they look like big pan cakes.

A. They are but that is what it means. There are more there but they are no different. Qualitatively they are no different, they are only quantitatively different. Do you know what I mean?

Q. I've always had trouble with this one.

A. Now, if you go there, there and there (INDICATES), the bands are there but they are much weaker than they were here. In fact, they are more like what is there (INDICATES). You see, if you look at these bands and you look there, it is very close. Here it is much more but the bands are there. They are there in all of them. In fact, again we corresponded with Bess and Bess agreed with us. As I said, we said to Bess that as far as we are concerned, all the proteins were present in all the bands. The only difference was a quantitative difference. We agree that you can come to the conclusion from geophosphatic patterns that you can come

to the conclusion that that is the only quantitative difference between HIV and microvesicles. So they agreed with us, that means they didn't have any proteins different between the HIV infected and the non-HIV infected band. If the non-infected HIV bands had the particles they call HIV who are indeed HIV, then in the infected bands they should have had some proteins which are not present in the non-infected, it is very simple, and yet they don't have that proof.

Q. If we go to 74 -

A. Yes, in 74, now -

Q. We are now looking basically at what was in 72 again.

A. Yes, this time Bess and his associates labelled the proteins, gave names to some of the proteins to some of the bands. Is it clear? You can see that.

Q. Perhaps just jumping ahead a little bit, the labels were added because one of his reviewers asked him to label that.

A. No, no, because we again - first of all, he put his labels, right.

Q. Explain the labels.

A. Now, they put labels. Now, the labels p24, p17 and p6/p7, they are HIV proteins. HLA, this is a cell protein. So they label everything around 32 as being cellular proteins. In the infected bands they also label 41 or the proteins around 41, because where the proteins move in the gel, some move quicker, some more slower, it depends, so you can't say exactly 41. It will appear 41, 45. It depends on the condition you are using and the label on the protein. So they label all their proteins around 42.7 as being actin and they left all the bands higher than 40, 41, unlabelled, but they are mainly HIV proteins, including two of the most important HIV protein, gp120 and gp160, which they did not label. So again, the question is why they have not done it or if it appears that there are some very good reason. The gp120, what is said to be a unique HIV protein, two unique HIV proteins, gp120 and gp160, it

was shown in 1989 that there actually are 41. There are proteins that is gp41 and they are trimers and tetramers that are 43 and 41 joined together. So they are not three different HIV proteins, they are one HIV protein, 41, which according to Montagnier, is actin, and this is accepted also by Elizabeth Dax and Schupbach, and Schupbach is one of the main collaborators with Gallo in 1984. As far back as 1987, Henderson, some American researcher, has shown that this is actually again cellular protein, and in Schupbach, Elizabeth Dax, co-author in 2005, wrote: 'After viral bands appear to be cell associated with the most common being in the

molecular weight range of 70' - that is 70,000 molecular - '51-55,000 molecular weight.' So Elizabeth Dax and many other HIV experts accept that all the proteins with molecular weight higher than 24,000 are cellular proteins. They call them proteins but they are actually cellular proteins. And it must also be mentioned that the lower molecular weight, the p7 and p6, they are also fragments of the proteins which have a molecular weight higher than 32 which again we end up as them being cellular protein. We ask -

Q. Can I just interrupt you for a second. If you look at 76 through to 86, somewhere on 86, just before your conclusion, I need you to look through those quickly. Most of them are pretty self-evident as to what they mean. Perhaps 78 on the bottom, it is just a photograph of the fish. I think they are probably all fairly self-evident until you get down to your final conclusions. Is that right.

A. This is again the head of correspondence.

Q. I know, but if you look ahead to the next dozen or so slides, they are all pretty self-explanatory.

A. Yes, they are.

Q. Perhaps we could leave his Honour to look at those himself and then take him straight to the conclusion. Let me just go a little bit because as I said, we asked Bess what evidence he had to label p24, p17 and p6/p7 as

viral proteins and he responded: 'Several bands are labelled as either actin HLA DR, p24 CA, p17 MA, p6/7 NC and you are wondering how we determined the identity of these ... these labels were added when one of the reviewers asked for them. He felt it would help orientate readers when looking at the figure - the reviewer is correct. We did not determine the identities of the bands in this particular gel'. So, Bess did not have any evidence that the protein he labelled as being HIV protein was actually HIV. Now, this is a very important response but we are left now with one protein, p24. Even if we forget what Bess

