BACK

The Perth Group

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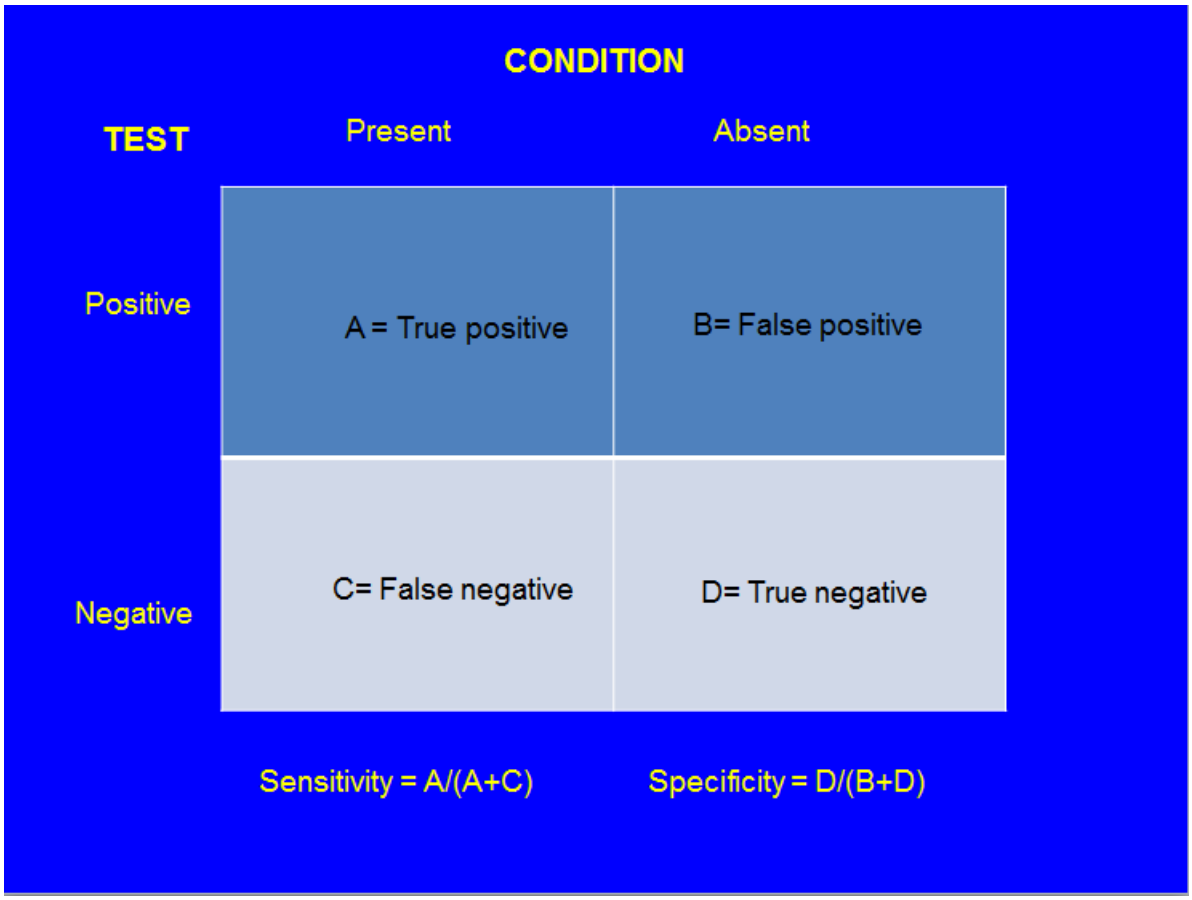
If there is no HIV why are there "HIV" antibody tests?

Exposure to foreign agents both living and non-living, generically known as antigens (from ANTIbody GENerating), results in the production of antibodies from cells known as B lymphocytes or B-cells. Antibodies are proteins which immunologists describe as "directed against" or "to" a given antigen. Antibody reacts with (binds to) the immunising antigen forming an antigen-antibody complex. Infection with HIV is claimed to produce antibodies directed against its several proteins. Serological diagnosis of HIV infection is based on the detection of such antibodies. In *Harrison's Textbook of Internal Medicine* Anthony Fauci writes "a positive EIA [enzyme immunoassay] with a confirmatory Western blot remains the "gold standard" for a diagnosis of HIV infection".[1]

