I will be discussing the HIV antibody tests.
These are the tests that doctors use to diagnose HIV infection in their patients.
According to the HIV experts these tests are extraordinarily accurate.
I will present scientific evidence there is no proof the antibodies that react in these tests are caused by HIV and also present other evidence that the antibodies have causes which are not retroviral.
PLEASE NOTE

The speaker notes in this file are not the literal court transcripts

However, with the exception of text marked EXTRA, all information in
the speaker notes was provided as testimony
Antibodies are proteins produced by cells of the immune system known as B lymphocytes. The B stands for bone marrow derived. These are different from the T lymphocytes which are depleted in the blood stream of AIDS patients.

Any substance that generates antibodies is known by the generic title “antigen”. For example, proteins act as antigens.

Normally the body does not generate antibodies directed against its own constituents. This is because the normally operating immune system can distinguish between self and non-self. However, there are situations where this breaks down and the body does produce antibodies against self components. These are called auto-antibodies and this happens in AIDS.

The concentration of antibodies in healthy people is about 15 gm/litre. AIDS patients typically have much higher levels. Up to 25 gm/litre, which means in terms of the concentration of antibodies, AIDS patients are in surplus. They are not deficient.
Humans are thought to have a repertoire of about one million different antibody molecules.
Antibodies are present in the blood stream.

If a specimen of blood is left on the laboratory bench, after about twenty minutes the blood will have separated into blood clot and serum. The antibodies are dissolved in the serum.

Because serum is used to detect antibodies, antibody diagnosis is often referred to as serology. For the same reason a person with a positive antibody test is sometimes referred to as being “seropositive”. Or if negative as seronegative.
The antigen that induces an antibody also reacts with that antibody. That is, the antigen and antibody combine chemically with each other.

The reaction can be demonstrated outside the body by taking some serum and adding it to the antigen in a test tube. As the reaction takes place it produces some physical alteration in the reaction mixture. Often this is a colour change, which can be measured by some means. The colour change is how the laboratory scientist knows there has been a reaction. This is what an antibody test is.

Antibody tests can be used for diagnosis because if a person is infected with a foreign agent, such as a bacterium or a virus, that person will produce antibodies directed against that agent or its biochemical constituents. Such as its proteins.

However, antibodies are not a virus. An antibody test is only an indirect means of proving the presence of a virus. Everyone knows that no test is perfect so why not test for a virus directly? Shouldn’t that be the gold standard method? Free from ambiguity? The answer is that virus isolation is relatively complicated, time consuming and expensive. Antibody testing on the other hand is relatively simple, quick and cheap. And
it is acceptable as long as its accuracy for proving a viral infection has been established before being introduced into clinical practice.
In order to perform an antibody test for a virus three things are needed. A blood sample from which to obtain the patient’s serum, the proteins of the virus and a method of determining the criteria for a positive result.
Eleni Papadopulos has presented electrophoretic evidence that the “HIV” proteins are cellular proteins.
There is also evidence that the antibodies said to identify certain proteins in cultures from AIDS patients as “HIV” may also react with the same proteins in non-HIV infected tissues.

For example, by this means p24 has been found in the blood of healthy, non-infected blood donors and also in non-infected organ transplant recipients.
Placentae from 25 normal term pregnancies were collected by vaginal delivery...Antigens gp120 and p17 were identified in normal chorionic villi...Antigen p24 in villous mesenchymal cells...localized to HLA-DR positive cells”


Three of the HIV proteins have been found in the normal human placenta, and since the same particles Montagnier described as “HIV” also occur in most placentas, and there is ample evidence that placental tissue has reverse transcriptase activity, why aren’t pregnant women regarded HIV infected?

However, for the sake of argument, let us assume the proteins in the antibody test kits are the unique proteins of a retrovirus HIV.

In Australia as in most of the developed world two methodologically different antibody tests are used.
These are the ELISA and Western blot tests.

ELISA stands for enzyme linked immunosorbent assay. Sometimes this test is called an EIA which means enzyme immunoassay.
In the ELISA test the proteins are present as a mixture. When serum is added and the test developed a colour change ensues which is quantified by measuring how much light passes through the reaction mixture using a spectrophotometer. The greater the amount of antibody the higher the reading. But the ELISA cannot distinguish which proteins are reacting.

In the Western blot test the proteins are electrophoretically separated along the length of a thin nitrocellulose membrane. When serum is added and the strips developed the laboratory technician can name which proteins have reacted. The sites of the antibody/protein reactions are referred to as "bands" and their intensities are usually judged by eye.

Please note that in this and subsequent Western blot diagrams the proteins are not in electrophoretic order. Rather, for convenience, they are grouped according to which "gene" is said to produce them. These genes are known as gag, pol and env.

EXTRA

In regard to reporting the Western blot the National Reference Laboratory recommends “At the completion of each run, reactions should be read immediately…and then the strip can be dried and stored. If stored, strips should not be taped but enclosed in a plastic binder. Because the reactions may fade with time, a written record of the reactions (including intensity) or a photocopy of the strips immediately after completion of the assay must also be included”. Page 188.
This slide illustrates how these two tests are used.

First an ELISA test is performed. Almost everyone produces some colour change in the ELISA but it has to be over a certain amount to be reported reactive. If it does not exceed that particular amount it is reported as non-reactive and that is the end of the matter for that person.

