Do antibody tests prove HIV™ infection?

A blood-curdling interview with Dr. Valendar F. Turner

Photo: David Smith of Perth

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Photo : Joan Shenton 1998

HC: Good afternoon Downunder.
VFT: Good morning Huw.
HC: The Perth Group publications[1-13] seem to cover just about every facet of HIV and AIDS but what I want to go over again is the antibody tests.
VFT: Fine.
HC: I'm particularly interested in trying to make this subject plain and simple for ordinary folk who haven't read the arguments published in the Group's papers over the past decade. Or if they have, don't quite understand. I mean it's pretty much in-your-face to read an abstract telling you Eleopulos et al don't accept HIV antibodies tests as proof of HIV infection in anyone.
VFT: I know but that's how Eleopulos et al read the data.
HC: Could you start with an overview?
VFT: Sure. Let's consider the two words 'antibody' and 'test'. In this context 'test' has two meanings. The first is something you do in an attempt to indicate the presence or absence of some substance or property. For example, does a patient have appendicitis? Or is a woman pregnant? The second is something you do to ascertain something's worth. For example, if you develop a blood test for pregnancy, how well does it perform?
HC: And antibodies?
VFT: Antibodies are proteins produced by cells of the immune system known as B lymphocytes. Not to be confused with T lymphocytes, the immune system cells which HIV allegedly kills making people immune deficient. The present theory of antibody production is that each B lymphocyte and its descendants, known as clones, elaborates one and only one unique antibody molecule.
HC: What switches B-cells into producing antibodies?
VFT: Two things. Firstly, when a B-cell encounters a substance known as an antigen. That word is derived from the letters of ANTIbody GENerating. Antigens are always large molecules and are often proteins. In fact proteins are the most powerful antigens. Even more so if they gain direct access to the blood stream.
HC: How does the antigen get the B-cell to make the antibody?
VFT: In the old days it was thought antigens instructed B-cells in the art of making antibodies. Like reading out a recipe while someone else makes the cake. But that's no longer believed. Nowadays the theory is that each B-cell already knows the recipe. But for only one type of cake. Each is programmed to make a unique antibody. Many times over of course but all the same. It's estimated B-cells have a combined repertoire of about one million distinct antibody molecules. It's just a matter of an antigen meeting up with the right B-cell. When it does that's the key which turns the switch as you suggest. The cell divides and produces a clone and out come the antibodies. That antibody then unites chemically with the antigen.[14]
HC: What else induces antibodies?
VFT: B-cells can be stimulated non-specifically. You give the immune system a belt and an assortment of B-cells go into production. For all we know this might be quite common. The only way to find out is to test for antibodies to everything except what you used to belt the immune system.
HC: What is the biological purpose of the antibody/antigen union?
VFT: Supposedly antibodies neutralise the untoward effects of antigens.
HC: Are germs antigens?
VFT: Yes, but with some qualification. Obviously antibodies and antigens must combine at particular places on their molecules. It's like hugging your grandmother. Your arms are only a small part of you and make contact only over a small part of grandma. The business end of the antibody molecule is called the combining site and the part of the antigen it joins on to is the antigenic determinant. There are many possible antigenic determinant sites on each antigen and any of these can induce a corresponding clone of B-cells to produce a particular antibody.
HC: So the antibodies produced to a germ are really a mixture of many different molecules to many different bits of the germ?
VFT: Yes. The technical term is that the antibody response is polyclonal.
HC: How do you give the immune system a belt?
VFT: Let loose with drugs or infectious agents or foreign proteins. Things to which all the HIV/AIDS risk groups are exposed. Of course these may act as conventional antigens but they can also act

on other B-cells. This may produce arcane reactions. A good example is that of Epstein-Barr virus, the virus that causes glandular fever.

HC: Wha's arcane there?

VFT: Somehow the virus switches on a set of B-cells programmed to make antibodies which react with the red blood cells of horses. And another which makes antibodies to sheep blood. But these aren't antibodies destined for EBV itself. They're something completely different. One wonders why we would ever need to produce such antibodies but we can. In fact as doctors we make use of this to diagnose glandular fever. This is an antibody test but it doesn't look for antibodies to the causative virus. Instead it looks for the horse blood antibodies.

HC: Curioser and curioser. What's the basis of using antibodies to prove HIV infection?

VFT: The belief that because HIV is foreign it will induce the production of antibodies directed against HIV.

HC: The theory is that an antibody to a virus can only arise if B-cells have encountered that virus?

VFT: Yes.

HC: Why not prove HIV infection by growing the virus?

VFT: Antibodies is technically easier and a lot quicker and cheaper.

HC: And you detect the antibody by taking some blood, mixing in some virus and seeing if the two react?

VFT: That's the theory but before we get to that let me explain something else very important. What we can call the age old antibody problem: why you can't reason backwards from antibodies to germs. It comes about because a particular antibody may also react with an antigen or antigens that did not stimulate its production.[14-22] This can be due either to non-specific stimulation or because antibodies cross-react.

HC: What does cross-react mean?

VFT: Two different antigens may share the same determinant. So the same antibody can get hold of either antigen by reacting with that part. Even though they're otherwise different proteins. You can also prove the existence of cross-reactions by doing a little thought experiment. Antibodies are large proteins and can themselves act as antigens. So that's at least two things an antibody can react with. The antigen that produced it and the antibody to it when it acts as an antigen.

Photo : Murex

A UK-produced ELISA test kit from Murex including rack of testing wells (centre)

HC: Why are these phenomena a problem?

VFT: Because they spoil what would be a nice theory that a person who has an antibody to 'X' must automatically be infected with 'X'. It's scientifically impossible to make such a claim merely from a chemical reaction.

HC: Even if it is beyond question that 'X' is a constituent protein of a unique virus?

VFT: Yes. You may never be infected with what your antibodies react with. Otherwise we'd have to say patients with glandular fever are infected with horse blood. As well as sheep blood. Or AIDS patients are infected with laboratory chemicals.

HC: AIDS patients have antibodies to laboratory chemicals? Can you name some?

VFT: Off the top of my head I can name one. Trinitrophenyl antibodies.[23]

HC: And it's not known how that arises?

VFT: Not precisely.

HC: How does one get around the antibody problem?