In the EIA test (aka ELISA, enzyme-linked immunosorbent assay) the antigens are a mixture of "HIV" proteins. In the Western blot the proteins are separated along the length of a nitrocellulose strip or other medium. To perform an antibody test a sample of blood is obtained, the serum separated, diluted, and then added to the proteins in the EIA and Western blot test kits. If antigen-antibody complexes form these are detected as a physical alteration in the reaction medium, typically a colour change. The colour change is the evidence an antibody has reacted with a protein. In the EIA the colour is quantified by the change in optical density, which is read with a spectrophotometer. The Western blot produces a series of horizontal lines known as "bands" which are read and interpreted visually. With 12 antigens there are 4096 possible band combinations (one of which is zero bands). In many countries (including the US and Australia) an initially reactive (positive) EIA is repeated and if twice reactive a "confirmatory" Western blot is performed. Different combinations of Western blot bands are interpreted as either positive, indeterminate or negative. Negative is no bands. Indeterminate is a Western blot in which the band combination does not fulfil the criteria for being positive. If the Western blot is positive that person is classified "HIV antibody positive" and reported HIV-infected. According to the HIV experts, HIV antibody testing is extraordinarily specific, that is, there are virtually zero false-positives. There are several reasons to question this assertion, or indeed whether any "HIV antibody positive" individual is infected with a retrovirus HIV.[2-10]

The aim of all tests used in clinical medicine is to distinguish between having or not having a particular condition or disease. For example, does a patient with chest pain have or not have an acute myocardial infarction? Is a woman whose menses has ceased pregnant or not? Does a person with fever, lassitude and myalgias have influenza? Most tests are not the condition, disease or infectious agent being sought. They are indirect and considered far preferable because, in general, they are less invasive, less technically demanding and time consuming, and cheaper than directly detecting the condition, disease or object.

Before a test is introduced into routine clinical practice its parameters must be verified against the actual condition for which it is to be used. The test parameters of principal interest are sensitivity and specificity. These are determined by tabulating the presence or absence of the condition sought against positive and negative tests. The condition is the gold standard for the test and the means of determining the condition must be independent of the test. A test cannot be its own gold standard.[11] The results are put in a 2 X 2 table where the columns are the presence of absence of the condition sought and the rows the test result. Although obvious, it is important to stress that what is used to validate the test result decrees the condition for which the test parameters apply.



Ideally, every time a condition is present the test is positive (100% sensitivity) and every time the condition is absent the test will be negative (100% specificity). A false-positive occurs when the condition is absent but the test is positive. One or more false-positives means the test cannot be 100% specific. Determining the parameters of a test can be illustrated using the example of a blood test for pregnancy. Positive and negative tests are verified against the gold standard of pregnant/not pregnant (babies born/not born or positive/negative ultrasound scans).

In the case of a test for HIV infection the gold standard is HIV. That is, is the presence or absence of HIV according to the results of virus isolation/purification experiments. However, nowhere in the scientific literature have such data been reported. What has been reported is "*de facto*" gold standards for HIV/no-HIV. Under this guise HIV-infection ≡ AIDS patients and no HIV-infection ≡ healthy blood donors. Nowadays it is common to use commercially available "reference sera" obtained from such individuals.[12]

Neither AIDS nor healthy blood donors are valid gold standards for a retroviral infection because they are not the presence or absence of a retroviral infection respectively. Test manufacturers are obviously aware of this flaw, typically including the following preamble in their test kit packet inserts. "At present, there is no recognized [gold] standard for establishing the presence or absence of antibodies to HIV-1 and HIV-2 in human blood". Not only is this wrong (HIV isolation is the self-evidence and recognised gold standard), the statement acknowledges the problematic nature of the *de facto* standard. Use of the *de facto* standard leads to the following.

If healthy, non-HIV-risk blood donors are substituted for the absence of HIV, then all positive antibody tests in healthy blood donors are, by definition, false-positives. Given the number

of current blood donors, and the far greater number of potential blood donors worldwide, there must be large numbers of healthy, HIV positive individuals who are not infected with HIV (see addendum).

There are ample data that non-HIV antibodies react with the protein antigens in the HIV test kits.

1. Antibodies do not react exclusively with their inducing antigens. They may also react with other antigens, that is, they cross-react. The immunological community was "shocked" by the discovery of this behaviour, so much so it was they who applied the descriptor "promiscuous".[13]

2. AIDS patients are exposed to a plethora of foreign antigens resulting in a surfeit of antibodies, any or all of which are capable of cross-reacting with other antigens including the "HIV" protein antigens.

3. The surfeit includes autoantibodies, the latter being antibodies directed against "self" antigens, that is, a person's own proteins. Thus, following arguments presented elsewhere that the "HIV" proteins are cellular, the association between a positive "HIV" antibody test and AIDS is predictable. In other words, "HIV" antibodies are subset of a much larger set of autoantibodies present in AIDS patients that react with many "self" antigens that are not "HIV" proteins.[14', 15]

