

AFFIDAVIT

I, VALENDAR FRANCIS TURNER of Dalkeith, Western Australia, MAKE OATH AND SAY as follows:-

1. I am a registered medical practitioner in the State of Western Australia.
2. I graduated in Medicine from the University of Sydney in 1969.
3. I was awarded the Fellowship of the Royal Australasian College of Surgeons in 1977.
4. I was made a Foundation Fellow of the Australasian College for Emergency Medicine in 1983.
5. I am a senior consultant emergency physician and since 1978 have practised in all Perth teaching hospitals as well as a number of peripheral hospitals.
6. My professional activities have included clinical and administrative duties, teaching medical students and junior hospital staff, lecturing at the University of Western Australia, Head of Department at the Royal Perth and Swan District Hospitals and attendance at national and international conferences and meetings.
7. I have authored and co-authored several papers in peer-reviewed, scientific journals (Annexure 2).
8. My present employer is the Department of Health, Western Australia where I am medical co-director of the West Australian Health Call Centre.
9. Since AIDS appeared in 1981 I have belonged to a group of scientists known as "The Perth Group" led by biophysicist Eleni Papadopulos-Eleopulos.
10. Over the past 25 years the Perth Group has extensively researched the HIV/AIDS scientific literature and published several papers in peer-reviewed, scientific journals (included in Annexure 2) as well as the popular press and on the Internet (Annexure 3 and The Perth Group website www.theperthgroup.com).
11. I am an invited member of the South African Presidential AIDS Advisory Panel and spoke at this conference on behalf of the Perth Group in July 2000.
12. My report is attached (Annexure 1).
13. The views expressed in this report are my own and do not reflect those of my previous or current employer(s).
14. The statements made in this affidavit are my opinion based on study of the scientific literature and are correct to the best of my knowledge, information and belief.

SWORN by the Deponent) -----
)
At Perth)
)
On the ---- day of -----)
2006)

Before me

Justice of the Peace

Western Australia

ANNEXURE 1 TO AFFIDAVIT by VALENDAR FRANCIS TURNER

A. VIRUS ISOLATION

1. According to the HIV/AIDS experts the HIV theory of AIDS is as follows: There exists a unique virus, classified as a retrovirus and referred to as human immunodeficiency virus (HIV). This entity is transmitted from person to person principally by blood, sexual contact and from infected mothers to their unborn children. When HIV gains access to the body it (a) infects and causes the death of a subset of white blood cells of the immune system known as CD4 lymphocytes; (b) causes the immune system to produce antibodies that react with biochemical constituents (proteins) of the virus particle. Detection of such antibodies is used to diagnose individuals infected with HIV. After infection and typically over many years, the numbers of CD4 cells gradually diminish leading to a state known as acquired immune deficiency ("AID"). In turn AID is followed by the development of a number of different ("AIDS indicator") diseases which constitute the clinical AID syndrome ("S"). Hence a person has AIDS when he or she has HIV and develops one or more of these diseases. HIV does not directly cause the approximately 30 different AIDS indicator diseases but indirectly by its effect on the immune system.
2. The research conducted by my colleagues and I over the past two decades leads me to conclude this theory is unproven.
3. A virus is a microscopic particle (a minute piece of matter) made up of a nucleic acid genetic "blueprint" (RNA or DNA) and some proteins. Viruses are so small they lack the space necessary to contain the raw materials from which to produce the substances and energy required for their replication (reproduction). Hence, in order to replicate, viruses, unlike bacteria for example, are obligate parasites of living cells.
4. Particles with the appearances of a virus are not regarded as a virus unless there is proof they replicate in this manner. Virus-like particles fulfilling this property are referred to "infectious particles" and then and only then can such particles be regarded as a virus.
5. Retroviruses belong to a Family of virus particles which have in common RNA as their genetic blueprint and a protein enzyme (a biological catalyst that accelerates the rate of chemical reactions) called reverse transcriptase (RT). The function of this enzyme is to copy the viral RNA into DNA, a process known as reverse transcription. It is referred to as "reverse" because its direction runs contrary to the previously held but no longer accepted "biological dogma"— that in biological systems information flow is one way only. That is, from DNA to RNA.
6. Retrovirus particles are spherical in shape with a diameter of approximately 100nm. Ten thousand such particles could fit side by side in the length of one millimetre.
7. Retrovirus particles can only be visualised and their morphology studied using the electron microscope (EM). The latter is an instrument in which

- an electron beam, rather than light, is used to 'illuminate' the object being studied. The advantage of the EM is its ability to visualise and resolve objects and features of those objects not possible to perform with the light microscope. The resolving power of the EM is about 0.2 nanometers, about the same distance separating two atoms in a solid. In this regard the EM performs about a thousand times better than the light microscope.
8. Morphology is the branch of biology that concerned with the form and structure of organisms. In regard to retroviruses such study elucidates the size, shape and general and distinguishing features of the viral particles.
 9. Virologists claim to prove the existence of viruses by carrying out a number of laboratory procedures collectively referred to as "virus isolation". In regard to "HIV", the interpretation of these data as proof of virus isolation is highly problematic. This is because (a) each phenomenon has well known and accepted causes other than a retrovirus. Some were discovered decades before the AIDS era by scientists some of whom are now HIV experts; (b) the "isolation" experiments were not accompanied by correct or sometimes even by any controls. The latter are experiments carried out at the same time on material from patients who are sick with similar clinical, biochemical and immunological abnormalities as AIDS patients but who do not have AIDS nor are in a risk group for AIDS. Controls are an essential component of retrovirus isolation experiments because "retrovirological phenomena" may arise, even spontaneously, in material known not to be infected with a retrovirus.
 10. Professor Luc Montagnier and his colleagues are accepted to be the scientists who first isolated and hence discovered HIV. Their experiments were reported in the May 20th 1983 issue of *Science* and typify the problems listed in (9). Montagnier's paper is entitled "Isolation of a T-lymphotropic retrovirus [HIV] from a patient at risk for acquired immune deficiency syndrome (AIDS)". However, what Montagnier reported as "isolation" was detection of an enzyme activity, that is, reverse transcription—not purification of virus-like particles proven to be infectious. In fact Montagnier did not purify "HIV".
 11. Subsequent researchers have not performed experiments substantially differently from those reported by Montagnier and his colleagues. Hence, based on the currently available data it is not possible to claim that a unique retrovirus has been isolated from the tissues of AIDS patients.

