HIV Tests Are Not HIV Tests

Henry H. Bauer, Ph.D.

ABSTRACT

Tests for human immunodeficiency virus (HIV) do not detect HIV; they respond "positive" to a wide range of physiological conditions. The seminal papers from Gallo's laboratory did not demonstrate HIV to be the cause of AIDS. The patent based on those papers did not demonstrate that the proposed HIV tests, which are actually for HIV *antibodies*, are specific for HIV antibodies.

From the original announcement that "HIV is the probable cause of AIDS" to "HIV antibodies demonstrate active infection by HIV" was an unwarranted progression that came almost subliminally. HIV test kits were approved only for blood screening and do not claim to diagnose infection. There is no gold standard for an HIV test; the existing tests are at most adjuncts to clinical diagnosis of actual infection by HIV.

Consequences of misapplication of "HIV" tests include subjecting healthy individuals to iatrogenic harm through life-long intake of highly toxic medication.

Introduction

So-called "HIV tests" have not been proven to detect infection by HIV (human immunodeficiency virus, a retrovirus), even though for more than a quarter century these tests have been widely used to diagnose such infection. Manufacturers of the test kits do not claim that the tests detect infection.[1] The tests are for *antibodies*[2] and were approved only for screening blood, where sensitivity rather than specificity is the prime criterion and false positives are of relatively little concern. Technical discussion[3] of how to detect HIV infection makes plain that in themselves the tests are insufficient to diagnose infection. In point of fact, "positive" "HIV" tests may be the result of dozens of such conditions as hypergammaglobulinemia, tuberculosis, vaccination against influenza,[4] receipt of tetanus immune globulin,[5,6] or even pregnancy.[7-9]

The story behind these circumstances has a number of parts:

1. The initial claimed discovery or identification of a retroviral cause of AIDS;
2. The patented method for detecting antibodies claimed to be specific to HIV and constituting a claimed "HIV test";
3. Extrapolation of the claim that HIV tests detect antibodies to the presumption that a positive test signifies active infection; and
4. In absence of any gold standard test, use of the first (unproven, unvalidated) antibody test as the basis for supposed validation of all later tests. (A gold-standard test would have to be based on data from pure virions of HIV obtained from an HIV-positive individual.)

The mistaken equating of "HIV-positive" with "infected by HIV" has the gravest consequences: healthy individuals who happen at some time to test "HIV-positive" have suffered physical, psychological, or financial damage.

In the following, when citing early work that employed the nomenclature of human T-lymphotropic virus (HTLV-III) and lymphadenopathy-associated virus (LAV), those terms are used rather than the now-agreed term "HIV."

Announcement of an AIDS-causing Retrovirus

HTLV-III was the probable cause of acquired immunodeficiency syndrome (AIDS), announced the secretary of the Department of Health and Human Services (HHS) at a press conference on Apr 25, 1984, introducing the purported discoverer, Robert Gallo. Four later publications in *Science* revealed the basis for that claim.

Popovic et al.[10] described an immortalized T-cell line in which large quantities of HTLV-III could be obtained by co-culture with putatively infected T-cells. Retrovirus was said to be detected through the presence of reverse transcriptase (RT), and RT activity was also the criterion for choosing a sucrose density of 1.16 g/ml as the "band" in which retrovirus was said to be found under ultracentrifugation. However, RT activity is present in normal cells,[11,12] a fact that vitiates a central element in this work as well as in much subsequent research on HIV. Electron microscopy was claimed to show large amounts of "extracellular viral particles"; however, that could only have been demonstrated by isolating and purifying those particles and establishing that they were indeed virions, and this had not been done. The virus was also said to be detectable via antigen-antibody reaction, but this involved a tautology. As discussed below, "isolation is not isolation; purification is not purification."