4. HIV positive individuals with or without AIDS characteristically have raised levels of the gamma globulin fraction of the plasma proteins (up to 70%). This is because, as Fauci affirms, "Aberrant immune activation [stimulation] is the hallmark of HIV infection and is a critical component of the pathogenesis of HIV disease. This activated state is reflected by hyperactivation of B cells leading to hypergammaglobulinemia".[1] The gammaglobulin fraction of plasma includes antibodies and the greater the number of antibodies the greater the probability of cross-reactions. In fact raised gammaglobulins predict a positive antibody test. For example, using the Western blot as a "gold standard", hypergammaglobulinaemia identifies HIV infected children with a specificity of 97%.[16]

5. Data published between the mid and late 2000s show that "Oxidation–reduction (redox) reactions can "unmask" autoantibody activity in blood and other body fluids from normal, healthy individuals…The autoantibodies unmasked by redox reactivities represent a growing list of specificities, many that are responsible for modulating and/or regulating intracellular functions".[17] Since HIV positive and AIDS patients have undergone cellular oxidation,[18-22] "HIV" antibodies may be nothing more than unmasked "non-HIV" antibodies bearing no relationship to a retroviral infection.

6. Even if the proteins in the test kits were retroviral, autoantibodies, like all antibodies, may also cross-react. Hence "non-HIV" autoantibodies may react with the "HIV" proteins to cause positive tests.

7. "HIV" antibodies including positive WBs occur where there is no HIV or AIDS. For example, dogs do not develop AIDS but in 1991 Strandstrom and colleagues reported that 72/144 (50%) of dog blood samples "obtained from the Veterinary Medical Teaching Hospital, University of California, Davis" tested in commercial WB assays, "reacted with one or more HIV recombinant proteins" (gp120 – 21.5%, gp41 – 23%, p31– 22%, p24 – 43%)[23]

8. 62% (45/73) of sera in patients with measles demonstrated Western blot bands corresponding to HIV-1 *pol* and *gag* antigens. The most prevalent bands were p18,

p24, p41 and p55. The authors concluded that “these findings suggest that the immune response to natural measles virus infection results in the production of antibodies to HIV-1”[24] although they are not “HIV antibodies”.

9. In 1991 based on antibody/antigen reactions Faulk and Labarrere documented the p18, p24 and p120 “HIV” proteins (test-kit antigens) in the placentas of 25 normal term pregnancies of healthy women.[25] They wrote “It must be stressed that the reactive cells [antibody identified “HIV” proteins] do not imply infection with HIV, for none of the mothers had histories of HIV infections, all the pregnancies were normal”.

10. Patients transfused with HIV negative blood develop antibodies to the “HIV” proteins. Genesca *et al* conducted Western blot assays in 100 EIA negative samples of healthy blood donors; 20 were found to have HIV bands which did not fulfil the then (1989) criteria used by the blood banks for a positive WB. These were considered indeterminate WB, (WBi), with p24 being the predominant band (70% of cases). Among the recipients of WBi blood, 36% were WBi 6 months after transfusion but so were 42% of individuals who received WB negative blood. Both donors and recipients of blood remained healthy. They concluded that WBi patterns "are exceedingly common in randomly selected donors and recipients and such patterns do not correlate with the presence of HIV-1 or the transmission of HIV-1", "most such reactions represent false-positive results".[26] Genesca also noted that “48-64% of donors repeatedly reactive for anti-HIV-1 by EIA have WBi patterns. The frequency of such patterns in low-risk populations is so high as to suggest that, as with EIA, most such reactions represent false-positive results”. As discussed above, if low risk populations with their relatively low abundance of antibodies have so many false positives why not high risk populations with their much greater abundance of antibodies?

11. Among 89,547 anonymously tested blood specimens from 26 US hospital patients at no risk of AIDS, from 0.7% to 21.7% of men and 0-7.8% of women aged 25-44 years were found to be EIA and HIV WB positive.[27] This study not only excluded patients in the known AIDS risk groups but also patients with almost a hundred other diseases including "gunshot and knife wounds” all of which pose meagre if any risk of HIV/AIDS.[28] If one hundred different diseases bearing no relationship to AIDS may result in antibodies considered “non-HIV”, why are the same antibodies “HIV” when present in another 29 “AIDS” diseases?

12. The time course decay of antibodies in newborn infants of HIV positive mothers is sufficient proof that “HIV” antibodies cannot be the result of “HIV” infection.[29]

13. As mentioned, in the Western blot the individual “HIV” proteins are separated along a nitrocellulose strip. Serum (antibodies) is added and the proteins that react undergo a colour change producing a series of horizontal lines known as “bands”. Prior to 1987 a positive Western blot consisted of a p24 or p41 band or both. Under these criteria 15-40% individuals not at risk of AIDS tested “HIV positive”,[30-33] a finding that even the HIV experts recognised as problematic. Beginning in 1987 the problem was remedied by arbitrarily changing the number and mix of bands thereby making it “harder” for an individual to be classified HIV positive. Since then the criteria (position and number of bands) have progressively varied between countries, institutions and laboratories.[10, 15, 34, 35]

