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The Australian Response to AIDS

Recorded interview with : ELENI PAPADOPULOS-ELEOPULOS AND VALENDAR TURNER

Interviewer: Stuart Reid
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NOTE TO READER

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ELENI PAPADOPULOS-ELEOPULOS
VALENDAR TURNER
interviewed by Stuart Reid

TRC-2815/80 : Tape 1

Stuart Reid: Eleni Papadopulos-Eleopulos has followed a career in medical physics and is currently a biophysicist in the Department of Medical Physics at Royal Perth Hospital. The interview is also with Dr Valendar Turner who has followed a career in emergency medicine and general surgery and is currently a staff specialist in emergency medicine at Royal Perth Hospital. Eleni Papadopulos-Eleopulos will be speaking with me, Stuart Reid, for the Oral History Project conducted by the National Library of Australia and the Australian Federation of AIDS Organisations.

On behalf of the Director-General of the National Library, I'd like to thank you for agreeing to participate in the program. Eleni, you do understand that copyright is shared by yourself and the Library?

Eleni Papadopulos-Eleopulos: Yes.

Stuart Reid: That being so, may we have your permission to make a transcript of the recording, should the Library decide to do so?

Eleni Papadopulos-Eleopulos: Yes; please do.

Stuart Reid: We hope you'll speak as frankly as possible, knowing that neither the tapes nor any transcripts produced from them will be released without your authority. This interview is taking place today, the 25th of the 11th, 1993, at Eleni Papadopulos-Eleopulos's office at Royal Perth Hospital.

Dr Turner, also on behalf of the Director-General, I'd like to thank you for participating in this program. Dr Turner, you do understand that copyright is shared by yourself and the Library?

Valendar Turner: Yes.

Stuart Reid: That being so, may we also have your permission to make a transcript of the recording, should the Library decide to make one?

Valendar Turner: Certainly.

Stuart Reid: We hope that you too will speak as frankly as possible, knowing that neither the tapes nor any transcripts produced from them will be released without your authority.

Eleni, could we begin by getting some background on yourself; some of your biographical background, as it were?

Eleni Papadopulos-Eleopulos: What is really interesting here, in relation to my work, I can say that I have studied nuclear physics at the Bucharest University in Romania but I never worked in nuclear physics. Also my speciality is medical physics. I finished university and I came straight to Australia. So, from the beginning, I've been working in the medical field. So all my working life has been in the medical field.

As a physicist, initially I was doing routine work and development. In time I start being interested, not only in the physical side of my work but, because it involved patients and diseases, I started being interested in the biological side of my field. So I really studied and I self-taught myself biology, especially cellular biology. I have done a fair amount of theoretical research regarding cellular function in health and disease.

Stuart Reid: Did you originally come from Romania?

Eleni Papadopulos-Eleopulos: No, I'm from Greece. I was born in Greece but I studied in Romania.

Stuart Reid: What was it brought you to Australia?

Eleni Papadopulos-Eleopulos: My parents were here. In fact, the whole family was here. The extended family was here. So once I finished, I decided to come here as well.

Stuart Reid: And what's the nature of your work at Royal Perth Hospital now?

Eleni Papadopulos-Eleopulos: I'm a physicist. Most of my work is occupied with UV radiation and radiation in general.

Stuart Reid: What about your history of publication. What have been the main areas that you've published in?

Eleni Papadopulos-Eleopulos: I consider my major work is a paper which has been published in full [in 1982]. There have been some short abbreviated versions before 1982 but in 1982 I published a paper which I consider that would be my major work. It was called “A Mitotic Theory” and discusses cellular function in general and goes into theories of muscle function and cancer and disease in general. That has been published in the *Journal of Theoretical Biology*. Vol 96 pps. 741-758 (1982).

That paper has a fair amount of prediction regarding biological function in disease. We have tried in our laboratories here in the Department of Medical Physics, in

cooperation with other departments, to demonstrate some of the predictions of the theory. So far we have been successful with everything we tried and we've had a fair amount of publication, especially in regard to muscle function.

Stuart Reid: I'd like to take up some of the issues that arise from that work in cellular biology in just a moment, but first of all I'd like to get some biographical information from Val as well. Val, what about yourself? Where are you from and what's your academic and medical background?

Valendar Turner: I'm a graduate of the University of Sydney in 1969 in MB BS and I did a fellowship in general surgery from the Royal Australian College of Surgeons in 1977, and a Fellowship from the College of Emergency Medicine in the early eighties. I've practised as a general surgeon for a brief period of time but I've principally been involved medically in emergency medicine and have been at Royal Perth Hospital since the late 1970s.

Stuart Reid: Your research background: what sort of research work have you done while you've been working in the area of emergency medicine?

Valendar Turner: I've published... and it was clinical research. I don't do any laboratory research whatsoever. I've published some papers in the area of clinical medicine. I'm interested in major trauma and have published about diagnostic methods of diagnosing intraperitoneal bleeding and other, not too interesting, topics. I'm currently involved in a project in the Department of Medical Physics trying to establish a device to non-invasively detect the presence of intraperitoneal bleeding.

Stuart Reid: If I could come back to you, Eleni, what was it that led to your interest in HIV and AIDS?

Eleni Papadopulos-Eleopulos: My theory of cellular function predicts a fair amount regarding biological disease. When AIDS came to be, when first diagnosed, from the theory, I concluded, or I hypothesised, that the disease may not be caused by an infectious agent, as it was then assumed. Really, my main interest was... because when first AIDS was diagnosed in gay men, the main disease was Kaposi's sarcoma which is malignancy, a neoplasm. And the main topic of my theory was cancer. So I got very interested in Kaposi's sarcoma because it was a rare cancer. I got involved and I tried to study Kaposi's sarcoma and, because my theory predicted what is the cause of cancer or what is the mechanism of cancer, I came to the conclusion that - 'AIDS' was not what it was called then - AIDS was really equal to Kaposi's sarcoma and pneumocystis carinni pneumonia and was due to some parameters in the lifestyle and not an infectious agent. And since then I've been trying to prove that hypothesis.

Stuart Reid: It might be useful for us to get a kind of a lay person's explanation of your theory about cellular biology because at the... in order to see how you developed your theories about HIV/AIDS, it's probably important that we understand what it was you were thinking about cellular biology in general.

Eleni Papadopulos-Eleopulos: The theory predicts... and as I say, this is a theory and we have proved a few of their predictions but not all of them. The theory postulates that biological function, normal function, is due to oxidation and reduction of the cells and in particular a charge transfer between proteins - some of the proteins which exist in the cell - which is induced by changes in the redox stage. That is, the function is due to oscillation of the cellular redox - between the cell becoming more or less oxidised which is manifest as a permanent oscillation.

Now, the theory predicts that in disease there is a relative oxidation, so whatever causes the disease, apart from... you can have diseases due to a relative reduction because, for function to take place, you have to have all the time an oscillation between more oxidised to less oxidised or more reduced. Once you stop this oscillation, then disease ensues. That can be done either by too much reduction or too much oxidation. In general, you don't get reduction. Reduction is only when you give to a patient - when you inject or when you feed someone with a very high dose of reducing agents. But, in usual life, shall we say, in our life span, we get more and more and more oxidised. That's why we get all diseases, especially the chronic diseases like cancer, like cardiovascular diseases, like rheumatism. They are due to oxidation.

Stuart Reid: Is it possible to measure this oxidation and the oscillation from oxidation to reduction?

Eleni Papadopulos-Eleopulos: Yes, it is possible, and many people have done it and we tried it here, but unfortunately we don't have enough money to continue to prove that. It requires a lot of expertise and work. So that's why we did not try to actually measure the oscillation and the oxidation with time, because it's easier to prove the muscle function in regard to this theory. We have done it and we proved it, that oxidation plays a key role in muscle contraction and thus in cardiovascular diseases.

Valendar Turner: Could I just make a comment, because as a clinician I was impressed by this theory, although it takes a non-physics graduate a long time to come to grips with the ideas. These ideas are not easy. They're very general which, to a clinician's mind, somewhat detracts because we like specific solutions to specific problems. But I was particularly impressed with one of the predictions of the theory, that is that magnesium would benefit people with acute myocardial infarction, that is heart attacks. And a long time ago - ten years or more - Eleni's theory was able to predict that the use of magnesium would benefit patients with acute myocardial infarction. This has now been discovered to be true, almost by accident by some of the trials on the thrombolytic agents that are used in myocardial infarction.

In fact, in our emergency department, in our cardiology department, patients with acute myocardial infarction are now routinely given 80 millimoles of magnesium ion intravenously over a 24-hour period, and there's a marked reduction in the mortality, the incidence of malignant arrhythmias and also an improvement in the overall heart

function. So, as Stephen Hawking says, a theory is judged by how good its predictions are. This is a very good theory.

Stuart Reid: In relation to the question about measuring this oscillation, you say that what's required is money. Is it something that's been tested elsewhere? Can you point to any particular research that would support the actual physics of your hypothesis?

Eleni Papadopulos-Eleopulos: My theory is based on actual experimental evidence, the theory generally. There are experiments done in completely unrelated laboratories. Nobody relates ... thinks. What I have done, really, is to relate a vast amount of experimental evidence and come out with a theory. But there is plenty of evidence, especially now, as Val said, of things which the theory predicted and when everybody was thinking that I am a nut - you know, not a few people told me that - and now they are seeing that the evidence which came since then, although not based on a theory and is all more or less accidentally come upon; it proves my theory.

Valendar Turner: Surely, you should mention the work in AIDS patients. I mean, it's important for the present topic surely?

Eleni Papadopulos-Eleopulos: That is one of the... I thought maybe we should not... we don't want to talk about that. We'll come to that, but let's start from now. The theory predicted that all AIDS patients... my theory on AIDS now, coming to the theory of AIDS, which is based on my theory on the cellular function... the theory predicted that all AIDS patients will be oxidised in general and in particular the concentration of their sulphydryl groups will be decreased. This has now been shown to be the case.

The theory also predicted that AIDS patients should be treated with reducing agents. At present there are a few institutions around the world who are trying to use reducing agents to treat AIDS patients.

Valendar Turner: The frustrating thing is that they don't really know why they want to do it.

Eleni Papadopulos-Eleopulos: Yes, they don't know why. They are doing it, but they don't know why. They don't know why it is. They just found out... you see, there are different laboratories who specialise doing different tests, so when a disease comes about they just try and repeat the test to a given group and then they may or may not have a result. So that's what they do, you know. They have done the sulphydryl groups without knowing why they're doing it. They found out that they are decreased but they have no explanation why. My theory predicts - not only has an explanation, but predicts - that that's how it's going to be, and what shall we do to reverse it.

Valendar Turner: They also have the benefit, these agents, of being plentiful and cheap.

Eleni Papadopulos-Eleopulos: That is really the main thing. That is from the point of view of practice - of clinical practice.

Valendar Turner: And probably non-toxic.

Eleni Papadopulos-Eleopulos: Less toxic than everything which is used today.

Stuart Reid: And are they being used here in Western Australia in Perth?

Eleni Papadopulos-Eleopulos: No.

Stuart Reid: We might come to some discussion of that later on when we talk about the reactions to your research, but I'd like to still focus on the research itself and the stage at which the first cases of AIDS started to appear in the literature and generate some interest. At that stage, as I recall it at least, infection wasn't the first hypothesis. There were people speculating that it had more to do with lifestyle, sexual practice, drug intake and so on. Can you recall that period of time and your thoughts developing about the disease during that period prior to the acceptance of an infected agent?

Eleni Papadopulos-Eleopulos: There were many, many people, as you say, initially who did question lifestyle and specially drugs and they were saying that maybe this would be the cause. But, from the very beginning, the CDC, the Centre for Disease Control, in Atlanta, Georgia and Gallo from the National Cancer Institute, they postulated that an infectious agent... The fact that the disease was restrained in the gay community in which at least some of them were sexually promiscuous, they postulated that it was an infectious agent which was sexually transmitted. More or less all the efforts were put in to proving the infectious theory of AIDS.

Gallo, again because it was Kaposi's sarcoma which was the main disease, and Gallo was working nearly all his life in proving a viral etiology of cancer. In fact, he claimed to have proven that at least one human virus, a special virus called retrovirus, HTLU-I, as to being the cause of one neoplasm. He postulated that AIDS, that is Kaposi's sarcoma, and thus AIDS, in gay men was due to the same retrovirus which he isolated a few years before.

Stuart Reid: What's your assessment of his work at that time?

Eleni Papadopulos-Eleopulos: To be honest with you, at that time I did not know much about Gallo's work and I did not try to find what was the infectious theory of AIDS. In fact, my first writing on the cause of AIDS did not mention either Gallo or any of the other people who are, shall we say, pushing the infectious theory of AIDS. The only reason that I came to look at the other side was the strong advice or, shall I say, impetus which John Papadimitriou gave to me, because he said if I want to be credible, I have to look at both sides; that is, the other side which by 1983... all of this

time to be accepted and my view, or my hypothesis, I have to talk in parallel about that. So that's why I started getting involved to look in the viral etiology of AIDS.

Valendar Turner: I can just add to that briefly, that since the viral etiology has become more or less universally accepted, Eleni's theory has become unknown or almost unknowable, and most of our work has centred on - rather than expounding and explaining the original oxidative theory - has centred on attacking the HIV theory.

Stuart Reid: Which do you think would be the better area to start on first in this context? Should we discuss your criticism of the HIV theory first?

Eleni Papadopulos-Eleopulos: Yes, I think that would be best.

Stuart Reid: What is it that's fundamentally wrong with the HIV theory - HIV as the cause of AIDS? What's wrong with that theory?

Eleni Papadopulos-Eleopulos: There are many. First of all, to say that an agent - it doesn't matter if it is infection or non-infection - is the cause of a disease, you have to prove that the agent exists. Once you prove that the agent exists, you have to prove that the agent is capable of causing the disease and then to prove that that disease, in a given population, is indeed caused by that agent and not by something else - because many diseases have multiple etiological reasons.

