NIAID/NIH "Evidence"

REBUTTAL

A rebuttal to the NIAID/NIH document
"The Evidence That HIV Causes AIDS"

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The Evidence That HIV Causes AIDS, is a document that was created by the Office of Communications and Public Liaison of the National Institute of Allergy and Infectious Diseases/National Institutes of Health (NIAID/NIH): www.niaid.nih.gov/factsheets/evidhiv.htm (November 1994, updated on November 29, 2000). It is the most comprehensive document we know of to attempt to answer the many arguments that HIV does not cause AIDS. In the following rebuttal we argue that the NIAID/NIH document is seriously flawed for both failing to respect the standards of scientific discourse and failing to provide credible evidence to support its fundamental claims.

Science is normally advanced through publications that are:

- are signed by its authors;
- include a comprehensive list of citations;
- include contact information for at least one author;
- are modifiable only through additional publications (including errata);
- are open to rebuttal through published letters in the same journal;
- are peer reviewed
- rely largely upon other peer-reviewed literature to back up their assumptions.

The NIAID web page has none of these characteristics. On the contrary:

- it is anonymous;
- citations are restricted to the protagonist case;
- it changes occasionally without warning or explanation;
- it has no forum for those who disagree with parts of it to respond;
- it gives no information about the review process, if any, used to validate it;
- prejudges the issue by labelling the dissident case as "myth" and the protagonist case as "fact";
- fails to detail the dissident case in the same details as the protagonist case.

This rebuttal limits itself to revealing the fallacy of the key assertions made in the NIAID/NIH document:

- (A) that "HIV fulfills Koch's postulates as the cause of AIDS";
- (B) that "modern culture techniques have allowed the isolation of HIV in virtually all AIDS patients, as well as in almost all HIV-seropositive individuals" and that a human immunodeficiency virus (HIV) has been clearly identified;
- (C) that so-called HIV antibody tests, when positive, indicate specific "antibodies that indicate HIV infection." That is, such antibodies are caused by HIV infection and nothing else.

Here we present evidence that all the various "HIV tests" (antibody, antigen, PCR (viral load) or by "modern culture techniques") have never been validated the only possible standard of viral purification, nor has a unique AIDS-causing retrovirus ever been properly identified. Without the unequivocal gold standard of viral purification all the correlations put forward in the NIAID/NIH document as evidence that HIV causes AIDS have no solid foundation.

A point of clarification: This rebuttal draws largely on two major critics of the HIV/AIDS hypothesis: Papadopulos-Eleopulos et al. (known as the "Perth Group") and Peter Duesberg. On one essential point they hold fundamentally opposing views; Peter Duesberg accepts the isolation of HIV and the antibody tests as proof of infection (albeit with a harmless passenger virus) while the Perth Group does not. Readers might like to read The Last Debate (1999) http://www.virusmyth.net/aids/data/epdebate.htm which the Perth Group wrote to compare and contrast their views to those of Peter Duesberg. You can find Duesberg's defence of his position here: http://www.healtoronto.com/nih/continuum_award.html

A CLAIMS THAT KOCH'S POSTULATES HAVE BEEN FULFILLED

NIAID: "HIV fulfills Koch's postulates as the cause of AIDS."

THE FOLLOWING IS A SHORT SUMMARY SHOWING HOW HIV FAILS KOCH'S POSTULATES:

Koch's Postulate #1:
>> EPIDEMIOLOGICAL ASSOCIATION
the suspected cause must be strongly associated with the disease.

NIAID: "…virtually all AIDS patients are HIV-seropositive; that is they carry antibodies that indicate HIV infection."

The assumption that a positive "HIV antibody" test indicates active HIV infection in every case is not supported by the scientific evidence. Also, the supposed correlation between a positive HIV antibody test and AIDS is extremely weak", based on circular logic and has been flawed from the very beginning.

On April 23, 1984 Dr. Robert Gallo filed a patent application for an antibody test, now generally referred to as the "AIDS test". The same day Gallo announced at an international press conference that he had discovered a new retrovirus that he called HTLV-III (now known as HIV), and that it was "the probable cause of AIDS". This announcement caught even the scientists in the audience by surprise. Gallo had circumvented an essential part of the scientific process; he had not published his research findings in any scientific or medical journal or subjected them to the normal process of peer review prior to being announced to the public.

When Gallo's "evidence" was finally published weeks later there were some serious problems. The laboratory procedure Gallo and his colleagues considered to prove isolation were observed only in 36% of his AIDS patients and only 88% were positive on the "HIV-antibody" test. Also, in order to ensure that only the AIDS patients and not the healthy control group came up positive on his antibody test, he had to dilute the blood an extraordinary 500-fold. At lesser dilutions too many healthy controls would also test positive. These facts alone should have been enough to cast serious doubt on Gallo's claim that he had discovered a new retrovirus or the "probable cause of AIDS" (Gallo, 1984; Papadopulos-Eleopulos, 1993a; Koliadin, 1998). An excellent summary of how corrupt, deceitful (and possibly even criminal) his research was can be found in the book Science Fictions by John Crewdson, a Chicago Tribune science writer (Crewdson, 2002)

There are tens of thousands of people who have been reported with AIDS to the CDC's HIV/AIDS Surveillance "without laboratory evidence regarding HIV infection":

> Upon request, the CDC's director of the HIV/AIDS division, Harold Jaffe, stated that the HIV status of 43,606 out of the 253,448 American AIDS cases recorded by the end of 1992 was "not tested" (per. com., 1993). However this figure seems to be an understatement. Obviously, all 10,360 American AIDS cases diagnosed before the HIV antibody test, i.e., before 1985, were not tested (HIV/AIDS Surveillance, February 1993). In addition, the CDC published that "Approximately one third of AIDS patients in the United States have been from New York and San Francisco, where, since 1985, 7% have been reported with HIV-antibody test results, compared with 60% in other areas" (Confronting AIDS-Update 1988). Thus, between 1985 and 1987, 58% (93% x 1/3 + 40% x 2/3) of the 56,807 AIDS cases recorded in that period, or 32,948, have not been tested. For 1988, the CDC reported that 27% or 9,039 of the 33,480 AIDS cases recorded for that year were not tested for HIV (Selik, R. M., et al., 1990, J. AIDS 3, 73-82). According to the CDC's Technical Information Activity, 3682 AIDS cases without an HIV-test were recorded in 1989, 2888 in 1990, 1960 in 1991, and 1395 in 1992 (per. com., 1993). Thus, at least 62,272, or 18,666 more than Jaffe reports, were not tested (Duesberg, 1993).

There are thousands of people who have been diagnosed with AIDS even though they were found to be HIV negative on the antibody tests:

> Determination of the HIV-AIDS correlation is further obscured because HIV-free AIDS cases are not recorded in the CDC's HIV/AIDS Surveillance. By 1993, at least 4621 HIV-free AIDS cases had been documented in the U.S., Europe, and Africa with the clinical AIDS definition. […] Some of these proved to be HIV-free even by PCR amplification of viral RNA and DNA (Duesberg, 1993).

In addition, HIV antibody tests are rarely used in Africa and other poor nations where the majority of people with AIDS supposedly reside, so there is no way of knowing how many test positive on the HIV antibody tests. In these parts of the world AIDS case definitions like the Abidjan/WHO, Bangui/WHO (clinical), Caracas/PAHO and revised Caracas/PAHO leave the door wide open for AIDS surveillance "without laboratory evidence regarding HIV infection". Surveys indicate that more than half of

Africans who qualify for an AIDS diagnosis test HIV-negative:

- **227 patients with "AIDS": 59% test HIV-negative** (Lancet 340, p971, 1992)
- **122 patients with "AIDS": 69% test HIV-negative** (Am. Rev. Resp. Diseases 147, p958, 1993)
- **913 patients with "AIDS": 71% test HIV-negative** (J. AIDS 7:8, p876, 1994)

Finally, since positive HIV tests are now required in most wealthy nations to diagnose AIDS, even people suffering from one of the 29 "AIDS indicator diseases" with symptoms exactly like those of AIDS, are not diagnosed with AIDS. This creates a ridiculous artificial correlation in which the definition of AIDS requires an HIV positive test, and then the artificial correlation is used to try to show that HIV must be causing AIDS. (Note: Under a few exceptional circumstances the CDC does not require proof of HIV infection e.g.; PCP pneumonia with no other apparent cause.)

In summary: If HIV antibody positive = HIV infection, clearly HIV is not found in all cases; and the definition also allows HIV to be absent. so HIV fails Koch's Postulate #1.

KOCH'S POSTULATE #2
>> ISOLATION
It must be isolated from the host and grown in pure culture (NIAID).