Table 1. Global Variation in the Criteria for a Positive Western Blot.*

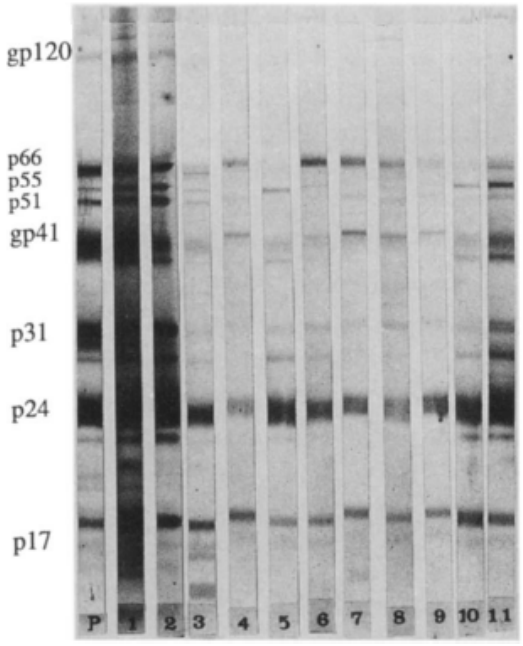
Organization	Criteria
CDC and ASTPHLD	Two bands of GP41 or GP120/GP160 or p24
FDA (United States)	p24 and p31 and either GP41 or GP120/GP160
SFTS (France)	
Unequivocally positive	Two ENV bands (GP160 and GP120) with GAG or POL
Probably positive	ENV (GP160) and GAG (p24)
Probably positive	Two ENV bands only (GP160 and GP120)
World Health Organization	Two ENV bands, with or without GAG or POL
CRSS and Pan American Health Organization	One p24 or p31 band and one ENV band
American Red Cross	One GAG band, one POL band, and one ENV band
Paul Ehrlich Institut (Germany)	Two bands; one must be ENV
China	Two ENV bands or one ENV band and p24
Singapore	Two ENV bands (GP160/GP41 and GP120) and any GAG or POL band
Australia	One ENV band and any three GAG or POL bands

* CDC denotes Centers for Disease Control and Prevention, ASTPHLD Association of State and Territorial Public Health Laboratory Directors, GP glycoprotein, FDA Food and Drug Administration, SFTS Sanguine Nationale Transfusion Sociétés, and CRSS Consortium for Retrovirus Serology Standardization. Data are from Genelabs.[5]

14. The most obvious example of "tuning the tests" is that of Africa where the correlation between what constitutes the African clinical syndrome[36, 37] and a positive antibody test is particularly poor. (In one study 83% of patients with suspected AIDS were HIV positive but so were 44% with malaria, 97% with herpes zoster, 43% with pneumonia, 67% with amoebic dysentery and 41% with carcinoma.[33] In another study 42% of women with recurrent abortions, 67% with vaginal ulcerations and 33% with haemorrhoids had a positive HIV antibody test[38]). The high prevalence of positive tests in the "wrong" patients in Africa was an even greater problem than in the developed world. This is why in 1990 HIV experts adopted the revised WHO criteria for Africa to "increase the specificity of the positive [Western blot] criteria" by mandating "at least two *env* bands", that is, two of p41, p120 and p160 bands, regardless of the presence or absence of any other bands[39] (whose presence in various combinations is mandated everywhere else in the world). The new criteria replaced the 1987 WHO criteria that consisted of "one *env* band (gp4l, gp120 or gp160) plus any other virus-specific band". (Strecker and his colleagues' criteria were even more lenient – "at least [any] two immunologically distinct bands must be present on the strip for a positive result"[40]). The revised WHO/African criteria create several paradoxes.

    In 1994 Oscar Kashala and Max Essex[41] reported antibody test data on leprosy patients and their contacts ("family members and other persons living within 1.6 km of the leprosarium who interacted daily with the patients"). Leprosy is caused by *Mycobacterium leprae*, a bacterium that "shares several antigenic determinants with other mycobacterial species, including *M. tuberculosis*". They reported that "WB were indeterminate [neither positive nor negative] in 46 (83.6%) of 55 leprosy patients and 19 (3.9%) of 482 HIV-negative pregnant women (table 1)". In fact "indeterminate patterns were also found in a higher proportion of leprosy contacts (25/39; 64.1%)". In their Figure 1 they published several representative Western blot strips of leprosy contacts and patients.