However, if the ELISA is reactive it is repeated and if still reactive a Western blot is performed. Serum is added to an unused strip and, according to which bands appear, the result is classified as positive, negative or indeterminate.
In Australia a positive Western blot is reported when there is at least one of the p41, p120 or p160 bands, plus three other bands. Negative means no bands. Indeterminate means a band pattern that is neither positive nor negative. A positive Western blot is regarded as proof of HIV infection. A negative Western blot is reported as no infection. An indeterminate Western blot is almost always not caused by HIV infection. When the Western blot report is issued it is recommended practice to list the actual bands and their intensities as well as interpreting the result for the clinician.

The reason testing authorities give for doing these tests in this order is as follows:

The ELISA is not specific enough to make a definite diagnosis.

Hence, if the ELISA is reactive, the mixture of HIV proteins could be reacting to either genuine or non-genuine HIV antibodies. HIV experts claim by separating the proteins in a Western blot test some of the 1023 possible band combinations are caused by genuine HIV antibodies and the rest are not. The question is, “how do they know that these reactions are specifically due to HIV infection”? 
The word specific is one that occurs frequently in regard to antibodies and antibody testing. Hence we need to understand precisely what it means.

It means that the thing under consideration has only one cause.
For example, this is an extremely specific test for a certain brand of motor car. So specific I don’t have to name it.
On the other hand this is not a specific test for that motor car because this test is positive for other makes of cars.

Hence a 100% specific test is one in which a positive result points only to one cause. There is no other cause.

And if an antibody is said to react specifically with an antigen, it means it reacts with that antigen and with no other antigen.
Are the tests specific for “HIV”?

Infect human with a virus
Protein induces antibody
Antibody and protein react?
  YES

Discover an antibody and protein react
Proof that protein induced that antibody?
  NO

Why? Antibodies are not monogamous

Let us address the point- what is the proof the antibody tests are specific for HIV infection.

We know that if we infect a human with a virus, because it is foreign and its proteins are foreign, the body will produce antibodies that react with those proteins and this will show up in an antibody test.

Because of this we may think that whenever we find an antibody that reacts with a particular protein, that proves the antibody arose because of that protein. Unfortunately, this is not true. This is because antibodies are not monogamous. Antibodies induced by one antigen can and do with other antigens.

There are many examples of this including one pertinent to the HIV tests.

One percent of healthy people, not infected with HIV, which means some 200,000 Australians, have a reactive ELISA test. And 40% of people, which could amount to 8 million Australians, have at least one Western blot band, but they’re not infected either. So it’s obvious that non-HIV antibodies react with the test kit proteins. Even dogs and
mice, who do not get HIV or AIDS, develop antibodies that react with these proteins, including the proteins regarded as the critically important envelope and core proteins.
This phenomenon, an antibody reacting with more than one antigen, has been known for decades..
35 years ago the eminent Australian scientist and immunologist Sir Gustav Nossal wrote “an antibody molecule made following the injection of one antigen frequently can combine also with a second antigen of a related or similar shape…In other words, the antibody cross-reacts with the second antigen”. Which means that a reaction with a test kit protein is no guarantee that the antibody arose because of that test kit protein.
20 years ago Avrameas, a scientist from the Pasteur Institute who is a specialist on antibodies, also addressed the fact that an antibody may react with many, different antigens and that the antigens do not have to be similar.
In 2001 Dr. John Marchalonis, another antibody expert from the University of Arizona, went even further calling antibodies “promiscuous” and may bind to multiple antigens that “are ostensibly unrelated to one another”
To illustrate this point here are some data on reactions between two antibodies, one called E7, the other D23, and twelve, chemically dissimilar antigens. The number of plus signs is an indication of the strength of the reaction.

It’s not difficult to see that unlike the three pointed star and that motor car, there is no one to one relationship between either antibody and a particular antigen.
If you add antibody E7 to actin it reacts. If you add it to renin it reacts, just as strongly. Is E7 an actin antibody or a renin antibody?

Or is D23 the renin antibody?

If this seems complicated let me illustrate the point with some kitchen chemistry. Lemon juice and vinegar both curdle milk. That is, they react with the milk proteins to produce a precipitate. That's what curdle is. But by looking at curdle you cannot identify which agent was added. It could be either vinegar or lemon juice. You can't tell.

This means that just because you find an antibody in blood that reacts with the antigen in a test kit, this does not prove the antigen caused that antibody.

There is always room for doubt about the ability of an antigen to identify an antibody although it does not mean that particular antibodies never react specifically. They may and if they do then that is highly propitious for a scientist developing an antibody test. The problem is proving this is the case.
To illustrate how such proof should be obtained let us consider the example provided in our affidavits.
How may a scientist may prove a test for pregnancy is specific?

There is a blood test for pregnancy which detects a protein secreted by the placenta which, as we know, is present in the pregnant state.

However, every doctor knows that pregnancy tests can be misleading. They can miss a pregnancy, even an advanced pregnancy. A pregnancy test can also be positive when a woman is not pregnant. There are even situations where a man may have a positive pregnancy test.
When a woman is tested there are only four possibilities.

She can be pregnant with either a positive or negative test. That’s 1 and 2 on this slide. Or she can be non-pregnant with a either a positive or negative test. That’s 3 and 4. These four situations are called the test parameters and given the names on the right hand side of the slide.

There are two factors to appreciate.

