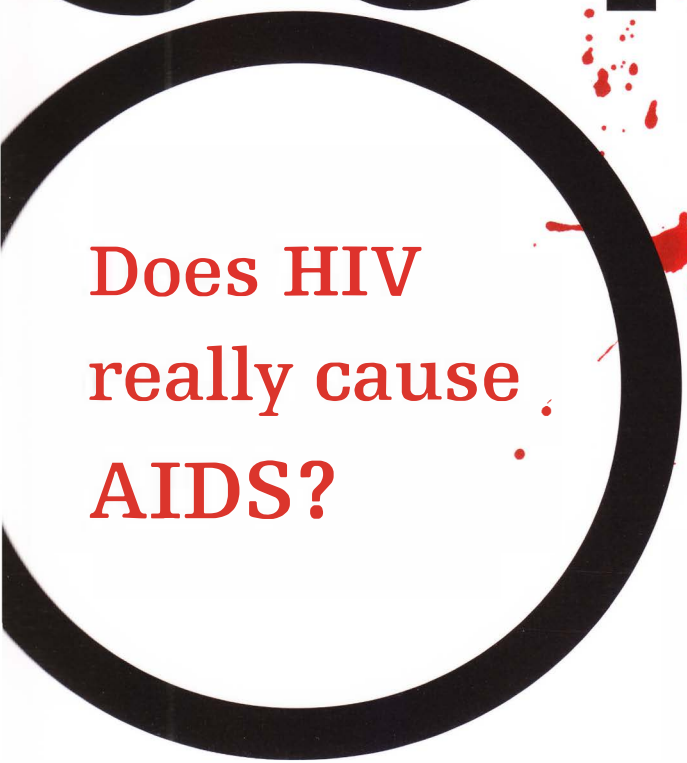
REBECCA CULSHAW

Science Sold Out

Does HIV really cause AIDS?



Foreword by
Harvey Bialy

author of *Oncogenes, Aneuploidy, and AIDS: A Scientific Life & Times of Peter H. Duesberg*

Science Sold Out

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I appreciate the support of all the people, too numerous to mention, who wrote or called with words of encouragement.

I thank, from the bottom of my heart, the scientists, journalists, and activists who have worked tirelessly for justice and fairness regarding HIV/AIDS. I'm sure I would miss some people, so I won't even try to name any names.

AUTHOR'S NOTE

THE PURPOSE OF this note is to explain a little about the format of the book and, in particular, what has been left out.

After much consideration, I decided not to include a chapter or section on what I think *does* cause AIDS. This decision was not made lightly. I know that either way, I will be attacked. If I don't hypothesize as to the true causes of AIDS, if not HIV, I will be attacked for having "not even suggested an alternative explanation." If I do present such a theory, I'll be attacked because, inevitably, my theory will not be able to explain every case of AIDS in the way that purported HIV infection does, although HIV infection only explains AIDS *because it is part of the definition of AIDS.*

Ultimately, the reason that I did not include such an analysis is because, quite simply, that is not what this book is about. This book purports to illuminate critical flaws in the HIV hypothesis. Those who criticize a theory have no obligation to propose an alternative. In order to exonerate one accused of a crime, in no way must we prove who actually did commit the crime—we need only present sufficient evidence that it was not the accused. HIV is falsely accused of causing AIDS.

Having said this, however, it is my opinion that AIDS is complex, and there is no possible way that one single agent can explain all cases of AIDS. Certainly drug use explains some cases of AIDS. Certainly multiple infections can explain some cases of AIDS. Certainly malnutrition can explain some cases of AIDS. Psychological terror in and of itself can cause massive immune dysfunction, and there is no lack of

psychological terror evident in the HIV campaign. And finally, there are simply some cases in which a person dies, and there is no apparent explanation. The boyfriend of a woman I work with died suddenly this year from a raging infection. He became very ill, and his immune system collapsed, unable to handle the infection, and he died. He was not HIV-positive, but if he had been, he would have been an AIDS case.

Please note that 100 percent of the profits from the sale of this book will go to support the Serge Lang Memorial HIV/AIDS Archive.

