WHY THE "AIDS TEST" DOESN'T WORK
IN AFRICA

by Christine Johnson

Last summer’s events at the 13th International AIDS conference in Durban, and South African President Thabo Mbeki’s refusal to adhere to the "conventional wisdom" on AIDS, have made it more crucial to reevaluate all aspects of AIDS in Africa.

It is widely believed that Africa is being devastated by a plague of "AIDS." This is in spite of the fact that, according to the World Health Organization’s (WHO) Weekly Epidemiological Record, 19 years’ worth of AIDS cases for the entire continent of Africa has amounted to only 876,009. (In the US, more people than this die in one year of heart disease.) Africa is generally blamed as the origin of AIDS, yet statistics point towards a more likely source of this disease: the United States.

It was not until 1997 that the cumulative number of AIDS cases in Africa surpassed those in the United States. The most current statistics (November 2000) show that the cumulative tally stands at Africa 876,009 and the United States 733,374—not much of a difference considering WHO’s estimate that 25.3 million Sub-Saharan Africans have HIV/AIDS, whereas in the United States it is well below one million. Why is there this huge discrepancy? The main reason is many Africans test positive on HIV antibody tests—while very few Americans do—and few HIV-positive people in any country go on to develop AIDS.

Researchers originally targeted Africa as the source of AIDS for three rather feeble reasons:[21]

1. Robert Gallo’s discredited theory that AIDS was caused by HTLV-1, another retrovirus similar to HIV, and thought to be endemic in Africa;
2. the prevalence of Kaposi’s sarcoma in Africa (even though Kaposi’s sarcoma was a new disease in American gay men, it had existed in Africa since ancient times, and hence could not indicate a brand-new disease there); and
3. a small number of AIDS patients of African origin who were living in Europe.

When researchers began taking HIV antibody test kits to Africa around 1985, they immediately found verification of the above ideas. Small groups of Africans were tested and found to be positive on these tests, and these numbers were extrapolated to the entire continent. On this basis, and although only a few thousand AIDS cases had been reported in Africa at that time, the WHO immediately began estimating that millions of Africans were infected with HIV and that Africa would have to contend with an imminent plague.

In the mid-80s when HIV antibody tests first became available, it became apparent that there were problems associated with using these tests in the African population.[1-5] In 1985, Hunsmann found that positive HIV (then called HTLV-III) ELISA tests had a low frequency of confirmation using a different type of antibody test, the immunoprecipitation method. This led him to question the specificity of ELISA in African blood samples.[1]

Biggar found correlations between positive HIV antibody tests and age and poverty.[2] He also found correlations with malaria and parasitic diseases in Africans (but not in Asians or South Americans). Labius Mutanda of the Ugandan Public Health Service and guest lecturer at St. Louis University (US) in 1991 reported that "existing ELISA and Western Blot assays may not always be able to reliably ascertain HIV infection in many African individuals."[3] Mutanda told me that his experience with both ELISA and Western Blot in Uganda was that often an individual could be positive if tested with the test kit from one manufacturer and negative if tested with the kit of a different manufacturer.

Serious questions have arisen as to whether HIV antibody tests are specific in any population,[6] although mainstream AIDS researchers still believe they are accurate, and considerations of test failure in Africa have never prevented the tests from being used there for many purposes including estimating HIV infections. Mulder in 1994 demonstrated that HIV-positive Africans died at a much greater rate than HIV-negative Africans, and offered this as definitive proof that HIV causes AIDS.[7] In reality, the only thing Mulder proved was the utility of HIV antibody tests when employed as generalized indicators that something is wrong, i.e., they can be used as surrogate markers of AIDS risk.

The ELISA test contains a mixture of broken-up HIV proteins called a "whole viral lysate." In theory, if a person’s blood contains any HIV antibodies, the ELISA will react. The Western Blot is more sophisticated (and much more expensive). The HIV proteins are separated into bands on a strip. That way, if any antibodies cause a reaction, it can be determined exactly which HIV protein they are reacting to. The most important HIV proteins are p24, p32, gp41, gp120, and gp160.

In the US, ELISA is considered to be very inaccurate, and no diagnosis of HIV infection is made without a Western Blot (considered to be more accurate) as confirmation. Interestingly, in the UK, just the opposite is true and Western Blots are considered to be inaccurate!

For the most part, Africans aren’t tested. It’s simply too expensive. But when they are tested, the ELISA is used. HIV ELISAs are not accurate enough to diagnose an American with HIV infection, but
they are accurate enough for Africans!