says, he labelled them but he did not have any when they labelled the HIV. We have only one protein. We are left with, as we started, with one HIV protein, p24, but what evidence do we have that this was HIV, because so far we didn't have any, and since, becoming even worse for it, when we see what Montagnier responded in the 1997 interview, Montagnier said - Gallo, he asked him repeatedly why he did not publish any pictures from what they called purified HIV. Montagnier's response was stunning. He said this was because they did not find any particles which looked like retroviruses. He said, let me quote, 'We found some particles but they did not have the morphology typical of retroviruses', and he repeated 'I repeat, we did not purify', and then he was asked if they were purified and he says 'I do not know if', that is Gallo, 'they were really purified. I don't believe so'. So we have now Montagnier finding a protein in a material which he did not even have a retrovirus-like particle and he found that protein to react with antibodies which are present in the patient's cells and he said the protein was HIV and the antibodies were HIV; he is infected with HIV. Now, this is no different. Montagnier likes to compare himself with a fisherman. He likes himself to fish big fish, or what Montagnier did, let's continue it with analogy. What Montagnier did is to throw his net, because the net

catches fish according to the size. He catches the particles by their intensity. So what Montagnier did is to throw his net in the sea, pull it back, like, for a fish, find not one single fish and he says 'Well, nothing there which looked like fish' and he said what he has in his net is nothing else but fish. In fact, a very specific fish, one single type of fish. In continuing in his interview, once Montagnier accepted that what he called 'purified virus' didn't even have virus-like particles, he continued to ask it and he said 'Do pictures from purification exist?'. Montagnier said 'Yes, of course'. Tahi: 'Have they been published?'.

Montagnier: 'I couldn't tell you. We have some somewhere but it is not of interest, not of any interest'. Now, we have asked Gallo. He sent an email to Gallo and Gallo didn't know who he was and he responded and he said you are asking if either he or Montagnier published any pictures to show that what they had was purified virus. He replied 'Montagnier subsequently published pictures of purified HIV particles as, of course, we did in our first papers. You have no need of worry. The evidence is obvious and overwhelming'. In fact, there was not one single picture published by Gallo in 1984 or at any time since of a purified virus. Never did Montagnier publish any such pictures.

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In 2001, Djamel Tahi interviewed the Pasteur Institute Luc Montagnier, wanted to see what is in the purified - what will they call purified virus. His response was 'We have never seen virus particles in the purified virus. What we have seen all the time was cellular debris, no virus particles'. So we have now, we reach the most specific HIV protein originated from a material which did not have immunovirus particles. Now this is as good as a scientific proof a scientist can have that these proteins is nothing more than a cellular protein. In conclusion, at present HIV experts claim that there are 10 HIV protein but it is no evidence to prove this claim, and all the evidence points out that the proteins which are called, or which are said to be HIV proteins are cellular proteins. 84, a summary, viruses are particles. Now, each particle has unique morphological characteristics. Even today, no agreement exists as to what are the morphological characteristics of the particles said to be HIV. No HIV particle has all the morphological characteristics of retroviruses. Knobs are fundamental to the definition of a retrovirus but no knobs ever prove to exist on the particle which are said to represent HIV. Retrovirus particles may appear in cultures not infected with HIV. 85, now, viruses are infectious, by the definition, they are transmittable. Particles even with RT are not proof that they are

viruses, knobs absolutely necessary for infection, no knobs with HIV particles, so if there are no knobs they cannot be infectious. The only evidence of transmission in isolation is evidence they exist in HIV cultures, but retrovirus activity is not specific to retroviruses, so finally it may not be detected if hundreds of viruses is not proof of infection. Summary, again, the proteins, each virus contain unique proteins, purification absolutely to prove their existence, no proof for purification of HIV by anyone to date, and all the evidence shows that they are cellular proteins. 87, conclusion, no proof for the existence of unique HIV

particle. No proof of HIV transmission, no proof for the existence of unique HIV proteins which means that there is no proof for the existences of a unique human retrovirus.

MR BORICK: We won't worry about 88. There are just two other topics of evidence to cover. You remember I referred to the paper dealing with mortality rates and the paper of the TV broadcast and I'll hand up just a summary of the slides we'll present on that.

DOCUMENT HANDED TO HIS HONOUR

HIS HONOUR: So what's this document you've now handed to me?

MR BORICK: It's the final 10 slides of this witness's presentation, so it can be A5 continued, A5, and then numbers it through to start at 89.

HIS HONOUR: Starts at 89, yes.

MR BORICK: Yes, 89.

HIS HONOUR: 89 really is a slide dealing with - it's got Ms Eleopulos's name, Mr Turner's name, so that should be 89.

MR BORICK: Right, yes.

HIS HONOUR: So the first slide is 90, which is headed 'The HIV theory of AIDS'.

MR BORICK: Yes, thank you.

XN

Q. Could you explain the purpose of HIV theory of AIDS

slide.

A. According to the HIV theory of AIDS, HIV infection itself or CD4 cell leads to the decrease of CD4 cells. HIV infection kills CD4 cells. The decrease in CD4 cells leads to the clinical syndrome that is AIDS. Now, if this is the case, then the more HIV you have the more killing of CD4 cells you will have and the higher the rate of death from AIDS and the higher the rate of AIDS. But this is not what is all the evidence shows.