NIAID: "…modern culture techniques have allowed the isolation of HIV in virtually all AIDS patients, as well as in almost all HIV-seropositive individuals…"

HIV has never been "isolated from the host and grown in pure culture." Proof of purification requires an electronmicrograph showing [pure] viral particles with the morphology of retroviruses — and nothing else. Such an electronmicrograph has never been provided for HIV (Papadopulos, 1998b). Attempts to show the purity of so-called HIV isolates have been a complete failure (Gluschankof, 1997; Bess, 1997; De Harven, 1998a). Even Luc Montagnier, regarded as the discoverer of HIV, has admitted that his research team failed to purify a virus. He also said "analysis of the proteins of the virus demands mass production and purification. It is necessary to do that". His team "had not enough particles produced to purify and characterize the viral proteins" (Tahi, 1997). Since viruses and cells are made of proteins and nucleic acids, and especially since there are thousands of proteins and nucleic acids in cells, even if HIV did exist how is it possible to tell which constituents are viral and which are cellular? Attempts to show the purity of so-called HIV isolates have been a complete failure (Gluschankof, 1997; Bess, 1997; De Harven, 1998a).

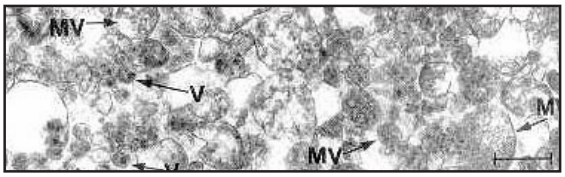
The word 'isolation' is commonly used in HIV research papers and, to most people, implies that the virus has been purified. Yet, in reality, the term has been degraded to utter meaninglessness because all that is looked for are surrogate markers (e.g. certain enzymes or proteins) which are believed to come from HIV. Not even one single intact viral particle is identified, and none of the surrogate markers that are used are actually specific to HIV. The process starts with unpurified extracts of body fluids being added to a culture of cancerous cells along with some stimulating chemicals. After several days or weeks, the culture is usually examined for one of the following signs: the reverse-transcriptase enzyme, the protein 'p24', retrovirus-like particles or proteins that bind with antibodies from AIDS patients. None of these are specific to retroviruses. Cultures are radically changed by being exposed to atmospheric oxygen levels (21%) rather than the 0.5-10% found within the body. It seems that this is just another indication that culture systems are unrepresentative of the real world: Roy S et al. Oxygen sensing by primary cardiac fibroblasts a k key role of p24. Circ Res. 2003; 92. (Ranki, 1988; Roy; Papadopulos, 1993a).

The assumption that HIV can be isolated from "virtually all AIDS patients, as well as in almost all HIV-seropositive individuals" will be discussed in greater detail in sections B and C.

See: Appendix A: The Origin of the "HIV" Proteins

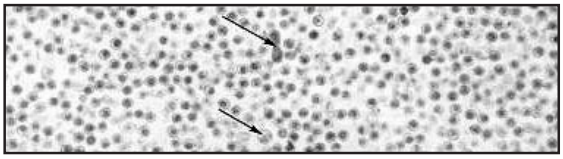
See: What "HIV" Researchers Mean by "Isolation" and "Cloning" http://healtoronto.com/cloning.html

"Purified" HIV (primarily cellular debris)



The first electronmicrographs (EMs) of "purified HIV", published in the March 1997 issue of Virology, disclose "major contaminants". In the example above, note that the arrows labeled 'V' point to the few particles that are "retrovirus-like". The authors of these studies concede that their pictures reveal the vast majority of the material in the density gradient is cellular contamination.

Truly Purified Friend Virus



By contrast, this electronmicrograph of the Friend virus shows, at a magnification of 19500 x, an almost pure population of typical "type C" viruses (not yet called retrovirus in 1965). The arrows point at contaminating debris and microvesicles. The interpretation was that virus purification was satisfactory and that contamination rate was extremely low (De Harven, 1965).

Figure 1.

Koch's Postulate #3
>>TRANSMISSION PATHOGENESIS
Transfer of the suspected pathogen to an uninfected host, man or animal, produces the disease in that host.

NIAID: "Postulate #3 has been fulfilled in tragic incidents involving three laboratory workers with no other risk factors who have developed AIDS or severe immunosuppression after accidental exposure to concentrated, cloned HIV in the laboratory."

Of the three people who supposedly got AIDS from 'purified' or 'concentrated' virus, only one has been discussed in any credible detail in the scientific journals. Duesberg is puzzled why these cases have not been written up in medical journals, as this would have been an opportunity to show that one of Koch's postulates of causation by a microbe has been fulfilled (Presidential AIDS Advisory Panel, 2001). There have been no scientific papers written about two of these three people and there have never been any further reports, even though these situations must have occurred no later than 1993 and since then millions of needlestick injuries have occurred.

NIAID: "In all three cases, HIV was isolated from the infected individual, sequenced and shown to be the infecting strain of virus."

Weiss et al report:

> In a prospective cohort study of 265 laboratory and affiliated workers, one individual with no recognized risk factors for human immunodeficiency virus type 1 (HIV-1) infection was HIV-1 seropositive at the time of entry into the study. Molecular analyses of two HIV-1 isolates derived in two independent laboratories from a blood sample from this worker showed that the isolates were indistinguishable from a genotypic form of HIV-1 present in the H9/HTLV-IIIB cell line (Weiss. Science 1988, 239(4835):68-71).

This sounds impressive, but what does Weiss mean when he claims "the isolates were indistinguishable"? Valendar Turner offers a good analogy:

> To speak of HIV DNA (i.e.; "molecular analyses") first one needs to prove that a particular sequence is unique and is present (as the RNA equivalent) in Retroviral-like particles proven to be a retrovirus (they aren't a virus until you prove they and they alone are what induce the appearance of more such particles). To proceed without such proof is no different from conducting a paternity suit where there is no proof that the DNA of the alleged father has been taken from the defendant or even a man. What defines the alleged father's DNA is solely that it comes from his body. It's no different with retroviruses. The operative expression is virologica habeas corpus. Amplification, sequencing, hybridization is wondrous stuff and keeps masses of scientists employed but their work is utterly meaningless in terms of a retrovirus if you don't have proof that's where it comes from.

To see the basic fallacy underlying these molecular analyses, consider the results of attempts at HIV isolation from a cell line like H9/HTLV-IIIB. Two papers in the leading science journal Virology in March 1997 provide astonishing data on the purification and isolation of HIV (Gluschankof et al, 1997; Bess et al, 1997). For the first time in the history of AIDS, elusive electron microscope images of 'HIV' collected or 'banded' at the official density required for purifying retroviruses, 1.16 gm/ml, were published. The electronmicrographs (EMs) disclosed "major contaminants" in "pure HIV" — an oxymoron to say the least. The authors of these studies conceded that their pictures reveal the vast majority of the material in the density gradient was cellular contamination. HIV expert Hans Gelderblom, of Berlin's Robert Koch Institute, co-authored the first paper which describes the contamination as "an excess of vesicles" — particles of cellular proteins, that may contain DNA or RNA. In a consecutive paper, a U.S. research team from the AIDS Vaccine Programme in Maryland reveal carefully, "It is unknown how these cellular proteins associate with the virus" and warn, "The presence of microvesicles in purified retroviruses has practical implications". One has to wonder how it could be possible to sequence an "HIV" genome or obtain pure HIV proteins from "isolates" like these. (See Figure 1.)

Eleopulos and colleagues summarize the problem:

> The "HIV RNA" and "HIV DNA" are defined on the basis of length (approximately 10,000 nucleotides) and chemical composition (adenine rich) of all the RNA present in cultures of tissues of AIDS patients, NOT on the basis of RNA extracted from a particle first isolated and then proven to be a retrovirus.
>
> In 1990 the HIV genome was said to consist of ten genes. This year Montagnier reported that HIV possesses eight genes and according to Barr,-Sinoussi, HIV has nine genes. Neither is there constancy of the number of nucleotides in the "HIV genome". Also, to date, only 11 full length "HIV genomes" have been sequenced and accordingly, HIV genotype consignments are derived from sequence analysis of subgenomes measuring 2% to 30% of the total. The data is that such "genomes" vary between 3-40%. (If 30% of the HIV genome varies as much as 40%, how much does 100% of the HIV genome vary? In the HIV Western blot, how can an HIV producing one set of proteins detect antibodies that are produced in response to the set of all other disparate "HIV genomes"? When does "HIV" become some other entity?). Thus, not only are there no two HIV genomes of the same length or nucleotide composition, there is no single genetic entity "HIV DNA" to describe the myriads of "HIV genomes". It is also estimated that patients contain between one and one hundred million distinct HIV DNAs at the one time. Neither is it correct to encompass such DNAs under the umbrella of a quasispecies of "closely related genomes" (Eleopulos, 1996).

NIH: "In another tragic incident, transmission of HIV from a Florida dentist to six patients has been documented by genetic analyses of virus isolated from both the dentist and the patients."

