correlation. But there are more sinister forces at work here. The use of the term "HIV disease" is an effective way of obscuring the fact that "AIDS" today is as ephemeral and difficult to isolate as the retrovirus itself. In the early 1980s, AIDS consisted of only five diseases, Kaposi's sarcoma (KS), *Pneumocystis carinii* pneumonia (PCP), candidiasis, cytomegalovirus, and "gay bowel syndrome." There was also a state referred to as pre-AIDS or "AIDS-related complex," consisting of various systemic abnormalities including weight loss and persistent lymphadenopathy (swelling of the lymph nodes). Despite the fact that KS and PCP have absolutely nothing in common other than being linked by their appearance in a particular segment of society, at least AIDS had a somewhat consistent clinical presentation.

Not only has any specific clinical presentation for AIDS become impossible thanks to the list of twenty-five to thirty, depending on where one lives, AIDS-defining conditions, many of which have absolutely nothing to do with one another or with immune deficiency at all, but the existence of a particular clinical picture that we can call "AIDS" has become confounded by a number of factors.

First, patients are living longer than ever expected. There are people alive and well today who were diagnosed not only HIV-positive but also as having AIDS itself back in 1984. Popular consensus would say that the increased life expectancy is completely attributable to the antiviral drugs. This is negated by the fact that many of those so diagnosed have either not taken antiviral drugs, or have taken them very briefly. There is another item to consider, however, and that is the fact that dosages of drugs given today are far lower than in the days of AZT monotherapy. Consequently, people who would never have developed AIDS in the first place—if they had not been coerced into starting antiviral therapy—are simply developing illnesses more slowly than they would have under AZT monotherapy or aggressive HAART.

AIDS is looking less and less like a disease or even a syndrome at all, as all uncomfortable contradictions are swept under the rug, and "HIV disease" has become a name for some combination of the results of three blood tests—antibody, CD4+, and viral load—often in the presence of no disease at all.

*Five*

## PROBLEMS WITH THE HIV TESTS

BY NOW, MANY members of my generation, including me, have by now had an "AIDS test." But what exactly is an AIDS test? We already know AIDS isn't a disease, so what are we testing for?

The easy answer is: antibodies to HIV. Everyone knows that. A positive result indicates you were exposed to HIV at one time, developed antibodies to it, and surely the virus is hiding in your body somewhere—because everyone knows that HIV antibodies are *not* protective, quite the opposite: are a sure sign of imminent death and doom. Brave new viruses follow brave new rules, evidently.[7]

It may come as a surprise that no HIV antibody test has been approved by the FDA to diagnose HIV infection *on its own.* Each test must be tested against or used in combination with another unvalidated test, and depending on where you live, it takes a magic combination ranging from three, two, one, or no positive result(s) on three, two, or one unvalidated test(s), to be "confirmed" HIV-positive.

It is also relevant to note that the HIV antibody tests were *never* originally intended as diagnostic tools, but rather as screening tests to guarantee the safety of the blood supply.

The implications of this are so far-reaching as to be, to my mind, absolutely scandalous. Even if we throw away the causation issue, even

if we assume for the sake of argument that HIV absolutely does cause AIDS, the fact remains that the HIV antibody tests have been used as a weapon of discrimination ever since testing began. I can think of no medical test that is used the way the HIV antibody test is used.

Ignoring the fact that no medical test should be used to discriminate against anyone, ever, this situation becomes far worse when one considers that the tests being used in this way are some of the worst tests ever manufactured in terms of standardization, specificity, and reproducibility.

Media advertisements—particularly on music video channels such as MTV, VH1, and BET popular among preteens, teens, and young adults—have long advocated the concept that "everyone is at risk" and that we should *all* get an HIV test. We've probably all heard the slogan "knowing is beautiful," which leads to the question: knowing what, exactly?