B. ANTIBODY TESTS

12. Notwithstanding, virus isolation is not the routine method of diagnosing HIV infection because it is technically demanding, time consuming and expensive.
13. From 1983/84, that is, from the time reports of the discovery of HIV appeared in the scientific literature, scientists have attempted to use tests that detect antibodies to diagnose infection with HIV. Such tests became generally available in 1985 and their current widespread availability and

use are largely dependent on test kits supplied by biotechnology companies.

14. Individuals who fulfil criteria deemed a positive test result, (which vary considerably), are referred to as being “HIV antibody positive”. This term is synonymous with “HIV positive” and neither term means “HIV” particles have been isolated from a person.
15. Antibodies are not viruses. Antibodies and hence a positive antibody test may be indirect evidence of a viral infection but if and only if the antibodies are proven specific. An antibody test that is 100% specific means that a virus is the only cause of a positive test. Nothing else but the virus is capable of causing a positive test. HIV/AIDS experts claim their antibody testing methods are virtually 100% specific for HIV infection.
16. Antibodies develop because the immune system has the inherent ability of distinguishing between “self” and “non-self”. That is, the immune system can detect the presence of foreign material such bacteria and viruses that gain access to the body. Any substance that induces the formation of antibodies is known by the generic term “antigen” (from ANTIBody GENerating). Hence when a person or animal is infected with a foreign substance, such as a protein from a virus, one can predict that antibodies will develop. For example, antibodies are formed following natural infections with measles or chicken pox. The same occurs following immunisations for the same viruses. Antibodies are detectable in the bloodstream about ten days after an infection and reach their peak concentration in about three weeks.
17. Hence within days of a natural infection or immunisation one can also predict that if one obtains serum from a subject and mixes it with the viral proteins a reaction will occur.
18. Serum is the yellowish fluid in which red blood cells are suspended and in which all the person’s antibodies are dissolved. Serum forms about half the blood volume and is separated from the red blood cells by spinning the blood specimen in a centrifuge. Because serum is used to detect antibodies, using antibodies to diagnose infections forms part of the practice known as serology.
19. Hence the presence of antibodies is demonstrated by the fact they react with the inducing antigen. The laboratory scientist detects the occurrence of a reaction because it results in a detectable, physical alteration in the reaction mixture. Commonly this is a colour change which can be quantitated using a machine such as a spectrophotometer. In some antibody tests the colour change is noted and interpreted by the laboratory technician.
20. To perform a test to determine whether there are antibodies that react with “HIV” two things are required: (a) the “HIV” proteins. (b) a serum specimen from the person being tested.
21. To obtain the HIV proteins first it is necessary to purify the virus particles. This is because viruses replicate only in cells and cells themselves, like viruses and living matter in general, are also made up of RNA and

- proteins. Luc Montagnier, the discoverer of HIV, agrees with this commonsense requirement. During an interview in 1997, in response to a question about what was necessary to characterise the HIV proteins, he answered "...analysis of the proteins of the virus demands mass production and purification. It is necessary to do that".
22. Yet in their 1983 *Science* paper, in which Montagnier and his colleagues claimed to have first isolated and purified HIV, they did not publish any electron micrographs to prove that the material which they called "purified virus" contained particles bearing the morphology of retroviruses. Or that it contained any particles of any kind, retroviral or non-retroviral. In the same 1997 interview, when asked about this omission, Montagnier replied such EMs were taken but, despite a "Roman effort", none revealed particles "with the morphology typical of retroviruses".
 23. Also at the same interview, Montagnier repeatedly stated that he did not purify HIV. And in his opinion neither did the principal US research team led by Dr. Robert Gallo when his team reported their isolation of HIV on May 4th 1984.
 24. Hence, the discoverer of HIV had no proof of isolation or purification of a new retrovirus, making it impossible for Montagnier or anyone else using the same method to characterise particular proteins as those of a retrovirus infecting individuals with AIDS.
 25. Research published since confirms that particles claimed to be HIV have not been purified.
 26. Research published since shows that the proteins considered unique to HIV may be found in "non-HIV-infected" cells.
 27. Nonetheless, HIV experts apparently believe there are proteins belonging to a retrovirus HIV and claim to use them to detect "HIV antibodies" and thus prove "HIV" infection.
 28. Even if there was proof these proteins are those of a purified, infectious particle proven to be a retrovirus the fact that patients have antibodies that react with these proteins is not proof the antibodies are caused by infection with HIV. This is because antibodies induced by a particular antigen react not only with that antigen but may also react and with other antigens. This is a critically significant issue and one which I believe has been disregarded in the quest to discover the cause of AIDS. The implications of this fact are explained in the following examples (29, 30, 33).
 29. Humans who are blood group A contain antibodies that react with the red blood cells of humans who are blood group B. And *vice versa*. If blood from either person is inadvertently transfused into the other, antibodies in the recipient will react with the red blood cells from the donor, thereby forming intravascular clots and shutting down the recipient's circulation. The result may prove fatal. However, no scientist would argue that the antibodies are caused by "infection" with another person's blood or claim their presence proves "infection" with human blood. In fact scientists believe such antibodies develop soon after the infant leaves the sterile

