SULAN J	Т
NO.65/2006	2
	3
R V ANDRE CHAD	4
	5
WEDNESDAY, 20 DECEMBER 2006	6
	7
RESUMING 10.06 A.M.	8
+ELENI PAPADOPULOS-ELEOPULOS CONTINUING	9
HIS HONOUR	10
Q. Ms Eleopulos, I know it is difficult because it is har	d 11
to change one's style or speed of speaking, but the	12
reporters, particularly when you are using technical	13
language, have some difficulty understanding and keepi	ng 14
up with you because you speak quite quickly, so if you	. 15
could try and just slow down a little bit. I know it	is 16
difficult but if you could just slow down a little bit	17
so that we could try and get it down accurately.	18
A. I will try. I have been promising to myself and that'	s 19
why I'm so nervous and it is becoming a vicious circle	20
I promise to myself I'm not going to be nervous and I	21
will speak - this is my problem. I am not good in	22
expressing my views.	23
Q. I understand. You are not alone when you say that you	24
are nervous. Lots of people are when they come into t	he 25
witness box. Anyway, you do the best you can and I wi	11 26

	slow you down, if you don't mind, if you are going too	27
	quick.	28
A.	I don't mind at all.	29
MS	MCDONALD: Before I continue, my learned friend has	30
	advised this morning that there are some slides which	31
	are going to be withdrawn from your Honour's	32
	consideration, given that the material they are based on	33
	can't be find. I would invite my learned friend to	34
	indicate that before I continue so I don't cross-examine	35
	on slides which are not before your Honour.	36
MR	BORICK: Slide 88 in the isolation presentation;	37
	slides 18 and 19 in the sexual transmission	38

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	presentation.		1
MS	MCDONALD:	Can I check? It seems to me that No.20	2
	also comes fro	om the same study as 18 and 19.	3
MR	BORICK:	I will check that. Slides 39 and slide	4
	52, and in the	e antibody testing presentation, slide 26.	5
	The Consta	antine book is on an aeroplane between here	6
	and Perth. It	is in every library in the country other	7
	than in South	Australia. It was supposed to be here at	8
	huge expense e	early this morning but they haven't got it	9
	to us. We exp	pect it mid morning.	10
HIS	HONOUR:	Thank you.	11
+CR	OSS-EXAMINATION	BY MS MCDONALD	12
Q.	Before I pick	up where I left off yesterday, I just want	13
	to go back and	l ask you a couple of questions about the	14
	evidence you h	have given already. Yesterday when I was	15
	asking you som	ne questions about the number of people who	16
	are reported t	to be HIV positive in the world, I asked	17
	you this quest	ion and you gave this answer. I am going	18
	to read it to	you and I invite you to listen to it.	19
	P.218. You mi	ght recall it was at a point in time when	20
	that UN docume	ent was in front of you.	21
A.	Beg your pardo	on?	22
Q.	It was at a po	oint in time when that UN document, the	23
	booklet, was i	n front of you.	24
A.	The UN?		25
Q.	Yes.		26

A.	Yes.	27
Q.	I asked you this question: 'What do you think is wrong	28
	with these 39.5 million people', and you gave this	29
	answer: 'If you have just a small glance of this, now	30
	HIV existed or AIDS existed for 25 years, HIV existed	31
	for more because they claim it was in the population	32
	even before 1980, right'.	33
HIS	HONOUR: What page are you on?	34
MS	MCDONALD: I have moved to 219.	35
XXN		36
Q.	'And it is sexually transmitted. Why, even today, this	37
	virus, if you go through the document, you will find out	38
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	that this virus is still only restricted to blacks or	1
	to, say, Africans and Asians. Why? What's happened?	2
	We live in a global village. People moving left, right	3
	and centre'. The first question is: do you agree that	4
	was the answer you gave to that question yesterday.	5
Α.	Yes.	6
Q.	What did you mean when you said 'even today, this virus,	7
	if you go through the document, you'll find out the	8
	virus is still only restricted to blacks or to, say,	9
	Africans and Asians'.	10
Α.	If you read the document and you find out that the vast	11
	majority of people who were reported to be positive are	12
	either from Asia or Africa, or migrants to Europe from	13
	Africa and Asia, or blacks in America. That is, the	14
	vast majority out of that 39 million supposedly belong	15
	to these groups. If you go to England, for example, a	16
	few years ago, I can't recall the exact number, but it	17
	was about 100, no more than 250 white British citizens	18
	born in England and whites who are HIV positive.	19
Q.	But yesterday you didn't talk about the vast majority,	20
	you used the words 'You'll find out this virus is still	21
	only restricted to blacks or to, say, Africans and	22
	Asians'.	23
Α.	By 'restriction', I meant relative. It is all there.	24
	Relatively speaking, it is very, very, very minor	25

proportion of people who are reported to be HIV infected 26

	in Europe, whites in Europe, Australia or the USA.	27
Q.	Can I move on to another topic for a moment. Yesterday	28
	I asked you some questions about the web site of the	29
	Perth group. Do you recall that.	30
A.	Yes.	31
Q.	And you indicated - and these are my words, not yours -	32
	really, you don't have very much to do with that web	33
	site.	34
A.	As I said yesterday, we are a group of people which	35
	others call us the 'Perth group', and we inherited this	36
	name. So to make sense easier, we call now ourselves	37
	the 'Perth group', but not one of us does HIV/AIDS	38

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	research. It is a minor part of our duties in hospital.	Τ
	As I said to you yesterday, my work is not HIV research.	2
	I do even less than 50, no more. In fact, much less	3
	than 50% of my research in the hospital is related to	4
	HIV/AIDS. In fact, now even less, because what we have	5
	to say about HIV and AIDS -	6
Q.	I'm going to stop you.	7
A.	We have said it -	8
Q.	I'm going to stop you.	9
A.	Yes.	10
Q.	Because this has nothing to do with the question I just	11
	asked you.	12
Α.	Sorry, yes.	13
Q.	The question I just asked you is about the web site.	14
Α.	Yes, and I was going to come back to answer that answer	15
	because we are -	16
Q.	Let's just deal -	17
Α.	Because we are a group, each of us, who divide your	18
	tasks to make things easier to be able to do the work.	19
	We don't have staff to do the work for us and we don't	20
	ourselves - are not doing this work all the time. So we	21
	have to come up with data. We have to divide to make	22
	things easier. I have to do. Everybody is responsible	23
	for something.	24
Q.	Let's try and actually go back to the question. My	25
	question is: do you agree that yesterday you told the	26

	court that really you don't have very much to do with	27
	this web site.	28
Α.	Yes, I did. Dr Turner is mostly responsible for the web	29
	site.	30
Q.	When you say 'mostly responsible', do you have a role.	31
A.	Not much.	32
Q.	Looking at this document, the document you have in front	33
	of you is two pages.	34
Α.	Yes.	35
Q.	If you look on the left, there is a column headed 'Home'	36
	on the first page.	37
A.	On the first page? On the front page.	38

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Q.	There is a column to the left, a dot point, headed	1
	'Home'.	2
A.	Yes, yes.	3
Q.	And then if you go to the second page, you will see that	4
	column continues down.	5
A.	Yes.	6
Q.	And it ends with 'Perth group and VirusMyth'.	7
A.	Yes.	8
Q.	Do you see that.	9
A.	There is there the context to other web sites.	10
Q.	So those points that run down the page on the left,	11
	that's like the index or the menu, if you like, of	12
	different areas of the web site. Is that right.	13
A.	Sorry?	14
Q.	That column on the left, the dot points -	15
A.	Yes.	16
Q.	- that is the index or the menu into the web site.	17
A.	It is contact list. It gives the contact list and the	18
	contributors, and the Perth group -	19
HIS	HONOUR: They are talking about the VirusMyth.	20
XXN		21
Q.	So if someone was to go into this web site and look for	22
	who the contributors are, we have seen what we see on	23
	page one on that document in front of you; correct.	24
A.	Yes, you will see this.	25
Q.	Your name is the first name on that list.	26

A.	Yes, I am the leader of the Perth group and this is the	27
	Perth group web site.	28
Q.	And the second name is Dr Turner.	29
A.	Yes.	30
Q.	And then the left two names, what's their role.	31
A.	The last two names are, Dr Turner is the person who is	32
	actually keeping the web site going, technically going.	33
Q.	So that's it. You and Dr Turner are the contributors to	34
	the web site.	35
A.	No. No, the Perth group is not only me and Dr Turner.	36
	If you go through, I'm sure you will find all our names.	37
Q.	Why aren't there other names listed under the	38

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	contributors' heading if that is the case.	1
A.	I don't know. They should be there. All the Perth	2
	group should be there.	3
EXH	IIBIT #P15 COPY OF WEB SITE ENTITLED 'PERTH GROUP, THE	4
HIV	//AIDS DEBATE' TENDERED BY MS MCDONALD. ADMITTED.	5
		6
Q.	I want to ask you about another answer you gave	7
	yesterday at p.195. This was very early in the	8
	cross-examination yesterday when I was asking you some	9
	questions about whether you had the backing or support	10
	of your employer in expressing the views that you had	11
	put to the court. In that context, I asked you this	12
	question at line 21: 'To make it quite clear, the	13
	question is simple. Do you have the backing -' and then	14
	you interrupted me and this was your answer: 'Simple, I	15
	said it. It is in my duty, 30% is research and	16
	development of which, according to my head of	17
	department, I can use about 50% of it in HIV/AIDS but my	18
	research in HIV/AIDS is mostly done in my private time'.	19
	Firstly, do you agree that's the answer you gave	20
	yesterday.	21
A.	Yes, I agree.	22
Q.	Are you saying, in that answer, you have the authority	23
	of the head of your department to use 50% of your	24
	research time in relation to researching HIV and AIDS.	25
Α.	Yes.	26

Q.	So you are saying that you have the authority of the	27
	Royal Perth Hospital to spend 50% of your research time	28
	on HIV/AIDS.	29
A.	In 1988 it was agreed there has been some complaint from	30
	the HIV experts that I contacted one of the AIDS	31
	patients, and they know this, and especially one	32
	haematologist, because the gentlemen who was a	33
	haematologist complained to the medical superintendent	34
	that I have interfered with their patients, and he wrote	35
	a letter, that haematologist - the patient I had	36
	contacted was a haemophiliac who was the president of	37
	the AIDS Council in the Haemophiliac Society in Perth.	38

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I was trying to find out from the haematologist, which	1
member I was friendly, to ask what per cent of	2
haemophiliacs were testing positive in 1988. Nobody	3
would give me an answer, not even a private answer.	4
Since we were friends, I thought I would be able to get	5
that information either as a scientist or as a friend.	6
Nobody would give me that response. So there was the	7
the gentlemen who was very often on the news on our TV,	8
and as I said, who was the president of the Haemophiliac	9
Society and then he had an article. There was an	10
article in Western Australia about him. So one day I	11
write him and I ask him, not as his capacity, I didn't	12
know that he was HIV positive, I asked him 'Will you	13
please tell me how many of the haemophiliacs test	14
positive for HIV?' and he replied about 75% of the ones	15
that have been tested.	16

CONTINUED 17

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	ones which have been tested' and I said 'Yes, I	2
	understand not all of them have been tested but of the	3
	ones who have been tested'.	4
Q.	I am going to interrupt you.	5
A.	But you asked me to explain to you what is going on, is	6
	my work on AIDS, so I have to give you an answer. Your	7
	Honour please allow me to say what happened and what I	8
	was being told by the medical superintendent of the	9
	Royal Perth Hospital.	10
MS	MCDONALD: She has just directed a question to your	11
	Honour.	12
HIS	HONOUR	13
Q.	You go ahead and answer the question.	14
A.	Thank you. Now, as I said the - I told the President of	15
	the Haemophilia Society and he said 'About 75%' and	16
	after he gave me all the information I said 'But how	17
	many of them have AIDS?' And he said '2'. And I said	18
	'What do they have?'. He said 'What do you mean what do	19
	they have?'. I said 'What diseases they have' and he	20
	said 'They haven't got any disease'. 'They don't have	21
	AIDS'. But he said 'They do, I am one of them'. And I	22
	said 'What do you have?'. He said 'Apart from	23
	haemophilia he said I have low T4 cells' and I said 'But	24
	low T4 cells, you are haemophiliac, are you injected	25
	with T4?' And he said 'Yes'. He said 'Yes, I am	26

So I said 'That high a percentage?' and he said 'Of the 1

	injected with and I am on AZT'. Then the T4 cells are	27		
	made - are dual to the factor 8 you are given. He said	28		
	'But that cannot - factor 8 I know that AZT is toxic but	29		
	factor 8 can't decrease the T4 cells'. But I said 'But	30		
	yes, it can decrease'. He said 'Who are you - '	31		
Q.	Ms Papadopulos -	32		
A.	To make the short story - to make the long story short,	33		
	well after the complaint they have - they wrote letters	34		
	- the haematologist wrote letters to the medical			
	superintendent and the medical superintendent asked the	36		
	head of the department and after all we had a meeting in	37		
	the medical superintendent's office and it was decided	38		

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that I can do as much AIDS research and that was with my	1
head of the department, then head of the department, and	2
the medical superintendent, and I can publish in	3
scientific journal as much as I want but, I should not	4
talk to the public or press on behalf of the hospital	5
and I fully understand this; the hospital cannot have	6
two policies totally opposite policies in regard to AIDS	7
and I kept that promise, I promised and I did that. We	8
never wanted to give interviews and we always, when	9
people arrived and they asked me, I always specify what	10
I say and I did say in this court what I say is not on	11
behalf of the hospital, I am talking here in my behalf,	12
Eleni Papadopulos-Eleopoulos, as a private citizen, not	13
as a representative of Royal Perth hospital.	14
	15
You didn't say this in your evidence-in-chief you only	16
said that when you were cross-examined about it.	17
Toward and Taidule loss that There to any that	1.0

Q.

XXN

- A. I wasn't asked. I didn't know that I have to say that. 18
- Q. Can we go back to the question I asked you some minutes 19 ago now: is it your position in court today that 20 currently, not 10 or fifteen years ago, 50% of your 21 research work at the hospital is HIV/AIDS. 22
- A. No, it is not, but I can use up to 50% of it but I am 23 not using that much because I don't need to do that 24 because now - by now we have published everything which 25 can be said about HIV and AIDS. The only work now I am 26

	doing is when we have some papers like the ones from	27				
	Europe by May on HAART.	28				
MR	BORICK: It is not a person.	29				
Α.	Drugs. So when such a paper is published, like, as I	30				
	said, which are very, very fundamental regarding the HIV	31				
	and AIDS, then I read the papers - in fact I don't even					
	have to look in the journal because there is a professor					
	in America who always he has access. He said he'll send	34				
	the papers before you see the copy out on the street or	35				
	papers like the British paper where HIV is said to be	36				
	responsible only for 4-6% of the decrease in T4 cells	37				
	and something else causes the decrease of the over 90%.	38				

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	These papers doesn't tell me - I read them in bed. So I	1
	don't have to use that 50% and I am not using it.	2
XXN		3
Q.	You still not have answered my question which is do you	4
	say you had the authority.	5
A.	Yes, I do, I did say.	6
Q.	Let me finish the question. Do you say you have the	7
	support of your employer, the Royal Perth Hospital, to	8
	use 50% of your research time researching HIV and AIDS.	9
A.	I said the hospital knows about my work, the hospital	10
	decided in 1998 I can do this research and I was doing -	11
	I was spending about 50% of my time then, now I don't	12
	have to spend.	13
HIS	HONOUR	14
Q.	The question is though do you say you don't spend it now	15
	because you don't have to.	16
A.	But I still have authority.	17
Q.	You still have the authority.	18
A.	Yes.	19
XXN		20
Q.	You have the authority of the hospital to use that time	21
	researching HIV and AIDS.	22
A.	My head of the department - I don't go directly to the	23
	medical superintendent. Is the head of the department	24
	who is directly responsible, I am directly responsible	25
	to.	26

Q.	We will come back to that later when I put some	27
	documents to you, but I want to move on to a different	28
	topic for the moment. It relates to some evidence you	29
	gave about slide 26. This is slide 26 from that first	30
	component, the virus isolation. Looking at A5.	31
Α.	P.5, slide 26.	32
HIS	HONOUR	33
Q.	Virus taxonomy.	34
Α.	Yes.	35
Q.	On the right-hand side at the top.	36
Α.	Yes.	37
XXN		38

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Q.	Tota will see the silde after hamber 27 down on the next	
	row to the left was also related to slide 26 in that it	2
	came from the Hans Gelderblom publication, is that right	3
A.	Yes, two slides there, taxonomy.	4
Q.	What do you mean looks like -	5
A.	Yes.	6
Q.	Did that image come from that publication.	7
A.	This is what is generally accepted. This is generally	8
	accepted. I do not give - this is taken from Montagnier	9
	from Luc Montagnier's book 'Virus'.	10
HIS	HONOUR	11
Q.	That is slide 27.	12
A.	Slide 27, yes.	13
XXN		14
Q.	Is that your evidence, it didn't come from the	15
	Gelderblom publication you have referred to in slide 26.	16
A.	Slide 26, no, that is virus taxonomy, that is the same.	17
	Gelderblom has a similar thing in the web site. Both -	18
	almost everybody present the same. This is a standard	19
	presentation of HIV you can find in all - slide 27 can	20
	be found in nearly everything including Harrison's	21
	textbook of medicine. In one - Harrison in one - I	22
	forgot now which edition - in one of the editions it is	23
	presented the core is presented cylindrical and in	24
	another edition - I apologise, don't know which edition	25
	but later edition - the core is presented as cone-shaped	26

	and yet give exactly the same editor, the same	27
	electromicrogram. The same electromicrogram in the	28
	Harrison book is interpreted diagrammatically in one of	29
	them as the core being a cylinder and in the other the	30
	same electromicrograph is presented as the core being	31
	totally different, cone-shaped core, the same	32
	photograph, and one doesn't know what to think which is	33
	the right one. How can you have the same	34
	electromicrograph in the most prestigious textbook of	35
	medicine being represented in totally different forms.	36
Q.	Have you finished.	37
A.	Yes.	38

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Q.	I will remind you of what your evidence was about slide	1
	26 to put it in context, some questions that I am going	2
	to ask you.	3
A.	Yes.	4
Q.	When we got to slide 26 in your PowerPoint presentation,	5
	you said the following and this is at p.29 line 25 I am	6
	going to start part-way through an answer because it is	7
	a very long answer that goes for over a page but we will	8
	start from the point of the answer where you moved on on	9
	to deal with slide 26. Slide 26 'The viruses are also	10
	divided into families, genera and species. By	11
	definition particles belonging to the family of	12
	retroviruses are "Enveloped viruses with a diameter of	13
	100 to 120 nm budding at cellular membranes. Cell	14
	released virions, that is, individual virus particles,	15
	contain condensed inner bodies known as cores and are	16
	studded with projections which are known as spikes or	17
	knobs".	18
A.	Yes.	19
Q.	That from a paper by Hans Gelderblom.	20
A.	Yes.	21
Q.	One of the best known microscopia -	22
A.	Elctromicroscopia.	23
Q.	In general, in HIV in particular. This is a diagram of	24
	what a retrovirus looks like. Then you talk about the	25
	diagram. Firstly, do you agree that was your evidence.	26

A.	Yes.	27			
Q.	And you were there referring to an article produced by a				
	number of authors, but particularly Gelderblom.	29			
A.	Yes.	30			
Q.	Have you subsequently provided the prosecution with a	31			
	copy of that article.	32			
A.	No, I don't know if we have done.	33			
Q.	That was one of the ones you didn't provide the	34			
	prosecution with that we had to ask for.	35			
A.	That is possible.	36			
Q.	Looking at an article headed 'Fine Structure of Human	37			
	Immunodeficiency Virus'.	38			

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MS MCD	ONALD:	There is a copy for your Honour and I	1
wi	ll tender th	at article.	2
HIS HO	NOUR:	Any objection?	3
MR BOR	ICK:	No.	4
EXHIBI'	T #P16 ARTIC	LE HEADED 'FINE STRUCTURE OF HUMAN	5
IMMUNO	DEFICIENCY V	IRUS (HIV) IMMUNOLOCALISATION OF	6
STRUCT	URAL PROTEIN	S AND VIRUS CELL RELATION BY GELDERBLOM	7
AND OT	HERS' RECEIV	ED 3/12/1987 TENDERED BY MS MCDONALD.	8
ADMITT	ED.		9
			10
XXN			11
Q. Fi	rstly, do yo	u agree that is the article that you	12
re	lied upon fo	r those slides, 26 and 27, in your	13
Por	werPoint pre	sentation.	14
A. If	I gave this	reference, then, yes.	15
Q. Th	is was the a	rticle. During your evidence you have	16
be	en at pains	to tell us, haven't you, that there are	17
HI	V - HIV has	never been photographed.	18
A. So	rry?		19
Q. Du	ring your ev	idence you have told us many times that	20
HI	V has never	been photographed.	21
A. No	, no, no, no	. I never said that HIV has not been	22
pho	otographed.	I never said - just saying here - what I	23
mea	ant - let us	make it clear now, you can take	24
pho	otographs fro	om the culture there are numerous	25
nha	otographs no	t only by Hans Gelderblom but by many	26

	including Montagnier and including Gallo. Also, a	27
	lawyer found out what Gallo represented as his	28
	electromicrograph actually was Montagnier's	29
	electromicrograph but let us not go into those details.	30
	There are numerous - let us forget what Gallo did.	31
	There are numerous photographs of what is called - what	32
	is meant to represent HIV particles from the cultures.	33
	I can give you hundreds of papers. What we are saying	34
	is as in this document you have given us yesterday -	35
HIS	HONOUR	36
Q.	P 4.	37
Α.	Is that there are no electromicrographs - what is meant	38

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	to represent apart from Best and Oshenko 1997 papers	Т
	there are no photographs of the banded material, to show	2
	that what they are saying is pure HIV actually is pure	3
	HIV.	4
XXN		5
Q.	You see in this very article that you relied on -	6
Α.	I am relying on many articles, that is not the only	7
	articles we are relying on.	8
Q.	I realise that but talking about this one at the moment.	9
	In this very article you relied on it is full of	10
	photographs that the author purports are of HIV.	11
Α.	We corresponded with Hans Gelderblom - I can give you	12
	all our correspondence with Hans Gelderblom for years.	13
	He never denied that there are no pictures of the pure	14
	virus, that is what we are interested in, and, as I say,	15
	this is what is required to prove the HIV RNA and to do	16
	what you repeatedly yesterday said, molecular studies of	17
	the HIV: you cannot do molecular studies if you don't	18
	have this, this is banded. If you don't present this	19
	kind of evidence we can forget all the molecular	20
	studies, all.	21
Q.	In fact in this article what the authors were doing was	22
	comparing two strains of HIV, HIV 1 and HIV 2.	23
Α.	Yes, you can - I am not saying that they don't say they	24
	have HIV, I am not saying that they don't have HIV -	25
	that is all the debate. That is all the debate between	26

	what is called dissidents and what is is called HIV	27
	protagonists and apropos the dissidents - there are at	28
	least two Nobel laureates including Walter Gilbert, a	29
	physicist, who has given the Nobel for his work in	30
	biology without ever having a formal study in biology.	31
HIS	HONOUR	32
Q.	Is he a protagonist for the view that HIV has not been	33
	identified as a virus.	34
Α.	He - there isn't only one group of people who are	35
	questioning the isolation of the HIV. We have many	36
	supporters but we are the first who ever say that HIV -	37
	there is no evidence for existence of HIV. As far as I	38

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know, he says there is no evidence that HIV - because he	1
is close to Peter Deusberg - that there is no evidence	2
that HIV causes AIDS. I am not fully familiar with what	3
he exactly says.	4
CONTINUED	5
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XXM			1

Q.	So let's go back to this article that you relied on and	2
	cited in your presentation. Now that was an article in	3
	which the authors set about comparing the morphology and	4
	the appearance of HIV 1 and HIV 2. Do you agree that is	5
	what they were doing in this article.	6
A.	Yes, they are saying that. But, as I said, I didn't	7
	give them any other articles which say that.	8
Q.	So is it your evidence that 'Well, they're wrong about	9
	that'. What are you saying about it.	10
A.	They are seeing some particles there but seeing	11
	particles, every retrovirologist, including Gallo; in	12
	fact in 1976 Gallo published a paper - I have to	13
	answer - Gallo published a paper in which he says,	14
	finding virus particles, that is particles which have	15
	the morphology of retroviruses, which have, even in	16
	scriptures, is not proof that they are viruses, these	17
	particles just have the right condition in your culture	18
	and you will get these particles. There is no argument	19
	about this between us and the retrovirologists.	20
Q.	So when you decided to include an exert from this	21
	article in your PowerPoint demonstration you chose not	22
	to put some of the photographs of particles said to be	23
	HIV 1 and HIV 2.	24
A.	If you look at the slide, the slide says 'Virus	25

taxonomy' and we are not talking about if these 26

	particles there represent HIV or not. We only say what	27
	the viruses - in fact, here exactly define what HIV	28
	should be and this is again something which you find in	29
	all papers on HIV. It's nothing, it's nothing unusual	30
	about it and I do not have to put the pictures here.	31
Q.	Do you think it might be a fair representation of what	32
	this article was about if you had included the pictures.	33
Α.	No, there is no need for a picture here.	34
Q.	Because -	35
A.	There is no way you have to put a picture. We do this	36
	on viruses, on retroviruses what HIV should be, that is	37
	what we describe here.	38