The push for mass HIV testing appears to be reaching a fever pitch lately, possibly due to the fact that the general public seems to sense that we are *not* all at risk—a conception that AIDS advocates, for reasons which may be entirely altruistic but which are equally likely to be sinister or at best self-serving, believe needs to be changed. A recent campaign by the shoe manufacturer Aldo featured well-known entertainers such as Christina Aguilera and Charlize Theron urging "AIDS awareness and testing"—as though we are not already aware of AIDS, after twenty years of mass media campaigns. Furthermore, the shoe designer Kenneth Cole, recently designated chairman of the board of the American Foundation for AIDS Research (AmFAR), has launched a campaign recently that states, bluntly and absurdly, "We all have AIDS."

With such alarm bells being sounded throughout the mainstream media, it is no wonder that at this time, nearly half of all adults have had at least one HIV test (Bauer 2005). This test is accompanied by significant anxiety on the part of the person submitting to it, made worse by the fact that one has to wait on tenterhooks for the results to come back, sometimes as long as two weeks. It might seem reasonable for a person to be curious about what, exactly, the test is actually testing *for*, given the stigma associated with a positive result (or even with the fact that one "had to" get tested) and the supposed death sentence associated with this result.

It might seem reasonable to be curious—and it is curious indeed that most people never ask the question.

We assume, based on what we've been told for years by television, newspapers, politicians, and celebrity activists, that this test is measuring the presence or absence of a virus that will eventually kill you in a very nasty manner indeed. No wonder the testing campaign seems at times like a campaign of terror.

When you look at the medical literature and at the documentation provided by the test manufacturers themselves, though, you find out something quite different than what you had first imagined.

Even more shocking than the disclaimers placed in all test kits asserting their lack of validation and *lack of FDA approval to diagnose HIV infection* is that patient serum (blood) must be diluted by a factor of fifty to four hundred times before it is tested for HIV antibodies (Giraldo 1998, Kremer 1998).

The two major test kits routinely used for HIV diagnosis are the enzyme-linked immunosorbent assay (ELISA) test and the Western Blot (WB) test. The ELISA is run first, as a "screening" tool, and was first approved on the basis that it would be helpful in screening donated blood for HIV antibodies. Depending where you live, if your first ELISA is reactive (what we call "positive," a label that we shall soon see is quite misleading), you may get a second ELISA. If this ELISA is also reactive, you are tested with a different test, the WB. This is the final "confirmatory" test for HIV infection. It is extremely important to realize that these tests are *all* antibody tests, and they are all used to detect the presence or absence of certain "HIV-specific" antibodies.

Why is this so important? Remember, we're testing for antibodies here. In most cases, antibody tests are used to determine *prior* infection, because the pathogen itself is long gone. In certain cases, such as herpes and syphilis, there is concern about latent infections possibly becoming reactivated some time after the production of antibodies,[8] and so an antibody test is a reasonable measure to take. Antibody tests are done in general because they are cheaper and easier to do than to directly test for viruses or bacteria. However, in all of these cases, the antibody tests have been rigorously verified against the gold standard of microbial isolation—that is, the microbe was isolated in pure form and determined to consistently and specifically generate exactly those antibodies being tested for.

Of course, antibody tests all have a certain degree of nonspecificity due to the fact that certain proteins do cross-react. Some false positives occur with all antibody tests, but the rate of false positives for HIV is a particularly outrageous example of this phenomenon. Most of this is no doubt due to the fact that the tests are not verified against viral isolation, but part of the fault lies with the fact that the proteins contained in the test kit are not specific to HIV.

The reason that the HIV tests can never be used to diagnose true infection with an exogenous retrovirus is the same reason there is a reasonable correlation between testing HIV-positive and the risk of developing AIDS (and this risk is magnified in the high-prevalence groups). In the early days of AIDS, when the antibody tests were being developed, it was not possible to actually isolate HIV particles and prove the presence of those particles in people diagnosed antibody-positive as well as their absence in those antibody-negative. Instead, cell cultures from AIDS patients were activated using powerful chemicals called mitogens and after this activation, about thirty proteins were found in this mixture, all of which gathered at a density characteristic of retroviruses. A subset of these was specifically attributed to HIV and nothing else, and ten of these are used to define reactivity on the ELISA and Western Blot HIV antibody tests.