Stuart Reid: What's your view on the existence then of the virus? Does the virus actually exist?

Valendar Turner: Well, it sounds heretical to say so: We don't believe that the evidence which is currently available proves beyond reasonable doubt that the virus does exist.

Eleni Papadopulos-Eleopulos: Or that AIDS patients, any AIDS patients, are infected with HIV.

Valendar Turner: There are a number of reasons for saying this, and this is obviously the, if you like - forgive the term - the guts of this particular part of the debate. I mean, people who do AIDS research are all, I'm sure, very intelligent and very skilled and they are obviously seeing something down their microscopes. The argument we have is that we do not think that the appellation HIV is earned by the various phenomena that they see down their microscopes. I'm using that term...

Eleni Papadopulos-Eleopulos: Not only microscope, but in their petri dishes, microscopes and everything else. The phenomena which are...

Valendar Turner: I'm using the term "microscopes" in the metaphorical sense. It's partly microscopes, electron microscopes to be specific, and other laboratory phenomena. I am very keen on the use of the word "metaphor" to explain it. I mean,

we will explain it, but my personal view is that I regard the term HIV as a metaphor. As Aristotle said, a metaphor is a name you give something that belongs to something else. We regard HIV as a metaphor for a whole lot of other related, unrelated - who knows - laboratory phenomena which occur in cultures of tissues from AIDS patients under various circumstances. Now, we can go into those in a minute.

Stuart Reid: Are these viral phenomena?

Valendar Turner: That's debatable.

Eleni Papadopulos-Eleopulos: Oh, what a surprise. [Both laugh about the question.] [Tape stopped momentarily.]

Stuart Reid: The question of whether what the researchers are seeing is actually something which is viral - it may not be HIV; but are they seeing viral phenomena when they're looking at this cellular material that they're finding?

Eleni Papadopulos-Eleopulos: We don't know, because all the phenomena they are seeing, they can appear independently of viruses, like, you know, reverse transcriptase, that is one enzyme which when it's detected is considered to be unequivocal evidence of the presence of a retrovirus. It's not the case because reverse transcription can be found in normal cells and many other viruses including hepatitis B virus which is ... It has been proven beyond doubt, the existence of reverse transcriptase in hepatitis B virus. A significant number of AIDS patients, in fact most of the haemophilia patients for sure, are infected with hepatitis B virus. So the detection of reverse transcriptase in the culture of these patients, in our view, cannot be considered as proof for the existence of a retrovirus.

Valendar Turner: And it's important to note that hepatitis B virus has been discovered in lymphocytes - in human lymphocytes as well.

Stuart Reid: Are they not though finding sufficient characteristics of a virus which is different from these viruses which appear - retroviruses - elsewhere for them to say, "This is something different in this case."?

Eleni Papadopulos-Eleopulos: You can't... you don't have HIV... what do you mean by HIV? As we said, by HIV you mean a number of phenomena which you will detect, but not one of the phenomena is characteristic to HIV or to any other retrovirus, so you can't say really... You know, if you have one characteristic, one phenomena, which belongs only to HIV and to nothing else, then you can say when you detect that phenomena, that you detect HIV. But this is not the case.

Stuart Reid: Would you not also be able to say that with a particular combination of things which only appeared in these circumstances?

Eleni Papadopulos-Eleopulos: No, they don't. They don't appear only in these circumstances. They appear in...

for example, what is called a retroviral particle, it can appear in other circumstances in connection with reverse transcriptase.

Stuart Reid: Eleni, I'd like to get this point as clear as possible: why is it that the data do not validate precisely the presence of unique exogenous retrovirus?

Eleni Papadopulos-Eleopulos: Well, by an exogenous retrovirus, we mean a virus which comes from outside, say one person who comes in contact with another person and the second person is infected with it. Then, if you find that virus in the second person, you say that that virus has been transmitted from one person to another. With retroviruses, this is not the case. Every single one of us has information in his or her cells to, under given conditions, under the right conditions, to synthesise viral particles. So it doesn't matter who you are, even if you're the most healthy person, you still, when your cells are taken out and especially when they're put into the petri dish, the cells and the right condition will start to produce retroviruses. So this is one part.

Now, to prove the existence of a retrovirus, of a unique retrovirus, you have to show that the parameters which you are detecting in the petri dish, because that's usually how retroviruses or viruses are proven - their existence is proven usually in the petri dish - you have to prove that these retroviruses or HIV has unique phenomena associated with it. Now, for HIV, what is called HIV, is the detection in the petri dish of reverse transcriptase - a few phenomena, including the reverse transcriptase, virus-like particles and an antibody-antigen reaction. That is, when you take some of the proteins which are in the petri dish and you react them with blood from patients, there is a reaction between them which is interpreted as being that the patients have antibody directed against this virus.

So let's start and see each of them... if any of them is specific to HIV. As we said, we have published a paper this year in the *Journal of Biotechnology* - Val, myself and John Papadimitriou - and we explain there. We give detailed explanation why the data which is presently interpreted as proving the existence in AIDS patients of such a virus does not prove it. So reverse transcriptase we have already mentioned. In fact what they're detecting in the petri dish is reverse transcription which means you make DNA from RNA. So you have RNA and from the RNA with a given enzyme you can make DNA. Now, the detection of this phenomena is interpreted by all the HIV experts as proof of the existence in the petri dish not just of a retrovirus but of HIV.

Now, this cannot be the case. First of all, even if it is [that] the reverse transcription is specific to retroviruses, it cannot be specific to HIV because what you detect there may be a retrovirus which you already had in the cells there. Then you detect a retrovirus which was just synthesised because of your condition. Secondly, the reverse transcription is not specific to retroviruses. As we already mentioned, reverse transcription can be found in all cells, infected or non-infected and, in many viruses its existence has been proven which are not retroviruses. So this is another problem.

And thirdly, the way reverse transcriptase is detected in the petri dishes of AIDS patients cannot even prove that it is a reverse transcriptase. It is because all the normal enzymes which make DNA in our cells can induce the same phenomena. So you don't know really, even if you detect reverse transcriptase in the petri dish or you detect one of the other enzymes which exists in all our cells and which make DNA. So that's the problem with reverse transcriptase - one of the phenomena, the main phenomena, which is considered to be proof of the existence of HIV.

Stuart Reid: What about the viral-like particles?

Eleni Papadopulos-Eleopulos: Well, virus- or retrovirus-like particles have been found for a long time not only in the petri dish but even in tissue; in fresh tissue from animals and even humans. In the petri dish these particles can appear. If you put cells in the petri dish and you keep them for a long time and you have the right condition, the cells will start producing particles. If you radiate the petri dish or if you put substances which these days are put in there, so-called AIDS cultures, these particles will appear, you know, in normal non-infected cell cultures. So, again, do not prove that what is seen as a particle in the culture is proof of HIV. In fact, the same particles, exactly the same particles, have been seen in fresh tissue of people, of individuals, who have diseases other than AIDS; exactly the same morphological characteristics.

So, finding a particle, in our view cannot be considered as proof of the existence of HIV.

Stuart Reid: So, while each of these things can't be considered as proof, taken altogether, aren't they indicative, strongly indicative?

Valendar Turner: Why? Why should they be? If individually they are not specific, how can combining them make them more specific? I don't see...

Eleni Papadopulos-Eleopulos: It's not only individually. What you see in the petri dish. You put normal cells and in normal cells you can find both reverse transcription and particles. In time you get in these cultures reverse transcription and particles. So how can we say... You can find these two together in AIDS cultures and in normal cultures. So how can you say that they are...

Valendar Turner: And it's fair to say that Gallo himself discovered some of this in the seventies.

Eleni Papadopulos-Eleopulos: In fact, not only that, but let's be fair to Gallo because Gallo repeatedly said that finding reverse transcriptase and particles is not proof of the existence of a retrovirus. It's not proof of isolation of a retrovirus in the culture and even of its detection. The only thing which Gallo considers as proof for the existence of a virus in individuals is to find antibodies which react - antibodies in the sera of people - which react with the proteins which are found in the test tube.

Stuart Reid: And you have problems with the antibody theory as well?

Eleni Papadopulos-Eleopulos: Yes. In fact, that is our main... one of the main themes in our...

Valendar Turner: Could I just add here, speaking as an outsider in a sense, that the term "virus" is used interchangeably. It's very important for people who listen to this tape to realise that the term "virus" is used in two different ways. When electron microscopists look at these particles, they use the term "virus" in a morphological sense; that is, they describe what they see. When an ordinary person uses the term "virus", they think of something like the flu or a cold, something which has associated with it the ability to infect an individual and then pass the same infection on by means of this agent. Now, it's fair to say that strictly scientifically, before you can use the term "retrovirus" in the sense that it's an infective exogenous agent of disease, that you have to have proof that the latter occurs. And it's unfortunate that the term is used interchangeably. Does that come across to you?

Stuart Reid: Yes, it strikes me that you're talking about the difference between what they're actually seeing in the dish and what's actually making people sick. But people are getting sick. There is no question that there is something that is making people sick, and we'll come to what you think it is in a moment. But, in looking at the cellular material from people who are sick or who are infected, to use the generally accepted term for what's happening here, are you not seeing a combination of things when you look at them - electron microscope or petri dish or whatever - which is different from people who are healthy?

Eleni Papadopulos-Eleopulos: No, you can see the same combination of things, you know, in a petri dish even if people are healthy. That's one.

Stuart Reid: But you're not... you wouldn't get the antibody reaction?

Eleni Papadopulos-Eleopulos: No, the antibody reaction you won't get with a healthy person - although there are many healthy people, you know, which... in fact, 10 per cent... with the most stringent criteria they're using to define a positive test, 10 per cent of healthy people test positive. So there are many healthy people, or people who are not at risk of developing AIDS, who test positive. Now, the other thing is: the fact that AIDS patients test positive, they have a positive antibody test, does not mean that the antibody which is in these people is really an HIV antibody. The antibody can be directed towards many other infectious and non-infectious agents which the AIDS patients suffer from. Then it can cross-react with the so-called HIV proteins.

So, we don't have proof that really the antibodies which react with the HIV proteins... let's say the proteins or the particles there, they are HIV, you can't define by definition,

you know - just because you find a reaction you [can't] say that that is a reaction due to antibodies or due to infection of that person with HIV.

Valendar Turner: Perhaps we need to explain in a little more detail what the AIDS antibody tests actually are because this must be of interest in the clinical dimension, because people go to doctors and have AIDS tests.

Eleni Papadopulos-Eleopulos: How can you determine a proper test? How can you evaluate an antibody test?

Valendar Turner: The basics of it are that it is believed that foreign material injected or getting access to the body causes the body to make proteins called antibodies which react with these and are supposed to neutralise their effects. In the case of HIV, to do the test you need to have two things. You need what they call HIV and you need some blood from the patient and these are reacted together and the ensuing reaction is somehow measured.

Now, the source of HIV is not, as you may imagine, a pure suspension of millions of tiny little retroviral particles. It is, in fact, a number of proteins derived from cultures of tissues from AIDS patients with other added chemicals - the petri dish we referred to before - in which some purification or separation has taken place by means of ultra centrifugation.

Eleni Papadopulos-Eleopulos: Or filtration.

Valendar Turner: Or filtration. Now, we have to assume therefore that the proteins that are obtained by this methodology are in fact specific for HIV, and there is no evidence... there's no data to support this. In fact it's admitted by the AIDS experts themselves that 80 per cent of the proteins obtained in this manner are in fact cellular and non-viral.

Eleni Papadopulos-Eleopulos: The only reason that they are called HIV proteins is because they react with AIDS patient sera, but you can't have that as proof that they are HIV. First, we have to prove that they are HIV and then you have to look at the reaction. You can't go the other way round, you know, like Gallo and Montagnier, you know. You find that this protein reacts with AIDS patient sera and, because they react, they're HIV.

Valendar Turner: Just to continue in that vein, the preparations are not pure and, even if they were pure let us postulate, then if we look at the particular proteins which are believed to be specific for HIV, the important proteins, then there is data which is discussed in our *Biotechnology* paper that for each one of these important proteins, there is evidence that indeed these proteins are not specific for HIV.

Now, if we assume these proteins are specific; that is when you react the patient's blood with these proteins, that you're dealing with proteins which come from a unique

agent, that is HIV, that still does not prove that the antibodies present in the patient's blood were made in response to the foreign stimulus, HIV, because antibodies by their very nature - and there's no disputing this fact - may cross-react with many other proteins, other antigens.

The only way to prove that the antibody-antigen reactions that are seen in these tests mean the presence of a particular agent, in this case HIV, is to verify the tests in parallel with a gold standard, that is another independent, not related to antibody antigen reactions, way of determining the presence of HIV. And in the case of HIV this has to be the isolation of HIV itself. There's no work has ever been reported that this has ever been done. In our *Biotechnology* paper we go into this fact and we also explain why, in our opinion, if this was ever attempted, there would be considerable problems in achieving this end.

Stuart Reid: What does this say about the clinical usefulness of the tests?

Valendar Turner: We say, as a succinct summary of the situation as we see it, that the relationship between the antibody tests, the so-called HIV antibodies, and the virus is completely and utterly unknown because of this gold standard problem.

Eleni Papadopulos-Eleopulos: Now, if you want to use the antibody tests as a marker for future development of AIDS, then that's all right. But you cannot say because you have a positive antibody test, you prove the existence in the patients of HIV. As long as you take that into consideration, as long as you don't say a positive antibody test is proof of HIV infection, it's all right.

Valendar Turner: And in this regard, it's useful to give a couple of examples of this that people will understand. It's well known that if you get an attack of glandular fever, for example, which is a disease most people have heard of, then the doctor can do a test on your blood to see whether you've been infected with the putative agent of this condition. The test that the doctor actually performs - that he orders and is performed in a laboratory - is a test to detect antibodies which react with the red blood cells of sheep. Now, this is a well known test, it's called the Paul-Bunnell test. It's ordered every day in surgeries all around Australia and it works. But one cannot say from this information that people with glandular fever are infected with sheep red blood cells or that sheep red blood cells are the cause of glandular fever.