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Q.	Because later in your presentation when we get to slides	1
	30 -	2
A.	In fact, may I clear something here, now that I look	3
	again. No, here we describe what the retroviruses are	4
	in general, not HIV 1, HIV 2, or any other HIV. What	5
	retroviruses are; this is a textbook -	6
HIS	HONOUR	7
Q.	Definition.	8
Α.	Everywhere. Nothing, nothing special about it and of	9
	course we didn't have to put a picture. It is not	10
	misrepresentation at all.	11
XXN		12
Q.	Because you did go on at slides 30, 35, 36, they are	13
	over the page -	14
A.	Yes, I know what is here.	15
Q.	- to include some photographs -	16
A.	Yes.	17
Q.	- of particles said to be HIV.	18
Α.	Yes, they are particles.	19
Q.	You were critical of those photographs and what they did	20
	or do not show.	21
Α.	I haven't got these particles because I am discussing	22
	now paragraph by paragraph and I can say to you	23
	paragraph by paragraph what is in this paper and	24
	Montagnier had some pictures there. Now, this is	25
	relevant here. Montagnier had these nictures and this	26

	picture is said to be the first picture of HIV. I had	27
	to put it there. It is a totally different matter.	28
Q.	I suggest you have been very, very selective in what	29
	you've put in your PowerPoint presentation and	30
	misrepresented a number of articles including this one.	31
A.	I'm sorry, but there is no scientist, and I mean no	32
	scientist will concede that this misrepresentation, and	33
	you can bring here and you can give it to any scientist	34
	these papers and let's see anyone who say that this is a	35
	misrepresentation. I'd like to see one scientist saying	36
	that.	37
Q.	Can I just turn to Montagnier and Gallo, given that	38

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	you've again mentioned them. Would you agree that at	1
	the time it was said that Montagnier and Gallo had	2
	isolated HIV as the cause of AIDS -	3
A.	No, sorry, may I correct you here because the question	4
	is I don't - with due respect -	5
HIS	HONOUR	6
Q.	Just let Ms McDonald ask a question and then see if you	7
	can answer it, all right.	8
XXN		9
Q.	What I'm asking you is, do you agree with this	10
	proposition, so if you listen to it first: do you agree	11
	with the proposition that Montagnier and Gallo were	12
	reported as being responsible for isolating HIV. We	13
	will break it down, do you agree with that proposition	14
	first of all.	15
A.	I agree that now initially there was a big argument	16
	between Montagnier and Gallo because they wanted, each	17
	of them wanted to be the discoverer of HIV. There was	18
	an agreement between the French and the American	19
	government that Montagnier was the discoverer of HIV,	20
	there was an agreement between governments that	21
	Montagnier was the discoverer of HIV and Gallo was the	22
	one who proved - Montagnier discovered HIV in 1983 in	23
	his science paper, in May 1983, and Montagnier and	24
	Gallo, or Gallo proved that HIV is the cause of AIDS and	25
	in his May 1984 paper, papers, four papers.	26

Q.	Back then wasn't part of the controversy or the issue	27
	about whether or not the virus that Montagnier was said	28
	to have discovered and the virus that Gallo was said to	29
	have discovered was in fact the same thing.	30
A.	Yes, that was what the controversy was about. Because	31
	Gallo was accused of two things. First of all, he was	32
	accused of misappropriating the French virus.	33
	Secondly - and he was found, the principal paper, there	34
	were four papers in science in 1984. In the principal	35
	paper the principal author is Popovic. He is the	36
	principal author and Popovic and Gallo were found by	37
	the, by a senate committee to have committed scientific	38

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	misconduct. Now, but as I said, ultimately this was	1
	agreed that still, after all this, it was agreed that	2
	Montagnier discovered it and Gallo proved that it is the	3
	cause of AIDS.	4
Q.	Do you agree that it's since been acknowledged that part	5
	of the issue was confused, if you like, by the genetic	6
	instability of the virus, the fact that the virus is	7
	genetically unstable.	8
A.	Yes, they said it cannot be because we don't have, we	9
	don't have, it cannot be - first of all, Popovic - Gallo	10
	did not have anything - Montagnier did not have anything	11
	for Gallo to steal, but let's forget that part and,	12
	secondly, Gallo could not steal Montagnier - could not	13
	misappropriate Montagnier's virus because Montagnier	14
	sent to him a cell free substance HIV, a cell free HIV	15
	cannot infect even if we admit that there is HIV and	16
	that there are HIV particles, the particles lose their	17
	knobs and the knobs are, everybody agrees, absolutely	18
	necessary for infectivity. By the time the Montagnier	19
	material reached Gallo all the knobs will have	20
	disappeared so it will have been impossible for Gallo to	21
	steal Montagnier's virus if Montagnier had a virus.	22
Q.	Have you finished.	23
Α.	Yes.	24
Q.	Would you accept that both Montagnier and Gallo have	25
	been internationally recognised and acclaimed for	26

	isolating the virus HIV said to cause AIDS. There is	27
	international recognition for that now.	28
A.	Yes, I do agree, but let us think a little bit.	29
MR :	BORICK: Excuse me, I would like to make sure the	30
	transcript has one word in it, that Montagnier and Gallo	31
	have international recognition 'now'. That was the	32
	question but I'm not sure whether the 'now' would have	33
	been picked up.	34
A.	I've answered the question. Did you say the AIDS virus	35
	or HIV, excuse me?	36
XXN		37
Q.	HIV as the cause of AIDS.	38

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A.	You called it?	1
Q.	The HIV virus as the cause of AIDS.	2
A.	Yes. That is the accepted - no, there is a debate. It	3
	is a debate that this is a cause of and it is a debate	4
	if they really prove the existence, so none of them is	5
	generally accepted. It is accepted by many and I will	6
	say the dissidents I will have to agree are minority,	7
	but there - you cannot say that there is a total	8
	agreement, or agreement beyond reasonable doubt.	9
HIS	HONOUR	10
Q.	That might be a matter for others to decide.	11
A.	No, no, sorry -	12
Q.	Not me, but for others in a certain forum. That is what	13
	your view is.	14
A.	No.	15
Q.	You're expressing your view.	16
A.	Your Honour, what I meant is that there is no agreement	17
	between scientists.	18
Q.	You say there is a group of scientists, of which you are	19
	one, who don't accept that it has been scientifically	20
	established that HIV exists as a virus.	21
A.	Yes.	22
Q.	And there are a group of scientists, of which you are	23
	one, who don't accept that, whatever it might be, if you	24
	called it HIV, that it is sexually transmittable.	25
Ζ	No and there is a group of scientists who say that even	26

	if HIV exists there is no proof that it is the cause of	27
	AIDS. In fact, that was accepted by a judge in a court	28
	of law in Argentina.	29
XXN		30
Q.	Are you one of that group that says that there is no	31
	proof that HIV causes AIDS.	32
Α.	Is the cause of AIDS? We say much more than that. That	33
	is what all this is all about.	34
HIS	HONOUR	35
Q.	But assuming for a moment that HIV does exist, are you	36
	part of the group that would say that it has not been	37
	established that HIV causes AIDS. I know that you say	38

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	it has not been established that HIV exists as a virus,	Τ
	that is your starting point, but assume for a moment	2
	that HIV does exist, are you part of the group who	3
	question whether HIV causes AIDS.	4
A.	But your Honour, I cannot assume that.	5
Q.	You can't move from the first premise.	6
A.	I cannot. If there is no evidence I cannot assume, I	7
	cannot as a scientist, I cannot work on assumptions.	8
Q.	So you don't even need to enter into that debate, is	9
	that accurate.	10
A.	Sorry?	11
Q.	You would not even enter into the debate as to whether	12
	HIV causes AIDS because you don't acknowledge that HIV	13
	exists as a virus.	14
A.	That's true.	15
XXN		16
Q.	Didn't you give evidence in this court when Mr Borick	17
	was asking you questions that there is no proof that HIV	18
	causes AIDS. That was part of your evidence.	19
A.	Sorry, I don't understand the question?	20
HIS	HONOUR	21
Q.	The question is, was it part of your evidence when you	22
	were answering questions from Mr Borick that there is no	23
	proof that HIV causes AIDS.	24
A.	I gave evidence - the first thing to say that HIV is the	25
	cause of AIDS, the first step is to prove its existence	26

	and since HIV, according to us, the evidence that not	27
	proven the existence of HIV, then HIV cannot be the	28
	cause of AIDS.	29
Q.	And it can't be sexually transmitted - if it isn't	30
	proved to exist.	31
A.	Yes, true.	32
Q.	So all of your evidence, and I want to make sure I	33
	understand it, all of your evidence is premised on your	34
	view that it has not been established that HIV exists as	35
	a virus.	36
A.	No, as I said is what you have to start with, but we	37
	published in many of our papers, we assume that the HIV	38

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	exists.	1
Q.	I thought when you were giving your evidence a moment	2
	ago you said that you were not prepared to assume that	3
	or you could not assume that in answering the question	4
	whether you accept or reject that HIV causes AIDS.	5
A.	Yes.	6
Q.	I thought that was your evidence, but I might have	7
	misunderstood you.	8
A.	No, I say it now as well that is the first step.	9
Q.	Assume for the moment that HIV does exist because you	10
	seem to have acknowledged that in some of your papers.	11
A.	In published papers, yes.	12
Q.	Assume it does exist, the question is do you take issue	13
	with those scientists who say or have concluded that HIV	14
	causes AIDS.	15
A.	Yes, of course, of course I do. In fact, not only that	16
	I do but I have an alternative hypothesis, as I said,	17
	which I put in the beginning of the HIV arena and	18
	interestingly enough many of the HIV experts have proven	19
	my - the prediction, a theory. It was - I have here	20
	that I may not be an expert witness because I don't do	21
	experimental work. There is a big difference between	22
	experimental work and between theories, both play a	23
	vital role, I'm not denying the role experimental	24
	research plays, but you can have a lot of experimental	25
	data and that is what now, for people who work in	26

	biology, that is the big discussion. In biology now	27
	there is so much data but nobody can put it together,	28
	there is a big problem and if anyone goes into biology	29
	you will see that everybody expects that physicists like	30
	me, who do not have training in biology, to come and	31
	answer this question. In fact, I have a few slides here	32
	and maybe I will, if I, with your permission, can	33
	present this?	34
CON	TINUED	35
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		37
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Q.	Well you can, I'm not sure it's answering the question.	1
	It's going further than answering the question. Perhaps	2
	we'll go back to Ms McDonald's question and if it needs	3
	further elaboration -	4
A.	Yes, there is no evidence that HIV causes AIDS. If you	5
	assume HIV is this, this there is no evidence that HIV	6
	causes AIDS.	7
XXN		8
Q.	Do you accept that AIDS exists.	9
A.	Yes, I accept that. What I accept is that in 1981 in	10
	gay men a very high frequency of some existing diseases,	11
	but relatively uncommon, come - start to appear in very	12
	high frequency. And then this later on became known as	13
	AIDS. The main diseases were Kaposi's Sarcoma, a	14
	malignancy of the skin and internal organs, and	15
	Pneumocystis Carinii Pneumonia; PCP. They are the main	16
	diseases. And they become AIDS. In fact, up till 1985	17
	they were the main diseases, are known as AIDS. Then in	18
	1985 there are a few more diseases then. By 1987 the	19
	number of AIDS cases start to decrease. So AIDS was	20
	redefined and then suddenly the number of AIDS cases	21
	jumped by, I think by 90%. Then by 1993 AIDS cases	22
	started to increase again. So AIDS was re-defined	23
	again. And now, we don't even have AIDS, what usually	24
	is in the press you hear AIDS-related. HIV-related	25

diseases or HIV-AIDS. The reporting had changed, all

26

	the way along the years. Now, Kaposi's Sarcoma by now,	27
	which was the main reason for the introduction of the	28
	retrovirus epithelial phase, is accepted as not - it's	29
	caused not to be HIV. And it's interesting that in 1988	30
	Dr Turner and I sent a paper to the Medical Journal of	31
	Australia giving reason why this disease cannot be	32
	caused by HIV. We accept that HIV exists. And the	33
	paper was rejected. And it may be interesting to know	34
	how, why it was rejected.	35
HIS	HONOUR	36
Q.	We won't go to that now.	37
A.	That is what is happening all the time, how the HIV	38

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	experts reject things. Now everybody accept Kaposi's	1
	Sarcoma, one of the two main diseases, is not caused by	2
	HIV. Now we have many other diseases which have been	3
	added by definition of AIDS. And now the vast majority	4
	of what is called 'AIDS' is found in Africa and Asia,	5
	according to Richard Horton, the editor of Lancet. And	6
	about 90% he said, I think, roughly, and then he said of	7
	these most of them are TB. So the vast majority of AIDS	8
	in the world is TB.	9
Q.	Tuberculosis.	10
A.	Tuberculosis; a disease which existed forever, and it	11
	was caused by totally different microbes. Now, do I	12
	agree with HIV? I don't have to say any more your	13
	Honour, that HIV is not a - cause diseases, because	14
	Montagnier is with me.	15
XXN		16
Q.	Isn't the reason, the largest number of people with HIV	17
	is in Africa is because the government has refused to	18
	give them access to antiretroviral treatment.	19
A.	Let me come to why give the antiretroviral. Now we have	20
	the evidence from the main paper on HAART diseases, with	21
	whom we have responded -	22
Q.	Have you finished your answer.	23
A.	No.	24
Q.	I didn't think so.	25
Α.	With the HAART we have the main paper of 22,000 people,	26

	over 22,000 people where according to Professor Cooper	27
	the finding is paradoxical; is paradoxical. Paradoxical	28
	means you have evidence -	29
Q.	We know what paradoxical means.	30
HIS	HONOUR: Maybe you do, Ms Papadopulos-Eleopulos	31
	may understand it differently.	32
Q.	What do you understand to be paradoxical.	33
A.	Paradoxical means totally opposite to what you expect.	34
HIS	HONOUR: I think we are all agreed on that.	35
A.	That is Professor Cooper's, not my interpretation. That	36
	is that you expect with HAART to have less HIV but you	37
	have more.	38

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XXN 1

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Q. Do you accept that a disproportionate number of people,

A. Not die - if the majority of people who have AIDS, who

who have been diagnosed with HIV, die of AIDS.

are said to have AIDS test die from TB, then I cannot	5
say they die from AIDS. Let me say, a very good reason,	6
why you say AIDS and if the people who die, we agree on	7
that, that the vast majority of people who die from AIDS	8
are TB patient. Now TB, before the HIV era, it was	9
known that TB, just the infection, leads to a decrease	10
in T4 cells. In fact, even when the disease is not	11
active the T4 cells still remain low. Essex - and Essex	12
is a big name in HIV-AIDS - Essex himself in 1993 proved	13
that more than 60% of people who have TB, who test	14
positive for HIV, even if they don't, even if they're	15
not infected with HIV, in fact they will be positive for	16
HIV with the most rigid criteria, which is used in	17
Australia or was used at that time. Now we have relaxed	18
our criteria I believe. So the vast majority of TB	19
people who have low T4 cells, and you have TB like this	20
AIDS, AIDS tests for low T4 cells. You have TB because	21
that is what the disease is. It will test positive even	22
if they are not infected. So if you want to call them	23
AIDS, you call them AIDS. But, you name - you give just	24
another name to TB, that's all. And -	25

HIS HONOUR

Q.	Let Ms McDonald ask the next question.	27
XXN		28
Q.	Do you accept it's extremely rare for anyone in	29
	Australia to die from TB these days.	30
A.	Yes, I accept that.	31
Q.	Do you accept that in Australia a disproportionate	32
	number of people who die of AIDS have tested HIV	33
	positive.	34
A.	Yes, they're gay men; the vast majority of people in	35
	Australia who die of AIDS are gay men.	36
Q.	Not all though.	37
A.	I said the vast majority are gay men.	38

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Q.	Do you accept that you may have been wrong in your views	1
	that HIV doesn't exist.	2
A.	I accept that, and we accepted it, and we've been	3
	asking, including in our publication, if you read our	4
	publication, in the titles, asking for people to tell us	5
	that we are wrong. We have written to people, we have	6
	asked Montagnier, and I - in 1992, in a meeting in	7
	Amsterdam, in a meeting in Amsterdam, Montagnier was	8
	present and he came to the meeting in the morning, and	9
	after the session -	10
HIS	HONOUR	11
Q.	I think you told us all about that meeting in your	12
	evidence-in-chief.	13
XXN		14
Q.	Taxi stand meeting, we know about that.	15
A.	I don't know if I said what Montagnier said?	16
HIS	HONOUR	17
Q.	I think you did.	18
A.	That's all right. So he himself could not come with	19
	evidence, he told me the p24 is the only evidence that	20
	we have that people are infected with HIV. Because we	21
	know now by 1997 he said the p24 was in the materials,	22
	he found it in material which he did or didn't have	23
	retrovirus-type particle. So if that is the evidence	24
	then Montagnier - in fact, I wrote back to him and I	25
	said 'Here it is, what's going on with p24? It cannot	26

be.' He never responded, so I don't know. Now, when it	27
comes to AIDS, in - because of AIDS, and Montagnier who	28
is the discoverer of HIV, in 2003 there was a meeting of	29
the European Parliament and Montagnier gave a talk, the	30
meeting was on AIDS in Africa. There were some of the	31
dissidents there, and Montagnier was there and he gave a	32
talk, and Montagnier in his talk said that - this is the	33
relation from French, I am not relating word by word and	34
even if I do it may be wrong, he said that the decrease	35
in T4 cell is due to apoptosis, and apoptosis is due to	36
oxidation. And oxidation in Africans is due to	37
malnutrition. That is the sum of his talk. In other	38

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	words, AIDS in Africa is due to malnutrition which	1
	should be same from day zero. So Montagnier agrees	2
	totally with us. Even with my oxidation theory and the	3
	cause of AIDS in Africa.	4
XXN		5
Q.	Do you know how it is that blood is screened in this	6
	country, when someone gives a blood donation or there is	7
	to be a blood transfusion.	8
A.	The blood is screened before it's transfused?	9
Q.	Do you know how it is, how it's done? I'll start that	10
	again to make it clear for you. Firstly, do you accept	11
	that if someone donates blood or there is about to be a	12
	blood transfusion that blood is screened for HIV.	13
A.	Yes, I do. It is screened -	14
HIS	HONOUR	15
Q.	Just say 'Yes, I agree' and let the next question come.	16
	You'll have an opportunity to explain if the question	17
	presumes something you don't agree with.	18
A.	But your Honour, if I say they screen for HIV that	19
	means -	20
Q.	It's simple, the answer is 'Yes, I do agree that in	21
	Australia they screen blood nor HIV'. I accept that you	22
	don't, I accept what you say, in the sense of I	23
	understand what you say, when you say as far as you're	24
	concerned it's not proved that HIV exists -	25
A.	No, I say the tests do not prove HIV.	26

Q.	Just answer the question and if it needs clarification	27
	you'll have an opportunity.	28
HIS	HONOUR: Yes, you go on Ms McDonald.	29
XXN		30
Q.	Do you know what method is used in this country to	31
	screen blood donations and blood that is going to be	32
	used for transfusions for HIV.	33
A.	I think they use antibody test or they use a polymerase	34
	chain reaction; viral lot.	35
Q.	Do you know which method is used.	36
A.	I don't know exactly which one is used. I know these	37
	tests are used in general for screening blood, donated	38

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	blood.	1
Q.	Well, isn't it the case that what usually occurs, they	2
	look at the nucleic acid of the virus.	3
A.	That's what I said, that's what PCR is.	4
Q.	Are you aware the reason they do that now is because a	5
	young child in Sydney was tested positive for HIV,	6
	having been given a blood transfusion. Are you aware of	7
	that case.	8
A.	When?	9
Q.	Are you aware in general terms of an incident in Sydney	10
	in which a child, in fact the child of a surgeon, was	11
	given blood and then was diagnosed as being HIV	12
	positive.	13
A.	The child may have tested positive, but it's not because	14
	the child was given a virus.	15
Q.	Are you aware of the case.	16
A.	I'm aware that people who are given blood, and this is	17
	accepted even by Elizabeth Tucks and by many other HIV	18
	experts, that people who are given blood, including	19
	Professor Calici, one of the best HIV researchers in	20
	Italy, said that blood transfusion leads to causative	21
	antibody tests.	22
Q.	Let's go back to this particular case. So are you	23
	saying you are aware.	24
A.	I don't know this particular case. I am saying people	25

who are given blood may test positive, and there is

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	ample evidence for that. People who are given - a	27
	Russian scientist who has said it himself, before he	28
	took his own blood, he tested himself. Put the blood in	29
	the blood bank, then he re-transfused it, into himself,	30
	his own blood, and he found out that after that he had	31
	at least some of the proteins, HIV proteins reacting	32
	with antibodies from himself.	33
Q.	You see, I suggest that what happened with that child is	34
	that when they went back and traced who had donated that	35
	blood it was discovered that person also tested positive	36
	for HIV.	37
A.	You may find out, that is possible to find out. I'm not	38
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	saying that you don't find that out. But if you do	1
	mainly, this is the thing that is not done, we have not	2
	got controls in HIV-AIDS. What should be done there is	3
	not to test one child randomly, because of one - or the	4
	other reason, what you do there is test all of the	5
	people who have been given blood transfusion. And then	6
	go back, then look back, what this kind of test said to	7
	be looking back. They should do for each patient, for	8
	each patient who is transfused, test him and see if	9
	there is only one person which is test positive. This	10
	has never been done.	11
Q.	Do you accept that transfusion recipients or there have	12
	been transfusion recipients who have tested HIV positive	13
	and gone on to die of AIDS, without there being any risk	14
	factors present.	15
A.	There are very few.	16
Q.	Do you accept there are case studies of that situation.	17
A.	No, they are not controlled cases, they are not, not	18
	randomly controlled study of this type. No way there is	19
	proof.	20
Q.	So in this context -	21
A.	Patients who are given transfusions are usually sick.	22
	In fact, in America the vast majority of people who are	23
	given transfusion die within a year.	24
HIS	HONOUR	25
Q.	No, that was not the question.	26

HIS	H0NOUR:	Ask the	question	again.		27
XXN						28
Q.	Do you accept	that ther	re are rep	orted stud	dies of	29
	incidents of t	ransfusio	on recipie	nts testi	ng positive f	or 30
	HIV and then d	ying of A	AIDS when	there are	no other ris	k 31
	factors present	t.				32
A.	A. We have here a slide with all the transfusion people who					
	are said to be - not all, but studies with the					
	transfusion pe	ople who	are said	to have b	ecome positiv	e. 35
	Now, as I say,	blood tr	ansfusion	, blood t	ransfusion wi	11 36
	lead to a posi	tive test	. And pe	ople who	are given blo	od 37
	transfusion the	ey are si	.ck, and t	hey will o	die. In fact	, 38

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the vast majority die within a year. So yes, some of	1
these patients will die. And maybe some of them will	2
have one of the diseases which is said to be AIDS. But	3
it doesn't mean they die from HIV. They will die, of	4
course they will die. The question is what was the	5
cause of their death?	6
CONTINUED	7
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	I accept people who are transfused will test positive,	1
	but also, we must remember that HIV experts say that	2
	people who are transfused will test positive even if	3
	they are not given HIV and people who are given	4
	transfusion are very sick people, most of them, and half	5
	of them will die within a year. So some of them will	6
	die of diseases who are said to be HIV or the way now it	7
	is called, HIV-related disease. So if you test positive	8
	and you die, no matter from what, it will be HIV-related	9
	disease.	10
Q.	Are you aware that in this State, when someone is	11
	diagnosed as having HIV they are subjected to the ELISA	12
	test and then the Western blot and then an RNA viral	13
	load test is done, that is a nucleic acid test, and then	14
	the genotype of that person's virus is profiled.	15
A.	Which one? Which person?	16
Q.	In this State, with every person who is diagnosed HIV	17
	positive, that is on the basis of the ELISA test, a	18
	Western blot test and RNA viral load test, or a nucleic	19
	acid test, then on top of that, their virus is	20
	genetically profiled.	21
A.	I'm aware that the people who are said to be HIV	22
	positive have an ELISA test, but all the HIV experts	23
	admit that a positive ELISA doesn't mean HIV infection.	24
	Then I admit that there is Western blot but there is no	25
	proof of the Western blot, nobody, and this does not	26

mean - as I said before the HIV expert, including	27
Blattner, including Mortimer and including many other	28
HIV experts and test kit manufacturers, say that is not	29
possible to say that the person who has a positive	30
antibody test is infected with HIV. Now, the viral	31
load, according to the CDC and every other HIV expert,	32
the viral load cannot be used to prove HIV infection in	33
adults, adolescents or in children, apart from	34
children - and this is really incredible - apart from	35
children who are set to have acquired the disease, to	36
have acquired HIV from their mother. So, you can have a	37
child or two children, one is said to be infected from a	38