The stunning part of this story is how, out of thirty or so possible retroviral proteins, those ten were selected as being specifically from HIV and nothing else. Remember, HIV had not been properly isolated at this point and there was no way of knowing directly that any of these proteins was specific to HIV. So, in an amazing display of circular logic, they simply selected the proteins that most commonly reacted in blood samples of AIDS and pre-AIDS patients (Petricciani et al. 1987, Schochetman et al. 1994). No wonder there is a correlation between testing HIV-positive and developing AIDS in some risk groups.

Although this reasoning is absolutely scandalous, the problems with the HIV tests do not stop there. The initial ELISA test must be run on serum that has been diluted four hundred fold with a special diluting agent provided by the test manufacturer. This seems rather strange, particularly considering that most antibody tests—for example, the test for antibodies to hepatitis B—are run on undiluted serum, and even those that are diluted are diluted by a very low factor, such as for Epstein-Barr virus, which is diluted tenfold. The only antibody test that has a dilution factor that could possibly be described as approaching that of the HIV ELISA is the rheumatoid factor (RF) antibody test, which must be diluted fortyfold—which is still an order of magnitude lower than the dilution required for the HIV ELISA. (The HIV WB is run at a dilution factor of 50:1.)

A crucial fact about the rheumatoid factor antibody test is that it is testing for elevated levels of antibodies that are very common, and whose elevation (rather than mere presence) indicates some sort of autoimmune response that is not normal. Without dilution, it would be impossible to distinguish those with elevated levels of antibodies from controls with normal levels of antibodies.

One wonders what would happen if the HIV ELISA were run undiluted. Amazingly, there is an answer to this question available. Dr. Roberto Giraldo, a medical doctor working at the Cornell University hospital, ran an experiment in which he tested over one hundred *undiluted* patient samples, including a sample of his own blood, all of which reacted "negative" on ELISA as it is run according to normal testing protocol. He discovered that *every sample reacted* on ELISA when undiluted. This means that 100 percent of samples tested "positive" when undiluted (Giraldo 1998).

While this example alone should be enough to cast significant doubt as to what it is, exactly, that these tests actually detect, it gets worse.

The HIV antibody tests contain a mixture of ten or eleven "HIV-specific" proteins. In the ELISA, the proteins are present as a mixture, and the serum reacts with the proteins in such a way as to cause a color change. The color change is not discrete—meaning that everyone has varying degrees of reaction. It isn't as though those who are really "HIV-infected" have the reaction, whereas those who are not show no difference. There are varying degrees of the color change, and a cutoff value has been established, above which the sample is considered reactive or "positive" and below which it is considered "negative."

Clearly, this language is absurd, since *positive* and *negative* are polarities and not positions on a sliding scale. Moreover, the decision as to where the cutoff is placed is not universal but is determined by the testing venue and depends on what the test is intended for (Papadopulos-Eleopulos et al. 1993, Turner et al. 1999). This is patently ridiculous—like deciding that in Texas "cold" will be 32 degrees but in New Hampshire it will be 25 degrees. Hence I strenuously

object to the terms "positive" and "negative" in the context of HIV tests, since clearly these words are not well defined. "Reactive" and "nonreactive," though still not perfect descriptors of what is actually happening, are more realistic.

With the WB, the proteins are separated out according to their molecular weight in kilodaltons and are then presented as "bands" on a thin nitrocellulose strip, so that a reactive test is determined by a particular combination of reactive protein bands. As with the ELISA, a "positive" result on the WB is not consistently defined. Depending upon the lab or the country in which the lab is located, different combinations of two, three, or four bands are sufficient to diagnose HIV infection (Papadopulos-Eleopulos et al. 1993).