Duesberg summarizes the evidence disproving the allegations against Dr. Acer:

> Based on their own research, insurance companies concluded that the HIV strains in the five patients were different from that in Acer, meaning each caught it from a different source. (116) A study out of Florida State University has backed this conclusion. (117) Even the CDC acknowledged this evidence, though it still preferred to believe the dentist had infected Bergalis. But the CDC's own numbers give away the reality. An estimated 1 million Americans have HIV, in a total population of 250 million. Thus, 1 in 250 Americans have the virus. Five HIV -infected patients of Dr. Acer, out of 1,100 tested, comes to 1 in 220, virtually identical to the national average. [...] These HIV positive patients merely represent random samples from the general population.
>
> The CDC's theory that AIDS was transmitted from Dr. Acer to his patient [Kimberly Bergalis] began to crumble in the mainstream press in 1994 when an investigative reporter researched the alleged victims of Dr. Acer. "He found weak evidence, shoddy science, and the work of a very accomplished malpractice attorney." (118)
>
> The report first casts doubt on the time course of AIDS transmission from Dr. Acer to his patients. "[Bergalis] developed AIDS just two years after the surgery, and only 1 percent of HIV positive patients develop the full-blown disease that quickly." (119) The investigation disclosed that one of the six other patients that Acer presumably infected had visited the dentist's office only once for a cleaning by a hygienist, not by Acer himself. (120) The report further calls into question the exclusive reliance of the CDC and the malpractice attorney of the "Acer six" on the DNA fingerprinting technique to match Acer's virus with those of his patients. [...] Several experts have directly challenged the DNA fingerprinting that linked Acer to his patients, claiming that instead Bergalis's virus matched other HIV strains much more closely. (121) In view of this, a writer in the New York Times commented, "The CDC owes it to the public to reopen [Acer's] case." (122) (Duesberg, 1996)

Not one paramedic, emergency medical technician or surgeon in the U.S. has contracted AIDS from on-the-job exposure (CDC, 2000). Out of 733,374 total U.S. AIDS cases through 1999, only 25 are thought to be occupationally acquired, based on presumptive evidence — meaning they admitted no other risk factor (CDC, 2000). In Canada, out of 17,389 AIDS cases through 2000, only 5 are reported as occupationally acquired (PPHB, 2001). Only one case has been documented, and the evidence was weak and circumstantial (CCDR, 1992). Consider that the 1 million needle-stick injuries among health care workers in the U.S. each year result in about 1,000 cases of hepatitis among health care workers annually. That means that in the 18 years of AIDS, health care workers contracted 18,000 cases of hepatitis and 25 cases of AIDS. Pretty strange if HIV is a blood-borne virus!

B CLAIMS THAT HIV HAS BEEN ISOLATED.

NIAID: "HIV can be detected in virtually everyone with AIDS."

NIAID: "Recently developed sensitive testing methods, including the polymerase chain reaction (PCR) and improved culture techniques, have enabled researchers to find HIV in patients with AIDS with few exceptions. HIV has been repeatedly isolated from the blood, semen and vaginal secretions of patients with AIDS, findings consistent with the epidemiologic data demonstrating AIDS transmission via sexual activity and contact with infected blood (Bartlett, 1999; Hammer et al. J Clin Microbiol 1993;31:2557; Jackson et al. J Clin Microbiol 1990;28:16)."

New testing techniques cannot both be validated against existing antibody tests and used to support current tests. They must, instead, be independently validated using the only technique that unambiguously detects HIV — virus purification.

In a letter to the editor of the British Medical Journal, Eleopulos et al simply states the problem and invites speculation as to how all those "extra" Africans become HIV positive:

> EDITOR - According to Lamptey, in Africa slightly more than 80% of infections are acquired heterosexually.1 The high rates of heterosexual infection in Africa have been generated by actuarial models and antenatal data.2 These high rates are not supported by data originating from prospective epidemiological studies.
>
> In 1997 Padian et al published the results of a 10 year study on heterosexual transmission of HIV in northern California.3 The data were divided into two parts, cross sectional and prospective. From the cross sectional study it was estimated that infectivity for male to female transmission is low, approximately 0.0009 per contact, and approximately eight times more efficient than female to male transmission. Using this estimate of male to female transmission, it would take 770 or 3333 sexual contacts respectively to reach a 50% or 95% probability of becoming infected. If sexual contact were to take place repeatedly every three days this would require a period of 6.3 and 27.4 years respectively. Based on the estimate of female to male transmission by Padian et al it would require 6200 and 27 000 contacts and a period of 51 and 222 years, respectively (table).

Numbers of years to attain 50% and 95% probability of transmission of HIV in United States and Uganda assuming sexual contact once every three days

	Probability per contact	No of years for 50% probability	No of years for 95% probability
United States			
Male to Female	0.0009	6.3	27.4
Female to Male	0.0001125	51.0	222.0
Uganda			
Male to female	0.0009	6.3	27.4
Female to male	0.0013	4.4	19.5

> In 2001 a community based study was reported from Uganda, where 174 monogamous couples, in which one partner was HIV-1 positive, were retrospectively identified from a population cohort involving 15 127 people.4 The probability of transmission per sexual contact was 0.0009 for male to female and 0.0013 for female to male respectively (table). The authors concluded that the probability of HIV transmission per sex act in Uganda is comparable to that in other populations, suggesting that infectivity of HIV subtypes cannot explain the explosive epidemic in Africa (R H Gray et al, eighth conference on retroviruses and opportunistic infections, Chicago 2001). In other words, there is no more heterosexual transmission of HIV in Africa than anywhere else, including Britain, the United States, Australia, and Europe. (Papadopulos-Eleopulos et al, 2002)

"Recent data has challenged the whole notion that the majority of HIV infections in Africa are heterosexual." (Brewer DD, 2003; Gisselquist D et al, 2002; Gisselquist D et al, 2003a; Gisselquist D et al. 2003b)

The statement quoted above from the NIAID document cites three references to support its claim that newer tests have allowed HIV to be 'repeatedly isolated' from people diagnosed with AIDS. When these three references are examined, however, one finds that they actually point out many inconsistencies and problems with the tests currently being used, and in some cases these uncertainties are openly admitted. This is a perfect example of the paucity of evidence and misleading references put forward by the NIAID/NIH in support of their claims. Following is a brief analysis of how the three references for the above NIAID/NIH statement are inadequate and misleading:

1 Bartlett, 1999; 2001-2002 Medical Management of HIV Infection by John G. Bartlett, M.D. and Joel E. Gallant, M.D., M.P.H. Copyright © 1997-2002 The Johns Hopkins University on behalf of its Division of Infectious Diseases and AIDS Service. "Production of the 2001-2002 edition of Medical Management of HIV Infection has been underwritten by an unrestricted educational grant form GlaxoSmithKline, Inc."

The relevant section reads as follows:

> Viral Detection: Other methods to establish HIV infection include techniques to detect HIV antigen, DNA, or RNA (Table 2-1). HIV-1 DNA PCR is the most sensitive and can detect 1-10 copies of HIV proviral DNA. **None of these tests is considered superior to routine serology in terms of accuracy**, but some may be useful in patients with confusing serologic test results, when there is a need to clarify indeterminate test results, for virologic monitoring in therapeutic trials, and for HIV detection when routine serologic tests are likely to be misleading such as in patients with agammaglobulinemia, acute retroviral infection, neonatal HIV infection, and patients in the window following viral exposure. **In most cases, confirmation of positive serology is accomplished simply by repeat serology.** The sensitivity of tests for detection of HIV varies with the stage of disease and test technique, but is usually reported at >99% for DNA-PCR, 90% to 95% for quantitative HIV-RNA, 95% to 100% for viral culture of peripheral blood mononuclear cells (PBMC), and 8% to 32% for p24 antigen detection (J Clin Microbiol 1993;31:2557; N Engl J Med 1989;321:1621; J AIDS 1990;3:1059; J Infect Dis 1994;170:553; Ann Intern Med 1996;124:803). None of these tests should replace serology to circumvent the informed consent process.

None of this sounds like evidence that there are valid tests for detecting HIV that could be considered a gold standard for "routine serology", that is, HIV antibody tests. It is clear that antibody tests do not prove presence of a virus. DNA, RNA and protein antigens are contingent upon proof they are extracted from pure culture of virus.

A foot note at the bottom of the page of the internet version of this publication states: "Physicians and other health care professionals are encouraged to consult other sources and confirm the information contained in this site...." So perhaps we had best move on.

2 Hammer et al. J Clin Microbiol 1993;31:2557. Use of virologic assays for detection of human immunodeficiency virus in clinical trials: recommendations of the AIDS Clinical Trials Group Virology Committee.

This is a set of guidelines or tutorial similar to the Medical Management of HIV Infection. Rather than providing evidence that "HIV can be detected in virtually everyone with AIDS", it strongly suggests the opposite. In the section on Viral Isolation two assays are discussed; PBMC culture and plasma culture. Regarding the PBMC culture:

> (iv) Indicated use: diagnosis. For a specimen to be considered positive by either the quantitative microculture assay or the qualitative macroculture assay, the sample supernatant must contain 30 pg or more of p24 antigen per ml, as determined by the standard p24 EIA. **This arbitrary positive value has been determined historically on the basis of average positive values from various kit manufacturers and is unrelated to that used for determining positivity in sera or plasma.**

In the following discussion of plasma culture Hammer et al. state:

> **Plasma viremia is not apparent in all HIV-positive patients. Infectious virus is rarely detectable in patients with CD4+ cell counts above 500/mm3.** As the CD4+ cell count drops, the percentage of patients with detectable plasma viremia will increase. For practical purposes, plasma viremia cultures should only be attempted when CD4+ counts are <200/mm3 and are accompanied by a positive baseline cell culture.