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	blood transfusion and another one to be infected from	1
	the mother. You can use viral load to prove HIV	2
	infection from the mother but you cannot use viral load	3
	to prove infection in the child who got blood	4
	transfusion. As I said, normally everybody says that	5
	you cannot use - not only CDC, you cannot use viral load	6
	to prove HIV infection but you can use viral load to say	7
	how many virus particles you have in the plasma. This	8
	is like saying 'I can use viral load to count how many	9
	apples in the basket but I cannot use to tell of these	10
	apples'.	11
Q.	But isn't that test for measuring the viral load	12
	specific to the HIV virus.	13
Α.	As I said, if it was specific it would have been used to	14
	prove infection. How can it be specific if you cannot	15
	use it to prove infection? Even if we admit that there	16
	is - 'viral load' means molecular testing. That is HIV	17
	genome, HIV RNA, not that you prove its existence. Now,	18
	as far as profile is concerned, this is not done	19
	routinely. This is done only in a way to prove	20
	transmission from one person to another.	21
Q.	I suggest to you, in this State it is routinely done and	22
	kept on a database.	23
Α.	It is routinely done. Why do you do it routinely? For	24
	what purpose? For what purpose, unless you want to	25
	prove the infection from one person to another,	26

	otherwise it is a very expensive thing to do, and still	27
	I will give evidence, you cannot use viral load itself	28
	much less profiling to prove anything.	29
Q.	Do you accept that mothers who are HIV positive have	30
	children who are tested at birth and are also HIV	31
	positive.	32
A.	Yes, if the mother is positive. If the mother has	33
	antibodies which react with the HIV test kit, then the	34
	child will have the same antibodies because the	35
	antibodies are transmitted through the placenta and it	36
	will be there until the child becomes about nine months.	37
	The mother's antibody will be in the child, so up until	38

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	nine months we expect that - at birth all of them will	1
	be positive, and after nine months we will have none -	2
Q.	You are saying that the antibodies just go after nine	3
	months.	4
A.	Yes, the antibodies from the mother are gone, yes.	5
	These antibodies, the infant cannot make any antibodies,	6
	so the antibodies are transmitted from the mother to the	7
	child but then the child little by little starts to make	8
	antibodies and during that time the mother's antibodies	9
	disappear.	10
ADJ	TOURNED 11.33 A.M.	11
RES	SUMING 11.48 A.M.	12
Q.	Can we go back to a document I was asking you about	13
	yesterday that you wanted some time to consider	14
	overnight, P12, the Intrafamilial Transmission. Having	15
	read that particular article, did that jog your memory	16
	as to whether you were aware of Professor French working	17
	on this case.	18
Α.	No, I didn't know that he was working on this case.	19
Q.	Let me just take you through it. The objective is	20
	described at the beginning and it is described as 'to	21
	describe the clinical epidemiological and molecular	22
	evidence for transmission of HIV infection from a person	23
	with unrecognised HIV infection to a family member in	24
	two unconnected families where the route of transmission	25
	could not be conclusively determined'. Do you agree	26

	that was the objective of this study.	27
A.	Yes.	28
Q.	Basically, there were two family groups in which a	29
	person tested positive for HIV.	30
A.	Yes.	31
Q.	And there was one family - I withdraw that.	32
A.	I can explain.	33
Q.	I'm asking the questions. That these were two families	34
	in different Australian cities who both had received a	35
	blood donation.	36
A.	Yes.	37
Q.	Correct.	38

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A.	Correct.	1
Q.	And that with one of the females in particular, when it	2
	was found that she was HIV positive, it was found that	3
	she had a very unusual strain of HIV. Do you agree	4
	that's what the article indicates.	5
A.	That's what it said.	6
Q.	And that when they went back and checked her family	7
	members, it turned out her sister also tested HIV	8
	positive with the same unusual strain.	9
A.	No, the strains were determined after the two sisters	10
	were found positive.	11
Q.	And both of them had the same unusual strain of HIV.	12
A.	That's what it says here.	13
Q.	One that hadn't been seen in Australia.	14
A.	That's what it says.	15
Q.	And when they dug a bit further and looked at the	16
	epidemiology of the situation, it turns out the older	17
	sister had had an affair or a sexual relationship with a	18
	Russian sailor.	19
A.	That's what it says.	20
Q.	And low and behold, he had the same HIV strain.	21
A.	No, that is not said. There is no such evidence there.	22
Q.	Isn't it the case that that strain that the two sisters	23
	had, had never been seen in Australia before.	24
A.	Well, it don't say that we have never seen. They say it	25
	is not a strain which is usually found in Australia.	26

	That's what they say. Now, it doesn't mean that I agree	27
	with it.	28
Q.	So you would accept, on the basis of that testing, it	29
	would appear that one sister has given the virus to the	30
	her.	31
A.	No. That's what they claim but that is not proof and	32
	that is not only what I say. May I remind you about a	33
	court case which just took place in London not long ago	34
	in which a gay man was accused to have transmitted a	35
	virus to another gay man, and because what they have	36
	done through genetic analysis, that the virus from what	37
	is called virus from the two people was found to be	38

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	related, the accused gay man was advised to plead guilty	1
	but then he changed his legal team and ultimately he was	2
	found not guilty, and this was because an HIV expert, an	3
	expert on the so-called HIV genome and through genetic	4
	analysis from London gave court evidence and it was	5
	accepted that you cannot prove transmission with this	6
	kind of profiling or this kind of testing, and here it	7
	is, she had a power point presentation.	8
MR E	ORICK: 'She' is the expert witness. Perhaps	9
	that name could go on the transcript.	10
HIS	HONOUR: The expert witness in London had a	11
	PowerPoint presentation.	12
MR E	ORICK: Maria Garetti, she had a PowerPoint	13
	presentation and she gave evidence that it is not	14
	possible to determine transmission by this kind of	15
	testing.	16
	Your Honour, I think they say we don't give you more	17
	information. I have already indicated that but it was a	18
	prosecution witness.	19
XXN		20
Q.	Isn't it the case that one of the key pieces of evidence	21
	that HIV exists is that the virus is being cultured and	22
	vaccines created that had successfully treated HIV.	23
A.	They have - in 1984, when it was announced by the	24
	Secretary of Health, I don't know what exactly they are	25
	called in America, that the Americans discovered the	26

course of AIDS, she said, and Gallo said, that in two	27
years we will have a vaccine. Now, more than 20 years	28
after, we still haven't got a vaccine and never has been	29
a vaccine, and according to David Hall, one of the best	30
known HIV experts, he says we are not going to have an	31
HIV vaccine in our lifetime or even in our children's	32
lifetime.	33
CONTINUED	34
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Q.	Do you	accept	there	are millio	ons o	f people	around	the	1
	world v	who have	been	diagnosed	as H	IV positi	ve who	are	2

managing their condition with antiretroviral medication. 3

A.	They are. There are many people around the world who	4
	are given antiretrovirus. That is what it is,	5
	antiretroviral drugs. Now, as I said, that is the only	6
	study which has ever examined and actually properly	7
	conducted a study which has examined the relationship	8
	between treatment and the outcome. Montagnier's study,	9
	that was the main study. That study found out that	10
	although the viral load, the more HIV you have - HIV is	11
	the cause of AIDS, which means the more HIV you have the	12
	higher the viral load because the viral load is assumed	13
	to determine how many HIV particles there are and the	14
	higher the viral load, the more AIDS you have. The	15
	lower the viral load, the less HIV you have. According	16
	to this study, this study found out that, yes, the	17
	antiretroviral caused a decrease in the viral load.	18

- Q. I am going to cut you off because I'll turn to that

  study in due course and you'll have an opportunity to

  put that then. I'm asking you about general

  probabilities. Do you accept that it is now the case

  that pregnant women are treated with antiviral

  medication and they're having children who don't have

  antiretroviral bodies.
- A. There is no evidence that the antiretroviral drugs 26

	decrease the transmission or the HIV.	27
HI	S HONOUR	28
Q.	The question was a fairly straightforward question. Do	29
	you accept that there are pregnant women who are HIV	30
	positive who are being treated with antiretroviral	31
	drugs, whose children are now testing not to have HIV.	32
Α.	There are many women that test positive for HIV and	33
	their children do not test positive but there is no	34
	evidence that the antiretroviral decreased the so-called	35
	HIV transmission.	36
XX	ZN	37
Q.	Would you be prepared to tell Mr Parenzee to give up his	38
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	antiretroviral medication then.	1
A.	We're asked repeatedly by many people around the world	2
	what advice we give regarding antiretrovirus. We never	3
	give any advice.	4
HIS	HONOUR: That is not the question.	5
A.	I wouldn't advise neither Mr Parenzee nor anybody else.	6
	I never do it and I won't do it. The physician who	7
	cares for the patient, they will advise them. I won't	8
	advise them what treatment they have.	9
Q.	Would it be your view that the antiretroviral drugs that	10
	Mr Parenzee is taking are of no assistance to his	11
	condition.	12
MR I	BORICK: That assumption ought not to be made.	13
HIS	HONOUR: I just asked -	14
MR I	BORICK: You shouldn't phrase assuming because,	15
	frankly, he's not on any drugs.	16
HIS	HONOUR: I will withdraw the question.	17
XXN		18
Q.	Are you aware of a situation in Thailand in recent	19
	years, a scheme under which pregnant Thai women who had	20
	tested HIV positive were given antiretroviral medication	21
	for a limited period of time. They all had their	22
	children, they were all born without being HIV	23
	positive - they tested negative.	24
A.	There is no such a finding as far as I know.	25
HIS	HONOUR: Let Ms McDonald finish.	26

XXN	27
Q. I didn't suggest there was a study, I'm saying the	is is a 28
situation in Thailand in recent years.	29
A. I am aware of studies that were conducted in Thail	land 30
but I am not aware of any studies in Thailand that	t have 31
shown that when given antiretroviral that no child	d had 32
HIV.	33
Q. I will put the question to you again and I'll fin:	ish it 34
this time before you answer. Are you aware of a	35
situation in Thailand in recent years, an internat	tional 36
program, whereby women who were pregnant who teste	ed HIV 37
positive were given medication for a limited period	od to 38
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	enable them to have their children and the children,	1
	when born, were HIV negative.	2
A.	As far as I know there is no such study. I will be	3
	grateful if you give it to me.	4
Q.	For completeness, I'll take you through the second half.	5
	Within a few years, a large percentage of those children	6
	were orphaned because their mothers died of AIDS.	7
A.	No.	8
Q.	I suggest that was the situation that occurred in	9
	Thailand only some years ago.	10
Α.	I should have the scientific study. I have based my	11
	views on scientific papers or scientific evidence. I	12
	have to have the scientific evidence. I cannot comment	13
	on some claims, I can't comment on that.	14
Q.	I want to ask you more questions about the	15
	antiretroviral medication. Are you aware of the	16
	approval process that a drug has to go through before it	17
	can be used in a particular country and then subsidised.	18
A.	They have the clinical trials and this was done. This	19
	is done only to test for toxicity. It is not done for	20
	what is called efficacy. The clinical trials used to be	21
	done, blind studies. There are very rare studies in HIV	22
	testing now which are introduced in clinical trials,	23
	falling this time with clinical trials. As I said, the	24
	clinical trials are only done for toxicity, not for	25
	efficacy.	26

Q.	Are you aware that in Australia, before any drug can be	27
	prescribed, it has to be approved by the Therapeutic	28
	Goods Administration.	29
A.	Yes.	30
Q.	That has occurred here with the antiretroviral.	31
A.	That is most likely what has happened, otherwise it	32
	won't be on the market.	33
Q.	Is the advisory body the Australian Drug Valuation	34
	Committee.	35
Α.	Yes.	36
Q.	They assess and evaluate whether that drug should be on	37
	the market.	38

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A.	They evaluate it on its toxicity.	1
Q.	They weigh up the evidence by the drug companies about	2
	whether their claims are valid -	3
NOT	ANSWERED	4
Q.	Part of the process to get approval is that that body	5
	weighs up the evidence that comes from the drug	6
	companies to determine whether or not their claims about	7
	the effectiveness of the medication.	8
A.	Yes, usually on HIV - I have to answer it.	9
Q.	Please don't cut me off.	10
HIS	HONOUR: If you answer the question.	11
XXN		12
Q.	Are you aware that is what occurs.	13
A.	I don't know what the question is, sorry.	14
Q.	Are you aware that the Therapeutic Goods Administration,	15
	being advised by the Australian Drug Valuation	16
	Committee, weighs up the evidence provided by the drug	17
	companies to determine whether their claims about the	18
	effectiveness of the medication are valid.	19
A.	They do it but it is not always right. Let me give you	20
	an example. The Food and Drug Administration in America	21
	approved the introduction of AIDS and now in very high	22
	doses and yet now it is known that it hasn't got any	23
	frequency and it is shown by the study after the drug	24
	was also approved by the Food and Drug Administration.	25
	It is shown by the study, a laboratory study between the	26

	English and French scientists, that the drug, if	27
	anything, leads to higher mobility than mortality in the	28
	patient.	29
Q.	So, it was incorrect before when you said that the only	30
	thing that is assessed is the toxicity of a drug. What	31
	they in fact -	32
A.	Usually now, that's all they do.	33
Q.	Not usually, you told us before that all that was	34
	considered is the toxicity. Are you now agreeing that	35
	in fact this organisation or body weighs up how	36
	effective the medication is.	37
Α.	They evaluate - they won't introduce in the market if	38

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	they don't do any evaluation, it has to be evaluated.	1
	You cannot introduce a drug into the market if it is not	2
	evaluated.	3
Q.	In Australia, with the antiretroviral medication, you're	4
	aware that is the subject of a government subsidy; in	5
	other words people get it cheaper than what it actually	6
	costs.	7
A.	Yes.	8
Q.	Again, for that to occur there's another hurdle that has	9
	to be jumped. It has to go through the pharmaceutical	10
	benefits committee.	11
A.	That is true.	12
Q.	Again another body who looks at the costs benefits	13
	analysis of this medication.	14
A.	That is true.	15
Q.	To determine how much of the taxpayers' money should be	16
	contributed.	17
A.	That is true.	18
Q.	On that body, again there are scientists who look at how	19
	effective the medication is.	20
A.	That is true, there are scientists.	21
Q.	For the antiretrovirals to have the status they do today	22
	and that is being widely prescribed in Australia and	23
	subsidised, the drug companies have had to jump through	24
	all those hoops.	25

A. Yes.

26

Q.	In the United States, are you aware there's a similar	27
	sort of body.	28
A.	The Food and Drug Administration.	29
Q.	They have approved the antiretrovirals.	30
A.	Yes.	31
Q.	Right across the United States.	32
A.	It is an international body, the Food and Drug	33
	Administration. If it is approved, it goes and it is	34
	valid throughout all of the United States.	35
Q.	In Europe it is the European Medicines Assessment	36
	Committee.	37
A.	Yes.	38

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Q.	They have approved the antiretrovirals.	1
A.	Yes, but I don't know what this has to do with this	2
	case.	3
Q.	In Thailand, given recent events, the government there	4
	also are providing antiretroviral medication; do you	5
	accept that.	6
A.	Yes.	7
Q.	In Africa, although there is some resistance in the	8
	government, the mining companies are providing	9
	antiretroviral medication to their workers: were you	10
	aware of that.	11
A.	Yes.	12
Q.	To keep their workers alive.	13
A.	No. Let me tell you what the United Nations	14
	representative said in one of the latest interviews he	15
	gave. He said there is a big problem. People on drugs,	16
	11% of them died, so there is a problem there. We have	17
	to do something about it, the people who are on drugs,	18
	11% died. They are very worried about this fact,	19
	including Lewis.	20
Q.	If you dispute that the reason that the mining companies	21
	in Africa are giving their workers antiretroviral	22
	medication is to keep their work force alive, why do you	23
	say they're giving them medication.	24
A.	Everybody tries to do what they think is good for their	25
	people. Their action, I cannot blame them, nobody would	26

	plame them. They have based their action on what the	2 /
	scientists are telling them.	28
Q.	Before I move onto some specific studies, I want to deal	29
	with the question of publications and that is	30
	publications by yourself and overnight we have been	31
	provided with a list of publications. I propose to	32
	tender that. Do you have your own copy with you.	33
A.	I don't have it with me but they are my publications.	34
EXH	IBIT #P17 DOCUMENT HEADED 'PUBLICATIONS ELENI	35
PAP	ADOPULOS-ELEOPULOS' TENDERED BY MS MCDONALD. ADMITTED.	36
		37
		38

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XXN		1
Q.	Do you have a copy of this.	2
A.	Yes, I do.	3
Q.	Yesterday you were telling us about a letter that you	4
	had written in recent times.	5
A.	Yes.	6
Q.	Which of these particular publications were you talking	7
	about there.	8
A.	I don't have it, sorry.	9
Q.	The letter you were telling us about yesterday, which	10
	publication is that.	11
A.	It is the first one, it says 'Would Montagnier please	12
	clarify whether HIV or oxidation by the risk factors is	13
	the primary cause of AIDS', medical hypothesis 2006	14
	volume 67(3)666-8.	15
Q.	You told us yesterday that is a letter that consequently	16
	was not peer-reviewed by anyone.	17
A.	Yes.	18
Q.	In effect, a letter to the editor.	19
A.	I never said it was peer-reviewed.	20
Q.	That particular journal, the medical hypotheses, that is	21
	not one of the mainstream prestigious journals, is it.	22
A.	It is a very prestigious journal.	23
Q.	The next one you have listed, 2006, what form did that	24
	publication take.	25
Α.	That is a response to an article which was published in	26

	emergency medicine, a comment on an article. It is a	27
	comment on an article which was published in Emergency	28
	Medicine Australia, which claimed that a very high	29
	percentage of people are infected in Papua New Guinea.	30
Q.	When you say a 'response' or a 'comment', we're talking	31
	about something, again, in the form of like a letter to	32
	the editor.	33
Α.	You can say it like that.	34
Q.	You had this article last time, this is what we say	35
	about it.	36
A.	It is the people which responded. What I did was I	37
	commented on their paper.	38

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HIS	HONOUR	1
Q.	Commentary or critique.	2
A.	Yes.	3
XXN		4
Q.	You're not talking about a full article that went	5
	through and critiqued everything that was in the paper,	6
	we're talking about a short -	7
A.	Yes, it is a short article.	8
Q.	Like in the form of something like a letter to the	9
	editor.	10
A.	It is a short article, it is not a letter to the editor.	11
Q.	Was that peer-reviewed.	12
A.	Yes, and it was sent to the authors and the authors	13
	replied but they did not respond to their question or to	14
	the problems we arrived at.	15
Q.	Was it peer-reviewed by the journal.	16
A.	I beg your pardon?	17
Q.	Was it peer-reviewed in the journal.	18
A.	Yes, as far as I know it was peer-reviewed by the	19
	journal. It was peer-reviewed by the journal.	20
Q.	That particular journal; Emergency Medicine Australia.	21
A.	Yes.	22
Q.	That is not a specialist journal, of course.	23
A.	It is a specialist journal. Turner is an emergency	24
	physician and it is a journal of the association. It is	25
	a specialist journal.	26

Q.	Not a specialist journal, in the sense that it relates	2/
	to urology, epidemiology or biology, it is emergency.	28
Α.	It is a clinical journal, that is what I am saying.	29
	There is a big difference between a clinical scientist	30
	and a research scientist. There is a difference between	31
	them.	32
CON	FINUED	33
		34
		35
		36
		37
		38

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Q.	You see if we go through their list of articles, what we	1
	see is they are all critiques of other people's work.	2
A.	No, they are theories. They are theories and let me	3
	tell you what is the difference between a theory and	4
	experimental work. Theory: experiments produced data.	5
	The experiments produced data. The theories unite this	6
	data and make predictions. Maybe you will allow me to	7
	read something of - one page of what the experts is	8
	saying about AIDS; prediction. That is what Montagnier	9
	did, that is what Val did and that is what I did. The	10
	theories, they unite and they come out with a united	11
	few, and they make prediction in and a theory is -	12
	theoretical research is the one which makes science to	13
	progress. The theories make science progress. Maybe	14
	you will allow me to read something from the editor -	15
	from the ex-editor of Medical Hypothesis. In fact I	16
	will read from HIV expert.	17
Q.	What are you reading.	18
A.	I am reading what is a theory.	19
Q.	Where is it from, who wrote it, what document are you	20
	referring to.	21
A.	It was written by a gentlemen who is a HIV expert	22
	published in 'Genetica'. This is a journal, 'Genetica',	23
	published in 1995.	24
MS	MCDONALD: Could I see what the witness is referring	25
	to?	26

HIS	HONOUR:	Show the document to Ms McDonald.	2.
MS I	MCDONALD:	No, I don't consent to the witness	28
	referring to the	hat document, it is a secondary document.	29
	Can I indicate	from now on, if the witness is going to	30
	refer to other	documents, it should be the primary	31
	document and re	eferred in court.	32
A.	There is a slice	de, I am just reading.	33
HIS	HONOUR		34
Q.	We need the pr	imary document. There is no point in	35
	referring to so	omething that was prepared from a primary	36
	document. If	you are going to refer to something you	37
	need the prima:	ry document.	38

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MR BORICK: To say it is a secondary document in this	2
jurisdiction is taking it a bit far. She has got a note	3
of what was said. It is an accurate note of what was	4
said and she wants to refer to it.	5
MS MCDONALD: I don't necessarily accept it is an	6
accurate note.	7
HIS HONOUR: It is supposed to be a quote I assume	8
Ms McDonald.	9
MS MCDONALD: Therein lies the problem. As your Honour	10
will see as we go through these articles now, the quotes	11
are quite often out of context and quite misleading.	12
MR BORICK: I object to that because there has so far	13
been one allegation of misrepresentation put and that	14
had no legs in it whatsoever. If they want to make	15
these claims of misrepresentation, get them out in the	16
open.	17
HIS HONOUR: Right.	18
HIS HONOUR	19
Q. Is the original of this article available.	20
A. The original?	21
Q. The actual Genetica 1995.	22
A. It is. I have the whole - I can ask somebody in my	23
department to photocopy the whole article and send it to	24
me. I have the whole journal because I published two	25
papers there and they gave me - send me the actual	26