There is an important question here waiting to be asked: If all these proteins are specific to HIV, shouldn't only one protein be sufficient to diagnose infection? On the other hand, if a person is truly infected, shouldn't their serum react with all ten bands, not just two or three or four?

It turns out that there is ample evidence in the medical literature that cross-reactivity with several of these proteins is *extremely* common in the general, low-risk population. It has been found that between 20 and 40 percent of blood donors from the general population show "indeterminate" WB results, meaning that they have one or two reactive bands, or some combination that "does not fit the criteria for positivity" (Proffitt 1993). This means, if the HIV tests are accurate, that these people have antibodies to one or two HIV proteins. (However, in Africa two reactive bands are enough to diagnose infection, and in most places in the U.S., Canada, and the U.K., three bands suffice. The most stringent criteria of four reactive bands—but not the same four—is adhered to by only two countries, France and Australia.)

An extremely comprehensive review of the Western Blot test was published in the journal *Bio/Technology* (now *Nature Bio/Technology*) (Papadopulos-Eleopulos et al. 1993). It was shown that of the proteins present in the Western Blot HIV antibody test, the following nonspecificities can be noted:

The protein gp120, which is considered to be a component of the envelope of HIV, and as such being part of the "knobs" or "spikes" on its surface, which enable it to enter an uninfected cell, is not specific to HIV. The proteins gp41, p80, and gp160, are all associated. Specifically, p80, gp120 and gp160 are all considered to be "oligomers" of gp41—which basically means they consist of the appropriate number of gp41 proteins hooked together. Gp41, itself, has been shown to be nonspecific and is considered to be a component of cellular actin, ubiquitous in human cells and certainly not specific to HIV (Barré-Sinoussi et al. 1983, Stanislawsky et al. 1984).

The p24 protein is considered to be synonymous with HIV infection. In fact, newborns are often tested for p24 antigen as a surrogate marker for HIV infection, since antibody tests cannot be used due to the persistence of "ghost" antibodies inherited from the mother that persist for up to eighteen months. However, p24 is frighteningly common among individuals at no risk of HIV infection. Serum from blood donors that is nonreactive on ELISA has a 20 to 40 percent chance of being "WB indeterminate," and p24 is the most commonly cross-reacting protein, appearing in 70 percent of indeterminate cases. Furthermore, 41 percent of multiple sclerosis patients who are not ELISA-reactive test positive for p24 antigen. Even more puzzling is that p24 is detectable in nowhere near 100 percent of AIDS patients.

In other words, of ELISA-negative serum, 14 to 28 percent tested have non-HIV-specific reactions to p24. Further, considering that not all AIDS patients have detectable p24, this means the presence of p24 is neither necessary nor sufficient to diagnose HIV infection.

The p18 protein is the second most frequently detected protein in blood donors at no or very low risk of HIV infection. Along with the HIV *pol* protein p32, it has been detected in many situations in which HIV infection is extremely unlikely, and thus cannot be considered to be indicative of HIV infection.

It is germane to note at this point that in all the labs, criteria for positivity of the Western Blot test consists of some combinations of the above mentioned proteins—gp160, gp120, gp41, p24, p18, and p32. However, since none of these proteins is specific to HIV, this would be like saying that since dogs have four legs, are furry, wag their tails, and enjoy eating steak, that *any* entity that is furry and enjoys steak must be a dog.

Of course, antibody tests must satisfy three criteria: they must be specific (meaning very few people truly "negative" would test positive), sensitive (meaning very few people truly "positive" would test negative), and they must be precise, or reproducible. The issues of specific-

ity and standardization have been addressed, and following one further comment regarding the specificity of the HIV antibody tests, we shall discuss their lack of precision.