VFT: First by realising the problem exists. If you like analogies, diagnosing infections using antibodies, that is, serological diagnosis, is like trying to identify objects from the shadows they cast on the ground. There's a connection but clouds, buildings, trees and so forth all produce shadows that may look the same or similar. The way around the dilemma involves an appreciation of both meanings of that word 'test'. According to the first meaning what we want is some method of finding HIV in the body - HIV infection. That's what we're really chasing. The best way to do that would be to find the actual object itself. HIV. Prove the existence of HIV in every patient by means that are unambiguous for a unique retrovirus.[24-25] The gold standard. Any other way, including antibody tests, is indirect and must therefore be validated by comparison alongside the gold standard. The second meaning of 'test'.

HC: How?

VFT: By running the two sets of data concurrently. The antibody test and whatever you do independently to prove the existence in the person of the virus.

HC: Virus isolation versus the antibodies?

VFT: Yes but there's more to proving the existence of the virus than isolating a particle. After Eleni's [Eleopulos] interview[26] I'm sure your readers must be a full bottle on this topic.

HC: I wonder! How is an antibody test for HIV actually done?

VFT: As you said. Take some blood from a patient, remove the red cells and then add what's left, the serum in which the antibodies are dissolved, to some proteins the experts claim are unique constituents of HIV.

HC: What do you see if the test is positive?

VFT: If the antibodies react with the proteins there will be some detectable change in the solution or in whatever medium the test is performed. It may change colour or a precipitate may form. Or there is some other measurable effect.

HC: Things light up? That's all there is to it?

VFT: Basically. But there are refinements. For example, the ELISA versus the Western blot. The ELISA has all the proteins mixed together and in the Western blot you can see each reacting individually, side by side along a thin nitrocellulose strip.

HC: How is the comparison with HIV gold standard done?

VFT: What everyone wants to know is whether the test can be positive when there is no HIV infection. In other words, is my test a false positive? So, what a scientist is obliged to do long before the test is introduced into clinical practice is to determine what's known as the specificity of the test. That's a measure of how often a positive test turns up given HIV is known to be absent. Proved by viral isolation. If the test is one hundred per cent specific the answer of course should be never.

HC: Yes. I think people tend to get confused here. Can we go over these two words, sensitivity and specificity?

VFT: Sure. Sensitivity is a measure of how often a test is positive when you already know what you're testing for is present. For example, if a thousand women are pregnant, does the test diagnose them all? If it picks 980 then it's only 98% sensitive. And is it specific, in other words, is it ever positive when a woman is definitely not pregnant? For example, if, from a thousand women known not to be pregnant there was one positive test, the test would be 99.9% specific. You'd never dream of putting a pregnancy test into practice until you'd sorted out these parameters.

HC: If we take the HIV ELISA test, which is the first and sometimes the only type of test patients have performed to diagnose

HIV infection, how is the sensitivity determined?
VFT: First let's examine the way it should be determined. The correct procedure is to assemble say a thousand people proven by HIV isolation to be infected with HIV and see how many have a positive ELISA. Now the ELISA is made positive because the solution in which the antibodies react turns cloudy and the degree of cloudiness can be measured with a special instrument that gives out a number.
HC: Is any degree of cloudiness positive?
VFT: No because there is always some non-specific background activity. If you set the degree of cloudiness for a positive test very low then everyone might be positive. If it were a pregnancy test for example, even men could be pregnant. So you set some limit or sets of limits for the comparison.
HC: How is this determined?
VFT: Here there are some very unscientific practices. Basically, a group of healthy individuals is tested to estimate the background activity. This will have a range of values and from this range researchers select an upper limit which is maybe two or three standard deviations higher than the mean value. Any reading greater than that is defined as positive.
HC: It's arbitrary?
VFT: Yes.
HC: They don't set the level according to the results of virus isolation?
VFT: No. And setting a level doesn't prove the antibodies are genuine anti-HIV antibodies. You can't say antibodies are to HIV just because there's more of them. Higher levels might just be more of the same that caused the lower level of cloudiness. Or lower levels might be the real thing. The only way to prove the antibodies are a reaction to something called HIV is first to prove you have the virus.
HC: What about the sensitivity of the Western blot?
VFT: Again, you have to set criteria for what constitutes a positive test and then apply this to a population of known infected people. Again there are no such data for even one of the multitude of different criteria which are said to define a positive HIV Western blot. But, as I'm sure you know, the sensitivity is not of prime importance to the HIV experts because in most parts of the world the Western blot is put forward as a means of sorting out which positives ELISAs are due to HIV infection and which are not. What's important for the Western blot is its specificity.
HC: How does one perform an experiment to measure specificity of the HIV antibody tests? ELISA and Western blot?
VFT: Take a thousand people including AIDS patients, as well as people who are sick with similar illnesses and laboratory abnormalities as AIDS patients, as well as those at risk and some healthy people, perform HIV isolation to prove none have the virus and amongst this group see how many are antibody positive by whatever criteria you set for each test.
HC: Why such a diverse range of individuals?
VFT: Because these tests measure antibody reactivity and you need lots of antibodies and lots of variety to produce lots of chances of reactions to prove that the reactivity which defines a positive test is restricted to those individuals who are HIV infected.
HC: Well, if sensitivity of either antibody test has never been measured against the guaranteed presence of HIV, has the specificity ever been measured against the certified absence of HIV?
VFT: No one has ever reported an experiment performed to draw this comparison. Not for the ELISA nor the Western blot. This is one of the great AIDS mysteries. However, if you look at Gallo's 1984 Science papers,[27] what Gallo and his colleagues called HIV isolation was positive in only a third of their AIDS patients. Yet nearly three times that number had antibodies.[28]
HC: That's a huge disparity. That's nearly twice as many people with antibodies and no virus as with antibodies and virus! It's a much better correlation between antibodies and absence of infection. So right from the start it should have been obvious the test was grossly non-specific?
VFT: Yes.
HC: How did Gallo explain this discrepancy?
VFT Gallo didn't admit to any discrepancy in virus isolation. Instead his group believed all the patients with antibodies were infected. They blamed the low yield of virus isolation on failure to receive or handle their tissue specimens under "optimal" conditions.
HC: Yet the Gallo lab was considered expert in culturing retroviruses?
VFT: Yes over a decade of experience and nowadays it's claimed that the blood of untreated AIDS patients is teeming with HIV.
HC: Has the discrepancy between antibodies and HIV isolation narrowed over time?
VFT: Not in the least. If you remember our reply to Peter Duesberg,[11] between 1992-93 several reputable, international laboratories in the UK, Germany and the USA tested 224 specimens from antibody positive individuals. These labs also claimed to have performed viral isolation but like all HIV researchers, they're forever perverting the meaning of that word. What they called HIV isolation was another antibody test. This time for detecting just one protein, p24. And under this guise 'isolation' was positive only 83 times.[29] That's 37%. Substantially the same rate as Gallo in 1984.
HC: Do HIV experts really refer to an anti-p24 antibody test as virus isolation?
VFT: Most of the time. And some report just finding reverse transcriptase as virus isolation.
HC: Is the failure to perform the gold standard comparison the reason why the Perth group claims not one antibody positive person in the world is infected with HIV?
VFT: Principally on that basis we say there is no proof that one person is infected. Yes. But the other reason of course is that no one has yet proven the existence of HIV using the proper method. The method based on the definition of a virus and as discussed at length at the 1972 Pasteur Institute meeting.[24-25]
HC: Which the Perth group was the first to argue over a decade ago?
VFT: Right from day one.
HC: Nonetheless, it still seems an intrepid claim. No proof that even one antibody positive person in the world is infected?
VFT: Look Huw you just can't put the words "HIV" and 'antibodies' next to each other and claim you've proved they exist. Or a virus exists. All the test indicates is that some antibodies in patients react with some proteins present in cultures of tissues from the same patients. But given that information what a scientist is obliged to do next is make the comparison with the virus gold standard. Before pronouncing the test highly specific for diagnosing HIV infection. In fact, do you see that the origin of the proteins used in the tests doesn't matter? They don't have to come from HIV. I mean we diagnose Epstein-Barr virus infection without using proteins from the Epstein-Barr virus. Horse red blood cells are not constituents of that virus. What counts is the correlation between certain reactions and the presence or absence of the virus.
HC: But surely it makes sense to use proteins from the germ?
VFT: It does because if there is a germ there is a possible connection, forwards, between the germ's antigens and the patient's antibodies. But just because you use the germ doesn't mean you can ignore the problem of antibody cross-reactivity and everything else.
HC: So it's incorrect for scientists to say the HIV antibody tests are better nowadays because they use purer proteins?
VFT: That's right. It doesn't follow. Even if genetically engineered proteins are used in the test. You could take the purest protein in the world and find a patient with an antibody to that protein. That doesn't create an axiom that a person with that antibody is infected with a germ containing that particular protein. This is an extremely important but frequently unappreciated concept. In fact you could take a genetically engineered protein and make the test worse.
HC: How?