Q. 91.

A. Now, this is so - let me have - this means that only HIV and nothing else.

Q. Perhaps if I interrupt you a minute, it might be easier if you go through to 95, which is the bottom one on the bottom left page you're looking at there. Can you go through to 95.

HIS HONOUR

Q. The one headed 'HIV infection' with an arrow 'CD4'.

A. This is the main study. This is a study published this year, so again, as I said, the more HIV you have the more AIDS you have, the more death from AIDS you have. However, a paper published this year by Europe, it was a European study, there were 22,000, over 22,000 patients treated with Heart. That is active and retroviral therapy. These are the drugs which are presently used to treat HIV infection. All they found, this drug came into clinical practice in about '96, but with time they are - the HIV experts claim that they improve the treatment, improve the combining of the virus and that led to better control of HIV. Now, by viral law, that mean the number, they say that viral law means the number of HIV particles in the population. So they found out that the better the retrovirus control, that is the - from 1996 till 2003 they had success in decreasing HIV. But this did not translate in having less mortality from AIDS. In fact, they said that the rate of AIDS in most recent period increases. This is the - Professor Cooper made a comment, he wrote a

commentary in Lancet about this paper and he said that - this is his word - a 'paradoxical finding', or it is paradoxical if you can see that the AIDS theory, because the less HIV you have, the less AIDS you should have. They found the opposite. The less HIV they have in the last few years, not only the mortality did not decrease, the rate of AIDS increases. So something else must be involved in causing AIDS and increase in the cell. And indeed this is the case by even more recent paper, in fact, in paper published in September. In that study - I don't know if we have it here, but let me - there it is.

Q. Are you moving to the Rodriguez study now.

A. Yes.

Q. Just before you do that, in relation to your reference to Professor Cooper, in his report at para.G after dealing with 3, he refers to a dramatic decline in the prevalence of AIDS and HIV related mortality since the introduction of antiretroviral therapy which inhibits HIV replication; is that right.

A. There is a lot of claim.

Q. That is what he says in his report.

A. He may, yes.

Q. The study you've just referred to contradicts that.

A. Yes, but let me see.

Q. Excuse me - and Professor Cooper has subsequently described that as a paradox.

A. Yes.

Q. Thank you.

A. Let me say that this paper, this paper will show you that if you control HIV, according to the HIV theory of AIDS you should be able to control AIDS, the rate of AIDS and AIDS mortality, and this paper shows that this is not the case. But to prove, the only way to prove that Heart leads to a decrease in mortality is to have a double blind control study. That is, to have people who are on Heart and people who are not on Heart and never a doctor, not a patient, knows what's going on, who gets

what, who gets placebo, who gets what, and then to follow them up and to come to a negative that will show that people who are on Heart at the end of the trial have less AIDS and less mortality. No such study has ever been published.

HIS HONOUR

Q. It would be a bit difficult to publish such a study, wouldn't it.

A. Sorry?

Q. It would be a bit difficult to publish such a study, would it not, because it would mean that you would have to deny a whole group of people a form of medication

which some experts at least say certainly reduces the mortality rate.

A. Yes, but you have to have -

Q. That means you have to get a whole group of people who have AIDS and not treat them.

A. Yes.

Q. A whole group of people who have AIDS and treat them; unlikely that kind of study is going to take place.

A. No, because how do you treat them with something which you don't know if it benefit the patient or it made them worse? You have to have some indication. You just cannot base your treatment on a claim, on a wishful thinking.

MR BORICK: Perhaps if we just move on, your Honour, I'll discuss that with you later. My understanding was that with the concept of the antiretroviral treatment, Heart as it's called, with that you would expect a decrease in mortality. That hasn't happened.

HIS HONOUR: I understand what the proposition is, but I just query the proposition that the only way you'd ever confirm it is by a blind study. I only asked a question about that. It may be it was not relevant.

MR BORICK: I'd like to take an instruction on that and we'll think about that and talk about that tomorrow.

XN

Q. Do you want to go to slide 96, are you ready. You have

already.

A. Yes.

Q. Could you explain this.

A. This is, as I said, even a more recent paper and in this study the authors examined HIV infected individuals who are not on any drugs, and they call Heart to find out if it was - if HIV was the reason for the decline of the CD4 cells, that is for AIDS, for immune deficiency. They concluded - now, really important that 'We report that plasma HIV RNA level can account for only a small proportion of the variability in the rate of CD4 cell loss in chronic untreated HIV infection' and concluded

'Presenting HIV RNA level predict the rate of CD4 decline only minimally in untreated persons. Other factors as yet unidentified, likely drive CD4 cell loss in HIV infection. This finding have implications for the treatment decisions in HIV infection and for understanding the pathogenesis of progressive immune deficiency'. So they're two important things which one concludes, from these conclusions you draw. One, the HIV is responsible for only - what the words they use - for a minimal decline of the CD4 cells. That's for acquired immune deficiency. There are other factors which cause the decline. Secondly, the risk get very important implication regarding the HIV theory and regarding treatment of HIV infected patient. And these authors are - I think I have another slide where it said.