Test manufacturers and AIDS educators commonly claim sensitivity and specificity levels for the HIV antibody tests of 99 percent or better. While this sounds like an impressive figure, it is meaningless in light of the fact that the aforementioned sensitivity and specificity are estimated by comparing antibody tests against one another and not against HIV itself. However, the problems are considerably worse than this.

Suppose for the sake of argument that these values reflected the true accuracy of the HIV test. HIV is thought to be present in about 0.4 percent of the US population, or in about one of 250 randomly selected Americans. Suppose that we were to administer an HIV test to ten thousand randomly selected Americans. In such a random sample, we would expect forty "true positives," with the remainder, or 9,960 people being negative. A 99 percent sensitivity would mean that 1 percent of those truly positive would actually test negative. With forty people positive, *perhaps* one person would register false negative. So it appears that the test is really quite acceptable as far as eliminating false negatives is concerned.

However, a 99 percent specificity level means that 1 percent of those truly negative would test positive; 1 percent of 9,960 is approximately one hundred people, so we can see that the number of false positives would outnumber the number of true positives by a factor of one hundred to forty, or 2.5! This is because the prevalence of HIV in the population is so low. As the prevalence increases, we get fewer false positives. This factor of true positives to total positives is also known as the positive predictive value (PPV) of the test, and it indicates what percentage of all positives we can expect to be true positives. A PPV of 40/140 means that in the total population, *we can expect only about 35 percent of all positive tests to be "true" positives.*

If we test outside the risk groups, the prevalence of HIV goes down to about one in five thousand, or 0.02 percent. Testing ten thousand non-risk group Americans would yield *two* true positives. However, we would obtain approximately one hundred false positives in this case, and the PPV is less than 2 percent! Clearly, testing outside the risk groups would mean that almost everyone who would test positive would be a false positive, and, extrapolating to the general population, tens of thousands of people would be terrorized and put on poisonous drugs for no reason—a medical disaster.

Repeat testing would eliminate many of these false positives, but not all of them, as we will see. Perhaps the most striking example of the imprecision, or nonreproducibility, of the WB test, can be found in the Army study by Colonel Burke and coauthors. In all, 135,187 military applicants at very low risk for HIV infection were selected and tested using the protocol of an initial screening ELISA, followed by a second ELISA if the first was reactive, then a WB if the second ELISA was also reactive, and finally a second WB if the first WB was also positive (Burke 1989). They found that on initial ELISA screening, six thousand individuals tested positive. Upon repeating the ELISA, two thousand people were negative, leaving only four thousand positive specimens. These four thousand specimens were then tested. Among those whose first WB was reactive, eighty had a positive WB followed by a negative repeat WB. In the clinical setting, the testing would have stopped at the first positive WB, leaving eighty people determined to be truly negative in the Army study who would have been given a death sentence if they were tested by their doctors. How many, if all Americans were tested as per the CDC's recommendation, would be given a death sentence *even with repeat testing?* Since eighty of 135,187 false positives would not have been eliminated by accepted test procedures, this means *more than 170,000 Americans would be given a death sentence for no reason.*

This problem is further confounded in the ELISA test, since the proteins are present as a mixture, and there is no way of knowing what sort of cross-reactivity may be occurring. It certainly seems as though virtually every human would have a reactive ELISA test if the test were run undiluted, so what does this mean about the specificity of the test? There is no other interpretation than to say that the test is a nonspecific test, like the test for RF antibodies. If the tests were highly specific (which is doubtful), the only possible explanation would be that more or less everyone has been exposed to HIV at some time, but some people simply produce more antibodies than others, and these people's antibodies still react even under a four hundred–fold dilution.