The other example we often use is the example of syphilis. One of the syphilis antibody tests which predicts the likelihood, the propensity, to develop syphilis in individuals, is where they have antibodies to ox heart proteins. And this is used as a screening test, if you like, for syphilis. Again, no one is postulating that patients with syphilis are infected with ox heart or that ox heart is the cause of syphilis. So, it's important to always examine what you think a test is telling you and what you really are entitled to know.

Eleni Papadopulos-Eleopulos: And to interpret... how you're interpreting. It's the difference between what the actual test tells you and how you interpret it. If you want to, you know, use the so-called HIV antibody tests for a prediction of AIDS, that's quite all right.

Valendar Turner: It's quite all right in the high risk groups.

Eleni Papadopulos-Eleopulos: Exactly. You have to be very, very precise here. It's quite all right in the high risk groups. But the same test in the population at large doesn't say anything, even about the development of AIDS.

Valendar Turner: And the reason we can say that is because there is no data on the predictive value of these tests in otherwise healthy people, and it would be now very difficult to obtain this data because persons who are told they are positive believe that they are infected with a lethal human retrovirus for starters and secondly, they may be treated with drugs as a result of this which in turn may make them sick for other reasons. It's almost an experiment that one cannot do at present. It's terribly important to get this point across: that it's fine to use the tests as a marker in high risk groups but to turn around and say to a healthy individual that "You are infected with a human retrovirus, HIV, on the basis of these tests" is not scientifically allowable.

Eleni Papadopulos-Eleopulos: Not even a healthy... even a non-healthy... even an AIDS patient, you can't tell him, because the test is positive, he is infected with the retrovirus, unless you have proof that these tests indeed mean the presence of a retrovirus which, to date, we haven't got.

Stuart Reid: There is a great deal of testing of healthy people, particularly through the blood transfusion service, where blood is tested routinely and very, very few cases of positive findings are emerging in those studies. So it would be highly indicative if there were... if it was positive, that there was something else there, especially if you could do the test again in a week's time and find that it was still positive.

Eleni Papadopulos-Eleopulos: Let's continue with the example which we already gave with syphilis. If you test normal people, blood donors, for antibodies to ox heart, you hardly find any one of them testing positive, but if you go and test IV drug users who are not infected with the agent which causes syphilis, you find about 20 per cent of them testing positive, although they don't have the infectious agent - 20 per cent of them will test positive. So testing normal healthy blood donors does not prove anything about the specificity of the HIV antibody tests. What you have to do is to test sick individuals who you know they are not at risk and they never develop AIDS.

If you look in the literature, you'll find out that many of these groups of people, including people with lupus erythematosus, people who have organ transplants, a high per cent of them have positive HIV antibody tests, but that does not prove that they are infected with HIV.

Valendar Turner: I think it's important to emphasise this point about specificity in relation to testing healthy blood donors, because we know from our correspondence with the authorities in Australia that the specificity of the HIV antibody tests is verified by testing five thousand healthy blood donors. Now, you would not expect to find healthy people making antibodies to anything new or different. That's what health is all about. But the way the specificity is defined, defined as the number of negative tests in people who do not have HIV infection, you will conclude, erroneously, that these tests are highly specific as an artefact of the way you've selected the population you test for specificity on.

In America there was a study called the Sentinel Study which is at odds and supports our view that in fact people... we don't know how healthy these people were but they were in hospital but they had everything else excluded that was vaguely AIDS related, even from the point of view of being people who'd had car accidents, because...

Eleni Papadopulos-Eleopulos: Gunshots.

Valendar Turner: Gunshots... and it depends which hospital they looked at. I think there were twenty-six hospitals looked at and they found antibodies in somewhere round one per cent to around 1.6 or 1.7 per cent.

Eleni Papadopulos-Eleopulos: More than that, 1.7 per cent I think it was.

Valendar Turner: But this was interpreted as proving that HIV is prevalent in this particular population rather than the obvious, that is, that the antibody tests are non-specific.

Stuart Reid: You also argue some specific problems with the western blot test which is used to confirm cases when the Eliza test indicates that a person is positive in the first place.

Eleni Papadopulos-Eleopulos: Well, in addition to the fact that nobody has proven the specificity of the western blot, although it's been considered to be 100 per cent specific, there is no antibody test or any other test used in medicine, I'll say, which is 100 per cent specific. Yet, the western blot is considered to be 100 per cent specific without ever its specificity being determined. So that is really a big problem with the western blot. But, in addition to that, there are many other problems associated with the western blot. One is that the western blot is not standardised. Every single laboratory, major laboratory, in the USA uses a different criteria for interpreting western blot. So it depends where you're going - in which laboratory your test has been done. In one laboratory you may be called positive; in the other laboratory you will be called intermediate or negative.

Valendar Turner: Indeterminate.

Eleni Papadopulos-Eleopulos: Indeterminate. In Africa and in Asia the tests are not routinely performed. AIDS is defined only on clinical grounds, but when a test is performed the criteria used in those continents is completely different to the criteria used in Europe and the United States. So it is really impossible to come to any conclusion in regard to the relationship between a positive for western blot - doesn't matter what it means - and the development of AIDS because there are no standard criteria.

The second problem is that the test is not reproducible. In the same laboratory, the same blot could give different results from one day to another and, certainly, the same blood - we do give one example in our paper - in nineteen different laboratories gave completely different patterns, because the western blot is read as a pattern - bands. Each laboratory gave a different pattern.

Stuart Reid: But in all cases, close enough to be, say, all positive or all negative?

Eleni Papadopulos-Eleopulos: It's not close enough. Depends where, you know... what criteria you are using.

Valendar Turner: Let's just expand on the western blot. The western blot is a technique for actually visualising the individual so-called HIV proteins in a strip - a little strip of nitrocellulose. You can look at it. Various reagents are used to colour the bands so you can see them. They're read by eye. The bands are the various proteins separated out according to their charge and molecular weight. Now, there are, as Eleni said, different criteria for positivity. The only thing the laboratories will agree on is that if there are no bands at all, including, which is rather intriguing, bands which do not represent reactions with non-viral proteins, the test is negative; everyone agrees. They all have different criteria for positivity. Anything that doesn't fit either of those is called indeterminate.

Now, the criteria for positivity vary and, to make it quite simple and to bring it home, you can get on an aeroplane in one country or in one state in the United States and you can travel from Canada and you can travel back to the United States and you can even travel to Australia or you could travel to Thailand and you can be positive or indeterminate depending on where you find yourself. Now, a person may wish to know what is going on. It makes a big difference to know whether you're indeterminate or whether you're positive. Who can blame people for asking this question? Unfortunately, in some centres, the western blot bands are not actually reported. The judgment is made according to whoever reports them, so the clinician may not even be aware that this is an issue.

Eleni Papadopulos-Eleopulos: And, in fact, you know, because we're saying here with... in the indeterminate results. If you give people negative blood (Not recorded)

Valendar Turner: The western blot is a technique for visualising the individual so-called HIV proteins. You see bands where the antigen-antibody reactions occur. It's read visually and the proteins are accorded a place by their molecular weight and charge. For example, P41 is a molecular weight protein of 41,000. Now, in western blot reporting there are three criteria. One is positive, the second is indeterminate and the third is negative. All laboratories agree that a negative test is no bands reacting whatsoever, including, rather intriguingly, bands which do not represent reactions with viral proteins. A positive test is defined in various ways by different laboratories and an indeterminate test is one which is neither positive nor negative.

To bring this down to a thing that people will understand is that you can actually... because criteria for positivity vary, you can get on an aeroplane somewhere in North America and travel around, say, Canada and the United States, even take a trip to Thailand, and you can be indeterminate or positive depending on where you find yourself. Such a person may wish to know, not unreasonably: am I infected definitely with this lethal retrovirus or aren't you sure? And you can't answer in certain states, because of this conflicting data on what constitutes a positive test. I mean, we wonder when people make these pronouncements that their set of criteria is positive and the next state's not, how do they know? How do they work this out? No one has ever explained this.

Eleni Papadopulos-Eleopulos: Nobody tells you why, you know, some people consider this positive and the other people consider the other. In fact, there is no agreement, not even between two laboratories. Now, I think we already talk about standardisation and...

Valendar Turner: Also this problem illustrates that one has to have the gold standard test done to sort this out. That is the only way that it can be done; there is no other way.

Eleni Papadopulos-Eleopulos: This is for specificity but, you know, we still can agree. You know, even if you don't have that, you still... before you determine the specificity... you can't determine specificity. Before you use the gold standard, you have to agree as to what you consider are positive tests. There is no agreement what you consider a positive test, so you can't even go to the second step to prove it is specific. And that is the problem for reproducibility. The test is not reproducible.

As we said, the test is not standardised and is not reproducible, so unless you have, you know, before you even talk about specificity, before you use a gold standard, you have to have the test... being able to reproduce the test from day to day and from laboratory to laboratory. This is not the case with the HIV antibody test. The same blood in two different days could give a different pattern and the same blood in different laboratories can give you different patterns of western blot. In fact, we have in the paper one example with the same blood being tested in nineteen laboratories and each laboratory came up with a completely different pattern of western blot.

Valendar Turner: We also have examples of... in the reproducibility where tests that were known to be positive were occasionally negative.

Eleni Papadopulos-Eleopulos: And the other way round, you know, in the same laboratory.

Valendar Turner: These are not just any laboratories. These are in reference laboratories. This data is from reference laboratories.

Eleni Papadopulos-Eleopulos: The best laboratories. Apropos of indeterminate western blots... in fact not indeterminate. For example, let's go with people who are given HIV negative blood... negative testing, completely negative, the western blot pattern completely negative. You give them and...

Valendar Turner: What Eleni means is you transfuse patients with blood known to be HIV western blot negative.

Eleni Papadopulos-Eleopulos: Negative. And then you test them again after their transfusion and you find that 40 per cent of them have an indeterminate western blot, 30 per cent of them have a p24 band, this protein, p24, which Montagnier considers - you know, the Pasteur Institute which are today accepted as being the first to have isolated HIV - p24 as the most specific protein or band to HIV. In fact up... we don't know what are the criteria in France now, but up till 1987, the p24 was considered proof of HIV presence in people.

Valendar Turner: This is rather a chilling statistic, because there were a lot of people tested in the early days of the so-called... of the epidemic, the AIDS era, especially haemophiliacs who were tested back in the mid-eighties...

Eleni Papadopulos-Eleopulos: Not mid-, before.

Valendar Turner: Well, the tests only started in 1984 but they haven't been tested since. Maybe some of them would not be classified as positive. I mean, I'm sure some of them at least would be classified as indeterminate if they were retested and had the same band patterns as they did then. Some of them didn't have a western blot. Some of it was done on the other antibody test, the Eliza. The important thing also about p24 is that we definitely know that this is the case for Montagnier because Eleni actually has met Montagnier and discussed this. I mean, that's just a little aside to make it interesting. But there's no doubt about it that they believe this.

There's a fascinating study from - is it Russia? - where patients have had blood... their own blood taken from them, removed and then irradiated and then put back, and I'm not sure of the percentage...

Eleni Papadopulos-Eleopulos: The blood was negative, the people tested before it was reinjected, and yet, when the people were reinjected with their own blood which was irradiated, after that they tested positive for the p17.

Valendar Turner: And in a similar manner there's some data from experiments done in mice where, if you take the blood from a healthy mouse and inject it into another unrelated healthy mouse, they produce antibodies to these HIV proteins. And how one can claim that these are specific for HIV is a complete mystery as far as I'm concerned.

Eleni Papadopulos-Eleopulos: The mice are not infected with HIV. That is accepted. Yet, you know, when they're injected with blood... in fact when they're injected, mice which are used as a model for lupus, when they're injected with foreign blood - blood from other mice - they test positive to the two most specific HIV proteins, that is p24 and p41. So this raises questions certainly to us. These pose questions regarding the specificity of the antibody tests.

Valendar Turner: In regard to this, this is one of the most frustrating aspects of the work, especially the *Biotechnology* paper, is although the paper which is quite long - it's about ten thousand words - has a lot of data in it, all in a similar vein to what we've been discussing about the tests, we've never had any scientific criticism. As one of our commentators has said, none of the big shots have ever attempted to answer the issues that we raise.

Stuart Reid: Why do you think that is?

Eleni Papadopulos-Eleopulos: We do not know.

Valendar Turner: We don't really know. We don't know whether they just haven't read the paper. They certainly must know about it; it's well publicised. It's in a very well known journal. They choose not to deal with these very fundamental issues. I mean, I can't imagine anyone listening to this not wanting to know answers to these questions. These are reasonable questions and people have a right to know about the answers.

Stuart Reid: And are these refereed journals that...

Eleni Papadopulos-Eleopulos: Certainly. *Biotechnology* is a sister journal of *Nature* which is... they're both extremely...

Valendar Turner: Top class.

Eleni Papadopulos-Eleopulos: They're considered to be some of the best journals.

Stuart Reid: What about the people that they've sent your work to to be checked before it gets published? Have they not taken it up at all?

Eleni Papadopulos-Eleopulos: They're reviewers. The reviewers did make...

Valendar Turner: You mean taking it up as an issue to propagate our views, you mean? We don't... reviewers are always anonymous. We don't know who they are. If they have, then we certainly don't know about it. But it was done in the United States, not here.

Eleni Papadopulos-Eleopulos: No, we sent it to the United States. We don't know where the paper was reviewed. We don't know who the reviewers are or where they're coming from. And we did make a lot of changes to the paper. We sent in fact to the journal about three revised manuscripts. We had to change it again and again before it was published. We don't want to antagonise people. We're just putting scientific questions. In fact we really love to have scientific exchange, not personal, not political, not anything unrelated to scientific questions. In fact we really... I mean, I will love to have any criticism, even if somebody proves to us that what we're saying is rubbish, we will be more than willing to accept it if we are proven that way.