Q. Yes, if you look at 97.

A. They was from four very prestigious universities in America and their authors, including Harvard, including the University of California and Washington and they're people who involved directly in HIV and AIDS research. In fact, they're epidemiologists and HIV experts from such institutions. Now, there was a commentary related to this study, and the commentary was even according the people who made the commentary, it was very highly regarded, HIV expert and they said 'The provocative main

finding from their study, that is Rodriguez study, was that the HIV load predicted -' in fact, I think they said 10 '- predicted no more than 10% of the observed CD4 loss in patient with chronic untreated HIV infection. What factors explain the other 90%? 25 years into the HIV epidemic, a complete understanding of what drives the decay of CD4 cells, the essential event of HIV disease is still lacking'. And they also wrote 'The findings presented by Rodriguez et al. provide support to those who favour non-virological mechanisms as the predominant cause of CD4 loss'. That is, the AIDS is caused by factors other than HIV. In fact, the

subtitle on their commentary was '25 years and still a puzzle'.

Q. And the final reference to Montagnier on cloning, 89.

A. Cloning is not important for today. You can characterise HIV without any cloning. The characterisation is that the virus density, when you couldn't find any in the body, which is again nonspecific. There are many other papers and I don't think we have the slides here, which appeared from the - we have the papers from two of the main and best, the largest best control studies engaged in. One is from Africa and one is the MAC study, the multicellular AIDS study in America.

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A. And in Amsterdam, just - I can't remember 2004 - 2003 - I can't remember. There wasn't concluded that the immune deficiency - that first all that is shown that people before they become HIV positive they have decreased cd4 cells. And I say the decrease in these cd4 cells before HIV infection is a risk factor for the development of the clinical syndrome. In the MAC study, even earlier than that, it also shows that there are factors other than HIV which augment or determine progression to AIDS so there are many studies today which show - and there are in the more recent time actually there can become more and more frequent that the cause of AIDS one must look for something else than HIV for the cause of AIDS.

HIS HONOUR: Mr Borick I think your client wants to speak with you for a moment. That's right?

MR BORICK: I don't know.

XN

MR BORICK: Mrs Eleopulos can have a break now and she will be repleased by Dr Turner.

HIS HONOUR: Yes, thank you.

MR BORICK APPLIES TO INTERPOSE WITNESS

VALENDAR FRANCIS TURNER

LEAVE GRANTED

WITNESS STANDS DOWN

+THE WITNESS WITHDREW

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MR BORICK CALLS

+VALENDAR FRANCIS TURNER SWORN

+EXAMINATION BY MR BORICK

Q. Will you take his Honour through your qualifications.

A. I have an MBBS in the University of Sydney 1969 FRACS, FRACM.

HIS HONOUR.

Q. FRACS is a Fellow of the Royal Australasian College of Surgery.

A. Yes, and FRACM is a Fellow of the Royal Australasian College for Emergency Medicine.

XN

Q. And your work history.

A. I have been an emergency physician since 1977 and I have worked in several - in fact I have worked in all major emergency departments in Perth. I have spent over 20 years in the Royal Perth Hospital and I was at one stage in charge of the Royal Perth Hospital emergency department. I am currently employed on a part-time basis by the Department of Health in Western Australia, in a clinical advisory capacity and in the project development unit. I would like to stress that the views I am going to express in this court case are not the views of the Department of Health of Western Australia, if I may say that.

Q. Now, you are experienced with what I generally call HIV.

A. I became interested in HIV back in 1981 like a lot of doctors did because it was new, something interesting, terrifying at the time as I recall, and of necessity because just about everything that can happen in medicine happens in emergency departments and we had to learn about this disease. I possibly became more interested in it than a number of my colleagues and I knew a lot about - in the years before we had HIV there was a couple of years between 1981 and 1983 before HIV was accepted to be the cause of AIDS when people were wondering what it was caused by and I cooperated with her and I became interested in it and another reason I

became interested in this topic, especially the antibody test, because we in medicine treat needle stick injuries which involves the antibody test and I was concerned to know that the tests we were ordering were rigorous and could be relied upon and I had lots of patients with needles stuck and I had colleagues needle stuck and I have been needle stuck myself and I developed an interest in this topic because of that, and I suppose I have spent 25 years reading about this, studying it, thinking about it. At one stage my children asked me how much time I had spent on this and I worked out I had spent the equivalent of two undergraduate medical degrees studying the literature. I have written several papers. I have co-authored several papers and I have spoken at the South African Presidential AIDS Council Meeting and I was invited to that and I have published some invited papers as well and I supplied those with my affidavit.