- confines of the uterus and is exposed to a wide variety of foreign environmental substances including germs. However, the antibodies produced as a result of these antigenic stimuli produce perchance antibodies that react with the antigens present on the red blood cells of other humans. Hence the absolute need to cross-match blood before transfusions are undertaken.
30. Glandular fever is a common disease caused by infection with the Epstein-Barr virus. Infection with this virus not only results in antibodies that react with the Epstein-Barr virus but also antibodies that react with the red blood cells of sheep and horses. In fact, when faced with a patient whose history, symptoms and signs are suggestive of glandular fever, physicians order tests for the latter rather than the Epstein-Barr virus antibodies. Yet such patients are not infected with animal blood and neither is animal blood the cause of this disease.
 31. Hence we must conclude that it is not possible to claim *ipso facto* that a reaction between an antibody and an antigen proves that person has been exposed to or infected with that antigen. Or with a bacterium or virus that contains that antigen.
 32. When an antibody reacts with an antigen other than the antigen that induced it, the reaction is referred to as a "cross-reaction". The potential to produce confounding and hence misleading reactions is a well known characteristic of all antibody molecules. (Wanted) "reactions" and (unwanted) "cross-reactions" can be regarded analogous to drugs having (wanted) "effects" and (unwanted) "side effects". In both cases what is "unwanted" detracts from the ability to achieve a desired outcome. This is why serology has been described as "similar to determining the exact shapes of clouds by the shadows they cast on the ground".
 33. A germane example is the fact that 62% of patients who suffer an attack of measles develop antibodies that react with six of the ten, so named, "HIV" proteins. HIV experts accept these are not antibodies caused by "HIV" infection. They are measles antibodies which cross-react with the proteins present in the "HIV" antibody test kits.
 34. Immunologists believe humans are capable of elaborating perhaps as many as one million different antibody molecules. Given this repertoire and their proven proclivities for cross-reactions it is impossible to conclude, merely on the basis of reactions, this proves the identity of the participating antibodies.
 35. The only means by which antibody reactions can be proven specific for a putative agent is to compare the reactions with that agent. This is a wholly empirical exercise best illustrated by another familiar example.
 36. Pregnancy tests are antibody tests. To prove the veracity of a blood test to detect pregnancy one compares positive and negative test results against the presence or absence of babies being born. In the case of a 100% accurate test one would expect all women who had babies to have a positive test and all women who did not have babies to have a negative test. In other words, the test parameters, including specificity for detecting

- pregnancy, are proven by using the baby as the “gold standard”. In the case of “HIV”, the antibody tests are claimed to prove HIV infection. Hence the gold standard for such a test must be HIV itself, as proven by isolating the virus. In this case HIV is “the baby” that authenticates whether or not the reactions between the antibodies and the test kit proteins are caused by infection with “HIV”. This gold standard principle is used to verify tests throughout clinical medicine but has been ignored by the HIV/AIDS experts in regard to determining antibody test parameters for HIV infection. Nowhere in the scientific literature are there reports of antibody tests verified independently of an antibody/antigen reaction against a virus isolation gold standard.
37. Since HIV isolation itself is problematic this gold standard verification cannot presently be done.
 38. Hence in my view there are no scientific reasons for asserting that a person who is “HIV antibody positive” is infected with a retrovirus HIV.
 39. This conclusion does not negate that facts that (a) the antibodies are present; (b) whatever their genesis, within the AIDS risk groups they predict the presence or development of illness.
 40. HIV/AIDS experts are aware that persons may have antibodies that react with one or several of the “HIV” proteins and yet not be infected with HIV. In fact they explain these as “biological false positives” caused by cross-reacting, “non-HIV” antibodies.
 41. HIV experts claim they can distinguish between “true” (caused by HIV) and “cross-reactions” (not caused by HIV) by using second, third and fourth generation antibody tests and arranging these into various test algorithms. By developing such methods they claim HIV infection can be diagnosed with the utmost accuracy. I reject such claims because no amount of “technological tinkering” can obviate the fundamental need to verify all antibody tests, no matter what methods are used and in what arrangement they are conducted, against the virus isolation gold standard.
 42. One such testing algorithm, used in most countries including Australia, includes an antibody test known as the Western blot. This test is said to act as a “supplementary” test to “confirm” other positive antibody “screening” tests which HIV experts themselves consider less than ideally specific for diagnosing HIV infection. In the Western blot procedure the ten or so “HIV” proteins are impregnated at separate sites along the length of a nitrocellulose strip. The sites where each protein is present are identified by ‘p’ (for protein) followed by a number (which is that protein’s molecular weight in thousands). For example, p18 or p24. Three of the proteins are labeled ‘gp’ (for glycoprotein) because these proteins (gp41, gp120, gp160) incorporate sugar moieties in their structure. When serum is added and the strips developed the sites of antibody/protein reactions show up as coloured bands. The laboratory technician visualises these bands and hence determines which proteins have antibodies reacting with them. The Western blot test is reported according to the number and pattern of bands that appear on the strip. HIV experts assert that certain

- Western blot band patterns prove HIV infection and only these patterns are reported as positive. In Australia a negative Western blot is one with no bands. Any pattern that is neither positive nor negative is reported as indeterminate. Most indeterminate results are considered not due to HIV infection.
43. It should be noted that 40% of healthy blood donors have at least one Western blot band. HIV experts assert these bands are not caused by HIV antibodies but by cross-reacting “non-HIV” antibodies. Hence antibodies that react with the “HIV proteins but which are not caused by “HIV” are highly prevalent in healthy people at no risk of developing AIDS.
 44. Healthy people have relatively fewer antibodies than AIDS patients who typically have elevated levels of antibodies in general. The more antibodies one has the greater the opportunity for cross-reactions. Hence a scientist would expect unhealthy individuals including AIDS patients to have a much higher prevalence of “non-HIV” antibodies reacting in the “HIV” test kits than healthy individuals. Without viral isolation gold standard data it is impossible to determine what proportion, if any, of “HIV positive” individuals react because of HIV antibodies rather than “non-HIV” antibodies.
 45. Even if we accept that the proteins in the Western blot strips are HIV in origin there are many problems with this “confirmatory test”. The most significant is that, like all antibody tests used alone or in combination, the specificity of the Western blot has not been determined using a virus isolation gold standard.
 46. The Western blot is not standardised. That is, combinations of bands that “confirm” HIV infection in one laboratory, institution or country may not “confirm” it in another. For example, the criteria defining a positive Western blot in New York City are not the same as the criteria used in Australia or Africa. To use an analogy, no doctor would accept the existence of different electrocardiographic criteria for diagnosing a heart attack around the world. A patient cannot have a “heart attack positive ECG” in New York City which is not a “heart attack positive ECG” in Sydney, Australia (Annexure 4).
 47. The global variation in the criteria for a positive Western blot make it impossible to claim the specificity of such a test can even be determined.
 48. For the reasons above I am of the opinion there is no basis in antibody testing to consider Parenzee infected with a retrovirus.
 49. Thus I conclude there is no scientific proof Parenzee transmitted a retrovirus to his sexual partners.