First, in an ideal pregnancy test all numbers would fall into 1 and 4. All pregnant women would test positive and all non-pregnant women would test negative.

And if there are positive tests in women who are not pregnant, which are called false-positives, then obviously the test cannot be specific. Number 3 on the slide.

The question is: how do we determine these parameters and thus know how much trust doctors and patients can place in this test?
This brings us to the second factor: Before we can evaluate the test we need a method of knowing for sure whether or not a woman is pregnant. And this method must be independent of the test. A test cannot test itself. That would be cheating. We call the independent method the gold standard because this is going to tell us whether it is TRUE or FALSE that the woman is pregnant. It is against this certain knowledge that we compare our test results and obtain our numbers. Whatever numbers arise, they can only relate to and be as good as our gold standard. So we would like to have the most accurate, unambiguous gold standard for pregnancy possible.

What could we use as a gold standard? First up we might consider using the woman’s “clinical status”. That is, knowing that pregnancy causes cessation of menstruation, nausea, urinary frequency and weight gain we might consider these a suitable gold standard. However sooner or later we would see that will not work because these symptoms have so many other causes, even in combination.

Eventually we come to the realisation that the gold standard for a pregnancy test is a baby and that’s what we use to check the results.

These same principles apply to the evaluation of any diagnostic test. That is, you must compare your test results with a gold standard that best represents the entity you are testing for. We know the aim of antibody testing is to prove infection with HIV. In this instance we can think of HIV as “the baby”. So we require a gold standard for HIV infection against which we can find out whether the antibodies really are caused by HIV and not for some other reason. What can it be? It can only be HIV. Diagnosing HIV is the reason for the test and the only yardstick for HIV is HIV. As determined by HIV isolation. However, when we search the HIV literature it is apparent that experiments comparing HIV antibody tests with HIV isolation have never been reported. And in our view cannot be reported because of all the problems Eleni Papadopulos has discussed.

HIV experts themselves acknowledge there is no gold standard for these tests.
Dr. William Blattner is a retrovirologist and a director of the Institute of Human Virology at Baltimore in the United States.

According to him there is no gold standard.
Manufacturers of antibody tests admit there is no gold standard.

Here is what one manufacturer repeatedly includes in the test kit packet insert:

“At present there is no recognized standard for establishing the presence or absence of HIV-1 antibody in human blood”
According to Dr. Philip Mortimer, Director of the Sexually Transmitted and Blood Borne Virus Laboratory in the UK,

"Diagnosis of HIV infection is based almost entirely on detection of antibodies to HIV, but there can be misleading cross-reactions between HIV proteins and antibodies formed against other proteins, and these may lead to false-positive reactions. Thus, it may be impossible to relate an antibody response specifically to HIV infection” (emphasis added).

It means that if someone has a positive test we can’t be sure if it’s caused by HIV infection.
Yet despite all these caveats such tests are claimed to be extraordinarily accurate.

Before we consider why HIV experts claim antibody tests can diagnose HIV let us consider several scientific problems in regard to the “confirmatory” Western blot test.
First, this is a text book promoted by the Australian National Reference Laboratory. One of its authors, Dr. Dax, is the head of that laboratory.

In this book there is confusion about the identity of two of the diagnostically “extremely important” p120 and p160 glycoproteins in the Western blot strips.

The p41, p120 and p160 proteins are also called glycoproteins because they incorporate carbohydrates, that is, various types of sugar molecules, into their structures. Hence the optional prefix “gp”.
Confusion about the identity of the diagnostically “extremely important” gp120 and gp160 proteins

gp120, gp41 are “viral antigens that reside within specific areas of the virion”

The gp160 precursor is a “true gene product”, that is, a true viral protein

In the Western blot the gp120 and gp160 proteins are integer subunits (trimers [X3] and tetramers [X4]) of gp41 (Pinter et al)


In one part of the book it states gp41 and gp120 are viral proteins that reside in the HIV particle and that gp160 is “a precursor, being subsequently cleaved to form gp120 and gp41…and although “not a structural component of the virus, it is a true gene product”, that is, a true viral protein and this protein is present in the Western blot.  Page 60/61

But another part of the book they agree with Pinter et al who showed that the p120 and p160 proteins in the Western blot are not different proteins but are composed of 3 or 4 subunits of the same, p41 protein. The same protein that Montagnier regards as cellular actin.
Confusion over the identification of these bands has resulted in incorrect conclusions in experimental studies. Similarly, some clinical specimens may have been identified erroneously as seropositive, on the assumption that these bands reflected specific reactivity against two distinct viral components and fulfilled a criterion for true or probable positivity. The correct identification of these bands will affect the standards to be established for Western Blot positivity: it may necessitate the reinterpretation of published results” (emphasis added)


These findings led Pinter to warn that confusion over these bands may have led to wrong diagnoses and such diagnoses may need to be revisited.

Apparently no one heeded this warning.

This means that whenever a WB diagnosis requires 2 or more gp bands, what you actually have is only one band, a p41 which, according to Montagnier, is actin.

So when the criteria say you need two or three, but you really only have one, you are not fulfilling the criteria for a positive test.
Second, why should separating proteins in a Western blot make the test specific?

Why is it that of the 1023 possible band patterns that could occur in a Western blot, only genuine HIV antibodies cause certain patterns while non-genuine antibodies avoid making the same patterns but do make others?