Q. You have made a reference in relation to HIV to 1981, I think I heard that right, was that right.

A. Sorry I meant AIDS first appeared officially in July 1981 and there was a lag period of a couple of years before anyone knew what caused it or what the current accepted cause is.

Q. And you use the expression 'needle struck'. You had better just explain that.

A. It is common. Unfortunately it is common for people working in emergency medicine, and especially also in hospitals in general, to get stuck with needles that have been in patients. Taking blood, you turn around, it is crowded and there are too many controlies in the cory door and you get jabbed yourself and so everyone thinks if that happens to them there are going to die of AIDS. And so we deal with those people and there is a protocol which, as I said, involves performing antibody tests.

Q. Now, moving to your presentation and as with Mrs Eleopulos, basically you present it. We start with the

proposition the HIV infection is diagnosed by using antibody tests; is that correct.

A. That is correct.

HIS HONOUR: There are a series of slides we are about to deal with.

EXHIBIT #A6 SERIES OF SLIDES CONSISTING OF NINE PAGES TENDERED BY MR BORICK. ADMITTED.

A. Your Honour, may I just ask you a question?

HIS HONOUR: Yes, certainly.

A. Would it be permissible if I sometimes refer to speaker notes during this presentation?

HIS HONOUR: Any objection?

MS MCDONALD

Q. I suppose I should just see what there are. Are there any more than what is contained in the PowerPoint documents that we have.

A. There are.

MS MCDONALD: I can take an opportunity to look at them after.

HIS HONOUR

Q. Doctor, you refer to your notes. Can there be made available for Ms McDonald to have a look at.

A. I can make them available, not today unfortunately because there have been revised a little bit from the originals but I can certainly make them available to the

court.

Q. It is really Ms McDonald rather than me.

A. Okay. Well, I mean if there can be photocopied.

MS MCDONALD: Just so I can have a quick look.

MR BORICK: It will be a bit difficult to have a quick look.

HIS HONOUR

Q. Can you just hold them up for me.

A. That is the first page.

Q. And how many pages are there.

A. There is a page for each slide so I know which slide to tell you.

MR BORICK: It is the notes of his evidence in effect.

HIS HONOUR

Q. So, what have we got, about 40 pages odd or something like that, maybe 50.

A. 55.

HIS HONOUR: They can be photostatted overnight.

MS MCDONALD: I am content for the witness to use them for the remainder this afternoon.

HIS HONOUR: They can be photostatted overnight.

MR BORICK: Yes they can but what's happening is you are going to hear it in evidence. There will be few changes, not many.

HIS HONOUR

Q. If a copy of the notes can be provided overnight, that's fine.

A. My pleasure.

Q. So you refer to your notes if you need to.

A. Thank you very much.

XN

Q. Now, we will start with A6, starting again with 1, which we are now looking at and you proceed.

A. Slide 2 actually. Antibodies are proteins produced by cells of the immune system known as B-lymphocytes. The B stands for bone marrow derived. These are different from the T-lymphocytes which are depleted in the

bloodstream of AIDS patients. I should apologise, some of this is repetition from my colleague's talk but I am sorry, it is difficult to avoid that. Any substance which generates the antibodies is known by the generic title 'antigen' which is just derived from the initial syllables of 'antibody generating', as is on the slide. Now, normally the body does not produce antibodies against its own self components because normally the immune system can discriminate between itself and non-self but there are instances where this breaks down and the body does produce antibodies against itself and they are called auto-antibodies and I bring this up at

this stage because it is important for the argument that I present later on. The concentration of antibodies in normal healthy people is about 15 g per litre and AIDS patients, and in fact HIV positive patients, typically have levels which are higher than that and add up to 25 g per litre, about 50% higher. So at least in terms of antibody concentrations, they are not deficient, although you could say there are in surplus. And I bring this up because this is also part of the argument I am going to present later on, those two facts: auto antibodies and high levels of antibodies in AIDS patients. Out of interest, the human body is thought to have a repertoire of about 1 million different antibody molecules to be able to produce that.

Slide 3, just to remind you that serum is where all the substances that you need to live are dissolved and it also includes the antibodies. And as my colleague said, because serum is used in antibodies sometimes this practice is known as serology and people are referred to as sero-positive or sero-negative as appropriate.

Slide 4. The antigen that induces the antibody reacts with the antibody. That is, two actually combine chemically, it is a chemical reaction. You can demonstrate this reaction outside the body by taking some serum and adding it to the antigen which is in a test tube. As the reaction takes place, some physical

alteration in the reaction mixture occurs. Often this is a colour change which can be measured by some means. It is the colour change that tells the laboratory scientists that there has been a reaction. This is essentially what an antibody test is. Now, how come we can use antibody tests for diagnosis? Well, as I said at the beginning, it relies on the fact that foreign substances induce antibodies. So if a person is infected with a bacteria, bacterium or a virus for example then that person will produce antibodies directed against the antigens in that - because they are foreign, such as protons, and these can be detected and

tested.