It was further stated that HTLV-III proteins had been found in 85% of sera from AIDS patients and that HTLV-III is related to HTLV-I and -II, retroviruses previously discovered by Gallo. In contrast, the LAV described earlier by Barré-Sinoussi and Montagnier was not related to HTLV-I or -II, and antibodies to it were present in only 37.5% of sera from AIDS patients. Those claims were soon shown to be false. HTLV-III indeed was LAV, a sample of which Montagnier had sent to Gallo; and the 2009 Nobel Prize for discovering the virus went to Barré-Sinoussi and Montagnier, to the exclusion of Gallo. Numerous other flaws have been uncovered in the work reported from Gallo's laboratory, and it was owing only to a technicality that Gallo himself failed to be formally charged with scientific misconduct.[13]

In a companion article, Gallo et al.[14] claimed to have isolated HTLV-III from 26 of 72 AIDS patients, 18 of 21 individuals with pre-AIDS, 3 of 4 healthy mothers of children with AIDS, and 1 of 22 "normal male homosexual subjects." However, as an illness becomes more severe, one might expect to find more of the pathogenic agent at work rather than less, so it seems strange that "the primary cause of AIDS" could be found in only 36% of those suffering from the active disease when it could be found in 86% of those with pre-AIDS. Moreover, the symptoms of "pre-AIDS"

were fever and chronically swollen lymph nodes, seen in many illnesses and not specifically precursors to AIDS; those symptoms were presumed to be such precursors only when encountered in people belonging to the groups in which AIDS had appeared, gay men or intravenous drug abusers. Beyond that, the term "isolated" is seriously misleading, as shown below.

A third article published at the same time[15] again asserted that HTLV-III is "a true member of the HTLV family" yet "clearly distinguishable from HTLV-I and -II." There were somewhat confused claims concerning which antigens are associated with which of these retroviruses. Thus, p61 and p65 are "encoded by HTLV-I," yet often recognized by sera from AIDS patients. Antigens of HTLV-III are "similar in size" to those of other HTLVs but include three "serologically unrelated" groups: p55 and p24, which are "group-specific"; p65, which is "envelope-related"; and a third group "of unknown affiliation." In one place it is said that "antibodies to the structural proteins of HTLV, notably p24 and p19…are not detectable in most AIDS patients…." Yet later p24 is included among the "most prominent…antigens" of HTLV-III, namely p65, p60, p55, p41, and p24; less prominent antigens were said to be p88, p80, p39, p32, p28, and p21. Some cross-reaction of p65 with HTLV-I was acknowledged, as well as cross-reactions with nonspecific Gag-related antigens. Nevertheless, specificity was claimed for p65, p55, p41, p39, p32, and p24 as "newly expressed after viral infection"; but of course this does not preclude the possibility that these antigens might be found also in association with other agents than HTLV-III. It is pertinent that the immunological abnormalities seen in AIDS patients are also present in people suffering from, for instance, tuberculosis, diabetes, malaria, macroglobulinemia, aplastic anemia, or thalassemia, and that they can be induced by (for example) adrenalin, prednisone, or Epstein-Barr virus.[16]

The fourth article[17] reported antigens of HTLV-III to be reactive with sera of 88% of AIDS patients and 79% of gay men with pre-AIDS symptoms as well as with less than 1% of "heterosexual subjects." This time the major reactivity was said to be against p41, a presumed viral-envelope antigen. Note that HTLV-III antigens were reactive with 88% of AIDS sera even though HTLV-III had been found[14] in only 36% of such sera.

It has often been remarked that these papers fall far short of demonstrating that HTLV-III is a necessary and sufficient cause of AIDS; for example, "the evidence does not constitute proof of the isolation of a retrovirus, that the virus is exogenous or that the virus is causally related to AIDS."[18] Indeed, the Gallo opus actually represents a circular procedure:[19,20]antibodies in sera from AIDS and pre-AIDS subjects were shown to react with material taken from cultures infected with unidentified agents present in sera from AIDS and pre-AIDS subjects. The most serious deficiency is the failure to show that similar reactivity is not present in other physiological conditions or in other illnesses than AIDS, and later studies have indeed found many instances of such false-positive HIV tests.[4-9]

The Patent for Detecting the Virus

U.S. Patent 4,520,113, granted to Robert Gallo and co-workers Mikulas Popovic and Mangalasseril G. Sarngadharan, dated May 28, 1985, claims that "antigens associated with the infection of human cells by this virus are specifically recognized by antibodies from AIDS patients. Specifically, HTLV-III isolated from AIDS patients and transmitted by co-cultivation with an HT cell line is specifically detected by antibodies from human sera taken from AIDS patients."

