MS MCDONALD CALLS

+PETER JAMES MCDONALD AFFIRMED

+EXAMINATION BY MS MCDONALD

Q. You have you produced to the court a curriculum vitae.

A. I have.

EXHIBIT #P87 CURRICULUM VITAE OF PETER JAMES MCDONALD TENDERED BY MS MCDONALD. ADMITTED.

Q. Have you also produced for the court two separate reports in relation to this matter.

A. I have.

Q. I think the first was dated 27 August 2006.

A. Correct.

Q. And then you've more recently done a supplementary report dated 28 January 2007.

A. Correct.

EXHIBIT #P88 REPORT OF PROFESSOR MCDONALD DATED 27/08/06 TENDERED BY MS MCDONALD. ADMITTED.

EXHIBIT #P89 REPORT OF PROFESSOR MCDONALD DATED 28/01/07 TENDERED BY MS MCDONALD. ADMITTED.

XN

Q. You are at least semiretired.

A. Yes.

Q. I use the expression semiretired because do you still have some involvement in relation to HIV.

A. Yes, I probably prefer to say I retired from full-time academic employment at the end of 2003 but I have actually maintained a fairly intense involvement in many of the initiatives that I was involved in into retirement.

Q. Can you give his Honour a bit of an overview of what your current involvement is.

A. My current involvement is dominated by HIV AIDS in as much as I remain a member of the Principal Federal Ministerial Advisory Committee on AIDS, Hepatitis and Sexually Transmitted Disease, as it grew out of the

original National Council on AIDS. I'm the chair of the Scientific Advisory Committee for the Special National Centre that Professor Cooper was - who is the director of. I am also on the Scientific Advisory Committee of the Virology Initiative. In addition to that I chair the Vaccine Management Committee of the big NIH grant on vaccine development and a similar management committee, which is basically the equivalent of a sort of management board that the National Institute of Health require for the development of the vaginal microbicides.

Q. What are vagina microbicides.

A. They are a fairly recent development in the fight against AIDS whereby its recognised that women are less - there is an imbalance of power between men and women in the ability of women to protect themselves against sexually transmitted HIV and there is a series of compounds that have been developed to insert vaginally prior to exposure or sexual intercourse and possibly thereafter. It so happens that there is an Australian developed product called Viva Gel, and internationally there are seven or nine other compounds that are now into human studies.

Q. I want to go back in time a bit because would it be fair to say that you've been involved with HIV since it really was introduced to Australia.

A. Well almost. I came back to Australia from the US in 1975 and had nothing to do with HIV, in fact the first encounter I had with HIV was at an Australasian Society of Infectious Diseases meeting we hosted in Adelaide and brought some Americans out and they described as 'gay playing' at that time, I think that was 1981. In 1982 as part of my international role on the General Infectious Diseases or Society of Infectious Diseases Committee, I can't remember which one at the time, I was in Vienna when Professor Gallo and others presented the case for HTLV or retrovirus as causing AIDS. I personally had nothing to do with it until about 1987/1988 when two things occurred. One is at Flinders

Medical Centre where I was the head of Microbiology & Infectious Diseases we began to get the first few cases of AIDS and in a sense we had to set up a system for looking after them and those were the days when we ended up with a whole six-bedded bay and four separate additional rooms to look after these people that mostly died, and died a horrible death actually, panting away with this nudistas pneumonia and it was really quite a challenge to manage them and the staffs and the families associated with them. At the same time I'd had a fairly, I'll say successful career in research, mostly to do with pharmacology of antibodies and clinical infectious diseases and trials of drugs, mostly in surgical sepsis and then in abscess management, so I had a fairly prodigious output, not so much from me but I got a good team around me. At about the same time I began to play a role nationally through National Health and Medical Research Council and National Granting Agencies, in what I might vaguely call or generally call research management. And then when HIV came along back in about the mid, 85/86/87 there began to be some investment of earmarked funds for research into HIV and AIDS, and that was because it was recognised that certain important findings were needed to guide the management of the epidemic in this country. And at that time there were two streams of, I'll say activity, focussed on HIV AIDS. One was through the National Health and Medical Research Council led by a Professor David Pennington who is ex-Vice Chancellor of the University of Melbourne and still quite active, and that was focussed on there was a sort of biomedical based sciences aspect of it, then there was a separate initiative led by Ita Buttrose and social sciences and community concerned people they didn't actually always see eye to eye with each other in terms of where the resources should go and what was causing this problem and it was Minister Neil Blewett who undertook a community-wide consultation and came up with the first

national strategy on HIV AIDS, which progressively got together, over about '87/'88, it was formally promulgated through parliament in '89. Now under that national strategy they, that is Commonwealth Government, made a financial allocation to cover a whole range of things, from blood transfusion screening through implementation of community-based education and research, and there was an amount of money that incrementally went up from about $4 million to about $10 million over the period '88 through to 1990. I hadn't personally, in fact I had made a deliberate choice not to get involved in HIV research and so on because I knew it was a fairly competitive field shall I say, but Mr Blewett rang me up one day out of the blue and said would I take on the chairmanship of the Commonwealth AIDS Research Grants Committee which was responsible for allocating - no-one was responsible really for outlaying the research that was needed, allocating the funds, monitoring the progress of the research in the country and then applying that research to policy and practice as the epidemic unfolded. One of the provisos in taking that job on was that I was - neither I nor the other experts in research were able to allocate to ourselves any research moneys, it was deemed to be a conflict of interest. So I basically spent from about '88/'89 through to 2002 as chair of this committee of experts that turned over fairly regularly and set about establishing Australia's research capability in HIV, monitoring its effectiveness and then translating it into policy and practice.

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Q. How did you go about monitoring the research and whether it was effective or it wasn't effective.

A. The first thing we did was create what I call the building blocks of research. We knew we needed to be able to grow the virus in Australia - we hadn't got that capability. We knew we needed to track where it was - the epidemiology, who was infected and so forth. We knew that there were various behavioural elements to do with sexuality and needle use or injecting drug use and the capability within Australia to address those building blocks - and I suppose there are the high level ones and there are lower level ones - was sort of patchy, if I could put it that way. We were quite strong in immunology, very weak in virology in this country at that time. The social sciences were not at all well developed in the areas that we were concerned with - that is to do with sexuality and injecting drug use. It was really a matter of identifying those building blocks, putting up a request for researchers to address specific issues and we also established the three national centres in HIV to do with the major areas of interest: the first being the Virology Centre that was headquartered in Melbourne at Fairview Hospital and had reflections all around the country. The second was Professor Cooper directed the Centre of Epidemiology and Clinical Research and the third was the Social Sciences National Centre - that originally went to Brisbane and then moved to Macquarie University and is now at the University of New South Wales. To answer your question: how do we evaluate the effectiveness of that research? It was really two-fold, unlike the free-flowing investigator driven research where people say 'I want to do a research on this topic' and they go and do it if they can get the money. When we - I'll say - let research contracts really as much as grants, we specify the topic and review the output on it annually. For example, when you see Professor Cooper, he's got a lot of activities in his unit and every year that national

centre puts up a work plan to do with where they propose to go in the next year. It was my and my committee's job to review that work plan and to see whether it was consistent with what we believed was needed, in research terms, for the epidemic and, at the same time, we reviewed how much progress they had made in the previous year to sort of fulfilling the goals of the work plan. It was very much a hands-on activity and I personally regularly visited these places around the country to observe what was going on.

Q. Would it be fair to say that over those years, if there was commonwealth money involved, you had some role in overseeing and ensuring that the money wasn't wasted.

A. That was the primary concern - I do really believe at the beginning, for the first several years, the real concern of the commonwealth, and certainly the politicians, was to make sure that this epidemic was adequately addressed.

Q. You have been in this court throughout the course of the evidence of all of the witnesses but, in particular, those witnesses called by the prosecution.

A. Yes.

Q. The views that those witnesses expressed, in terms of the existence of HIV, the question of whether it is sexually transmitted and the developments there have been, in terms of treatment of HIV, do you agree with the opinions that they offer the court.

A. There is nothing that any of the expert witnesses for the prosecution said that I would disagree with.

Q. The evidence they gave in this court was typical mainstream scientific opinion.

A. Yes.

Q. Were you involved or did you play any role in relation to the AZT trial.

A. The AZT trial - you mean in Australia?

Q. Yes.

A. The evidence that AZT was effective was published in mid-to-late 1986 and that was a placebo controlled study

in the New England Journal of Medicine, that we can table, if you wish - I think we might have already done so. When that information came out, Australia was probably at the peak - I'll say - of the death of people with AIDS. Every week there was a funeral that people were going to and some quite high profile people were involved. There was a lot of concern to get on and if there was something that was successful in therapy, then we should take it up. Through the National Centre in Epidemiology and Clinical Research, we provided funds to conduct a trial in Australia on the effectiveness of AZT, which was both a reconfirmation, if you like, of the original study but without a placebo but, just as importantly, it was really to help develop the health service infrastructure needed to distribute and monitor patients and so on. This was the very first time this sort of infection had been treated and, subsequently, we developed viral loads and CD4 counts and established the National Reference Laboratory and quite a lot of things happened over a small number of years.

Q. You have heard evidence from a number of witnesses about the impact of antiretroviral medication, both within Australia and internationally, and comments to the effect that that's one of the real achievements so far in the science, in relation to HIV. Firstly, do you agree with those general observations.

A. Absolutely.

Q. You yourself observed the effectiveness of the antiretroviral medication.

A. Oh, yes. Like the others, in the early days in Adelaide I was the only physician actually treating HIV, for a start, until the Royal Adelaide developed bigger clinics. As I said earlier, at Flinders we had a whole bay and some side rooms that were always full of dying people with AIDS - not just HIV. Within a year of AZT access, the ward was nearly empty and has remained so to this day.

Q. You have had some involvement internationally with

different research programs on the international stage.

A. Yes, mostly through those vaccine and therapeutic trials. I should say that there's an organisation called HIVNAT - HIV Netherlands Australia Thailand - it is headquartered in Bangkok but it is a joint venture between the Australian national centre that David Cooper directs, the Thai Red Cross AIDS research centre in Bangkok, but the Thai Red Cross actually has a huge role in HIV because it runs hospitals and community clinics and orphanages and it is a very big organisation, and the equivalent to Professor Cooper's organisation in Amsterdam, called NATEC, led by Professor Joep Lange. The purpose of that was to bring together the developed world expertise and capability with the Asian one - primarily with a view to undertaking clinical research on the use of drugs in that region and establishing the laboratory infrastructure that is essential for managing treatment. I became involved because David Cooper basically said he wanted someone on the board of that international advisory board for that initiative, so I joined that, together with an American and another Dutchman and some officials from the Red Cross. That led to quite a few trips - that is not the right word - comings and goings to Bangkok and then, subsequently, Cambodia around the range of trials. When we started this, the most heart-rending thing from my perspective was the paediatric AIDS, where between 20% and 40% of the HIV-positive mothers, and there were many, many, many thousands up in Thailand at the time, their offspring with infected with HIV and invariably died that horrible death over a small number of years. As soon as AZT came in and that 076 trial that's been referred to earlier, demonstrated some efficacy in preventing maternal child transmission, it just worked like a charm in preventing the babies getting infected - when I say 'worked like a charm' it wasn't 100% protective but, over the years, it improved dramatically. A consequence of that was that instead of

having sick babies dying at age two or three or four with AIDS, the babies were born uninfected by HIV but their parents - the mother in particular, and father - often died within the first year of delivery because the treatments that were provided to the mother during her pregnancy or late pregnancy were not able to be carried on because of the expense and the fact that there weren't all that many drugs. There was this whole, nearly a decade, where there were all these AIDS orphans, as a consequence of treatment which sort of poses a few ethical dilemmas which are now solved because the United Nations global fund is providing treatment for the parents of babies who are born to HIV-positive mothers and treated. That has been one of the most startling observations on a population basis, even though I never actually practiced in Thailand and looked after them hands-on.