HIS HONOUR

Q. Can you just put that in some plainer language that I and others who are not medical people might understand.

A. My fist is an antigen, it is foreign to you. My body has a system, the immune system that actually recognises its foreign and produces an antibody - here it is. And it has the right size and shape to grab hold of that and combine with it. And if I can somehow demonstrate that I have got this in theory then I can demonstrate that you have been exposed to that. And I can demonstrate it by actually getting a bit of this from somewhere.

Q. That's the antibody.

A. No the antigen creating from the virus, putting it in a test tube, adding some serum from you and if I see it change colour I can say 'Yes there has been a reaction between the antigen and the antibody' and that is presumptive evidence that in fact you have been exposed to that antigen.

Q. So, let's say I have got an infection.

A. Sorry?

Q. Let's say I have got an infection of some kind.

A. Yes.

Q. It doesn't matter what it is.

A. Measles.

Q. Well, let's take measles. So, if I have got measles

will I be creating some antibodies.

A. Yes.

Q. To fight off the antigen.

A. To fight off the antigen. What the antibodies do in the body is they are said to help neutralise the toxic effects of that organism.

CONTINUED

Q. So if I get an infection, say, of measles, I will create antibodies for the measles.

A. Yes, and there are many tests used in clinical medicine to prove that somebody has been infected with a particular agent. So, as I just explained, any antibody test can be used for the diagnosis for the reason I just explained to his Honour.

HIS HONOUR

Q. So if I come into the hospital with what appears to be an infection and I have got certain symptoms -

A. Yes.

Q. - you could take some blood from me, that goes up to the laboratory.

A. Yes.

Q. And they test that blood to see if I have got antibodies; is that correct.

A. That's correct. The common scenario is you would present with a fever and sweating and headache and all sorts of things and you could have virtually anything and we might test you for everything and we might come up with something but I can tell you most of the time we don't actually find out. People have a virus. That's how it is. But sometimes - but it depends what the suspicion is.

Q. Sometimes they can identify it, sometimes they can't.

A. Sometimes the antibody test is positive. Then you have

to decide whether the antibody test fits the critical picture. But there are some - so you can use diagnosis because of what we have discussed, however, antibodies are not a virus and it is only an indirect means of proving a virus or a bacteria and everyone knows that no test is perfect, and no test is perfect. Even x-rays are not perfect. I have operated on patients who look like they have got fractures on X-ray and they haven't because when I actually open them up and look at the bone it is not even broken. So not even x-rays are perfect. So why do we do this? Why don't we just try and find the thing straight off directly without mucking

around basically, if you forgive my expression? The reason is because, in the case of a virus, virus isolation is complex, it is time-consuming, it is expensive and antibody tests are not. They are easy, quick and cheap and it is just a blood test, so they are very popular and they are perfectly satisfactory providing that you establish their bona fides before you introduce them to clinical practice. Slide 5, please. Now, in order to form an antibody test for a virus, three things are needed. You need a blood specimen in which to obtain serum from the patient, you need a source of the viral proteins and you need to determine some criteria if you are actually going to label a positive test. Slide 6, this is just to say that in terms of HIV, Eleni Papadopulos-Eleopulos has already presented the argument for the HIV proteins as cellular. I am not going to discuss that any further. I will get to the point of this in a minute. The next slide, which is slide No.7, Eleni also said that using antibodies, scientists have identified certain proteins in tissue samples of AIDS patients as being HIV. Now, you can take the same antibodies. It is technically possible to get hold of those antibodies and use them to test other tissues. In fact, by doing this, some proteins, for example p24, have been found in situations where everyone agrees there is no HIV. For example, p24 has

been found in healthy blood donors and healthy individuals and also in non-infected organ transplant recipients. Slide No.8, three of the HIV proteins using antibodies have been found in normal placenta, and when you consider the same particles. Also, there is ample evidence that placental tissue reverse transcribes, that is has reverse transcriptae activity, one could reasonably ask: why aren't pregnant woman regarded HIV infected? However, for the sake of argument, I'm going to proceed assuming that the proteins in the HIV antibody tests are those of a unique virus HIV. Slide No.9, now, we are talking about the tests that doctors

order, the routine tests used to prove humans are infected with HIV. This is what is meant by being HIV positive. Now, there are two different tests. One is called the ELISA and one is called the Western blot. Slide No.10 - perhaps if you give me a version of what Trudy has printed, I can use that to refer to the slide numbers because my slide numbers may be different.

HIS HONOUR: No, your slide numbers are the same as mine.

MS MCDONALD: Sorry, I simply have the wrong version again.

HIS HONOUR: We are up to 10.