Assuming that this explanation is not reasonable, which I suspect to be the case, the other possible reason for the results indicated above is that the tests are simply nonspecific and cannot in any way diagnose

infection with a *particular* microbe. The best they can do is to detect a condition called *hypergammaglobulinemia,* meaning having too many antibodies to too many things. This interpretation is perfectly consistent with the finding of reactive specimens in most AIDS patients. It has been known since the beginning of the AIDS epidemic that AIDS patients had generally been exposed to a vast number of infections and recreational drugs prior to testing positive. Since infections, as well as drug use, induce antibodies, it is no surprise that the likelihood of cross-reactions will increase. It is also known that having so many antibodies indicates a problem with the antibody arm of the immune system, and that having such problems typically accompanies a deficiency in cell-mediated immunity—exactly what is observed in AIDS patients.

It is relevant to note that about 40 percent of the human genome is composed of what are called *RNA transposable elements* (Griffiths 2001). *RNA* is composed of a single strand of nucleotides (rather than the familiar double helix of DNA) and replicates differently than does DNA. The word *transposable* means that they can move or "jump" around, as well as cleave and form *endogenous retroviruses.* Endogenous retroviruses are the same in structure as "conventional" exogenous retroviruses, as HIV is purported to be, having at least three genes, *gag, pol,* and *env.* This is significant because, among other reasons, it is impossible to distinguish an endogenous retrovirus from an exogenous retrovirus simply by looking at a picture. This is part of what makes retroviruses so different from "ordinary" viruses.

Human beings are full of retroviruses that start out as retroviral sequences in the genome. They are expressed as endogenous retroviruses whenever cells are decaying at a higher rate than normal and often when cells are dividing and growing at a higher rate than normal. This is a major confounding factor for the HIV tests because during times of disease or growth, such as pregnancy, a higher than normal level of endogenous retroviruses will be expressed, and we form antibodies to their proteins. This greatly increases the chances of cross-reactivity, and it at least partly explains why people whose health is compromised in the first place are more likely to test HIV-positive, as well as why people who test HIV-positive are more likely to become ill. The retroviruses are simply a marker for cell decay and/or division.

Furthermore, some of the known human endogenous retroviruses (for instance, HERV-K and HERV-W) not only produce antibodies that cross-react with the HIV test (Vogetseder et al. 1993), but they have RNA sequences that are similar to those of HIV, and these sequences are very likely to be mistaken by the viral load PCR as fragments belonging to HIV. (Viral load PCR does not measure intact viruses but rather fragments believed to belong to HIV, as we will discuss further later in this chapter.)

Endogenous retroviruses are primarily transmitted perinatally, from mother to child. Perinatal transmission is presumed to be the most efficient mode of HIV transmission, which should raise suspicions as to whether there is sufficient information to conclude that HIV is even exogenous at all, particularly given the lack of solid evidence of sexual or perenteral (blood-to-blood as via infected needles) transmission (Bruneau et al. 1997, Gray et al. 2001, Hugonnet et al. 2002, Padian et al. 1997).

The idea that the HIV tests might measure a nonspecific marker for an immune system with a broken antibody arm is further strengthened by the fact that these tests have never been validated against the gold standard of HIV isolation. Since the diagnosis HIV-positive carries with it such a stigma and the potential for outrageous denial of human rights, it is only humane that doctors, AIDS researchers, and test manufacturers would want to make absolutely certain that the tests they are promoting are completely verifiable in the best possible way.

This is not happening. The tests have never been verified against the presence of HIV because, to date, there is no clear evidence that HIV has been isolated in such a manner as to be acceptable as a gold standard for antibody tests. By isolation, HIV researchers usually mean successful culturing, which merely means that certain chemical reactions indicating phenomena consistent with HIV have been observed.

Etienne de Harven published a paper in 1998 that was highly critical of the methods used for isolating HIV and the other human retroviruses, as well as the subsequent development of the antibody tests.