The Test Is Nonspecific

This nascent test was hardly foolproof, since sera from only 88% of AIDS patients and from 79% of pre-AIDS gay men were positive, and since positive reactions were also seen in some (presumed uninfected) blood donors,[17] from the very beginning the test was anything but specific.

One reason may be the claim of p41 as the most characteristic and prominent HIV-specific antigen, and that p65, p60, p55, and p24 too were "not detected in normal sera." However, antibodies to p24 have been found in substantial proportions of patients with multiple sclerosis, T-cell lymphoma, and generalized warts.[21] Moreover, in about 15% of healthy blood donors, p24 was "the predominant band" on Western blot tests, and p41 has also been found in blood platelets of healthy individuals.[18] Far from being specific to HIV or AIDS patients, then, p24 and p41 are not even specific to illness.

Isolation Is Not Isolation; Purification Is Not Purification

In the patent documents as well as in the earlier articles in *Science*, and across the HIV/AIDS literature to date, "isolation" and "purification" do not have the meaning those terms convey in common parlance, namely that isolation means to extract the pertinent entity from its original setting (in this case, an HIV-positive individual) and purification means to remove all contaminants from the isolate in order to leave only the pertinent entity.

In HIV/AIDS parlance, by contrast, "isolation" and "purification" do not mean extracting and purifying HIV from an AIDS patient or from an HIV-positive individual. Rather, white blood cells from that individual are cultured together with immortalized T-cells from a line originally established by Gallo, using in addition such stimulants as phytohemaglutinin or IL-2 that are presumed to make the cultured cells express the putative retrovirus. The "isolation" is then said to have been made if (1) the culture displays RT activity—which, however, is now known not to be specific for retroviruses,[11,12] or (2) an extract from this culture is able to do what the original isolate did, i.e. to further "infect" other cultures. Such a procedure falls short of demonstrating that any virions were actually present in the original isolate from a human being: Any authentic virions present in the culture might have been generated by the culturing procedure. One cannot exclude that the culturing actually produced *ab initio* an entity that can generate a positive "HIV" test-response. Indeed, normal human genomes include some DNA sequences homologous with "HIV," and these may be expressed by culturing techniques like those used in HIV/AIDS research.[18]

"Purification" in HIV/AIDS parlance means that material thought to contain HIV is ultracentrifuged in a specified medium, and what sediments at a particular density is regarded as "HIV." In point of fact, published electron micrographs of such "purified" "isolates" show a motley mixture of cellular debris. It clearly does not contain pure virions, and indeed there is no proof that it contains any virions at all.[22,23]

Gallo has even asserted that there is no need to purify HIV since his method of culturing produces so much of it that it does not matter what else might be present:

> You have to purify…. [A] retrovirus comes out of [cellular] membrane. In so doing, it incorporates some cellular proteins in the virus…. [B]y putting it through a sucrose gradient it would do hardly anything when you have very little virus. So the ratio of cellular material to virus, I

don't want to say this is an accurate number but I will give an example. Let's say it would be a thousand to one but when we succeeded in mass producing the virus in a continuous culture, you have got an enormous purification far beyond the sucrose gradient alone because you are now producing loads of virus with little amounts of cell.[24] ["Cellular membrane" is a correction for what was at first mistranscribed as "chromosome membrane."]

This claim, that a high ratio of putative virus to cellular debris is as good as or better than pure virus, cannot stand. The presence of even tiny amounts of some unknown, undetected, active agent can confound experiments. Impurities present in amounts too small to be detected directly can nevertheless produce measurable effects if those effects are of a catalytic or enzymatic nature.