A. I was just explaining that there are two tests, ELISA and Western blot, and now I'm explaining the difference between them. In the ELISA test, the HIV proteins are present as a mixture represented here by these rectangles, and when you add serum, the test and the antibodies react, you get a colour change which can be quantified by seeing how much light passes through the solution using a spectrometer and this gives you a number. So it is an objective. It gives you - it is an objective test. The greater the amount of antibodies the higher the reading, but the ELISA test obviously can't distinguish which protein is reacting. In the Western blot, what happens is the proteins are electrophoretically separated from each other. Shall I

explain electrophoretic?

XN

Q. Yes.

A. Proteins have a molecular weight and they have a charge and if you put that mixture of proteins at one end of the gel, something like gelatin, to a pole, just a blob of it and you stick a voltage of about 100 volts on the other end, the voltage grading in the gel will drag the proteins through the gel and as they do this they separate because the fast ones don't move - don't go as far as the little ones and so they get separated out on the basis of how heavy they are. But also on the chart,

that's why sometimes you will see p32, for example, is p31, because not every laboratory can reproduce exactly the same conditions in their gels. 'P' stands for 'protein' and the number, as the lady has told you, is for the molecular weight in thousands. So in the Western blot, these proteins have been separated electrophoretically in a thin neutro-cellular membrane and when you add serum to these and there is a reaction and there is a colour change, you can tell the sites, you can tell the actual proteins that have actually reacted with the antibodies. So an antibody to p39 would produce a colour change here and P41 would produce one up here. Normally these are invisible. If you pull a strip out of a kit, you don't see any of these. I have had to put these in to indicate that they are there but imagine that they are invisible. Now - and these are called 'bands'. Don't confuse this with the 116 grams per million band that the lady has just talked about. These are called bands but the sites of the protein antibody reactions are called Western blot bands. So when you hear the word 'band', it means an antibody has reacted with the protein in a certain position by given the name of the protein's molecular weight. Please note, in these Western blots in the diagrams and in the subsequent diagrams that I am going to show you, the proteins are not in electrophoretic

order. I have grouped them for convenience for another reason according to which gel is said to produce them. The genes are known as 'gag', 'pol' and 'end'. It will become apparent later why I have had to tell you that. The next slide is No.11. In Australia, there is an antibody testing which goes like this. These are the two tests I have discussed is the ELISA and the Western blot and they are put together as a system. First an ELISA test is performed. If you are having HIV testing, if you are being needle stuck, first they do the ELISA test. Almost everyone produces some colour change in the ELISA test but you don't get to be called reactive

unless it is over a certain amount. If it is below that certain amount it is called non-reactive and in most cases that is the end of the story for you, you are out, but if it does exceed that particular amount if it is reactive, then you have a Western blot test. Now, a Western blot is done because it is said to confirm whether this is a true positive or not. I will explain that term in a moment but the Western blot is a supplemental test and when you do it you add serum to the strip, as I said, and certain bands may or may not appear, and according to which bands appear, the result of the Western blot is classified as positive, negative or indeterminate. Slide 12, is this understandable? This is a blank Western blot except I show where the protein are, the HIV proteins. You add serum and some of these bands have lit up. In this one it hasn't lit up, it didn't do anything, it is negative. This one here is positive and this one here is not positive or negative so it is called indeterminate.

HIS HONOUR

Q. You are going to explain why it is indeterminate.

A. Yes, I am. In Australia, a positive Western blot is reported when there is at least one of these bands p41, 120, 160, plus three other bands will come from these here.

Q. So it has to be one of those, plus three areas.

A. At least one plus three areas. That's the criteria in Australia. So, you can see that that does fulfil those criteria. We have got one of these three and we have got three others.

Q. That's one of either p41, p120 or p160.

A. Yes, exactly.

MR BORICK: If you go to page 6 of your document, you will see that we will be coming to this in more detail. We will be comparing the different conditions at that level.

A. As I said, the Western blot is regarded as positive, negative or indeterminate in Australia. In fact, these

terms are used everywhere where the Western blot is used and the bottom line is that the positive Western blot is regarded as proof of HIV infection, that is that your reactive ELISA was, in fact, due to HIV infection. A negative Western blot means that you don't have HIV infection, and an indeterminate Western blot, which means it is neither negative or positive on the criteria, in most cases, in the majority of cases, is not due to HIV. When the Western blot report is issued to a clinician, its recommended practice is to list the bands and their intensities as well as interpreting the reports of the clinician. This is typical of all tests. We don't get test results back as normal, high or low, we get the number. We like to deal with the numbers because ultimately the clinician has to tell the patient the news, not the laboratory technician. So we would like to have as much information as possible and that is well accepted in clinical practice. Now, why is there this overview of the ELISA followed by the Western blot. The reason is this. According to the HIV experts, the ELISA is not specific enough to make a definite diagnosis. Hence, if the ELISA is reactive, the mixture of HIV proteins could be reacting to HIV antibodies or they may be reacting to some other antibodies caused for some other reason. Now, the experts claim that by separating out the proteins in the Western blot, some of

the 1,023 possible band combinations are caused by general HIV antibodies while the rest are not. The question is: how do they know that? How do they know which band patterns are specifically due to HIV and which aren't? Now, the word 'specific' is one we have been using a lot in this court today and one which gets used frequently in regard to antibodies and antibody testing. Hence, we need to understand precisely what it means. If we go to slide 13, and I don't mean to be trite but I thought this might help to get the point across. This is an extremely specific test for a certain brand of motor car. In fact, it is so specific