A. It is article published in the journal 'Genetica'.

is	sue.						27
HIS HO	NOUR:	On the a	ssum	ption that	the journal	can be	28
pro	oduced I wi	lll allow t	he e	vidence de	bene esse.		29
MS MCD	ONALD:	Can I as	sk if	there are	going to be		30
fu	rther artic	cles relied	l on,	we get co	pies of them	. We	31
are	e being amk	oushed ever	ry st	ep of the	way.		32
HIS HO	NOUR:	I unders	stand	the diffi	culty.		33
MR BOR	ICK:	You are	not !	being ambu	shed because	there	34
is	a huge amo	ount of mat	eria	l and it i	s impossible	for a	35
la	yman to kno	ow where th	ne sc	ientist is	going to co	me from	36
in	this. It	could take	up	this whole	courtroom.		37
HIS HO	NOUR:	Ms Papac	lopul	os-Eleopou	los said she	would	38
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get a copy of the article, it can be faxed across, or I
                                                                 1
   presume that the publication might be available in some
                                                                 2
    medical library here.
                                                                 3
                  Could well be available here, I need to
MR BORICK:
                                                                 4
    check.
                                                                 5
HIS HONOUR:
                   In any event, I will allow the evidence
                                                                 6
    to be given rather than have an argument about whether
                                                                 7
    it can be given on the basis that the original article
                                                                 8
    be produced.
                                                                 9
HIS HONOUR
                                                                10
Q. You were saying.
                                                                11
A. He says -
                                                                12
MR BORICK:
                  Sorry, I am not sure we got who 'he' was.
                                                                13
HIS HONOUR
                                                                14
Q. Who is he.
                                                                15
A. Harris, an HIV expert in an article published in
                                                                16
    Genetica 1995. He says 'The power of prediction is thus
                                                                17
    all important in evaluating candidate causal factors for
                                                                18
    the cause of effects which cannot be directly
                                                                19
    manipulated such as in AIDS' he says. 'It has been
                                                                20
    observed by the late Karl Popper, noted philosopher of
                                                                21
    science, that almost no theory is ever absolutely wrote
                                                                22
    out, "falsified" ' in parenthesis 'by experiment because
                                                                23
    with enough imagination nearly any theory can be linked
                                                                24
    after the fact so what' continues to explain all data.
                                                                25
    That is continuing to explain all data. 'If one causal
                                                                26
```

factor not explained, results statistically in a given	27
situation, it is not necessary to adopt it. One may	28
instead postulate an additional factor which explains	29
results in the case where the first one fails. In fact,	30
if one persists in hypothesising new factors each time	31
an old factor fails, one will need to never adopt any	32
hypothesis at all. At some point however, during this'	33
- during this - 'makes any theory simply too ugly and	34
unimaginative to be believed. In that "point", in	35
parenthesis, 'if a better alternative is not in view	36
many scientists may decide to discard the whole theory	37
or at least most scientists will, as Max Blank pointed	38

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	theories".' So you can keep a theory going. You need a	2
	theory. First thing is you need a theory including	3
	AIDS.	4
XXN		5
Q.	Can I check what you are referring to, still the	6
	PowerPoint slides.	7
A.	It is a PowerPoint slide. So, you need the theory in	8
	any science. In any scientific subject you need theory	9
	including AIDS according to Harris. According to	10
	Harris, a theory, if you want it, can go and go and go	11
	forever. But at one point you have to stop it.	12
	Unfortunately, this is what happened with HIV. It has	13
	been modified so many times that I think it is reaching	14
	- it is my view - is reaching a point where you can't	15
	modify it any more. But, what I want to stress here,	16
	research is not what everybody thinks. Research is most	17
	of all a theory and in fact, maybe it will come later,	18
	if I can find - I can't find now - the purpose of the	19
	medical hypothesis, it was to encourage publication of	20
	theories of hypothesis because according to the author,	21
	to the editor, an MD, he says - and I will find and give	22
	later the PowerPoints - he says in medicine and biology	23
	we have too much data but we do not have theories to	24
	connect all this data and he thinks that physicists and	25
	chemists will be able to do that.	26

out, "only death remove the last diehard belief in some 1

Q	. I want to ask you about one of your PowerPoint slides,	27
	A5, slide number 95. If the witness could have to	28
	PowerPoint print-out in front of her.	29
A	. I think if you read it, doesn't matter if I have it.	30
	Slide?	31
Н	IS HONOUR	32
Q	. 95.	33
A	. Yes.	34
XX	KN	35
Q	. You gave some evidence about that slide and before we	36
	start to go into the study I want to remind you of what	37
	your evidence was in relation to this particular slide.	38

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A.	Yes.	1
Q.	It appears at p.73 of the transcript, looking at number	2
	95, this was your evidence 'This is the main study.	3
	This is a study published this year, so again, as I	4
	said, the more HIV you have, the more AIDS you have, the	5
	more death from AIDS you have. However, a paper	б
	published this year by Europe, it was a European study	7
	there were 22,000 - over 22,000 patients treated with	8
	HAART, that is active retroviral therapy. These are the	9
	drugs which are presently used to treat HIV infection.	10
	All they found - this drug came into clinical practice	11
	in about '96, but with time they are - the HIV experts	12
	claim they improve the treatment, improve the combining	13
	of the virus and that led to better control of HIV. Now	14
	by viral law'.	15
HIS	HONOUR: That should be 'load' I think.	16
XXN		17
Q.	'By viral load, that means the number, they say that	18
	viral loads means the number of HIV particles in the	19
	population. So they found out that the better the	20
	retrovirus control, that is from 1996 until 2003, they	21
	had success in decreasing HIV, although this did not	22
	translate in having less mortality from AIDS. In fact	23
	they said the rate of AIDS in the most recent period	24
	increases. This is the - Professor Cooper made a	25
	comment he wrote a commentary in Langet about this	26

	paper and he said that - this is his word - a	27
	"paradoxical finding", or it is paradoxical if you can	28
	see that the AIDS theory because the less HIV you have	29
	the less AIDS you should. They found the opposite. The	30
	less HIV they have in the last few years, not only the	31
	mortality did not decrease, the rate of AIDS increases.	32
	So something else must be involved in causing AIDS and	33
	increasing the cell'. Then you move on to another	34
	paper. Firstly, do you agree that was the evidence you	35
	gave.	36
A.	Yes.	37
Q.	Are you aware that people who are on antiretroviral	38

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	medication for a period of time build up a resistance to	1	
	the drug.	2	
A.	No. No, no resistance to the drug you said.	3	
Q.	People on antiretroviral medication for an extended	4	
	period of time can become resistant to the drug.	5	
A.	Yes, they can and they say if you become resistant to	6	
	the drug you don't increase the viral load, that is what	7	
	resistance means, you don't decrease the viral load.	8	
Q.	Is it also the situation at present it is believed that	9	
	the period of time for which someone can be on an	10	
	antiretroviral medication before building up a	11	
	resistance for it, is about ten years.	12	
A.	Yes. Ten years? No, they have much - no they have said	13	
	- even after a few years people start having increased	14	
	viral load again. So, yes.	15	
Q.	This particular slide, and part of your evidence was	16	
	based on an article called 'HIV treatment response and	17	
	prognosis in Europe and North America in the first	18	
	decade of highly active antiretroviral therapy: a	19	
	collaborative analysis', is that correct,	20	
A.	Yes.	21	
MS	MCDONALD: I tender that and I have a copy for your	22	
	Honour.	23	
EXH	IBIT #P18 ARTICLE ENTITLED 'HIV TREATMENT RESPONSE AND	24	
PROGNOSIS IN EUROPE AND NORTH AMERICA IN THE FIRST DECADE OF 2			
HIGHLY ACTIVE ANTIRETROVIRAL THERAPY: A COLLABORATIVE			

ANA	LYSIS' TENDERED BY MS MCDONALD. ADMITTED.	27
		28
XXN		29
Q.	The paper that has been produced to you -	30
A.	Yes.	31
Q.	That is the one you have referred to.	32
A.	Yes.	33
Q.	I will go through this in a little detail. There is a	34
	summary at the beginning.	35
A.	Yes.	36
Q.	Which says 'Background to highly active antiretroviral	37
	therapy (HAART), the treatment of HIV infection was	38

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	introduced a decade ago. We aimed to examine trends in	1
	the characteristics of patients starting HAART in Europe	2
	and North America and their treatment response and short	3
	term prognosis. You agree that is a summary of what	4
	this is all about.	Ē
A.	Yes.	6
Q.	There are two things they're looking at there, aren't	7
	there. Firstly, the characteristics of someone who is	8
	going onto HAART for the first time.	9
A.	Sorry?	10
Q.	There are three things that summary would suggest that	11
	the authors of the article were looking at.	12
A.	Yes.	13
Q.	Firstly, the characteristics of a patient who was	14
	commencing with the antiretroviral medication.	15
A.	What do you mean by 'characteristics'?	16
Q.	Look at that summary. Can you see that.	17
A.	Yes, I see the summary, yes, for the treatment of HIV	18
	infection introduced a decade ago.	19
HIS	HONOUR	20
Q.	You need not read it to me, I can read it. If you look	21
	at the summary the question is: was one of the purposes	22
	of the article to examine trends in the characteristics	23
	of patients starting HAART.	24
Α.	Yes. Yes.	25

26

Q. In Europe and America.

A.	Yes.	27
XXN	XXN	
Q.	One of the things looked at what were the	29
	characteristics of the patients first starting on the	30
	medication.	31
A.	Yes.	32
Q.	Secondly, their treatment response, how they responded	33
	to the treatment.	34
A.	Yes.	35
Q.	And the third thing the authors were looking at was the	36
	short term prognosis of those individuals.	37
A.	Yes. I agree.	38

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Q.	If we go down the article, we see that in relation to	1
	methods they set out how they approached this. I won't	2
	take you through all the detail. They looked at a	3
	number of - 22,217 people who were first starting on	4
	HAART and some other criteria.	5
A.	Agree. Yes.	6
CON'	TINUED	7
		8
		9
		10
		11
		12
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		14
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		25

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Q.	We then have what's described as the 'Results'.	1
A.	Yes.	2
Q.	And they are that the proportion of heterosexually	3
	infected patients increased from 20% in 1995 to 47% in	4
	2002-2003. Do you agree that's what the author has	5
	reported.	6
A.	Yes, that's what it says there.	7
Q.	This is a paper that you relied on in your PowerPoint	8
	presentation; isn't it.	9
A.	Yes.	10
Q.	These authors seem to think that it's sexually	11
	transmitted.	12
A.	That's what they claim but where is the evidence?	13
Q.	These authors suggest that the work they have done	14
	indicates that heterosexual transmission is increasing.	15
Α.	That is all they claim, but as I presented in my	16
	evidence there is no, in my evidence in October, there	17
	is no evidence that there is any heterosexual	18
	transmission. There are no studies, scientific studies	19
	in which heterosexual transmission has been proven.	20
Q.	You don't agree with that bit of the paper so you didn't	21
	bring it to our attention in the PowerPoint, is that the	22
	situation.	23
A.	The PowerPoint was not about sexual transmission. This	24
	is not a study on sexual transmission. This is the	25
	study on the effects of antiretrovirals on the	26

	progression to AIDS. It has nothing to do with a	27
	scientific study to prove heterosexual transmission,	28
	nothing.	29
Q.	Wasn't one of the purposes of this article to examine	30
	the trends in the characteristics of patients starting	31
	HAART.	32
Α.	Yes. They were told there that this person had	33
	heterosexual - it does not prove, they don't present	34
	evidence that these people were heterosexually	35
	transmitted. What HIV - let us assume that there is	36
	such a thing, they accepted that, they accepted that	37
	these people got HIV, become HIV positive, they did not	38

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	present any data for that.	1
Q.	One of the things they have reported as finding in the	2
	very first line of their results is an increase from 20%	3
	to 47% of heterosexually transmitted HIV.	4
A.	Their study population - in their study population they	5
	had more people who have claimed over the years, who	6
	were claiming that they had acquired HIV heterosexually,	7
	but there is no proof of that and there is no need for	8
	that because this study was not conducted to prove	9
	heterosexual transmission. This study was conducted to	10
	prove or to find out what is the effect of	11
	antiretrovirals on the outcome of HIV, on AIDS, that is	12
	what it was all about.	13
Q.	Is the next result that the authors reported on an	14
	increase in the proportion of women from 16% to 32%.	15
A.	Yes, that's what they say there.	16
Q.	So, again, looking at the characteristics -	17
A.	That is what they have. They have more women at this	18
	time, that is true.	19
Q.	Is the next result that they report at finding a change	20
	in what's described as the median CD4 cell count.	21
A.	Yes.	22
Q.	I will read it in context: 'The median CD4 cell count	23
	when starting HAART increased from 170 cells' - I will	24
	put it in general terms rather than putting too much	25
	detail - 'then decreased to about 200 cells'.	26

HIS HONOUR:	'Increased', '269 cells', is that right,	27
what you're put	ting Ms McDonald?	28
MS MCDONALD:	They have described it as 'decreasing'.	29
HIS HONOUR:	From where are you reading?	30
MS MCDONALD:	Sorry, under 'Results': the median CD4	31
cell count'.		32
HIS HONOUR:	'When starting HAART increased from -'	33
MS MCDONALD:	Sorry.	34
XXN		35
Q. 'Increased from	a 170 cells to 269 cells in about 1998 but	36
then decreased	to about 200 cells'. Do you see that.	37
A. Yes, I do.		38

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Q.	And you agree that's another characteristic they were	1
	looking at in terms of people starting out on HAART, or	2
	antiretrovirals.	3
A.	What they are doing there, they divided patients in	4
	different periods of time and they found out that when	5

different periods of time and they found out that when	5
the patients were given in the first period of time the	6
HAART they had a CD4 count of 170, then they increased	7
to 269, but then in the next stage they increased again,	8
but this is so small variation, scientifically	9
insignificant. If you go and measure your CD4 counts in	10
the morning and then you do a different count in the	11
afternoon you will find much bigger variation than this,	12
in fact the variation can be 2-300 counts, so this	13
statistically and scientifically, biologically is not	14
significant, but, if you see, that HAART did not have	15
any affect on the CD4, that is Professor Cooper too. So	16
immunologically, HAART did not have any significant	17
effect.	18

- Q. And the last line in terms of the results is the 19
  'Interpretation' I'm still looking at the first page. 20
- A. The first page?
- Q. Yes, under the heading 'Results'.
- A. You're going back to the first page? 23
- Q. Under the heading 'Results', 'Interpretation: 24 virological response after starting HAART improved over 25 calendar years', so the medication seemed to work. 26

A.	The proportion - is that what you're reading, you're	27
	reading the results?	28
HIS	HONOUR	29
Q.	No, the interpretation on p.1.	30
A.	Yes, the 'virological response after starting HAART' -	31
XXN	XXN	
Q.	'- improved over calendar years', so there is an	33
	improvement with HAART.	34
A.	Yes.	35
Q.	But in statistical terms that improvement hasn't	36
	decreased - sorry, but such improvement has not	37
	translated into a decrease in mortality.	38

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A.	Exactly, it's what I am saying.	1
Q.	We will go to some of the details about what the authors	2
	say about that. For that purpose we might go to p.454,	3
	the page numbers are at the bottom of the page.	4
A.	That is in this?	5
HIS	HONOUR	6
Q.	Yes, the page numbers are at the bottom.	7
A.	Yes, which page?	8
Q.	454.	9
A.	Right, yes.	10
XXN		11
Q.	We will go to the heading 'Discussion'.	12
A.	Yes.	13
Q.	So it starts off with a summary of the situation.	14
A.	Yes.	15
Q.	'The results of this collaborative study, which involved	16
	12 prospective cohorts, over 20,000 patients with HIV 1	17
	from Europe and North America, show that the virological	18
	response after starting HAART has improved steadily	19
	since 1996'. Would you agree that's what it says there.	20
A.	Yes.	21
Q.	So things have improved since 1996.	22
A.	What has improved, the HAART, the viral load decreased,	23
	but as it said in the interpretation, but such	24
	improvement, that is the decrease in the viral load, has	25
	not translated into a decrease in mortality. So that	26

	is, we have a decrease in HIV, in viral load means HIV,	27
	but to have no decrease in mortality. The reason we	28
	give HAART is to decrease mortality, not to decrease the	29
	viral load.	30
Q.	Can we go back to the section headed 'Discussion' where	31
	the authors set out their understanding of what's	32
	occurring here.	33
Α.	They start then to make assumptions.	34
HIS	HONOUR	35
Q.	One moment. Let Ms McDonald put the question and then	36
	you can answer it.	37
		38

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XXN		1
Q.	I might follow up on that. Did you then say the authors	2
	then started 'to make assumptions'.	3
A.	I'm trying to explain why this happened.	4
Q.	They actually offer an explanation for why there is that	5
	paradoxical reaction in the discussion; don't they.	6
Α.	They don't have evidence for that. Please, just tell me	7
	please what is their evidence?	8
Q.	The authors go through in some detail why it is that	9
	this sort of outcome has occurred; don't they.	10
Α.	They try to explain it.	11
Q.	So you select from that paper or study a couple of	12
	sentences about an observed outcome and you just	13
	disregard the explanations provided by these authors for	14
	why that outcome might be so.	15
A.	No, I have quoted there exactly what they say. A vast	16
	virological improvement which is not translated being a	17
	decrease in mortality and I reported - even Professor	18
	Cooper and Professor Cooper's interpretation is that	19
	this is a paradoxical finding - you have a decrease in	20
	viral load, no matter who are the people, whether it	21
	were women, it was blacks, because you treat all these	22
	people. The HAART is given to women, to men, to	23
	Africans, black, white, they all are on HAART and if	24
	HAART is to be used to decrease mortality they should	25
	have an effect in all of them, not in an effect in men	26

	and not an effect in women and vice versa, and not an	27
	effect in blacks and not whites; HAART should be	28
	effective in everybody. As I said, I do not have to say	29
	anything, I don't have to make any interpretation.	30
	Professor Cooper's interpretation is that these findings	31
	is paradoxical and we quoted what Professor Cooper says.	32
	We didn't take anything out of context, neither from the	33
	main paper, not from what Professor Cooper says,	34
	comments.	35
Q.	We will let Professor Cooper comment on that. In	36
	relying heavily, as you have, on this study and in	37
	giving your evidence you didn't think it appropriate as	38

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	an objective scientist to give his honour the	1
	explanation given by the people who conducted the study.	2
Α.	We are saying what was the finding. What they're trying	3
	to explain, even then, they don't say 'This is the	4
	interpretation'. They don't say that.	5
Q.	Let's go to what they say then, shall we.	6
Α.	Yes.	7
Q.	So we have dealt with the first sentence which starts	8
	'The results of this collaborative study which involved	9
	12 prospective cohorts, 20,000 patients with HIV 1 from	10
	Europe and North America, showed that the virological	11
	response after starting HAART has improved steadily	12
	since 1996'. We have dealt with that. They go on to	13
	say -	14
Α.	No, please read the next sentence.	15
HIS	HONOUR: She is about to.	16
XXN		17
Q.	They go on to say 'However there was no corresponding	18
	decrease in the rates of AIDS or death up to one year of	19
	follow-up. Conversely, there was some evidence of an	20
	increase in the rate of AIDS in the most recent period'.	21
	I pause there. That's what you've been referred to as	22
	paradoxical outcome.	23
Α.	That's what Professor Cooper interpreted as the paradox	24
	and that's what we presented in our slides, so we did	25

not misquote anything or misinterpret.

26

Q.	Don't keep going back to Professor Cooper. It's your	27
	evidence that this is a paradoxical outcome.	28
HIS	HONOUR	29
Q.	You agree with Professor Cooper.	30
A.	I do agree with Professor Cooper.	31
XXN		32
Q.	The authors go on then to discuss that, don't they.	33
	They say 'The trends were accompanied by changes in the	34
	characteristics of patients starting with HAART. In the	35
	early years when HAART was being introduced most	36
	patients were men who were having sex with men but by	37
	2002 most patients starting HAART had been infected	38

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	through neterosexual transmission. Over the same time a	1
	proportion of female patients doubled'. I will pause	2
	there, then. You disagree with all of that, you say	3
	that's not proof that it's sexually transmitted and so	4
	forth.	5
Α.	I do not disagree that the number of women may have	6
	increased. I do not know how much we have increased,	7
	from one to two. I do not know how much the increase	8
	was, but - yes, but I would - what I disagree is that	9
	these people got HIV by heterosexual conduct.	10
Q.	Then there is another important detail that they	11
	provide, isn't there, that 'The median CD4 cell count	12
	when starting HAART has declined in recent years'.	13
Α.	Yes.	14
Q.	So what that means is that when you look at the	15
	statistics that over the years people starting HAART	16
	were getting lower and lower CD4 counts.	17
Α.	Yes, that is what I'm saying. That was one of the	18
	paradoxes. The less HIV you have the less CD4 you have,	19
	should be that way around.	20
Q.	What I'm suggesting to you is that if you read what the	21
	authors say there, is that a person starting HAART in,	22
	say, 1998 on average had a higher CD4 level than someone	23
	starting HAART in, say, the year 2000.	24
A.	What is the difference?	25

Q. That you're having people later in time -

26

A.	No, no, I'm saying what was the difference because we	27
	have to have the number?	28
Q.	The difference is in the results.	29
A.	Where? One second. In the early years - what is the	30
	sentence you read me?	31
Q.	I'm sorry.	32
A.	The sentence that you have read me?	33
Q.	I've read to you the last sentence in the first	34
	paragraph under the heading 'Discussion'.	35
HIS	HONOUR	36
Q.	'The median CD4 cell count when starting HAART has	37
	declined in recent years'.	38

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A.	Which place, sorry?	1
Q.	Under 'Discussion' on p.454.	2
Α.	Yes, 'Discussion' in the second paragraph or the first?	3
Q.	The first paragraph.	4
A.	I see, 'The CD4 cell count when starting HAART has	5
	declined in recent years'. What was the decline? We	6
	have to have a number there.	7
Q.	Do you say that you don't accept that statement.	8
A.	It may have been but I don't know the exact number.	9
ADJ	OURNED 12.58 P.M.	10
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RESUMING 2.02 P.M.	1
MS MCDONALD: There is a matter I wish to raise before	2
I continue cross-examining. At the beginning of the	3
luncheon adjournment I raised with my learned friend,	4
Mr Borick, whether he would speak to the witness about	5
not speaking to the other witness about her evidence.	6
Mr Borick has declined to do that on the basis they are	e 7
experts. In my submission, he should be instructed his	8
witness is not to confer about the witness's evidence	9
during cross-examination. In the absence of that I	10
would invite your Honour to say something to the	11
witness, in my submission that is just not appropriate.	12
MR BORICK: It's again emotive, I didn't decline to,	13
I had to go off somewhere in a hurry and I said to both	n 14
of the witnesses not to talk about it at all during the	15
lunch hour.	16
Now, I would like to clarify with you at some point	17
in time, perhaps later in the afternoon, what they can	18
and can't talk about. My view is very clear, they have	e 19
been cited as collaborating together for 25 years.	20
There is nothing new that has come out of this	21
cross-examination and there can be no possible prejudic	e 22
to the prosecution. In return I record a clear	23
understanding that when the other side start their case	24
they can all come and sit in here and all come and talk	25
with each other as much as they like. Perhaps we'll	26

	talk about it later.	27
HIS	HONOUR: Ms McDonald, we'll speak about it later,	28
	in the meantime you continue your cross-examination.	29
	Mr Borick my preliminary view is though, whether it's an	30
	expert or not, whilst the witness is under	31
	cross-examination they really shouldn't discuss their	32
	evidence with another expert. Because it's their	33
	opinion which is being asked for and although there has	34
	been a collaboration - I'll hear you if there is any	35
	challenge about it - I think it's desirable that she not	36
	speak about her evidence whilst under cross-examination.	37
MR	BORICK: Yes, but sometimes there's reference to	38

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documents they haven't got or something. I need to know	1