C. VIRAL LOAD TESTS

1. According to the HIV/AIDS experts HIV is a retrovirus with a unique RNA genome. The term genome is defined as the full complement of genes and the genome is necessary for the HIV particle to reproduce the virus particles.

2. To identify RNA as that of a retrovirus a scientist must first purify the viral particles. This is because the cells in which viruses replicate also contain RNA. Since the particles said to be “HIV” have not been purified then it is not possible to claim a particular RNA is that of “HIV”.
3. Experts claim they are able to determine the number of RNA molecules in a specimen of blood using several methodologically different tests based on a biochemical technique known as the polymerase chain reaction (PCR). The PCR is a technique which utilises a small piece of the RNA of interest to quickly multiply and detect the same RNA if present in test material. Experts refer to the number obtained by the PCR tests as the “HIV “viral load” and state such measurements are essential for the clinical management of patients who are HIV positive. The “viral load” is said to be the most reliable prognostic indicator for HIV infected individuals and is also said to guide the choice and determine the effectiveness of “antiretroviral” drug therapy.
4. In 1996 one leading international HIV expert published a paper in *Science* in which he stated “In the total blood volume, the number of virions can equal 10^6 [one million] particles per milliliter, or an estimated 10^9 [1 billion] HIV particles per milliliter”.
5. However, (a) there are no published correlations between the “viral load” (number of RNA molecules) and the number of particles considered to be “HIV” in blood. This is because to date no HIV researcher has published even one electron micrograph demonstrating the existence of even one such particle in the blood of even one AIDS patient; (b) RNA molecules are not viral particles and viral particles are needed for infection to take place. Hence the term “viral load” is both unfounded and misleading.
6. HIV experts acknowledge there are problems measuring the actual “viral load”. Different laboratories and different PCR tests obtain markedly different results for the same “viral load” on the identical specimens (Annexures 5 and 6).
7. These “viral load” data confirm, for example, that in one particular test the “viral load” could be 60% lower or higher than the mean value; (b) if another test is used on the same specimen the mean obtained is halved with a 30% variation around that mean. In other test data a “viral load” could be 295,000 or less than 400 (considered zero) depending on which assay is used to obtain the measurement. Mathematically this range (295000/0) is infinite.
8. Inter-laboratory and inter-test variability is used to justify expert recommendations that patients should always be tested by the same laboratory using the same assay. In other words, HIV experts are not concerned with the actual value of “viral load”. This leads one to question how is it possible (a) to make general, categorical statements about the biological relevance of “viral load”; (b) transpose such statements to individual patients whose “viral load” is being monitored for the same purposes. That is, making management decisions in regard to “antiretroviral” therapy and advising on prognosis. If the reliability of

- measurements of body chemistry were as insubstantial as “viral load”, managing fluid and electrolyte balance would be unworkable. For example, if one method of measuring serum sodium produced a result half that of another, the latter would make no sense because it would be incompatible with life.
9. Roche, the manufacturer of the Amplicor HIV-1 assay, one of the most commonly used RNA “viral load” tests, has withdrawn their test from sale.
 10. In order to count RNA molecules a scientist must have a test able to distinguish between “HIV” RNA and all other RNAs. To use an analogy, if one wishes to count the number of apples in an orchard where all manner of fruit is grown, first one must be able to recognise an apple.
 11. If, as HIV experts assert, the viral load measures “HIV” RNA, then this test must be capable of distinguishing between “HIV” RNA and all other RNAs. That is, by recognising “HIV” RNA, *ipso facto* the test proves HIV infection. However, according to the US Centers for Disease Control (CDC), “In adults, adolescents, and children infected by other than perinatal exposure, plasma viral RNA nucleic acid tests should **NOT** be used in lieu of licensed HIV screening tests (e.g., repeatedly reactive enzyme immunoassay” (emphasis in original). (“The “licensed screening tests” and “enzyme immunoassay” are antibody tests).
 12. However, according to HIV experts, the role of “viral load” tests is confined to measuring the “quantity of virus” in patients whose “HIV” infection has first been proven by antibody tests.
 13. One group of HIV experts states “Plasma viral [RNA] load tests were neither developed nor evaluated for the diagnosis of HIV infection”.
 14. Roche, the company that manufactured and sold the AMPLICOR HIV-1 RNA MONITOR test include the following statement in the test kit packet insert: “The COBAS AMPLICOR HIV-1 MONITOR Test v1.5 is not intended to be used as a screening test for blood or blood products for the presence of HIV-1 or as a diagnostic test to confirm the presence of HIV-1 infection”.
 15. Hence the test that the HIV experts assert able to count “HIV” specific RNA molecules is not considered capable of diagnosing “HIV” infection.
 16. I conclude these tests are meaningless in terms of their ability to identify RNA as “HIV” let alone measure the “viral load”.