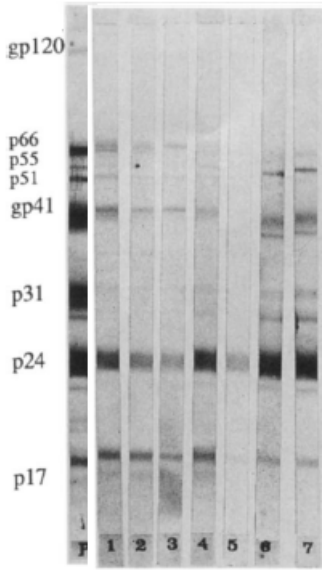
Kashala *et al* Figure 1



"ELISA [EIA]-positive sera from leprosy patients and contacts. p, positive control serum. Lanes: 1, 2, HIV-1-positive sera from 2 leprosy patients; 3-5, sera from 3 leprosy contacts with indeterminate Western Blots (WB); 6-11, indeterminate WB patterns of sera from 6 leprosy patients".

The authors reported strips 1 and 2 positive using the revised WHO criteria for Africa. That is, "WB was considered diagnostic for HIV-1 if there was reactivity with two of three envelope bands (gp 160/120 and gp41)". All the other strips were considered indeterminate. However, every "indeterminate" strip in this figure is diagnostic (HIV positive) according to the criteria of every other laboratory, institution and country, including Australia where the criteria are the most stringent.

The fact is that EIA screening is the Western blot gatekeeper, thereby obviating the awkward question "Is a positive Western blot proof of HIV infection when the EIA is negative"? As Kashala further showed, in leprosy patients HIV-1 EIA negative sera can be HIV positive on the Western blot using the criteria of several institutions.



Western blots of HIV-1 EIA-negative sera from leprosy patients (lanes 1-4) and controls (lanes 5-7).
F = positive control (WHO criteria).

Tuberculosis is the principal AIDS-defining disease in Africa. Hence it is a mystery that Kashala and Essex did not test tuberculosis patients for "cross-reacting" antibodies. Especially, as the authors pointed out, "*M. leprae* shares several antigenic determinants with other mycobacterial species, including *M. tuberculosis*, and are expected to produce antibodies with similar patterns of reactivities. Despite this omission, in concluding their paper the authors stressed this possibility, "HIV-1 ELISA and WB results should be interpreted with caution when screening individuals infected with *M. tuberculosis* or other mycobacterial species. ELISA and WB may not be sufficient for HIV diagnosis in AIDS-endemic areas of Central Africa where the prevalence of mycobacterial diseases is quite high".

15. There have never been uniform criteria for defining a positive "confirmatory", Western blot test. The implication of the variations between countries, laboratories and institutions is that one and the same patient may be antibody positive under one set of criteria and not positive under another or several others. This fact rarely if ever reaches the ears of physicians or the hundreds of epidemiologists whose studies are based on antibody testing, let alone patients and the general public.[6-8, 10, 15, 34, 35] This issue is the subject of an exchange between Brent Leung and microbiologist Dr. Claudia Kücherer at the Koch Institute in Berlin in *House of Numbers*[42] (at time 31:06).

16. There have also been changes in the definition of AIDS (the most important in 1987 and 1993) which kept the incidence and number of cases increasing.[43] For example, "There is a threefold increase in patients in the Edinburgh City Hospital Cohort [532 HIV-seropositive individuals] defined as having AIDS under the 1987 and the proposed 1992 [1993] CDC definitions".[44] Under the 1987 definition[45] AIDS could be diagnosed when (a) evidence of HIV infection was "not performed or gave inconclusive results" or (b) even when all tests were negative, that is, when there was definite evidence the patient was not infected with HIV or (c) in the absence of any evidence of immune deficiency and even when the cause of immune deficiency could have been other than HIV. Under the 1993 definition[46] it was legitimate to diagnose AIDS in "All HIV-infected persons who have <200 CD4 + T lymphocyte counts per microliter", that is, without an indicator disease. Hence a terminally ill patient with PCP and an asymptomatic person are both reported as AIDS.

The number of AIDS cases was further increased by several factors including the introduction in 1987 of “mild and moderate disease” as AIDS and permitting a presumptive diagnosis of indicator diseases based on non-specific findings. In other words throughout the AIDS era the correlation between “HIV” antibodies and AIDS has been maintained by a process of selective adjustment – by discrimatory testing and changes to both the definition of a positive test as well as the clinical definition of AIDS.[43, 47] As a result correlation between a positive antibody test and AIDS has been at the behest of manipulation by committees, not the putative agent.[6-8, 10, 15, 34, 35]

If there is no retrovirus HIV why are the antibody tests correlated with AIDS?

In the early 1990s UK science journalist Neville Hodgkinson and Professor Ronald Penny, clinical immunologist at St. Vincent’s Hospital in Sydney, were interviewed by Philip Adams on ABC radio (Australia). Professor Penny presented a seemingly powerful defence against the nascent questioning of the HIV theory of AIDS. He said: “Wherever you have AIDS you have HIV and wherever you don’t have AIDS you don’t have HIV”. It is crucial to understand that what Penny meant by “you have HIV” was “you have a positive antibody test”. He did not, indeed could not assert “wherever you have HIV you have a retrovirus HIV isolated from cell cultures of your blood or tissue” because even today there are no such data “wherever you have AIDS”.