Discarded Assertions

A number of assertions in the patent have since been quietly forgotten, for example that there is a certain cross-reactivity with Gallo's HTLV-I and HTLV-II. Gallo had earlier reported isolation of HTLV-I from an AIDS patient[25] and described HTLV-I and HTLV-II as "*the only known specific cofactors* for AIDS" [emphasis in the original].[26]

The patent assigns the main specificity of the test to p41, with some reactivity also to p65, p60, p55, and p24. But the contemporary criteria for Western blot include p160, p120, p68, p55, p53, p41, p39, p32, p24, p18; only three of the five antigens said by Gallo to be specific for HIV are among the 10 now supposedly specific for HIV, and there are seven others as well. Even worse, there is currently no agreement over which combination of these is supposed to be specific for HIV.[27] For example, in Africa any two of p160, p120, or p41 suffice to constitute a "positive." On the other hand, a positive test in Australia, Germany, or Britain requires any *one* of those together with, in Australia any three of Pol- (p68, p53, p32) or Gag-related antibodies (p55, p39, p24, p18), in Germany any *one* Pol- or Gag-related antibody, or in Britain specifically p32 and p24. In France, by contrast, *all three* of p160, p120, p41 are required together with any one Pol plus any one Gag. No fewer than five different criteria have been used by different groups in the United States.

Arbitrary Criterion for "Positive"

The enzyme-linked immunosorbent assay (ELISA), the primary antibody test, measures a color intensity. No controls are perfectly colorless, however. The only objective way to identify a color intensity that would correspond to guaranteed complete absence of purported HIV antibodies would be to have samples from controls known not to have been exposed to HIV, which is an impossibility. As the best practical but admittedly imperfect approach, repeat blood donors are used as controls.[3]

The patent's Example 1 reports that "absorbance readings greater than three times the average of 4 normal negative control readings were taken as positive." Under that criterion, 88 percent of AIDS patients, 79 percent of pre-AIDS individuals, 60 percent of intravenous drug abusers, and 27 percent of gay men tested positive; 0.5% of controls also tested positive. That is hardly foolproof, specific detection of whatever might be uniquely characteristic of AIDS. It does not exclude that one of the "normal controls" may have harbored small amounts of HIV antibodies, nor does it exclude that absorbance readings three times greater than the average "normal control" might be produced by cross reactions.

In other ways, too, the patent is less than impressive. Example 1 and Example 4 both refer to the data in Table 1 and describe the same procedure, though in somewhat different words; why they are given as distinct separate examples is puzzling. In a single paragraph, Example 5 asserts a finding of specificity without stating how many experiments were carried out.

Not an HIV Test

In effect, the manner in which this test was developed makes it at best an AIDS and pre-AIDS test, not an HIV test—one which is even more sensitive to and specific for pre-AIDS than for AIDS. Furthermore, since the symptoms of pre-AIDS—swollen lymph nodes and fever—are seen also in many other illnesses, the test is evidently a nonspecific illness test. Patients with many illnesses may react "HIVpositive." After the test had been in use for some time, moreover, it turned out that it could read "positive" for conditions that are not even illnesses, such as vaccinations or pregnancy.[4-9]

Antibodies as Denoting Infection

The scientific publications and the patent claiming HTLV-III to be the probable cause of AIDS were clearly insufficient to establish that claim. How then did it come about that a less-than-specific antibody test became a basis for asserting active infection by HIV?

Rodney Richards, who worked on the development of the first ELISA HIV test (marketed by Abbott Laboratories), has provided a detailed chronology showing how this unprecedented equating of antibodies with active infection came about.[28] The story would be literally incredible were it not fully documented by the authoritative material in the public domain cited by Richards.