what they are about.	2
HIS HONOUR: You can certainly speak to her and you	3
can speak to Dr Turner, I mean he is in court, so he is	4
hearing what she's got to say. So if you need to speak	5
to either of them about locating documents or getting	6
instructions that is entirely appropriate. It's just	7
that it would in my view be not appropriate for	8
Ms Papadopulos-Eleopulos to speak to Dr Turner about the	9
evidence that she is giving and to get his views perhaps	10
about what she is saying in the witness box.	11
MR BORICK: I'll frame something during the course of	12
the afternoon which they will both have and both can	13
understand, I'll give it to your Honour and my friend.	14
HIS HONOUR: You don't need to frame anything	15
Mr Borick, unless you take a different view to me then	16
I'll hear any submission about it. But I can certainly	17
tell the witness - clearly they are probably staying at	18
the same hotel, they are probably eating together and so	19
forth, but what they should not - and you know, they're	20
professional people, but what would concern me is if	21
they're discussing the substance of the evidence that	22
Ms Papadopulos-Eleopulos is giving, that's all.	23
MR BORICK: They've heard that, I'll make it clear to	24
them.	25
XXN	26

Q.	have you been discussing your evidence with Di luther as	۷ /
	events have proceeded in court so far.	28
A.	We have not discussed anything, we cannot, we just went	29
	to lunch together. But we did not discuss anything	30
	about the court case. I had a look at the paper, but I	31
	never discuss it with Dr Turner.	32
Q.	What about before the luncheon adjournment, when	33
	Mr Borick spoke to you.	34
Α.	No.	35
Q.	At no stage have you spoken to -	36
Α.	Not in relation to my evidence.	37
Q.	Can I go back to the document I was asking you about,	38

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	P18. If the witness could have that exhibit.	1
A.	27?	2
HIS	HONOUR	3
Q.	P18; that's the HIV treatment response.	4
A.	HIV treatment response, yes.	5
XXN		6
Q.	We were dealing with the page with the heading	7
	'Discussion' which is 454.	8
A.	Yes.	9
Q.	We'd got to the end of that first paragraph which reads	10
	'The median CD4 cell when starting HAART has declined in	11
	recent years'. You agree with what that says.	12
A.	It says that, yes; that is what is written.	13
Q.	You were asked before lunch 'Well, where are the	14
	figures'. If you go back to the first page, where it	15
	says 'Results', it sets out the figures. It indicates	16
	that in the very early stages, when the treatment was	17
	introduced, people started with a lower CD4 count, about	18
	170, and then 95/96 it peaked if people were starting	19
	HAART.	20
A.	If I could look -	21
Q.	If I could finish. Starting HAART they would have 269,	22
	then at the end of the study period that had decreased	23
	back to about 200 cells. Firstly, do you agree that's	24
	what it says there under 'Results'.	25
A.	Under 'Results' that's what it says. And that is what	26

	is in the figure as well, in the table.	27
Q.	That's what they're indicating isn't it, that a factor	28
	that is relevant here is that in the more recent times	29
	in the study, people who were starting HAART	30
	antiretroviral medication had a lower CD4 count; which	31
	in effect they were sicker.	32
Α.	No, I do not agree with that, because the difference -	33
	the first period was 170, that figure was the lowest	34
	CD4, 170. Now after that period, in the other periods,	35
	it increased. The CD4 increased. And in the last	36
	period it was 202, still higher than it was in the first	37
	period. But the changes which took place are not	38

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	statistically significant, and certainly not	1
	biologically significant.	2
Q.	Well, the authors seemed to think it was statistically	3
	significant didn't they.	4
A.	They couldn't say. They do say that there was a lower,	5
	but they don't say that was statistically significant.	6
Q.	They say it was a significant.	7
A.	The median - sorry, the median CD4 cell counts when	8
	started HAART declined in recent years; that's what it	9
	says.	10
HIS	HONOUR	11
Q.	One must assume, must one, that they would have thought	12
	that was significant otherwise they wouldn't have come	13
	to that conclusion.	14
A.	Sorry your Honour, but you can say that they are, and	15
	some people they decline, but not significantly	16
	statistically significant difference, or they say they	17
	are higher statistical difference, they have to clarify	18
	that.	19
XXN		20
Q.	We'll go through and look at how they clarify it then	21
	shall we.	22
A.	Yes.	23
Q.	Continue under what the authors say about 'Discussion',	24
	starting at the second paragraph under that heading.	25
	'The discrepancy -', do you have that.	26

A.	'The discrepancy between the -' yes.	27
Q.	Yes, they then say 'The discrepancy between the clear	28
	improvement we recorded virological response and the	29
	apparently worsening rates of clinical progression might	30
	be related to the change in the demographic	31
	characteristics of study participants, with an	32
	increasing number of patients from areas with a high	33
	incidence of tuberculosis. For example, in the Swiss	34
	HIV Cohort Study there was a steady increase in the	35
	number of patients from sub-Saharan Africa. These	36
	patients were younger, more likely to be female and more	37
	likely to have been infected heterosexually than other	38

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study participants. Also, they had lower CD4 counts at presentation and the most frequent AIDS defining event 2 was tuberculosis'. I'll keep going for a little bit 3 more. 'Similar trends have been seen in other european countries and in North America. In the USA the rates of 5 tuberculosis are increasing in foreign-born people, and 6 outbreaks are increasingly common in other groups at 7 high risk of HIV infection, including prisoners, 8 homeless people, and gay and transvestite and transsexual HIV infected men.' I'll pause there. So 10 you agree, the authors say they are actually 11 characteristics about the people who are embarking upon 12 a series of antiretrovirals which may explain why it is 13 there has been this paradoxical result. 14

A. These people, they are AIDS patient, in the vast 15 majority of people who are said to have AIDS, as I said 16 before, TB. If HAART works then it should work more 17 than anywhere else in people who have TB. If it doesn't 18 work in this type of people then we can't, it cannot be 19 an effective treatment. Now, may I your Honour respond 20 to this, because there is evidence that we have to look 2.1 at how HAART is supposed to work and how HAART - what is 22 meant by HAART has a virological effect. By decreasing 23 the viral lot there is no proof that the HAART has a 24 virological effect. And it is a very good reason for 25 it. HAART, the branch which are included in HAART by 26

define on the HIV DNA, they do not act directly on the	27
HIV RNA, they act by decreasing the HIV DNA, in the	28
profiles, what is called HIV DNA is incorporated in the	29
cellular DNA. And if there is such a thing as an HIV	30
profile, as an HIV DNA, by design these drugs should	31
decrease the HIV DNA, and then in return because of the	32
decrease of the DNA then we should have a decrease in	33
the RNA. There is no other way. And this - in no study	34
this has happened. In fact, we have a study from	35
Australia which Professor Cooper, as the core author,	36
they have shown that HAART does not decrease the HIV	37
DNA And we sent a letter to the journal the Journal	3.8

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	or infectious biseases, patering several questions now	_
	can this be explained. How can we say that the HAART	2
	has an antiviral effect? And the journal published our	3
	letter and asked the authors to respond. Well they	4
	responded but they did not answer any of our questions.	Ę
	It is published there, they who can give their article	6
	and our letter and their response. It is impossible for	7
	HAART to have an antiviral effect. Similarly, a group	8
	from Italy they have studied the effect of HAART on the	٥
	viral DNA, that was on HIV. And they can find out	10
	paradoxically - they use another word, I haven't got the	11
	word but is the same meaning - that following HAART	12
	treatment the HIV DNA increases instead of decreasing.	13
	So whichever what happens, whatever these drugs have it	14
	cannot be an antiviral effect.	15
Q.	So you are referring there to some Italian study.	16
A.	An Italian study, and study from here, I'll give you	17
	both studies.	18
Q.	Sorry?	19
HIS	HONOUR: She'll give you both studies.	20
A.	I'll give you both studies.	21
XXN		22
Q.	Before you finish your evidence tomorrow.	23
A.	Yes.	24
Q.	Now, going back to this article, the one that you relied	25
	on, there's reference in that passage I just read to you	2.6

	Tit was the most frequent AIDS defining event. Now	27
	what do you understand by the term 'AIDS defining	28
	event'.	29
A.	Is a disease which is said to prove AIDS; AIDS in	30
	communicable diseases.	31
Q.	Said to be an opportunistic infection arising as a	32
	result of a person having AIDS.	33
Α.	Not of having HIV infection, not to having AIDS as a	34
	result of having HIV infection not to having AIDS. You	35
	have the disease, that is you have AIDS resulting from	36
	HIV infection.	37
Q.	Isn't tuberculosis one of the opportunistic infections	38

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	that arise from someone having HIV.	1
A.	That's what I've been saying all this morning. That the	2
	vast majority of people who are said to have AIDS are TB	3
	patients; that's what I'm saying.	4
Q.	What I put to you that the experts in this field say is	5
	that TB is in fact an opportunistic infection that	6
	people with AIDS get.	7
A.	TB is an AIDS indicator disease, that is what is said;	8
	that's what I've been saying all this morning. I've	9
	been saying, the vast majority of people that have AIDS	10
	today are said to have TB. This - AIDS equals - I'll	11
	say, put it in a different way. TB equal AIDS, if you	12
	have a positive test.	13
HIS	HONOUR	14
Q.	For HIV.	15
A.	For HIV antibodies. TB equals TB if you have a negative	16
	test.	17
XXN		18
Q.	Aren't there many people who have had positive HIV tests	19
	who then have gone on to develop TB.	20
A.	I said yes there are. But let me repeat, is that proof	21
	for HIV infection?	22
CON	TINUED	23
		24
		25
		26

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Q. Go back to this article. The authors there, don't they, 1 seem to be providing, in fact, there are more than but at least two reasons, why it is that we might see this 3 paradoxical outcome that you have referred to so often. 4 One, is a change in the characteristics of those people 5 starting antiretrovirals in that they are coming from 6 countries like Africa where there is high TB, and 7 secondly, that people starting the antiretrovirals at 8 the time most recent to the end of the study, in fact, 9 got to a point where they had a much lower CD4 count 10 than they had previously in the mid 90s. 11 You repeat the question so let me repeat the answer. 12 Sorry? 13 0. I said you repeated the question so let me repeat the 14 Α. answer. The answer is, that from what I can see, the 15 decrease, there is no decrease. There is no 16 statistically significant decrease and the papers do not 17 say there is a significant decrease. If you take the 18 first period and the last period, the first period was 19 the period with the lower CD4 count. Now, that may have 20

changes. Now they say most of the cases - they were

doing the study, now we are doing the study - most of

Now, that may have been the case, I'm not deny it, but

TB is AIDS. If TB is AIDS, we have to treat AIDS no

matter what they have. So if TB say it is AIDS, it

the cases which are presenting now they say had TB.

21

22

23

24

25

26

	should not have worse result. If HIV is the cause of	27
	AIDS when you have less of TB, when you have less HIV	28
	you should have less TB. It is as simple as that.	29
Q.	Isn't it the case that the authors of this report, the	30
	people who conducted this study, were clearly of the	31
	view that the decline of the CD4 cell count in a person	32
	starting heart treatment was very significant in terms	33
	of the outcomes of this study.	34
Α.	Significant?	35
Q.	Let me take you to their words.	36
Α.	Yes.	37
Q.	P.455.	38

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A.	455?	1
Q.	Yes.	2
Α.	Yes.	3
Q.	We will go right to the end to the ultimate comments.	4
	Starting with the last two lines on that page, don't	5
	they say there: 'The decline of CD4 cell count when	6
	starting the heart in recent years must also be of	7
	concern. Patients starting treatment with CD4 counts,	8
	less than 200 cells are at higher risk of disease	9
	progression and death in the long-term compared with	10
	those with higher base line CD4 cell counts. Early	11
	diagnosis and treatment is, therefore, of great	12
	importance to prevent clinical progression. A survey of	13
	new HIV diagnosis in the US and Ireland show that many	14
	opportunities for earlier diagnosis are missed. Our	15
	results indicate that such oversights could be common in	16
	many countries and settings and that, therefore, an	17
	expansion of voluntary and cost-effective screening in	18
	health care settings is likely to be beneficial. The	19
	ART cohort collaboration will continue to monitor the	20
	characteristics and prognosis of HIV infected patients	21
	starting heart and updating analysis at regular	22
	intervals'. Firstly, do you agree that that is the	23
	concluding paragraph in that report.	24
Α.	That is what they are saying they should do. If that's	25
	what they can do, they can do it. I'm not arguing with	26

	that. I am only concerned about the effect of heart on	27
	the mortality and CD4. Now, 200, if you look at the	28
	CD4s, even in the last period, it was 202. So there was	29
	not more than 200, the medium count. So, this is what	30
	they are advising people to do. I'm not disagreeing	31
	with that.	32
<u>)</u> .	And aren't they also saying, in this report, that there	33
	are a number of explanations for why it is that they are	34
	saying what you have called the paradoxical outcome and	35
	a large part of it is the fact that people, when	36
	starting medication, have lower CD4 counts than they had	37
	previously.	38

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A.	But we said it again and again. They did not have lower	1
	CD4 counts.	2
Q.	I want to move on.	3
Α.	The first period had 170. In the last period they had	4
	202. In between, all of them had higher.	5
Q.	I want to move on now to deal with another slide which	6
	you have produced to the court, slides 96 and 97 in A5.	7
Α.	Which page?	8
Q.	It is slides 96 and 97.	9
Α.	Yes.	10
Q.	And these slides related to what has been referred to as	11
	the Rodriguez study.	12
Α.	Yes.	13
Q.	What I have done, as I have done before, is I will just	14
	remind you of what you have had to say about these	15
	slides in your evidence-in-chief before I ask you some	16
	questions.	17
A.	Sure.	18
Q.	P.75, line 28, you were asked to explain slide No.96 and	19
	your response was this: 'This is, as I said, even a more	20
	recent paper and in this study the authors examined HIV	21
	infected individuals who are not on any drugs and they	22
	call Heart to find out if it was - if HIV was the reason	23
	for the decline of the CD4 cells, that is for AIDS for	24
	immune deficiency. They concluded - now, really	25
	important - that "We report that plasma HIV RNA level	26

can account for only a small proportion of the	27
variability in the rate of CD4 cell loss in chronic	28
untreated HIV infection", and concluded, "Presenting HIV	29
RNA level predict the rate of CD4 decline only minimally	30
in untreated persons. Other factors as yet unidentified	31
likely drive CD4 cell loss in HIV infection. This	32
finding have implications for the treatment decisions in	33
HIV infection and for understanding the pathogenesis of	34
progressive immune deficiency". So, they're two	35
important things which one concludes from these	36
conclusions you draw. One, the HIV is responsible for	37
only - what the words they use - for a minimal decline	38

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	deficiency. There are other factors which cause the	2
	decline. Secondly, the risks get very important	3
	implication regarding the HIV theory and regarding	4
	treatment of HIV infected patient. And these authors	5
	are - I think' and then you go to slide 97. Firstly, do	6
	you agree that was your evidence.	7
Α.	Yes.	8
Q.	Then you discuss slide 97. You talked about the people	9
	who are named there. I won't go through all of that.	10
	Then you refer to some commentary from those people in	11
	relation to the Rodriguez study and you quoted them as	12
	saying: 'The provocative main finding from their study,	13
	that is the Rodriguez study, was that the HIV load	14
	predicted no more than 10% of the observed CD4 loss in	15
	patient with chronic untreated HIV infection. What	16
	factors explain the other 90%? 25 years into the HIV	17
	epidemic, a complete understanding of what drives the	18
	decay of CD4 cells, the essential event of HIV disease	19
	is still lacking'. Then you said 'And they also wrote	20
	"The findings presented by Rodriguez et al provide	21
	support to those who favour non-virological mechanisms	22
	as the predominant cause of CD4 loss"', and then you go	23
	on to say 'That is, the AIDS is caused by factors other	24
	than HIV'. Do you agree that was your evidence.	25

26

A. Yes.

of the CD4 cells. That's for acquired immune 1

Q. I want to deal with that study, the Rodriguez study.	27
NOT ANSWERED	28
EXHIBIT #P19 PAPER HEADED 'PREDICTIVE VALUE OF PLASMA HIV	29
RNA LEVEL ON RATE OF CD4 T-CELL DECLINE IN UNTREATED HIV	30
INFECTION' BY RODRIGUEZ AND OTHERS REPORTED IN THE AMERICAN	31
MEDICAL ASSOCIATION JOURNAL DATED 09/2006 TENDERED BY	32
MS MCDONALD. ADMITTED.	33
	34
Q. This is the paper you rely on at that point of your	35
presentation.	36
A. That's what I was saying, yes, this is the paper.	37
Q. Do you have it in front of you at the moment.	38

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A.	Yes.	1
Q.	You will see at the beginning there is a heading	2
	'Context'. That is the context in which this study	3
	occurred.	4
A.	Yes.	5
Q.	And then 'Objective'.	6
A.	Yes.	7
Q.	And that is to estimate the proportion of variability in	8
	rate of CD4 cell loss predicted by presenting plasma HIV	9
	RNA levels in untreated HIV infected persons.	10
A.	Yes.	11
Q.	So it was to look at the relationship between CD4 cell	12
	loss and RNA.	13
A.	Yes.	14
Q.	Viral RNA. Sorry, HIV RNA. That was my mistake.	15
A.	Yes.	16
Q.	If we go down further, there is a heading 'Results' and	17
	it starts 'In both cohorts, higher presenting HIV RNA	18
	levels associated with greater subsequent CD4 cell	19
	decline'. Do you see that.	20
Α.	Yes.	21
Q.	Do you agree that's what was said there.	22
A.	Yes.	23
Q.	And then, if we go down to 'Conclusions', 'Presenting	24
	HIV RNA level predicts the rate of CD4 decline only	25
	minimally in untreated persons. Other factors are	26

	defined, likely drive CD4 cell loss in HIV infection.	27
	These findings have implications for treatment decisions	28
	for HIV infection and for understanding the pathogenesis	29
	progressive immune deficiency'. Do you agree with that.	30
Α.	Of course. That's what I said in the slide.	31
2.	You see, you have suggested in your evidence and by the	32
	way you have presented the PowerPoint in relation to	33
	this study that the authors of this paper concluded that	34
	it is something other than HIV, something quite	35
	separate, that is leading to the decline in the CD4	36
	count.	37
Α.	That is all there was in the commentary to the authors	38

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by Layne, which is one of the best HIV e	xperts, said 'I	1
didn't say that'. I mean, I said that b	ut it was	2
something else. Layne, that is the comm	entary. They	3
say 90%. See, the viral load is respons	ible for 10%.	4
In fact, in the text, he says 4 to 6%.	Layne said 10%.	5
Layne gives 10%. If you subtract 10 fro	m 100, you are	6
left with 90, so something else leads to	90% of CD4	7
decline, or if that something else is no	t HIV - they say	8
it is something else, not me - so anythi	ng which is	9
responsible for 90% must be considered t	o be the main	10
cause of AIDS.		11
Let's go back to what I was asking you.	Is it your	12
evidence that the authors of the Rodrigu	ez report who	13
undertook this study are saying it is so	mething other	14
than HIV that is leading to the depletion	n of the CD4	15
count.		16
That's what they say. It is not me who	say, that's what	17
I say. You read it. That's what they s	ay.	18
I'm about to take you through it. I sug	gest to you that	19
that is not what the authors are saying.	All the	20
authors are saying is that it is a more	complex equation	21
than that. You just can't look at viral	load and CD4	22
count. There is not necessarily a corre	lation.	23
Well, that's what HIV material says. Th	ey don't say it	24
is more complex. They say that is the p	roblem. The	25
problem is that there is a more complex	relationship	26

Q.

Α.

Q.

A.

	between AIDS - It is more complex between AIDS and its	2 /
	causes.	28
Q.	You see, didn't two of the authors, the primary authors,	29
	Rodriguez and Lederman, actually subsequently publish a	30
	further paper to clarify what they meant in their	31
	conclusions and observations in the study, that is P19.	32
A.	Are you meaning the paper or the request of Professor	33
	John Moore?	34
Q.	The paper that is headed 'What our work means' by	35
	Mr Rodriguez and Lederman.	36
A.	Yes, it was something published, a commentary in AIDS	37
	Truth, where nobody can respond.	38

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Q.	Is that -	1
А.	Do you want me to say what they say there?	2
Q.	Have you seen -	3
Α.	It is in the paper here. I have seen the paper.	4
Q.	You have seen their response.	5
А.	Yes, I have seen their response.	6
Q.	To your understanding, did that response come about	7
	because there was a perception that this study was being	8
	misused by some members of the scientific community.	9
А.	A commentary inside this magazine by HIV expert,	10
	including Furuchi, who says that the decline in AIDS	11
	with CD4s is due to stimulation, immune stimulation not	12
	immune suppression, that is a commentary in science. It	13
	was not me commenting on that paper in print anywhere.	14
CO	NTINUED	15
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EXH	IBIT #P20 DOCUMENT HEADED 'WHAT OUR WORK MEANS' BY	1
BEN	IIGNO RODRIGUEZ AND MICHAEL LEDERMAN TENDERED BY MS	2
MCD	OONALD. ADMITTED.	3
		4
XXN	I	5
Q.	Have you seen this document prior to putting together	6
	your power point presentation.	7
Α.	We did not discuss it because it came after our power	8
	point presentation.	9
HIS	HONOUR: Has this document got a date?	10
MS	MCDONALD: The only date I pick up is the site was	11
	developed in March 2006. I will make some more	12
	inquiries about that.	13
XXN	I	14
Q.	Just to make it clear, had you seen this response before	15
	you put together your power point presentation.	16
Α.	I am repeating, we could not get proof in our power	17
	point presentation of this because it came after we	18
	presented our evidence.	19
Q.	At the beginning, the author's explain what it was that	20
	they did in relation to producing that report or study	21
	commencing 'predicted value of plasma'. They then go on	22
	and make this statement - this is two-thirds of the way	23
	in the paragraph - 'Positive, as we believe	24
	cross-examination of scientific findings to be, we have	25
	learned with growing concern about interpretation of the	26

	works that are not only inaccurate but misleading and	27
	potentially dangerous to HIV-infected persons	28
	everywhere, thus, we are writing here to clarify the	29
	significance of this work, its implications for the role	30
	of HIV viral load measurement in clinical practice and	31
	its meaning to persons living with HIV and AIDS'. Do	32
	you agree that's what it says there.	33
Α.	That is what is written.	34
Q.	When you read through this paper, that is clearly what	35
	it sets out to do, to make absolutely crystal clear what	36
	their position is in terms of the outcomes of these	37
	findings.	38

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A.	They	change	the	interp	retatio	on of	their	own	data.	1
^	TuTh a +	+ha d		a malea	a +	.h.+ .				2

- Q. What they did was make sure that no-one or attempted 2 to make sure that no-one would go around 3 misrepresenting what their initial data had been. 4
- A. If Furuchi misrepresents the data, it is not my problem. 5

  They should talk with Furuchi. Furuchi, as you know, is 6

  the main person in HIV/AIDS in America. He's 7

  responsible of distributing over \$2 million a year for 8

  HIV research \$2 billion, sorry. 9
- Q. The authors go on to explain what they did. 'Briefly 10 used complex modelling to calculate the estimated speed 11 which HIV-infected persons not receiving treatment for 12 HIV would lose their CD4 cells over time' and then asked 13 a simple question, based on a single measurement of 14 those persons' viral loads: 'How well can one account 15 for the variation in the rate of CD4 cell loss from one 16 person to the next. To the surprise of many the answer 17 is, very poorly, to the tune of about 4-6%'. Firstly, 18 do you agree with what that says there. 19
- A. Yes. 20
- Q. Secondly, do you agree that that is what they did in the 21 paper that's been tendered as P19.
- A. They say, to the surprise of many, and to their own
  surprise they found out that there is a very poor
  account for the decline of CD4, only 4-6%.
  25
- Q. I am asking you, do you accept that summary. 26

A.	I accept the summary, yes.	27
Q.	As a summary of what it was, that was the outcome of the	28
	initial study.	29
A.	This is the study. That's what they say in the study	30
	and that's what they say here. They say 4-6%.	31
Q.	Then they explain it. They say 'Most disturbing amongst	32
	all the interpretation of this finding, this has been	33
	taken by some to mean that our data raise doubts about	34
	HIV being the cause of AIDS. Some have gone as far as	35
	to affirm that our results prove that it is not. As	36
	this is the most damaging of all the interpretation of	37
	our work, we will address it first'. That is what you	38

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	have been suggesting in this court, isn't it, that there	1
	is something else, other than HIV, that is causing	2
	people to die of AIDS.	3
A.	I am not saying - this is not my saying, let me repeat.	4
	This is what the commentary to these papers said, that	5
	there is something else. They are their words, not	6
	mine.	7
HIS	HONOUR	8
Q.	What are your words.	9
A.	My words are too. If HIV is responsible for 4-6%, then	10
	there must be something else.	11
Q.	You agree with those critiques.	12
A.	I agree with the commentary.	13
XXN		14
Q.	That is the opinion that you have been giving in this	15
	court.	16
NOT	ANSWERED	17
HIS	HONOUR	18
Q.	That is your opinion.	19
A.	That is my opinion. I am doing nothing more than	20
	repeating their findings.	21
XXN		22
Q.	Let's go on to see what these authors had to say in	23
	response to the sort of opinion that you are expressing.	24