See Table 1 in the Piatak paper to get some perspective on how much "plasma viremia will increase."(Piatak, 1993) Hammer et al. also suggest that a DNA PCR assay could be used for diagnosis, but with this qualification:

> For the diagnosis of HIV-1 infection, two specimens collected at different times must be HIV-1 positive by PCR as defined above. **There is still some question at this time regarding confirmation of the PCR positive results by culture.**

3 Jackson et al. J Clin Microbiol 1990;28:16. Human immunodeficiency virus type 1 detected in all seropositive symptomatic and asymptomatic individuals.

This paper claims to 'isolate' HIV in 100% of people who had been previously diagnosed HIV-positive using the antibody tests, but there are a number of inconsistencies in its claims. The first major inconsistency is that the vast majority of similar studies have been able to 'isolate' HIV in much less than 100%, and even as low as 20%, of people diagnosed 'HIV-positive', so that using this study as a reference is misleading, at best. Another major inconsistency is that the word 'isolation' is used to describe the detection of surrogate markers which are not specific to HIV, as discussed above. Papadopulos-Eleopulos et al. provide a thorough refutation of this study's conclusions:

> In their 1990 paper Jackson et al reported that "Between February 1987 and October 1988, peripheral blood mononuclear cells (PBMC) from 409 individuals who were antibody positive for HIV-1 by Western (immuno) blot (56 AIDS patients, 88 patients with ARC, and 265 asymptomatic individuals) were cultured". "Using a sensitive technique previously described", the p24 assay noted above, they reported that "HIV-1 can be isolated from 100% (56 of 56) of AIDS patients, 99% (87 of 88) of ARC patients, and 98% (259 of 265) HIV- 1 antibody positive asymptomatic individuals". Not one of "131 HIV-1 antibody-negative individuals has a positive culture". Using the same p24 assay (Abbott) they tested the serum from 403 out of the 409 individuals. The test was positive in 23/56 (42%)

AIDS patients, 31/88 (57%) ARC patients and 44/259 (17%) asymptomatic antibody positive individuals. For unstated reason(s) a positive serum test is considered proof for the detection of "HIV-1 antigen in serum" while the same positive culture test is considered proof for "HIV-1 isolation" from the culture. There are many reasons to question the interpretation of the p24 assay: (a) The p24 assay is an antibody/antigen reaction and is subject to ubiquitous background reactivity. In this context, even if "two serial supernatant samplings with the later sampling showing greater reactivity", even if double or triple, for example, 30 and 60 or 30 and 90, both readings may be nothing else but background readings. Jackson and colleagues' criteria are not even in agreement with those used by Ho et al which are equally as arbitrary […] Even without a gold standard [i.e.; HIV isolation], the non-specificity of the p24 antigen test is so obvious that it is accepted by no less an authority on HIV testing than Philip Mortimer and his colleagues from the UK Public Health Laboratory Service, "Experience has shown that neither HIV culture nor tests for p24 antigen are of much value in diagnostic testing. They may be insensitive and/or non-specific".(236) […] Jackson et al's claims are not even confirmed by other laboratories. According to Jackson et al, up until 1990 only three small studies reported "100% isolation rates of HIV-1 from AIDS patients". In all the other studies, "HIV-1 was not isolated from 6 to 50% of HIV-1 seropositive AIDS cases reported. The culture recovery rate of HIV-1 from HIV-1 antibody positive asymptomatic patients has generally been even lower, only 20 to 42% in some studies". (Papadopulos-Eleopulos, 1996)

An example of a study with typically low rates of viral 'isolation', was published by Piatak, et al.(1993). This study not only found that many people diagnosed HIV-positive using antibody tests had no 'culturable' virus, but also found that only 53% of people with measurable 'viral loads' had positive viral cultures. See Table 1 (Piatak, 1993).

In the table to the right from Piatak et al.(1993) the column entitled "HIV RNA" shows the "viral loads" of the individual people listed, while the column entitled "Plasma culture/TCID" shows how much virus they could find using techniques claimed to "culture" the virus — note that most people with viral loads have no culturable virus, even with viral loads in the hundreds of thousands. One patient with CDC Stage IV AIDS had a viral load of 815,000 per ml of blood plasma even though he had no "culturable" virus (not shown in this detail of the table).

Patient ID	CD4+ cells* (cells/mm³)	HIV RNA† (copies/ml)	HIV p24 Ag		Plasma‖ culture (TCID/ml)
			ICD‡ (pg/ml)	Reg§ (pg/ml)	
CDC stage I¶					
HOBR 0961	920	2,617,400	386	660	125
SUMA 0874	853	1,485,000	892	1,250	625
BORI 0637	817	2,398,600	218	258	1#
INME 0632	739	2,427,900	275	360	1,000
WEAU 0575	358	355,400	258	329	0**
FASH 1057	262	21,783,600	5,072	5,406	10,000
CDC stage II or III					
RIPH 0179	1080	40,800	39	0	0
HIDO 1099	823	36,000	0	0	0
ATKA 0381	760	9,400	0	0	0
HOJU 0143	720	38,900	0	0	0
MAMA 0341	705	113,400	31	0	0
ADFR 0194	703	24,200	0	0	0
TIMI 0852††	678	100‡‡	0	0	0
BECH 0171	678	13,800	8	7	0
MALE 0264	644	198,800	0	0	0
ROJO 0331	640	78,700	0	0	0
BELI 1233	627	4,400	0	0	0
HAJO 0940††	624	13,800	0	0	0
ADDI 0101	616	128,300	0	0	0
FOMA 0784††	521	34,400	0	0	0
JUJA 0156	458	586,100	0	0	5
WATH 0272††	350	67,200	0	0	0
BAMA 0037	323	91,000	154	53	0
WAJO 1286	281	46,100	183	35	0
BAST 0514††	270	9,400	0	0	0
STMI 0862	257	92,900	574	350	5
STBO 1287	231	8,600	0	0	0
SLMI 0843	197	84,900	0	0	0
CDC stage IVC2					
ALFR 0229	615	91,800	0	0	0
DOBE 0859	533	114,500	0	0	0
CALI 0950††	456	40,800	35	5	0
ARLA 0846	435	49,100	0	0	0
WOAL 0263††	425	32,800	0	0	0
GADA 1162††	424	192,500	0	0	0
MCSE 0176	360	341,000	5,000	1,070	100
CHJI 0774	332	33,300	0	0	0
EDWI 0817	267	73,100	0	0	0
GRJO 0849	243	94,700	0	0	25
MIWI 1278	236	173,600	79	0	0
SMDO 0157††	141	2,200,000	740	284	3,125
HEMI 0562	117	223,000	123	101	125
DAJO 0306	109	36,900	361	120	0
SMST 1012	95	738,900	765	90	0
DUSE 1021	82	655,500	179	119	5
WHWI 1106	67	104,500	221	27	25
NOWR 1192	54	469,900	209	20	3,125
MIGE 1132††	29	191,800	0	0	0
DABE 0775	27	448,200	173	153	5
EMJA 0809	21	625,900	187	226	25
CLRA 0703	10	687,900	396	40	25
LARO 0833	7	479,300	823	238	5
CDC stage IVC1					
DEDA 0006	57	1,309,000	233	14	10,000
VAST 0181††	56	664,500	450	25	0
DODO 0116	50	1,867,000	357	385	100,000
SZHO 1173††	37	806,300	631	195	625
MILA 0284††	32	815,100	0	0	0
BIJA 0205	32	4,744,000	1,920	1,050	10,000
NAPH 0073	14	1,804,000	480	480	1,000
TIMI 0018	14	3,445,000	330	390	100
WATI 0855	7	9,300,000	606	302	625
MCMI 0063	7	232,000	221	12	625
LENA 1029	<5	2,500,000	53	0	125
FARO 1042	<5	4,800,000	199	204	625
RUTH 1145	<5	424,800	230	143	125
YOAL 0522	<5	2,600,000	205	5	125
JOJI 0070	<5	1,606,000	2,460	885	100,000

*CD4+ cells determined by flow cytometry within 6 weeks of sampling for virologic assessments. †HIV RNA copy number per milliliter of plasma, as determined by QC-PCR (*13, 17*). ‡Immune complex–dissociated HIV p24 antigen (*19*). §HIV p24 antigen (*19*). ‖Tissue culture infectious doses of virus per milliliter of plasma (*18*). ¶Centers for Disease Control classification system for staging of HIV-1 infection (*2*). #Three days previously, plasma cultures were positive at 10 TCID/ml, with HIV-1 RNA level of 1,350,600 copies/ml. **Eight days previously, plasma cultures were positive at 1,000 TCID/ml, with HIV-1 RNA level of 216,400 copies/ml (Fig. 2.). ††AZT or dideoxyinosine therapy at time of study. ‡‡Extrapolated value.

Table 1. Virologic and clinical summary for 66 consecutively studied HIV-1-infected patients.