> When, around 1980, Gallo and his followers attempted to demonstrate that certain retroviruses [can cause disease in humans], to the best of my bibliographical recollection, electron microscopy was never used to demonstrate directly viremia (the presence of viruses in the blood) in the studied patients. Why? Most probably electron-micrographic results were negative, and swiftly ignored! But over-enthusiastic retrovirologists

continued to rely on the identification of so-called "viral markers" attempting to salvage their hypothesis ... ELISA, then Western Blot tests were hastily developed, at sizable profits eagerly split between the Pasteur Institute and the US. "Seropositivity" (based on these two tests) became synonymous with the disease, itself, plunging an entire generation into behavioural panic, and exposing thousands of people to "preventative" AZT therapy which actually hastened the appearance of severe or lethal immunodeficiency syndrome. (de Harven 1998)

HIV researchers will swear up and down that HIV has been properly isolated and that such apparently sensible criteria as separation of viral particles from everything else and proof of their existence as shown by clear electron micrographs are not necessary.[9] You might think that with the hundreds of billions of dollars spent so far on HIV, there would have been by now a successful attempt to demonstrate HIV isolation by publication of proper electron micrographs. The fact that there has not indicates quite strongly that no one has been able to do it. Since the "isolation problem" has long been an argument put forth by scientists questioning HIV, it seems that if it were possible to resolve this problem, mainstream researchers would be eager to do it if only to shut such dissenters up.

While this may be alarming enough in and of itself, it is of particular concern when one considers that every day people are given a diagnosis of imminent death based on a test whose value as a diagnostic tool is very dubious indeed. One need only consider some of the disclaimers included in any of the popular test kits:

ELISA testing alone cannot be used to diagnose AIDS.
—*Abbott Laboratories test kit (Abbott 1997)*

Do not use this kit as the sole basis for HIV infection.
—*Epitope Western Blot kit (Epitope 1997)*

The amplicor HIV-1 monitor test is not intended to be used as a screening test for HIV, nor a diagnostic test to confirm the presence of HIV infection.
—*Roche viral load kit (Roche 1996)*

As to so-called viral load, most people are not aware that tests for viral load are neither licensed nor recommended by the FDA to diagnose HIV infection. This is why an "AIDS test" is still an antibody test. Viral load, however, *is* used to estimate the health status of those already diagnosed HIV-positive. But there are very good reasons to believe it does not work at all. Viral load uses either polymerase chain reaction (PCR) or a technique called branched-chain DNA amplification (bDNA). PCR is the same technique used for "DNA fingerprinting" at crime scenes where only trace amounts of materials can be found. PCR essentially mass-produces DNA or RNA so that it can be seen. If something has to be mass-produced to even be seen, and the result of that mass production is used to estimate how much of a pathogen there is, it might lead a person to wonder how relevant the pathogen was in the first place. Specifically, how could something so hard to find, even using the most sensitive and sophisticated technology, completely decimate the immune system? While not magnifying anything directly, bDNA nevertheless only looks for fragments of DNA believed, but not proven, to be components of the genome of HIV—but there is no evidence to say that these fragments don't exist in other genetic sequences unrelated to HIV or to any virus.

While at first glance it might seem completely reasonable to estimate the quantity of a pathogen by amplifying it and then using the amplification formula to back-calculate for the true quantity, there are serious problems with this approach. As Mark Craddock explains, the efficiency of PCR must be *perfect* in order to obtain an accurate value (Craddock 1996). This is rarely the case. If the efficiency is off by even a small amount, the error has the potential to increase (or decrease) exponentially because PCR amplifies up to forty-five times. Even the mainstream literature (Piatak et al. 1993) admits that viral load testing overestimates infectious virus by a factor of at least sixty thousand. This means that a viral load of sixty thousand corresponds to at most one infectious viral particle. In the aforementioned Piatak paper, fully one-half of their patients with detectable viral loads had no evidence of virus by culture.