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FOREWORD

AS DR. CULSHAW'S text so clearly speaks for itself, there is really no need for any analytical preface, and I will not even attempt one. Instead I will tell a story of how this *Terra Nova* book came to be, in the hope that this story will make the reasons for its publication now more apparent.

In February of this year, Darin Brown, a mathematics professor at Eastern New Mexico University, and Frank Lusardi, a computer programmer in New York City, began a Wikipedia-style website devoted to providing a "fair" (meaning data rich) but by no means "balanced" forum for the Internet presentation of HIV/AIDS dissent. When I was directed to this AIDS Wiki, one of the first things I saw was a page devoted to "Petitions," and because I had just launched one of my own, I of course checked to see if these "newcomers to Internet AIDS insurgency" had included it. Indeed they had, but described it quite incorrectly and thus confirmed some of my worst fears concerning the petition.

The petition, which I thought a simple and quantitative way to test the truth of one of the favorite orthodox shibboleths for denying credibility to dissenting arguments—that the vast majority of scientists had already considered the Duesberg critique for many years and found it lacking in substance, and so saw no reason at all to further engage in a useless and possibly dangerous debate—had turned out to be not simple at all. In fact, it was apparently so confusing that almost nobody who read it understood either its semantic content or its purpose.

The text of the petition called on the editors of *Nature* and *Science* to take an anonymous, electronic straw poll of their readers asking them

whether they thought a series of debates sponsored by the National Academy between Peter Duesberg and David Baltimore (the two most prominent and best-credentialed scientists on opposite sides of the AIDS-causation question) would be a waste of time. Like the two blackboards with "A Bird in the the Hand" and "Paris in the the Spring," it was very difficult for most people to see that the petition did not actually call for such a debate.

And so I wrote an e-mail to the moderator of this new website pointing out the correction. Not fifteen minutes later I received a reply apologizing profusely for the oversight (and of course correcting the web entry), excusing the lapse only by saying how pressured he had been in trying to build the site's content quickly and that he was really much better at solving differential equations than his misreading of the simple text might have indicated. This began my acquaintance with the remarkable Dr. Brown.

Some short while later, Donald Miller, MD, a very highly regarded cardiac surgeon as I was to learn, published an online review of my biography of Peter Duesberg. The review appeared on the extremely popular website of Lew Rockwell, and I was surprised to see it quickly become a "most popular" item.

Some short while after that, I learned about Dr. Brown's good friend Rebecca, who was thinking of leaving the "Sunny Brook AIDS Farm" after ten years of making hay for it as an HIV mathematical modeler and had written a cathartic first-person account of her journey from unquestioning believer to convinced heretic. She was wondering if I would be kind enough to look it over and to please not be too hard on her writing as it was her first go at this kind of prose.

What she sent was an essay entitled "Why I Quit HIV," and it stunned me, as it did Lew Rockwell (to whom I suggested she send it without any line editing at all) and everyone else who has read it, including Richard Grossinger and Lindy Hough, the publishers of North Atlantic Books.

The rest, as they say, is her story.

Harvey Bialy
Cuernavaca
June 12, 2006

Introduction

THE PARADOX OF THE PREVALENCE CURVE

ANY BOOK THAT purports to reveal and explain the many flaws, paradoxes, and examples of circular logic—and often just plain *il*logic—in the HIV=AIDS=DEATH theory should introduce the reader to one such fatal flaw straight away. And so I present to you the paradox of the U.S. HIV prevalence curve (Duesberg et al. 2003a).

Before I present the curve itself, please note that although many of the arguments presented in this narrative refer specifically to North America (and, by extension, Europe, as part of the First World), essentially all of them apply to HIV/AIDS anywhere else in the world. The virological and immunological arguments I present are, of course, applicable no matter what geographic location one wants to consider. But this applies to the epidemiology as well because most of the reports we hear about HIV rates in places like Asia and Africa are simply statistical contrivances with no basis in reality.[1] Although it is true that the raw prevalence of HIV in sub-Saharan Africa is indeed higher than it is in North America and Europe, the fact is that in no case does HIV prevalence ever fit with AIDS incidence.