Valendar Turner: Absolutely. We want to keep the debate scientific. In fact, it would be nice in this interview to just paraphrase what John Papadimitriou said about how we regard ourselves. That is that we do not regard ourselves as AIDS experts; we are not AIDS experts. All we have done is taken the hypotheses that are put up by the AIDS experts and using their own data, we point out the inconsistencies in what they say. And we would like to know what their response is.

Eleni Papadopulos-Eleopulos: Not only the AIDS data but the AIDS data and in addition the data presently available since 1911...

Valendar Turner: 1911, when Roux started all this.

Eleni Papadopulos-Eleopulos: Right, in relation to retrovirology in general. So it is discussed in view of the presently available data as well as the data in retrovirology in general. We are not... we never have seen... I mean, I never have seen an AIDS patient. I mean, I've seen people I know but not...

Valendar Turner: Should we just, in anticipation, mention the PCR because in private discussions people say that our criticisms of Gallo's and Montagnier's isolation methods are now outdated because we have the polymerase chain reaction data, and this is such an exciting new development in molecular biology that people don't often think about it critically what it actually means. Eleni, you can explain this better than I.

Eleni Papadopulos-Eleopulos: I think suffice to say here that the Pasteur researchers, and not one, many, do not believe that HIV infection can be defined in terms of molecular biology because they say that there are many problems, you know, including the fact that no two HIVs have been isolated which are the same. The

specificity of the polymerase chain reaction never has been determined. Even if the specificity is determined and reproducibility and everything else which is required with the tests, even if they are all set out and shown to be proper, and even if all HIVs were one and the same, you still can't define it because finding... with a polymerase reaction you can detect only a very small part of what is called the HIV genome. But finding a small part of the HIV genome does not mean that the whole genome is there because the genome for retroviruses is... most of the retroviruses are defective. That is, you have one bit but you don't have the rest. So finding one bit... and in fact they say that about 99 per cent of the HIV genomes are defective. So finding many bits of positive PCRs does not mean that the HIV is there.

Valendar Turner: So although it's attractive to think you've found a tiny piece of this genetic material of a virus that we don't believe has been properly demonstrated, this is not - given the nature of retroviruses, that they can arise from pre-existing information that we all have - you can't equate these two phenomena.

Eleni Papadopulos-Eleopulos: In fact today, you know, there is evidence, of course presented by others, that even the normal human genome has HIV sequences.

Valendar Turner: That's referenced in the *Biotechnology* paper.

Eleni Papadopulos-Eleopulos: And since then we had more information. So, you know, finding a bit of what is called HIV genome in an AIDS patient does not prove that that person is infected with HIV.

Stuart Reid: I wonder if we could turn to what you think is actually causing the condition that we know of as AIDS or AIDS related condition?

Eleni Papadopulos-Eleopulos: In our view, as I said, again this is a hypothesis which has to be proven, is that each AIDS risk group has a different set of causes of HIV... sorry, of AIDS. You know, each group, although the disease appears to be caused by the same agent, in fact it doesn't even appear because the set of conditions which are called AIDS in one AIDS risk groups are not the same with the set of conditions which are called AIDS in another AIDS risk group.

Although some of the diseases overlap, for example, you know, most of them don't. Kaposi's sarcoma is a disease. In fact, the disease for which the HIV hypothesis has been put forward, is exclusively restrained to gay men. So how can you know... if HIV was the cause of AIDS in all AIDS risk groups, then we know viruses produce the same disease - doesn't matter in which person they are - all AIDS risk groups would have had Kaposi's sarcoma.

Valendar Turner: I think this is another point that needs to be emphasised from the clinical point of view. That is, if you take the gonococcus which causes gonorrhoea, it causes urethral discharge and cervicitis in those it affects, but it also has other spectrums. It can cause arthritis, it can cause meningitis, it can cause myocarditis, but

these diseases occur in all people who get them. The people who get it in Sydney get the same diseases, the same spectrum, as people who get it in Perth. It would be a very curious pathogen that actually had a preference for one part of Australia or one city. And yet, HIV, which ties up a number of diseases in fact shows preference for which continent it's in, because it's different in Africa from it is in Europe and the Americas... in America, in North America...

Eleni Papadopulos-Eleopulos: In fact, there is no relationship, hardly any relationship, between what is called AIDS in Africa and what is called AIDS in gay men in Australia or in the United States or Europe.

Stuart Reid: What sort of differences are there?

Eleni Papadopulos-Eleopulos: For example, the main and the most specific, shall we say...

Valendar Turner: Just to carry this statement on, not only does it show continental preferences, it shows preferences for which group you're in, as Eleni has said. This is an important point, that if you're a gay man, you get a different subset of diseases from if you're a haemophiliac, for example. Haemophiliacs almost never get Kaposi's sarcoma, so the epidemiology which is often thrown up as showing that HIV is the cause of AIDS in fact is inconsistent, it's unusual.

Stuart Reid: Couldn't that just be that people who are haemophiliacs die before they get Kaposi's sarcoma?

Eleni Papadopulos-Eleopulos: No, no, no, because, in fact, of all the AIDS risk groups who test positive, haemophiliacs live the longest.

Stuart Reid: My other point (Inaudible).

Eleni Papadopulos-Eleopulos: They live the longest. In fact, the rate of AIDS in haemophiliacs is much, much lower. This is another point. Thank you for mentioning it. I mean, if HIV causes the disease, then once you are infected, the rate of disease development should be the same in all the AIDS risk groups. In fact, if anything, in haemophiliacs it should be much higher than in gay men because gay men to start with are healthy, whereas haemophiliacs are born with a disease, they are abnormal, at least in one respect, compared to gay men. Yet AIDS in haemophiliacs, the rate is much lower than the gay men.

But again about... because you ask me, you know, in what respect it differs... again, because we start with Africa. As I said, the more specific and principal clinical syndrome or diseases of AIDS in gay men is PCP and Kaposi's sarcoma. Now, pneumocystis carinni is a ubiquitous organism, it exists everywhere in all continents. Yet, African AIDS patients never develop pneumocystis carinni pneumonia. They do have Kaposi's sarcoma but Kaposi's sarcoma existed in Africa for ever, and in fact...

Valendar Turner: Kaposi's is interesting because it's recorded in one of the ancient Egyptian papyruses, the Ebers papyrus which dates to 3,500 years ago, long before the time of Christ. So we can say with certainty about Kaposi's sarcoma that it did antedate in Africa AIDS by a long time.

Eleni Papadopulos-Eleopulos: Not only that, but it was very extensively studied in the 1950s and sixties because it was... like, in gay men now, there it affected young individuals, it was very aggressive. But all the studies... and they were looking really... they were looking to find out an infectious agent, to correlate Kaposi's sarcoma with an infectious agent. In fact, that was the reason that they went... it was done by non-African doctors and they still could not. The conclusion was that Kaposi's sarcoma is not caused by an infectious agent in Africa but by some either environmental or other agent. We don't know what.

Valendar Turner: And in fact in that regard, it's interesting to note that the official view now from the CDC in America is that Kaposi's sarcoma in gay men is no longer caused by HIV.

Eleni Papadopulos-Eleopulos: Not caused by HIV, is not associated. Not only is not caused... it's not indirectly caused by HIV, but it is not associated with HIV. In fact, they had to admit that because in the other AIDS risk groups, Kaposi's sarcoma doesn't exist.

Valendar Turner: So it's almost come full circle. Kaposi's sarcoma was the reason the HIV hypothesis was originally put forward, and now it's dissociated. So one wonders whether those gay men who only have Kaposi's sarcoma as one of their AIDS diseases should be declassified.

Eleni Papadopulos-Eleopulos: But to me as a physicist, in fact that was one of the... we wrote a lot of papers on Kaposi's sarcoma, and to me the fact that the HIV hypothesis was put forward to explain Kaposi's sarcoma, and its prediction is admitted now to be erroneous, is sufficient to question the HIV hypothesis as a whole.

Stuart Reid: Just looking at that situation of there being Kaposi's sarcoma in Africa, wouldn't the alternative hypothesis regarding that though be that AIDS was around in Africa and was indicated by Kaposi's sarcoma?

Eleni Papadopulos-Eleopulos: No, because as... there are many reasons of that. First of all, Kaposi's sarcoma existed forever and if Kaposi's sarcoma was caused by HIV, now... and it was concluded it was not an infectious agent, doesn't matter what, but if it was... let's assume it was caused by HIV, then why was Kaposi's sarcoma and thus AIDS and HIV restricted for so many decades in the African continent when, you know, there was plenty of relationship... or how do I say, there was plenty of travel or people being...

Valendar Turner: There was the slave trade; that's plenty of travel.

Eleni Papadopulos-Eleopulos: No, I mean, you know, in the... say the fifties and sixties, you know.

Valendar Turner: I see. You mean in recent times?

Eleni Papadopulos-Eleopulos: In recent times. You know, there was plenty of opportunities for the virus to travel to Europe and to America.

Valendar Turner: And also if this is the case, that Kaposi's sarcoma in Africa has always been AIDS, why does Africa have a population problem?

Eleni Papadopulos-Eleopulos: Yes, exactly, you know, because if AIDS existed there, you know, for so many years in AIDS and HIV, and if AIDS is such a...

Valendar Turner: In fact, in Africa it is said that the incubation period of AIDS is actually four years versus ten years in the west, so even more so.

Eleni Papadopulos-Eleopulos: We should not have left one African by now, we should not have had one African, but, you know, it's not. In fact, there are so many hypotheses, many people including Gallo... I think Gallo was the first one to hypothesise that HIV somehow appeared in Africa. Nobody went to say that HIV was present there as far back as the 1940s and fifties or sixties even. And secondly, now many people are trying to... they change their mind and we don't believe any more that HIV originated in Africa.

Valendar Turner: The other important thing is that modern studies where tests have been done on Africans, because it's a study because tests aren't usually done in Africa, have clearly shown that if you take the WHO clinical case definitions, only about half of the people who fulfil that definition of AIDS are in fact HIV positive.

Eleni Papadopulos-Eleopulos: Not even half... in Zaire 27 per cent of women who fulfil the WHO definition test positive for HIV.

Valendar Turner: So what have the others got? As one of the commentators has said, is this AIDS by definition? You tell us.

Eleni Papadopulos-Eleopulos: The thing is... as I said, pneumocystis (Inaudible) is the best example, you know. It is everywhere. If anybody, Africans should have the pneumocystis pneumonia and yet there are not African AIDS patients with pneumocystis pneumonia. What we have in Africa is diseases which existed forever in Africa. There is nothing different today than it was a hundred years ago.

Valendar Turner: And the only difference is they just happen to have antibodies, some Africans, against the proteins in the western blot strips.

Eleni Papadopulos-Eleopulos: That is not new. They have had that forever but we do not know; we are testing them now. Again, Gallo, when he did the Uganda study in 1973, he had some blood and they tested that and they count 67 per cent of children tested positive for HIV, western blot positive.

Valendar Turner: And he was puzzled by that.

Eleni Papadopulos-Eleopulos: And he was puzzled by that and he said, "Why then we don't have AIDS"?

Stuart Reid: Could we turn now to the actual mechanism of oxidative stress in AIDS. How is it that oxidative stress actually leads to AIDS?

Valendar Turner: The answer to that is: we don't know specifically how each episode of oxidative stress in a particular individual causes a particular disease.

Eleni Papadopulos-Eleopulos: But we know that oxidative stress will lead to pathology. I tried in my theory because I thought that there is not an infectious agent which causes the AIDS. I had a big problem because, say, by 1983 AIDS was diagnosed in gay men, in haemophiliacs, in drug users, so if it was not an infectious agent, how could all these groups have... which was assumed then and even I do not know the diseases are different... it can be caused by... what would be the cause, what would be the relationship between all this... what would be the common denominator in all these groups? And the conclusion which I came to was that they all must be subjected to oxidising agents by either a common oxidising agent or different oxidising agents.

Stuart Reid: What sorts of oxidising agents?

Valendar Turner: If you look at the risk groups, the obvious choices are... in the gay men is semen or some constituent of semen, sperm or cellular material or something contained in semen.

Stuart Reid: Is there evidence that there is oxidising material in semen?

Eleni Papadopulos-Eleopulos: Oh yes, that is everywhere.

Valendar Turner: In fact, yes, we could go on about that, but the short answer is yes, there's plenty of evidence. The use of certain drugs, at least in the early days of AIDS, by the gay community was prevalent. The use of nitrites in particular which we've described particularly in our paper on Kaposi's sarcoma was practically ubiquitous, and these are certainly potent oxidants. I mean, that's how they work.

That's the standard pharmacology, chemistry. Of course, so in this group there are two contenders. As far as haemophilia is concerned...

Eleni Papadopulos-Eleopulos: It's factor 8 itself. Factor 8 and most probably the proteins which are there. The way factor 8 is made, the other proteins will be oxidised as well. But, again, for factor 8 to produce its biological effect, you have to make it a strong oxidant. If you reduce it, its biological potency is destroyed. They have that and in addition they have many... then we have the drug users. The drugs they are using again are not only nitrates, because some of them all use nitrates but even the drugs which are used for intravenous injections, they're again oxidising agents.

And on top of all this, the three AIDS risk groups are subjected to many viruses and infectious agents which again when in the body... the only way to survive, they have to take reducing equivalents from the body for them to survive. So in the body they will oxidise. All these viruses which these people... you know, the gay community have a lot of infectious diseases - suffer from a lot of infectious diseases. And in fact they are treated then for these infectious diseases again with oxidising agents. So you build up a lot of factors which maybe each separate will have no effect, but when they all come together, they add up, you know, they'll have a synergistic effect which will culminate in pathogenesis.

Valendar Turner: I mean, when you get to specifics, it's difficult, as I said initially, to predict which person is going to get what disease, but we know that AIDS patients have reduced cellular and plasma thiols, that's the sulfhydryl groups - which is an indicator of the oxidation/reduction status of the body. It's known that certainly innate lymphocyte functions which are measured by immunologists and which they refer to as ... in immune terms are impaired by the cell being oxidised, and that these can be reversed by giving the opposite of an oxidation reagent, that is a reducing agent.