Q. You have just touched on the United Nations there. I want to ask you some questions about some of the big developments internationally. His Honour has some documents already that relate to these issues. The first is the Durban Declaration, just tell us about that.

A. The Durban Declaration - prior to the Durban conference, which was an international AIDS conference, there was increasing concern about the statements by the leadership, if I can put it that way, in South Africa, denying the existence of HIV and attributing it to other factors and it was very clear that life expectancy was dropping dramatically and the only sort of common factor was HIV. I think it was about 5,000 scientists around that time developed the Durban Declaration, which I think has been tabled. That led to some changes in South Africa, at the time, but really not too many for quite a while longer. What it did lead to was a greater awareness by the United Nations, in particular, who was gathering all these statistics from around the world and getting concerned about sub-Saharan Africa, in

particular, but also other parts of the world, where there was not only concern for the welfare of the individuals but the GP and GMP of the countries affected was dropping off and the world bank got involved. It is seen as a serious global impact of HIV, that something needed to be done about it.

Q. There's been reference to a United Nations General Assembly Special Session.

A. Yes, the so-called UNGAS, I think you could probably say it was the Durban Declaration, but Kofi Annan, in particular, and several people in the United Nations really took up the AIDS cause and it took several years to actually get a United Nations general assembly special session up. It is not something that happens too often and that was the one that gave rise to all countries signing up to the final statement that was put out, which, the landmark of that UNGAS, as far as I'm concerned, is that it actually linked treatment to prevention, which is something in Australia, I think, we had long experience with and were agitating for years and years and years, to make treatments more widely available, particularly in the developing world. The reason for that is that you can only actually acquire HIV from somebody who's already got it and if you make treatments available to people who are symptomatic and whose life you can prolong by provision of antiretroviral treatment, you have identified little cohorts in the community and you can direct various activities, from education and condom use and so on, to those pockets of the population. In addition, we didn't know this exactly at the time but we had a feeling that transmission was directly related to viral load of the individual and by provision of treatment you lowered viral load and therefore diminished the infectivity, and that's a public health approach that's been used for TB control for many, many years.

Q. Was there some sort of fund put in place to enable this to occur.

A. Yes. That's the so-called global fund which was established to combat HIV and AIDS, malaria and TB, because there was some concern that HIV was the only focus of attention and, actually, they all go together and it doesn't make a lot of sense to just pick off HIV.

Q. I want to turn and deal with a couple of particular topics; firstly, there was some evidence given by Ms Papadopulos about a conversation that she purported to have had with you in about 1990.

A. Yes. I must say I heard her give that evidence and I do recall - as part of my travelling around the country - reviewing research initiatives, calling on Professor French and Professor Shellum, actually, who was doing work in WA at the time. I was vaguely aware of the witness and when I was at the Royal Perth Hospital I do recall seeking her out and I think she was anxious to talk to me because I was in a position of - I won't say allocating funds for research - but chairing the funds allocations committee. The reason that I was interested to meet her is that at about that time the whole mechanism, whereby HIV caused AIDS, was far less certain then than it was now and from other sources - largely French, actually - there was some evidence that this oxidative metabolism process was involved in something called apoptosis, which is a fancy word for cell death. The disease AIDS is typified by the cell death of these CD4 cells, so I thought it was reasonable to meet with her. I can't honestly recall the nature of the conversation in the way that the witness related. I assume I said things as she reported it. I did, however, form the impression that she was not undertaking a level of research that would be of much value, in terms of the chemistry and so on, because that's not what she was on about - she was a radiation physicist. I'm sure I said some nice things to her - like she's eligible to put in an application but not to be too hopeful about the outcome.

HIS HONOUR

Q. I presume you'd tell anybody that you could to put in an application.

A. In that sort of position you have to because you can't be seen to prejudge whether someone is going to be able to deliver on a research project. That is my standard response. A lot of people came seeking funds.

XN

Q. In terms of the theory she was putting to you, did there appear to be any scientific relativity to what she was saying.

A. You are testing my memory of a fairly brief conversation that I probably didn't diarise other than 'I attended her office' or something. My recollection was that the discussion with her was about sort of these oxidative cellular activities and not about her theory that HIV didn't exist, or that AIDS didn't exist. My memory could and probably is flawed. That is certainly the understanding that is still in my mind about it.

HIS HONOUR

Q. But even if she had come to you and said 'Look, I'm doing some experiments from which I think you can conclude that those who claim that HIV is a virus' - 'exists as a virus that results in AIDS, if untreated', I assume you would have said to her 'If you have scientific research you want to put before our organisation seeking a grant, put it before us and we will assess it'.

A. I'm certain I would have said this. At about that time we had what I call a full suite of research categories. We had project grants for small groups, we had program grants for teams coming together to solve a problem and we had these national centres, we had training awards, and within each of those we had sub-categories, and I was often approached by potential researchers to say 'Well, which category, which grant, which whatever, am I eligible to apply for?' and I usually pointed that out. If they applied and were successful, that is good,

otherwise, just like the National Health and Medical Research Council, or the National Institute of Health in Washington, anyone can apply for anything. It doesn't guarantee success.

XN

Q. Moving on to another topic: reference was made during the evidence of another witness to the Sydney Blood Bank Cohort Study. I can't remember which witness it was, but whoever it was said that you might be able to tell us about that particular study.

A. Yes. An observation was made - John Kaldor probably said that in his evidence, might have referred to that, or Martyn French, or Dominic Dwyer, because they were all involved one way or the other. It refers to a blood donor in 1984 and 1983. This was prior to screening of the blood bank donations. Surprisingly, this donor - there were 15 recipients of his transfusion, or it might have been 14, I'm testing my memory a little bit about that, but I can get the particular articles that were written about that, or they are probably in John Kaldor's CV, actually. Anyway, there were 15 HIV-infected blood transfusion recipients. One of them died and another was lost to follow-up, but they remained well for at least 10 years after receiving that contaminated transfusion. That created a little bit of excitement amongst epidemiologists and virologists and transfusion services and, ultimately, they cultured the virus out of both the donor and most of the recipients who were still well and had no other signs of progression on to AIDS, but they were still HIV antibody positive. The actual virus that was grown out of those people was identical in the terms that have been previously described to this court, but it had a deletion in the nef gene. I think we've had enough discussion about various genes in HIV. The excitement related to the fact that maybe this was a rearrangement of genes in the nef region, and, if that was a strain associated with long-term non-progression, which is the

term used for these, then it might be pointing the way to vaccine developments. So, quite a lot of effort was put in looking at nef gene deleted, potential vaccinating strains. Unfortunately, as that work was proceeding, some of the long-term non-responders began to become ill, so it was a lead worth following that was terminated about seven or eight years after that research commenced. I personally played a role in assessing the value of investing in that research and so on, so I kept a fairly close watch on it.

Q. You have referred already, I think, to vaccine trials that you have been involved in.

A. Yes. When I say I'm 'involved' in them, I suppose I would describe myself as the person who puts these things together, knows where the expertise is and creates the teams. In the late 1980s there was pessimism about the ability to develop a vaccine for HIV. The pessimism was around the fact that this was a hugely variable virus within an individual and between individuals. All vaccines developed were merely developed for a strain of flu, or a measles, or something with a stable antigenic expression. If you have a moving target, as Dr Gallo said, it is very difficult to make an antibody to one and then a variant pops up, so there was a bit of pessimism at the time. The other basis for pessimism is this integration of the RNA of the virus HIV into the DNA of the host and how can you get a vaccine to address that issue, but at the same time there were major developments in immunology of T cell immunity. Australia has a long and proud history of immunology, particularly in that T cell area, arising largely from the work of Sir McFarland Burnett in Melbourne, and then at the Australian National University with, I suppose, Frank Fenner and Gordon Ada, and then Peter Doherty and Ralph Zinkernagel, and they explicated the whole process of T cell or cellular immunity. I might just indicate that there is antibody-based immunity and there is T cell immunity.

The body's response to things like bacteria and foreign particles is to mount antibodies and code those bacteria and they are engulfed by fagocitic cells, the neutrocytic cells in the blood and tissue. If you have a cancer cell or a virus-infected cell, the very presence of the cancer genes that are called oncogenes and the presence of the virus inside a cell cause what are called neoantigens on the outside of the cell that the virus or the cancer is in, and it is those cell surface antigens that attract the T cell immune response, and in order to eradicate the virus, basically you kill the cell that contains the virus and then the virus is sort of eradicated, the same, potentially, with cancer cells. So, we have a lot of expertise in that in this country. Coincidentally, the CSIRO was doing great things with animal vaccines around, for the court, I'll say a variant of small pox virus that was the fowl pox virus. That is a virus into which you can put a large number of genes that will then express in that fowl pox the proteins that the genes code for. So, you can get this pox virus to express nef, or gag, or pol, or all of those things, and the intention was to introduce those into animals and then move down to humans ultimately. By the early 90s there was an effort mounted internationally under the banner of the International AIDS Vaccine Initiative, and the National Institute of Health in the US also put a huge amount of money into vaccine development, there was a special call, selected tender, I suppose, put out to groups that were deemed to be in a position to go down this path. The Australian group, if I can summarise, was invited to put a proposal together, and we did, and we have been going down that path ever since. Now, this is not myself. I'm chairman of the board, I suppose. It is all these other much more skilful scientists and clinical trialists and social researchers even that do all the work and I just keep them going in the right direction. This Australia group identified the genes, inserted them into the fowl

pox, did all the pre-clinical safety studies in rats and mice and then makak monkeys and ultimately got to the point of administering to humans. We are right at the cusp of that now because, in the next two or three or four weeks we are commencing in Thailand, after some initial studies in Sydney, a group of Thais being vaccinated with this Australian-developed product.