	'There is absolutely no doubt that HIV is the cause of	25
	AIDS. Far from challenging the veracity of this	26

statement, our work further confirms it. This is easily	27
appreciated from our initial analysis of the data which	28
shows that, on average, individuals with higher viral	29
loads tend to lose CD4 cells more rapidly than those	30
with lower viral loads. There is no contradiction	31
between this finding and our main message because the	32
overall trend among a group of subjects cannot be	33
directly translated into a prediction of what will	34
happen to a single individual within that group.	35
Importantly, this finding replicates, rather than	36
disputes the substances in the paper by Mellors et al	37
and the citation. This demonstrates this 10 years ago,	38

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	conclusions are invalid precludes a combination of	2
	sloppy thinking, sloppy reading or malicious intent,	3
	which two you choose to believe'. Is that what it says	4
	there.	5
A.	That's what it says there. I don't think it was	6
	misinterpreted and I don't think Furuchi did.	7
Q.	Are the authors in this paper directly refuting the use	8
	that you seek to make of that study.	9
A.	Of course not. They said in their summary and in here	10
	that the viral load predicts only 4-6% and that's what	11
	it is and they do say it here. I'm talking about what	12
	their data says and Furuchi said that and Layne said	13
	that.	14
Q.	Do the authors then go on over the page to explain or to	15
	use an analogy to try and explain the findings they have	16
	arrived at. Going to the paragraph starting 'An often	17
	cited'; do you see that.	18
A.	I know the analogy very, very well.	19
Q.	Let's go back to it 'An often cited analogy deposits	20
	that the clinical course of HIV infection can be thought	21
	of as a train approaching a broken bridge. The CD4 cell	22
	count is the distance that separates the train from	23
	certain doom, whereas the viral load is the speed at	24
	which the train is travelling towards that point.	25
	Expanding on this image, we propose the train's fuel,	26

thus using our work to claim that those previous 1

	rather than a single material, can be thought of as a	27
	mixture of combustibles of which the number of viral	28
	particles in the blood, i.e. the viral load is but one	29
	of the components. As the relative component to the	30
	mixture changes, so does the efficiency of combustion	31
	and hence the power of the engine and speed of the	32
	train. From this follows that were the train to run out	33
	of fuel it would cease to move'. Do you agree with	34
	that.	35
A.	Yes, I know their analogy, as I said.	36
Q.	It goes on to say 'Thus in two persons with the same	37
	amount of HIV in blood, the efficiency of combustion and	38

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	hence the speed of the train, rate of CD4 cell decline,	1
	may vary. That is precisely what our work shows. For	2
	the HIV-infected patient this means that it is very	3
	difficult to predict what the pace of his or her CD4	4
	decline will be, just based on the measurements of the	5
	amount of HIV in the blood'. Do you agree that's what	6
	it says there.	7
A.	Exactly and that's what we are saying. They say, can I	8
	explain their analogy please?	9
Q.	Certainly.	10
A.	The analogy, until now it was told that the number of	11
	CD4 cells tells you how far you are from the broken	12
	bridge. If you have high level of CD4 you are very far,	13
	if you have low, you are very close to the broken	14
	bridge. This is one viral load, the other viral load is	15
	the speed of the train and the speed, until now, is	16
	determined by the viral load. The higher the viral	17
	load, the higher the speed and, thus, the sooner you	18
	reach the broken bridge. Now, the Rodriguez paper tells	19
	us that the speed is not determined solely by the viral	20
	load, in fact the viral load is responsible only - the	21
	fuel for the speed is HIV only - is responsible of only	22
	4-6%. The rest of the fuel, which speeds the train, is	23
	something else. It is exactly what they say in the	24
	paper. The rest is something else, what I told you here	25
	at the beginning, if you start with a low CD4, you can	26

start with low CD4 - in fact there is a very recent	27
paper from Amsterdam where the authors, in a study for	28
gay men, is one of the largest studies in gay men, apart	29
from the second largest study - the Max study. And the	30
authors, again HIV experts from Amsterdam, they found	31
out that there is a decline of CD4 cells - statistically	32
significant decline of CD4 cells, before a patient tests	33
positive. That decline determines how quick - that is	34
how quick - you are reaching the bridge before you are	35
HIV-infected. Whatever causes that decline of the CD4	36
cells before you reach - you become positive, there was	37
a factor there, these factors - surely they don't	38

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disappear straightaway after you become positive, they 1 will continue to be there and they will continue to 2 decrease the CD4 cells. That is what the Max study has shown. The Max study has shown, again this is the 4 largest - I'm repeating - is the largest study in gay 5 men and the longest. It started in 1984, it was about 6 5,000 men and if we had no other study but the Max 7 study, we would have known everything which has to be 8 known about AIDS in gay men. The papers from there are 9 very, very good. At one stage they publish a paper and 10 it was shown that after you become positive there are 11 other factors which determine - they use another word -12 determine the development of AIDS, so it means that 13 there are factors there which are not HIV because they 14 found out when you are HIV-infected and they study the 15 patients after they were HIV-infected and they found out 16 that people who continue to have sex and the higher the 17 sexual frequency of passive anal intercourse, the higher 18 the probability of them developing AIDS. That cannot be 19 HIV because once infected with HIV - when they're 20 infected with, say, the bacteria that caused syphilis, 2.1 then you can have as much sexual contact with people who 22 have syphilis as well, or who are infected with the 23 bacteria and it won't make any difference to how you 24 develop syphilis. Here, they found out that the most 25 sexual intercourse, and specifically passive anal 26

	intercourse, you have, the higher the probability that	27
	you will develop AIDS and you will develop it soon. It	28
	must be something else apart from HIV which determines -	29
	this is their word - determines the development of AIDS.	30
	We have now two studies - the best ever studies in gay	31
	men which say there are other factors which determine	32
	how quick the train or the speed of the train towards	33
	the broken bridge.	34
Q.	You just told the court then, the words of the author	35
	was that it is something other than HIV that causes	36
	AIDS. The authors of this report never said that. At	37
	its highest they said there is not necessarily a direct	38

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- link between the level of viral load and the level of 1
  depletion of the CD4 cells. 2
- A. AIDS is depletion AID stands for the decrease in T4 3 cells and there's a decrease in T4 cells as shown in the slide there. According to HIV, HIV infection leads to 5 the increase in T4 cells and T4 cells is the hallmark of 6 AIDS, that is anyone who has the HIV serum, the increase 7 of the cells is the hallmark of HIV infection and AIDS. 8 That means only people who have HIV will have a decrease 9 in T4 cells, which is totally shown to be not true. 10 There are many people who have a negative test for HIV 11 and they have low T4 cell counts. The second part of 12 that hallmark means that only people who have low T4 13 cells will develop the disease. That is, again, not 14 true. There are people who first develop the disease, 15 first develop pre-existing PCP, and then develop 16 decrease in T4 cells, that is the disease - as the cause 17 of the decrease in T4 cells. There are people who have 18 HIV who have positive tests and who have a decreased T4 19 cell - zero T4 cell - and they can live, from the 20 evidence we have, up to five years without developing 2.1 any disease. There goes your hallmark of HIV infection 22 and AIDS. 23
- Q. Isn't it as simple as this: you have relied on this study as supporting your opinion that HIV doesn't cause AIDS, when in fact the very authors of that study have

25

26

	come out and said that is wrong, that is not what that	27
	study means at all.	28
Α.	That is what they said in the paper and that is what the	29
	commentary says. If the commentary was not written by	30
	me, it was written by Layne and if you read the paper	31
	and if you read what is here, their analogy tells you	32
	that is exactly what the analogy tells you. I know	33
	they're saying it, they said it in the paper, they said	34
	it in their analogy and that is what Layne said in the	35
	commentary to this paper and this is what Furuchi said	36
	in the commentary to this paper in science.	37
Q.	Moving on to ask you questions about some of your slides	38

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	in relation to sexual transmission. Looking at Exhibit	
	A8, slide 6 in A8, that is your presentation on sexual	2
	transmission, relating to contact tracing.	3
Α.	Slide 8?	4
Q.	Slide 6.	5
A.	Yes, sexually transmitted diseases.	6
CON	TINUED	7
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Q.	You gave evidence in this court that there is no contact	1
	tracing in relation to HIV.	2
A.	That is what Haverkos said.	3
Q.	Wasn't that article you were relying on an article	4
	published in 1988.	5
A.	Yes.	6
Q.	An article that was published in the United States.	7
A.	Yes.	8
Q.	You see it is a matter of fact contact tracing occurs	9
	throughout Australia in relation to HIV.	10
A.	I cannot find that published in any paper.	11
Q.	You have prepared -	12
Α.	I am discussing on scientific evidence. Scientific	13
	evidence is from scientific papers.	14
Q.	You are prepared, on the basis of an article from 1988,	15
	18 years ago now, from America, to say, make a blanket	16
	statement, there is no contact tracing in relation to	17
	HIV.	18
A.	I cannot - found any scientific papers where contact	19
	tracing can be done in a population or - people said to	20
	be sexually - to have been infected by sexual contact.	21
Q.	I suggest this is a really good example of you just	22
	pulling out snippets from articles and studies to	23
	support your argument.	24
A.	No, I totally disagree with you.	25
Q.	A phone call to Department of Health would have told you	26

	that contact tracing occurs in relation to HIV in this	27
	country.	28
A.	I don't - I have to have scientific publication. I am	29
	discussing scientific publication.	30
Q.	So what if you made a phone call to the Department of	31
	Health and they told you that as a matter of course	32
	there is contact tracing for everyone diagnosed with	33
	HIV, you wouldn't have included that in your	34
	presentation.	35
A.	There is no contact tracing from everyone who has been	36
	said to be HIV infected by sexual contact.	37
Q.	You see, in this state - we will deal with this state	38

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	for a moment - if someone has a positive result to ELISA	1
	test and Western Blot, their doctor, that is the doctor	2
	who ordered the tests, is advised to contact the Public	3
	Health Authority.	4
A.	Yes.	5
Q.	Do you know that.	6
A.	Yes.	7
Q.	You did know that.	8
Α.	Yes, I know that.	9
Q.	Then the Public Health Authority either speaks to the	10
	person who has HIV directly or does it through the	11
	treating doctor.	12
Α.	Sorry, can you repeat the question?	13
Q.	Once that notification is made to the Public Health	14
	Authority, once the doctor gets the results, is told	15
	that he has to notify the public health authorities.	16
A.	Yes.	17
Q.	I should just say, as an aside, are you aware also	18
	separately to that the IMVS is obliged to notify the	19
	Public Health Authority.	20
Α.	Yes.	21
Q.	Did you know that.	22
A.	Yes.	23
Q.	And that the Public Health Authority, either directly	24
	with the person who has a positive result, or through	25
	that person's treating doctor, then speaks with that	26

	person. Did you know that.	27
A.	I do, yes.	28
Q.	With the purpose of finding out recent sexual contacts.	29
A.	Yes.	30
Q.	Did you know that.	31
A.	Yes, they will do that, they may do that now.	32
Q.	Isn't that contact tracing.	33
A.	That was - there is contact tracing which is done with	34
	one person, it is not a contact tracing when HIV was	35
	said to be sexually transmitted.	36
Q.	What I am saying to you is that that is contact tracing	37
	now done as a matter of course around this country.	38

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- A. There is but this is not what other studies are on 1
  - sexual transmission, the studies which claim 2
  - heterosexual transmission, are based on. None of the 3
  - respective studies in which sexual transmission is 4
  - studied have done contact tracing. The cross-section of  $\,$  5
  - studies could not have done contact tracing. 6
- Q. You gave evidence at p.114 not about studies, about 7
  - generally how one proves the disease is sexually 8
  - transmitted, and said this, starting at line 4, 'To 9
  - prove that disease is sexually transmitted you have 10
  - first of all to find the agent in genital secretions. 11
  - It has to be in both partners, the passive and the 12
  - active partner. As I said, it must be by sexually 13
  - transmitted. The evidence for a sexually transmitted 14
  - disease is usually obtained, or always is obtained by 15
  - contact tracing, that is, if a man or woman is found to 16
  - have a sexually transmitted disease then the doctor 17
  - tries to trace her sexual partners before she became 18
  - infected and sexual partners after she became infected 19
  - and this goes on as far back as they can. This is not 20
  - done for HIV '. That was your evidence, wasn't it. 21
- A. This has not been done in any of the studies in any of 22
  - the studies which claim truth for sexual transmission, 23
  - heterosexual transmission, and you can go through all 24
  - the studies which claim heterosexual transmission and 25

there is - never has been done. In fact, if you read -

	if you read the latest commentary everybody now seems to	27
	comment on HIV/AIDS, on AIDS Truth, where nobody can	28
	respond. If you see the commentary by Nancy Padian and	29
	Bear, she implies there that there are contact tracing.	30
	In fact she - I think 6 to 9, numbers 6 to 9 - and when	31
	you go and look there, there is nothing like that, what	32
	you have - there is some mathematical orders in	33
	population, it is not contact tracing, but the studies	34
	that concluded heterosexual transmission -	35
Q.	You mention Padian. She is someone else who has also	36
	written a response as a result of people doing what she	37
	describes as misusing the results of her studies.	38

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Α.	That is what I said, I know the thing, that is what	1
	everybody does now, they go and publish in AIDS Truth	2
	where you cannot respond. It is such a truth that seems	3
	to be beyond reach of any scientist. They have - they	4
	are the gods, they have the truth, and nobody else can	5
	have it. So you just publish there and everybody who	6
	reads the - all you have to do is reach conclusion,	7
	editor finds it - he said - that is he said to Joan	8
	Moore whose web site it is, 'I disagree with your title.	9
	I don't like your - the name of your web site. It	10
	implies that only you have the truth'. That is what	11
	they want to portray, that only they have the truth and	12
	nobody else have the truth.	13
Q.	Let us go back to what you told this court. You didn't	14

- Q. Let us go back to what you told this court. You didn't tell the court, you didn't talk about studies, no 15 studies on contact tracing, you put to this court that 16 the normal method for tracing sexually transmitted 17 diseases was contact tracing and this is not done for 18 HIV.
- A. This was not done for in this study say to prove 20 sexually heterosexual transmission. 21
- Q. You weren't talking about a study at that point in time 22 you were making a general statement to the court. 23
- A. Yes, and that was not that is what I implied. I 24 implied that was not done in the studies in which it is 25 claimed to have proven heterosexual transmission. 26

Q. I might turn to that study that you excised that quote	27
from, it is the epidemiology of immunodeficiency	28
syndrome amongst heterosexuals. I indicate it is a 1988	29
paper.	30
MS MCDONALD: I tender that.	31
EXHIBIT #P21 ARTICLE 'EPIDEMIOLOGY OF IMMUNODEFICIENCY	32
SYNDROME AMONGST HETEROSEXUALS' AUTHORS HARRY HAVERKOS AND	33
ROBERT EDELMAN, PUBLISHED 7/10/1988 EDITION OF THE JAMA	34
TENDERED BY MS MCDONALD. ADMITTED.	35
	36
MS MCDONALD: I think that is the Journal of American	37
Medicine Association.	38

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XXN		1
Q.	This is the article that you excised that quote from.	2
A.	Yes.	3
Q.	I will just take to you the relevant page, p.1927, the	4
	third to last page.	5
A.	Yes.	6
Q.	If you go into the middle column.	7
A.	Yes.	8
Q.	There is the word 'prevention' then, underneath that,	9
	the words 'sexual contact tracing', do you see that.	10
A.	Yes.	11
Q.	That is where you have extracted that passage that we	12
	see in slide number 6.	13
A.	Yes.	14
Q.	You just cut and paste it straight out of this paper.	15
A.	That is what he said.	16
Q.	In America in 1988 he is talking about, isn't he.	17
A.	I am not talking only in America, I am talking all the	18
	studies which claim heterosexual transmission.	19
Q.	Other than that little snippet, under 'sexual contact	20
	tracing', you didn't tell us anything else about this	21
	study in your PowerPoint, did you.	22
A.	No.	23
Q.	Let you just see what the study actually says there.	24
	Are you familiar with it.	25

A. I am familiar.

Q.	Starts off with the purpose of this article. The	2/
	purpose of this article is 'To inform health care	28
	professionals about the extent to which human	29
	immunodeficiency virus, HIV infection, and acquired	30
	immunodeficiency syndrome, AIDS, are spreading to the	31
	heterosexual population in this country', correct.	32
Α.	Yes.	33
Q.	That is what this paper is about.	34
A.	Yes, that is what it says.	35
Q.	I will take you to some details in a moment, but would	36
	agree that the authors of this report absolutely express	37
	the view that HIV can be heterosexualally transmitted.	38

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A.	But they don't give evidence for it, and the evidence I	1
	discuss on the papers which have data, scientific	2
	evidence, this study does not have any evidence.	3
Q.	You don't include anything about that aspect of this	4
	paper because there is no evidence about why it is -	5
A.	I don't have to say.	6
Q.	Could you let me finish please. Just let me finish.	7
A.	Yes, sorry.	8
Q.	Is it your evidence that the reason you have included	9
	none of the details of this paper in terms of the	10
	opinion of the authors, heterosexualally transmitted, is	11
	because they don't have the studies and the data there.	12
A.	Look, there are most probably about maybe more than	13
	100,000 articles where the authors say heterosexualally	14
	transmitted AIDS. Do I have to include all this	15
	article - I will have done nothing else but	16
	photocopying, to give them to you, all my life.	17
Q.	There is an article you chose to rely on to put before	18
	the court.	19
A.	I am - but this is an article which what is said in	20
	relation to what - how heterosexual - how sexually	21
	transmitted diseases are proven. I don't have to say	22
	every single article. As I say, if I say every single	23
	article - if I present to the court every single article	24
	where it is said heterosexualally transmitted AIDS, I	25
	will do nothing all my life but photocopying to give	26

	them to you.	27
Q.	Lets us go back to the part you did choose to include in	28
	your PowerPoint, the part in relation to sexual contact	29
	tracing.	30
A.	Yes.	31
Q.	You have just told us that the reason you didn't include	32
	any of the rest was because it didn't have the studies	33
	then.	34
A.	Yes.	35
Q.	There is no evidence to back up that there is no sexual	36
	contact tracing in this paper, is there.	37
Α.	Well, that is what he says.	38

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Q.	Well, why are you prepared to rely on what he says in	1
	that one paragraph without anything to back it up and	2
	not the rest of the article.	3
A.	I have backed it up there, I have backed it up then with	4
	all the evidence I have presented and I have put - I	5
	didn't omit any of the major studies. If I omitted	6
	anything it was by, inadvertently, but I did not omit	7
	any studies to my knowledge which prove heterosexual -	8
	evidence for heterosexual transmission, either in	9
	Europe, Africa or America or anywhere else.	10
HIS	HONOUR	11
Q.	I just take you to p.1924 of this article	12
A.	Yes.	13
Q.	You see the heading 'Heterosexual transmission in the	14
	United States'.	15
A.	Yes.	16
Q.	He says 'In 1984 and 1985 there were initial reports of	17
	AIDS transmitted from men to women amongst spouses of	18
	military personnel and haemophiliacs in the United	19
	States'.	20
A.	Yes.	21
Q.	'More recently transmission of HIV infection and AIDS	22
	from women to men has been reported.'	23
A.	Yes.	24
Q.	'Isolation of HIV from cervical secretions of women at	25
	risk for AIDS supports the epidemiological evidence for	26

	female to male transmission.' Now, in that paragraph he	27
	gives a number of references.	28
A.	Yes.	29
Q.	Commencing at 33 through to 42 and, if you go then to	30
	the references, 33 to 42, refers to various studies.	31
A.	Yes. Yes, 32.	32
Q.	33 is Redfield.	33
A.	Yes, I know where - I presented that study.	34
Q.	33 through to 42 are various studies.	35
A.	Yes, I am aware of all these studies.	36
Q.	You are aware of all of these studies.	37
Α.	Yes, I am aware of all these.	38

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۷.	bo you charrenge the authors of this article that those	_
	studies are evidence of what they claim to be the	2
	position in 1984 and 1985;	3
Α.	Yes, and it is not only me for challenging, it is Nancy	4
	Padian herself who challenged this article.	5
Q.	I am really only interested in your views.	6
Α.	Yes.	7
XXN		8
Q.	If one goes to the end of this paper to where it has a	9
	list of references, there is a very long list of source	10
	material references that the author has relied on to	11
	produce this paper. So when you say the evidence for	12
	what they are saying is not there, that is just not	13
	true, there are 90 different studies or references that	14
	form the basis of their opinion.	15
Α.	But they are not related to that heterosexual	16
	transmission.	17
Q.	The whole paper.	18
Α.	There is nothing - I am sorry, they are not, these	19
	papers are not giving data on heterosexual transmission.	20
	His Honour gave us - he was much quicker than both of us	21
	to find out which are the references which relate to	22
	this topic.	23
CON'	TINUED	24
		25

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Q.	Let's go to some of what the authors say in the	1
	references they rely on. On the first page there is a	2
	heading 'Heterosexual transmission in Africa and Haiti'.	3
	The author goes on to say 'Convincing evidence that	4
	heterosexual transmission could occur first came from	5
	epidemiologic studies of AIDS in Africa and Haiti.	6
	Heterosexual promiscuity and contacts with female	7
	prostitutes had been associated with HIV infection among	8
	Africans and Haitians'.	9
A.	Yes, yes, that's what it says.	10
Q.	He then goes on and breaks it down.	11
A.	Yes.	12
Q.	And littered throughout the following paragraphs in	13
	relation to Africa and then Haiti are studies that	14
	support the propositions the authors are putting	15
	forward.	16
A.	No, they are not, no, I'm sorry. Give me one reference	17
	where he says evidence of heterosexual transmission.	18
Q.	That is my point. Let's deal with the annexure under	19
	the 'Epidemiology of AIDS in Africa', so I'm on p.1923,	20
	the first column, second paragraph down.	21
A.	'Immediately'?	22
Q.	Yes, 'Immediately, African, American and European	23
	investigators collaborated to survey parts of Africa for	24
	further evidence of AIDS. Similar findings strongly	25
	suggested heterosexual transmission with HIV, namely,	26

	the nearly equal sex distribution and a lower mean age	27
	for female patients among subjects who are in their	28
	sexually most active years of age, were subsequently	29
	reported from Zambia and Rwanda. Acquired	30
	immunodeficiency syndrome in Africa epidemiologically	31
	resembled other sexually transmitted infections'. You	32
	agree that's what it says.	33
A.	Yes, I agree.	34
Q.	And if you look there we have got footnotes 14, 15 and	35
	16.	36
A.	Footnotes, yes, in the many references.	37
Q.	If we turn then to 14, 'Evidence for heterosexual	38

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	transmission and clinical manifestations of human	1
	immunodeficiency virus infection and related conditions	2
	in Lusaka, Zambia. Published in Lancet; it's a very	3
	reputable publication, isn't it.	4
A.	Yes, I agree, so is Jama.	5
Q.	15, 'Seroepidemiological studies of HTLV-III antibody	6
	prevalence among selected groups of heterosexual	7
	Africans'; do you see that, and HTLV-III is HIV;	8
	correct.	9
A.	Sorry?	10
Q.	HTLV-III.	11
A.	Yes.	12
Q.	The next one, 16, 'Prevalence of HTLV-III/LAV in	13
	household contacts of patients with confirmed AIDS and	14
	controls in Kinshasa, Zaire in JAMA'.	15
A.	Yes.	16
Q.	So there is one little paragraph and there we have three	17
	separate studies in reputable journals that support that	18
	proposition that have been relied on for that	19
	proposition.	20
A.	This time they are not even - I'm aware of all the	21
	studies. If you want I will send you photocopies of all	22
	these studies. These are the studies which I have read	23
	again and again. Maybe you won't be able to read them	24
	because I have so many other ones there, but in not one	25
	of these studies there is evidence for heterosexual	26

	transmission. In fact, these studies are not considered	21
	by anybody, definitely not by Padian, or any other	28
	research as proving heterosexual transmission of AIDS.	29
HIS	HONOUR	30
Q.	What do you say is the basic requirement necessary to	31
	prove heterosexual transmission.	32
Α.	What you have - all these studies did, let me say, they	33
	went there and they took some population of Africans and	34
	they tested them for HIV. They collected the HIV	35
	antibody. At that stage you go and test people and if	36
	you find their blood to have a p24 band reactive or 41	37
	band reactive - this all done by pens in Africa - and	38

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	they will say you are infected. By this standard, 30%	1
	of us here will test positive, would be HIV infected,	2
	will have heterosexually acquired HIV. So these studies	3
	are not considered as proof for heterosexual	4
	transmission.	5
XXN		6
Q.	What about in a situation in which, using the same	7
	country in Africa, using the same method of testing, so	8