For example in Australia the Western blot on the left is not positive but indeterminate. And, according to the experts, in most cases these bands are not caused by HIV antibodies. But add a p120 band, as on the right, making the test positive, and the three bands that were not caused by HIV now are caused by HIV.

This raises two questions:

How is it that in the right strip the p24, p55 and p32 antibodies are caused by HIV but in the left strip they are not?
If non-HIV antibodies can cause the bands in the left hand strip why can’t the gp120 band in the right hand strip also be caused by non-HIV antibodies?
Especially given, as I said at the beginning, AIDS patients have far more antibodies than healthy people, each one with the potential to cross-react with different antigens, why can’t four or even more Western blot bands be non-HIV? What’s to stop all the bands in the Western blot being non-HIV?

We have tried many times to find this out. By writing to medical journals and the Australian National Reference Laboratory but have never received a scientific answer.

EXTRA
Theoretically, with ten bands to choose from, there are 1024 different bands patterns that can occur in a Western blot. Applying the NRL Australian criteria 693 of these will be positive and the remaining 331 either indeterminate or negative.
The third issue involves some history.

Originally, that is, in most cases before 1987, a single p24 or p41 band, or both, was considered confirmatory proof of HIV infection.

For example, in 1985 four Australian women undergoing artificial insemination were reported to have become HIV infected from donor semen from an HIV positive man. The man and women were diagnosed on the basis of one or two of these particular bands. Nowadays these Western blots would not be reported positive.

Haemophiliacs

Then it was discovered that about 40% of healthy individuals, at no risk of AIDS, have at least one Western blot band. Most often a p24 with or without other bands.

Obviously 40% of individuals could not have HIV infection so HIV experts solved this problem by arbitrarily increasing the number of bands and designating only particular patterns of bands as positive.
The issue is that testing authorities have defined a positive Western blot in many different ways such that the criteria vary between laboratories, institutions and countries. So much so that a person positive under one set of criteria may not be positive under another.
Here are several of the major jurisdictions that have published criteria for a positive Western blot test.
And nowadays manufacturers also provide their own criteria.
Here are the criteria for each of these jurisdictions.

For example, in Africa, the left most column, for a person to be diagnosed HIV positive he must have at least two of the three bands p41, p120 or p160.

But in Australia and in some parts of the United States one band will suffice while in France a person must have all three.

In the right most column we see that in the MultiCenter AIDS Cohort Study, an ongoing study of 5000 gay men, even up till 1990 just a single, “strong” band was still considered a positive Western blot.

Here are some examples to illustrate the outcome of the global variation in the HIV Western blot test.
In this slide the Western blot has two bands, a p24 and a p41, and is positive in the jurisdictions indicated by the flashing star but not in the other jurisdictions.

A person with this Western blot would be HIV positive in some parts of the United States but not in Australia or Africa or France.
In this slide the Western blot has four bands and is positive in the jurisdictions indicated by the flashing star but not in the others.

A person with this Western blot would be HIV positive in Australia but not in Africa.

HIV experts respond to these different Western blot criteria in two ways.

First they claim many people with positive Western blots have so many bands they would be positive under many or even all jurisdictions. If this is the case then why are there different criteria?

Second, without defining what they mean, they claim the differences are slight. If the same test result has a person HIV positive in one country and not HIV positive in another we would argue the differences are not slight.

It also impossible for the HIV antibody tests to be “extraordinarily accurate” when the result depends on which laboratory does the test.
We should remember viruses and not committees of humans determine which antibodies arise and hence which Western blot bands develop. And if these different criteria for a positive test really do reflect some mystical geographical variable in the way HIV infects people, one would think that the criteria one should apply to diagnosing “HIV” infection should be those that operate where that person was infected. So if a person was infected in Africa for example, the African criteria should be used, not the Australian criteria.

Which means if you don’t know where someone has been infected you would not know which criteria to use.
IMPLICATIONS

According to the HIV experts, in Australia approximately 1% of non-infected people have a reactive ELISA while 40% have a single band Western blot test. It is also estimated that that approximately 1/1000 Australians are HIV infected. Which means that 0.1% of people must have a four band or more positive Western blot test.

Given that 40% of Australians have one Western blot band and 0.1% have four or more bands it is difficult to believe that a number of Australians, a number somewhere between 40% and 0.1% of the population, do not have 2 or 3 bands on the Western blot test. This number of bands would be regarded as indeterminate in Australia but under the criteria of several overseas testing authorities some of these tests would be positive.

How many such people are there? Would such people be ill advised to be tested in other countries? Would overseas public health authorities regard such individuals at risk of transmitting “HIV” in their countries? Or advise them to take antiretroviral medications? On the other hand, how do Australian public health authorities rate overseas individuals whose Western blot bands are positive overseas but indeterminate in Australia?
And what is someone wishes to emigrate? Whose criteria determine the infection status?
Interpreting the Western blot test in Australia

NRL: “Positive: the presence of a glycoprotein (envelope) band plus three other viral specific bands, or now some laboratories use the band combinations specified by the manufacturer as their interpretation criteria”

Manufacturer GENELABS recommend “following the accepted policy to be in accordance with local regulations” BUT

their criteria (including 2 of the p41, p120, p160 bands) are different from the “local [Australian] regulations”

*Dax EM et al. Advances in laboratory testing for HIV. Pathology 2004;36:551-60.