Q. To make these vaccinations, do you need to have an understanding of the genome of the virus.

A. It is vital.

Q. Can you explain, in very simple, lay terms, why that is.

A. It is vital because, firstly, you can't give HIV, or an attenuated form of HIV, to a human; it is unethical, unsafe. The history of vaccines is that you can fool the immune system, if you like, into responding to the important antigens of the infecting organism, or potentially infecting organism, by taking another harmless virus, like this fowl pox one, and selecting out the genes that code for the proteins or elements of HIV that attach to the host human cell and enter and then sort of undergo the replication cycle. So, you need to know exactly what is in the genome and where it's coding for, I'll say gag, pol, nef, env, we don't know what the best ones are, other than, the more you get in, probably the better it is. So, that is why you need to have a fairly precise idea, not generally of the genome, but actually very specifically, because you have these clades that have been referred to, and if we are framing a vaccine, as we are doing basically for Asia, which is a mixture of clade A and E, you would want to take genes from HIV molecular clones, they are called, for the A and the E clade, so that is why you need to know a lot about them, splice out the segments you are interested in, and, at the same time, make sure that you take out things that might potentially be harmful in human use. The reverse transcriptase gene would be a good example of you wouldn't want to put that into a human. Even though we have heard many, many times in

this court that there is a specific HIV reverse transcriptase that is different to the reverse transcriptase of HTLV1 or other endogenous viruses, and is distinct from the similar sort of enzyme that is in human tissue, and that is a safety thing, so you need to know exactly where things are so you can splice them out and not put them in your final product. So we in this country have just been going down that path for quite a long time.

Q. On to a different topic: you have heard a number of witnesses questioned about a virology publication by White and Fenner.

A. Yes.

Q. And the third edition was the one that Ms Papadopulos referred to on numerous occasions during her evidence, then we have heard reference to there being a fourth edition during the evidence of, I think it was, Professor Dwyer. Have you obtained for the court a copy of chapter 35 in the fourth edition which relates to retroviridae.

A. Yes.

EXHIBIT #P90 CHAPTER 35 OF THE 4TH EDITION, MEDICAL VIROLOGY, BY WHITE AND FENNER, TENDERED BY MS MCDONALD. ADMITTED.

Q. Is there direct reference to HIV in this chapter.

A. Yes, there is.

Q. I take you to p.538.

A. Yes. This is a chapter covering the field of retroviruses and starting on p.538 is a segment on human immunodeficiency viruses.

Q. I won't take you through it all. His Honour can read it for himself. For example, at p.547, there is discussion of the genetic variation in HIV.

A. Yes.

Q. Then, over the page, HIV is the cause of AIDS.

A. Yes.

Q. Under the heading, the authors there note 'The case of

declaring HIV the sole etiologic agent is now overwhelming'.

A. Yes.

Q. It goes on to describe 'HIV can be cultured in virtually all cases of AIDS, it grows preferentially in CD4 T lymphocytes' and so on.

A. Yes. I thought this was sort of a neat little overview of HIV or the retroviruses in general, but I must say this fourth edition was copyrighted in 1994, so it is actually quite dated, but the third edition was copyrighted in 1986, and if you recognise that it takes at least a year between deciding to write a book and actually copyrighting it, it is more likely you are using information of two years back.

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So that third edition would have been being developed at almost exactly that famous 1984 period that people weren't sure whether HIV actually existed. So it's not unsurprising that there was almost no reference to HIV or retroviruses in the third edition and there was in the fourth edition. My recollection is that Mrs Eleni Papadopulos referred to the site for identification and speciation of the viruses that was specified in the third edition. It's my view - and it went through isolation, purification - it's all in the transcript. It would be my contention that all of those things have actually been undertaken with HIV. And I think this just confirms that it's the same textbook 10 years after the previous one, and it clearly accepts, according to their criteria, that HIV and retroviruses exist. So, your Honour the other day had an exchange with Dominic Dwyer between the third and fourth edition. I just thought this might be helpful to confirm that that was a very perceptive exchange and that things have moved a long way in those 10 years.

HIS HONOUR

Q. Could I take you to p.540.

A. Yes.

Q. There are some electron micrographs, a figure of 30-4 at the top of the page.

A. Yes.

Q. Could you just explain to me what that figure represents.

A. It represents electron micrographs, and they haven't reproduced all that well here, of cells of the type that Dr Gallo described this morning. And the viruses - the retrovirus itself is perhaps best seen in - I think it's H, the middle lower panel - where you've got that outer coat with the gp120 projections or things that have been referred to as knobs so that's sort of isolated from the grunge around. Now they are not all HIV-1. A to E is HIV-1 and visna viruses, which is an animal lentiviruses that you heard about this morning, is demonstrated in

some of those other lower panel things. And the reference is that Gonda, who was again referred to this morning. The quality of these is not at all good in its photocopying.

Q. It really comes back to this question, it might be very simplistic and you tell me if it's a meaningless question or simplistic. One of the criticisms that have been made by Ms Papadopulos-Eleopulos and Dr Turner is that the viruses has never been photographed. There have been a number of witnesses who have said that is not correct.

A. That amazed me to be quite honest because it started back in 1983 when people thought they had a strange virus and it was photographed by electron microscopy, at the time. I mean it's in those science articles that Gallo articled, Montagnier did it, took some pictures of it. It was less than perfect because of the nature of the material at the time. Progressively over the decades as cultures have become purer and purer, almost cauldrons of retroviruses have been developed and there has been enormous opportunities to photograph the viruses, and several of the witnesses produced pictures I think which have been tabled with the court. It's quite a sophisticated - I was going to say game of electron microscopy these days because there is different computer-assisted approaches to doing it. I mean there is absolutely no doubt in my mind that this has been photographed many, many times. You can Google it.

Q. Are any of these depictions photographs. That's what I was going to say.

A. Sorry. Well, they are. A-E is HIV and, if you start at A, you've got the process of that virion -

Q. Coming out of the cell.

A. Coming out of the cell. You recall various people have described the fact that the viruses has got to parasitise off the host cell, it has got to process and then bud out. In that process it takes some of the host

site. So you've got that process showing the beginning to emerge in A, in pinching off almost in B and then it becoming a separate particle in C, and the definition in D and E is really not very satisfactory here and you've got a similar thing for visna viruses. And I guess a point that could be made here, and Gallo and Dominic Dwyer all pointed out that photographically there is not a lot to tell in terms of differences visually between a HTLV1 virus and a simian virus or a visna virus. And if you look at C above and C below, it's a terrible rendition, they don't look amazingly different to the naked eye. The value of electron microscopy is actually getting much better preparations than this and particularly with sophisticated approaches and computer assistance. You can actually work out what the external and internal structures look like in very great detail. At that sort of ultra microscopic level there are differences but very subtle between the various forms of retroviruses.

Q. Just one other thing to help me because I'm not sure that I necessarily understand all of these things. Looking at the diagram below, that's 35.5, it's a schematic diagram of the HIV-1 virion. The strands that you can see right in the centre of the diagram, there are two of them, which are doubled sided, and in between there are some dots which have a label. Are they what you would call a strand of DNA.

A. Yes. On the right-hand side we have got reverse transcriptase, and then under that single-stranded HIV-1 RNA, and it refers to two strands curly ones of RNA. So there is a pair of RNA gene sequences if you like there.

Q. You'll see on the right-hand side between two - I'll call them knobs for want of a better word, I think the previous witness called them envelopes. There is a dark single not squiggly but not straight line. Do you see that.

A. You mean -

Q. If you look above the title 'Single stranded HIV RNA 1

A' you'll see an envelope or a knob.

A. Yes.

Q. Above that there is a black line with a kind of a squiggle at the end. Can you tell me what that represents.

A. It's the same as on the left where it says 'Host proteins'. I don't know which particular ones - in order to - which ones they are referring to there. But the knobs are absolutely virus specific gp120 that's made as a consequence of the gene sequence in the RNA, or actually it's made as a consequence of the DNA that is integrated into that host cell. And in this process of budding you do create a unique enveloped virus where you wrap up the internal nucleic acid and, things like reverse transcriptase in an outer coat that is a mixture of structural proteins which are often taken from the host cell intermingled with absolutely virus specific knobs, if you like. And this is where you can get into some sort of issue with what is a virus at the end of the day. Because clearly the dotted dark bits between the gp120 knobs and the little black extensions reflect elements of the host cell, or the host cell membrane more likely, that are integrated into that outer coat of the virus. So the outer coat is basically a mixture of HIV gene specific viral products plus some host cell products. It makes the whole virus absolutely unique in the sense that it a mixture of some host cell products, together with viral specific things creates a specific entity.

XN

Q. We might finish up with some more photographs. Looking at this document, produced to you is a two-page document. The first page shows two electron micrographs, and the second page just relates to where the images are sourced from with the heading 'HIV molecular clones' handwriting onto the document.

A. Yes.

EXHIBIT #P91 DOCUMENT HEADED HIV MOLECULAR CLONES TENDER BY MS MCDONALD. ADMITTED.

Q. Can you tell us what we see in those two images.

A. Yes. What you are seeing in the upper panel of the photograph is a series of images of HIV, and on the right-hand side that you see one budding out of a cell just - it's a better photograph in fact than the ones we were previously discussing. The cell - the thing down the bottom out of which that virus is budding on the right is one of those continuous culture cell lines that Dr Gallo was describing which actually produces lots and lots and lots of virus. Now a molecular clone is essentially the entire gene of the HIV or any cell livised. And by using complex biotechnology systems you can get the gene into a plasma and transfect the gene into that cell culture and it begins to produce lots more virus, and those viruses are a very fertile ground for studying various protein manufacturers, processing a virus and so on and so forth. That is what the topic about the article is about, it's how it prohibits the gag mechanism in the list. Ms McDonald, you've actually taken me to the photographs. But I suppose - against the background of discussion of purification isolation in this court this would be perhaps the best evidence that HIV exists, its gene has been sequenced and taken out of the type of virus we were discussing previously in that textbook, and the gene is taken out without any of that outer coat and core and then just the naked RNA, if you like, put into a totally separate cell and it then produces more HIV that's actually got gp120 and all the knobs on that come in part from the cell that its transfected. So, basically that's evidence that you can take the whole gene sequence, put it an uninfected cell culture and then cause more virus to be produced. And that has become pretty much the standard for producing large amounts of virus. And the lower panel is really just a close-up of the upper panel. And the darker core

is outlined there. And I suppose that can give your Honour some idea about - you can actually take a photograph of that and sort of get quite good images. If you want to. But this is a molecular age. And in a sense images done be electron micrographs are far less frequently taken these days because if you know it's a unique gene it's nice to have a photograph to put in an article, and it confirms in a sense that it looks like HIV would be expected to be. Perhaps a light hearted comment; one of my earlier colleagues when we were talking about this stuff Ian Gus used to say 'If it looks like a duck and quacks like a duck it is a duck'. A diagnostician I know of is prone to saying 'You can say this is a mouse, it looks like a mouse, it squeaks like a mouse. But then you can go further and say this is a valve C mouse, we have its entire geno' and we don't actually have to see the mouse, we can take a hair from the mouse or the tip of its tail and say this comes from a mouse. I mean it's probably a bit trivial but it sort of indicates that it's the molecular end of the business these days that provides the vast amount of information about the virus or the animal.