The word *curve* is actually a misnomer when it comes to describing the HIV prevalence graph shown below because as you can clearly see, with the exception of a small *drop* in case estimates in 1995, the

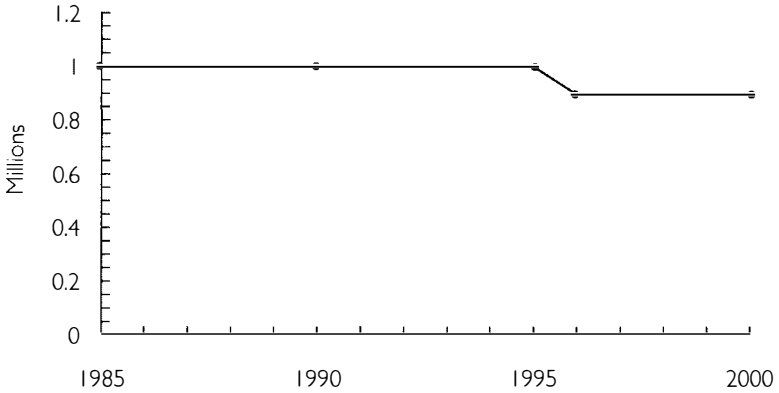

FIGURE 1: Prevalence of HIV in U.S. population

prevalence of HIV in the U.S. population has remained, for all intents and purposes, perfectly constant since testing began in 1985 (See Figure 1).

Please note also that although the graph terminates in the year 2000, official estimates remain similar, and the latest CDC estimates for HIV prevalence state that approximately one million Americans currently test positive for HIV (CDC 2003, CDC 2005), a fact that would change the graph little.

It is important as well to point out that although, yes, this curve is estimated—largely owing to the fact that because not everyone tests for HIV, we can never really be sure *exactly* how many Americans truly test positive—the estimations on which this graph is based depend upon what are still very high levels of testing. HIV prevalence estimates in the U.S. are in fact based upon more actual testing than almost any other disease testing (Bauer 2005).

In contrast to the HIV prevalence curve, U.S. AIDS cases peaked in 1993–94 (Duesberg et al. 2003a). Although this was due, at least in part, to the expansion of the AIDS definition by the CDC in 1993, it is clear that the AIDS epidemic is nonconstant, and indeed, after increasing slowly prior to 1993, it has gradually declined up to the present day. We often hear phrases in the lay media such as "The number of AIDS cases is double what it was *x* number of years ago," which creates a false sense of alarm because it implies that *many more people are now*

developing AIDS than ever did before. What the media reports don't mention, however, is that the numbers given are cumulative totals, in which all new cases for a given year are added to all the cases for all the years prior to yield a running total. Of course, if you continually add up all the AIDS cases since the beginning of AIDS record keeping, it will be impossible to ever obtain a decrease.

The true numbers of annual AIDS cases, however, are not reflected by cumulative totals but rather by annual incidences. The figure above displays the estimated number of HIV-antibody-positive people in the U.S. for each given year, and as the figure clearly conveys, this number has remained almost perfectly constant since 1985 at about one million. With a U.S. population of about 295 million people, this amounts to only 0.4 percent of U.S. citizens testing positive for HIV antibody.

This data should sound a clear alarm when one considers the supposed "infectious" nature of AIDS (and possibly HIV). First, if HIV is a new pathogen, then its prevalence should not have remained constant—it should have clearly increased, according to Farr's Law, which asserts that a new contagion spreads exponentially throughout the population. More damning, however, is the following.

HIV is said to cause AIDS on average eight to ten years after infection. If HIV causes AIDS, then the incidence of AIDS should have mirrored the prevalence of HIV, only shifted eight to ten years into the future. If HIV causes AIDS, the AIDS incidence curve should be flat. This is not the case.

The discrepancy cannot be explained away by AIDS drugs because this cannot account for the sharp rise in AIDS incidence between 1987, when the first AIDS drugs were marketed, and the drop that began in 1993.

There are many more flaws in the HIV theory of AIDS, and the following pages will highlight some of the more damning of these. It should certainly be clear to anyone who was around during the 1980s that AIDS now looks nothing like what it was predicted to look like twenty years ago. The growing focus on Africa—and to a lesser extent Asia—is merely a tactic to keep people supportive of AIDS, and thus maintain the funding of scientists and activists who work on AIDS, because it is clear that if we were to base our decisions upon what is happening at home, the AIDS industry would all but disappear. Instead, we remain transfixed by the notion of a deadly sexual plague decimating all

of Africa and potentially decimating any of us because of our inherent human need to focus our collective fears and insecurities on a tangible, concrete threat.