I don't actually have to tell you what it is. On the other hand, this is not, slide 14. This is not a specific test for that motor car because this test is positive for all makes of cars. 100% specific test is one in which a positive result points to only one cause. There is no other cause and if an antibody is said to react specifically with an antigen, then it means it reacts with that antigen and no other antigen.

HIS HONOUR

Q. You have just referred to slides 13 and 14. 13 is the positive, 14 is non-positive.

A. No, no, no.

Q. Slide 13 you can positively tell what vehicle it is, slide 14 you can't.

A. Slide 14, yes - you are right, sorry, you are right.

Q. 13 is a Mercedes Benz, isn't it.

A. Yes, but it is not my Mercedes Benz, unfortunately. I own the tyre. So let us address the point. What is the proof that the antibody tests specifically for HIV infection. I should make the point that HI V experts, including world health organisations, say that these tests are extraordinarily accurate for HIV infection, for diagnose of HIV infection.

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Now, we know if we infect a human with the virus, because it is viral, it will produce proteins, 14 proteins, which will react with that virus and that will show up in any test. Because of this we may think - we may be tempted to think that if we come along with any old thing and even if you know a measles virus protein, for example, find an antibody in reaction to it, that proves that the antibody because of that, but that is not true, unfortunately that is not true, nature does not work like that, it's not that simple. This is because, this is repetition, antibodies are not monogenous, antibodies can react with other antigens. There are many examples of this including one pertinent to the HIV tests. In fact, there's one in relation to measles - you discussed measles previously. It's known, for example, that patients who develop a measles infection develop antibodies, which are measles antibodies, which react with six of the HIV antibodies, and they disappear when measles antibodies disappears. That's the scientific literature. But they are not HIV antibodies, they are measles antibodies. I just point that out because we discussed measles. Another example which is probably more pertinent to the present discussion is that 1% of healthy people not infected with HIV which means some 200,000 Australians have reacted to the ELISA test but they're not infected and

40% of people, which could relate to 80,000 Australians, but that's a jump, but 40% of people from a sample of hundreds, so it's possible, there are probably millions of Australians who have one western block band, in other words, they have one of those bands that light up in the serum and they're not infected with HIV either. So I'm just bringing this up to say -

HIS HONOUR

Q. They'd be in the indeterminant range.

A. Yes, they are, correct. I'm just bringing this up as an example to show, to illustrate that non-HIV antibodies react in the HIV test kits. Not everything that reacts

in those tests is caused by - is an HIV antibody and that's what it is. We admit that. The argument I'm developing, to race ahead a bit, is perhaps all the antibodies that react to these tests are non-HIV antibodies, but we'll get to that.

Q. You tell me when it's a convenient time because we're about to adjourn. So when it's a convenient -

A. Are we adjourning for the day?

Q. Yes, we'll be adjourning for the day.

A. All right, well, I think I know where to finish in a few slides.

Q. Yes, you finish when it's convenient.

A. I'm making the point that non-HIV antibodies can react with a test kit. Even dogs and mice who do not get HIV or AIDS also develop antibodies that react with some of these proteins, including critical envelope proteins which say it's crucial for diagnosing HIV infection. The next slide, 16.

XN

Q. Yes, 16.

A. Elaine has already reported this is Gus Nassal's book written 35 years ago. He was talking about this phenomena, antibodies reacting with antigens, and we've already had this slide. Just to repeat it, that an antibody molecule following the injection of one antigen frequently can combine with a second antigen of a

related or similar shape. That's called a cross-antigen. The next slide, 20 years ago Stratis Avrameus, a scientist from the Pasteur Institute who was a specialist on antibodies, also addressed the fact that an antibody can react with different antigens but he added the antigens don't have to be dissimilar. And the next slide, No.19, just to repeat what Dr John Marcionus, an expert says - it's worth repeating this - the immunological community was shocked to find that antibodies would be polyreactive in binding multiple antigens that ostensibly were unrelated to one another. There was obvious a stage when reactions were thought to

be specific but, as technology has advanced, antibodies are not only monogenous, they are promiscuous and combine with multiple antigens. This creates a very big problem for serological diagnosis which I could develop further tomorrow.

DISCUSSION RE TIMETABLE

ADJOURNED 4.32 P.M. TO WEDNESDAY, 25 OCTOBER 2006 AT 10.15 A.M.

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