CONTINUED

HIS HONOUR

Q. It may be professor that lawyers like to see, feel or touch. They find it a bit difficult when they can't.

A. You can see the photograph.

Q. We've seen the photograph.

XN

Q. I just finish up by asking you this: clearly in the medical and the scientific world there's room for differences of opinion and debate about developments and new discoveries. The sorts of arguments that we heard advanced by the two defence witnesses in this case, does that form part of any legitimate scientific debate that's going on in the world at the moment.

A. I don't believe so. I've sat in this court for many days and I sort of wonder if the world began and ended in 1983 and '84. There were some important observations made then and they were seminal, but there's been a huge amount of sort of development since that time. This particular discussion we just had is an example of that much more sophisticated later development. That's the year 2000 so it's even a few years old now but that represents a much higher level of sophistication than the original 1983 stuff. In 1982, '83, '84, molecular biology, PCR, all of that stuff was just beginning to emerge and be refined, and now there are machines that do this sort of thing, so it's a vast development over a period of time and I think we should be looking at the recent evidence and not the - we should acknowledge the seminal observations of Montagnier and Gallo, but they certainly have been superseded the work of those people by many hundreds if not thousands of others. Much of the time, of course, has been focused on sort of the theories advanced about the lack of evidence to support the fact that HIV even exists and how it might be associated with disease or spread and so on. There's an element about that whole process and timeliness, as I call it, that the court hasn't really I think been apprised of. I am now speaking as someone who has been

at the interface between the research, almost commissioning it, and then using the value of that research. In the early stages, and I am speaking from the Australian perspective, we knew that there was a new disease around which we called AIDS. It was pretty apparent that in Africa and South America and so on it was spread heterosexually. It was also fairly clear in the US and in Australia that it had a concentration in the gay community and amongst some drug users, so back in the late 80s there was certainly recognition of the potential for harm in Australia, but we hadn't quantified it. There was not a lot of precision about the epidemiology in the late 80s in Australia, so a decision was made - I was part of that I suppose, I sort of wonder whether it was a good idea or not - to go very public with that Grim Reaper campaign that was clearly targeted at the whole of the population against an uncertain knowledge base about its epidemiology in this country. In a sense, we got it wrong because subsequent analysis of all of the people becoming infected and transmissions in Australia shows it was really focused in the gay community, or men having sex with men. Over the next three or four years the whole focus of the campaign changed to just targeting those risk groups rather than spending a lot of money on television ads that interestingly resulted in a major reduction of other sexually transmitted infections in the community like gonorrhea and so on, but really it didn't have a lot to do with an AIDS risk for the population as a whole.

HIS HONOUR: Mr Borick, you would like me to adjourn until 10 a.m. tomorrow?

MR BORICK: Yes.

ADJOURNED 3.55 P.M. TO TUESDAY, 13 FEBRUARY 2007 AT 10 A.M.

SULAN J
NO.65/2006

R V ANDRE CHAD PARENZEE

TUESDAY, 13 FEBRUARY 2007

RESUMING 10.01 A.M.
+PETER JAMES MCDONALD CONTINUING
+CROSS-EXAMINATION BY MR BORICK

Q. In your original report to the court you said 'Indeed, Eleopulos raised some valid questions about transmission of HIV infection in the 1990's' and in the second report you said 'The denial argument about the existence of HIV tends to focus on early experiments and methods which were the subject of legitimate debate, for example the use of p24 as a marker of growth of the HIV. Since that time methods have been extensively refined, become routine',' and then over the page you said, in relation to the evidence of Papadopulos and Turner 'Much of their evidence was anchored in reports of the 1980's and 1990's when there was significant debate about the cause of AIDS in the role of HIV' then you go on about the changes that have been made. Could you elaborate on that, for example what valid questions did Papadopulos raise in the 1990's.

A. Well, I personally lived through this, from before HIV was identified through to today when I believe there is absolutely no doubt. I also, because of my position of having to allocate funds for establishment of diagnostic systems and national reference laboratories and the sorts of things that we should be focussing on, did spend a fair bit of personal time addressing the validity of assays, the types of laboratories we should have and one of the things that challenged me in that process was the fact that sort of 1983/4/5 was right at the cusp of the introduction of molecular techniques into scientific endeavour in this sort of sense. And,

for example, the early detection by Montagnier and Gallo of what they said is a unique virus, whether or not it was the ultimate cause of AIDS, was at a time when p24 and reverse transcriptase were primarily identified through this Western Blot technique, which is basically where you put some mixture of proteins and so on and so forth, in a well and you sizzle them with electricity and the proteins migrate at different levels. And prior to molecular capability of analysing these things it was a chemical analysis or an antibody based one. Now that's not nearly as precise as the analysis by genetic means of what that band is that runs at what is say the GP120 level or what is acknowledged as the molecular weight. Basically those Western Blots separate out the molecular weight proteins and all that a Western Blot tells you is that you've got a black spot that is consistent with a molecular weight, say of 24,000 or of 18,000 or whatever, and then you can extract that and do further tests on it. At about 1983/4/5, before the virus had actually been separated out and I'll say purified in the context that Gallo yesterday outlined, in vast numbers of, there was no gene or gene sequence on which to base the more precise molecular diagnostic techniques that can say for absolute surety 'This is a p24 from HIV', which has been identified out of these cultures. So there was a, I would have said a two to three year period around then. The whole world didn't react as quickly as Dr Gallo reacted and there was some questioning about whether this was real and there was a lot of controversy. So around that time I felt it was my responsibility to examine the sorts of questions that Ms Papadopulos was raising. So that's the basis for my comments in the reports, which I still stand by, and from my perspective there really was a period of a few years, from 1983 or 4 onwards, that, you know, around the world those questions were being raised.

Q. And those questions were continuing to be raised and needed answers into the 1990's because you do say that

she raised some valid questions about transmission, diagnosis and pathogenesis of HIV infection in the 1990's'. The scientific debate you refer to was still going in the 90's.

A. Yes, well three things. Epidemiology, diagnosis and pathogenesis, I'll take those sequentially -

Q. Transmission, diagnosis and pathogenesis.

A. Sorry, transmission. Well, in the early 1990's there were still some uncertain areas of transmission. For example, it was not absolutely clear that there was, let me say non-sexual, non-injecting drug, non-transfusion basis for transmission of HIV. At time there were lots of studies looking at toothbrushes in households where one person had HIV and others not. There was great concern, you might recall, about children going to kindergarten where, you know, there was an HIV positive child and other blood-borne viruses like hepatitis have been acknowledged as being able to be spread by, I'll say blood contact, you know, getting cuts in the schoolyard or football players with blood, blooded things and so on. In fact there was quite a deal of concern at a sort of public health level about those things and I suppose that's what has focussed my mind most on, you know, really clarifying that you just don't get it off the door knob or from blood that's been sitting on a bench or shaking hands or toothbrushes or that sort of thing and there are quite a few studies addressing that issue and by the late 1990's, in fact, I think it was very clear that it was an extremely rare situation to have non-sexual, non-injecting drug, non-transfusion - transmission of HIV. So that's the -

HIS HONOUR

Q. Can I ask you this question: that period in the 90's that you speak about, was there any question about sexual transmission.

A. No, I mean the sexual transmission was crystal clear.

Q. Both heterosexual and homosexual.

A. Yes.

Q. Anal and vagina.

A. Yes, both.

Q. What about blood transfusions.

A. Well there was - the blood transfusion thing was sort out in the 80's, quite clearly, because once HIV donor screening was introduced there were -

Q. So there was no issue that it could be transmitted by blood transfusion.

A. Beyond 1985/6 in Australia - well almost none because there's this window period between when someone is exposed to HIV and when they mount the high tita antibodies.

Q. I understand that. That was a question of whether you could actually detect it in that early stage but there was no issue about it being transmissible once you were infected.

A. No.

Q. What about intravenous drug users.

A. They are a more difficult group. Australia, I don't know by luck or planning, escaped a major injecting drug use epidemic but -

Q. The question was, was there any issue in the 1990's that that's how HIV could be transmitted.

A. No, the data to support injecting drug use transmission largely came from the US where they had these so-called shooting galleries that people would put their arms in and someone would take the syringe full of whatever around to several people and in Northern Europe, particularly Spain and Scandinavia and so on, Southern Europe was a sexually transmitted thing and northern Europe - and the epidemiology and ability to track identical strains was available at that time. So there was no doubt in the early to mid 90's that injecting drug use transmission was well established.

Q. So the issue surrounded not so much whether it could be sexually transmitted, transmitted through blood transfusion, transmitted through drug injection but more about whether it could be transmitted in other ways.

A. Yes.

Q. That's where the controversy was.

A. That's where the controversy was. You might recall problems about football teams and kindergartens, and I have chaired a couple of committees -

Q. We've still got the blood rule in Australia in football.

A. Yes, we do - but, mind you, it's not absolutely certain because Professor French presented in this court about a few cases that there didn't seem to be a basis for transmission other than someone had open sores and so on and so forth.

Q. So they're cases that one might say in non-medical terms, as far as research is concerned, are at the margins.

A. Yes, absolutely.

Q. So that's where the controversy was.

A. Yes.

XXN

Q. I think you've probably covered transmission now. You said you wanted to deal with diagnosis of -

A. Yes, diagnosis. I hope it's plain to the court that there are really what I would call two phases of diagnostic testing; one was what I might call the pre-clone protein diagnostic systems and the other is the post-clone protein. You heard Dr Gallo yesterday describe how he managed to make huge amounts of HIV in fairly pure form and, for many years, it was the viruses produced out of cell culture that were used to generate the proteins that were put on the ELISA tests and Western blots and, as this court has heard many times, it's actually not possible to produce absolutely 100% pure proteins from a cell culture mix. I mean, serious attempts are made to purify it as much as possible, but, at the end of the day, you can have it 99% pure but

there is often some remaining serum components because they're grown in foetal calf serum or human serum and so on and, when they're put on the platforms, as they are often called, for the basis of the ELISA or the Western blot tests, you will inevitably have some contaminated proteins at very small concentrations. Then, when they identified the gene sequence and could use specific elements of the gene of HIV to produce proteins, that was the point at which the proteins for the diagnostic tests were made what I might call absolutely pure, without any contaminating material from the cell cultures they were grown in. When did that occur - I would have said between 1995 and '98, and it sort of merged from cell-produced proteins to clone proteins over several years, and some companies had cell-produced proteins and others progressively had clone proteins produced. A lot of the propositions - and they were quite accurate - about sort of false positive results and so on were largely related to the cell culture produced material on the ELISAs and Western blots, and there is a whole range of conditions that generally I would call inflammatory; things like TB, measles, intercurrent viral infections, pregnancy - not that that's inflammatory but it alters the amount of antibodies that are present in the blood.