Initially, in 1984, the Centers for Disease Control and Prevention (CDC) had quite properly acknowledged the possibility of false-positive antibody tests owing to "an antigenically related virus or nonspecific test factors." In people at high risk of AIDS, it "probably" meant prior exposure; however, "[w]hether the person is currently infected or immune is not known" and "the frequency of virus in antibody-positive persons is yet to be determined."[29]

Six months later, CDC admitted that there would be a high proportion of false positives when screening low-risk populations: No one should be informed of testing positive before the test had been duplicated.[30]

Three months beyond that, the Food and Drug Administration approved Abbott's ELISA test *for blood screening*. Obviously it makes sense to take all possible precautions against the presence of a possible pathogen in blood that is to be used for transfusions: better to discard 100 donations of good blood than to allow one infected sample to be transfused. It is very different, however, to inform someone on the basis of a highly unreliable test of an infection with a deadly pathogen for which there is no cure. The package insert with the Abbott test had the appropriate caveats: "There is no recognized standard for establishing the presence or absence of HIV-1 antibody in human blood…. The risk of an asymptomatic person with a repeatedly reactive serum sample developing AIDS or an AIDS-related condition is not known." The same caveats apply to the Western blot antibody-test approved in 1987 and widely used as supposed confirmation of duplicate positive ELISA tests.[28,p339] Similar disclaimers from various manufacturers are found in more recent test-kit inserts.[1]

Some months after the Abbott test had been approved for blood screening, data from blood donors revealed that 44% of samples strongly positive for "HIV" antibody contained no virus detectable by culture. Similarly, 40% of gay men testing antibody-positive had

no detectable virus.[31] In nearly half of all cases, then, both in a high-risk group and in a low-risk group, a positive "HIV" test occurred in the absence of HIV.

Then CDC stood this evidence on its head. The data showed that absence of virus accompanied antibody positivity in almost half of all cases. Looking instead at the other half, CDC asserted: "Since a large proportion of seropositive asymptomatic persons have been shown to be viremic, (5) all seropositive individuals, whether symptomatic or not, must be presumed capable of transmitting this infection."[32] [Reference (5)[30] conceded that it was not known what proportion of seropositive donors was actually infected].

Perhaps CDC was so concerned about preventing transmission of HIV that it used the same reasoning as with blood screening: better that some large number of actually non-infected people be warned against passing on a possibly fatal infection than that a small number of infected persons unwittingly infect others. But that ignores the devastating psychological effect on the many uninfected people who were thereby doomed to believe that they were harboring an incurable, inevitably fatal infection.

CDC then went even further than "presuming" that seropositive might mean infection. In 1986, in an article in the *Journal of the American Medical Association*—which is much more widely read than the *Mortality and Morbidity Weekly Reports* in which the *presumption* had been stated—CDC researchers *defined* seropositive as *equivalent to infection.*[33] As Richards points out,[28] all the cited data reported that virus could not be cultured from a high proportion of seropositive individuals. The CDC was now dismissing this evidence from culture by asserting that seropositive equals infection, even though culture is supposed to be a *direct* demonstration of infection whereas seropositivity can only be an indirect indication of possible infection.

CDC has continued to dispense with all caveats, asserting that "presence of antibody indicates current infection"[34] not as a precautionary measure in screening blood, not as a precautionary measure to prevent transmission, not when the seropositive individuals are in a high-risk rather than low-risk population, and irrespective of whether they are symptomatic or asymptomatic. It is asserted dogmatically without any exception that seropositive means infected. Surely this constitutes public-health malpractice based on junk science.

Furthermore, Richards[28] points out that CDC was actually transgressing its mission to safeguard public health by this assertion, which dictates what doctors should do on the basis of a particular test, even in the face of what the Food and Drug Administration, charged with protecting consumers, had said just a few months earlier: "The significance of antibodies in an asymptomatic individual is not known."[35]

No Gold Standard for HIV Tests

To this point, I have only reviewed work from the 1980s. Were the deficiencies later corrected?

Unequivocally and simply, "No."

For one thing, all later work built on and presumed the soundness of the seminal articles. For another, it remains understood by the actual experts that a positive "HIV" test does not in itself signify infection, be the test an ELISA, a Western blot, a "viral load" measurement, or a culture; but this understanding is not broadcast outside the technical literature.