"Circulating levels of plasma virus determined by QC-PCR also correlated with, but exceeded by an average of nearly 60,000-fold Table 1 [27], titers of infectious HIV-1 determined by quantitative endpoint dilution culture of identical portions of plasma. Several virologic and immunologic factors already identified in HIV-1 infection, including neutralizing antibody [28], viral envelope shedding [29], deterioration of other viral components [25], and genotypically defective virus [30] likely contribute to the differences in levels of circulating virus determined by QC-PCR and titers of culturable virus. However, the minimum requirements for establishment of productive infection of primary mononuclear cells are not known. If more than one intact viral particle is required to attain productive infection of a host cell, this would exaggerate the discrepancy observed between viral titers in plasma determined by QC-PCR as compared with those determined by endpoint dilution culture. For HIV-1 propagated in vitro, total virions have been reported to exceed culturable infectious units by factors of 104 to 107 [25], ratios similar to those we observed in plasma."

M. Piatak et al. "High levels of HIV-1 in plasma during all stages of infection determined by competitive PCR" (1993) Science 259: 1749-1754.

C CLAIMS THAT HIV ANTIBODY TESTS ARE VALID

NIAID: "Current HIV antibody tests have sensitivity and specificity in excess of 98%."

NIAID: "Diagnosis of infection using antibody testing is one of the best-established concepts in medicine. […]. Current HIV antibody tests have sensitivity and specificity in excess of 98% and are therefore extremely reliable (WHO, 1998; Sloand et al. JAMA 1991;266:2861).

"Progress in testing methodology has also enabled detection of viral genetic material, antigens and the virus itself in body fluids and cells. While not widely used for routine testing due to high cost and requirements in laboratory equipment, these direct testing techniques have confirmed the validity of the antibody tests (Jackson et al. J Clin Microbiol 1990;28:16; Busch et al. NEJM 1991;325:1; Silvester et al. J Acquir Immune Defic Syndr Hum Retrovirol 1995;8:411; Urassa et al. J Clin Virol 1999;14:25; Nkengasong et al. AIDS 1999;13:109; Samdal et al. Clin Diagn Virol 1996;7:55."

As with the claim "HIV can be detected in virtually everyone with AIDS", the anonymous authors have provided spurious references that do not support their assertions. Seven of the eight references have nothing to do with attempting to confirm the validity of the antibody tests. Only the Jackson et al. reference can be considered appropriate, but their conclusions are based on the flawed logic discussed above. See previous discussion of Piatak et al.

Even if one were to accept the existence of a unique "HIV" retrovirus with an outer coat composed of unique proteins ('antigens'), this still does not prove that antibody tests are accurate. No antibody test can distinguish between HIV antibodies (if they exist) and cross-reacting non-HIV antibodies unless the specificity of the test is established by comparing the presence of antibody reactivity (a 'positive' test result) when virus purification is successful, and the absence of antibody reactivity when virus purification fails. No data in the scientific literature has reported this essential verification. HIV antibody tests are therefore not grounded in reality, and may be meaningless.

The same applies to "viral load" (PCR) tests:

> If you have to use PCR to find something, that automatically means that what you find has no pathological relevance. Piatak, et al.(1993) were able to successfully "isolate" virus from only 53% of people with measurable viral loads. See Table 1 (Piatak, 1993). Thus the diagnosis "HIV-positive" is of questionable scientific value and needs to be reappraised.

Eleopulos el al remind us of this revealing meta-analysis of the sensitivity and specificity of PCR:

> Researchers from several institutions in the USA performed a meta-analysis of studies published between 1988 and 1994 that evaluated the sensitivity and specificity of PCR (Owens DK, et al. 1996). They "accepted positive results on conventional antibody tests (if they included a confirmatory Western Blot analysis or similar test) or viral cultures as high-quality evidence of infection", that is, as a gold standard. In a search of 17 computer databases, they "identified 5698 titles of potentially relevant articles. After independent review by two readers, 1735 titles were judged to be potentially relevant". Then they "reviewed the associated abstracts and then selected 379 studies published as full articles for further review. Of these 379 articles, 96 met the inclusion criteria and were analyzed". They found that: "Measured performance was extremely variable. When indeterminate PCR results were excluded, sensitivity ranged from 10% to 100% and specificity from 0% to 100%". They concluded "Our investigation produced two main findings. First, the false-positive and false-negative rates of PCR that we determined are too high to warrant a broader role for PCR in either routine screening or in the confirmation of diagnosis of HIV infection. This conclusion is true even for the results reported from more recent, high-quality studies that used commercially available, standardized PCR assays...We did not find evidence that the performance of PCR improved over time" (Papadopulos-Eleopulos E, et al. 2001).

Why does there seem to be a correlation between so-called HIV antibodies and AIDS defining illness?

Without carefully controlled, unbiased studies to determine the clinical significance of HIV tests, it's hard to know what positive results may or may not indicate. It seems clear that people who fall into the AIDS risk groups and test positive on these antibody tests are more likely to develop the illnesses classified as AIDS — as well as diseases which are not classified as AIDS. The so-called AIDS diseases occur principally in people with a history of repeated exposure to foreign proteins and /or toxins: multiple STDs and other viral, bacterial and parasitic infections; various pathogens via injecting contaminated street drugs; recreational and pharmaceutical drugs; commercially-made clotting factor and blood transfusions. They may also occur in the chronically

malnourished due to their weakened immune systems and subsequent exposure to more pathogens. These exposures to various pathogens, foreign proteins and toxins may elicit enough antibodies to register on the non-specific HIV antibody tests. Such a high level of antibody activity is not usually found among healthy people who are not in any AIDS risk group.

Papadopulos-Eleopulos points out that it is practically impossible to obtain clear data on this issue:

> In other words, follow people with a positive test over a period of years and see who developed AIDS and who did not. The trouble is it would be very difficult for most people knowing they are HIV positive, as well as their physicians, not to believe that sooner or later they're going to get very sick and eventually die of AIDS. And that mindset may greatly effect the results of such an experiment. (Johnson, 1997)

The "modern culture techniques" and the various indirect markers that HIV researchers regard as the equivalent of HIV isolation were discussed in detail above. Here it is worth noting that Eleopulos et al. argue that the various markers thought to indicate HIV infection can only be detected when cultures derived from the patients' tissue are subjected to extreme biological and chemical stress. Known as oxidative stress, these conditions are comparable to what happens in our bodies when exposed to the long list of stressors described above. This kind of oxidative stress may cause the conditions that then cause the HIV test to react positive, as well as increasing the risk of a person developing AIDS-like illnesses (Papadopulos-Eleopulos. 1992; Giraldo RA.).

See: Appendix B: The correlation between "HIV" antibodies and AIDS diseases

There are no simple answers to the mystery of AIDS. The illnesses do exist, even though they probably do not have a single cause. The HIV antibody tests may not indicate the presence of a virus, but people who come down with AIDS-like diseases are more likely to test positive. It is likely that an association between the diseases and the antibodies is due to a number of health risk factors. In Western countries, the biggest factor in illness appears to have been drug use, whether injected, ingested or inhaled, with different patterns of use leading to different diseases. In Third World countries, malnutrition can lead to a wasting condition that meets the Bangui definition of AIDS. Antiviral drugs are highly toxic, and can also lead to many of the diseases defined as 'AIDS'. Addressing known health risks is often much safer, cheaper and more effective than placing millions of people on lifelong pharmaceutical therapy. (Duesberg, P, Rasnick, D.)

Many HIV antibody positive people - some diagnosed over 17 years ago – remain healthy without HIV drugs and have never developed AIDS. For many people, being diagnosed HIV positive may have no bearing on their probability of living a long, healthy life. Clearly, testing positive on such a non-specific test does not always mean that one is in danger of acquiring immune deficiency. On the other hand, the idea of "HIV positive" has taken on devastating significance in the popular consciousness. The patients' health - physical and psychological — may be seriously compromised by an "HIV positive" diagnosis. Their physicians may feel compelled to offer treatments, even in the absence of clinical symptoms, with drugs well documented for their toxic effects. To confound things further, many of the toxic effects are indistinguishable from symptoms of AIDS (DeSimone, 2000).

Alive and Well founder and spokesperson Christine Maggiore recently summarized her perception off the human toll of the failed HIV/AIDS hypothesis:

> Whenever I speak in public someone invariably asks, "If HIV doesn't cause AIDS, why do a lot of people who test HIV positive get sick and die?" I think a better question is: How does anyone who tests positive remain well? Between the initial devastation of the diagnosis, the subsequent social isolation, dire predictions by doctors, lab tests measuring out our remaining time, medical care that assumes our inevitable early demise, AIDS organizations poised to usher us into death, negative expectations of friends, family and the public, constant media reports on the incurable fatal virus, pressure to consume toxic drugs, to regard ourselves as infected, abnormal, and ill, pitches to sell off our life insurance, exchange work for disability, and get a handicapped parking pass, and official orders to keep a safe, latex-covered distance from people we love, I wonder how so many of us manage to live.