Q. Is that list I gave to you earlier this morning relevant to what you are just saying.

A. Yes, it is, I suppose.

MR BORICK: Before we go on, I showed Professor McDonald an article 'Whose Antibodies Are They Anyway? - Factors Known to Cause False Positive HIV Antibody Test Results', and Professor McDonald said he was prepared to comment on that and I think it's relevant to his answer now. Could I tender the article now?

HIS HONOUR: You can tender it for the purpose of putting it to the witness.

EXHIBIT #A18 DOCUMENT ENTITLED 'WHOSE ANTIBODIES ARE THEY ANYWAY? - FACTORS KNOWN TO CAUSE FALSE POSITIVE HIV ANTIBODY TEST RESULTS BY CHRISTINE JOHNSON, CONTINUUM SEPT/OCT 1996' TENDERED BY MR BORICK. ADMITTED.

Q. Could you continue with your answer now.

A. In a sense I was responding to my comments in one of the reports about certainties of diagnosis, and, in the early stages, when cell culture-produced proteins were used in these diagnostic tests, there was the probability of very low level contaminants from cell culture, and I noticed this paper you presented, it says September/October 1996, so that would have probably - would have had to reflect the earlier cell culture-based diagnostics, and I was about to say I think that there is a variety of inflammatory conditions and human conditions, including pregnancy and anything that stimulates the immune system to produce a high level antibody. This can occur in acute senses, in most of the conditions listed here, malignant disorders, primary biliary cirrhosis, and I've only had a brief opportunity of looking at that, but, from my assessment, they're diseases or conditions in which you would expect a higher level of circulating globulins or antibodies. In those circumstances you are likely to have more of the sorts of antibodies that would react with the non-specific or the contaminating proteins in the diagnostic tests at fairly low levels. And I think before this court has previously been produced a description of the body's reaction to HIV where, in the early phases, you have a sort of non-specific low affinity antibody produced in the very early phases of the infection and then, after a few weeks, the affinity and direction of the antibody produced by the human becomes 10 to 100,000 more avid and there is no doubt that that is a very specific high titre antibody directed at HIV. The problem about false positives in the, I would say, the pre-1996 days was really focused

around the fact that these tests were designed to pick up people at the earliest possible stage of infection, maybe with a view to screening out potential positive blood donors, and I think it was Professor Gordon who described these facts that are around the fluorescence that occurs with these enzyme immuno assays or ELISA tests, where the antibody is directed to, say, contaminating serum proteins that might be present in very low titre, actually causing a lowish level of fluorescence and, in the setting of the criteria for whether a test is positive or negative, the bar has got to be put somewhere in terms of how much brightness of fluorescence actually represents high titre antibody versus low titre antibody, and, because the emphasis was on screening out blood donors who are HIV positive, the bar was put down fairly low, but it was backed up by the Western blot that's a lot more specific. I suppose I personally have a sense of this because I spent a lot of my time engaged in debates about how low the barrier should be put and at what level of false positives would we accept it, and, ultimately, the whole problem got solved, as it were, when cloned proteins were used, because that level of cross-reacting antibody to contaminating proteins was - it was no longer a problem, and that's the point at which the tests went from 95, 96% sensitive and specific up to 99.5 or 9, but, from my perspective, it was an important debate to have at the time and it was solved really by the cloned protein production.

Q. Could you just define for me precisely what you mean by 'clone'.

A. You've heard how the whole gene of HIV has been extracted, and then that's RNA and it's copied into what is called CDNA, and that CDNA can be put in the Los Alamos bank or whatever and that becomes - it's the DNA copy of the RNA that is the material used for producing proteins, so you can then clone the DNA either back to RNA or use it to produce proteins.

HIS HONOUR

Q. Cloning means, basically, artificially reproduced doesn't it.

A. Yes.

Q. That's what it means.

A. Yes, a molecular clone is actually a mirror image of the gene and is a convenient way of storing and keeping and subsequently using it.

XXN

Q. Does that complete what you want to say about -

A. I haven't got on to pathogenesis.

Q. What do you mean by that, for the record.

A. That's a study of the process whereby diseases evolve, and for infectious diseases it refers to the process whereby a micro-organism produces disease.

Q. All right, now, you want to comment on that then.

A. Yes, well, there is still a lot of unknowns into the tiny but important detail as to why or how HIV manages to cause AIDS or reduction in CD4 and other cells, and, I mean, it quickly became evident that infection with HIV - the population based analyses said that, on average, 8.5 years between the point of transmission - sorry, it was 8.5 years between when someone either had a blood transfusion or a sexual encounter and they began to develop the symptoms of AIDS; that was an average of 8.5 years. However, it's very clear that there are some strains of HIV that appear to be particularly lethal and people will die within two years of acquisition of it, yet on the other hand there are these long-term non-progressors who ultimately manifest some symptoms but can go 15, 20 years before demonstrating symptoms. Now, what it is about either the strain of HIV or the host and their ability to deal with the virus that brings about that change is not yet certain.

HIS HONOUR

Q. There is nothing unusual about that, is there.

A. No, not at all. I mean, tuberculosis, some people -

Q. Die quickly, some people die slowly, some people never

die.

A. That's absolutely correct.

Q. Same with cancers, is it not.

A. Yes.

Q. There are some cancers where some people last longer with them, some people die very quickly.

A. Yes.

Q. There is a saying that 'you die with prostate cancer rather than of it'.

A. Correct.

Q. But some people die of it.

A. Yes.

Q. So there is nothing unusual about the fact that people who are diagnosed with HIV demonstrate different rates of progress.

A. No; that there is standard biological rates of progression. My interest in this particularly are what are those factors either about the virus or the host that supports longevity, if I can put it that way, because that is where the clues to vaccines and so on probably reside.

Q. Again there is nothing unusual about that.

A. No.

Q. In other viral conditions those questions are asked, are they not.

A. Absolutely.

Q. In the case of cancer those questions are asked and are still being asked.

A. Yes.

Q. That's what medical research is all about.

A. That's right.

XXN

Q. I think you were still continuing with your answer in the context of the valid question Papadopulos-Eleopulos was raising in the 90's in that context.

HIS HONOUR: I don't know of any valid question she raised about pathogenesis of the virus.

MR BORICK: I put to the witness the statement that

he made 'Eleopulos raised some valid questions about transmission, diagnosis and pathogenesis of HIV infection in the 1990's'.

HIS HONOUR: It's not clear to me what question he's talking about.

MR BORICK: That's precisely why I asked this question. He's answered the first two parts of it.

A. I can clarify that, Mr Borick, in the sense that in 2007 we know a whole lot more about pathogenesis in terms of the ways the CD4 cells are activated and influences on them and so on and so forth, but in the early 90's, I think it was a legitimate question for Eleopulos-Papadopulos to say it is uncertain as to what that mechanism between infection with HIV and destruction of lymphocytes actually is. As I said yesterday, I sought her out because she had a theory about oxidation of cellular pathways being one of the factors in that, so my comment in the report was a reflection of the fact that in the early 90's there were a wide range of endeavours to look at why it was that this mutating virus brought about the reduction of CD4 cells, and so I think it was a legitimate question for her to raise. I don't think I said that she actually was a scientist who was contributing to that in addressing the problem with experimentation and so on.

CONTINUED

HIS HONOUR

Q. In the early '90s, did you have any doubt in your mind that HIV caused AIDS.

A. Oh, no, not at all. The doubts that I had were related to the mechanism.

XXN

Q. That completes your answer to the question.

A. Yes.

Q. In your report you say 'With the advent of molecular nucleic acid detection systems, it is now routine to probe for HIV with molecular techniques rather than culture but that does not diminish the fact that HIV can be and is routinely isolated and cultured from white blood cells in affected individuals. In fact, the routine test that is applied to infected people is to measure the amount of HIV specific nucleic acid as a measure of their disease status. The viral load, as detected by molecular analysis, is an indicator of disease status'. That is your description of the molecular technique which has, in a sense, taken over from the original ELISA and Western blot tests. Is that a fair proposition.

A. Not entirely, actually. Molecular techniques, viral load detection, or viral RNA in the plasma, or even the cDNA in the lymphocytes is a highly accurate way of saying that is HIV, because it is detecting the genes of HIV. In time, the refinement of that technology came after the ELISA and antibody based systems of detecting infection with HIV and a huge amount of effort was gone into confirming that if you had HIV antibody, using a very specific and sensitive test, you could culture a virus or you could use molecular techniques to confirm that anybody who had the HIV antibody, also had a virus detectable with both cultured and molecular techniques. The way the system has evolved is that the antibody systems are the first pass, as it were, in identifying people with HIV and the molecular techniques, and I'll say viral load for shorthand. Molecular techniques are

sort of confirmatory or used for following the progress of disease, both in terms of prognosis and response to treatment and they're also widely used now in the blood transfusion services, where there is some concern about the window period when antibodies don't develop and these nucleic acid detection systems are routinely used. They're not actually a viral load for the transfusion services, they just sort of say 'yes' or 'no' there is HIV gene sequences in this blood.

Q. I think you would agree with me that one thing that all of the experts that have been called by the prosecution have made very clear that Ms Papadopulos and the Perth group are living in the past - times have changed. Every one of them has made that fundamentally clear.

A. I would absolutely agree with that. The major advances in virology, and particularly with HIV, occurred around molecular biology. Once the gene was identified and cloned, things could happen very quickly and I think it was professor Dwyer who drew the comparison between SARS and HIV. SARS came along in the mature molecular biology era and it was literally a matter of weeks between when a few people in Hong Kong and other places got this rapidly often fatal pneumonia. Within a few weeks they actually had the gene, the epidemiology was then able to be worked out and, I suppose, if HIV entered the population today, we might have taken a somewhat different approach to its identification and the epidemiology, I suspect we would have used molecular techniques as the primary diagnostic and used the antibody test as the backup. It is perfectly acceptable and it has taken 20 years to get to this point in time and it is sensitive and specific and the two tests - that is the antibody-based ones and the molecular ones are well worked through, they're focused on the same germ, if I can put it that way, and they are probably the best quality tests in all of medicine.

Q. That's the viral load tests.

A. Both.

Q. Could you put a figure on the mature molecular era - I think that's the expression you used.

A. It is. I would say between 1995 and '98. Prior to '95, it was really hard work to sequence a gene and splice and you had to use basically manual methods. By about the mid-'90s, plus or minus a year or two, there were machines that were developed that could do things much more rapidly than the manual techniques. Certainly by 2000, these sequencing machines' ability to produce proteins and so on almost have become black boxes, if I can put it that way - you put something in one end and it comes out the other end. There's been an explosion of capability to do these things.

Q. I think we all know what the argument is about the ELISA tests and about the Western blot tests and the argument about viral loads - questions have been put and issues have been raised, but I'm having difficulty understanding precisely what you mean by the genetic sequence. Lawyers like to look at things, see things. Could you explain to me precisely what is meant by the genetic sequence and can we see it, can we look at it.