Obviously Penny’s argument is premised on a positive antibody test ≡ HIV infection but there are no virus isolation gold standard data to authenticate this premise.[7, 8, 15] However, that does not devalue the correlation between a positive test and AIDS or its clinical and public health benefits. There are many situations in clinical medicine where an abnormal test points to or predicts a health problem. Fever is a common example, as an elevated peripheral blood neutrophil (white blood cell) count, C-reactive protein or erythrocyte sedimentation rate (ESR[48]). 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"[49] although the latter is claimed to be the direct cause of the clinical syndrome. If the rate at which red blood cells fall under gravity in a test-tube is a better predictor than “immune deficiency”, why not some antibodies that react with some proteins? The fact there is a link between a test and a disease does not prove causation. A correlated phenomenon may be nothing more than an epiphenomenon. The reason at risk individuals have positive “HIV” antibody tests is the predictable result of antibody promiscuity and the *milieu* of immune stimulation caused by many factors and the elevated antibody concentrations that typify the AIDS risk individuals.

Further reading

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Addendum

Using and perpetuating the wrong gold standard

In 1985, in the first of many methodologically identical publications, Stanley Weiss, Robert Gallo and their colleagues in The AIDS Seroepidemiology Collaborative Working Group reported a "Screening test for HTLV-III (AIDS agent) [HIV] antibodies. Specificity, sensitivity, and applications".[50] They concluded "The sensitivity and specificity of this HTLV-III ELISA for correctly identifying patients with AIDS were extremely high for a single-stage screening assay".

Weiss *et al* Figure 1

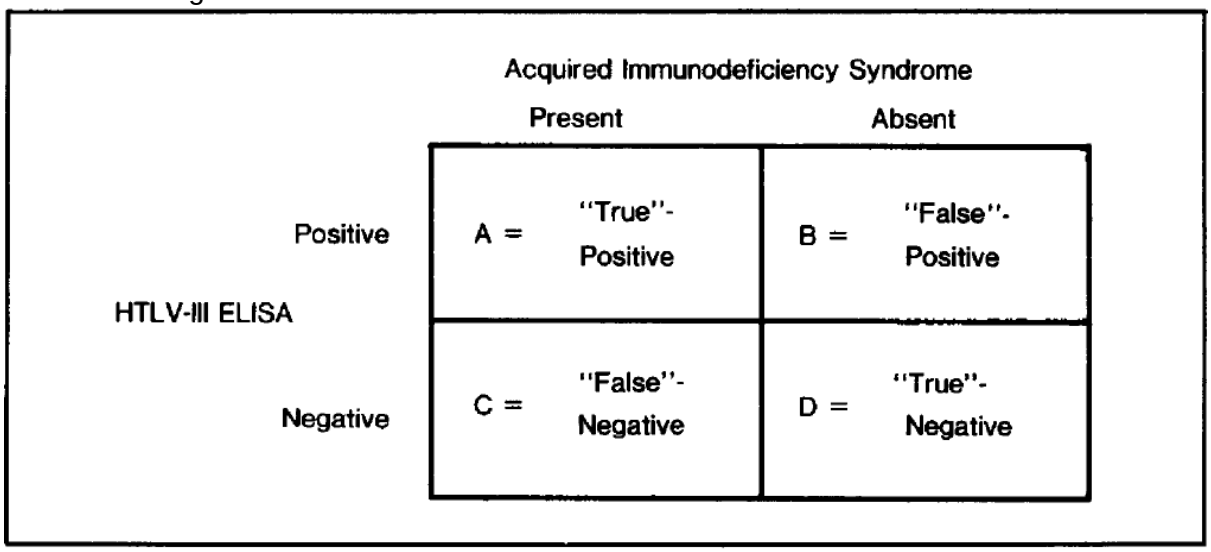

Fig 1.—Binary table for calculation of sensitivity [*A*/(*A*+*C*)] and specificity [*D*/(*B*+*D*)] of test results. HTLV-III indicates third member of the human T-cell leukemia (lymphotropic) retrovirus family; ELISA, enzyme-linked immunosorbent assay.

There are several problems with this interpretation.

1. Despite claiming their data determine the "Specificity, sensitivity" of a "Screening test for HTLV-III (AIDS agent) [HIV] antibodies" what the authors' table and data clearly document is a test for AIDS. The column title reads "Acquired Immunodeficiency Syndrome" which is either "Present" or "Absent". The boxes that read "True positive", "False Positive", "False Negative" and "True Negative" respectively document the relationships between a positive / negative test and having AIDS / being a healthy blood donor. These data do not define relationships between the test results and HIV. AIDS is not HIV. All Weiss *et al* can claim is that the antibody test reliably distinguished 88 AIDS patients from 297 healthy blood donors. There is no need for such a test. These individuals can be distinguished clinically (as did Weiss *et al* in order to write their paper).