More damning evidence against the use of viral load as an indicator of clinical health is given by Mark Craddock in his rebuttal to the Durban Declaration. In his letter, which remains unpublished to this day,[10] he examined the patients in the Piatak paper. Using their CD4+ T-cell counts, viral loads, and measurements of virus by culture, he computed correlation coefficients on all pairwise combinations. A correlation

coefficient is a numerical value that measures the strength of the relationship between two variables. A correlation coefficient close to 1 means a nearly 100 percent association, whereas a correlation coefficient near 0 means there is no association. Statisticians generally view any correlation coefficient less than 0.5 as indicating very poor correlation.

Craddock's computations revealed that among all pairwise combinations, the correlation coefficients were close to zero. This is extremely relevant, because it means that T-cell count has no effect on viral load, viral load has no relation to infectious virus levels, and infectious virus levels have nothing to do with T-cell count. In other words, *all laboratory tests used to assess the severity of HIV infection are virtually worthless.*

It is worth noting at this point that viral load, like antibody tests, has never been verified against the gold standard of HIV isolation—bDNA uses PCR as a gold standard, PCR uses antibody tests as a gold standard, and antibody tests use each other. None use HIV itself (Johnson 2001).

It is also germane to note that Kary Mullis, the *inventor* of the PCR technique, which is the primary tool used in assessing viral load, wastes no opportunity to publicly decry the misuse of PCR to quantify viral load. Dr. Mullis has called the HIV/AIDS hypothesis "one hell of a mistake" and has stated many times that "quantitative PCR is an oxymoron" (Mullis 1996).

However, I would argue that the real problem with the administration of HIV antibody tests lies not with the tests themselves but with how they are used essentially as weapons of terror. This medical terrorism reached new heights in June 2006 with the CDC's new HIV testing guidelines, which recommend that everyone between the ages of thirteen and sixty-five be tested for antibodies to HIV. Prior to the publication of these guidelines, HIV tests were not standard practice, due partly to the fact that pre- and post-test counseling was to be given alongside the tests, making the testing process expensive and time-consuming. In general, to get an HIV test, one either had to visit an STD or HIV clinic and request to be tested, or one needed to specifically ask one's doctor. (Other portions of the population, such as blood donors, military recruits, and patients undergoing certain hospital procedures, are subject to mandatory testing, but these segments of society do not comprise a large proportion of the population.)

Hence, it is not surprising that the vast majority of HIV tests have traditionally been sought by individuals in risk groups or people who had some good reason to believe they had contracted HIV. The new testing guidelines could change all this, and as a result, the number of false positives will soar. This is owing to Bayes's Law, which states that the higher the prevalence of a pathogen in the population, the higher will be the positive predictive value (PPV) of the test—that is, the lower the rate of false positives will be. The problem, as we have seen, is that in a population with low prevalence, the PPV will plummet and the rate of false positives will soar. Of course, many of these false positives can be eliminated by repeat testing, but as the Army study noted above clearly demonstrates, repeat testing will not eliminate all of these false positives.

Why is this a problem? Aside from the fact that many people who are perfectly healthy will be coerced into undergoing a regimen of medication that will inevitably cause long-term toxic effects (and often death), a more sinister complication is the violation in human rights that occurs following a positive HIV test. Every state in the U.S. and every province in Canada maintain a list of "HIV carriers" in that region. Once diagnosed HIV-positive, medical and life insurance can be denied, some careers may be terminated, but worst of all, a death sentence is given and, contrary to every other disease known to man, even cancers that are generally 100 percent fatal, hope is not allowed. Women are encouraged to abort their babies, and if they choose to carry their pregnancy to term, in many states they are forced to take antiretroviral drugs, and these drugs are forced on their babies as well. The babies themselves must be born by Cesarean section, and in many states the highly beneficial practice of breastfeeding is illegal.

Clearly, the "HIV test" needs to be thoroughly reappraised as a diagnostic tool. Results of this test should not be used to discriminate against anyone, especially since the test itself is so unreliable. But more urgently, *at the very least,* the HIV antibody tests ought to be rigorously verified against the actual presence of HIV itself. This has never been done.