A. No, you can't. You can look at it by way of a graph that's printed out that shows you all of the nucleic acid proteins, the ACTG sequence that make up the genes and I will refer to them as machines. You can extract either RNA or DNA and then put it in a machine, it will print out the sequence of the nucleotides that are the basis for the Watson and Crick outline of what a gene is. A gene is really just a pre-planned sequence of what are called nucleotide bases and there's only four of them in nature - A, C, T and G - adenine -

Q. Adenine-rich RNA, is that what you're going to refer to.

A. No, no. Both RNA and DNA are made up of the nucleotide sequences and you can convert between RNA and DNA using this reverse transcriptase or enzymes that have somewhat different names but similar functions - polymerises - and this is all part of life. I am trying to explain - so when it comes to something like HIV, they have got

the entire gene sequence mapped out and you can get a print-out, if you like, of the sequence of those nucleotide bases that constitute the change of nucleotides associated with elements of the virus - like the nef gene or the env gene and that sequence of nucleotide bases. You can splice it off and produce the proteins that are codes for - like the envelope protein or the gp120 protein or you can make an RNA copy of that gp120 sequence, for example, and then use that as a probe for the gp120 sequence in patient material and it tells you if - you put tags on this system to confirm that the sequence of gp120 has actually bonded with the gp120 sequence in the patient's material. There are a range of detection systems to confirm that there has been a bonding between your diagnostic sequence. The application of these molecular diagnostic techniques is really two-fold. On the one hand you could use the whole 9,600 bases, nuclear type bases, that constitute the whole genome of HIV and look for that in patient material, but it is expensive, it is cumbersome and we know that HIV is prone to a lot of variation - up to about 30% of the genes might drop out or alter and that is part of the make-up of HIV - it is genetically variable. Some elements are variable but there are non-variable or less variable elements of the gene and they're what has been referred to as the conserved elements. If you really want to use nucleic acid probing, or molecular diagnostics - the terminology can be confusing, I can understand that, because some words are interchangeable but if you're looking for the presence of HIV, you would look for the genes and you would have spliced them out of a clone and so you would know you were looking for, say, gp120, which is a constant conserved gene and if that - I'll say - lights up in your test, you know that there is gp120 gene sequence in the material you have probed. On the other hand, for people on treatment, they can develop resistance to the drugs that are being used and that

resistance is expressed in a resistant gene sequence that's present in the virus and this is one of the variable components because it reflects the ability of the virus to adapt to resisting the noxious impact of the antiretroviral agent. You really have two systems that are routinely used: one for the conserved, always identified or always present genes, the other for the variable one, for resistance, and the gene sequences associated with resistance are now fairly well identified and it has become common practice if someone's been on treatment for a year, plus, to do again nucleic acid probing for resistance genes, which are the variable one - they may or may not be there.

Q. *What are resistance genes, is this something to do with* viral load or am I missing something.

A. No. After a period of time, the virus, because it is capable of high level genetic variation, can develop metabolic pathways to get around the toxic effects of the antiretroviral treatment. The development of the new metabolic pathways or dropping an existing metabolic pathway in HIV is reflected in the genes of that virus. Perhaps it is easier to explain with bacteria which are less complicated than HIV. We know in - say the golden staph, it becomes resistant to penicillin or it has become resistant to penicillin over the ages. Its resistance to penicillin is based on the fact that somewhere and somehow, out of all nature, it developed a gene - could have been by mutation or it could have been by acquisition from another strain of bacteria. It gained a gene that enabled that golden staph to produce an enzyme called penicillinase, that breaks down penicillin. You can actually use a genetic probe on a staphylococcus to see whether it has got the gene that produces the enzyme that breaks down the penicillin. A similar but more complicated set of genes, either by mutation or acquisition from other strains of HIV, occurs with resistance to these various antiretroviral drugs. Because the antiretroviral drugs work in a much

more complicated way than antibiotics for bacteria, they have got a much more complicated set of genes associated with those resistances.

Q. Does Parenzee have a genetic sequence that we can look at or is there a graph that we can look at to see what it is.

A. I think earlier in this court a graph was tabled of his gene sequence.

HIS HONOUR: It was during his trial.

XXN

Q. That is the gene sequence that you're talking about, is it.

A. That, and I only glanced briefly at it, that is the gene sequence of his virus, compared to lots of other viruses, which enable people managing him and this court to be provided with evidence that this gene sequence for this virus - Parenzee's virus - is the same as the gene sequence for other people's viruses and that's how you can accurately say that there is almost certain to have been transmission of this virus between this person and that person.

Q. Leaving Parenzee out of it, is there an actual gene sequence for the HIV virus that is unique to the HIV virus, so that anyone who is testing for HIV can pick it up and test it against the virus.

A. Yes. Many people in this court have indicated that there is - that the whole gene of HIV has been identified. I think that one of the factors that is difficult to comprehend is that the whole gene sequence of HIV isn't absolutely identical for each strain of HIV.

CONTINUED

One of the attributes of HIV is its genetic instability and capability of mutation, which is why in the world we have got clades A, B, C, D and individuals. It keeps mutating slightly. So when you say 'Is there an ageing sequence for HIV?', I say yes, there is a gene sequence that always has a gp120 and a gag and a pole and an fg, but there are other genes there which are the more variable component that contribute to the fact that there are umpteen different strains of HIV, if I can put it - they don't use the term 'strain', they use the term 'quade' or 'quasi specie', and we have heard in this court about the so-called gene bank in Los Alamos, California which has thousands of varied strains of HIV. Now, the purpose for that gene bank is really to keep the scientists honest, I suppose. If you submit an article to a journal saying 'I have found this new strain of HIV out in South Australia', for example, 'that has certain characteristics that differentiate it from other ones and I think these characteristics are important', you then have to bank that gene sequence with the gene bank so that any other scientist can get a copy of that and confirm your observations.

HIS HONOUR

Q. I presume the same applies to influenza; there are many strains of influenza. There would be a gene bank of various strains of influenza at Los Alamas.

A. And there is a bank of strains of tuberculosis and so on. It has become part of the validation of science.

XXN

Q. There were some questions raised about the Los Alamos HIV database during the course of the evidence. Are you aware that Dr Brian Foley, who is a custodian, admitted that the adenine in which DRN was obtained from material was abandoned at 1.6 density but there was no proof that the material was purified, and he agreed with Gallo and Ehrlich, RNA is not specific to retroviruses. That is something that Gallo said in 1972.

HIS HONOUR

Q. What does that all mean, firstly. Try and put it into some language that lay people can understand.

A. It was the first time I was aware that Brian Foley was the custodian of the Los Alamos gene bank, that is the first thing. I heard what Gallo said and I heard the other evidence that Brian Foley was purported to have put forward. I really didn't understand what they were getting at. Sorry, I understood what Gallo was saying but in the context of the purported comments of Foley about what was in his gene bank, I really don't know other than to speculate that that gene bank has been going for a very long time. I don't know whether it was around in the 1970s -

OBJECTION: MS MCDONALD OBJECTS

MS MCDONALD: The witness is now getting to a point where he is speculating because my learned friend is not putting the source document or material before him.

HIS HONOUR: I'm not sure he is even qualified to talk about it, frankly, but that's another issue.

A. I agree with that. I don't have enough information on which to pass comment.

XXN

Q. I think you said you agreed with Gallo - and I will put this to you - that in 1972, he did show that retroviral RNA is an adenine rich RNA and this type of RNA is not specific to retroviruses and can be found in cells and cellular fragments. You may or may not be able to comment on that.

A. I don't recall the exact details of his comment. I haven't actually read the transcript from him and I don't think I'm in a position to comment.

Q. In relation to the electron photographs, I will call them, I think each witness in turn has said they are not expert in electron microscopy; is that right.

A. Yes, and I would say that about myself. On the other hand, each of - I will say 'us' - has used electron microscopy as part of our diagnostic armentarium. It is a bit like radiology. We are not the highly skilled

technicians and specialists who know exactly how to set up a machine and take high quality photographs but we do use the photograph produced by these experts as a diagnostic medium. In virology, the advent of electron microscopy brought significant insights into the causes of certain infections, particularly diarrhoeal diseases. There are a number of really tiny viruses that go by the agent 'Norwalk agent'. I mean, sometimes you have a little epidemic of diarrhoea, like on cruise ships and so on and so forth, and you can't grow a virus about it but the diarrhoeal stool, if you put it under an electron microscope you can often see in these epidemic situations tiny little viral particles with spikes on them that are typical of the agents associated with Norwalk virus diarrhoea, and Rotavirus diarrhoea is another one, and we did go through an era, before molecular biology again, where electron microscopy was a major diagnostic tool. These days we don't use electron microscopy to diagnose diarrhoea due to, say, Rotavirus because we have got a molecular-based test. So that is why I think many people have said it is superseded as a technique for viral diagnosis and speciation and so on because detecting the genes is much more accurate than looking at a photograph. Nevertheless, I think all of us have looked at those photographs and accept that all retroviruses, for example, have a similar outer coat with knobs on it that has been referred to, an inner core and they are circular, and there is very little difference between any of the retroviruses down an electromicroscope. They all butt out of the cell, they are circular, they have got a dense core. So the electron microscope gives you nice pictures and consistent pictures if you set it up correctly, but it isn't an essential diagnostic tool any more.

Q. But if the virus has been totally isolated and it is in the cells of every AIDS person and there are millions upon millions of them, why haven't we yet got one single photograph of the virus.

A. Well, I think many of them have been presented in this court by several witnesses. I don't know how to respond to that, Mr Borick.

HIS HONOUR

Q. Isn't the response that I have been shown pictures that witnesses tell me are pictures of the virus. Is that the response.

A. Correct.

HIS HONOUR: Mr Borick, you are putting these questions but where is the evidence? You have got a witness who says she has never seen a picture of the virus. I have now got half a dozen witnesses all who say they have seen a picture and so have produced it.

MR BORICK: When I present my outline of argument, I will say that that statement is wrong, they have not told you that.

HIS HONOUR: Mr Borick, I have been in a different courtroom to you.

MR BORICK: If you wouldn't mind, your Honour, just wait until you hear the full submissions on that.

HIS HONOUR: I have heard a lot of evidence.

XXN

Q. I will put another thing to you as the witness in this case. You heard Dr Gallo, yesterday, say that the photographs he had published had been photographs of the virus but that a mistake occurred, and I think he blamed the French for it, and they were not photographs of the virus at all.

OBJECTION: MS MCDONALD OBJECTS

MS MCDONALD: That is a misrepresentation of Dr Gallo's evidence. There was an issue about one photograph and one photograph alone.

HIS HONOUR: Correct.

MR BORICK: No, it was put to him, and he accepted it, that they had removed those photographs.

HIS HONOUR: Not every photograph, Mr Borick. You point me to the evidence.

MR BORICK: I haven't got the transcript with me

here.

XXN

Q. The photographs that you presented to the court yesterday that were at p.540 of the fourth edition of the Medical Virology, they are the Gallo photographs, aren't they.