To compound the problem in Africa, AIDS in Africa is diagnosed not with antibody tests but rather on the basis of clinical symptoms. This is called a “clinical case definition,” and was originally developed by WHO in 1985. It consists mainly of persistent fever, diarrhea, and weight loss. These symptoms are identical to many common African diseases. Only in Africa can you be diagnosed with AIDS on the basis of these symptoms alone.

To make matters worse, individual countries have felt free to develop their own clinical case definitions. Thus, there is no consistency between countries as to exactly what constitutes an AIDS case, and some of these clinical case definitions are extremely broad, making it easy to classify almost anything as AIDS.[34] Often new cases are registered which don’t fulfill even these extremely lax criteria.[34]

**Antigen/antibody reactions are nonspecific**

My search of the scientific literature on HIV antibody testing produced references to approximately 70 diseases or conditions that can possibly cause false-positive reactions on HIV ELISAs and/or Western Blots.[28] Many of the conditions are quite prevalent in Africa. These include tuberculosis, malaria, leprosy, Q-fever, tape-worms or other parasites, and leishmaniasis.

In order for these tests to work properly, it must be true that a protein (also called an antigen) will react only with the antibody that matches it. In reality, antigen/antibody reactions are nonspecific. Antibodies cross-react with antigens other than the ones that originally elicited them. Scientists routinely ignore this well-known phenomenon when it comes to HIV antibody tests.

This wide range of naturally occurring cross-reactivity does not in itself invalidate HIV antibody tests, or any antibody test. However, it does demand, as an absolute requirement, verification by an independent gold standard. The accuracy of any antibody test must be ascertained by determining that all people with positive antibody tests have the microbe in question isolated from their blood, and conversely that all people who are negative have no microbe isolated from their blood. The fatal flaw in HIV antibody testing is that virus isolation has never been used as a gold standard, and it is the only proper gold standard.[6] Without virus isolation, no one knows what antibodies are causing the reaction when the test comes back positive.

The problems of antibody/antigen cross-reactivity are compounded in relationship to the infectious disease burden of the person being tested. The more varying antibodies a person carries, the more likely that person is to possess some type of antibody that will cross-react on HIV antibody tests. Many Africans, exposed to a variety of diseases, tend to carry a multitude of antibodies. In this regard they can be compared to certain members of the recognized AIDS risk groups in the West (but not the general population of Westerners). The general rule is: The more diseases/microbes/foreign proteins, the more antibodies, and thus the more likely an HIV antibody test will be positive.

Test kit manufacturers “verify” the specificity of their tests (specificity is a measure of how often false-positives will occur) by testing several thousand random blood donors (by definition at low risk for AIDS or HIV infection), with 20 or 30 subjects thrown in who represent several of the more commonly recognized cross-reacting conditions such as rheumatoid arthritis or systemic lupus erythematosus. The other known cross-reacting factors[8] more prevalent in Africa are not added to the equation.

This practice of omitting Africans from the test sample (either healthy Africans or those with similar non-AIDS conditions that might elicit cross-reactions) results in a picture of test accuracy that fits only the type of population in the test sample. This creates severe bias and overestimates test specificity.[9] Constantine stated, “Test parameters thus obtained with this sort of a biased sample cannot be validly extrapolated to assess a test’s performance in different diagnostic situations.”[10] In other words, an HIV antibody test kit developed in the West will yield different results in Westerners and Africans.

ELISAs with estimated specificities in the high 90s have been used in Africa, with very poor results, for exactly this reason. Constantine reports "unsatisfactory test performance has been described in studies with east African serum from Tanzania and Egypt.”[10] Indeed, the specificity of one test dropped to an abysmal low of 51% when used in Africa.[11] (The way the math works out, even a specificity of 99% would produce extremely high numbers of false positives,[29] so you can imagine how inaccurate a specificity of 51% would be!).

Confounding this is the widely-acknowledged propensity of antibodies to one retrovirus to cross-react with the antigens of another retroviruses. [12,13] Gallo and his colleagues have repeatedly stated that the p24 of HIV and of two other human retroviruses, HTLV-I and HTLV-II, which Gallo claims to have isolated from humans, immunologically cross-react.[14] Since HTLV-1 is endemic in sub-Saharan Africa,[1] many people infected with HTLV-1 may be misdiagnosed as being HIV infected.