AIDS has become so mired in emotion, hysteria, and politics that it is no longer primarily a health issue. AIDS has been transported out of the realm of public and personal health and into a strange new world in which pronouncements by powerful government officials and ill-informed celebrities are taken as gospel, and no one even remembers when, a few years later, these pronouncements turn out to be false.

If we were to rewind the clock twenty-five years and superimpose today's beliefs about AIDS onto the landscape, it might raise a few eyebrows.

Some examples: First of all, there's the clinical latency period—the time from initial infection with HIV to the development of the syndrome AIDS. Initially, the asymptomatic phase from infection to AIDS was six months. This figure grew to a year, then five years, then ten, and now—as there are people who remain inexplicably healthy since the mid-1980s despite alleged infection with a supposedly deadly virus—fifteen years, twenty years ... Who knows?

Then there is the indisputable fact that neither AIDS nor HIV have spread like they were predicted to. The predicted heterosexual AIDS explosion never happened, and to even mention this prediction now is almost taboo as it is such an embarrassment to the AIDS establishment. As we observed from the prevalence curve, HIV has not spread at all, but rather it has remained constant in the population since its detection. The African epidemic looks suspiciously nothing like the American and European epidemic, and closer inspection reveals it likely that this African epidemic is pure fabrication.[2]

You might remember that in 1987 the CDC, via Oprah Winfrey, made the dire prediction that by 1990, one in five heterosexuals would likely be dead of AIDS. You might remember that both a vaccine and a cure were promised by 1986. You might wonder why people died so quickly on AZT, supposedly a "magic bullet." You might notice that they aren't dying so quickly on the new drugs—but then why do they look so wasted, drawn, and sick? Something's wrong here.

Scratching the surface just a little bit more, one uncovers many more problems than just some bad drugs and some clearly faulty predictions. The problems range from the fact that no one really understands how

HIV actually works—or even, for that matter, what HIV really is—to the paradox of how a disease could cause both vastly different epidemiologies and symptomatic presentations in the First and the Third Worlds.

As has been said by others, there are no paradoxes in nature, only flawed hypotheses (Duesberg et al. 2003b). Questions about HIV and AIDS have been raised since HIV was first discovered, and as the years pass, the questions accumulate but remain largely unanswered. Any such theory—one that cannot even answer questions for which it was put forth—should be looked at very critically.

One

HOW I CAME TO CHANGE MY MIND

SCIENTISTS HAVE BEEN criticizing the HIV/AIDS paradigm for over twenty years now. What makes me any different?

My chosen career has developed around the HIV model of AIDS. I received my PhD in 2002 for my work constructing mathematical models of the immunological aspects of HIV infection, a field of study I entered in 1996. Just ten years later, it might seem early for me to be looking back on and seriously reconsidering my chosen field, yet here I am.

My work as a mathematical biologist has been built in large part on the paradigm that HIV causes AIDS, and I have since come to realize that there is good evidence that the entire basis for this theory is wrong. AIDS, it seems, is not a disease so much as a sociopolitical construct that few people understand and even fewer question. The issue of causation, in particular, has become beyond question—even to bring it up is deemed irresponsible.

Why have we as a society been so quick to accept a theory for which so little solid evidence exists? Why do we take proclamations by government institutions like the NIH and the CDC, via newscasters and talk show hosts, entirely on faith? The average citizen has no idea how weak the connection really is between HIV and AIDS, and this is the manner in which scientifically insupportable phrases like "the AIDS

virus" or "an AIDS test" have become part of the common vernacular despite no evidence for their accuracy.