To add further confusion nowadays the National Reference Laboratory now permits Australian laboratories use the band combinations specified by test manufacturers.

But when you read the packet insert of GENELABS, one approved manufacturer, the advice is to follow “local regulations”. And although they do provide their own criteria, they are such that some positive Australian Western blots would be downgraded to indeterminate because this particular manufacturer requires not one but two of the glycoprotein bands.

So who decides what criteria to follow and how?
“Confirmatory tests for HIV [such as the Western blot] are sometimes called “supplemental” tests because they really don’t confirm infection…True, antibodies to HIV signal infection, but because of cross-reactive antibodies, positive results may not always be due to specific antibodies to HIV.”

Autoantibodies, high levels of antibodies in general, parasitic diseases, “other infectious agents”

“…pregnancy and syphilis, [which] are notorious for producing interference with serologic assays”


Constantine et al add more confusion by this statement.

“Confirmatory tests for HIV are sometimes called “supplemental” tests because they really don’t confirm infection…True, antibodies to HIV signal infection, but because of cross-reactive antibodies, positive results may not always be due to specific antibodies to HIV. False-positive assays have indeed been documented”.

Then they list a number of causes which include auto-antibodies, high level of antibodies in general, which is typical of AIDS patients, parasitic diseases, which are common in Africa, unspecified “other infectious agents” and pregnancy and syphilis.

Perhaps we can understand why HIV expert Dr. Philip Mortimer, who we quoted earlier, writing on the “Fallibility of the Western blot” said “Western blot detection of HIV antibodies began as, and should have remained, a research tool”.

In fact in England this test is not used.
We stress that nowhere in the scientific literature are there reports of HIV itself being used to define the true infection status of persons when validating the HIV antibody tests.

But needing something in place of HIV, something to act as “the baby”, HIV experts have resorted to certain de facto stand-ins which, in our view, are unscientific.

In the Constantine book, in addressing this issue, one reads that the true infection status has been determined by “clinical status, culture etc”. The “etc” is part of the quotation.

First, by clinical status is meant AIDS. And by AIDS is meant one or more of 30 diseases said to define AIDS. You could use clinical status as a gold standard for HIV but if and only if there is proof that these diseases are caused by nothing but HIV. But this is not true because all these diseases have causes which are not HIV. For example, TB today is the commonest AIDS defining disease in the world. Its underlying cause, before the AIDS era is poverty and malnutrition. So AIDS cannot be used as a gold standard.

On the other hand, if you choose to use AIDS as a gold standard you create a very big problem. Since the vast majority of individuals who test positive for HIV, including Mr.
Parenzee, do not have AIDS, then like women who test positive for pregnancy but do not have babies, the vast majority of positive HIV antibody tests must be false positives.

Second, culture cannot be used as a gold standard because both the antibody test and what is meant by culture is one and the same reaction. The only difference is for the antibody tests you have the antigen and you are looking for antibodies that react with them. And for culture, also known as “isolation”, you have an antibody and you look for antigens that react with it. They are both antigen/antibody reactions involving the same reagents.

Third, “ETC” conveys no meaning and its inclusion in a textbook of retroviral testing and quality assurance is surprising to say the least.

However, “ETC” may apply to another but flawed method which was put forward in the late 1980s by Burke and his colleagues who tested 1.2 million US military recruits in a paper that many regard put the Western blot test on the map.

EXTRA
When it comes to validating the Western blot, if we apply the Australian rules there are 1024 different possible Western blot band patterns. You can’t test each pattern with just one patient. Fifty would be a very conservative number and to test all patterns would require over 50,000 AIDS patients. But at the end of 2004 Australia counted 10,000 AIDS patients with 6500 deaths.
From his 1.2 million recruits Burke chose and tested a 135,000 subpopulation chosen because they were young and healthy and came from parts of the US that for all intents and purposes had no AIDS.

Burke defined a recruit antibody positive on the basis of 2 reactive ELISAs followed not by one but two positive Western blot tests. Now everyone else, including Gallo, regarded all positive tests in extremely low risk individuals as false positives. But Burke thought otherwise.

To find out Burke then performed four extra antibody tests on the positive soldiers, using another two different brands of Western blot and two other tests similar to the Western blot. If a soldier was positive on all 8 antibody tests he truly had HIV. If he wasn’t positive on all 8 he truly did not have HIV. Even though he already had 4 positive tests. Which is one Western blot more than we use in Australia to diagnose HIV.

In other words, for Burke and his colleagues, repeating the test was the gold standard for distinguishing between true and false HIV antibodies.
This is wrong. If a test can be positive for more than one reason, repeating the test will not resolve the ambiguity.
A photograph is a test for flowers. The basis of the test is a reaction between light and coloured pigments impregnated on a piece of celluloid. Real flowers and artificial flowers cause the same reaction in the film.

So Burke wanted to know if his flowers were true or false.
So repeated the test four time. I can repeat the test. In fact I could repeat it a thousand times, using the same or a different brand of camera or film but I still can’t tell if these flowers are real or false.
I can repeat the test and even if it’s negative I still can’t tell if the flowers are true or false.
Hence whatever the antibodies in Burke’s soldiers, HIV or non-HIV, they will be the same antibodies no matter how many times the test is repeated. Repeating a test is not a gold standard for determining the specificity of a test. When our group criticised this study in our 1993 Bio/Technology paper, a peer reviewed, international journal, not one scientist defended Burke’s methodology. Including Burke.