**Are the new "third generation" test kits any better?**

The World Health Organization (WHO) addressed this problem by providing local clinics test kits that use genetically engineered HIV antigens called recombinant antigens (as opposed to the usual whole viral lysate antigens which contained many cross-reacting contaminants). Local lab technicians were trained to use these tests properly. Gordon Stewart, a British epidemiologist (and member of RA’s editorial board) who had visited Kenya, described such a clinic to me. However, several years ago Panafriica News Agency correspondent Eliezer Wangulu described another part of Kenya where "most health facilities have dysfunctional laboratories that have also run out of reagents."

There are many centers where testing is performed by trained staff using recombinant antigen for ELISA tests. Western Blot is used by some, but certainly not even the majority, of centers as a confirmatory test. Stewart told me, however, he suspected that "much of the testing in Africa is done with miscellaneous test kits, unsupervised and unvalidated."

Proper performance and interpretation of Western Blots requires a high degree of expertise. Lab proficiency is highly variable and sometimes completely unacceptable.[30] Even reference labs, the highest quality labs in the United States, have quality control issues,[31] and it can be expected that the specificity of any test kit will deteriorate by an order of magnitude or more in less experienced labs, where most of the testing is done.[11,32] So one must wonder how excellent quality control could exist in Africa, where health care budgets are often minuscule, and lab experience is much less in comparison. In addition, the chaos of civil unrest and warfare in many countries has a profound effect on health care budgets and the abil-
ty to organize proper health care resources.

Whether or not a significant portion of African populations has access to properly run and equipped labs and testing programs, the use of recombinant antigen test kits will not solve the problem. It is claimed that these "third generation" test kit antigens are "purified" to the extent that unwanted cross-reactions, and thus false-positives, will not occur. However, recombinant antigens are derived from E. coli and may contain additional bacterial epitopes, and in test sera from some individuals with antibacterial antibodies, false positives occur as a result of the interaction of these antibodies with the antigens of the enteric bacilli.[15,16]

Other false positives can occur for reasons unique to recombinant technology, e.g., immunoreactive epitopes may rely on either primary amino acid sequence or conformational shape for antigenicity and therefore, nonspecific reactivity may result if similar epitopes exist on different viruses, such as the common flu virus.[15] The fact that other microbes share epitopes with HIV is amply documented.[17] Test systems based on recombinant HIV antigens have yielded positive results much more often than those based on whole viral lysate due to cross-reaction with antigens of enteric bacilli.[16] A study of two groups of random blood donors, which should have yielded similar results, showed positives to occur more than twice as frequently in the group tested with recombinant-antigen-based tests (617/119,004) as in the group tested with lysate-antigen-based tests (246/119,178).[18]

Another study was done in the former USSR to determine the positive predictive value (how often a positive test result indicates a true infection) of various confirmatory tests. This study yielded the following results in AIDS high-risk groups:[19]

Whole viral lysate antigens: 99.4% specificity
Recombinant peptide antigens: 95.1% specificity
Synthetic peptide antigens: 86.1% specificity

As mentioned above, a specificity of 95% indicates an extremely inaccurate test in terms of potential false-positives.

Yet another study demonstrated cross-reactions between the sera of people with autoimmune disorders (for example, systemic lupus erythematosus and Sjogren’s syndrome) and HIV synthetic peptides or recombinant gp120, gp41, and p24 proteins.[17] The purity of the antigens is really not the issue. Regardless of the source of antigens, all serological tests are subject to nonspecific and unpredictable reactivity.[15] It does not matter whether the HIV antigens are “natural or engineered, or even derived from HIV itself (e.g., a serological test for infectious mononucleosis employs sheep red blood cells).”[20] What does matter is whether the reactions of patients’ sera with these antigens are shown to be specific for the presence of HIV in vivo. A fundamental principle of antibody testing is that “for a test to be valid, regardless of time of development, generation, or appellation, its specificity must be authenticated by the use of an independent gold standard.”[20]

**Mycobacteria can cause false-positive HIV antibody tests**

In 1994, Essex found significant levels of false-positive reactions on both ELISA and Western Blot in people with leprosy, a disease associated with Mycobacterium leprae infection.[5] Antibodies to the carbohydrate structures found in the mycobacterial cell wall, lipoarabinomannan (LAM) and phenolic glycolipid (PGL), were noted to “[yield] significant cross-reactivities with the HIV-1 pol [p31] and gag [p24] proteins.” Essex stated that the “data suggest that mycobacterial cell wall antigens may share common epitopes with HIV” and warned that “ELISA and Western Blot may not be sufficient for HIV diagnosis in AIDS-endemic areas of Central Africa where the prevalence of mycobacterial diseases is quite high.”