Principally on that basis we say there is no proof that even one person is infected

VFT: Because every time you change the antigens there's a possibility you could introduce a new antigenic determinant. All antibodies know is how to react and there might be an antibody lurking that links up with that determinant but whose presence bears no relation whatsoever with whatever you're testing for. For example, lots of humans have antibodies to things like hepatitis A and even Pneumocystis carninii. In fact by the age of four most children have antibodies to the PCP organism. Without ever being sick from either organism. One of those antibodies might cross-react with the new determinant.

HC: And patients are tested for antibodies despite the fact that no one has done a gold standard comparison?

VFT: The tragedy is that these tests were introduced in the total absence of proof of their specificity. This is a fact. The moving finger has writ and all our tears cannot wipe out a word of it.

HC: That's from Omar Khayyam?*

VFT: Yes.

HC: The Perth group has claimed that the HIV proteins and antibodies as well as the existence of HIV are based on a circular argument. Could you explain that?

VFT: I'll try my best. When Montagnier and Gallo went hunting for retroviruses in 1983/84 they knew that merely finding a particle that looked like a virus, even if they were to isolate the particle and prove it could reverse transcribe RNA into DNA, did not prove the particle was a virus. That's because not all particles, even those that look like viruses, are viruses. And not everything that reverse transcribes is a retrovirus. Or even a virus. These phenomena are non-specific. And stringing together reverse transcription and particles doesn't cure the problem. The only scientific proof that a particle is a virus is purification and analysis followed by experiments to prove particles make more particles exactly the same. In other words, proof that the particles are infectious. These experiments have never been done. Proof of the existence of HIV is based on antibodies but unfortunately, picking up antibodies just added yet another nonspecific item to the list.

It's not the way a scientist should establish the existence of a virus or determine which are its antibodies

HC: But Montagnier and Gallo did discover antibodies from AIDS patients which reacted with some proteins in their cell cultures.

VFT: Yes they found a few but that doesn't prove the proteins which reacted with these antibodies are the constituents of a virus. Or that the antibodies were induced by contact with a virus. If you'd like another analogy imagine this experiment. In place of the AIDS-diagnosed patient's cell culture someone hands you a test tube containing milks obtained from half a dozen different animals. In other words, a mixture of several different proteins but you don't know from which animals. Now in place of a mixture of antibodies from AIDS patients you obtain a second test tube containing a number of different acids. You add the mixture of acids to the mixture of milks and produce curdles. Now you claim you've isolated a cow. Or a goat. And not just any cow or goat. A completely new species of cow or goat. One never seen before. There, in the culture. And then you claim that only a particular selection of the acids in the mixture produced that curdle. So, getting back to HIV, proteins reacting with antibodies makes them into the HIV proteins. But since these newly discovered proteins react with these particular antibodies that means these antibodies must be the HIV antibodies. It's called chasing your tail. It's not the way a scientist should establish the existence of a virus or determine which are its antibodies.

HC: Yet almost everyone believes these antibodies are the HIV antibodies and they're highly specific to HIV.

VFT: True and that's because of virtually the same circular argument. AIDS, the clinical syndrome, usually but not always, is accompanied by antibodies which are interpreted as proof that AIDS-diagnosed patients are infected with HIV. Then the antibodies are used to prove that HIV is the cause of AIDS. In other words, AIDS proves it's HIV proves it's AIDS. Naturally the antibodies seem specific. They and AIDS run around the same circle. What's important for anyone in this debate to realise is that when you pare down what the experts claim proves the existence of HIV, they are all non-specific phenomena including antibody reactions. That's all. It's not isolation. No viral-like particles are separated and analysed and then added to fresh cells to see if exactly the same come out.

HC: But regardless of where these antibodies come from, doesn't their relationship to AIDS-defining conditions mean something?

VFT: In the AIDS risk groups yes it does. If you have these antibodies you're at risk of either having or developing a number of diseases which constitute the AID clinical syndrome. But it doesn't prove the link is a retrovirus.

HC: Or that the illnesses are inevitable?

VFT: They may well not be inevitable. After all, we're talking statistics.

HC: All right. The Perth group has also written at length about the global variation in the HIV Western blot antibody test criteria. It was first presented in the Bio/Technology paper of 1993 and Continuum published your chart illustrating the same thing in the November 1995 issue.[30] Tell us about that.