Yet based on this study the Western blot was claimed to be 99.99% specific.

Hence it is our view that the specificity of the HIV antibody tests has not been proven against HIV and thus it is impossible to state how many, if any, of the antibodies that react in these tests are caused by a retrovirus HIV.

One may then reasonably ask, if not a retrovirus “HIV”, where do these antibodies come from?

Briefly, there are three possible reasons.
First AIDS patients have diseases such as mycobacterial and fungal diseases. Tuberculosis is caused by a mycobacterium for example. In fact mycobacterial and fungal disease together constitute a considerable number of AIDS diagnosis. It is known that antibodies that form in response to mycobacterial and fungal antigens react with the proteins in the HIV antibody tests.
For example, here is a series of Western blot tests performed on leprosy patients and their contacts. These are taken from a paper published by Myron Essex, one of the world’s leading HIV researchers from Harvard University.

Leprosy is a disease caused by a mycobacterium which is closely antigenically related to the organism that causes tuberculosis. According to the World Health Organisation, in Africa a positive Western blot requires two glycoprotein bands. These are present in the first three strips in this slide. One is a control serum and the other two are leprosy patients. However, none of the sixteen others are HIV positive because they have only one glycoprotein band. Yet based on the NRL Australian criteria, which are the most stringent in the world, if these individuals were tested in Australia, they would all be HIV positive.

The authors of this paper concluded that “HIV-1 ELISA and WB results should be interpreted with caution when screening individuals infected with *M. tuberculosis* or other mycobacterial species. ELISA and WB may not be sufficient for HIV diagnosis in AIDS-endemic areas of Central Africa where the prevalence of mycobacterial diseases is quite high”.
This paper is very significant. The majority of AIDS patients in the world are TB patients and they are AIDS patients because they have a positive test. Yet according to Essex these tests in these patients are not sufficient to prove HIV infection.

Mr. Parenzee was born in Africa where he lived until he was 15. Each year there are 250,000 new cases of TB in South Africa but this represents only a fraction of the people who have been exposed to the mycobacterium that causes TB.
Second, AIDS patients have auto-antibodies.

In fact they have a plethora of auto-antibodies that react with their own, cellular proteins. Which means if the HIV proteins are in fact cellular one would expect their tests to be positive on that basis alone. Or such antibodies could also react non-specifically with the test kit proteins.

Third, the AIDS risk groups are characterised by exposures to a very large number and variety of antibody inducing stimuli such as semen, blood, factor VIII, foreign proteins, infectious agents and drugs. Including oral drugs since a study of cocaine use in New York City prostitutes showed that positive antibody tests were almost twice as prevalent in those whose drug use was oral rather than intravenous. All these factors have the potential to induce antibody formation and it is not difficult to appreciate that the more antibodies one has, and the more varied the mix, the more opportunities there will be for cross-reacting antibodies to react non-specifically with the proteins in the test kits.

The same argument can be extended to non-AIDS sick individuals. Sick individuals in general are expected to have a higher number and greater variety of antibodies than healthy people, just as AIDS patients do. In fact one can predict that in sick individuals of
all kinds, not just those in the AIDS risk groups, we would expect a higher level of antibodies and at least some of these antibodies would increase the likelihood of reacting in the HIV test.

There are published data to support this contention.
In 1990, in a study never followed up or repeated, a research group from the United States reported the results of “HIV” antibody tests, including “confirmation” with the Western blot, on nearly 90,000 hospital patients. The authors took great pains to exclude any patient who had even the remotest chance of being in an AIDS risk group or having any disease that could remotely be connected to AIDS. So much so it took half a page of text to list over 70 exclusion criteria and they even excluded patients with gun shot and knife wounds because this group has a slight preponderance of positive tests. This study found that up to 22% of men and 8% of women in the AIDS age groups, that is, 25-44 years and in hospital, classified at no risk for AIDS, were antibody positive.

Read the paper at www.theperthgroup.com/RESPONSE/St.Louis.pdf

Read the exceptions at www.theperthgroup.com/RESPONSE/St.LouisExceptions.pdf
Here are the percentage rates of positive tests at the top 8 hospitals. As you can see the rate of positive antibody tests is not insubstantial.

And Mr. Parenzee was sick and attending a hospital at the time he was diagnosed HIV positive but to the best of my knowledge, he was not in an AIDS risk group.

And if there are factors in non-AIDS risk individuals that cause positive tests, why can’t the same factors also operate in individuals who are at risk for AIDS? After all, diseases don’t discriminate. Gay men and drug users still get the diseases that everyone else gets. They don’t just get AIDS.

As a corollary to this, one would predict that when health improves, at least some positive antibody tests in previously sick individuals may revert to negative. Again there is evidence for this.

A 1991 paper published by Lange et al reported that reformed drug addicts, HIV positive on the ELISA and Western blot, lose their HIV antibodies. But because HIV is said to be for life, but these addicts lost their antibodies, their original, positive tests, were regarded as false positives. Yet nowadays drug addicts with positive antibody tests are
regarded as true positives and are said to be infected for life and in fact are the second highest risk group for HIV.
Finally, there is also a highly significant historical precedent that illustrates how misleading antibodies may be in regard to diagnosing retroviral infections.