These carbohydrate-containing antigens are also present in other mycobacteria, in particular Mycobacterium tuberculosis. It is particularly significant to note:

1. Of the 661 million people in sub-Saharan Africa, 2-3 million have active TB with an annual mortality of 790,000.[21]
2. TB has now become an AIDS-defining illness, and 30-50% of African “AIDS” deaths are from TB;[21]
3. “HIV infection” as defined by a positive HIV antibody test does not precede TB infection but rather follows it;[21]
4. In a tuberculosis sanatorium in Kinshasa, Zaire, half of the suspected pulmonary cases, one-third of the confirmed cases and two-thirds of the confirmed extra-pulmonary cases had a positive HIV Western blot test.[21,22]

The presumption is that HIV infection leads to tuberculosis as an AIDS indicator disease. But from the above data it is more reasonable to conclude the opposite—that tuberculosis causes false-positive HIV seropositivity, without HIV infection being present.

**Anti-carbohydrate antibodies cross-react with HIV proteins**

It has been recognized since 1980 that naturally-occurring anti-carbohydrate antibodies cross-react with retroviral proteins.[23] Healy speculated that false-positive HIV Western blot gp41 bands were actually due to anti-carbohydrate antibodies, since gp41 and non-viral proteins share similar antigenic structures.[24] Tomiyama stated that “normal human serum contains antibodies capable of recognizing the carbohydrate moieties of the HIV envelope glycoproteins (gp41, gp120 and gp160).”[25] This is of particular significance when one realizes that African criteria for reading HIV Western blots allow a positive diagnosis based on two envelope bands alone.

Eleopulos states “Not only mycobacteria (M. leprae, M. tuberculosis, M. avium-intracellulare) but also the walls of all fungi (Candida albicans, Cryptococcus neoformans, Coccidioides immitis, Histoplasma capsulatum including Pneumocystis carinii), contain carbohydrate (mannans). One hundred per cent of AIDS patients (even those with ‘No candida clinically’) have Candida albicans antibodies...Since antibodies to mannans react with the ‘HIV proteins’ then, as Essex and his colleagues have pointed out for mycobacterial infection in Africa, one would expect the sera of all people infected with fungi and mycobacteria to cross-react with the “HIV-1 glycoproteins as well as to cause ‘significant cross-reactivities with HIV-1 pol and gag proteins.’”[17]

The vast majority of opportunistic infections experienced by AIDS patients in the West are due to PCP, candidiasis, cryptococcosis, coccidioidomycosis, histoplasmosis, tuberculosis or Mycobacterium avium-intracellulare disease (88% of AIDS cases diagnosed between 1988 and 1992 had one or more fungal or mycobacterial infections).[17] At the very least tuberculosis and histoplasmosis[26] are endemic in many parts of Africa, and if AIDS
in Africa and AIDS in the West are the same disease, it can be presumed that many African AIDS patients will be infected with the above organisms.[26]

**Estimates of HIV infections in Africa have no scientific basis**

In spite of the facts, the myth persists that Africa is suffering a catastrophic AIDS epidemic. Last year *Newsweek* joined the incessant proclamations that Africa is being ravaged by AIDS, citing “2.2 million [AIDS deaths] in 1998 alone.”[27] One should be astounded at this figure, given that only 876,009 actual cases have been reported in 19 years. However, *Newsweek* was merely doing its duty by repeating the estimates promulgated by WHO.[33]

According to Stewart, WHO bases its estimates on the numbers of both positive tests and of AIDS cases reported by member states, “accepted at face value and, with rare exceptions, unvalidated.” Estimates are extrapolated from these data using flawed mathematical models.

Christian Fiala, an Austrian doctor who has extensively researched the global epidemiological data on HIV and AIDS, states that WHO produces its estimates by multiplying reported cases by a certain factor (on the reasonable assumption that actual cases are more than reported cases). However, this multiplication factor arbitrarily increases every year. In 1996 it was 12; only a year and a half later it had increased to 38![33] Fiala states: “The well-known horror scenarios about an epidemic of a new infectious disease exist exclusively in the heads of the statisticians through untenable and escalating multiplications.”[33]

**Conclusion**

The huge, alleged AIDS epidemic in Africa is based on several factors which have no scientific basis: 1) WHO’s faulty estimates, 2) the nonspecific criteria of AIDS, and 3) grossly inaccurate HIV antibody tests which are not applicable in Africa.

While AIDS authorities proclaim that 25.3 million Africans are doomed to die, in reality, no one knows if a single one of them is infected with HIV.

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