D. ACQUIRED IMMUNE DEFICIENCY (Declining CD4 cell count)

1. Physicians treating HIV positive and AIDS patients monitor the number of CD4 cells in the peripheral blood. A decline in their numbers is interpreted as proof the cells are being killed as a consequence of HIV infection.
2. The fact that CD4 cells are diminished in the blood stream is not proof the cells are being killed. Their disappearance no more proves they are dead than the disappearance of the population from cities at Easter proves its citizens are dead.
3. CD4 cells are counted by means of antibodies that bind to a molecule on the cell surface known as the CD4 “receptor”. Evidence published by HIV

- experts shows that the measured decrease in CD4 cells may not be due to their selective destruction but by the loss of their surface receptors which are no longer available to bind to the antibody molecules.
4. In cultures of CD4 cells chemical agents induce such changes without killing the cells.
 5. *In vitro* (“test tube”/out of the body experiments) performed to prove HIV kills CD4 cells suffer from the fact it is not possible to add pure HIV to CD4 cell cultures. This is because to date no researcher has purified HIV. Thus such experiments, even if they did reveal declining numbers of cells in cultures, cannot distinguish between “HIV” as the cause and the many other substances which contaminate “HIV”.
 6. Data show that even when such “impure HIV” is added to cultures, “HIV” does not cause a more significant decline in CD4 cell numbers than that observed in control cultures to which culture material without “HIV” has been added.
 7. There are data that in the AIDS risk groups, such as drug addicts and haemophiliacs, individuals may develop low CD4 cells before they become HIV positive. In other words, the cause (HIV) follows the effect (low CD4 cells).
 8. Montagnier states “This syndrome [AIDS] occurs in a minority of infected persons, who generally have in common a past of antigenic stimulation and of immune depression before LAV [HIV] infection” (emphasis added).

E. SEXUAL TRANSMISSION

1. Infectious diseases are caused by microbes transmitted from person to person. Hence a person infected with a particular microbe transmits the microbe to another, uninfected person who in turn transmits it to others.
2. What distinguishes sexually transmitted infections from other infections is the fact their causal microbes are present semen and cervico-vaginal fluids.
3. There is not one single, published study from any country of sexual transmission of HIV based upon evidence of HIV in genital secretions.
4. The only evidence said to prove heterosexual transmission is epidemiological, that is, the study of the relationship between a positive antibody test and sexual behaviours. Such studies rely on inferences based on statistical associations between sets of observations.
5. In all the published studies of sexual transmission conducted in gay men, as well as many in heterosexuals, sexual partners are not linked either by known sexual contact with each other (contact tracing) or by sexual contact with individuals whose antibody status, positive or negative, is known.
6. The majority of studies said to prove heterosexual transmission are cross-sectional. That is, if both sexual partners are found to be HIV positive and the epidemiologist believes there is no other reason to explain the positive test, it is assumed one partner transmitted HIV to the other by means of

- sexual contact. The direction of transmission (male to female, female to male) is assigned arbitrarily.
7. There are a few studies on sexual partners where one is HIV positive and the other is not. The couples are followed over time to determine what happens to the antibody status of the HIV negative partner. These are known as longitudinal or prospective studies.
 8. Professor Nancy Padian of the UCSF Departments of Obstetrics, Gynecology and Reproductive Sciences has conducted the best available and longest studies of heterosexual transmission of HIV. From the cross-sectional component of her ten year study Padian calculated the "infectivity for male-to-female transmission is low, approximately 0.0009 per contact" and "approximately eight-times more efficient than female-to-male transmission". In other words, the per contact probability is 1/1111 for male to female transmission and 1/8888 for female to male transmission. These per contact risks are in marked contrast to gonorrhoea for example, where the per contact risk is 1/2 for male to female transmission and 1/4 for female to male transmission. One should note that the probabilities for female to male transmission were based on two cases, the validity of which Padian herself questioned. In fact she emphasised the limitations of cross-sectional studies and this was the impetus for her prospective study.
 9. One should also note that Padian accepted a positive antibody test as proof of HIV infection and thus transmission. However, the criteria she and her colleagues accepted as a positive Western blot and "confirming" HIV infection are not considered a positive Western blot and proof of HIV infection in most countries, institutions or laboratories. Including Australia.
 10. This table compares the probabilities of a female contracting infection from her infected male partner for both "HIV" and gonorrhoea after a number of sexual contacts with him.

CUMULATIVE PROBABILITY OF INFECTION

Number of Contacts	Probability of infection	
	Gonorrhoea	"HIV"
0	0%	0%
1	50%	0.09%
2	75%	0.18%
3	88%	0.27%
4	94%	0.36%
5	98%	0.45%
777		50%
3333		95%

This table compares the probabilities of a female remaining free of infection for both gonorrhoea and “HIV”

CUMULATIVE PROBABILITY OF NO INFECTION

Number of Contacts	Probability of NO infection	
	Gonorrhoea	"HIV"
0	100%	100%
1	50%	99.9%
2	25%	99.8%
3	13%	99.7%
4	6%	99.6%
5	2%	99.6%
777		50%
3333		5%

On average, to attain a probability of 50% of “HIV” infection the female would need to have sex 777 times with her male partner. To attain a 95% chance would require 3333 sexual contacts. Assuming both partners are capable of having sex every three days *ad infinitum* it would take 6.3 and 27.4 years respectively to transmit HIV to the woman. In regard to female to male transmission (which Padian calculated to be 8 times less efficient), on average it would take 6200 and 27000 contacts and a period of 51 and 222 years respectively for the man to become infected from his female partner.

11. The prospective component of Padian’s study recorded the outcome of HIV negative partners in couples where the other partner was already HIV positive. This part of the study lasted six years during which time the study participants underwent regular and intensive counseling in regard to safe sexual practices. Nonetheless, even after six years, 25% of couples still did not consistently use condoms. However, none transmitted or became infected with HIV.
12. Consistent use of condoms does not equate to no exposure to genital secretions. According the CDC, the typical pregnancy failure rate of male condoms during the first year of use was 15% for male condoms while “consistent” male condom users have a failure rate of 2%. In regard to the female condom “The estimated 12-month failure rate for pregnancy prevention among the 147 women was 26%. Of the 86 women who used this condom consistently and correctly, the estimated 12-month failure rate was 11%”.
13. In Africa, where the predominant mode of transmission of “HIV” is said to be heterosexual, a retrospective study concluded “The probability of HIV transmission per sex act in Uganda is comparable to that in other populations”.