Appendix A:

The Origin of the "HIV" Proteins

In regard to the antigens (proteins) used in the HIV antibody tests you may like add that in the paper by Bess et al shows that "infected" and "non-infected" cultures have a qualitatively identical protein electrophoresis pattern. That is, the "HIV" proteins are cellular. This evidence is corroborated by numerous other data. The proteins attributed to "HIV" have been found in non-HIV-infected tissues and tissues of healthy individuals at no risk of AIDS. These include the p18,[8, 10, 43, 44] p24,[43-48] p32,[49-51] p41,[24, 49, 50, 52-56] and p120/160 proteins.[43, 57, 58] From the beginning Montagnier has consistently stated that p41 is not an HIV protein but the ubiquitous cellular protein actin.[24, 52] In 1989 Pinter et al[57] demonstrated that the p120 and p160 proteins in the HIV Western blot strips are not distinct proteins but oligomers of p41 and that "confusion over the identification of these bands" has resulted in "incorrect conclusions".[58] Since the criteria for a positive Western blot in Africa[59] requires reactivity with any two of p41, p120 or p160 regardless of any other bands, we may conclude that Africans are infected with HIV if they possess antibodies which react with one of their own proteins. (Papadopulos-Eleopulos E, et al. 1993b)

Appendix B:

The correlation between "HIV" antibodies and AIDS diseases

1. The correlation between "HIV" antibodies (a positive test) and AIDS diseases is part contrived. Please note the following points: (a) Prior to 1987, one "HIV specific" WB band was considered proof of HIV infection. However, since 15%-25% of healthy, no risk individuals have "HIV specific" WB bands,[127,128] it became necessary to redefine a positive WB by adding extra and selecting particular bands, otherwise at least one in every seven people would be diagnosed infected with HIV. (Notwithstanding, in the MACS, one band remained proof of HIV infection in gay men until 1990[129]); (b) On the other hand, although AIDS began to decline in 1987,[130,131] this trend was countered by the addition of more and more diseases and, most recently, mere laboratory abnormalities[132] to each revision (1985, 1987 and 1993) of the first, 1982 CDC definition. The net effect of these changes was to maintain a correlation between "HIV" antibodies and "AIDS" amongst the "risk" groups while the risk of an HIV/AIDS diagnosis outside these groups remained slight; (c) This was further accentuated by avoiding testing outside the risk groups. However, when such studies were performed, for example, (a) amongst 89,547 anonymously tested blood specimens from 26 US hospital patients meticulously chosen to be at no risk of AIDS, between 0.7% to 21.7% of men and 0-7.8% of women aged 25-44 years were found to be HIV WB positive.[133] (It is estimated that approximately 1% of men are gay. Also, at the five hospitals with the highest rates of HIV antibodies, one third of positive tests were in women. Yet men vastly outnumber women as AIDS patients). (b) the US Consortium for Retrovirus Serology Standardization reported that 127/1306 (10%) of individuals at "low risk" for AIDS including "specimens from blood donor centers" had a positive HIV antibody test by the "most stringent" US WB criteria[127] (see below). Thus the correlation between "HIV" antibodies and AIDS, which experts accept as the only proof that HIV causes AIDS, is not a statistic related to the natural, unbridled activity of a virus but is instead a contrivance generated by mankind. Not only does correlation never prove causation, the artificiality of this particular "correlation" severely compromises its scientific analysis. (Turner V et al. 1999)

2. The fact that many people are not tested does not permit any conclusion in regard to the correlation. One can only operate on data where patients are tested. (Absence of evidence is not evidence of absence). In fact, as shown for example by the MAC studies, there is a high correlation between a positive test and having or developing AIDS indicator diseases. We must accept this correlation (despite the proviso above) but we are entitled to question whether the link is a retrovirus HIV. You may like to read the section on AB testing in our mother to child monograph to see our explanation of how the correlation can make sense but not in terms of a retrovirus.

Here are the same data reworked for this context:

The issue we must address how on the one hand can a positive "HIV" antibody test predict the onset of certain AIDS "indicator" diseases while on the other not be proof of infection with a retrovirus? In other words, if, as the EPE and her colleagues argue, the "HIV" proteins are cellular and not viral, but individuals develop antibodies which react with these proteins, could such a non-specific test, which has absolutely nothing to do with a retrovirus infection, behave in such a clinically useful manner? The answer is yes and in fact clinical medicine is replete with such tests. Take for example, the presence of a fever, or an alteration in a blood count. Such tests often do predict particular illnesses, or exclude others, and are also of considerable use in gauging the course of a disease or the effect of treatment, or confirming cure. No one has any difficulty with this concept but no one imagines a fever is a cause of a disease. When the doctor looks at your temperature chart every morning he can tell at a glance there is something wrong or whether or not you are on the mend. In this regard possibly the test nearest to the HIV antibody test is that which measures the rate at which a patient's red blood cells sediment down column of saline solution (the erythrocyte sedimentation rate or ESR). All this test does is measure how far down the column a drop of blood fall in one hour. Normally this is about 15 mm but in certain illnesses it can be over 100 mm per hour or higher. And a raised ESR may predate symptoms (like a positive antibody test), that is, it may occur while the patient is healthy. The ESR, first discovered in

Appendix B continued...

1918 by Fahraeus while seeking an test for early pregnancy, is a common but non-specific test which, when elevated, "is a measure of the presence and intensity of morbid processes within the body". Like a positive "HIV" antibody test, an elevated ESR also has the capacity to predict "a likelihood of death within the next several years far above" a normal ESR. A common cause of elevated ESR is infection and "Elevated ESRs are also seen with pregnancy, malignancy, collagen vascular diseases, rheumatic heart disease, and other chronic disease states [tuberculosis, osteomyelitis], including human immunodeficiency virus infection".[143] One important factor which affects the ESR is the size of the red cells, especially rouleaux formation where the red blood cells clump together. Rouleaux formation may result from changes in the negative charge of red cells, caused by "the dielectric effect of proteins in the surrounding plasma", especially by "fibrinogen, immunoglobulins, and other acute-phase reaction proteins", and their increased levels in some disease states.[143] Diseases such as tuberculosis are characterised by a raised ESR and the effect of treatment is assessed by serial measurements of the ESR. Diseases such as tuberculosis (and AIDS) are not caused by red blood cell clumping induced by "the dielectric effect of proteins" but the fact this can be demonstrated and measured in vitro is of great diagnostic and prognostic utility in clinical practice. Thus "HIV" seropositivity, like the ESR, may represent nothing more than a non-specific indicator, serendipitiously discovered in 1983/84, of altered homeostasis connoting a propensity to develop particular diseases. As long as the present interpretation of a positive test is accepted this may never be ascertained because knowledge of seropositivity by both patient and physician attracts multiple confounding factors virtually impossible to eliminate.

(Even asymptomatic, non-anaemic HIV positive individuals may have an increased ESR[144] and the test may be predictive for disease progression.[145] In HIV positive children a correlation exists between seropositivity, hypergammaglobulinaemia and an elevated ESR.[146] As far back as 1988 researchers from the Institut National de Transfusion Sanguine, Paris, France, found that: "An increased ESR in HIV-seropositive subjects seems to constitute a predictive marker of progression towards AIDS before the decrease of the CD4 count".[147] In other words the ESR is a superior predictive marker for the development of the clinical AID syndrome than is a decrease in the CD4 cell count, although the latter is said to be the cause of the syndrome). (Papadopulos-Eleopulos E, et al. 2001)

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For our critique of another notorious document see the Durban Declaration Rebuttal. A rebuttal to the "Durban Declaration" published in Nature on July 6 2000. http://thedurbandeclaration.org/

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Glossary

KOCH'S POSTULATES

In the late nineteenth century, German scientist Robert Koch established a set of procedures to isolate and identify the causative agent of a particular microbial disease. The following four steps, which are still used today, are known as Koch's Postulates.

1. A specific organism must always be observed in association with the disease.
2. The organism must be isolated from an infected host and grown in pure culture in the laboratory.
3. When organisms from the pure culture are inoculated into a susceptible host organism, it must cause the disease.
4. The infectious organism must be reisolated from the diseased organism and grown in pure culture.

RETROVIRUS

A retrovirus is a virus which has a genome consisting of RNA and relies on reverse transcriptase, an enzyme, to perform a kind of reverse-translation of its genome from RNA into DNA for insertion into the host's genome. The virus itself is just a storage form for its RNA; the reverse transcription takes place in the host's cytosol, the internal fluid of the cell. A retrovirus' genome integrated into the host's genome is called a provirus. One of the characteristics that distinguish retroviruses from a typical viruses is that they do not kill host cells but rather persist within them in one form or another. Human Endogenous Retroviruses (HERVs). are retroviruses that become dormant and exist as DNA copies in every cell in the body of the host and are passed down from generation to generation. Our bodies express these sorts of particles all the time and they encompass 1% of the human genome. This fact complicates identifying any retroviruses that might be exogenous (from outside the host) and have a unique and atypical ability to cause disease.

The retrovirus genome contains at least three genes:
• gag codes for core and structural proteins of the virus.
• pol codes for reverse transcriptase.
• env codes for the virus hull proteins.

GOLD STANDARD

"Gold Standard" in diagnostic medicine refers to the most specific and sensitive test to validate other tests. Often it is impractical to use the most specific and sensitive test for general screening tests for infection. Therefore, a cheap and relatively accurate proxy text is developed. To assess the accuracy of these tests studies are done comparing the proxy test's results against the results from the gold standard on the same test samples.