I have come to the conclusion that the massive scientific, governmental, and societal acceptance of the HIV/AIDS model has little to do with any real evidence implicating HIV. The paradigm has been supported from the beginning by government institutions that, perhaps inadvertently, encourage poor-quality scientific research standards. But the problem is even more complex than that. There is something truly bizarre about the fact that the announcement of the discovery of the causative agent of AIDS—via press conference, no less—was immediately accepted by scientists and citizens alike before any supporting evidence had been published or critiqued in the scientific literature. Although I believe that the decline in scientific standards is the major reason HIV researchers seem to suffer from tunnel vision and some sort of collective amnesia that enables them to consider no other cause for the complex phenomenon of immune deficiency other than a single virus, as well as to conveniently "forget" every few years when they announce a new and exciting discovery that will "explain everything" that a similarly new and exciting discovery from a few years back is now shown to be wrong, there are more subtle forces at work here. The sociological reasons behind society's immediate acceptance of the HIV theory are profound and far-reaching, and I will address these later.

As a child, I felt terrorized by the specter of AIDS. When it was announced in 1984 that the cause of AIDS had been found in a retrovirus that came to be known as HIV, there was a palpable panic. My own family was immediately affected by this panic because my mother had had several blood transfusions in the early 1980s as a result of three late miscarriages she had experienced. In the early days, we feared mosquito bites, kissing, and public toilet seats. I can still recall the panic I felt after looking up in a public restroom and seeing some graffiti that read: "Do you have AIDS yet? If not, sit on this toilet seat."

But as a teenager, I noticed that within a few short years, people stopped distinguishing between those who were "HIV-positive" and those who actually had AIDS, beginning to assume they were the same thing. I was no expert in the field by any means, but I paid attention to the news and have always had an interest in medicine, and I could not see the defining event that caused people to accept the change from HIV as "the virus associated with AIDS" to "the virus that causes

AIDS." I remember people referred to Magic Johnson as "having AIDS" and I objected, purely on the basis of logic, "No, he's *HIV-positive.* That's not the same thing."

However, years passed, and I simply assumed that HIV did cause AIDS, that more and more people were going to get sick and die, but that it was possible that some HIV-positives were simply carriers who might never get sick. I certainly heard enough stories about long-term, AIDS-drug-free survivors to plant a seed of doubt in my mind that HIV did not *always* lead to AIDS.

One of the reasons that I chose to write a master's thesis on mathematical models of HIV infection was my curiosity about this disease, and I figured this would be an excellent way to read as much of the medical literature as possible and to start getting some answers. Little did I know, as I completed my master's degree and continued to write a PhD dissertation on the same subject, that what I would learn would go a long way toward explaining why I'd always been so confused about AIDS.

Two

SCIENCE SOLD OUT

AIDS IS SAID to be caused principally by the HIV-mediated destruction of CD4+ T-cells. The first conundrum I encountered was the lack of agreement on, or evidence for, *any* mechanism by which HIV supposedly caused this cell death. The second problem, less troubling on a purely virological level, but much more disturbing in light of scientific standards, was that papers on the molecular biology of HIV seemed to have a very short shelf life—they go out of date very quickly. In mathematics a journal article takes a significant amount of time to write and at least several months to go through the review process. By the time a paper appears in print, it may well be years from the time the work was first started. On several occasions I submitted papers with fairly recent references regarding various aspect of HIV's molecular biology, only to be answered with the criticism from a reviewer that some of these references were now "out of date." Sometimes the references were only two or three years old. I later discovered that this is a common occurrence in HIV research. Science, of course, is meant to be self-correcting, but it seems to be endemic in HIV research that, rather than continually building on an accumulating body of secure knowledge with only occasional missteps, the bulk of the structure gets knocked down every three to four years, replaced by yet another hypothesis, standard of care, or definition of what, exactly, AIDS really is. This new structure eventually gets knocked down in the same fashion.

Even more disturbing is the fact that HIV researchers continually claim that certain papers' results are out of date, yet have absolutely no hesitation in citing the entire body of scientific research on HIV as massive overwhelming evidence in favor of HIV. They can't have it both ways, yet this is exactly what they try to do.

There are further problems with the scientific method surrounding HIV/AIDS, which shall be dealt with in later chapters. Among the major problems are the circumstances surrounding the publication of the initial papers by Robert Gallo's group that appeared in the journal *Science* following the historic 1984 press conference (Crewdson 2003); continuing difficulties in demonstrating a cell-killing role for HIV; continuing problems with (and an apparent lack of interest in) properly isolating HIV as an exogenous retrovirus; and, possibly the worst of all, the astounding lack of specificity, standardization, and reproducibility of the HIV antibody and viral load tests.