14. HIV experts claim that the presence of “non-HIV” sexually transmitted diseases facilitates the transmission of “HIV”. However, in a large, well designed and executed study on the effect of sexual behaviour intervention on transmission of “HIV” in Uganda, the authors reported a reduced incidence of herpes simplex virus type 2 (“HSV2- a proxy measure of unprotected sexual contact”), as well as a significant reduction in acute syphilis, gonorrhoea, and unprotected casual sex in the intervention group. Yet there was no effect on HIV incidence despite the fact that an “apparently appropriate intervention that reduced other STDs and was implemented on a huge scale with great care and commitment”.
15. These data beg the question of heterosexual transmission of HIV. In other words, there is no proof that “HIV” is sexually transmitted.
16. These epidemiological data are consistent with their being no proof that a retrovirus has been isolated from AIDS patients.

ANNEXURE 2 TO AFFIDAVIT by VALENDAR FRANCIS TURNER

PERTH GROUP PUBLICATIONS Scientific Press

1. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, et al. Mother to Child Transmission of HIV and its Prevention with ATZ and Nevirapine. Perth: The Perth Group, 2001:204.
2. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Alfonso H, Page B, Causer D. Questions about results reported with potent antiretroviral therapy for human immunodeficiency virus type 1 infection. *Journal of Infectious Diseases* 2000; 181:1518-1519.
3. Papadopulos-Eleopopulos E, Turner VF, Papadimitriou JM, Bialy H. AIDS in Africa: Distinguishing fact and fiction. *World Journal of Microbiology and Biotechnology* 1995; 11:135-143.
4. Papadopulos-Eleopopulos E, Turner VF, Papadimitriou JM, Causer D. Factor VIII, HIV and AIDS in haemophiliacs: an analysis of their relationship. *Genetica* 1995; 95:25-50.
5. Papadopulos-Eleopopulos E, Turner VF, Papadimitriou JM, Hedland-Thomas B, Causer D, Page B. A critical analysis of the HIV-T4-cell-AIDS hypothesis. *Genetica* 1995; 95:5-24.
6. Papadopulos-Eleopulos E. A Mitotic Theory. *Journal of Theoretical Biology* 1982; 96:741-758.
7. Papadopulos-Eleopulos E. Reappraisal of AIDS: Is the oxidation caused by the risk factors the primary cause? *Medical Hypotheses* 1988; 25:151-162.
8. Papadopulos-Eleopulos E, Hedland-Thomas B, Causer DA, Dufty AP. An alternative explanation for the radiosensitization of AIDS patients. *International Journal of Radiation Oncology and Biological Physics* 1989; 17:695-697.
9. Papadopulos-Eleopulos E, Hedland-Thomas B, Causer DA, Turner VF, Papadimitriou JM. Changes in thiols and glutamate as consequence of simian immunodeficiency virus infection. *Lancet* 1991; 338:1013-4.
10. Papadopulos-Eleopulos E, Turner VF, Causer DS, Papadimitriou JM. HIV transmission by donor semen. *Lancet* 1996; 347:190-1.
11. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Oxidative stress, HIV and AIDS. *Research in Immunology* 1992; 143:145-8.
12. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Kaposi's sarcoma and HIV. *Medical Hypotheses* 1992; 39:22-9.
13. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Has Gallo proven the role of HIV in AIDS? *Emergency Medicine [Australia]* 1993; 5:113-123.
14. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Is a positive Western blot proof of HIV infection? *Bio/Technology* 1993; 11:696-707.
15. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, et al. Global voices on HIV/AIDS. Heterosexual transmission of HIV in Africa is no higher than anywhere else. *British Medical Journal* 2002; 324:1035.

16. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, et al. A critique of the Montagnier evidence for the HIV/AIDS hypothesis. *Medical Hypotheses* 2004; 63:597-601.
17. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, et al. High rates of HIV seropositivity in Africa-alternative explanation. *International Journal of STD & AIDS* 2003; 14:426-427.
18. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Causer D, Alphonso H, Miller T. A critical analysis of the pharmacology of AZT and its use in AIDS. *Current Medical Research and Opinion* 1999; 15:1s-45s.
19. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Causer D, Page BA. HIV antibody tests and viral load--more unanswered questions and a further plea for clarification. *Current Medical Research and Opinion* 1998; 14:185-6.
20. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Stewart G, Causer D. HIV antibodies: further questions and a plea for clarification. *Current Medical Research and Opinion* 1997; 13:627-34.
21. Turner VF. Reducing agents and AIDS--Why are we waiting? *Medical Journal of Australia* 1990; 153:502.
22. Turner VF. The HIV Western blot. *Medical Journal of Australia* 1994; 160:807-808.
23. Turner VF. Inactivation of HIV in factor VIII. *Medical Journal of Australia* 1998; 168:366.
24. Turner VF. HIV drug remains unproven without placebo trial. *Nature* 2005; 434:137.
25. Turner VF. Detection of acute HIV infections. *New England Journal of Medicine* 2005; 353:631-3; author reply 631-3.