Eleni Papadopulos-Eleopulos: Not only the function of the T cells but even their survival depends on the redox state. If they're relatively oxidised, the function will be diminished. If you oxidise them even more, they will die. They can't survive if they are strongly oxidised.

Valendar Turner: The other factor in the equation of course is that... I shall preface that by saying that therefore this theory that Eleni expounded in the early 1980s is not an infectious theory of AIDS; it is a toxic theory of AIDS. And as with all toxic substances, what matters is firstly the nature, but secondly the dose, and one would find support for this hypothesis by exploring in one group whether the dose of whatever oxidant we're dealing with is somehow related to the development of the AIDS phenomena, the AIDS diseases, for example, and there's certainly support for this in the gay community.

Eleni Papadopulos-Eleopulos: Yes, I think this is a very good point. It's not homosexuality as such which will lead to AIDS, even if there is proof that semen contributes to the development of AIDS or causes AIDS or nitrates contribute or cause

AIDS or all the other agents act synergistically. Say, for example, that only semen and nothing else, you can exclude everything else, that semen is the cause. It is not being gay men who are practising homosexuality which will be, shall we say, the cause or which will lead to the development of AIDS.

Valendar Turner: Any situation where people are exposed to a large amount of sperm, including women. Women who practise anal intercourse, for example, we would postulate would have a higher than normal incidence of the AIDS diseases.

Eleni Papadopulos-Eleopulos: But not even who practise anal intercourse - this is a hypothesis - but women who are promiscuous, it is a known fact - this is not a hypothesis - have a much higher frequency of cervical cancer which has been shown to be related to the dose of semen.

Stuart Reid: I mean, being promiscuous isn't necessarily related to the amount of semen, to the dose, is it? Someone can be promiscuous by having ten partners over ten years or twenty partners over ten years as opposed to one partner, but one partner can give, you know... having sex more frequently would give more semen.

Eleni Papadopulos-Eleopulos: Sure. It's not the frequency or not the number of partners you have. In fact, there is good evidence that for AIDS from Amsterdam, Professor (Inaudible) and his colleagues, they have shown that there is a direct relationship between the number of episodes of passive anal intercourse and the development of HIV seropositivity and the development of AIDS. The same has been shown by the MAC study, that is the multi-centre AIDS cohort study in America. So it's not really the number of partners you have but the number of...

Valendar Turner: The dose of the oxidant.

Eleni Papadopulos-Eleopulos: The dose, shall we say, of... it's not promiscuity, it's...

Stuart Reid: But that's not the case with the cervical cancer you were talking about... using as a parallel though, is it?

Eleni Papadopulos-Eleopulos: Yes, it is, because, you know, shall we say, the nuns... nobody says that nuns... you know, there are no nuns who develop cancer... there are nuns who may develop cervical cancer but women like, shall we say, prostitutes, they develop a much higher frequency. Now, there you have two things. This is the easy way to find out because most probably some woman, you know, on the street, you know, who is not promiscuous, who is only with her husband, maybe she develops it because of that, but we don't know because of the other causes of cancer.

Valendar Turner: Unfortunately the word "promiscuous" has changed meanings, hasn't it? It's supposed to mean having a diverse number of partners. Obviously there's an association between promiscuity and dose of semen, but it's not a linear

relationship, I suspect. We need to be specific. We need to talk about the amount of semen. Promiscuity is a loose association. In fact, at this stage it's probably interesting to speculate how one can almost disprove the HIV/AIDS hypothesis with the data on passive anal intercourse.

Eleni Papadopulos-Eleopulos: Yes, sure. Some of the best evidence against HIV in fact comes from epidemiological studies in gay men. From the beginning, from the very beginning, before 1983 when HIV was said to be isolated, there was evidence that the only sexual act directly related to the appearance of AIDS in gay men was passive anal intercourse. In fact, even in 1984 Gallo presented evidence that... in fact I just have it here. You could read it.

Valendar Turner: Yes. In fact Gallo himself wrote in 1984, and I quote:

Of eight different sex acts, seropositivity correlated only with receptive anal intercourse.

That was in the *Lancet* in September 1984, written by Goerdhart et al.

Eleni Papadopulos-Eleopulos: Goerdhart was the principal author. Now, since then the data... in fact the best...

Valendar Turner: Shall we just develop that, because if receptive anal intercourse...

Eleni Papadopulos-Eleopulos: No, but let's see. There is much ... since then data from everywhere appeared which supports exactly... similar data appeared everywhere. The best will be from Amsterdam where they have in fact two very, very good studies which have shown exactly the same thing, and the multi centre AIDS cohort study in America where they have shown that the only sexual act directly related to the development of AIDS is passive anal intercourse.

Valendar Turner: Is that AIDS or seropositivity or both?

Eleni Papadopulos-Eleopulos: No, sorry, seropositivity... to development of seropositivity, and recently they have also shown that the only sexual act directly related to the progression of AIDS is again passive anal intercourse.

Stuart Reid: That could also be compatible with an infectious theory, given that that is a passage into the bloodstream that is...

Eleni Papadopulos-Eleopulos: No, it can't be, sorry, because, say the heterosexual... because we are told, you know, that HIV and thus AIDS is sexually transmitted. Now, we know... with gay men it's different because some gay men practise both passive and active sexual acts, but with the heterosexuals it's always... the woman is always passive and the man is always active. So if it is only the passive partner who develops the disease or who becomes HIV seropositive and who develops a disease...

Valendar Turner: And whose disease progresses.

Eleni Papadopulos-Eleopulos: And whose disease progresses, how we can... in this partner the woman cannot transmit it to the man.

Stuart Reid: But it's a physical difference with the walls of the vagina as against the walls of the rectum. You are actually physically getting the virus transferring through the...

Eleni Papadopulos-Eleopulos: No, we are not talking about how the virus is transmitted. We are talking that here it is only the passive person... you know, doesn't matter if it's vaginal or it is anal intercourse, right?

Stuart Reid: I see, yes.

Eleni Papadopulos-Eleopulos: So it is only the passive person, even if the woman practises anal intercourse which many women do. This is not a practice which is a sexual act which is practised only by gay men. There is evidence now that a significant percentage of women practise anal intercourse, so it's nothing new. We can't condemn gay men for that. But it is only the woman can get infected and can get a disease. The men never can get infected, never can get diseases, so it is like pregnancy: only women can get pregnant. The men cannot get pregnant.

Valendar Turner: And it can't be transmitted.

Eleni Papadopulos-Eleopulos: Cannot be transmitted sexually.

Valendar Turner: It can be acquired by the passive partner but how does the active partner get it?

Stuart Reid: So are you saying that there are no cases at all of people who have only been the active partner getting AIDS?

Valendar Turner: There's no scientific proof of this but we've had discussions with...

Eleni Papadopulos-Eleopulos: No, the only way to prove that is to have, you know, these studies. There are many reports where they say, you know, here it is one person and another, their case is reported as being due. Let's have one example about that. In New York, I mean, AIDS first was diagnosed there and one of the aims... I think it's 1991. By 1981 they had only eleven cases of AIDS which was heterosexually acquired, which was claimed to be heterosexually acquired. Now, that really I think is sufficient to tell us that it can't be.

Stuart Reid: Sorry, was that 1981?

Eleni Papadopulos-Eleopulos: I think it was 1991, sorry.

Valendar Turner: No, it was about 1990, 1991.

Eleni Papadopulos-Eleopulos: In fact I was looking today. I wanted to write it down and I can't find the paper, but it was ten years after AIDS was first diagnosed in the city. The cumulative number of AIDS cases which was claimed to have been acquired by heterosexual AIDS by men was eleven.

Stuart Reid: Anal intercourse has been around forever or for as long as we know. Why is it then that the AIDS condition didn't appear in homosexual men in large numbers until the eighties?

Eleni Papadopulos-Eleopulos: Not only homosexuals. As we said, you know, this is not only homosexual practice, you know. It is practised by everybody. We don't know what it was before.

Valendar Turner: We don't know what happened before. We don't know that gay men didn't get these diseases before. We don't know... all we know is that they got it in 1981.

Eleni Papadopulos-Eleopulos: We do know that there are much higher frequencies now for sure, that is certain, that they have much higher frequencies now that they...

Stuart Reid: Kaposi's sarcoma was a very rare disease before which only applied to... only really affected very old people.

Valendar Turner: That's not true. There's plenty of data to show that there are cases... in fact one estimate is 20 per cent of cases of Kaposi's sarcoma diagnosed in Europe and in America from 1900 till the beginning of the AIDS era would have been classified as AIDS had AIDS definition been around then. So no, we reject that assertion that it didn't happen. What we didn't know about, of course, pre-AIDS, was the number of gay men who developed these diseases. We probably don't even really know how many of the diseases developed because we're looking harder now. We know a little bit but we certainly don't know whether many were in gay men because being gay was something that was not broadcast before the late 1970s, eighties. It was a social change in the way... people's behaviour, if you like. People were no longer closeted.

Eleni Papadopulos-Eleopulos: But with Kaposi's sarcoma, you know, this is a malignancy which is... people have nearly normal life span. Even in gay men now, gay men with Kaposi's sarcoma live longer than gay men with infectious diseases, with opportunistic infections. Even in 1981-82, both the patient and the doctor had problem diagnosing Kaposi's sarcoma because, you know, they were thinking...

Valendar Turner: The true incidence of Kaposi's sarcoma pre-AIDS and the true incidence in gay men is totally unknown, so we're looking at evidence of absence. It's not evidence of... how does the saying go? Absence of evidence is not evidence of absence. But even we would agree that there was an increase in these cases because there were so many of them, that they could not be hidden, even if people were trying to hide them. So we would agree that there was an increase. Whatever the level was before in gay men, it was a lot higher in the 1980s. And you want to know how does one explain this?

Eleni Papadopulos-Eleopulos: Could be, you know, because of higher sexual activity. Another reason could be that, you know, in these cities they became... gay men from everywhere round the world were going to America.

Valendar Turner: To meet each other.

Eleni Papadopulos-Eleopulos: To meet each other. It was there where they had the freedom to express themselves because, you know, in Europe it was not; certainly, in Asia they could not.

Valendar Turner: And in Africa they certainly couldn't.

Eleni Papadopulos-Eleopulos: And in Africa they could not. So, you know, it is a combination of many factors which led to the true and apparent increase in these diseases in the gay community.

Valendar Turner: For which there was little prior knowledge - epidemiological knowledge.

Eleni Papadopulos-Eleopulos: Does that make sense or does not? Please ask.

Stuart Reid: I still have trouble, I guess, coming to terms with the idea that something as noticeable as AIDS, something as observable as AIDS, was not picked up beforehand when there had been communities of gay people in areas like San Francisco, for example, for a lot longer than just the eighties or the seventies and eighties.

Valendar Turner: We don't really know; we're guessing.

Eleni Papadopulos-Eleopulos: We don't know but, you know, Johnson and... what's their name, who studied...

Stuart Reid: Masters and Johnson.

Eleni Papadopulos-Eleopulos: Masters and Johnson. They say, you know, that really the practice of passive anal intercourse, you know, not only of gay people, but the practice of passive anal intercourse, it really increased much more in the seventies.

As I said, the drugs, they're there too. We cannot avoid two significant changes. I'll say three because... most people say two but I'll add the third one. The first is, you know, that for the first time gay men had reasons to be happy because they were not any more suppressed the way they used to be before, and when you have freedom you try to take opportunity of it. Then they had... unfortunately drugs came into the scene, which they never had them before, and then the third is - my addition is - that they came together. Gay men from everywhere were travelling to San Francisco or to Los Angeles and to New York, so all things together, to me, at least to me, make sense. I don't know if they, you know, make sense to others, but to me...

Valendar Turner: I don't think you can... I mean, you can't make any sense out of this without at least mentioning the fact that there is evidence that gay men at this period of history were exposed to high volumes of semen. Now, this is not meant to state that they were exposed necessarily to any more semen than any other groups practising sexual relations, but in absolute terms what counts is how much semen, in our theory, at least of our theory, our toxic theory, is how much. What the dose was. One doesn't have any basis for comparison really from previous times, but there is evidence that gay men were having many partners, sometimes up to a thousand a year in some cases. This would seem to indicate an unusually high exposure to semen; one could postulate more possibly than the human body had been exposed to in times past - not on an individual basis of course, but en masse, as Eleni has alluded to; the fraternity, if you like, the fellowship of being gay.

I mean, this is no reflection on gay people or the gay movement. We're only interested in the scientific issues and if it turns out that in fact exposure to large volumes of semen is a factor in inducing the oxidative stress which we postulate is a major etiological factor in the development of these diseases, then so be it. I mean, one cannot walk away from this.

Eleni Papadopulos-Eleopulos: But, you know, if it is proven... because this is another thing with the theory. All right, the theory is not proven but one thing which has been proven is that AIDS patients are oxidised. That is something which nobody can deny.

Stuart Reid: How is that determined?

Eleni Papadopulos-Eleopulos: It has been determined by measuring the cellular thiols.

Valendar Turner: By taking blood or cells and working out how much reducing substance is in them.

Eleni Papadopulos-Eleopulos: Right. The other thing which cannot be denied... our theory postulates that the phenomena which are called HIV are induced by oxidative stress. That was postulated in... from the very beginning once I came in contact... in 1984 I postulated that the phenomena which are called HIV are induced by oxidative

stress. Now today, again there is ample proof that HIV can be induced by oxidative stress in the petri dish. You put oxidative stress or substances or radiation which produces oxidative stress, you have HIV.

Valendar Turner: We didn't stress this at the beginning. We mentioned that the oxidative stress is capable of inducing pathology but we didn't say that in fact it also induces the very phenomena which people interpret metaphorically, if I may use that term again, as HIV. In fact, the theory predicts this and it's basic standard retrovirology. It's not as new as it sounds. It's knowledge which has been gained since 1911.