VFT: OK. The Western blot is a general laboratory technique for visualising individual protein/antibody reactions. The proteins are placed at discrete spots in a thin paper strip. In the case of HIV about ten of them. The human operator inspects the strip and declares which proteins react with antibodies. What you actually see is a series of dark horizontal rectangles called bands. You'd think that if there really were such things as HIV proteins, and that the HIV antibodies are highly specific, then just having one band light up would be proof that HIV is present. But according to the experts that's not the case.

HC: They say you need more than one?

VFT: With one single exception. The intriguing thing is this. Even if one or two bands are not sufficient to diagnose HIV infection there must still be a reason why they're there.

HC: Cross-reacting or non-specifically induced?

VFT: Right. Proteins in the tests lit up by part of the menagerie of antibodies present in AIDS patients. Or maybe a few present in a healthy person following some chance, B-cell stimulus. In fact, cross-reactions is the explanation given by all the HIV experts for "non-infected" Western blots. Non-HIV antibodies produced by non-HIV stimuli. But if one or two bands in a Western blot can be caused by non-HIV, cross-reacting antibodies why can't three or four, or five or six, or all ten bands be caused by cross-reacting, non-HIV antibodies?

HC: I don't know. You tell me.

VFT: Well, a scientist must admit to this possibility. And there's only one way to find out. Compare your favourite combination of antibodies with HIV itself.

HC: But that has not been done?

VFT: Not only not done. Not even possible to do because no research group has ever presented evidence for the existence of HIV according to the proper rules.[6-13, 26]

HC: What about the actual variation in the Western blot?

VFT: Another mystery. What is considered positive depends on where and by whom the test is done. Around the world different combinations of two or three or four of the ten possible bands are deemed proof of infection.[31-36] In Africa you need two bands but in France, the United Kingdom and Australia that wouldn't count. In Australia you need four and under the US FDA and Red Cross rules you need three.

HC: This is the basis of the Group's quip about emigration?

VFT: Yes. If you're positive in New York City just get on a plane and come to Perth. You'll no longer be positive.

HC: You mentioned an exception?

VFT: The US Multicenter AIDS Cohort Study or MACS. This excellent study began in the early 1980s and followed the fate of

5000 gay men. Under the study rules the Western blot could be positive with just one "STRONG" band.[36] Although that later changed. But until 1990 one band was considered sufficient to diagnose HIV infection.[31] That wouldn't count anywhere else. Not even in Africa. So there are gay men out there HIV infected on this basis. And perhaps given antiviral drugs as a result.

HC: Let me get this right. We are always conscious of our new readers and I think this is extremely important. You're saying that even the experts concede that some numbers or patterns of bands in the Western blot are not indicative of HIV infection because they're caused by non-HIV antibodies?

VFT: Yes. You can read what Anthony Fauci wrote about this in Harrison's Principles of Internal Medicine.[22] Maybe you could print the quote at the end of the interview.*

HC: So it's definite that non-HIV antibodies react in an HIV test?

VFT: Yes Huw. There are plenty of examples. For instance, 30% of people transfused with HIV negative blood develop antibodies to p24.[37] That's regarded as one of the most specific HIV proteins and it's present in the Western blot. And it was one way any one of those 5000 gay men could have scored a positive test in the MACS. So some gay men are infected with HIV on the basis of a test that turns up positive in one third of people transfused with blood that does not even contain HIV.

HC: I find that more than a bit disturbing.

VFT: So should any man in that study. Or any person Western blot tested before 1987.

HC: Why then?

VFT: Before 1987 anyone with a p24 or a p41 band was diagnosed positive and thereby infected. That is, if they were ever Western blot tested. Not everyone has had a Western blot. Some were diagnosed just on the ELISA. The way people are in most of the UK today, except in Scotland where the Western blot is still routine. For example, in 1985, using either p24 or p41 or both on the Western blot, Australian experts diagnosed HIV infection in a gay man and transmission of HIV from his semen to four women following artificial insemination. This was big news at the time because it was said to be direct proof for heterosexual spread. This is an oft quoted paper. In 1996 we questioned this in a letter published in The Lancet. In light of the current Australian criteria we asked were the man or the four women still considered infected? In their reply the Australian experts defended the original claim of HIV infection because all five people had progressed to AIDS and died. They implied that the reason extra bands were not present in 1985 was because in 1985 the Western blot was in its "infancy".

Western Blot "virus proteins"		Africa	Australia	U.S. Food & Drug Admin	U.S. Red Cross	CDC (1)	CDC (2)	CON'	MACS[2]	UK
ENV gene	p160	ANY 2	1 OR >	1 OR >	1 OR >	p120/ p160 AND p41	p120/ p160 OR p41	p120/ p160 OR p41	ANY 1 Strong OR 3 Weak bands from: p15, p24, p32, p41, p45, p53, p55, p64, & p120. Score '1' for each weak band, and '3' for each strong band - total of '3' or greater is positive	1 OR >
ENV gene	p120									
ENV gene	p41									
POL gene	p68	OPTIONAL	ANY 3	p32	ANY 1			p32 OR		p31 (sic)
POL gene	p53									
POL gene	p32									
GAG gene	p55			p24	ANY 1		p24	p24		p24
GAG gene	p40									
GAG gene	p24									
GAG gene	p18									

Criteria varying worldwide for a positive HIV test result on Western blot

HC: What's infantile about a test?

VFT: We don't know but if the test had not yet come of age, why was it being used? But there's two interesting points here. First, it confirms what I said earlier. HIV researchers use the diagnosis AIDS as proof that the antibodies are caused by HIV. The second is that if p41 and p24 were sufficient to diagnose HIV infection in Australia in 1985 and, according to the Australian experts, they were correct in these five patients, why aren't they sufficient now? They certainly still are in other parts of the world.

HC: What about the missing bands?

VFT: Although the WB criteria changed in 1987, apparently it was not until The Lancet published our letter that the sera from the gay man and one of the women were retested. On these sera the gay man and the woman now did have four bands.

HC: How would they arise?

VFT: The band that proved difficult was the p120 band. There was a belief that a protein of this molecular weight SHOULD be present in the Western blot. However, it took a lot of time and experimentation to work out how to produce one. In fact, it's impossible to have a "viral" p120 in the Western blot because we know from the work of Hans Gelderblom and his colleagues that HIV particles, once they're shed from the cell, rapidly lose all their knobs, and that's where the HIV experts claim the p120 protein is to be found. The real reason there's a p120 band in the Western blot has nothing to do with a virus. It's due to the fact that the HIV researchers eventually found the right chemical conditions to produce it when they prepare the Western blot strips. This was proven in 1989 when it was shown the p120 band is no more than a polymer of the p41 protein. We discuss this in our Bio/Technology paper..[1]

HC: Food for thought. What other instances are there of cross reactions?