2. From their data one cannot define even the sensitivity and specificity of the test for AIDS. The test ("HTVL-III ELISA") depicted in their table detects antibodies which react with a mixture of proteins claimed to be the "HIV" proteins. An antibody response to illness is not confined to the AIDS indicator diseases. Illnesses arising from many different pathologies stimulate the production of antibodies. For several of the reasons listed above, including most importantly antibody cross-reactivities, the Weiss *et al* "Acquired Immunodeficiency Syndrome" "Absent" patient group should have included sick individuals with clinical, biochemical and metabolic

features similar to AIDS patients but who do not have AIDS. Inclusion of such individuals is the only means by which antibodies producing "False-positive" tests for AIDS can be documented (see the Sentinel Hospitals Study (11) above). Healthy blood donors lack the impetus for antibody responses. By equating healthy blood donors with the absence of a particular condition a scientist could prove virtually all antibody tests highly specific. The use solely of healthy blood donors as absence of AIDS negates the Weiss *et al* data even as a test for AIDS.

3. Weiss *et al* assert their test is an "HTLV-III ELISA" and that the antibodies detected by the test are "HIV" antibodies because the antigens in the ELISA test kits are "HIV" proteins. In support they cite all four of the Gallo *et al* May 1984 *Science* papers. But in these papers the proteins were identified as "HIV" because they reacted with the same antibodies. That is, the antibodies present in AIDS patients. The argument - unknown antibodies X identify unknown proteins Y identify unknown antibodies X - is a scientific impossibility.

4. Weiss *et al* defined the "HTLV-III ELISA" as positive, borderline or negative based on optical density readings. Positive readings were defined such that 99% of the healthy blood donors would test negative. This is invalid. A positive test is one that is corroborated by a virus isolation/purification gold standard.

5. Weiss *et al* reported a few healthy blood donors with borderline ELISA results which were further evaluated using the Western blot, that is, with another antibody test whose parameters, like the ELISA, have never been verified using an HIV isolation/purification gold standard. On this basis Weiss *et al* concluded "Thus, the assumption (made on epidemiologic grounds) that any positive ELISA screening results among the blood donors could be assumed to represent "false-positives" appears to be valid". As discussed above, if "the assumption…appears to be valid", then all positive tests in healthy blood donors are "False Positive" and no healthy, antibody positive blood donor is infected with HIV. Nonetheless, HIV experts then assert there are "True", HIV positive healthy blood donors and healthy individuals in general. In fact, the majority of positive tests occur in healthy individuals. Which means globally most positive tests are "False positive". On any given day, one and the same healthy HIV positive blood donor would be counted a "False-positive" if enrolled in a study such as that of Weiss *et al* but a "True Positive" if tested by a private physician as part of a general medical examination.

The question that arises in regard to "HIV" positive healthy individuals is that of determining the risk to health. This is a different question from assessing the risk to AIDS patients and those at risk of developing the syndrome. The only way to determine the risk to healthy, low or no risk individuals is by testing a large group and following their clinical course over several years. However, since the mere knowledge of being antibody positive may prove detrimental to a person's health* regardless of a putative retroviral infection, the study must be conducted with both patients and physicians blinded to the test data until completion of the study. Since such data are unlikely to be forthcoming, forecasting outcomes for individuals who are HIV positive and healthy and not in an AIDS risk group is problematic. It is estimated that perhaps 20% of HIV positive individuals are unaware of their test status.[51] In the absence of the appropriate data, what benefit may ensue from testing is debatable.

Unfortunately, the use of AIDS/healthy blood donors as the "gold standard" for the "HIV" antibody tests that began with Weiss and his colleagues in 1985, has persisted throughout the AIDS era. For example, the 2002 Abbott EIA test kit packet insert states "Epidemiologic data **suggest** that the Acquired Immunodeficiency Syndrome (AIDS) is caused by at least two types of human immunodeficiency viruses, collectively designated HIV...Sensitivity for HIV-1 antibodies was computed based on the clinical diagnosis of AIDS. Specificity is based

on assay of blood donations from random donors" (emphasis added). In the 1997 version of the same packet insert, test parameters were reported with the following: Abbott reports "Sensitivity based on an assumed 100% prevalence of HIV-1 antibody in AIDS patients...Specificity based on an assumed zero prevalence of HIV-1 antibody in random donors". The only conclusion one can reach is that 33 years after the discovery of the "AIDS virus", the parameters of the "HIV" antibody tests remain unknown. Hence it impossible to know how many, if any "HIV positive" individuals are infected with a retrovirus "HIV".