In the mid 1970s Gallo discovered what he considered to be the world’s “first” human retrovirus in a patient with leukaemia. It was named HL23V and the evidence for its existence surpassed that of HIV because reverse transcription was found in fresh tissue without the need for culture and there was an EM showing particles in a density gradient at the retroviral density of 1.16 gm/ml.

Following the discovery of HL23V, some researchers decided to determine its prevalence using antibody tests. These included two of the best known HIV experts, Reinhard Kurth and Robin Weiss. They conducted a serological survey of humans and tested for antibodies that reacted with HL23V. They concluded that "The serological studies presented here and by others provide indirect evidence that the infectious mode of transmission remains a real possibility in humans, and suggests that infection with an oncornavirus [retrovirus] may be extremely widespread". One should add that the researchers also included three monkey viruses in their serological survey and found that humans also had widespread infection with these viruses.
Understandably, such studies raised a suspicion that such serological data were misleading. Was it possible that infection with a leukaemia causing virus was “widespread” while leukaemia itself was relatively rare? And since the vast majority of humans never come into contact with monkeys, how could infection with three monkey viruses be “widespread”?

EXTRA
One group of retrovirologists wrote “All investigators, however, pointed out that “the question of whether [retro]viruses have induced the antibodies…is impossible to decide. In an environment full of potentially related [antigenic] stimuli, nonviral antigens may also have been responsible for the induction of antibodies”. In their investigation they found “that the majority (if not all) of normal human sera contain naturally occurring…antibodies that react with the carbohydrate moieties of retrovirus envelope antigens”. These are the glycoprotein type of antigens considered “extremely important” for the serological diagnosis of “HIV”.
HL23V antibodies are non-specific—caused by exposure to “substances as diverse as normal components of serum, extracts of bacteria, and even nonprotein molecules such as glycogen”.

“The results are consistent with the idea that the antibodies in question are elicited as a result of exposure to many natural substances possessing widely crossreacting antigens and are not a result of widespread infection of man with replication-competent oncoviruses [retroviruses]”.

The answer came in 1980, five years after the discovery of HL23V, from two prestigious US research groups. HL23V antibodies are non-specific—caused by exposure to “substances as diverse as normal components of serum, extracts of bacteria, and even nonprotein molecules such as glycogen”. “The results are consistent with the idea that the antibodies in question are elicited as a result of exposure to many natural substances possessing widely crossreacting antigens and are not a result of widespread infection of man with replication-competent oncoviruses [retroviruses]”.

This discovery was of such significance that today nobody, not even Gallo, considers HL23V as being the first human retrovirus, or that such a retrovirus ever existed.

In our view the antigenic environment of the AIDS risk groups, which includes the fungal and mycobacterial diseases they get, may be the reason for positive “HIV” antibody tests. Not a retrovirus.

EXTRA
Following this Gallo accepted the evidence that the antibodies which reacted with proteins of HL23V were directed not against the proteins "but against the carbohydrate
moieties on the molecule that are introduced by the host cell as a post-transcriptional event, and which are therefore cell-specific and not virus-specific”. In other words, just as environmental antigens can induce antibodies that react with human red blood cells, which is why transfusions of uncrossed matched blood can kill a person, the environment can also induce antibodies that react with retroviruses. Both in the absence of exposure to foreign red blood cells or retroviruses.
This is a copy of Mr. Parenzee’s antibody test but it does not accord with the reporting recommendations set out by the NRL. It is unsigned, the word “REACTIVE” is used in regard to the Western blot but its meaning is undefined and although CONFIRMED POSITIVE the report does not document what band pattern was obtained.
Mr. Parenzee’s ELISA test was reactive but this does not prove he is HIV positive.

Since Mr. Parenzee’s “confirmatory” Western blot report does not document the band pattern his status as positive, indeterminate or negative cannot be verified.

One cannot rely on a “confirmatory” antibody test when a test done on the same specimen, is reported differently according to where or which laboratory performs the test.

Even if the Western blot test kit proteins are “HIV” and Mr. Parenzee has antibodies that react with them, this does not prove the antibodies are HIV.

CONCLUSION

This slide can be read
CONCLUSION

The only way to determine if the antibodies are HIV is to use HIV as a gold standard for comparison.

This has not been done.

At present this cannot be done

Presently there are no scientific data that prove a relationship between a positive antibody test and HIV infection.
In 1971 Sir Gustav Nossal wrote that antibody molecules possess “exquisite specificity...For each antigen there is a corresponding, different antibody. As with locks and keys only certain pairs fit”. Notwithstanding, in the same book Nossal acknowledged that “an antibody molecule made following the injection of one antigen frequently can combine also with a second antigen of a related or similar shape...In other words, the antibody cross-reacts with the second antigen”.

Since then many authors have embraced the term “promiscuity” to express the fact that antibodies may react with more than one antigen. Marchalonis states that ““epitope recognition promiscuity” is a property of antibodies of all vertebrate species...For many years, it was considered that a single antibody molecule bound only to the antigen to which it was raised, or at most to structurally homologous cross-reactive molecules. In fact the concept arose that monoclonal antibodies must be monospecific. The immunological community was shocked to find that B cells could be polyreactive in
binding to multiple antigens that were complex and ostensibly unrelated to one another”.