VFT: There are many more examples. Surely everyone knows about the dogs by now? Fifty percent of 144 dogs tested in the USA in 1990 were found to have antibodies to one or more HIV proteins.[38] But dogs don't get HIV or AIDS so those bands can't mean HIV infection. If a gremlin had mixed up the blood from the dogs and the men in the MACS no one could have told the difference. There's also non-HIV infected mice who develop HIV antibodies when they're injected with lymphocytes from similar HIV-free mice[39] and there's the study co-authored by the Australian expert Dr. Elizabeth Dax.[40] In 1991 her group re-analysed Western blot strips, not sera, performed in 1985 on sera originally obtained from ten intravenous drug addicts in 1971-72.

HC: What did that reveal?

VFT: Could I read the details from one of our unpublished papers?

HC: Go ahead.

VFT: Ten persons "with potentially positive WB patterns, when the more specific 1985 criteria were used", were traced. One patient had died from a motor vehicle accident and there were "no lymphoreticular changes at autopsy, and a thorough retrospective analysis provided no evidence of either current substance abuse or HIV infection". Of the nine living addicts, two could not be assessed clinically, seven were not chronically ill, (one was in prison but in good health, one had been successfully discharged from a methadone program, one was enrolled in a methadone program, another sporadically consumed illicit drugs). "The two former patients whose 1971-72 WB results were most strongly reactive had current ELISA and WB assays that were negative. The immune function parameters were inconsistent with immune suppression". Their data led the authors to conclude, "it is possible that antibodies to a non-pathogenic virus would have disappeared during the 17 to 18 years...follow up. Although this potential cannot be ruled out, it is more likely that the earlier results were false positives...definitive evidence of HIV infection in the United States' addict population as early as 1972 is still lacking".

HC: HIV antibodies can fade and even disappear over time?

VFT: Yes. Despite the fact that we're told HIV is forever, here

are drug addicts who gave up drugs, started to live a more healthy lifestyle and their antibody tests reverted to negative. And their T4s returned to normal. And most telling of all, they were alive twenty years later to tell the tale.

HC: And nowadays they'd be hailed as saved by the latest anti-HIV cocktails?

VFT: Quite possibly. It's worth stressing how great a dilemma these data create for the HIV experts. If these addicts had not attracted attention by being alive they would have died carrying a pathogenic HIV and most likely their deaths would be attributed to HIV. No doubt that was the official cause of death for many of their less fortunate brothers and sisters. But since they were alive and in relative good health this challenged the HIV theory of AIDS. So the experts toyed with the idea of a nonpathogenic HIV. That would at least rescue the tests. But it would also set the beginning of the AIDS era back to 1971. And place it not in Africa but in the United States. And make us wonder how lethal or relevant is a virus that hangs around for at least twenty years without killing the patient. And which disappears as the patients' health improves. So, for these particular addicts, who turned over a new leaf, it had to be false positives. Why couldn't all drug addicts all turn over new leaves and end up the same?

HC: Perhaps all AIDS patients? Stay well away from drugs, including anti-retrovirals, and live wholesomely and long enough for the antibodies, and the risk factors, to metamorphose into something kinder?

VFT: Maybe for some but don't forget AIDS patients have diseases. These should be evaluated and treated.

HC: Why is this paper unpublished?

VFT: We wrote the paper in early 1997 and called it A critical appraisal of the evidence for the isolation of HIV. I'm a Fellow of the College of Surgeons in Australia and we sent it there hoping to get the surgeons interested. The reviewing took months and there was a lot of correspondence. They declined to publish, not because of significant disagreement with the science but because the editorial board considered that debate about the existence or non-existence of HIV "would be of little interest or use to the majority of readers of the Australian and New Zealand Journal of Surgery".

Our HIV Reference Laboratory admits that one quarter of HIV-free blood donors have one or more reactive bands on the HIV Western blot

HC: Incredible.

VFT: Incredible but true.

HC: Where's the paper now?

VFT: On the Net. At the Reappraising Website[13] and also, thanks to the most generous efforts of Robert Laarhoven, at our own Website*. Last month Neville Hodgkinson told us that from the point of view of getting out the message about the existence of HIV, it was the most readily understood paper we have ever written.

HC: Getting back to Western blots, do the experts offer any explanation for the extreme variation around the world in the criteria for a positive Western blot?

VFT: Well there are a couple of things that emanate from our National HIV Reference Laboratory.

HC: What do they say?

VFT: First, it is claimed that the different WB criteria have become more closely aligned over time.

HC: Is that right?

VFT: How can it be? In 1985 it was all p24 and p41. Whatever side you're on, at least you'd have to say that was aligned. But a mere glance at the chart shows just how aligned the WB criteria are at present. If that's aligned what existed sometime in the past must have been close to anarchy.

HC: What about the different criteria for a positive test?

VFT: According to our experts it's perfectly legitimate to set the criteria for a positive test according to the prevalence of HIV infection in the community being tested.

HC: Meaning what?

VFT: Where the prevalence is low, as claimed for Australia, you set a lot of bands for a positive test. In fact we have four. But in Africa, where they claim the prevalence is up to 10%, you can get away with less, just two. And in the USA it's sort of intermediate. Two or three bands.

HC: Where's the problem?

VFT: First, what if I told you the Faculty of Medicine at the University of Western Australia teaches its students to interpret chest X rays differently in smokers versus non-smokers? Or in Catholics and Jews? Or in different countries? So in Iceland your chest X-ray shows lung cancer but not if you send the films to Perth. Second, the experts regularly make assertions about the prevalence of HIV infection but how do they know what this is? When you find out how this is estimated it turns out to be the same antibody test. You can't do that. You can't use an antibody test to determine the prevalence of a disease unless you know its specificity. No one knows the specificity of the HIV antibody tests. What the experts are doing is using a test of unknown specificity and setting it up as judge and jury over itself. This is the trouble with this so-called AIDS science. This is the sophistry used to determine the specificity of the HIV Western blot at an unbelievable 99.999%.[41]

HC: Could you explain what you mean by that?

VFT: HIV researchers perform an HIV antibody test in a number of individuals and then repeat it half a dozen times using a slightly different technique or a different brand of test. But they're all the same test. If the tests are positive and all match they say this proves the test is one hundred percent specific.