PCR (POLYMERASE CHAIN REACTION)

Polymerase chain reaction, (PCR) a rapid technique for in vitro amplification of specific DNA or RNA sequences, allowing small quantities of short sequences to be analyzed without cloning: oligonucleotide primers are annealed to single-stranded nucleotide sequences, which are copied by polymerase; the number of copies is geometrically amplified by repeated cycles of annealing and copying.

VIRAL LOAD TESTS

Quantitative competitive (QC-PCR) is the basic principle behind viral load tests. Early on, PCR promised the ability to determine the amount of target DNA present in a sample--a very enticing goal. Regular PCR determines whether the target is present. Quantitative PCR determines how much is present: Is it at a level that needs action, or is the level so low as to be irrelevant? Quantitative PCR should be simple. Theoretically, the amount of end product doubles with each amplification cycle. However, this makes several false assumptions, the worst of which is that reaction occurs at 100 percent efficiency in all amplification cycles.

For quantitative PCR, this efficiency problem is usually addressed with an artificial control: a competitor amplicon. Hence, this type of reaction is known as quantitative competitive PCR (QC-PCR). Once the control amplicon is created, it is quantitated, a known amount of control is spiked into the sample, and amplifications run for a fixed number of cycles. After the PCR reaction is complete, the products get quantitated to determine the ratio of target to control.

QC-PCR has numerous drawbacks. The main difficulty is creating and optimizing the control amplicon. Additionally, there needs to be a method for the accurate quantitation of the end products. This can be time consuming and prevents high-throughput applications. Finally, and not uniquely to QC-PCR, contamination is always a threat. These drawbacks have severely hampered the development and use of quantitative PCR. (Investigen Inc.)

ULTRACENTRIFIGATION

The process of separating particles into bands or layers based on their density in a sucrose medium. An ultracentrifuge has an exceedingly high rate of rotation which will separate and sediment the molecules of a substance.

Illustration: http://healtoronto.com/nih/images/ultracent.jpg

THE HIV WESTERN BLOT TEST

The HIV Western blot test consists of a thin nitrocellulose strip in which are embedded proteins claimed to be unique to HIV. Each protein is labelled with a "p" followed by its molecular weight in thousands. Serum is added to the strip and, if there are antibodies to a particular protein, this band will "light up". Even though the proteins are claimed to be unique to HIV, reaction with an insufficient number is considered an "indeterminate" test result, and usually is treated as negative, indicating that false positive reactions are quite common. The HIV Western blot is not standardised, and thus, around the world, different combinations of bands are considered positive. Hence a positive test in one country is not positive in another. An African would not be positive in Australia. A person from the MACS would not be positive anywhere in the world, including Africa. Yet the HIV Western blot is considered to be highly specific and is considered synonymous with HIV infection.

Illustration: http://healtoronto.com/nih/images/wbtests.gif

Glossary continued

Key: AFR = Africa;1 AUS = Australia;2 FDA = US Food and Drug Administration;3 RCX = US Red Cross;3 CDC = US Centers for Disease Control;3 CON = US Consortium for Retrovirus Serology Standardization;3 GER = Germany; UK = United Kingdom; FRA = France; MAC = US Multicenter AIDS Cohort Study 1983-1992

According to data presented in Lundberg et al.,3 when the US 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 HIV-positive by the same criteria.

Note: In February 1993, the US FDA relaxed its stringent criteria in order to "reduce the number of HIV-1 seroindeterminate Western blot interpretations"; that is, to increase the number of HIV-positive individuals.4

Endnotes

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ANTIGEN

Antigens are recognized by the immune system to be foreign agents, and therefore must be removed so that the body can continue with its normal cellular processes without hindrance. Antigens may be soluble substances, such as toxins and foreign proteins, or particulate, such as bacteria and tissue cells. These antigens produce an immune response by the organism in response to their presence. Think of the word as a short form for antibody generating.

IMMUNE CROSS-REACTION

Binding of an antibody or cell receptor site with an antigen other than the one that would provide an exact 'fit', i.e. an antigen-antibody reaction in which the antigen is not the same one that stimulated the production of that antibody.

AIDS RISK GROUPS

Definition extracted from the Medline Plus Medical Encyclopedia:

High risk groups include homosexual or bisexual men, intravenous drug users who share needles*, the sexual partners of those in high risk groups, infants born to mothers with HIV, and persons who received blood transfusions or clotting products between 1977 and 1985 (prior to standard screening for the virus in the blood).

** The qualification "who share needles" is not a distinction made when it comes to HIV/AIDS surveillance. This leaves open the question whether shared needles or IV drug use is the real AIDS risk factor.*

In reality the concept of AIDS risk groups is a product of the bureaucracy of HIV/AIDS surveillance driven by a need to categorize reported cases into a "hierarchy of exposure categories". This obscures the real factors that might be the root cause of the patients' disease(s).

From the CDC Technical Notes on HIV/AIDS Surveillance:

For surveillance purposes, HIV infection cases and AIDS cases are counted only once in a hierarchy of exposure categories. Persons with more than one reported mode of exposure to HIV are classified in the exposure category listed first in the hierarchy, except for men with both a history of sexual contact with other men and injecting drug use. They make up a separate exposure category.

"Men who have sex with men" cases include men who report sexual contact with other men (i.e., homosexual contact) and men who report sexual contact with both men and women (i.e., bisexual contact). "Heterosexual contact" cases are in persons who report specific heterosexual contact with a person with, or at increased risk for, HIV infection (e.g., an injecting drug user).

Adults/adolescents born, or who had sex with someone born, in a country where heterosexual transmission was believed to be the predominant mode of HIV transmission (formerly classified as Pattern-II countries by the World Health Organization) are no longer classified as having heterosexually-acquired AIDS unless they meet the above stated criteria. Similar to other cases among persons who were reported without behavioral or transfusion risks for HIV, these cases are now classified (in the absence of other risk information which would classify them in another exposure category) as "no risk reported or identified" (MMWR 1994;43:155-60). Cases among children whose mother was born, or whose mother had sex with someone born, in a Pattern II country are now classified (in the absence of other risk information which would classify them into another exposure category) as "Mother with/at risk for HIV infection: has HIV infection, risk not specified."

Cases among persons with no reported history of exposure to HIV through any of the routes listed in the hierarchy of exposure categories are classified as "no risk reported or identified."

CDC 2001 HIV/AIDS Surveillance Report (September 25, 2002)
Table 17. Adult/adolescent AIDS cases by single and multiple exposure categories, reported through December 2001, United States

CASE DEFINITION

Acquired Immunodeficiency Syndrome (AIDS)

from PAHO Epidemiological Bulletin, Vol. 22 No. 2, June 2001

Rationale for surveillance

AIDS is a disease targeted for reduced incidence, prevalence and transmission (9GPW, target 6.3). Control measures are based on prevention and care strategies. Surveillance is necessary to assess national needs in education, supplies, and health care and to anticipate spread in the community. Surveillance will provide epidemiological data used for national prevention and care plan and will be essential to evaluate the impact of control activities.

Recommended case definitions

Different case definitions are used in different countries, depending on population factors (children, adults, relative occurrence of opportunistic infections) and on the laboratory infrastructure and training available. Current case definitions include: (1–3: for sophisticated laboratory facilities; 4–6 for limited laboratory facilities)

(1) CDC 1987;

(2) CDC/CD4;

(3) European;

(4) WHO for surveillance (formerly Bangui/WHO/clinical);

(5) Expanded WHO for surveillance (formerly Abidjan);

(6) Caracas/PAHO & revised Caracas/PAHO.

1. 1987 CDC Surveillance definition for AIDS (1)

1A. Without laboratory evidence of HIV infection (no other causes of immune suppression)

(Indicator disease diagnosed definitively)

- Candidiasis of the oesophagus, trachea, bronchi, or lungs
- Cryptococcosis, extrapulmonary
- Cryptosporidiosis with diarrhoea persisting >1 month
- Cytomegalovirus diseases of an organ other than liver, spleen, or lymph nodes in patient >1 month of age
- Herpes simplex virus infection causing a mucocutaneous ulcer persisting >1 month; or bronchitis, pneumonitis, or oesophagitis for any duration in a patient >1 month of age
- Kaposi sarcoma in a patient <60 years of age
- Lymphoma of the brain (primary) affecting a patient <60 years of age
- Mycobacterium avium complex or M. kansasii disease, disseminated (site other than/in addition to lungs, skin, cervical or hilar lymph nodes)
- Pneumocystis carinii pneumonia
- Progressive multifocal leukoencephalopathy
- Toxoplasmosis of the brain in a patient >1 month of age
- In children <13: 2 or more bacterial infections within a 2-year period (septicaemia, pneumonia, meningitis, bone or joint infections…) or abscess of an internal organ or body cavity – excluding otitis media or superficial abscesses.