It is also asserted that “Promiscuity is not a new concept…many antibodies elicited against a particular antigen have also been shown to bind other, structurally unrelated antigens.” According to Avrameas “…antibodies are polyspecific, that is, they are able to react with various dissimilar antigens such as: proteins, nucleic acids” and "they are able to react with more than to self or non-self antigens, often without any apparent antigenic similarities". In 1997 Kramer et al noted that “high-affinity antibodies that have undergone antigen-driven somatic mutations are usually thought to be monospecific. Nevertheless, antibody cross-reactivity and polyspecificity have been observed since the earliest immunological studies” and “even high-affinity binding monoclonal antibodies are able to recognize more than one peptide epitope”.

Examples of the extent of antibody promiscuity are not difficult to find. In 1989 Baccala et al reported two monoclonal IgM natural autoantibodies (E7 and D23) that reacted with 11/12 unrelated antigens. In their 1997 study entitled “Molecular basis for the binding promiscuity of an anti-p24 (HIV-1) monoclonal antibody [CB1-4]”, Kramer et al reported reactivity against five unrelated peptides that competed with each other for binding to the paratope region of the antibody. The authors were able to construct binding supertopes derived from each peptide and “Data-base searches for proteins that match the supertopes resulted in the identification of more than 6000 heterologous proteins. A substantial number (>16%) [160] of those protein-derived peptides was able to bind CB4-1”. Furthermore, the authors were able to obtain and test 11 heterologous proteins containing CB4-1 binding supertope sequences found amongst the “50 strongest CB4-1 binding peptides”. These proteins included alcohol dehydrogenase (E. coli), UmuD (E. coli), candidapepsin (Candida albicans), myosin II heavy chain, non muscle (A. castellani) and X-Pro-dipeptidase (human)”. “All of them were recognized by CB4-1 in denatured and/or native from using solid phase enzyme-linked immunosorbant assay”.

In 2005 Predki et al stated “In the research lab, antibodies are commonly used tools for affinity purification, co-immunoprecipitation, quantitation and localization of proteins within tissues or cells. In the clinical setting, antibodies are used to quantitate protein
levels for diagnostic purposes, and their ability to either inhibit biological action or target specific cells for destruction forms the basis of their use as therapeutics. The success of each of these applications is largely due to, and contingent upon, the high affinity and specificity of antibodies for their antigen targets. Even though specificity is a hallmark of antibodies, cross-reactivity is not infrequently observed. Unrecognized, such cross-reactivity can have adverse consequences. The ability to assess and identify antibody cross-reactivity is an important but often inadequately addressed requirement for both research and clinical applications”. Predki et al also affirmed that “The literature is replete with examples of cross-reactive antibodies”; “Clearly, antibody cross-reactivity is very prevalent despite marketing efforts that suggest otherwise” and “The large number of cross-reactive antibodies is certainly cause for concern. However, perhaps more concerning are antibodies in current use with unrecognized cross-reactivity. Literature reports of cross-reactivity possibly represent the tip of a very large ‘iceberg’”.7 These authors presented the case for using protein microarrays as a “new tool for profiling antibody cross-reactivity” and tested a monoclonal antibody directed against a phosphopeptide from the kinase MAPK-APK2 in a microarray consisting of approximately 2000 proteins. “Signals from the protein microarrays were normalized by the amount of protein estimated on the array. The top ranked protein has the highest signal:protein ratio. The protein towards which the antibody was directed was ranked 17”. In other words, of the 40 proteins which reacted with this single antibody, binding by 16 was greater than that which occurred with cognate antigen. The authors also acknowledged that “the lack of a complete human proteome microarray prevents a comprehensive specificity analysis”. One can likewise note that the universe of antigens is not confined to the full complement of human proteins and reactivity is also dependent on many other factors including culture and testing conditions. Hence, at present, the true extent of antibody specificity is not measurable. Since a polyclonal antibody response is a set of monoclonal responses, this problem of defining antibodies and their cognate antigens is even further compounded.

In light of such evidence the scientific community has recognised that antigens and antibodies do not react monogamously and moved on from the notion of “one antigen,
one antibody”. Obviously, as Predki et al stated, “Unrecognized, such cross-reactivity can have adverse consequences. The ability to assess and identify antibody cross-reactivity is an important but often inadequately addressed requirement for both research and clinical applications”. Yet such “adverse consequences” appear to be under appreciated, including by clinicians. Indeed, in a straw poll undertaken by a colleague in a teaching hospital emergency department in 2006, all resident staff asked were of the opinion that antibody reactivity with a microbial protein proves infection with that microorganism.

It takes only moments to appreciate that if antibodies are promiscuous so too are antigens. Of primary interest to clinicians is not the chemistry of antibody/antigen interactions but whether or not the reactions observed in vitro between antibodies in a patient serum and a given antigen are specific for exposure to or infection with a particular antigen or microorganism. Our view is that the only way to obviate the problem of antibody promiscuity and determine the specificity of a serological test, (not to be confused with antibody specificity), is to measure it against a gold standard which best represents whatever the test is claimed to prove. However, in doing so one must distinguish between testing to confirm a syndromic diagnosis and testing to prove infection with a particular microorganism. If the former, the gold standard is the syndrome (however defined). If the latter, the gold standard must be the organism itself (isolation). In the literature there is a serious dearth of data in regard to the use of microbial isolation as a gold standard for serology, especially in the case of viruses. In view of this and the new appreciation of the unknowable extent of antibody/antigen cross-reactivities, what confidence can clinicians place in serological diagnoses of infectious agents?