ANNEXURE 3 TO AFFIDAVIT by VALENDAR FRANCIS TURNER

PERTH GROUP PUBLICATIONS Magazines, Popular Press and Online

1. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Bialy H. The Haemophilia Connection. Continuum 1995; 3:17-19
<http://www.theperthgroup.com/CONTINUUM/HaemophiliaConn.pdf>.
2. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Causer D. Why no whole virus? Continuum 1997; 4:27-30
3. Papadopulos-Eleopulos E. Looking back on the oxidative stress theory of AIDS. Continuum 1998; 5:30-35
<http://www.healtoronto.com/oxstress.html>.
4. Papadopulos-Eleopulos E, Turner V, Papadimitriou J, Page B, Alfonso H, Causer D. Distinguishing between true and "official" HIV infection. BMJ Online 2003
<http://bmj.bmjournals.com/cgi/eletters/326/7387/495#33450>.
5. Papadopulos-Eleopulos E, Turner VF. Deconstructing AIDS in Africa. The Independent Monthly 1994:50-51
6. Papadopulos-Eleopulos E, Turner VF. Reconstructing AIDS in Africa-Reply to Kaldor and Ashton. The Independent Monthly 1995; February:23-24
7. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM. Virus Challenge. Continuum 1996; 4:24-27
<http://www.altheal.org/continuum/Vol4no1.pdf>.
8. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, et al. HIV in South Africa. Brit Med J 2003
<http://bmj.com/cgi/eletters/326/7387/495#30348>.
9. Papadopulos-Eleopulos E, Turner VF, Papadimitriou JM, Causer D. The Isolation of HIV: Has it really been achieved? Continuum 1996; 4:1s-24s
www.virusmyth.net/aids/data/eprelypd.htm.
10. Turner VF. Where we have gone wrong? Continuum 1998; 5:38-44
11. Turner VF, McIntyre A. The Yin and Yang of HIV. NEXUS 1999; 6:29-36
<http://www.theperthgroup.com/POPPAPERS/yinyang.html>.

ANNEXURE 4 TO AFFIDAVIT by VALENDAR FRANCIS TURNER

GLOBAL VARIATION IN THE CRITERIA DEFINING A POSITIVE HIV WESTERN BLOT

HIV WESTERN BLOT STRIP*		AFR	AUS	FDA	RCX	CDC 1	CDC 2	CON	GER	UK	FRA	MAC
ENV	p160	ANY 2	ANY 1	ANY 1	ANY 1	p160/ p120 AND p41	p160/ p120 OR p41	p160/ p120 OR p41	ANY 1	ANY 1	ALL 3	3 WEAK BANDS OR ANY STRONG BAND
	p120											
	p41											
POL	p68	ANY 3 GAG OR POL	p32	AND	ANY 1	AND	AND	OR	ANY 1 GAG OR POL	p32	ANY 1	
	p53											
	p32											
GAG	p55	p24	AND	ANY 1	AND	AND	OR	ANY 1 GAG OR POL	p24	ANY 1	ANY 1	
	p39											
	p24											
	p18											

AFR=AFRICA;¹ AUS=AUSTRALIA;² FDA=US FOOD AND DRUG ADMINISTRATION;³ RCX=US RED CROSS;³ CDC=US CENTER FOR DISEASE CONTROL;³ CON=US CONSORTIUM FOR RETROVIRUS SEROLOGY STANDARDIZATION;³ GER=GERMANY; UK=UNITED KINGDOM; FRA=FRANCE; MACS= US MULTICENTER AIDS COHORT STUDY 1983-1992. * Bands not in electrophoretic order

NOTES:

- I. "The Association of Public Health Laboratories now recommends that patients who have minimal positive results on the Western blot, eg p24 and gp160 only, or gp41 and gp160 only, be told that these patterns have been seen in persons who are not infected with HIV and that follow-up testing is required to determine actual infective status".⁴
 - II. In February 1993 the US Food and Drug Administration relaxed their criteria in order to "reduce the number of HIV-1 seroindeterminate Western blot interpretations", that is, to increase the number of HIV positive individuals.⁵
1. WHO. (1990). Acquired Immunodeficiency Syndrome (AIDS). Proposed criteria for interpreting results from Western blot assays for HIV-1, HIV-2 and HTLV-I/HTLV-II. *Weekly Epidemiological Record* 65:281-298.
 2. Healy DS, Maskill WJ, Howard TS, et al. (1992). HIV-1 Western blot: development and assessment of testing to resolve indeterminate reactivity. *AIDS* 6:629-633.

3. Lundberg GD. (1988). Serological Diagnosis of Human Immunodeficiency Virus Infection by Western blot Testing. *Journal of the American Medical Association* 260:674-679. (Data presented in this paper reveal that when the FDA criteria are used to interpret the HIV Western blot less than 50% of US AIDS patients are HIV positive whereas 10% of persons not at risk of AIDS are also positive).
4. Mylonakis E, Paliou M, Greenbough TC, Flaningan TP, Letvin NL, Rich JD. Report of a false-positive HIV test result and the potential use of additional tests in establishing HIV serostatus. *Archives of Internal Medicine* 2000;160:2386-8.
5. Keinman S, Busch MP, Hall L, et al. (1998). False-positive HIV-1 test results in a low -risk screening setting of voluntary blood donation. *Journal of the American Medical Association* 280:1080-1083.

NOTE: Each horizontal band on the Western blot strip (extreme left of table) represents an "HIV" protein. Serum from a patient is added and when the strips are developed coloured bands appear at the sites where antibodies have reacted with the individual "HIV" proteins. The number and location of the bands that determine a positive test vary between laboratories, institutions and countries. Even today there are still no internationally agreed criteria as to what constitutes a positive Western blot. This gives rise to the situation where, for example, an individual positive in New York City on the CDC criteria may not be positive in Sydney, Australia. Or an Australian positive with p41, p32, p24 and p18 bands may not be positive in Africa. Or an African positive with a p41 and p120 band may not be positive in Australia, parts of the US or Europe.

Confusion over antibody reactivity is confirmed in diagnostic laboratory manuals. The Genelabs Diagnostic HIV BLOT 2.2 Western blot Assay Instruction Manual advises, "Specific guidelines for interpretation may differ depending on the local policies, GENELABS recommends following the accepted policy to be in accordance with local regulations". This is followed by seven different criteria for defining a positive Western blot issued by "different international regulatory bodies". Genelabs also append, "We recommend the following guidelines for the interpretation of the Genelabs Diagnostic HIV BLOT 2.2" and list an eighth set of criteria for a positive Western blot. This means that "different international regulatory bodies" or "local policies", and not the presumed pathogen determine patterns of antibody reactivity said to prove a retroviral infection. Manufacturer Bio-Rad advises "Each laboratory performing Western blot testing should develop its own criteria for band interpretation. Alternatively, band interpretation may be left to the clinician" (Bio-Rad Laboratory Manual 1993).