1B. With laboratory evidence of HIV infection

(Indicator disease diagnosed definitively)

- Coccidioidomycosis, disseminated (at a site other than or in addition to lungs or cervical or hilar lymph nodes)
- HIV encephalopathy
- Histoplasmosis, disseminated (other than or in addition to lungs or cervical or hilar lymph nodes)
- Isosporiasis with diarrhoea persisting >1 month
- Kaposi sarcoma at any age
- Lymphoma of the brain (primary) at any age
- Non-Hodgkin's lymphoma
- Any mycobacterial disease caused by other than M. tuberculosis, disseminated
- Disease caused by M. tuberculosis, extrapulmonary
- Salmonella (non-typhoid) septicaemia, recurrent
- HIV wasting syndrome
- Indicator disease diagnosed presumptively
- Candidiasis of the oesophagus
- Cytomegalovirus retinitis with loss of vision
- Kaposi sarcoma
- Mycobacterial disease, disseminated
- Pneumocystis carinii pneumonia
- Toxoplasmosis of the brain in patient >1 month of age
- In children <13: lymphoid interstitial pneumonia and/or pulmonary lymphoid hyperplasia

2. Conditions added to CDC surveillance definition for AIDS with laboratory evidence of HIV infection (1B above) (2). In addition to those in the surveillance definition:

- CD4+ T-lymphocyte count <200 x 106/litre (or a CD4 percentage <14%)
- Pulmonary tuberculosis
- Cervical cancer, invasive
- Recurrent pneumonia (more than one episode within a 12-month period).

3. European AIDS case definition [3]

Same as revised CDC definition (2 above) without CD4+ T-lymphocyte count.

4. WHO Case definition for AIDS surveillance (formerly Bangui/WHO/Clinical) [5]

WHO clinical case definition for AIDS in an adult or adolescents (>12 years of age) when diagnostic resources are limited. For the purposes of AIDS surveillance an adult or adolescent (>12 years of age) is considered to have AIDS if at least 2 of the following major signs are present in combination with at least 1 of the minor signs listed below, and if these signs are not known to be related to a condition unrelated to HIV infection.

Major signs (2 signs or more):

- Weight loss of at least 10% of body weight
- Chronic diarrhoea for >1 month
- Prolonged fever for >1 month (intermittent or constant)

Minor signs (1 or more):

- Persistent cough for >1 month
- Generalized pruritic dermatitis
- History of herpes zoster
- Oropharyngeal candidiasis
- Chronic progressive or disseminated herpes virus infection
- Generalized lymphadenopathy.

The presence of either generalized Kaposi sarcoma or cryptococcal meningitis is sufficient for the diagnosis of AIDS for surveillance purposes.

CASE DEFINITION continued

Acquired Immunodeficiency Syndrome (AIDS)

5. Expanded WHO case definition for AIDS surveillance (formerly Abidjan) [4]

For the purpose of epidemiological surveillance, an adult (>12 years of age) is considered to have AIDS if a test for HIV antibody shows positive results, and one or more of the following are present:

- 10% body weight loss or cachexia, with diarrhoea or fever, or both, intermittent or constant, for at least 1 month, not known to be due to a condition unrelated to HIV infection
- Cryptococcal meningitis
- Pulmonary or extra-pulmonary tuberculosis
- Kaposi sarcoma
- Neurological impairment sufficient to prevent independent daily activities not known to be due to a condition unrelated to HIV infection (for example, trauma or cerebrovascular accident)
- Candidiasis of the oesophagus (which may presumptively be diagnosed based on the presence of oral candidiasis accompanied by dysphagia)
- Clinically diagnosed life-threatening or recurrent episodes of pneumonia, with or without etiological confirmation
- Invasive cervical cancer

6. Revised Caracas/PAHO AIDS Definition [6,7]

A patient is defined as having AIDS when:

- cumulative points assigned for conditions listed hereafter equal or exceed 10, and
- HIV serology is positive.

Cases in which the total point score equals or exceeds the required score of 10, but HIV serology is pending are considered "provisional cases". Persons with cancer, or with immunosuppressive therapies, or where the sign / symptoms are attributed to conditions other than HIV infection are excluded.

Symptoms/signs/diagnostic Assigned points

- Kaposi sarcoma [10]
- Disseminated / extrapulmonary / non-cavity pulmonary tuberculosis [10]
- Oral candidiasis / hairy leukoplasia [5]
- Pulmonary tuberculosis with cavitation, or unspecified [5]
- Herpes zoster in a person of 60 years or less [5]
- Central nervous system dysfunction [5]
- Diarrhea one month or more [2]
- Fever of at least 38oC for at least a month [2]
- Cachexia or weight loss of more than 10% [2]
- Asthenia of at least a month [2]
- Persistent dermatitis [2]
- Anaemia, lymphopenia, and/or thrombocytopenia [2]
- Persistent cough or any pneumonia, and/or thrombocytopenia [2]
- Lymphadenopathy of at least 1cm at at least two non-inguinal sites [2]

Required point score 10 or more

Case classification

Depends on the case definition. Please check with National AIDS programmes.

Recommended types of surveillance

Routine monthly reporting of aggregated data from periphery to intermediate level.

Routine quarterly reporting of aggregated data from intermediate level to central level.

International: report updates every 12 months in the Weekly Epidemiological Record

Other sources of data:

- Hospitals
- Practitioners
- Consultations for sexually transmitted diseases (STD)
- Tuberculosis wards
- Mortality reports and statistics
- Active case finding.

Recommended minimum data elements

Case-based data for reporting

Unique identifier, age, sex, geographical area, mode of transmission (e.g., blood transfusion, drug use, other).

Aggregated data for reporting

Number of cases by age and sex, number of cases, mode of transmission (e.g., blood transfusion, drug use, other).

Recommended data analyses, presentation, reports

Graphs: Number of cases by age, sex, geographical area, risk factors.

Tables: Number of cases by age, sex, geographical area, risk factors.

Maps: Number of cases by geographical area.

Principal uses of data for decision-making

- Assess the magnitude of the problem
- Identify high risk areas for further intervention
- Plan public health measurements
- Assess impact on clinical services
- Plan health care services and supplies
- Validate HIV surveillance data

References

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3. Lancet, 1993;341:441 and AIDS Surveillance in Europe, Quarterly Report, 1993 ;number 37
4. AIDS 1993, Vol. 7 (suppl 1)
5. AIDS 1993, Vol. 7 (suppl 1)
6. Epidemiological Bulletin of PAHO Vol. 10 # 4 1990 Working group on AIDS case definition pages 9-11 o Journal of Acquired Immune Deficiency Syndrome Vol. 5 # 12 1992 " A simplified surveillance case definition of AIDS derived from empirical clinical data".
7. AIDS 1993, Vol. 7 (suppl 1)

Source: "WHO Recommended Surveillance Standards, Second edition, October 1999: , WHO/CDS/CSR/ISR/99.[2]

CASE DEFINITION continued

Acquired Immunodeficiency Syndrome (AIDS)

from PAHO Epidemiological Bulletin, Vol. 22 No. 2, June 2001

The 1993 AIDS Surveillance Case Definition of the U.S. Centers for Disease Control and Prevention*

A diagnosis of AIDS is made whenever a person is HIV-positive and:

- he or she has a CD4+ cell count below 200 cells per microliter

OR

- his or her CD4+ cells account for fewer than 14 percent of all lymphocytes OR
- that person has been diagnosed with one or more of the AIDS-defining illnesses listed below.

AIDS-Defining Illnesses:

- Candidiasis of bronchi, trachea, or lungs (see Fungal Infections)
- Candidiasis, esophageal (see Fungal Infections)
- Cervical cancer, invasive‡
- Coccidioidomycosis, disseminated (see Fungal Infections)
- Cryptococcosis, extrapulmonary (see Fungal Infections)
- Cryptosporidiosis, chronic intestinal (>1 month duration) (see Enteric Diseases)
- Cytomegalovirus disease (other than liver, spleen, or lymph nodes)
- Cytomegalovirus retinitis (with loss of vision)
- Encephalopathy, HIV-related† (see Dementia)
- Herpes simplex: chronic ulcer(s) (>1 month duration) or bronchitis, pneumonitis, or esophagitis
- Histoplasmosis, disseminated (see Fungal Infections)
- Isosporiasis, chronic intestinal (>1 month duration) (see Enteric Diseases)
- Kaposi's sarcoma
- Lymphoma, Burkitt's
- Lymphoma, immunoblastic
- Lymphoma, primary, of brain (primary central nervous system lymphoma)
- Mycobacterium avium complex or disease caused by M. Kansasii, disseminated
- Disease caused by Mycobacterium tuberculosis, any site (pulmonary‡ or extrapulmonary†) (see Tuberculosis)
- Disease caused by Mycobacterium, other species or unidentified species, disseminated
- Pneumocystis carinii pneumonia
- Pneumonia, recurrent‡ (see Bacterial Infections)
- Progressive multifocal leukoencephalopathy
- Salmonella septicemia, recurrent (see Bacterial Infections)
- Toxoplasmosis of brain (encephalitis)
- Wasting syndrome caused by HIV infection†

Additional Illnesses That Are AIDS-Defining in Children, But Not Adults

- Multiple, recurrent bacterial infections† (see Bacterial Infections)
- Lymphoid interstitial pneumonia/pulmonary lymphoid hyperplasia

** Entries on AIDS-defining illnesses can be found in the Encyclopedia of AIDS under the name given, unless otherwise noted in parentheses. Terminology may vary.*

† Added in the 1987 expansion.

‡ Added in the 1993 expansion.

(The Encyclopedia of AIDS