Eleni Papadopulos-Eleopulos: Only then they were not called... you know, they did not know that really all these substances... they knew that if you put radiation, if you put carcinogens, you'll induce retroviruses. What they... nobody apart from me, may I say so ...

Valendar Turner: She said modestly - modestly...

Eleni Papadopulos-Eleopulos: They did not see the relationship between all these substances which... and I claim... I don't say that I see but I claim that the relationship is oxidative stress. Now, what we know now... now it is called that and we know - there is ample evidence - that if you put oxidising substances or radiation or you radiate your petri dish, you produce the HIV phenomena. On the other hand, if you put reducing agents, you'll inhibit the HIV phenomena. So really what is important today to me, as far as I'm concerned, is even if we disagree on what causes AIDS, you know, with most people, when it comes to the principal and the most important fact, that is, how can we prevent and how can we treat AIDS, I think the evidence from our quarter and - call it the opposition. I don't call them opposition, but...

Valendar Turner: The HIV/AIDS protagonists.

Eleni Papadopulos-Eleopulos: The HIV theory and their HIV theory. We have to admit the evidence from... you know, the experimental evidence from there and the theoretical evidence from here all point to one thing: that HIV, in inverted commas, and AIDS can be prevented and reversed or treated with reducing agents. And I think at this point we all have to agree.

Valendar Turner: And when you encompass the HIV phenomena with the pathology, the theory has the attractive quality of being economical and explaining many things with one simple statement which we all seem to believe is one of the properties of a good theory. The other important thing is that surely there is now enough evidence around against HIV as being the theory of AIDS to make it mandatory as a public health issue, if nothing else, for this to be discussed and debated in proper scientific terms by scientists in open forum.

Eleni Papadopulos-Eleopulos: Apropos of public health issue, now, people are...

Stuart Reid: Can we move on to the public health issue on the next tape, please.

Eleni Papadopulos-Eleopulos: Apropos of the public health consequences of our theory and of the HIV theory, usually when we talk to people about our non-HIV theory of AIDS, people get a little bit upset and usually without knowing what we are saying, they say, "Oh, you're one of the Duesberg mob". That is Peter Duesberg who, like us, thinks that HIV does not cause AIDS, but there is a big difference between Peter Duesberg and us. In fact, Peter Duesberg... we came at about the same time, at exactly the same time, to this conclusion but from completely different points of view.

There are many differences between Peter and us, one of which was at the beginning he did not say what was the cause of AIDS and now he says that the cause of AIDS in all the AIDS risk groups is drugs and that as far as safe sex is concerned, he is of the opinion that there is no need to worry about condoms, just to put it... we are not of the same view. In fact we think safe sex is of extreme importance. That coincides with the HIV theory of AIDS as far as public health is concerned and, again, as far as drug users are concerned, in fact we go one step further: we not only say that needles should not be shared, we say that needles should not be used or even oral drugs because in our view it is the drugs which cause the abnormalities and not needle sharing. Also needle sharing could have an additional effect by transmitting infections such as hepatitis B and... I don't know what else.

Valendar Turner: Isn't it also important to stress the differences in perspective between Duesberg and us as far as HIV is concerned?

Eleni Papadopulos-Eleopulos: Peter does believe that HIV exists. He believes that the HIV antibody test, a positive HIV antibody test proves HIV infection but he says that if you test people who... for example, if you take haemophiliacs who test positive and those who test negative and you follow them up, you'll find out that both groups will develop AIDS with nearly equal frequency. We do not agree with that.

Valendar Turner: This is not a study, this is a postulate. Peter Duesberg has postulated that to prove or disprove the HIV theory, one way would be to conduct a study of two groups of haemophiliacs differing only in their seropositivity in their serostatus, that is with HIV positive or negative, follow them up for a reasonable period of time, several years, and see what happens as far as AIDS disease incidence occurs. He predicts that both groups will develop AIDS at the same rate.

We do not share that view. Because we believe that HIV antibodies, whatever that may mean, is a non-specific marker for the propensity of developing AIDS, we predict that in fact the haemophiliacs who are positive will develop AIDS at a greater rate. And thus this proposed study by Duesberg will not prove or disprove the HIV/AIDS hypothesis.

Eleni Papadopulos-Eleopulos: Really, from the public health point of view, we are different from Peter and we are very much in agreement with the HIV theory.

Valendar Turner: Except we probably wouldn't recommend mass testing of people for HIV antibodies for any reasons.

Eleni Papadopulos-Eleopulos: That's for sure. In fact, maybe there are much better markers than HIV antibodies for the development of AIDS in the AIDS risk groups.

Valendar Turner: There are. Not, "We believe..." There are better markers.

Eleni Papadopulos-Eleopulos: We do believe there are better markers, you know, and more standardised better tests, more specific, more reproducible, more everything, easier to do.

Stuart Reid: What sorts of things are you talking about here?

Eleni Papadopulos-Eleopulos: For example, in haemophiliacs it is known that, you know, 98 per cent of haemophiliacs who develop AIDS test positive for hepatitis B. This, you know, is an easier test to do. I don't know about expense but most probably it's less expensive.

Valendar Turner: There's also the breast cancer...

Eleni Papadopulos-Eleopulos: It won't be really that... psychologically it won't sound that bad than when you're told that you have a positive HIV test.

Stuart Reid: We've looked at possible ways of oxidative stress being the cause for drug addicts, for gay men and for haemophiliacs. What about people who have got AIDS through blood transfusion?

Eleni Papadopulos-Eleopulos: First of all the number of people who get AIDS from blood transfusion is very limited. The people who get blood transfusion... the fact that they get blood transfusion means that they are sick, to begin with. The people who get blood transfusion hardly ever develop Kaposi's sarcoma but they usually develop opportunistic infection and these opportunistic infections are nothing new. So, really, if somebody has the time and the money to conduct a study in people who test positive and people who do not test positive, it will find out that maybe... or if we look for these diseases before AIDS, maybe we'll find out that the frequency is...

Stuart Reid: They get the opportunistic infections but the key point is that something has affected their immune system to make it so they cannot compete with those infections.

Eleni Papadopulos-Eleopulos: The disease they have, the drugs they had... because as I said, the fact that they have this blood transfusion, they are sick. They are treated for this. In America I think 20 per cent of people who get blood transfusion get blood transfusion related to cancer. Now, people who have cancer... first of all cancer will

reduce your ability to fight infectious diseases. The chemotherapy and the radiotherapy, they are both known to induce opportunistic infection.

Valendar Turner: In fact, in the AIDS definitions within arbitrary time limits, you're excluded, or used to be excluded, as an AIDS case because you'd had something which was known to induce the same conditions. The whole problem with transfusion is one does not know what happened pre-AIDS. Don't forget that all these AIDS diseases are... none of them are new. You would expect them... these diseases occur every so often in people who are otherwise healthy. I mean, I've seen it myself.

Eleni Papadopulos-Eleopulos: In fact the first person who has been reported... it was a child who has been reported as having AIDS due to transfusion... if one goes and looks close... in fact the (Inaudible) I think to say that we do not know if this child has AIDS because of the blood transfusion or because he was premature and he really was prone to develop AIDS. In fact he did not have AIDS. Clinically he did not have AIDS.

Stuart Reid: What case are we talking about now?

Eleni Papadopulos-Eleopulos: It's the first case which was reported of HIV and AIDS acquired through a blood transfusion.

Valendar Turner: You see, one of the problems here is that apart from the fact you don't know what the statistics were before AIDS came along, you don't... we all know of people who say that they know someone who knows a case of AIDS that occurred after blood transfusion, but when you try and get the details, they're often very dubious. They're clouded in obscurity. And given that the disease has occurred before anyhow, that a lot of people who get given blood transfusions are, as Eleni says, sick, subject to other forms of therapy which themselves can cause these diseases, and given the fact that AIDS has consumed many diseases for itself by definition, one has to ask the question: is there anything different apart from HIV, serostatus if you like, about blood transfusion recipients than there ever was ever before? We don't know. We doubt it.

Eleni Papadopulos-Eleopulos: No, not only we doubt it, I don't think it's new, I don't think it's anything new.

Stuart Reid: But we would have expected to see the same pattern in blood transfusion patients after screening of blood if there wasn't the problem?

Eleni Papadopulos-Eleopulos: Yes, there is evidence for people who have received blood which was screened to be negative, the recipient developed AIDS and seroconverted and the donors remained for years sero negative and healthy, and this is CDC data.

Stuart Reid: But that would... you couldn't tell... unless you knew what other risk factors those people were exposed to, those specific cases wouldn't necessarily help you?

Eleni Papadopulos-Eleopulos: No, this is CDC data and they excluded every other single risk factor. In fact they said, there it is, we have, you know...

Stuart Reid: What about the cases of the famous AIDS babies in Queensland where they were able to trace the source of the blood to someone who also became sick with AIDS?

Valendar Turner: We don't know these cases. We can only comment if we saw the data. This is one of the problems, finding out the data, finding out what really happened. I mean, people put these cases up as if finding a case like this proves the whole HIV/AIDS hypothesis. I can tell you of cases where a man and a woman are married and live together and one gets melanoma and the other one gets melanoma. Now, does that prove that melanoma's transmitted? You cannot use these cases to prove your hypothesis. Hypothesis can only be proved really by injecting pure HIV into human beings and see what happens. I mean, obviously it's unethical to do that but, I mean, you have to... at some stage we have to agree what criteria we will accept as proof.

Eleni Papadopulos-Eleopulos: Apropos of that, when Montagnier first isolated HIV from haemophiliacs, he said... commenting, he said the only way to prove that HIV is the cause of AIDS is to have an animal model. Even today, ten years after, we have not got an animal model.

Valendar Turner: But they do... there are... they've infected chimpanzees that cost, what, $50,000 each and they're all healthy.

Eleni Papadopulos-Eleopulos: Doesn't matter how much they cost. They have injected them long, long ago and they're still healthy. There is no animal model available.

Valendar Turner: This isn't necessarily an argument that proves HIV can't be the cause of AIDS, not at all, but I mean, at some stage if we were debating this with a scientist, we would have to agree on what we would accept as being a chain of events that would be a causal link, and you certainly can't use one case of someone getting AIDS from a blood transfusion, given all the other constraints, plus the fact that 85 per cent of cases reported to the CDC only are actually subsequently shown to be AIDS. 15 per cent are over diagnosed. I mean, that surely must apply on average to blood transfusion recipients as well. We suspect that there is not really... if there's an increase in the number of cases of AIDS, in inverted commas, in blood transfusion recipients, it's probably due to the fact that people are looking for it or due to the test.

Stuart Reid: My understanding of the data, and I haven't got comprehensive... I haven't got detailed data to put to you, but my understanding of the data is that there was a short period of time when people whose only risk factor was blood transfusion became sick with AIDS or became HIV positive, and once the testing was introduced, then that number has now dropped to zero, dropped back to zero.

Eleni Papadopulos-Eleopulos: But today nobody tests transfused patients. They tested them when they thought it was due to it and now nobody tests transfused patients.

Stuart Reid: But if someone gets sick or shows any symptoms or anything like that, then they are tested and at that stage...

Eleni Papadopulos-Eleopulos: No, they are not.

Stuart Reid: I thought they would be exposed to testing through giving blood themselves.

Eleni Papadopulos-Eleopulos: If somebody gets blood transfusion and develops septicaemia or develops tuberculosis, even if he dies from septicaemia or from tuberculosis, they will be just people who have died from TB or have died from septicaemia. They are not going to be AIDS patients.

Stuart Reid: If someone was dying from tuberculosis in this hospital, would they be tested for AIDS?

Valendar Turner: Probably not. The problem... I say probably because CDC have recently added pulmonary tuberculosis, for reasons unknown, to their list of AIDS indicator diseases. So up until the beginning of this year, I can confidently say no, and I know from talking to my colleagues here that patients who are not dying of pulmonary tuberculosis but who could be very sick are not tested for HIV, not at all.

Eleni Papadopulos-Eleopulos: Or even for septicaemia, even dying from septicaemia which, you know...

Valendar Turner: And recurrent pneumonia is an AIDS defining disease. I mean, we see people who have pneumonia twice in a year. They don't get tested for HIV. I mean, it's a very biased testing, very biased indeed. And don't forget at the background of all this is this problem of it being a non-specific marker. It might be that all people who are destined to die become HIV positive. It might be a marker for impending death from many causes, like wrinkles or superannuation problems. You may laugh but, I mean, in logic there is no reason why it shouldn't be. We only think of it as a virus because we've been programmed to accept the fact that this must be the case.

Eleni Papadopulos-Eleopulos: In fact really, you know, this is I think a very good point because the only relationship which... or the only evidence we have or is presented as being... the only data which is presented as supporting the HIV theory of AIDS is the relationship between the antibody tests and AIDS. There is no other evidence.

Valendar Turner: Yes, that's right.

Stuart Reid: What you are saying is that that test is actually a truism?

Eleni Papadopulos-Eleopulos: Exactly.

Valendar Turner: It's a tautology. It's a loop. Self-referencing.

Eleni Papadopulos-Eleopulos: In fact that is why we decided to really look, to have a close look at the antibody tests.

Valendar Turner: See, initially we... as I was saying the other day before the interview, we decided that we didn't get anywhere by knocking HIV. The responses of the referees were mainly pejorative remarks about us from one of the medical journals we submitted it to. As I said to you before, we postulate this to be because of the attractiveness of the germ theory of disease, because, you know, it's nice and people will have a little agent which causes disease. If you get rid of that agent, you get rid of the disease. So we decided, well, everyone understands the tests. The man in the street understands the implications of the test. The clinical doctors... you know, the general practitioners and specialists who treat these patients relate well to the test. Let's look at the test. Let's see if the tests really mean what they say they mean, and we produced this huge article about the tests which we've referred to.

So every time we get away from that and forget that we're actually talking about a whole lot of non-specific stuff, then we're really not entitled to talk about HIV. Scientifically we're not allowed to talk about it. It's best said as... every time you say to us, what about people who are infected with HIV or have AIDS, you're talking about people who have antibodies to certain proteins which are stuck in western blot strips. That's what you're really saying. You're not entitled to say any more than that because there is no proof of any more than that.