SIMILAR TABLE BASED ON GENELABS PACKET INSERT

GLOBAL VARIATION IN THE CRITERIA FOR A POSITIVE WESTERN BLOT

CDC/ASTPHLD	Two bands of : gp41 or gp120/gp160 or p24
US manufacturer's (FDA)	p24 and p31 and one of: gp41 or gp120/gp160
SFTS France (Unequivocal POS) (Probably POS) (Probably POS)	Two ENV (gp160 and gp120) with GAG or POL Env (gp160) and GAG (p24) Two ENV bands only (gp160 and gp120)
World Health Organization	Two ENV bands with or without GAG or POL
CRSS Pan American Health Organization	One band of p24 or p31 and one ENV band
American Red Cross (USA)	One band each of GAG and POL and ENV
Paul Ehrlich Institut (Germany)	Two bands one must be ENV
China	Two ENV (gp160/gp41and gp120) and any GAG or POL
Singapore	Two ENV (gp160/gp41and gp120) and any GAG or POL
Australia	One ENV and any 3 GAG or POL

FDA=Food and Drug Administration; CDC=Center for Disease Control; CRSS=Consortium for Retrovirus Serology Standardization; ASTPHLD= Association of State and Territorial Public Health Laboratory Directors; SFTS=Sanguine Nationale Transfusion Societes, France.

Source: Genelabs Singapore and Genelabs Diagnostics HIV Blot 2.2 Western blot assay packet insert.

ANNEXURE 5 TO AFFIDAVIT by VALENDAR FRANCIS TURNER

MEASUREMENTS OF “HIV” “VIRAL LOAD”

The three assays frequently used to quantify the “viral load” are reverse transcription-polymerase chain reaction (RT-PCR), nucleic acid sequence-based amplification (NASBA) and branched chain DNA (bDNA). To assess the impact of the assays used and of “genetic variability in HIV-1 RNA quantification”, researchers from France “evaluated three commercial kits by using a panel of HIV-1 isolates representing glades A to H...These isolates were expanded in culture. Virus was collected by ultracentrifugation and resuspended in HIV-seronegative plasma. To standardize the quantities of virus to similar levels in each preparation, the p24 antigen was determined and the volume adjusted so that each specimen contained approximately 10pg of p24 antigen per ml”. The “HIV-1 RNA copies” per ml of plasma obtained were as follows (where <400 is considered zero RNA).

HIV-1 STRAIN	RT-PCR	BDNA	NASBA
DJ258	<400	111,500	100,000
DJ263	<400	79,800	60,000
SF2	225,500	38,000	240,000
III-B	54,000	17,000	360,000
ZAM18	78,300	70,000	66,000
ZAM20	178,800	125,800	420,000
UG270	179,800	29,200	170,000
UG274	320,000	41,400	32,300
CM241	18,800	72,800	35,000
CM235	4,700	52,000	15,000
163.3069	36,200	94,000	57,000
162.307	2,800	78,100	26,000
G98	254,700	269,000	<400
LBV21	184,500	295,000	<400
VI557	950,000	587,000	125,000

If this test is measuring one and the same thing, that is, the amount of “HIV” RNA in a patient’s plasma, then all the numbers in the rightmost three columns should be of identical order. Their gross variability should not be excused on the basis of “quantification of HIV-1 RNA is highly influenced” by the “HIV-1 strain” and the test kit used. It is incomprehensible how tests are used to quantify anything at all let alone what is believed to be a deadly microbe. If “viral load” were a pregnancy test or a test for cardiac enzymes following a heart attack clinicians could not use it.

SOURCE Coste J, Montes B, Reynes J, et al. (1997). Effect of HIV-1 genetic diversity on HIV-1 RNA quantification in plasma: comparative evaluation of three commercial assays. *J. Acquir. Immun. Def. Syndr. Hum. Retrovirol.*, 15, 174.

ANNEXURE 6 TO AFFIDAVIT by VALENDAR FRANCIS TURNER

"HIV" RNA sample tests reported by National Reference Laboratory Victoria

SAMPLE	TEST	NUMBER OF COPIES OF "HIV" RNA X 1000				CV
		MEAN	SD	LOWER	UPPPER	
QC101	TEST 1	40.8	24.6	16.2	65.4	60.3
	TEST 2	22.9	7.1	15.8	30	
QC102	TEST 1	228.4	198.4	30	426.8	86.9
	TEST 2	129.4	29.5	99.9	158.9	
QC106	TEST 1	421.9	249.2	172.7	671.1	59
	TEST 2	125.4	30.4	95	155.8	
	TEST 3	366.9	126.0	240.9	492.9	
	TEST 4	242.8	36.5	206.3	279.3	
QC108P	TEST 4	0.87	0.2	0.67	1.07	23
	TEST 5	0.13	0.1	0.03	0.23	
QC109P	TEST 4	11.7	1.8	9.9	13.5	16
	TEST 5	23.1	19.7	3.4	42.8	

Values given as mean +/- standard deviation (SD)

CV= coefficient of variation = mean/standard deviation

Lower = mean - SD; Upper = mean + SD

Shadings indicate greatest range between lowest and highest readings

These results are the means of quality control (QC) "HIV" samples measured by several laboratories in Australia. Each QC sample contains the same quantity of "HIV" RNA and data do not include "invalid runs".

Within assays the variation in the average values obtained measured between 16-86.9%.

In one experiment (data not shown) nearly one third of laboratories could not obtain a value within 2 standard deviations of the mean value.

From the data for QC101 and QC106, for example, a mean "viral load" of 40.8 or 421.9 X 1000 copies was reduced by approximately a half or two thirds respectively by measuring the same sample with a different assay. From the data for QC108P changing the assay reduced the mean "viral load" almost 7 times.

These data can be put into perspective by imagining the numbers represent the day's takings from a supermarket deposited in two banks that use different methods of counting cash. One can only speculate on the outcome.

Source: Best SJ, Gust AP, Johnson EI, McGavin CH, Dax EM. Quality of human immunodeficiency virus viral load testing in Australia. *Journal of Clinical Microbiology* 2000; 38:4015-20.