HC: Repeating the result is taken as proof of what caused the result? Unbelievable. How do they make an independent judgment as to the presence or absence of HIV?

VFT: That isn't done. What's done is like taking a chest X ray or an ECG on a number of different machines or in different hospitals and claiming that finding the same thing over and over proves lung cancer or a heart attack is truly present.

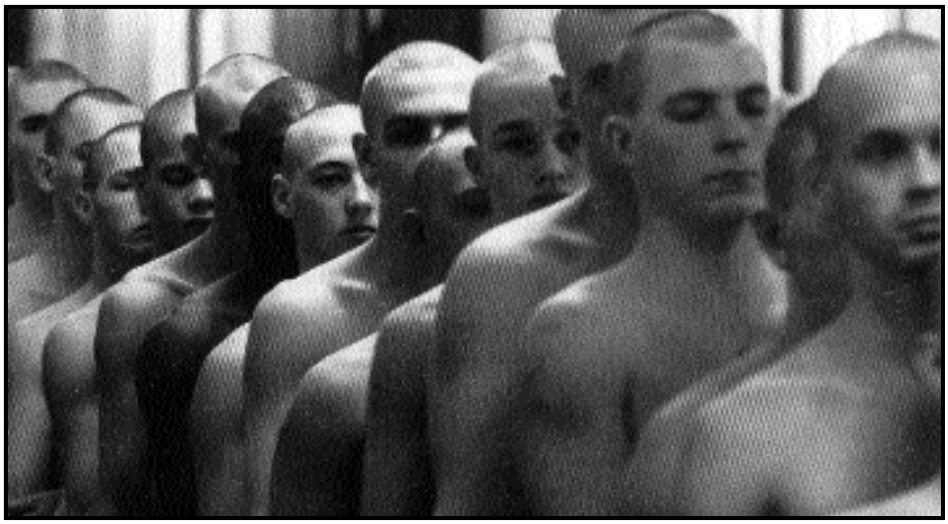
HC: So although everyone admits to interference caused by non-HIV antibodies, no one has really sorted out the magnitude of the problem. As the Perth Group says, they may all be non-HIV antibodies?

VFT: Yes. For example, our HIV Reference Laboratory admits that one quarter of HIV free blood donors have one or more reactive bands on the HIV Western blot. They concede these are caused by cross-reacting, non-HIV antibodies. Now, the way you get your cross-reacting, non-HIV-induced antibodies is to give your immune system a few belts. And the more belts, and the more closely spaced, the more likely a person tested will have cross-reacting antibodies. But we know that in places like Africa this kind of thing is happening all the time. And it happens across all the AIDS risk groups. So the very people you're testing for HIV are those with the greatest chance of having cross-reacting or non-specifically induced antibodies. So we have this grotesque paradox. One quarter of pristine, well fed, OZ* blood donors have one or more HIV WB bands, and that might include four bands, but they're not infected with HIV. But in Africa, poverty stricken, malnourished, Ugandan subsistence farmers with malaria or tuberculosis, or repeated attacks of dysentery, could have buckets of cross-reacting antibodies but if they've got just two bands on the Western blot, not four, they are infected with HIV. Do you know anyone who can explain this?

HC: It certainly seems at odds with what one would expect. I know of a lot of people who would avoid even trying.

VFT: It gets even more arcane. If our experts are right about the Western blot criteria becoming more closely aligned over time, since the Australian criteria haven't changed recently and since scientists seem obliged to set the number of bands according to the prevalence of HIV infection, one must deduce that the prevalence

of HIV infection in the rest of the world is approaching that of Australia.
HC: Which is deemed to be one of the lowest in the world?
VFT: Yes.
HC: Obviously it's been made much easier to diagnose HIV infection in Africa compared to Australia.
VFT: The World Health Organisation criteria make it much easier to report a positive test in Africa. But that doesn't prove a positive test is caused by HIV infection.
HC: The criteria should be the most stringent in the so-called developing world?
VFT: No one knows the correct criteria anywhere in the world but everyone does know about cross-reacting antibodies. And they are what create the confusion. It's like losing your five year old kid at the pictures. If you had to take him to something Adults-Only because your babysitter ran away, then it's simple. The theatre is most likely full of adults and any kid you see is likely to be your kid. But what if you took him to see Snow White? There's kids all over the place. You need far more stringent criteria before you can pick out your kid. If he had a look-alike, or even just dressed the same, you'd have to set the stakes higher still. If he had a twin brother you might need to take off his socks and look for the mole on his foot.



"In 1988 the US Army tested over a million soldiers and found...half of all the 12,000 first positive ELISAs were negative second time around".