	looking for the same proteins, the same bands, you see	9
	in a population a dramatic increase in HIV.	10
Α.	There that is not true. The first research by	11
	Montagnier and Gallo, they reported a very high HIV	12
	infection in Africans. In fact I think Gallo had	13
	about - may be confused here - Gallo or Montagnier, in	14
	one of their papers, even the controls had 12% been	15
	infected with HIV. So no, there is nowhere any evidence	16
	for an increase of HIV positive test in Africans.	17
Q.	I'm asking you to assume a hypothetical. Let's assume a	18
	hypothetical African country, we won't be specific about	19
	which one, and that testing was done in that country to	20
	reveal that there was about a 5% prevalence of positive	21
	HIV results - I'm making these figures up - and that	22
	using those same tests a year later that's increased to	23
	55% of the selection of the population tested, tested	24
	positive, the same community, the same country, the same	25
	testing for the same bands and proteins. Doesn't that	26

	indicate an increase in HIV or whatever it is that's	27
	causing these positive results.	28
A.	They never - you cannot have such paper and never has	29
	been one. Please give me one paper which has any	30
	scientific journal published where there has been such	31
	increase in positive test. In fact, in Uganda, which	32
	will have been the country more studied, they say we	33
	have a miracle now, we have a decrease by about 30% of	34
	the HIV and it is said that this is due to sexual	35
	education, but it is impossible to be due to sexual	36
	education your Honour because there is a study from	37
	Uganda where the population, they had about 16,000	38

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	people and they divided that and some of them were given	
	very, very intensive sex education, others were left -	2
	their education was not that intensive and others were	3
	left to their own. When it came after the end of the	4
	study, the others were tested for HIV and for other	5
	sexually transmitted agents. They found out a	6
	significant difference in sexually transmitted disease	7
	between the group which had intensive sex education and	8
	the group which did not, but there was no difference in	9
	regard to HIV. So if it was a miracle in Uganda, it	10
	cannot be because of sexual education.	11
Q.	Could it be because so many people have died of AIDS in	12
	Uganda that it wouldn't have increased.	13
A.	They couldn't have died. In Uganda the population	14
	increased. There is statistical - I cannot give you the	15
	exact data but the population in Uganda has	16
	significantly increased in the time of the AIDS era.	17
	There is a doctor, an MD, in Switzerland who is	18
	following this very closely and he came with all the	19
	data and he has it at his fingertips. I can ask and if	20
	the court will like that data I will be able to present	21
	it. There is no evidence that Uganda population has	22
	decreased, to the contrary.	23
Q.	Let's go back to this article. Still on p.1923, we have	24
	just gone through that paragraph commencing with the	25
	word 'Immediately' and finishing with the word	26

	'Infections'. You then go on to describe the situation	27
	in Nairobi and said this: 'Female prostitutes emerged as	28
	an important reservoir of HIV in central Africa' and	29
	they cite four studies in support of that proposition.	30
Α.	Yes.	31
Q.	'In a retrospective serosurvey, only 4% of prostitutes	32
	in Nairobi, Kenya, were HIV positive in 1980 through	33
	'81, while 59% of prostitutes tested in 1985 through	34
	1986 were seropositive' and again they have cited a	35
	study in support of that. Do you agree that's what it	36
	says.	37
A.	Sorry?	38

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Q.	Do you agree with that.	1
Α.	Yes.	2
Q.	So there is an example of a dramatic increase in HIV	3
	positive results.	4
Α.	Yes. How do they know the prostitutes - that all these	5
	prostitutes were not drug addicts? A scientific paper	6
	should exclude - if it's made to present sexual	7
	transmission it should exclude every other means of	8
	becoming positive including ever having TB or coming in	9
	contact with a TB person or a leprosy person or have	10
	leprosy or come in contact with any other person who is	11
	microbacteria infection. All of these things have to be	12
	excluded and the population should be prospectively	13
	followed up, not reduced to paper.	14
Q.	Are you suggesting that there might be an alternate	15
	explanation that 55% or more of prostitutes in 1981 to	16
	1985 become drug addicts.	17
A.	It is possible.	18
HIS	HONOUR	19
Q.	Read the next sentence. It says 'The seroprevalence	20
	among prostitutes increased with the number of sexual	21
	contacts and lower socioeconomic status; a higher number	22
	of partners was inversely related to socioeconomic	23
	status'. Does that suggest anything to you.	24
Α.	It would suggest to me that these people are poor people	25
	and they will have all kind of infectious diseases,	26

	including microbacteria infection which leads to a	27
	positive test.	28
Q.	It says that the seroprevalence amongst prostitutes	29
	increased with the number of sexual contacts.	30
Α.	Then we have anal intercourse. Anal intercourse is not	31
	limited to gay men.	32
XXN		33
Q.	So suddenly all these prostitutes, 4% before have HIV	34
	and 59, which everyone did know about it, started	35
	engaging in anal intercourse.	36
Α.	They have a mixture. Certainly these studies does not	37
	put heterosexual transmission and it is not me who says	38

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	that this study is not, does no prove heterosexual	1
	transmission. It is the people. Let's not repeat this	2
	again and again.	3
HIS	HONOUR	4
Q.	Would you agree that that is some evidence which might	5
	point to the fact.	6
A.	It might point but it's not proof.	7
Q.	It's a question of what you require as proof, isn't it.	8
	What you might require as proof others may not require	9
	that standard.	10
A.	No, I think all the people who do heterosexual, who	11
	study heterosexual transmission of HIV have the same	12
	criteria.	13
Q.	Certainly Harry Haverkos and Robert Edelman don't appear	14
	to have, do they.	15
A.	They do not say it is proof.	16
Q.	They used it as part of their material.	17
A.	No, they stated a fact but they don't say that this is -	18
Q.	If they are stating it as a fact you would assume that	19
	they think it's a relevant fact in preparation of their	20
	paper, wouldn't you. They wouldn't just state a fact if	21
	they didn't think it had some relevance.	22
A.	They may think that. I'm not excluding that, they may	23
	think that.	24
Q.	Do you agree that they are certainly people with high	25

qualifications.

A.	Yes, I agree. I correspond with Haverkos. In fact, he	27
	send us all his papers, he keeps us always informed.	28
XXN		29
Q.	Do you accept that in Kenya there is next to none	30
	intravenous drug use because of poverty and cultural	31
	issues, they just can't afford it.	32
A.	I do not think there is any country anywhere where there	33
	is no drug.	34
Q.	Next to none, very, very little.	35
A.	No, I don't - I don't have evidence of that and I doubt	36
	that anyone has that evidence.	37
Q.	Do you have any evidence one way or the other.	38

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Α.	No, not, I haven't got any evidence. I have seen but I	Т
	doubt that there is any evidence that in Kenya there is	2
	no drug addiction anywhere or next to none; will be very	3
	surprised.	4
Q.	I didn't put that to you. I suggested to you that there	5
	is very little intravenous drug use in Kenya.	6
A.	I said I don't know, but it's not only intravenous drug	7
	which lead to a positive test. In fact, there was a	8
	study done where prostitutes who switched from	9
	intravenous to oral drugs and vice versa and it was	10
	found that the prostitutes, when there was a higher	11
	grade in the prostitutes when they are taking oral drugs	12
	than it was when they are taking intravenous drugs.	13
Q.	Which study is this.	14
A.	It's a study - I will give you the study, the author is	15
	just escaping me at the moment.	16
Q.	The authors of this particular report then go on to	17
	discuss the epidemiology of AIDS in Haiti and say that	18
	the epidemiologic studies in relation to that country	19
	also suggest increasing heterosexual spread of AIDS.	20
A.	Yes.	21
Q.	And in the following passage relating to Haiti again	22
	they list numerous studies as support for that	23
	proposition.	24
A.	Support, but in science we don't go, we are suggesting	25
	you go with proof and all the studies which have been	26

	done following this, including the Padian study, show	27
	that there is no heterosexual transmission of HIV. Let	28
	me - there is a group of researchers from America,	29
	France and Germany who have done intensive studies on	30
	heterosexual transmission of HIV.	31
CON	TINUED	32
		33
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		35
		36
		37
		38

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RES	UMING 3.55 P.M.	1
Q.	Do you still have P21 in front of you.	2
A.	May I read this paper?	3
Q.	I would like to ask you some questions.	4
HIS	HONOUR	5
Q.	Ms Papadopulos-Eleopulos just allow Ms McDonald to ask	6
	you the questions, if you need to read further from the	7
	quote you can do so later. Just listen to the question.	8
XXN		9
Q.	Do you have P21 in front of you still.	10
A.	Thank you.	11
Q.	P.1923, we have gone to what has been happening in	12
	Haiti.	13
A.	Yes.	14
Q.	If I take you to the middle column of the page,	15
	paragraph commencing with the words 'In another study'.	16
A.	In the middle part of it?	17
Q.	Towards the top, middle column, second paragraph down.	18
A.	Middle column?	19
Q.	It commences 'In another study Pape et al reported'.	20
HIS	HONOUR	21
Q.	Have you got page 1923.	22
A.	Yes, 1923. The middle -	23
Q.	The middle paragraph, about here (INDICATES).	24
A.	In Haiti.	25
Q.	'In another study' it starts.	26

A.	Yes, 'In another study', the second paragraph?	27
Q.	Yes.	28
XXN		29
Q.	'In another study Pape et al reported that 60% of female	30
	spouses of Haitian men with AIDS and 63% of male spouses	31
	of Haitian women with AIDS were HIV positive, suggesting	32
	that female-to-male and male-to-female transmission were	33
	approximately equal. No differences in the types of	34
	sexual activities could be found between seropositive	35
	and seronegative spouses. Haitian men and women with	36
	AIDS were more likely than persons without AIDS to	37
	report having a large number of heterosexual partners'.	38

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	Do you agree that's what it says there.	1
A.	Yes.	2
Q.	The suggestion is there, if you accept what is written,	3
	that this isn't about some having anal intercourse and	4
	others not, I mean it says there was no difference in	5
	the types of sexual activities between these two groups.	6
A.	Yes, this study - let me see which is the study. The	7
	pop study yes, this study was severely criticised	8
	because a finding that equal number of men and woman	9
	test positive, it was interpreted that there is	10
	heterosexual transmission, that these people got	11
	infected through heterosexual transmission. Again, this	12
	study not me, but no HIV expert at present will consider	13
	right.	14
HIS	HONOUR	15
Q.	Well, Dr Edelman and Dr Haverkos were prepared to rely	16
	on it, were they not.	17
A.	I said previously, this was written in 1988.	18
Q.	Are Dr Haverkos and Dr Edelman still practising, do you	19
	know.	20
A.	They are still, and I think Haverkos is going to retire	21
	soon or - he may have left. I think he may have left in	22
	the middle.	23
Q.	To your knowledge, in any of the papers that they have	24
	published since 1988, have they indicated that they	25
	would no longer rely upon these studies or that this	26

	paper is inaccurate in any way.	27
Α.	As far as I know they don't discuss any more, that is	28
	more now on drugs, and in fact he is - the human	29
	papilloma virus paper is including human papilloma	30
	virus.	31
Q.	The question I'm asking, do you know of any paper or any	32
	statement made by Mr Edelman or Haverkos that suggest	33
	this paper, and the data upon which it relies, is no	34
	longer available.	35
A.	Yes, is no longer valid, yes. May I read from this	36
	paper which was published in 2003?	37
Q.	Who is it by.	38

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Q.	No, that's not the question. I'm asking whether	2
	Dr Haverkos or Dr Edelman, was there anything suggested	3
	from them that their paper is no longer valid.	4
A.	I don't know if they publish any paper to retract this	5
	statement. No, sorry, I didn't understand the question.	6
XXN		7
Q.	I'll try and speed up through this and move on to other	8
	things. If we move down the column we see 'Factors	9
	Modulating Heterosexual Transmission of HIV in Africa	10
	and Haiti'. They talk about condom use and that the	11
	epidemiology of condom use provides further evidence	12
	that HIV transmission occurs through heterosexual	13
	intercourse. That is that if you look at where condoms	14
	are used and where they're not used you find support for	15
	that proposition. That is that HIV is heterosexually	16
	transmitted. What do you say to that.	17
A.	This is not a study, as I said, these studies are not	18
	any more considered study to be valid for heterosexual	19
	or sexual transmission of HIV.	20
Q.	I might move up ahead to a further point. In the third	21
	column.	22

A. All this is a cross-section, and Professor Caldo will

agree that cross-sectional studies do not - you cannot

proof, you may suggest but it doesn't prove. You have

get information from cross-sectional study, or can't get

A. By researcher from the US, Germany and America.

1

23

24

25

	to have prospective studies to come to conclusion in	27
	regard to sexual transmission of HIV.	28
Q.	Or you can look at a whole load of studies and look at	29
	what the total picture tells you.	30
A.	The total picture tell me that there is no heterosexual	31
	transmission. It is not me who tells, it is the HIV	32
	experts, their evidence who proves that.	33
Q.	Let's move on to the third column. About halfway down	34
	there is a paragraph headed with the words 'It has also	35
	been suggested'.	36
A.	Yes.	37
Q.	It goes on to say 'It has also been suggested that HIV	38

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	may be acquired more readily from circumcised women or	1
	readily by uncircumcised men.' The first thing, do you	2
	agree with that proposition.	3
Α.	Firstly you have to prove it is heterosexual	4
	transmission. Before you can say condom and	5
	circumcision help you have to have proof of heterosexual	6
	transmission, such proof does not exist. So you can't	7
	talk about circumcision.	8
Q.	Let's talk about circumcision for a moment, you are	9
	aware of a number of studies occurring in Africa in	10
	recent times comparing the rates of HIV in men who are	11

A. No, no, these studies do not prove - you have to have, 13 as I said, proof of heterosexual transmission and the 14 studies, the best studies - the best studies from Africa 15 do not prove heterosexual transmission. We have study, 16 we have analysed this and published a letter, as I said 17 in my evidence, in Lancet. Which ends up that in Africa 18 there is no more heterosexual transmission than anywhere 19 else. Including Europe/Australia. 20

12

circumcised as compared to men who are not.

- Q. I'll go back to my question. Are you aware that in

  recent times there have been a number of studies,

  ongoing studies right up until in fact just weeks ago,

  comparing the prevalence of HIV in men who are

  24

  circumcised as compared to those who are not.

  25
- A. Yes, I'm aware that now there are advocate circumcision, 26

	based, there is no scientific evidence.	28
HIS	HONOUR	29
Q.	No Mrs Papadopulos-Eleopulos, we'll get through your	30
	evidence a lot faster if you answer the question. The	31
	question was: are you aware there are a number of	32
	studies in respect of HIV infection in circumcised and	33
	uncircumcised men. Now there is a short answer to that,	34
	'Yes, I am aware of those studies; no, I'm not aware of	35
	those studies'. What conclusions one may draw from	36
	those studies is not part of the question. The question	37
	is simply to ask you, are you aware of them.	38

but I don't know on what scientific evidence this is 27

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A.	Yes, I am.	1
Q.	So if you can keep your answers just to the question,	2
	we'll get through your evidence faster and perhaps it	3
	will be easier, because every time you give a long	4
	answer which is not responsive to the question it's	5
	difficult to get back to the line of questioning. All	6
	right. Do you understand.	7
A.	If I said 'yes' then it means that I agree with that.	8
Q.	No it doesn't, it means you are aware of it or you're	9
	not aware of it; that's all it means.	10
A.	I'm aware of it.	11
XXN		12
Q.	Are you aware also that those studies have been stopped	13
	recently because there was such a clear pattern showing	14
	that men who were circumcised were less likely to get	15
	HIV that wanted to give all of the other men in the	16
	control group who were uncircumcised an opportunity to	17
	become circumcised.	18
A.	I'm aware that's what they are saying.	19
Q.	That's what is happening now, those studies have been	20
	stopped for that reason.	21
A.	Yes, many studies were stopped because they thought	22
	prematurely. In AIDS many, many studies are stopped and	23
	they realise that they stopped it prematurely, so is no	24
	different.	25
Q.	And back in 1988, when this article was written and	26

	published, there was already some evidence that	27
	uncircumcised men would more readily contract HIV.	28
A.	That's what it says.	29
Q.	You disagree with that.	30
A.	That's what it says here.	31
HIS	HONOUR	32
Q.	The question was do you agree or disagree.	33
A.	I disagree with it.	34
XXN		35
Q.	I might go to the very end of this article. Can I take	36
	you to p.1927. There is a column headed 'Extent and	37
	Rate of Heterosexual Transmission of HIV.	38

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Α.	1927?	1
Q.	In the last column, third column over.	2
Α.	Yes.	3
Q.	The authors say 'It is not clear how far or how fast HIV	4
	infection will spread into the heterosexual population	5
	of the United States and the rest of the world; however,	6
	there can be no doubt that heterosexual transmission	7
	occurs and that, in a favourable environment, it can be	8
	an important factor in the dissemination of HIV	9
	injection and AIDS. Although AIDS is not "exploding"	10
	into the heterosexual population relative to other risk	11
	groups, the increase in the number of heterosexual cases	12
	is proportional to increases in other risk groups.	13
	These increases are resulting in a doubling of	14
	heterosexual cases every 14 to 16 months. From our	15
	perspective, AIDS is preponderantly a sexually	16
	transmitted disease and can be transmitted from	17
	man-to-man, man-to-woman, and woman-to-man. Recently,	18
	HIV transmission from woman-to-woman has been reported.'	19
	Firstly, do you agree with what's written there.	20
A.	If it's written I have no choice but to agree.	21
Q.	Do you agree with any part of what the authors have	22
	written there.	23
A.	I don't agree that this is happening.	24
Q.	And that they've even noted that there had been	25
	transmission from woman-to-woman.	26

A.	No.	27
Q.	The authors have noted that though, you might not agree	28
	with it, but the authors -	29
A.	I know that. I mean, you have read it, I accept what is	30
	written there. You have read it, yes, it is written	31
	there.	32
Q.	If we look at the references for that proposition, they	33
	have relied on a publication in Lancet, 87.	34
A.	Yes.	35
Q.	And two publications in fact, another one from Ann	36
	Intern Medicine Lancet.	37
A.	Yes.	38

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Q.	Lancet is a reputable journal.	
A.	Very reputable, Intern Medicine is very reputable; they	2
	are reputable journals, yes.	3
Q.	I just take you to other references in that list, 41 and	4
	42.	5
A.	In, sorry?	6
Q.	In the list of reference, 41 and 42.	7
A.	Yes.	8
Q.	We see the authors of this publication have relied on a	٥
	study entitled 'Isolation of HTLV-III/LAV from cervical	10
	secretions of women at risk for AIDS.' Published in	11
	Lancet.	12
A.	Yes.	13
Q.	There we go, there seems to be another reference to a	14
	person who is purported to isolate HIV.	15
A.	They found out a positive - they use p24 and they found	16
	out people with antibodies and they found a reaction	17
	with p24. Now pregnant woman would have a p24, a	18
	positive p24. Normal pregnant woman who have no risk,	19
	so can't say that they have HIV infection or HIV in	20
	their secretion. What they did is not proof of HIV	21
	relation. That is not proving for HIV detention.	22
Q.	The second one, 42.	23
A.	They're the same.	24
Q.	'Isolation of AIDS associated retrovirus from genital	25

secretions of women with antibodies to the virus'.

A.	Yes.	27
Q.	I want to turn to deal with a couple of other topics.	28
A.	May I your Honour, with your permission, read all the	29
	latest report? This is a reputable group of people.	30
HIS	HONOUR	31
Q.	Have you got the article from which you want to read or	32
	is it just a slide.	33
A.	I will present the article. I'll give the article.	34
Q.	Just indicate what -	35
A.	Thank you very much. As I say, the title, the article	36
	is called 'Mounting anomalies in epidemiology of HIV in	37
	Africa: cry the beloved paradigm'. And I quote: 'Though	38

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	heterosexual intercourse has been virtually the sole	1
	explanation offered for the AIDS epidemic in sub-Saharan	2
	Africa, to our knowledge in no other part of the world	3
	has penile-vaginal exposure; (as opposed to	4
	"heterosexual sex") been demonstrated to initiate or	5
	sustain rapid HIV propagation. HIV is not transmitted	6
	by sex -'	7
HIS	HONOUR	8
Q.	HIV is not transmitted by sex.	9
A.	HIV is not transmitted by 'sex'. Sex in inverted	10
	commas.	11
CON	TINUED	12
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	'But only by specific risk practices. Dispassionate	1
	assessment of our conclusions admittedly depend on	2
	willing suspicion of this belief since the current	3
	paradigm is deeply embodied'.	4
HIS	HONOUR	5
Q.	Where is that from.	6
A.	I will give the author, the journalist, Journal of STD	7
	in AIDS 2003.	8
XXN		9
Q.	You have given some evidence about the insert from the	10
	ELISA test. By that, I mean the instructions, if you	11
	like, that come with the ELISA test. Looking at Exhibit	12
	A7, that is the insert that was provided by your	13
	colleague, Dr Turner, as an insert as part of the	14
	packaging for the ELISA test.	15
A.	Yes.	16
Q.	We see a date on there.	17
A.	1995. Yes, there is a date there.	18
Q.	Is it 1995.	19
A.	Yes.	20
Q.	That has been superseded since then -	21
A.	Yes.	22
Q.	- by a new insert, if you like.	23
A.	A new test kit, do you mean?	24
Q.	New accompanying documents. What I am suggesting is it	25
	is now a 2001 -	26

Α.	Yes.	27
Q.	- instruction booklet -	28
A.	Yes.	29
Q.	That currently accompanies the kit.	30
A.	Yes.	31
Q.	Looking at this document, that is the one which is	32
	currently used as opposed to the one from 1995.	33
A.	I don't know. Most probably, yes. It is a more recent	34
	one. This will be the one which is used.	35
MR	BORICK: It would certainly help me if my friend	36
	could indicate the purpose of this document. Is it	37
	suggested there is a change to the instructions from A7?	38

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MS	MCDONALD: I haven't sat and compared the two. It	1
	has been put before the court as the most current and I	2
	was only taking the witness to the most current passage	3
	in the most current version.	4
EXH	IBIT #P22 DOCUMENT ENTITLED 'HIV REFERENCE 3D41-22 D3,	5
D4,	A0, 36-6381/R3 PRODUCED BY ABBOT AXSYM SYSTEM DATED	6
05/	2003 TENDERED BY MS MCDONALD. ADMITTED.	7
		8
XXN		9
Q.	Can I take you to the second to last page of the	10
	photocopy and there should be a heading on that page	11
	'Sensitivity and specificity'.	12
A.	Where? What page?	13
Q.	On the second to last page.	14
A.	Yes.	15
Q.	Do you see that.	16
A.	Yes.	17
Q.	You have already told the court or drawn to the court's	18
	attention -	19
A.	'Sensitivity and specificity', yes.	20
Q.	And there is an introductory sentence 'At present, there	21
	is no recognised standard for establishing the presence	22
	or absence of antibodies to HIV 1 and HIV 2 in human	23
	blood'.	24
A.	Yes.	25
Q.	It goes on to say though, doesn't it: 'Specificity is	26

	based on testing of random blood donors and hospitalised	27
	patient populations'.	28
Α.	Yes.	29
Q.	'Serum and plasma specimens'.	30
Α.	Yes, so the specificity is determined -	31
Q.	I have taken you there to that sentence commencing	32
	'Specificity'. It goes on to say: 'Sensitivity for	33
	HIV 1 (including HIV 1 group 0) and HIV 2 antibodies is	34
	expressed in terms of detection rate using confirmatory	35
	assay results eg (Western blot) as a basis for	36
	comparison'. Is that what it says in the document you	37
	have.	38

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- A. Yes. 1
- Q. It then indicates that all specimens in this study were 2 tested and the test results revealed the following: '1,
  - specificity based on zero prevalence of antibodies to 4
  - HIV 1 and/or HIV 2 in random blood donors' 6,340 were 5
  - tested 'is estimated to be 99.94% for the AXSYM HIV 6
  - 1/2 gO assay'. Do you see that. 7
- A. It says that. 8
- Q. Doesn't that mean it is 99.94% specific for those particular antibodies. 10
- A. You read the first sentence which tells you that this is 11 not truth. You don't use blood donors to determine the 12 sensitivity of - you don't use blood donors as a gold 13 standard to determining the specificity of an antibody 14 test no matter what test is that. The gold -15
- Q. Doesn't it -16
- A. Sorry, but I have to respond to this. Blood donors are 17 very healthy people and they have very low antibodies to 18 anything, to anything in their blood. So blood donors 19 would test negative to - no matter what antibody test 20 you are using, but to determine the specificity, the 21 only way to determine the specificity of an HIV antibody 22 test is to use HIV as a gold standard. The antibodies 23 are done to prove HIV infection and you have to compare 24 the 6,000 people who are there with the presence or 25 absence of HIV. The specificity of the antibody test is

determined, your Honour, by taking people, including	27
blood donors, people who are healthy, people who have	28
diseases, any type of disease, and people who have	29
infectious diseases and then you do, on the one hand in	30
this group, you do an antibody test. The whole thing is	31
done blindly, and on the other hand you do HIV	32
qualification, that is to prove, and then you compare	33
the two groups. The whole thing is done blindly, then	34
unblind. After you do the test, unblind it and then you	35
look. There is correspondence between the positive	36
antibody test and the existence of HIV. If every single	37
person - if it is 99% - if you had 100 people you tested	38

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with the antibodies and you had 100 people who you did	1
HIV qualification and you find out only one of these	2