The following is taken from Weiss and Cowan's chapter in the fourth edition of a standard textbook,[3] which can be regarded as authoritative on several grounds: Weiss has worked in this field since the beginning, having published since 1984, including with Gallo; and the book has been accepted and favorably reviewed, e.g. the 3rd edition in *JAMA*[36] and the 4th (2004 edition) in *Clinical Infectious Diseases*[37] and *JAMA*.[38]

In terms of substance, no more need be said than that "[i]n the absence of gold standards, the true sensitivity and specificity for the detection of HIV antibodies remain somewhat imprecise" (p. 150).

Obviously enough, "somewhat imprecise" is a euphemism: The lack of gold standard makes everything highly doubtful. Pure virions of HIV, the sine qua non for establishing a veritable gold standard, have never been obtained from an AIDS patient or from an "HIV-positive" individual. Twenty-five years of HIV/AIDS research have not brought valid HIV tests.

Weiss and Cowan make clear that inferring HIV infection is a matter of probability, not certainty, and that the assessment of probability requires interplay of laboratory testing with clinical information, very much including individual medical history and risk-category classification. They write:

> A *pre*-test probability assessment is required whenever test results are to be meaningfully interpreted [p 149; emphasis in original].
>
> An essential part of the testing process takes place even before testing is done; that is, the estimation of the probability of infection (the "pre-test" probability). This is necessary in order to interpret a test result appropriately, whatever the purpose—whether it is clinical, counseling or research—and can dramatically impact the predictive value after testing (or "post-test" probability) [p 159]. No test, per se, should be the basis for diagnosis on its own, but rather a test is merely an aid in correct diagnosis. The practitioner must use test results in the context of a clinical picture to reach an accurate diagnosis [p 172].[3]

The import of these uncertainties is illustrated in a table[3, p149] showing that in low-risk populations (prevalence of "HIV" 0.1%), a "positive" "HIV" test result has only about one chance in six of being a "true" positive; five out of six would be false positives. Conversely, at a prevalence of 99.9%, a negative test result would have only about one chance in six of truly being negative—once an individual has been designated "high risk," even a negative "HIV" test may not be accepted as definitively showing lack of infection, and further testing is recommended.

This illustration is based on a hypothetical test that is 99.5% sensitive and 99.5% specific, but the principal point is independent of the actual numbers—and no test, of course, is 100% sensitive or specific. Thus the initial belief that a person is high- or low-risk biases interpretation of the laboratory tests toward becoming self-fulfilling prophecies.

Conclusions

There is no gold standard for HIV tests. Current practice is to take positive tests as proof of active infection even though the antibody tests have not been shown to be specific for HIV antibodies, and even the presence of HIV antibodies has not been proved to signify active infection by HIV rather than past exposure and acquired immunity. As a result, healthy people may be doomed, without justification, to lifelong administration of toxic drugs. This applies particularly to people in groups traditionally regarded as "high risk." Outside Africa, that comprises gay men and injecting drug users, but tuberculosis patients and pregnant women should also be included since positive tests are so common among them. In recent years, black people in the United States have come to be

regarded as being at high risk because they persistently test positive at higher rates than other groups, in all economic and social sectors. Black women are particularly at risk because pregnancy conduces to false positives and HIV testing in pregnancy is mandatory in some jurisdictions. The risk of iatrogenic damage inflicted through improper applications of HIV tests must be considered.

Henry H. Bauer, Ph.D., is professor emeritus of chemistry and science studies and dean emeritus of arts and sciences at Virginia Polytechnic Institute and State University (Virginia Tech). Address: 1306 Highland Circle, Blacksburg VA 24060-5623. E-mail: hhbauer@vt.edu.

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Potential conflict of interest: I am the author of a book, *The Origins, Persistence and Failings of HIV/AIDS Theory*, which claims to show that HIV is not the cause of AIDS.

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