HC: So using only two bands in Africa means the test is worse quality than it is even in the West for example?
VFT: When you talk about tests you need to be careful with words. 'Quality' could refer to any test parameter. We don't know any of the test parameters because they've never been appraised against the gold standard. I must stress this again and again. Without knowing the sensitivity and specificity of the HIV antibody tests it is impossible to use the tests to prove HIV infection. But your question raises another interesting point. When you look at the mathematics of testing it's very easy to prove that where the prevalence of whatever you're chasing is high even a lousy test will get it right more than half the time. That's because the odds are stacked before a person even has the test. And 10% prevalence is very high. Diabetes is around five percent and migraine ten percent. So if one in ten Africans were HIV infected, and here I'm talking prevalence determined by bona fide means, not a circular abstraction based on antibodies, and the average African could afford to pay for a test, you could just about use anything. Even a test for Vegemite* antibodies might provide a reasonably good prediction of infection.
HC: Antibody tests aren't done routinely in Africa?
VFT: The World Health Organisation, Bangui definition of AIDS in Africa requires neither an antibody test nor a T cell count. I think this is something else extremely important to stress. People may not appreciate what the African data imply. First, no one would dream of diagnosing HIV infection or AIDS in the West without a blood test. But under the African definition it's OK. You can be an AIDS case just on symptoms, for example, fever, cough and diarrhoea for thirty one days fulfils the definition. Second, the only reason that heterosexuals in the West are deemed at risk of infectious immunodeficiency is because of how the African situation is interpreted. Because equal numbers of men and women in the reproductive age group have African AIDS diagnoses and when tests are done equal numbers have antibodies. Based on assumptions from these parallel but potentially misleading results, an African diagnosed under the Bangui definition, without an antibody test, is condemned to HIV and AIDS unlike anyone in the West. And under such diagnostic rigour the example of thousands of African men and women, who are essentially suffering from symptoms and diseases all called other names before 1981, is held up as proof that the West is menaced by the threat of heterosexually transmitted AIDS.
HC: Caused by the same virus?
VFT: Yes even though the antibody test used to diagnose the same virus is read differently in Africa. And might not be positive in other places. In fact, according to the CDC, in the United States, an African individual with an AIDS defining diagnosis is counted as heterosexual AIDS simply by the fact that he or she comes from a country where heterosexual AIDS is the claimed to be the "predominant" mode of transmission. Knowledge of actual sexual contact is not a requirement.
HC: It's assumed an African will invariably be heterosexual?
VFT: Apparently.
HC: Could an equal gender distribution of AIDS in sexually active adults prove sexual transmission?
VFT: It's consistent with sexual transmission but it's not sufficient proof. Equal numbers of sexually active adults develop appendicitis or meningitis. Or schizophrenia. Are these diseases sexually transmitted?
HC: Hasn't the Perth group recently published a paper reviewing cross-reacting antibodies?
VFT: Yes. Our last paper[12] reported a considerable amount of data showing that antibodies to the types of organisms which infect 90% of AIDS patients may also react with all the putative HIV proteins. Including in the Western blot. So, if 90% of AIDS patients are infected with either a mycobacterium or a fungus such as Pneumocystis carinii, how it is possible to diagnose HIV infection in such persons, or to assert that HIV is the cause of their diseases? The paper also examined cross-reacting antibodies in relation to proof for the existence of HIV. In fact, as a caveat, we go into great detail to explain how virtually overnight the world's first human retrovirus, Gallo's HL23V, became extinct when its antibodies were proved non-specific.
HC: And the Perth group posits a similar fate for HIV?
VFT: When someone finally takes on the isolation or specificity problem, they're really the same problem, we believe this is a distinct possibility.
HC: So compared to 1993, when the Bio/Technology paper was published, there's more evidence that positive antibody tests are caused by factors even the experts admit are non-HIV?
VFT: Definitely. The other thing that's important to remember is that patients are highly selected for antibodies before they ever get to the Western blot. WBs are done on people who first of all feel the need to go to a doctor and then have sufficient antibodies to make the ELISA react twice in a row.
HC: They're preloaded with a selection of antibodies?
VFT: Right. You see Huw, when you say someone is HIV negative, the truth is they're not ELISA negative, WB negative. They are actually ELISA negative either once or one out two, and Western blot not done. A negative is not confirmed with a Western blot, only a positive. But by choosing this particular testing strategy the HIV/AIDS experts have maximised the chances for the appearance of cross-reacting antibodies.
HC: Maximised cross-reactions? Is there evidence for this?

VFT: Yes. In 1988 the US Army[41] tested over a million soldiers and found that even in healthy military recruits, half of all the 12,000 first positive ELISAs were negative second time around. And after a second positive ELISA two thirds failed to react on a first Western blot. And some first Western blots failed to react on a second Western blot. So, what you set up with two positive ELISAs before a WB is ample opportunity to introduce confusion caused by cross-reacting antibodies. Snow White in a test tube.

HC: Might there be people who would test negative twice on ELISA and then positive on Western blot?

VFT: This happens but there are little data on how often because negatives usually aren't confirmed in this way.

HC: Are any other reasons put forward to justify the variation in the actual WB criteria?

VFT: None that I know unless of course HIV is endowed with some kind of global navigation system. It figures out where it is and then chooses which B-cells to engage. That skill would be extremely hard to encode in eight or nine or ten genes.

HC: Why eight or nine or ten genes?

VFT: It may be the most studied object in the universe but the experts still don't agree how many genes it has.

HC: In 1998 what advice would you give a patient wishing to know his or her HIV antibody status?

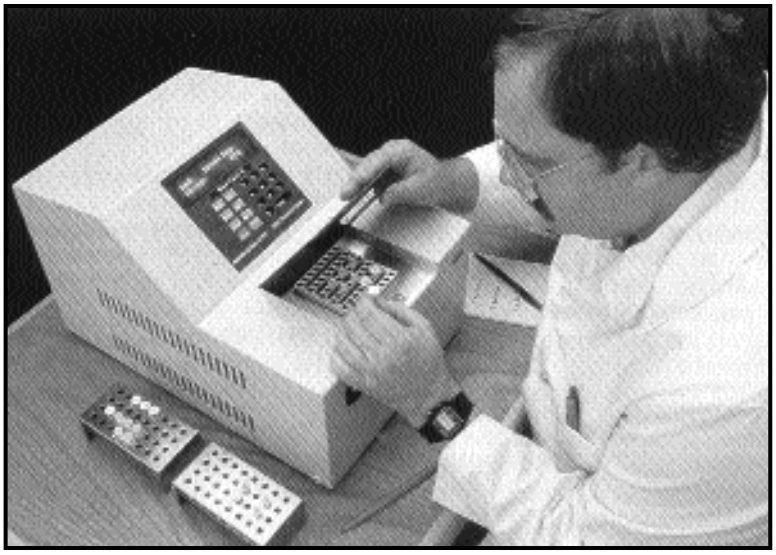
VFT: First of all, from the point of view of establishing the presence of HIV infection, I'd say don't have a test. Don't spread HIV testing. You wouldn't expect a woman who'd missed a period to have a pregnancy test if you didn't know how well the test performed. So why this one?

HC: What if someone, say in a high risk group, wants to know his or her chances of developing an AIDS-defining illness? Regardless of whether HIV is the cause?

VFT: I suppose there's two ways of looking at this. What are the chances of getting sick, which is how doctors tend to think, or what are the chances of remaining healthy? That puts a different emphasis from the point of view of the person. There's no doubt about the association between being in a risk group, having a positive test and developing certain diseases defined as AIDS. But that doesn't apply across the board. It's only statistical. So for an individual these two variables cannot be the whole story. Not all such people get sick and the risk varies up to fifty times between the risk groups. So, if you put aside the retrovirus link and all that goes along with that, you might look around for other factors. Now, like the ultimate causes of most diseases, some of these factors may be completely unknown and totally out of your control. But there might be some that are not unknown and are under your control. Maybe as simple as being in a risk group. You could, for example, decide to get out of your risk group or cease doing whatever is risky within your risk group. Remember what happened to the drug addicts. As far as explaining the association with the antibody tests is concerned, perhaps HIV researchers have inadvertently stumbled across a "something wrong test", like the ESR for example.

HC: What's the ESR?