Eleni Papadopulos-Eleopulos: And you don't know what these antibodies... I mean, AIDS patients have antibodies to everything you care to mention.

Valendar Turner: Yes, I can vouch for that because I had to type a list of the anti antibodies once in one of the papers and I got tired. I must have typed anti something thirty times. And I suspect they've got antibodies to substances which we haven't tested for. I'm sure that's the case, and antibodies to things they would never normally come into contact with showing the cross-reacting problem. So I mean, each test can't be specific when you find them in mice and...

Stuart Reid: You've called for a scientific debate about this issue. What kinds of opportunities have you had within your local medical community here in Perth to debate and discuss these issues?

Valendar Turner: Well, I think we've probably had opportunities but we haven't been terribly pushy. I mean, we could have been a lot more pushy, but that's to be quite...

Eleni Papadopulos-Eleopulos: No, we are not pushy because from... shall we say from the beginning, we are in very good relationships, shall we say, a very friendly relationship with all the immunologists and the people who look after... with John Armstrong, you know, who is considered to have been the first person to have shown HIV in AIDS patients. We're on very good terms with all of them, but from the beginning we agreed to disagree as to what causes AIDS, and we're continuing to do so.

Valendar Turner: I think we have been... I know that we've been regarded as an embarrassment to some people for our views, which is fine, but it's the fact, and it's an unpopular view. I mean, I don't know whether people think we're crazy, but...

Eleni Papadopulos-Eleopulos: We may be considered as an embarrassment and maybe it's nothing, but I never had any problems of getting help, apart from experiments. We cannot collaborate in... we did collaborate once.

Valendar Turner: We did have a little bit, but I mean, I don't know. I don't think I'm paranoid but I just think there's an unwillingness to embrace our ideas. And if there's an unwillingness, then obviously there's not enthusiasm to do the experiments. The medical superintendent of the hospital...

Eleni Papadopulos-Eleopulos: But you cannot blame them. Somehow I don't... not somehow. I don't blame them.

Valendar Turner: We understand them, that's all right. They don't believe this stuff.

Eleni Papadopulos-Eleopulos: We are putting so much work on this, on our theory, outside. I mean, we never got any money, we didn't get any help. We work a lot. We spend our weekends, our nights, our everything, doing...

Valendar Turner: And if we're right and they're wrong, if that... there's no doubt we both can't be right. I mean, there's no middle ground in this. I mean, toxic is not the same as infectious. Then we will naturally... it's... when you've got points of view which are completely opposing, it would be almost - in human nature - [impossible] for one side to help the other, in my view. The medical superintendent's been quite helpful in at least allowing us to sort of talk to journalists and newspapers and...

Eleni Papadopulos-Eleopulos: No, everybody's been... immunologists and everybody, I mean, they don't believe us but if you go to them for help... if I go and ask, you know, something, if I discuss their findings, they don't mind it at all.

Valendar Turner: One of the immunologists has just agreed to allow us to use some data from one of his patients in a paper we're writing at the moment. So I mean, they are...

Eleni Papadopulos-Eleopulos: They are very helpful and, as I said, on the other hand I don't blame them that they don't believe us because... how do you say?

Valendar Turner: They have to unbelieve themselves to believe us.

Eleni Papadopulos-Eleopulos: Not only that, not only to... no, it is to come to see what we are saying, to be able to criticise or to discuss what we are saying someone has to spend a lot of time and effort to do it.

Valendar Turner: That's true. There's a lot of prior knowledge needed on this.

Eleni Papadopulos-Eleopulos: You need so much.

Valendar Turner: But let's get on with the question. The other things we've done...

Eleni Papadopulos-Eleopulos: But this is important because, you know, you can't blame them that they don't believe us because to believe us or to criticise us, they have to do an enormous amount of work and they have not got the time.

Valendar Turner: It's probably not as important to them as it is to us because they're very busy people. I mean, we're busy people but they're busy in different ways. The other things we've done...

Eleni Papadopulos-Eleopulos: I spend my life...

Valendar Turner: Other things we've done to get noticed, we did present... about a year ago Eleni and I presented a short talk on this in Sydney at one of the college meetings, but it was to a group of people, emergency physicians who really couldn't expect to be a full bottle on this and who, quite frankly, I don't think believed a word of it. I've corresponded with a couple of people, AIDS experts within Australia, and we haven't really had much joy out of those people. I mean, I won't name them but they're well known. One person was very polite and commented on things which were sort of out of date but when pressed did not answer the specific questions which were asked.

We've done some funny things. We tried to get Elton John interested in providing money for this at one stage by writing to him, or at least to his publicists, but we didn't get anywhere with that. We've approached a few newspapers in Sydney.

Eleni Papadopulos-Eleopulos: No, we did not approach them; they approached us.

Valendar Turner: No, I approached some through my uncle who's a retired journalist. The fact that rather ironically, when the *Biotechnology* article was popularised through the London *Sunday Times* and newspapers from everywhere approached us, we were sort of tongue tied. We didn't quite know what to say because we do prefer the scientific debate rather than...

Eleni Papadopulos-Eleopulos: No, it's not that we don't know what to say and how to say it. It's that we don't want to have a debate on TV, radio or any other popular media because we think that neither side can gain by having that kind of debate, and certainly the patient cannot get... it will put the patient in a turmoil. So the only way this problem can be solved... I mean, we think there is a problem. The other HIV people do not think there is a problem, but the only way this problem can be solved is through the scientific media, through scientific journals.

Valendar Turner: Yes, and the short answer is that if enough experts or para experts, people close to the problem, people treating patients, even some patients themselves, I suppose, sense that all is not well with the HIV/AIDS hypothesis, then this debate will happen and will happen properly, but there's no way that we can get up and convince twenty experts they're wrong and change course.

Eleni Papadopulos-Eleopulos: I think the only way to solve the problem is to be able to convince the HIV people that there is something wrong.

Valendar Turner: But we have to convince them first that there might be something wrong so they debate it.

Eleni Papadopulos-Eleopulos: If the patient realises that, then it will be a big problem for everybody.

Valendar Turner: We think we've got the science part of this right, our view, but we haven't got the politics part right and we don't even know whether that's our game.

Eleni Papadopulos-Eleopulos: Certainly politics is not my game.

Valendar Turner: None of us are experienced in this sort of thing.

Eleni Papadopulos-Eleopulos: And John Papadimitriou and even you are not.

Valendar Turner: No, I'm not experienced. We'd probably get done on a television...

Eleni Papadopulos-Eleopulos: And we don't want to. I don't want to be...

Valendar Turner: We talk about writing a book but we haven't done it yet.

Stuart Reid: If there was to be the opportunity to do one set of experiments that would advance your case, what would that set of experiments be? What would you like to do to be able to firm up your case experimentally?

Valendar Turner: Well, to tell you the truth, we can't tell you the answer to that question. We know the answer to that question but we're not prepared to tell you in case someone else does the experiments before us.

Eleni Papadopulos-Eleopulos: That's not true.

Valendar Turner: That's exactly how I feel. These experiments can be very easy and if positive, they would be utterly devastating to the HIV/AIDS hypothesis, but I'm not prepared at this stage to tell you what they are, but I assure you we have them. All we need is some money, and not very much money, to do them. We're planning one. Do you want to add anything to that, Eleni? I've told the truth as I see it.

Eleni Papadopulos-Eleopulos: Experiments are not that easy. We don't need that much money, that is true. I mean...

Valendar Turner: We need some money.

Eleni Papadopulos-Eleopulos: Certainly we need some money. I mean, we are very... John and I... we're all... AIDS is not our main work. We are busy doing other things.

Valendar Turner: Earning a living.

Eleni Papadopulos-Eleopulos: Earning a living. AIDS is our... I was going to say our love; shall we say...

Valendar Turner: Obsession.

Eleni Papadopulos-Eleopulos: Obsession; is our scientific obsession, but we have not got the time and even the expertise to do the experiments. We can design them but, you know, when it comes to actually practically
doing them, I mean, for sure I haven't got the expertise. So we need money to employ people to do the experiments and to have the right equipment, to have everything. We do need money but not money... not even a very small proportion to... I mean, infinitesimally small proportion comparing to what is used today.

Valendar Turner: And this would be basic scientific research.

Valendar Turner: A little bit more about blood transfusions.

Eleni Papadopulos-Eleopulos: I discuss that in my first paper but I just can't find where I had it, because I really said... because it's a very, very... millions of people are transfused annually in America and the number of AIDS cases in transfused patients is really insignificant. As we said before, these people, you know... the reasons that they are getting the blood tell... they're getting blood because they're sick, and for sure some of the diseases, if not all, and the treatment which is used for them will induce the diseases which today are called AIDS. That has been going all the time, so we can't say that in blood transfused patients... in fact nobody will say that the clinical picture in blood transfused patients is different today than it was before the AIDS era. The only difference we have is that when these persons are tested, the ones who are... many are not, but the ones who are tested test positive for HIV.

Stuart Reid: We've been talking about AIDS and the acquired immune deficiency syndrome but we haven't really spoken about the immunodeficiency characteristic of AIDS. I'm wondering what you think about that? Does the oxidative stress account for the immunodeficiency or do you see immunodeficiency as a significant factor in all this? What is your view about immunodeficiency?

Eleni Papadopulos-Eleopulos: Immunodeficiency in AIDS really is a decreased number of T4 cells as determined by the use of antibodies, specifically raised against T4 cells. Now, the fact that when blood from an AIDS patient is tested or from a person who is at risk of developing AIDS, fewer T cells bind these antibodies is interpreted as being proof that the T4 cells in those individuals are killed - are destroyed. As far as we are concerned, the fact that fewer antibodies, or shall we say fewer cells, bind antibodies direct against T4 cells is not proof that the T4 cells are being killed.

Valendar Turner: Especially being killed by HIV.

Eleni Papadopulos-Eleopulos: By HIV or by any other agent. To us this proves that these cells do not bind the antibody and is not due to cell destruction. In fact there is evidence to show that this is the case. We still have not published that but we're going to publish it in the future.[1] That is one side. The other side is that we do not believe the T4 cells decrease... because according to the HIV theory of AIDS, HIV destroys, kills the T4 cells and the destruction of T4 cells or the decrease in T4 cells leads to the development of Kaposi's sarcoma and opportunistic infections, all different kinds of diseases which are called AIDS. In our view, T4 cells play no role in the... there is no relationship between T4 cells... put another way, there is no relationship between the T4 cell number and the development of either Kaposi's sarcoma or other neoplasms or opportunistic infections.

Valendar Turner: In fact, if we go back to the example of Kaposi's sarcoma, about the CDC view that HIV is neither directly nor indirectly the cause of Kaposi's sarcoma, some data from this study is that you can get Kaposi's sarcoma in the

[1] See Genetica Volume 95, pages 5-24 (1995)

absence of HIV and also in the absence of immune deficiency. So, as far as Kaposi's sarcoma is concerned, it is now not due to HIV and it is not always associated with immune deficiency.

Eleni Papadopulos-Eleopulos: No, it is agreed now, you know, and this is one thing which is agreed and is still the HIV theory.. nobody questions. To me that is... you know, if you do not have any other evidence, the fact that even the CDC today admits that Kaposi's sarcoma is not related either to HIV or to immune deficiency, this questions the HIV theory of AIDS. And they were forced to admit it. There are two reasons. One is that Kaposi's sarcoma was appearing only in gay men. Now, the only way to get by this is either to admit that AIDS is not caused by HIV, the different groups have different etiological agents, or to say that Kaposi's sarcoma is caused by a different agent, but HIV, never HIV, no T4s or immune deficiency plays any role. The thing is... but now today we have evidence that not only Kaposi's sarcoma but pneumocystis carinni pneumonia, the other...

Valendar Turner: The large group of indicator diseases.

Eleni Papadopulos-Eleopulos: ...can appear in the absence of HIV and T4 cell decrease in gay men.

Valendar Turner: So these are HIV negative, immune deficiency negative AIDS cases.

Eleni Papadopulos-Eleopulos: You can't say immune deficiency negative. You can call them immune deficiency negative, all right?

Valendar Turner: And that more or less wraps up AIDS for two of the diseases. I mean, not all cases but it happens. They are the exception to the theory which means the theory must be re-examined.

Eleni Papadopulos-Eleopulos: On the other hand, you can have people with chronic shortage of T4 cells, they never develop opportunistic infections.

Valendar Turner: Just summarise the evidence about the fact that there's no evidence that HIV kills T4 cells.

Eleni Papadopulos-Eleopulos: Nobody has proven either in-vitro... there is no evidence either in vitro or in-vivo that HIV kills T4 cells. Without going through all the evidence, suffice to say that in 1985 or '86, I don't know the exact date, Zagury and Gallo - Zagury is one other well known HIV expert - they have proven that you cannot get decrease in T4 cells in the petri dish unless you put another chemical which they are using and which is used in all the AIDS cultures, is called phyto haemogglectonin. * But if you have phyto haemogglectonin by itself without HIV, you'll get the same effect: you'll get a decrease in T4 cells. Now, last year - was it last year? - Montagnier's publication... they say now that the HIV kills T4 cells by a process called

apoptosis. Montagnier found out that when he has HIV and phyto haemogglectonin, PHA, he gets destruction of T4 cells.

Valendar Turner: ...by this mechanism, apoptosis. It's a morphological and biochemical description. It's a process which is well recognised.

Eleni Papadopulos-Eleopulos: It's a description of killing. Now, but if he has PHA and he says, "Incredibly". It must have been a translation from French. He said, "Incredibly, when we had only PHA and not HIV, we're still seeing the same phenomena". So, this is obvious to any scientist. If you have HIV by itself... if you have only HIV and you... HIV... now, let's admit that this HIV exists. So if you put in the petri dish HIV and you don't put PHA in, nothing happens to the cell. The cells are happily living forever after, maybe forever. They don't... you know, the cells can't live forever after, but they live... nothing happens to them. If you put PHA and HIV, you get cell destruction but if you put PHA by itself without HIV, you can still get cell destruction. So it is obvious what causes the destruction of T4 cells in the petri dish is not HIV.