A. I'm not sure. There is a reference around those photographs and I would - I hope I made it clear yesterday that those were not really very good photographs. They were sort of photocopied out of the text book, and I think if we are talking about photographs we should actually look at some of the others taken by the previous expert witnesses.

Q. Underneath the photographs it says that they come from the 1985 photographs of - I will call it the Gallo group.

A. If that's what it says, yes.

Q. I am saying that they are a group of photographs which certainly were challenged.

A. Look, I agree with that and I think Dr Gallo yesterday gave a very clear exposition as to the contamination of one out of - is it 46 culture lines?

HIS HONOUR

Q. 48.

A. Now, I can tell you, Mr Borick, that the sort of culture techniques and processes that Gallo and Montagnier and others went through at the time are hugely technically challenging and often prone to being contaminated and people make huge efforts to set aside those cell cultures that have become contaminated but there are some really - I will say 'sneaky viruses' - that can pop in and mimic retroviruses and it is not unsurprising that there was a contaminant in there. And you have got to remember that in those very early days before the virus had been regularly cultured and one culture was seen to be the same as the other culture and to the other culture and produced so-called productive infection, in a sense I could say that the gold standard

had not been established in 1983 and it was very quickly established because the gene was sequenced, and that is HIV.

XXN

Q. You gave us another photograph yesterday headed 'HIV molecular clones'. It appears to come from an article published in November 2000 headed by a person called Schubert. Do you know where they got the photograph from.

A. That's a photograph of a virus being produced out of a continuous cell line of the type that Dr Gallo described and that virus is being produced out of that cell that has been - I will use the term 'infected', he used the term 'transfected' - but essentially the gene of HIV has been put inside that lymphocyte and it has begun to reproduce and create new viruses butting out of the cell.

Q. Are you able to help us as to just where that photograph came from. Someone has handwritten on it 'HIV molecular clones'. Is that your writing on it.

A. No, that's Professor Gordon's writing actually.

HIS HONOUR: There is a source for it, Mr Borick, on the second page, figure 4 'Electron microscopy analysis of -'

MR BORICK: That wasn't my question. I appreciate what is there in figure 4 but my question is: where did the actual photograph come from itself? What virus, what year, what person?

A. Sorry, it came from that article that is an article describing how those molecular clones, that is a new virus which is in our terms absolutely pure because it has only come from the gene - there is no other person's contaminating material in that. So that is a system of producing pure virus and that creates an opportunity for the type of study that was the basis of that paper, which was analysing the internal mechanisms of the virus for - I forget what the title is but the operation of some of the genes or reproductive aspects of the virus.

That is now the common way in a research laboratory of creating a lot of virus for studies. That is really one example of the type of study that can arise from molecular clones.

XXN

Q. But you can't help us with the question of just where the authors sourced those photographs from.

A. It would have come from - it would have to have come from the cultures that they used to produce the virus to do their studies.

Q. So we come back to the point where you haven't got a photograph of an actual virus from an AIDS patient.

CONTINUED

A. I can't recall, your Honour, whether some of those photographs tabled by people were from an actual AIDS patient but I'm very happy to produce one. No, no, the court has seen a virus from an actual AIDS patient, there was the material introduced by Martin French from an article written by a John Armstrong that had electronmicrographs of lymph nodes from AIDS patients demonstrating the round viral particles with an inner core.

Q. Professor French has been questioned about that so I won't take up your time with it now but our position has been put to him.

HIS HONOUR

Q. Well, have a look at P70. Is that a photograph of a virus.

A. I believe it is.

XXN

Q. That's the one you are talking about we got from French, is it.

HIS HONOUR: No, Dwyer I think.

A. That was Dominic Dwyer produced that.

HIS HONOUR: Have a look at the photographs contained in A16, p.546.

MR BORICK: We don't have a copy of A16, can I just have a quick look at it?

HIS HONOUR: Yes, certainly.

MR BORICK: Yes, your Honour.

HIS HONOUR

Q. Is that a photograph of the virus. It says HIV1, HIV2.

A. Yes.

Q. And is that part of a paper by a Mr William Blattner, or Professor William Blattner.

A. Yes, it's part of a textbook I think.

Q. What was the date of that paper.

A. I don't think we know accurately -

Q. Approximately.

A. It's always hard with text books because unless you get the front page. But just looking at the references

dates on which it's based, just looking at this first page here, most of them are 1983, '82, '86 and I can't see a later reference date than 19 -

Q. There is 1987.

A. - 87. So I suspect it would be pre-1990. But, indeed, this court has seen the Gallo photographs of the virus. I sort of wonder if this sort of confusion about the central importance of a photograph, which I don't think any of the experts accept as vital -

Q. Any of the experts called by the Crown accept as being vital.

A. Absolutely. But I hope the court understands that in the cycle of producing new virus it buds out of a cell and you could technically say that it's not absolutely pure because it's got some host cell components as part of its make-up, and I have a feeling that that's where some confusion may be entering into this. If you take that line, no virus is absolutely pure. But I don't - I think that's an erroneous statement because the mix of proteins include some host cell elements for structural things, create a unique, reproducible, infectious particle, which we call a virus.

Q. Would you have a look at P62. Do they show some photographs of the HIV virus.

A. Yes, from my -

Q. P62A.

A. I mean these photographs, your Honour, are very similar if not identical to several that we have been presented.

Q. I know, I'm just asking the questions.

A. Well I mean, yes, P62A does demonstrate photographs of the virus.

XXN

Q. And when you say demonstrates a photograph of the virus, that's according to the people that publish those photographs, not you, you rely upon what they are saying.

A. Well, no, more than that Mr Borick, I can look at those photographs and say they have an outer cell membrane on

the virus, with projections, which are consistent with GP120, and an inner dark core and that is a characteristic feature of retroviruses. So wherever it came from if you showed me one of those pictures I would say that is consistent with a retroviral particle.

Q. That's right but you can't say it's a photograph of the HIV retrovirus.

A. No, I think we have heard many times that by electronmyography it's very hard to tell the difference between HTLV1 and HTLV2 and HIV. It marks them as belonging to that retroviral classification.

HIS HONOUR

Q. But it's not impossible to tell the difference, is it.

A. If you go into some of the more detailed sophisticated techniques like that colour photo, you can say 'This is really HIV morphology'.

Q. I think the photo I took you to with the four photographs on it, identified HIV1, HIV2, HTLV.

A. Yes -

Q. With differences between them.

A. But if you look at those, for someone like me I'm hard put to - if you show me one on its own I would say that's a retrovirus, whether it's HIV1 or 2, I don't think I'd be -

Q. No, you may not be but others may be.

A. Yes, and what they would be doing is actually using some of the techniques like on that colour photograph that actually show you much more internal detail. I'm sort of at the stage of saying is it darker on the outside and a core that's dark and for HIV1 and HIV2 and HTLV1, the make-up of that core and its configuration will be distinctive but you don't take those colour photographs every time from -

Q. No, I understand that and I understand your evidence about it. To put it in its simplistic terms for someone like me, as a clinician might ask for an MRI scan, someone who's a specialist in taking MRI scans and interpreting them will look at the scan, will interpret

it and send his interpretation down, with the scan, to the clinician, the clinician will look at the raw data and he will have to make up his or her own mind about the patient.

A. Yes.

Q. But the person who is trained in the skills of interpreting these is someone whose opinion the clinician will take into account.

A. Yes, absolutely. I mean I don't see -

Q. The clinician won't second-guess the specialist who specialised in interpreting these.

A. No. I mean I think Dr Gallo yesterday made that fairly clear that he wasn't an expert electronmyographer, that he had a colleague in Germany that he trusted to do the type of thing that an MRI specialist might do and he respected the opinion of the skilled electronmycroscopist, but by the same token Dr Gallo or I can look at that and say 'Yes, that looks like HIV'.

XXN

Q. I just want to ask you a couple of questions about Mr Parenzee's actual results back in 1998. Originally, blood was collected on the 19th of September 1998 and came back as a 'HIV antibody EIA positive' and then 'insufficient specimen to confirm HIV status by Western blot assay, please send a repeat specimen' and a repeat specimen, further blood sample, was sent on 22 September 1998 and it came back 'Western blot was performed and IMVS. This is considered to be a confirmed positive reaction indicating infection with human immunodeficiency virus'. It's validated by an unknown operator so we don't know who carried out this actual test. And then there is an expression 'HIV +ve? Lymphoma' on the bottom of it. First of all do you know what that expression 'HIV +ve'; plus positive, and then question mark, and then lymphoma, what that would represent.

A. No, I don't, I have never personally assessed Mr Parenzee. My understanding, and you can confirm this

Mr Borick, is that he presented with an enormous fever and swollen lymph nodes and so on, that led to the initiation of those tests, and one of the possibilities that can produce that fever and enlarged lymph nodes and be a complication of HIV is a lymphoma. But I am sort of interpreting that without -

Q. I think you have answered my question, but what about the wording 'This is considered to be a confirmed positive reaction indicating infection with human immunodeficiency virus'. Indicating sufficient to say that it is a confirmed positive.

A. Yes, I mean laboratory test results are sent out with the same risk management approach as insurance firms, for example, they tend - well, you heard Dr Dax say we don't call things positive we call them reactive. In fact the two are the same and mean the same and that set of words I would interpret, as a clinician, as indicating this is an absolutely positive HIV test that has been absolutely confirmed by Western blot and represents infection with HIV.

Q. A few more questions before I finish. The Perth Group are not on their own, are they, there are lots of other scientists out there who challenge what I will call generally the HIV/AIDS proposition and it takes two parts of the challenge at least. One is the very strong view taken by The Perth Group that the virus was never isolated, and the other headed by Duesberg that HIV does not cause AIDS.

OBJECTION: MS MCDONALD OBJECTS

MS MCDONALD: I object to the question formulated in that way. There are two different positions, my friend has melded them together. He may get an answer that may indicate that they are one and the same.

HIS HONOUR

Q. Answer it in two tranches. You understand the two tranches.

A. I understand where - I suppose a general comment about the denialist movement, whose magnitude I was frankly

not fully aware of until I got involved in this. There is huge websites and things flying around. But I would categorise them in the terms of 'The Perth Group' who are, what I might call, absolute denialists who deny that HIV exists, at all, -

Q. Proved, hasn't been proved to exist I think is what - I don't know that they put the positive.

XXN

Q. Proven to exist. That's the point.

A. Then there's another group of denialists who accept that HIV exists but that it's not responsible for producing the disease AIDS. Many of them maintain that it is or might be something else that is not yet detected. The third group of denialists maintain that it's toxins or poisons or blood transfusions or some overwhelming set of antigens that do something strange to the immune system to bring about the exhaustion of the CD4 cells that then result in pain. That's my general overview of the denialists.

Q. You recall Dr Turner was talking about the conference in South Africa and you had a look at one of the conclusions of that, that the scientists were never going to agree and that potentially more work was needed. Do you recall that.