VFT: The erythrocyte sedimentation rate. It's a test widely used in clinical medicine. It measures how fast a drop of blood falls to the bottom of a test tube of anticoagulant solution. The rate at which red blood cells sediment is affected by changes in the plasma in which they've been living, especially changes caused by alterations in the composition of the proteins. For example in inflammatory conditions such as rheumatoid arthritis and in tuberculosis, although non-diseases such as pregnancy also produce a high ESR. In fact, in the old days, the ESR was used as a pregnancy test. The point is this. Our group has long argued lack of proof for a retrovirus as the cause of these antibodies. But nonetheless, something must stimulate their production and understanding that this is a possibility might lead people to things which could undo their possibly harmful warnings. If the positive test is not caused by one of the actual diseases then maybe there are elements of the person's life which can be changed so that the stimulus to this warning system is turned down. Or even switched off. Again we come back to those drug addicts. They didn't have HIV, the experts say so, but they did have antibodies which reacted in an HIV test. Whatever the reason, when they altered their lives towards attaining better health, somewhere along the same road where they shook off their habit, they shook off their antibodies. I know the experts' explanation was that they never had "real" HIV antibodies but that, much more innocent interpretation, presents our side of the argument. These data are predicted by our theory. These data are a test of our theory and our theory has passed this test. The only difference is we say there are no proven, "real", HIV antibodies. So, maybe just the idea that these antibodies could have other causes might bring sufficient hope to neutralise the doom wrought by the explanation that they must be due to HIV. I think those of us who are not HIV positive cannot even begin to imagine how profoundly the psyche and health of an individual are affected by belief in the existence of a lethal retrovirus inexorably eating away at the immune system. It must take extreme valour to even question what almost the whole of the rest of world believes to be true.



A PCR (polymerase chain reaction) test kit - the basis of 'viral load': "Contact the manufacturers of the primers and probes, ask for the scientific justification."

HC: We should study long term survivors with HIV antibodies to delineate what factors lead HIV positive individuals towards diseases?

VFT: Or away from diseases. That would be of enormous interest and benefit.

HC: What about people with actual AIDS-defining diseases?

VFT: As I said before, the diseases should be vigorously and intelligently treated in their own right.

HC: What if someone not in a risk group is healthy but positive?

VFT: The only honest answer is that, from the antibodies point of view, there are no data upon which to pronounce a prognosis.

HC: Why do you say that?

VFT: Because from a purely scientific point of view, to determine whether these antibodies represent an independent hazard, one would have to take a hundred or so healthy, no risk, HIV positive individuals and follow them untreated for a number of years and see what happens. But you would not be able to tell them they're HIV positive.

HC: Why not?

VFT: Because, as we've just discussed, patients and physicians believe most fervently that being HIV positive is a death sentence. This belief and the possible administration of anti-HIV drugs may themselves produce illness. These two variables would severely confound the experiment.

HC: As a doctor yourself, what in particular would you say patients should ask their doctors?

VFT: Request scientific proof that the antibodies present in your body arise for no other reason than infection with a virus called HIV.

HC: What if the answer is don't worry, trust us and the tests are virtually perfect?

VFT: Then ask how, where and when and by whom this was established. Request citations, scientific papers, names, dates, places, researchers, journals. Get a copy of our 1993

Bio/Technology paper or our latest paper, or this or Eleni's interview, or some of the other stuff Christine Johnson has written about our research, and ask that each point is specifically answered. What you must find out is how the specificity of your test was determined. Since all the HIV experts declare cross-reacting antibodies affect both ELISAs and the Western blot, ask how they know your antibodies aren't all cross-reacting. Put that very question. And refuse to accept obfuscatory remarks and don't be put off by big names and big institutions.

HC: What if the answer includes advice to have a viral load test?

VFT: Then ask your doctor for proof that the RNA or DNA used in the test to match your RNA or DNA is a unique constituent of a particle proven to be an infectious retrovirus. I know the experts now regard virus particles as old hat but on the other hand, they still say a particle called HIV causes AIDS. So there has to be a direct link between the RNA and DNA and a particle. Where is it? Contact the manufacturer of the primers and probes and ask for the scientific justification for the label on the bottle. And since the PCR is quite capable of amplifying non-target sequences, how and where the sensitivity and specificity of the test for HIV infection was determined?

HC: What if one's told it's all too hard to understand?

VFT: It's not hard to understand. I know it takes time but basically most of this stuff is easy to understand. You know Huw, Papadopulos-Eleopulos et al have spent well over a decade behaving impeccably as scientists and all we've really proved is that even if you think you're right, that forms about three percent of the answer. The issues we've written about languish waiting for scientific responses. The trouble is so many of us, doctors included, accept the validity of the HIV theory and all the tests because of big names and big institutions. In good faith I must add but nonetheless without checking up for themselves or asking questions. Well, they're not usually the ones told they're infected with a lethal retrovirus. So patients must be their own advocates and thereby influence public opinion towards the debate. Let me remind you of what Galileo said: "In Science the authority embodied in the opinion of thousands is not worth a spark of reason in one man."

HC: Do you ever entertain thoughts that your ideas about all this may be totally wrong?

VFT: Yes. And if there was a scientific debate, and we were proved wrong, we would accept it.

HC: Finally, I believe you have written a book about some of your experiences?

VFT: It's nice of you to ask. The truth is I've written a manuscript. It's not yet a book because I'm still having a hard time doing the rounds of the publishers.

HC: What's it about?

VFT: It's a novel. A thriller[42] set in the US and Australia. About a biotechnology company trying to bump off an AIDS dissident because the Chairman of the Board perceives a huge threat to company profits. The story is woven around a Professor of Chemistry, a lady of course, and an HIV positive haemophiliac boy with a sceptical, politician uncle. There are several conversations and a court scene where our view of HIV and AIDS is aired.

HC: In plain language I hope?

VFT: That's for the reader to judge.

HC: Dr. Turner. Thank you very much for your time today.

VFT: Thank you Huw. I hope I've managed to stir a few hearts and minds. And if anyone out there wants to publish a highly controversial book, please let me know.

*The Moving Finger writes: and, having writ,
Moves on: nor all thy Piety nor Wit
Shall lure it back to cancel half a Line,
Nor all thy Tears wash out a Word of it.
- The Rubaiyat of Omar Khayyam

*According to Anthony Fauci, "the least likely explanation for an indeterminate [insufficient bands for positive but not the complete absence of bands=negative]western blot is that the individual is infected with HIV...The most likely explanation is that the patient being tested has antibodies that cross react with one of the proteins of HIV".

*http://www.virusmyth.com/aids/perthgroup/
*OZ - Australia
*Vegemite - A favourite Australian yeast-based sandwich spread.

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