Valendar Turner: The other problem with HIV and T4 cells is that there should be a demonstrable proper temporal relationship between the two. That is, the HIV theory is that you...

Eleni Papadopulos-Eleopulos: You don't have to go to... these are the experiments, you know, which...

Valendar Turner: I know, but it's just... I wanted to introduce the New York data on drug users, about...

Eleni Papadopulos-Eleopulos: Not only drug users, gay men and everybody. Now, if you go to... sorry, Val.

Valendar Turner: No, you carry on.

Eleni Papadopulos-Eleopulos: No, you better.

Valendar Turner: There's data from drug users in New York which show that the risk of seroconverting is whether you're immune deficient before you seroconvert. In other words, if you prospectively study a large group of drug users, you find that one of the risk factors for actually becoming HIV positive is whether you are immune deficient before you're seroconverted, not the other way round. This is just not on.

I mean, if the theory is that HIV infects T4 cells and kills them - and this is what the theory is, although the HIV protagonists do not know how HIV kills T4 cells, and we doubt whether it actually does kill them, it just sort of changes them in some way, because they are low. We're not saying they're not low. This shouldn't happen. I mean, this is the wrong... this is the cause before the effect. It's not on.

Eleni Papadopulos-Eleopulos: You know, instead of having first HIV and then T4 decrease, to have HIV you need T4 decrease. This is a good, prospective, well-designed study.

Valendar Turner: Studied over many years.

Stuart Reid: But couldn't that be explained by if your immune system is low, then you are more susceptible to infection, including infection with the HIV virus?

Eleni Papadopulos-Eleopulos: But then we don't have HIV. But then why do you need HIV? If you have to be first immune deficient and then you develop HIV, then you don't need HIV because...

Valendar Turner: You don't need HIV to become immune deficient.

Eleni Papadopulos-Eleopulos: Not only that you don't need HIV to become immune deficient, but you don't need HIV to develop all the other infectious diseases, because if immune deficiency... and you have first immune deficiency and then you have HIV, why you shall have HIV and then the other infectious agents? Why all these infectious agents don't come in parallel? HIV cannot have priority. All the other infectious agents will be equally as...

Valendar Turner: Well, the theory goes HIV, immune deficiency, then the diseases. I mean, HIV has factored itself out.

Stuart Reid: But what I guess I'm saying is that it can be more complex than that, that you can have a kind of... if you're infected with the HIV virus, once you're infected with it, it affects your immune system but if your immune system is low to begin with, then maybe you're more susceptible to getting infections. A certain number of people are exposed to the virus and only a certain number of those get... actually get infected with it.

Eleni Papadopulos-Eleopulos: That is true, not only with...

Stuart Reid: But more people would get infected who are immune deficient.

Valendar Turner: That could be true but that's not the explanation for AIDS.

Eleni Papadopulos-Eleopulos: Not only that is not the explanation for AIDS, it's the fact that why should... then why do you need HIV? You know, that's the question. Why do you need an intermediary? You don't need an intermediary. If you have the immune deficiency, and that's what they think in this prospective study, it's not one case. Here it is, a prospective study, a large study, and here it is they say "We don't have it. The only way you can get HIV seroconversion is to have immune deficiency,

and never the other way round". Not first HIV and then immune deficiency. Now, that is one.

Secondly, is the fact that if you have first immune deficiency and if the diseases which are called AIDS are due to an immune deficiency, then why do you need the intermediary? Why do you need HIV? You don't need HIV. We have to really stretch it then. I mean, you can assume anything you want. You can put any hypothesis you want but to me it is logical because all the other viruses... you know, it's not only HIV which induces immune deficiency. Epstein-Barr virus induces immune deficiency, cytomegalovirus induces immune deficiency - all the infectious agents, not only viruses, induce immune deficiency. Then why HIV has all this... why do we make this virus... why do we give all...

Valendar Turner: Why do we make so many exceptions?

Eleni Papadopulos-Eleopulos: It's not just the exceptions. I mean, we give to HIV so many qualities and so many...

Valendar Turner: That's right, including its continental preference; its subgroup preference.

Eleni Papadopulos-Eleopulos: And trying to avoid the immune deficiency changes - because that's the theory, that HIV changes to avoid immune deficiency. It's a very clever virus. I mean, we never had anything like this today. As I said, you can stretch the HIV theory to no end and really now it is stretched because Montagnier says that... that's what we did not mention because Montagnier now says that HIV is not sufficient.

Stuart Reid: That brings in cofactors?

Eleni Papadopulos-Eleopulos: That brings in cofactors, but if the cofactors can do without HIV, and that is all the evidence everywhere, if the cofactors... if you can't have HIV unless you have PHA, and PHA can do the same thing which is attributed to HIV without HIV, why do you need HIV there?

Valendar Turner: The other thing, Stuart, is that if you accept HIV as being a respectable, properly isolated, to use an Australian term, dinky-di virus and that it can cause AIDS, if you like, then how do you account for the fact that in the consortium study which was published in JAMA, I think, wasn't it, by Lundberg et al in 1988, using the least stringent criteria for diagnosis, only 79 per cent of AIDS cases are infected with HIV?

In other words, depending on what the criteria... this is the western blot criteria again - they vary. But if you take the most stringent criteria, 51 per cent of patients are not infected with HIV, given the most stringent criteria; and given the least stringent criteria, 21 per cent are not infected with HIV. Now, I mean, here is... what causes...?

What is the cause? How can this be? How can AIDS be due to a pathogen which is not present or not infecting the person? I mean... and sure, this is a question... another reasonable question that people can reasonably expect an answer to. There is no answer. It doesn't make sense. We could say, "Look, tests aren't perfect and you don't get all the things"... and people often... when we discuss this with our colleagues, they start arguing by analogy.

A typical answer is, "Look, we can't find the syphilis organism, we can't always find the virus in glandular fever, or some other disease we can't find the virus or the bacteria but we still say it's that and we do say it's that and we treat it". That is one very important difference which we've stressed between scientific debate and the pragmatism of clinical medicine. Clinical medicine is pragmatic. It has to be because it has to deal with real problems as they occur, but this is a scientific debate that matters because we're going round telling people who are positive on AIDS tests they're infected with a lethal virus which people... if we're going to say this, a reasonable expectation from the population is that there's a sound scientific basis for saying so. Like... you say, "When you take this pill you won't get pregnant", you can't tell people this unless there's good scientific reasoning to say it's the case. You can't argue by analogy, you can't say because the polio virus is not present in all people with polio or we can't find it, therefore it's all right for HIV not to be present. They've got nothing whatsoever to do with each other at all.

Eleni Papadopulos-Eleopulos: Especially the tests they are using for HIV are considered to be 98 per cent sensitive, the best, not only specific but 98 per cent sensitive.

Valendar Turner: It's unbelievable.

Eleni Papadopulos-Eleopulos: Even 98 per cent sensitive, then we shall have only 2 per cent of people with AIDS as being non-infected, but to have enough between 20 to 50 per cent of them non-infected for me is a big problem, for us is a big problem.

Valendar Turner: And yet when I mentioned this data to an expert in the field in Australia, I was told that this was old data. Now, I mean, that's just not an answer.

Eleni Papadopulos-Eleopulos: In 1988 all they say to you it is old data but today data is even less. Today we have even less data. Unfortunately now people are becoming more and more... how you say?

Valendar Turner: Complaining?

Eleni Papadopulos-Eleopulos: No, they don't complain.

Stuart Reid: Complacent?

Eleni Papadopulos-Eleopulos: Complacent, that is the word. We're not as thorough as we used to be earlier.

Valendar Turner: In that regard I think the medical profession has probably been a little complacent over accepting the HIV/AIDS theory because...

Eleni Papadopulos-Eleopulos: They have no other choice but to accept it.

Valendar Turner: No, but it's important for us to state this, that scientific theories are accepted because people accept the authority of those who are in power, if you like, and they have to because they haven't got time to go and check up on all this stuff. It's too hard, it takes too long, we know that, and so no one blames everyone for believing. The ordinary GP, physicians, patients, even the experts, don't read the original isolation. We know; we've asked them. I mean, it's not as if...

Eleni Papadopulos-Eleopulos: But they can't, they have not got the time. Immunologists sit down as we did... I mean, I devote my life to this. There would be very few people who devote their lives to find out why... about retroviruses. And why they should not accept what I have accepted if I do not have this theory? I must admit that I am biased and if I did not have... life would have been much easier, I agree.

Valendar Turner: What Eleni is saying is important. I mean, one of the AIDS commentators has addressed the question: how did we get into this mess, sort of question. That's exactly how it's been stated but it's: how did we get into this mess? I've often reflected on this and, as Eleni just said, you have a lot of... there's four specialties, there's four big bodies of knowledge. There's retrovirology which commenced in 1911. There's an enormous amount of work done over the decades. Immunology which started to sort of blossom in the...

Eleni Papadopulos-Eleopulos: In the eighties.

Valendar Turner: In the sixties it started seriously.

Eleni Papadopulos-Eleopulos: But really the immunology of AIDS is eighties.

Valendar Turner: It's almost AIDS related. And epidemiology which is a topic in itself, and clinical medicine. And the immunologists don't all speak retrovirology and vice versa. I mean, everyone's relying on the judgments of everybody else. It's been an enormous task to look at this knowing in my case very little and seeing it all in all those different forms. And you do cut across all of it and when you get the big view, then it's obvious that there's something wrong here, very wrong. How they can say a serological test - when we already know that antibodies cross react - with a specificity of nearly 100 per cent; I doubt if even X-rays are that specific. I mean, they're pretty unbiased type tests.

Eleni Papadopulos-Eleopulos: 99.9 per cent. In fact the latest thing... I told you they say that the Burke data presented evidence that one in a million western blots is false positive.

Valendar Turner: And they came to this conclusion by comparing their western blots with a whole series of antibody tests which were basically the same.

Eleni Papadopulos-Eleopulos: And you repeat them again and again.

Valendar Turner: Against themselves or, if you like, mirror images of themselves an arbitrary number of times without using a proper gold standard. I mean, the gold standard is the problem.

Eleni Papadopulos-Eleopulos: Yes, but you can't blame the immunologists then for accepting this because this is retrovirology or this is... you know, so he will say all right, they prove it. He has to accept it, he hasn't got time to go and check them. He will say they're proven that the tests which we're doing here are 100 per cent specific. I mean, one in a million is more than...

Stuart Reid: As three researchers, two of whom are here, working very much on your own in isolation, how important has it been for you to have one another?

Eleni Papadopulos-Eleopulos: Very.

Valendar Turner: Well, I think it's been very important. I mean, this work has been done over a period of what, ten years now. Most of the intellectual effort and most of the actual elbow grease has been done by Eleni, although she probably would have found it difficult without some physical support and help from John and myself.

Eleni Papadopulos-Eleopulos: Maybe I should talk on this.

Valendar Turner: I'm just trying to think how Eleni has helped me, and that's my part of the question. I know I've helped Eleni but I'm just wondering how Eleni and John have helped me. John has taught me patience and to be polite, very much so, and to be circumspect about what one says and not to take the frustrations of being not treated seriously out by having wars with my colleagues, and I hope I've succeeded in that way. My wife's not very keen on AIDS. You've heard of golfing widows. Well, I suppose in a sense my wife's an AIDS widow in a metaphorical sense, and it's been a bit difficult, there's been a bit of family neglect and I hope it ends soon.

Eleni Papadopulos-Eleopulos: I don't think knowledge of something is sufficient to be able to do the work. I mean, it is true that initially John and Val were only morally supportive in the first few years, but even without their moral support I could not have done anything. But, you know, for most of our papers recently... I published only one paper by myself. The others are always in cooperation with John and Val and other people in our department, but especially John and Val. Because we are completely

different, because each of us looks from a completely different point at the problem, we all contribute to it.

We argue between us, we've been arguing to no end, you know, and each of us tries to put his or my point across, but we always come to a compromise and it's not a compromise just for the sake of compromise. I mean, I never will accept something unless I am really convinced of John's and Val's point of view and I'm sure they will never compromise unless... I'm sure... I mean, we've been arguing. I know that we come to the compromise only when the other person puts the evidence down and the others accept it.

But I think it helps us a lot that we're different. In fact, as far as I'm concerned, our best weapon... I don't know if I can call it that way... if we are proven to be true, if our theory is proven to be true - not us, we are true - but if our theory is proven true, it will be the fact that we are outsiders, because I am sure if... I'm talking for myself. If I was an immunologist or a retrovirologist or... not so much epidemiology because epidemiology really supports our view, but if I was a retrovirologist, immunologist, I would not have behaved different than they are. The only reason that we are seeing differently because we are outsiders. The fact that we're outsiders I think helps us a lot.

Valendar Turner: Yes, I agree with that. In fact this is sometimes levelled as a criticism of us, that we are outsiders, that we don't do laboratory work and we don't treat AIDS patients and we don't do either of those things. I see the occasional AIDS patient through the emergency department and that's about it, but I fail to see why that should matter.

Eleni Papadopulos-Eleopulos: Yes, I think we are more objective. I mean, as I said, if I was that, I would be exactly the same. I can't see me being different, but the fact I am from outside I look at things differently.

Valendar Turner: I think we're fighting human nature here.

Eleni Papadopulos-Eleopulos: No, it's not human nature, it's human knowledge.

Valendar Turner: I disagree. I think it is a large dose of human nature.

Eleni Papadopulos-Eleopulos: You don't know if you are right or wrong. We don't know if we are right or wrong, so everybody finds for his...

Valendar Turner: We must say that we are the first to admit it; that if... we could be proven wrong tomorrow and if evidence was presented that we were proven wrong, we would accept it. We are not... that's it.

Stuart Reid: I think that's the point on which we'll have to leave it. Thank you very much, both of you.

[End of interview with Eleni Papadopulos-Eleopulos & Valendar Turner]