A. I recall him mentioning the fact that he went to South Africa and my understanding is that there were two conferences, one that had the scientists present as well as the denialists, if I could put it that way, and they couldn't come to any accommodation of each other's views, and the second conference which Dr Turner attended was of the denialists themselves. So I don't know if that answers your question Mr Borick.

Q. You heard him talking about that, and I'm not sure you are right about the second conference but the end result or the conclusion of that was that they were never going to agree upon anything. Is that right.

A. I think that's a reasonable comment. I mean this court's heard the disagreement as it were, but if I

could make a comment about the South African situation. President Mbeki had some doubts, he had a minister of health who's got a very long and complicated name, that was referred to as 'Dr Beetroot' because of her contention that beetroot, plus some sort of potato, plus garlic or lemon juice, I can't remember, was the answer to HIV. Now I don't quite know what the specific triggers were but she's sort of been set aside of recent years and South Africa has begun to treat HIV with antiretrovirals, out of the global fund and other things. So, despite the early concerns I suppose, expressed by the political leadership of South Africa, they've moved to acknowledging the existence of HIV and to implement antiretroviral treatment and to address that maternal child transmission, which is a huge problem in South Africa.

CONTINUED

And I think one of the documents tabled in this court was about a 240-page monograph of mother to child transmission from the Perth group who - I'm not sure whether it was part of that series of seminars in South Africa, but I thought that was particularly unfortunate for public health in South Africa, because it clearly states in several places that there is no such thing as transmission of HIV from mother to child and that there is no evidence that any antiretrovirals can prevent that transmission, and that flies so much in the face of hundreds of trials, and it's now routine around the world, including in South Africa, to use that.

HIS HONOUR

Q. With, as I understand it from other witnesses, very positive results.

A. Yes, absolutely. I've personally spent a bit of time on that, mostly in Thailand but observed it in other areas and, you know, it was a distressing situation. The first doses chosen of AZT reduced it by 50 or 30%, but then progressively there have been additional drugs, combination drugs, and it's now down to less than 1 or 2%.

XXN

Q. Just to finish off, you - apart from the experts that have been called by the prosecution - you did approach others I think, including Sir Gustav Nossal, as one example, who was unable to come.

A. That's correct, I think Sir Gus is on an aeroplane to Europe and, because of the timings and so on, was just not compatible.

Q. I think another one you approached was Kary Mullis.

OBJECTION: MS MCDONALD OBJECTS

MS MCDONALD: I object as to relevance.

HIS HONOUR: What's the relevance?

MR BORICK: The fact that another scientist whose name has been mentioned here was approached and gave a specific response which goes directly to the issue as to whether there is a scientific controversy.

HIS HONOUR: That's not evidence.

MR BORICK: Yes, it is.

HIS HONOUR: No, it's not. How can I rely on anything that this witness tells me about what Professor Mullis might have told him, told the prosecution, told anybody. It's not evidence. What do you expect me to do with it?

MR BORICK: The question at the moment is 'Did you write to Kary Mullis'.

HIS HONOUR: What's the relevance of it?

MR BORICK: I wanted to ask him the question of what was the response.

HIS HONOUR: What's the relevance? It's hearsay.

MR BORICK: It's not hearsay.

HIS HONOUR: Yes, it is. What do you want to establish? Assume this witness said 'I wrote to Professor Mullis and he wrote back to me and he said yes, there was a controversy'. What could you make about that evidence?

MR BORICK: Your Honour has made it very clear; I put the question you've ruled against me and I don't think there is much point -

HIS HONOUR: No, you challenged me when I said to you that I didn't see the relevance of it. If you want to pursue the question then you'll have to enter into a discourse with me to convince me that my ruling is wrong. In you don't want to contest my ruling -

MR BORICK: The first answer was it was relevant and I gave reasons why it was relevant and your Honour understood that. You came back with a statement as to the issue of the question about a controversy. As to hearsay, it doesn't prove the truth of a scientific controversy; it proves that another leading scientist in the world holds a different point of view. Time and time again it has been put the Perth group are idiots, they are on their own, just as we've been told there are web sites out there - there are a huge number of people talking about it, if you want to look at that.

HIS HONOUR: There are web sites out there; a huge

number of web sites which say that the Holocaust never existed, it never took place. That does not prove that there is any controversy about whether the Holocaust took place. All it proves is that there are a group of people who claim it never took place. Just because there are a group of people that claim it never took place does not prove that it is a controversial matter.

MR BORICK: The answer to that depends upon the quality and the standard of people who are claiming the Holocaust never took place. I don't want to get involved in that. It's not our position at all. We've put up a scientific argument, not a political argument, and that's all that could be.

HIS HONOUR: Mr Borick -

MR BORICK: I'm taking issue with your analogy with the Holocaust because that is a purely political issue.

HIS HONOUR: Ask the question get the answer and we'll deal with it how I'm supposed to deal with the answer in due course. I'll allow you to ask the question.

QUESTION ALLOWED

XXN

Q. You wrote to Kary Mullis.

A. I sent an email to Kary Mullis.

Q. And you were actually wanting to discuss with him - the email related to the effectiveness of PCR.

A. Yes; the reason I did that was because - I forget whether it was Dr Turner or Mrs Eleopulos-Papadopulos indicated that the inventor of PCR, who is Professor Kary Mullis, was purported to have expressed a lack of confidence in the *PCR for which he got a Nobel Prize in* chemistry.

Q. We've had some evidence on that, but Mullis responded by -

OBJECTION: MS MCDONALD OBJECTS.

MS MCDONALD: The witness should be able to finish his answer.

MR BORICK: I thought he'd finished his answer.

HIS HONOUR: I though he'd finished his answer.

A. I can continue and say -

HIS HONOUR: That's all right; I thought you'd finished your answer.

XXN

Q. Mullis responded by saying that the court has to realise - this is the effect of it - that the HIV issue is not settled scientifically and you can't prosecute people based on an unproven hypothesis, and he wasn't prepared to help. That was the effect of his answer, wasn't it.

A. The effect of his answer was to express great confidence in the PCR system that he invented, and to indicate that the controversy around HIV is not a controversy around whether PCR is a valid technology or technique. It's used millions of times around the world. He indicated that the controversy was really around whether and how HIV caused AIDS, and in his response he sent along a 10 year-old commentary that he made that essentially said there might be another virus out there that could do this, and could mimic it. That position is something that has not been accepted by his peers, Nobel Laureates and the like. I can go on about some sort of background -

HIS HONOUR

Q. He wasn't prepared to come to court to express that view.

A. No.

+RE-EXAMINATION BY MS MCDONALD

Q. What's Kary Mullis been doing in the last few years in terms of science.

A. I gather he's been surfing. He's an unusual character who worked for a biotechnology company called Cetus as a chemist, and it was in that position that he discovered the enzymes that underpin what is now referred to as PCR. He's quite a colour - I have never personally met him but this is comments from various people -

HIS HONOUR: Is this going to help me?

MS MCDONALD: No, it's not; the question has been

answered.

HIS HONOUR

Q. Can I ask this question. Has Mullis ever done any work specifically in the area of HIV.

A. None at all, your Honour. He's a chemist.

Q. He's a chemist.

A. Yes.

REXN

Q. Looking at A18, that's something that has been downloaded from the virus and home page. Professor, you were asked some questions about this particular article and I just want to make this clear: in terms of the list of things that purportedly can result in false positive HIV antibody test results, do you necessarily agree that all of those things can cause false positives of themselves. For example, let me direct you to the very last dot point.

A. Do you mean receptive anal sex?

Q. Yes.

A. I don't actually accept that. The reference is 39 and 64 - 39 is Papadopulos-Eleopulos, and 64 is National Institute of Justice, AIDS Bulletin. The reason I guess I don't accept that is that I know of nothing associated with anal intercourse which is likely to - unless there is serious trauma associated with it - that is likely to produce the type of inflammatory response that is typically associated with most of the conditions listed here; you know, leprosy, tuberculosis, lupus etc.

Q. Just to make it clear, when you were giving the answers that you gave to the questions about this article, were you or were you not saying that you agreed with this list of things as being conditions that can cause necessarily a false positive.

A. I was trying to draw out the fact that this list must be drawn up prior to 1996, which was in the era of antibody testing where the antigens on the testing material were not as absolutely pure as they are today, and these conditions are all likely to give a little tiny increase

in the sort of antibodies that would react with the contaminating material there. Now, it's correct I didn't look extremely carefully at the list, and I find it hard to understand how receptive anal sex could be a cause of a false positive antibody test. I might accept that people who engage in that sexual practice are likely to be infected with other things, like rectal gonorrhoea and that type of thing.

HIS HONOUR

Q. Who is Christine Johnson by the way.

A. I have no idea.

REXN

Q. Have you had time to go through each of those factors one by one and come to a view one way or the other as to whether or not those are conditions that may have resulted with false positives under the old test.

A. No; I only saw it five minutes before court.

HIS HONOUR

Q. But, as I understand your evidence, this list could be 25 pages long -

A. It could be.

Q. - but the question is irrelevant when you come to diagnosis of HIV by way of ELISA and Western blot.

A. Today it's absolutely irrelevant.

REXN

Q. Just on another topic, you heard yesterday Professor Gallo's evidence that by 1985 the full genome of the HIV had been sequenced, over 9,000 base pairs. Firstly, do you agree with that.

A. Yes.

Q. Today you talked about the mature molecular era and you referred to that I think commencing about the mid-90's. Could you just explain for us the difference, given that, on the one hand, we have evidence that we had the genome, the sequence, by 1985, but you describe the mature molecular era as being later.

A. In 1985 it would have taken a fairly large number of protein chemists and molecular biologists to grow up

enough virus to extract the RNA and use these enzymes that cleave - that break the gene apart into little bits and then analyse each of those little bits and then put all the little bits together to make a big bit, a big genome, and that's a process that takes many weeks if not months, and it's the sort of thing that, at that stage, only a few well-endowed organisations had the ability to do. The maturation of molecular biology I referred to was when, basically, machinery became available to do the splicing, automatically do the sequencing and print out the list of base pairs, and it went from a period where you got a print-out of a segment of the gene and then had to sort of thread it together and see what that little segment of gene produced when you created the protein that it coded for, and the machines got bigger and better and by the - well, in the last 10 years probably it's just been fairly routine to be able to produce the whole sequence in a machine.

NO FURTHER QUESTIONS

WITNESS RELEASED

+THE WITNESS WITHDREW

HIS HONOUR: Is that the case for the Crown?

MS MCDONALD: It is.

MR BORICK: That's the end of the evidence.

HIS HONOUR: There will be no evidence in rebuttal?

MR BORICK: There will be no further evidence, your Honour. A date was mentioned of 6 March - I could have my outline of argument in before that date, say around about the 1st.

HIS HONOUR: We'd set aside some days for any evidence in rebuttal, hadn't we?

MR BORICK: That's right.

HIS HONOUR: When were they?

MR BORICK: Certainly the 27th and 28th.

HIS HONOUR: I'm wondering whether you'd be in a position to be ready by around that time or the 1st or 2nd March?