*A situation akin to "pointing the bone", a traditional, ritualistic punishment practiced by Australian aborigines. A bone is pointed at an individual as a method of retribution. That individual soon becomes sick and death within weeks or months is an invariable consequence.[52]

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11. Donald Burke and his colleagues at the Walter Reed Army Institute of Research USA, are credited with proving that the Western blot has a specificity in excess of 99.9%. Yet this parameter was determined using the Western blot as its own gold standard. Burke tested a subpopulation of 135,187 military applicants chosen for an estimated very low HIV prevalence --1/10th that of a much larger pool of 1.2 million applicants. All applicants were screened with an initial ELISA, reactive ELISAs were repeated (in duplicate) and an initial WB performed. If diagnostic or reactive, a second WB was performed on another blood specimen. From the 135,187 applicants, there were 16 positive tests. In one the serum was unavailable for further testing and one applicant declined to provide a second sample. To distinguish between "truly HIV-infected" and "truly HIV-free" amongst the 15 positive

applicants Burke performed an additional four antibody tests, either WBs or a similar procedure. Fourteen samples were found positive by all four assays and all four were negative for one applicant. From this Burke and his associates calculated the false positive rate as 1 in 135,187 or 0.0007% with “a specificity of roughly 99.9993 percent“. Burke's erroneous definitions and derivations of the Western blot test parameters were flagged and accepted by the editorial writers for the purposes of advising physicians “The potential consequences of incorrectly informing a person that he or she is infected with HIV are severe: certainly anguish, fear, and depression; perhaps lost jobs, denied applications for health insurance, or aborted pregnancies; and possibly, suicide”.
See:
Burke DS, Brundage JF, Redfield RR, Damato JJ, Schable CA, Putman P, Visintine R, Kim HJ. Measurement of the false positive rate in a screening program for human immunodeficiency virus infections. N Engl J Med 1988. 319:961-964.
Weiss R, Thier SO. HIV testing is the answer--what's the question? N Engl J Med 1988. 319:1010-1012.

12. Biotechnolgy companies acknowledge non-use of the virus gold standard in their packet insert disclaimers: “At present there is no recognized standard for establishing the presence or absence of HIV-1 antibody in human blood” Source: Packet Inserts Axsym system (HIV-1/HIV-2) 1988, 1998. Abbott Laboratories USA.
13. According to immunologist John Marchalonis “For many years, it was considered that a single antibody bound only the antigen [protein] to which it was raised...In fact, the concept arose that monoclonal antibodies [every molecule identical] must be monospecific [react with only one protein in the universe of proteins]. The immunological community was shocked to find that B cells [whose surfaces have antibody molecules attached to them] could be polyreactive in binding multiple antigens to their surface that were complex and ostensibly unrelated to one another”. Marchalonis accorded this behaviour antibody "promiscuity". As long ago as 1969 the eminent Australian immunologist Sir Gustav Nossal wrote “An antibody molecule made following the injection of one antigen frequently can combine also with a second antigen…in other words, the antibody cross-reacts with the second antigen”.

In 2005 Paul Predki and his colleagues wrote: "The literature is replete with examples of crossreactive antibodies…the identity of cross-reactive proteins for the most part remains almost impossible to predict...Unrecognized, such cross-reactivity can have adverse consequences. The ability to assess and identify antibody cross-reactivity is an important but often inadequately addressed requirement for both research and clinical applications...”. The binding strength of a given antibody in a cross-reaction has been reported to be greater than for its “true” reaction, that is, for the protein against which the immune system produced the antibody in the first place. Predki documented this using a monoclonal antibody which reacted with 40 different protein antigens, binding to 16 of them more strongly than the antigen to which the antibody was raised. See Table 1 of Predki *et al*.

In 1997 Achim Kramer published data showing that a monoclonal antibody to the "HIV" specific p24 protein reacts with proteins found in bacteria, yeasts, amoebae, rabbits, monkeys and humans. The fungi include *Candida albicans*, the agent that causes one of the common AIDS indicator diseases. Nowadays a reaction between an anti-p24 antibody and proteins in a cell culture is considered proof of “HIV isolation”. Antibody promiscuity, that is, cross-reactivities, is one of the best kept secrets in immunology.
See:
Marchalonis JJ, Adelman MK, Robey IF, Schluter SF, Edmundson AB. Exquisite specificity and peptide epitope recognition promiscuity, properties shared by antibodies from sharks to humans. J Mol Recognit. 2001;14:110-121.

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48. The erythrocyte sedimentation rate (ESR) is the rate at which red blood cells sediment in a test tube in one hour. 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. The ESR is non-specific but when elevated, "is a measure of the presence and intensity of morbid processes within the body". A common cause of a raised ESR is infection and "Elevated ESRs are also seen with pregnancy, malignancy, collagen vascular diseases, rheumatic heart disease, and other chronic disease states, including human immunodeficiency virus infection". In fact 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.
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