people is HIV positive, you will say - sorry, I should	3
say if no person who has no HIV tests positive, then you	4
will say that the test is 100% specific, but if there	5
are people who have not got HIV and they test positive,	6
then you cannot say that the test is 99 or 100%	7
specific. The gold standard for the antibody test is	8
what we looking for, what the test is telling you. If	9
the test is done to prove HIV infection, you have to	10
compare it with HIV. You don't use gold standard with	11
blood donors, healthy people. This is not how	12
specificity is determined.	13

CONTINUED

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- Q. Do you accept that this packaging indicates that the 1 test is 99.94% specific for the presence of HIV. 2
- A. It is specified, if we use blood donors as the gold
  standard and that is what the problem is, they're using
  blood donors that are gold standard. You cannot use
  blood donors as a gold standard. This, I'm sure
  6
  Professor McDonald will say that. This cannot be used
  as the gold standard.
- Q. Let's go to the next paragraph and look at some 9
  unhealthy people. 'Do the manufacturers then report 10
  that specificity, based on zero prevalence of antibodies 11
  to HIV1 and/or 2 in a hospitalised population -' lots of 12
  sick people I suggest '- 1,670 tested is also estimated 13
  to be 99.94%'. Isn't that what it suggests or says. 14
- A. How they compare it, it depends. With gold standard

  used as blood donors, you cannot use blood donors that

  are gold standard. This is a standard procedure. If

  you test for HIV, you have to compare with HIV, full

  stop.

  19
- Q. Haven't we got then a group of blood donors, on your 20 assumption, who are healthy, a group of people in 21 hospital, presumably unhealthy, and with both of them it 22 is reported that this test is 99.94% specific for HIV. 23
- A. No, there is another test. There is a test this is

  the manufacture's claim but there are studies which are

  conducted in hospital patients and a very high percent

25

26

	of people - and Dr Turner presented that evidence, I	27
	think - a very high percent of people test positive. In	28
	this study, the St Louis study, the authors went to	29
	great lengths to exclude any person who had even the	30
	most remote possibility of being infected with HIV and	31
	this is a scientific study.	32
2.	Doesn't para.3 read 'HIV antibody detection rate, in a	33
	limited population of 581 HIV1 antibody confirmed	34
	seropositive individuals is 100%. This rate includes	35
	227 clinically diagnosed patients from different disease	36
	stages of HIV infections'. 100%.	37
Α.	This is not specificity. It has nothing to do with	38

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	specificity.	1
Q.	Does it have anything to do with sensitivity.	2
Α.	Yes, but we're talking about specificity, what is	3
	important is specificity.	4
Q.	100% detection rate.	5
Α.	Yes. What - what do you mean by 100%?	6
Q.	Para.4 'HIV2 antibody detection rate in a limited	7
	population of 304 -'	8
A.	Could you please say para.3 again?	9
Q.	We have just been through that, is there something you	10
	want to add to your answer.	11
A.	Can we please go to para.3?	12
Q.	Yes. Is there something you want to add to your answer	13
	about para.3.	14
A.	How are they confirmed? How are these people confirmed	15
	to be HIV positive? What test do they use to confirm	16
	that they were HIV positive? The only way to confirm	17
	that they were HIV positive is to have HIV	18
	qualifications and nobody has used. I'm sorry I am	19
	repeating this - these are the retrovirologists who say	20
	'We have not got a gold standard for the HIV antibody	21
	test and the gold standard in HIV qualification never	22
	has been done', so you cannot talk about specificity of	23
	the HIV antibodies. You have got to have HIV. That is	24
	one of the main ingredients, you have to have HIV. If	25
	you don't have HIV qualification, you cannot have HIV	26

	proteins, you cannot have HIV nucleic acid tests, that	27
	is PCR tests. You have to have qualifications.	28
HIS	5 HONOUR	29
Q.	We're back to your original proposition, aren't we.	30
A.	Exactly, but here it is stated that the antibody tests	31
	are 99% specific, this antibody test, and 100%	32
	sensitive. You cannot state that.	33
Q.	You say that that is a completely misleading document.	34
A.	It is.	35
MR	BORICK: I am confused between paras.3 and 4.	36
MS	MCDONALD: I will let my friend have a look at my	37
	copy.	38

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HIS	HONOUR	1
Q.	We're dealing with all of that document, aren't we.	2
	When you say it is misleading, it is misleading both as	3
	to specificity and sensitivity.	4
A.	Yes.	5
Q.	It comes back to your original proposition.	6
A.	Yes.	7
XXN		8
Q.	But you're prepared to rely on the first couple of lines	9
	of that document, though, in your evidence.	10
A.	Sorry?	11
Q.	You're prepared to rely on the first paragraph of that	12
	document in your evidence.	13
Α.	No, I say you cannot use the blood donors. I said you	14
	cannot use them.	15
Q.	Are you aware that these tests - and by that I'm	16
	referring to both the ELISA and the Western blot - are	17
	approved by the TGA.	18
MR I	BORICK: I'm sorry to interrupt but I want to take	19
	an instruction on the previous question.	20
HIS	HONOUR: Yes, we will wait while Mr Borick gets	21
	his instruction.	22
MR I	BORICK: The witness went back to para.3, 'HIV	23
	antibody detection rate in a limited population of 581,	24
	HIV1 antibody confirmed seropositive individuals as	25
	100%'. What it doesn't tell us is how -	26

OBC	JECTION: MS MCDONALD OBJECTS	27
MS	MCDONALD: I object to my learned friend giving	28
	evidence from the bar table.	29
MR	BORICK: One moment, I have a query. How does my	30
	friend say that it is proved from this document that the	31
	581 HIV antibody confirmed seropositive individuals is	32
	done? How do they prove that?	33
HIS	S HONOUR: That is the very proposition that the	34
	witness answered. My understanding was that she says	35
	her evidence is that that statement is presumptive of	36
	the fact that there is a virus called HIV and she	37
	doesn't accept that that's the position.	38

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Α.	I want to say more here. I'm saying even if we assume	
	that there is a virus HIV, in order to claim in this	2
	test, 99% specific and 100% sensitive, you have to	3
	compare the test with the actual virus, not to compare	4
	the specificity with blood donors and the sensitivity -	Ę
	we don't know what is what. What they have done, I	6
	assume here, is that they had some blood which they	7
	tested positive and how do they know that this blood is	8
	positive? What test did they use to prove that this	9
	test is positive?	10
MR 1	BORICK: That is the very question that I wanted	11
	to get clarified.	12
HIS	HONOUR: The witness has made the point.	13
Α.	They didn't tell you here what test they used to confirm	14
	that the blood was positive to start with and then they	15
	repeated it with ELISA and they found it in all the	16
	tests which all the blood was positive - it was positive	17
	with ELISA. They don't even tell us what test they use,	18
	so this is meaningless. You can't say - you cannot	19
	determine the specificity and sensitivity in this way.	20
XXN		21
Q.	Are you aware that these tests have also been approved	22
	by the Therapeutic Goods Administration.	23
Α.	If they are used in clinical productions, they have to	24
	be approved, yes.	25

Q. As you have already indicated in your evidence, you

26

	accept that for that approval to occur, the	27
	manufacturers' claims about the effectiveness of the	28
	tests are examined and scrutinised.	29
A.	I assume so.	30
Q.	Moving on to another topic and it relates to the genetic	31
	profile of the HIV virus. When I was asking you some	32
	questions this morning about the methods of testing that	33
	are used in this State and I suggested to you that in	34
	this State the sequence of each individuals' virus is	35
	established and put on a database, you said, I think,	36
	that you doubted that because it is a very expensive	37
	exercise.	38

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A.	No, I said - what I understood by profile, it was to	1
	compare it with somebody else. If you said that the	2
	sequences of some small part of HIV, or what is called	3
	HIV DNA, then that may be done.	4
Q.	Looking at this document, which was Exhibit P2 from the	5
	original trial, open it up so you can get a general	6
	overview of what it is.	7
A.	Yes.	8
Q.	There's been evidence that that document is a print-out	9
	of the genetic profile of every person in South	10
	Australia who is on the HIV database.	11
A.	Yes, it is the profile but you don't give here the	12
	sequence. They made the sequence, they generate this,	13
	yes, I agree with that.	14
Q.	Do you accept now, that in South Australia they don't	15
	only look at the ELISA and Western blot and nucleic acid	16
	test, but they do a sequence on each individual's	17
	genetic profile of the virus.	18
Α.	No, you cannot say they do the genetic profile of the	19
	virus. Let's assume that you have the HIV RNA or DNA	20
	and this is a long string. As you said yesterday, there	21
	is no agreement between the HIV expert as to how many	22
	genes the HIV genome has. I say very few, usually it is	23
	between 8 and 10. They come and go. Let's assume that	24

there's a string of RNA or DNA, which is HIV. When they 25

do the nucleic acid test, PCR, they don't do - they 26

	don't take the whole genome, they take a small part of	27
	what is said to be HIV. They are called primers and	28
	they amplify only a very small part - not even one gene	29
	but a very small - of what is called a gene - but a very	30
	small part of HIV of that string of RNA DNA which is	31
	called HIV RNA or DNA. A very small part.	32
COI	NTINUED	33
		34
		35
		36
		37
		38

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But that small part does not tell you that, first of	1
all, the whole genome is in that person because they	2
take the DNA from the cells of the patient and then they	3
look in that DNA of the patient if there are any	4
similarities between what is called HIV and in the cell.	5
So they don't, they take only a very, very small part.	6
So just my amplifying this small part you don't prove	7
that the whole genome is there, in fact maybe not even	8
one whole gene is there, maybe you just have a look at	9
only that part and, in fact, there is no proof that	10
nobody has done a test, a study to prove that the PCR	11
amplifies what you're looking for. It may be something	12
totally different. So this does not give you a profile	13
of the HIV genome, even if we assume that there is such	14
a thing of an AIDS patient and this is, again, let me	15
repeat, as I should, this is not only what I say and	16
this is accepted by a court in London on the basis of an	17
HIV expert. In fact, it is a pity that you don't have	18
the paper, if you need it I will supply it. There was a	19
paper published by a number of researchers from London	20
where they are saying that you cannot use DNA profiles	21
to prove epidemiologic studies in particular	22
I would ask you to produce that paper.	23
Sorry, I will produce the paper, definitely, and if you	24
want I will produce the paper. They say you cannot use	25
it. They give several reasons and if you do that you	26

Q.

Α.

	must do the whole HIV genome. Because, say, you have	2
	one person and you do the HIV genome and you do another,	28
	and you try to compare them, but you're comparing a	29
	very, very small part. It is like just taking a finger	30
	and a nose from one child and take a finger and a nose	31
	from another child, for example, and comparing them and	32
	say these children are twins. You have to have the	33
	whole person. This is not happening in HIV.	34
Q.	I want you to assume a few things for a moment.	35
Α.	Assume?	36
Q.	The first is this: the accused was diagnosed as being	37
	HIV positive via the ELISA test and the Western blot.	38

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	The woman with whom he had been in a sexual relationship	1
	for some time, some extended period of time, they lived	2
	together, also was determined to be HIV positive by	3
	virtue of the Western blot and the ELISA test. Both of	4
	them had their nucleic acid examined and both of them	5
	had the genetic profile, as much of it as we are seeing	6
	in that chart that has been produced to you,	7
	established.	8
A.	I have to have the genetic profile. I have to see -	9
HIS	HONOUR	10
Q.	Just listen.	11
XXN		12
Q.	Do you have all of that.	13
HIS	HONOUR	14
Q.	She is asking you to assume that.	15
XXN		16
Q.	Assume those factors, and assume that out of all of the	17
	people in South Australia on that database, the profile	18
	most closely linked to the accused is that of the woman	19
	with whom he has been in a sexual relationship for an	20
	extended period of time.	21
OBJ	ECTION: MR BORICK OBJECTS	22
MR 1	BORICK: I don't think it's possible to make the	23
	last assumption, that is the issue. There is a document	24
	there which the prosecution say was a profile. The	25
	witness is saying it's not a profile of anything.	26

HIS HONOUR:	All that the question asks the witness to	27
do is to ass	ume a whole lot of factors.	28
MR BORICK:	I appreciate that, but the last	29
submission ca	an't be made. My friend should reframe the	30
question and	say 'Assume there is a document held by the	31
South Austra	lian Forensic Science Department which has a	a 32
series of li	nes on it which demonstrates a profile',	33
something alo	ong those lines, but not asking her to	34
assume a gene	etic profile exists when the witness is	35
saying it do	es not, as I understand the evidence.	36
MS MCDONALD:	Your Honour has heard the evidence of	37
Dr Higgins.	In my submission, it's quite a proper	38

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	question. If at the end of the day these assumptions	1
	can't be made out, I can't make anything of it.	2
QUE	STION ALLOWED	3
XXN	I	4
Q.	Do you recall the question.	5
Α.	Could you repeat it, please?	6
Q.	It's a long one, so I will take my time.	7
Α.	Yes.	8
Q.	I would like you to assume that the accused has been	9
	diagnosed as being HIV positive by the ELISA test and	10
	the Western blot test. He has also had his viral load	11
	measured by way of nucleic acid test.	12
Α.	Yes.	13
Q.	And the genetic profile of his virus has been	14
	established. Assume also that he was living in a sexual	15
	relationship with a woman for an extended period of	16
	time. She subsequently, so after the accused was	17
	diagnosed, she too was diagnosed as being HIV positive	18
	with the ELISA, confirmed by the Western blot. Her	19
	viral load was measured by the nucleic acid test and the	20
	genetic profile of her virus was also established. I	21
	would like you to assume then that out of all of the	22
	genetic profiles of people who have been diagnosed as	23
	HIV positive, kept on a database at the IMVS in South	24
	Australia, the profile that was the closest to that of	25
	the accused was the woman with whom he had been living	26

	with in a sexual relationship. Doesn't that indicate to	27
	you that one of them has passed the virus to the other	28
	one way or the other.	29
A.	No, definitely not, and that is not what I say. This	30
	is not what the HIV expert say.	31
Q.	What is it that you say, you're the one presented as an	32
	expert.	33
Α.	I say no, and I base my knowledge on what the HIV	34
	experts say.	35
Q.	So it's just a coincidence then if their two profiles	36
	are the closest out of 850 or so profiles.	37
A.	There is no profile. What do you call HIV profile?	38

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Nobody has done any HIV profile. What they mean is a	1
small, a tiny bit of DNA. There is no profile of the	2
whole HIV genome on anybody. In fact, there is no	3
evidence for the existence of the whole HIV genome in a	4
human being. The HIV genome, as you say, the full HIV	5
genome it's existence is from cell culture, not from	6
people.	7
Put aside for one moment whether it's the full genome,	8
you can still have a genetic profile that is part of the	ne 9
genome, it doesn't need to be the full genome. If you	10
assume here that they are the two closest related, that	. 11
is the woman's is the most similar.	12
I understand the question.	13
- to the accused, are you saying that's just a random	14
coincidence.	15
No, because we don't know what's going on with the rest	16
The rest may be totally, totally - this is your	17
assumption that the HIV exists and the HIV DNA exists.	18
So if you find a small part, even if it's identical,	19
even if it's identical, the rest of the genome may vary	20
by 45%, because we say there the difference between our	21
DNA and that of the chimpanzee is 2%, the difference	22

being HIV in our DNAs is up to 40%, more than 40%, so.

particles, whatever they are, all the genomes, all these

But we still have HIV, we still say that all these

bits of DNA are HIV DNA. They accord for the came

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24

25

26

Q.

A.

Q.

Α.

	proteins, for the same particles, scientifically at	27
	least you can say it has to be questioned. You cannot	28
	have that big availability.	29
Q.	So if we accept what you say, let's assume for a moment	30
	that this is a very small sample of the genome that's	31
	been examined, that it doesn't necessarily reflect the	32
	full genome of that person's virus. It's a pretty big	33
	coincidence that they so closely match as compared to	34
	the 848 other people or profiles in the database.	35
OBJ:	ECTION: MR BORICK OBJECTS	36
MR :	BORICK: I don't understand the phrase 'genome of	37
	a virus', maybe I missed something, I don't understand	38

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MS	MCDONALD: It's the phrase the witness has been	2
	using. The whole genome.	3
MR	BORICK: The 'genome of a viruses' that's what I'm	4
	querying.	5
IXX	N	6
Q.	The point I'm making is a simple one. If that isn't all	7
	of the genetic profile of the virus, it's just a little	8
	bit from the woman and a little bit from the man and not	9
	necessarily reflecting the entirety of their genetic	10
	profile, it is a pretty big coincidence that those	11
	couple of random samples from each of them match more	12
	closely than anyone else on the database.	13
Α.	No, it's not. You can look for HIV, they should be the	14
	same. They should be the same in every person. They	15
	should vary no more than a few percent at most, but not	16
	by 45%, so, no. Unless, as I said, that is not what I	17
	say. This is how - what the HIV experts say, including	18
	Wain-Hobson.	19
Q.	That's not true, is it, that you would expect the virus	20
	to be the same in every person, because the HIV virus I	21
	suggest varies quite a bit.	22
Α.	That's what I said, varies by 45%. In fact, if the same	23
	person - in the same person you can have a million	24
	different HIV. Even if you're infected with one you	25
	have a million in a very short time and you cannot be	26

that expression.

	infected with one because everybody has another million,	27
	so you do not know what you're looking at. It is	28
	impossible to compare HIV genes. Each person has, and	29
	this is not my finding, this is what, as I said, the	30
	best HIV expert in the genome, including Wain-Hobson.	31
Q.	HIV is genetically unstable, isn't it.	32
Α.	It's unstable, of course it's unstable, that's what we	33
	are talking. Even if you - let's assume that there is	34
	HIV and you're able to put one HIV in a person, but how	35
	can you do it because each person has over a million, so	36
	if person X has sex with person B, person X already has	37
	a million, so when it has sex you can't, if it is	38

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transmittable, you cannot transmit one, you transmit	1
many of them, if not 1 million and then the person A	2
tells Mr person B, the person B in no time will start	3
making other viruses, in the end, within a very short	4
time, with totally different DNA and RNAs, so it is	5
impossible to compare.	6
CONTINUED	7
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Q.	I suggest it is because HIV is genetically unstable that	1
	the sequence of analysis which occurs when one is	2
	looking at the genes is based on what are called	3
	conserve genes, that is constantly express genes. What	4
	do you say about that.	5
Α.	No, constantly - conserve genes, that means constantly	6
	express genes. Conserve gene is a gene which is present	7
	in all of the HIVs and it does not vary, but they all	8
	vary. What they say when they said 'conserved', they	9
	mean that this gene is found in no retrovirus, including	10
	the genome retrovirus. That is what 'conserve' is meant	11
	by. It is found by a gene which is found in all the	12
	retrovirus.	13
Q.	I suggest, as I did to you yesterday, that there are six	14
	genes that are absolutely specific to HIV.	15
A.	How can you suggest that there is six genes, that they	16
	are specific to HIV? Do you mean that these genes are	17
	found nowhere else, these sequences are found nowhere	18
	else?	19
Q.	They are found nowhere else in a human body.	20
A.	Well, let me give you a paper and I have it with me.	21
	This time I have the paper with me. Please be patient	22
	for me to find. Your Honour, may I?	23
HIS	HONOUR	24
Q.	Yes.	25
Α.	Thank you.	26

XXN		27
Q.	Do you have copies of the paper for us.	28
A.	I will take it and I will give it to you. The paper is	29
	entitled 'Human Immunodeficiency Virus Type 1-Like DNA	30
	Sequences and Immunoreactive -'.	31
HIS	HONOUR: Can I have a look at it? I will read it	32
	out. It might be easier.	33
DOCT	UMENT HANDED TO HIS HONOUR.	34
HIS	HONOUR: The witness is referring to a paper	35
	entitled 'Human immunodeficiency virus type 1-like DNA	36
	sequences and immunoreactive viral particles with unique	37
	association with breast cancer'. It is a paper dated 22	38

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	December 1997 which was modified in 1998 and accepted on	1
	27 May 1998.	2
MS I	MCDONALD: I have no objection at the moment,	3
	subject to me getting a chance to read it overnight.	4
HIS	HONOUR	5
Q.	You wanted to refer to some paragraph or some part of	6
	that passage.	7
A.	May I read the abstract?	8
Q.	I will read it. It is easier for the reporters if I	9
	read it and you can tell me if you agree: 'RAK antigens	10
	p120, p42, and p25 exhibit molecular and immunological	11
	similarity to the proteins encoded by human	12
	immunodeficiency virus type 1 (HIV-1) and are expressed	13
	by 95% of breast and gynaecological cancer cases in	14
	women and prostate cancer cases in men. The binding of	15
	an epitope-specific anti-HIV-1 gp120 monoclonal antibody	16
	(MAb)(amino acids 308 to 322) to cancer RAK antigens has	17
	been found to be inhibited by a peptide derived from	18
	variable loop V3 of HIV-1. Breast cancer DNAs of 40	19
	patients were PCR amplified with HIV-1 gp41-derived	20
	primers, and all of the samples were found to be	21
	positive. The DNA fragments amplified in seven blindly	22
	selected breast cancer samples were sequenced. The	23
	breast cancer DNA sequences showed at least 90% homology	24
	to the HIV 1 gene for gp41. Antisense oligonucleotides	25
	complimentary to the HIV 1-like sequences inhibited	26

	reverse transcriptase activity and inhibited the growth	27
	of breast cancer cells in vitro. Viral particles	28
	detected in breast cancer cell lines were strongly	29
	immunogold labelled with the anti-HIV 1 gp120 MAb. The	30
	results obtained strongly suggest that the	31
	long-postulated breast cancer virus may, in fact, be	32
	related to HIV 1'.	33
Α.	So you can't be specific. If they are found in breast	34
	cancer, in other genome cancers and in prostate cancers,	35
	and, in fact, they are 90%, the DNA is 90% commodious	36
	with the HIV 1. So, in fact, it appears they are closer	37
	to the HIV genome than the HIV genome between them, and	38

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this is not - this is only just one paper. I have here	1
many papers who have exactly the same thing. They are	2
found in all general community. They are found in	3
people who have a disease in which they have very low	4
globins and, in fact, it appears they are tissues in	5
which you could not find HIV DNA with ordinary	6
hybridization techniques - I'm not talking about PCR -	7
it was tissue cells, and as I said, Gallo admitted this	8
in 1994. They could not find - in 1984 they published a	9
paper. Then, in 1994, he said we did not find any HIV	10
DNA in the Karposi's sarcoma cells and he said, in fact,	11
we did not find any HIV DNA in T4 cells but they found	12
in many other tissues, and I have here, as I said, it	13
looks - I mean, they were found because at one stage at	14
the beginning of the HIV thinking the HIV is transmitted	15
to insects and people have tested insects for the HIV	16
genome and they found the HIV DNA insects in a number of	17
them, different types. So, you can find HIV DNA - you	18
don't find it, only if you don't look.	19
MR BORICK: Logistically, we have got some documents	20
to get from Perth.	21
HIS HONOUR: Do you want me to adjourn now?	22
MR BORICK: I think so, because we are going to need	23
every bit of time we can get.	24
HIS HONOUR: Ms McDonald, how much longer do you think	25
you will be with this witness?	26

MS I	MCDONALD: I'm not getting to ask many questions at	27
	the moment, so I'm not getting through things very	28
	quickly at all.	29
HIS	HONOUR: We will just have to start at 10 o'clock	30
	tomorrow. If we don't complete her by 1, then we will	31
	have to start again in January.	32
MS I	MCDONALD: Also, obviously the sooner I can get any	33
	quickly at all.  HONOUR: We will just have to start at 10 o'clock 3 tomorrow. If we don't complete her by 1, then we will 3 have to start again in January. 3 MCDONALD: Also, obviously the sooner I can get any 4 further articles which are being referred to, the 5 better. Also, if the witness is going to be coming 5 prepared with more articles tomorrow, if we can have 5 them in advance then we won't have this problem again 3	34
	better. Also, if the witness is going to be coming	35
	prepared with more articles tomorrow, if we can have	36
	them in advance then we won't have this problem again	37
	tomorrow.	38

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HIS HONOUR:	We will adjourn until 10 o'clock tomorrow	Τ		
and we will see	e how we go.	2		
MS MCDONALD:	That article probably should be tendered	3		
at this stage.		4		
HIS HONOUR:	Do you want it tendered, Mr Borick? It	5		
has been refer	ced to.	6		
MR BORICK:	Yes. That is the one which has just been	7		
referred to?		8		
HIS HONOUR:	Yes.	9		
MR BORICK:	Yes.	10		
HIS HONOUR:	I will make it available to you,	11		
Ms McDonald, if	you want to copy it overnight.	12		
EXHIBIT #P23 DOCUME	ENT HEADED 'HUMAN IMMUNODEFICIENCY VIRUS	13		
TYPE ONE-LIKE DNA SEQUENCES AND IMMUNOREACTIVE VIRAL				
PARTICLES WITH UNIQ	QUE ASSOCIATION WITH BREAST CANCER'	15		
TENDERED BY MS MCDONALD. ADMITTED.				
		17		
HIS HONOUR:	The document is only about 10 or 12	18		
pages. I will	have my associate copy it when I adjourn	19		
and provide eac	ch of you with a copy.	20		
MR BORICK:	I would be grateful for that.	21		
ADJOURNED 5.12 P.M	. TO THURSDAY, 21 DECEMBER 2006 AT 10 A.M.	22		
		23		
		24		
		25		
		26		

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