The question still remains: How could science have gone so far astray? Why did the scientific community accept the HIV hypothesis so readily before any papers were published to support it? And how has this belief persisted so long despite results becoming "outdated" every few years? Why is there such disagreement between dissenting and orthodox scientists regarding the standards to which such crucial cornerstones as isolation procedures and antibody testing should adhere? How could scientists have so readily allowed their research to settle into one narrow, *unproven* channel of investigation? It's been over twenty years—surely, if there were something wrong with the theory, this fact would have been discovered. Corrective action would have been taken, and a "diverse portfolio of research direction" would have been explored (Strohman 1995).

The answer to these questions is twofold. The easy part of this answer is that, in point of fact, there are literally thousands of people, most of whom are credentialed doctors and scientists, who have insisted for many years that AIDS researchers have been entirely on the wrong path, or at the very least, have closed off legitimate lines of inquiry. There are many scientists who do not ascribe a pathogenic role to HIV at all, and yet more who contend that HIV alone is not the primary cause of AIDS. The latter include scientists such as Gordon Stewart, Robert Root-Bernstein, Joseph Sonnabend, Michael Lange, and Harry Rubin.

The most well known of the scientists who believe that HIV is harmless is undoubtedly Peter Duesberg, who is often cited as having been discredited despite the fact that there is no record of this "discrediting" anywhere in the scientific literature. By contrast, Duesberg has provided the most exhaustive critique to date of all the reasons HIV cannot possibly cause AIDS, and his criticisms have never been refuted anywhere in the peer-reviewed literature. The only "refutations" to Duesberg's arguments can be found in anonymously authored, non-peer-reviewed documents such as the NIH publication "The Evidence That HIV Causes AIDS" (NIH 2003) and the Durban Declaration (*Nature* 2000), both of which have been thoroughly rebutted themselves (Johnston et al. 2001a, Johnston et al. 2001b).

Perhaps just as telling as Duesberg's experience is the fact that the inventor of the polymerase chain reaction (PCR)—to date *the* method of choice for quantifying HIV viral load—Nobel laureate Kary Mullis, states categorically that quantitative PCR is invalid and should absolutely not be used for viral load testing.

The renowned expert electron microscopist Dr. Etienne de Harven became frustrated at the very onset of the HIV/AIDS paradigm. He shares the distinction of having produced the first electron micrograph of a retrovirus (the Friend leukemia virus). Since the beginning, de Harven has been skeptical not only that HIV could cause any disease but, further, that HIV has ever been properly isolated. He was at the time, and remains to this day, highly critical of all viral isolation procedures employed by HIV researchers. He contends that retrovirologists began using "shortcut," indirect methods not because of their increased efficiency, but because they couldn't get the results they wanted using the standard methods (de Harven 1998).

Dr. Rodney Richards, a chemist who worked for the company AmGen developing the first HIV antibody tests, contends that HIV has never been properly isolated and that the antibody tests are at best measuring a condition called *hypergammaglobulinemia,* a mouthful of a word that simply means having too many antibodies to too many things.

Dr. David Rasnick, who received his PhD in biochemistry for studying human proteases and holds several patents on protease inhibitors for various human diseases, has been critical of the HIV hypothesis since 1985. Furthermore, he strongly contends that the AIDS era has

rendered clinical-trial standards so low as to be nearly nonexistent.

John Lauritsen, a gay journalist and historian, has doubted the HIV hypothesis since its inception and has been extremely vocal about the incredible disservice a virus-only theory of AIDS has done to the gay community. His background in statistical survey research led to his extreme frustration with the lack of standards in epidemiological research and clinical trials. His exposé of the fraud and astonishing lack of standards that affect HIV clinical trials, in particular those that led to the initial approval of the drug AZT, are documented in his book *Poison by Prescription: The AZT Story*.

To put it plainly, HIV science has sold out to the epidemic of low standards that is infecting all of academic scientific research.

I have now been employed at the faculty level in university academia for four years, and prior to that I spent a cumulative total of four years doing graduate-level research. (The gap perceived by my having stated that I first began working on HIV ten years ago owes to the fact that following my Master's degree I spent two years working in industry.) I have also observed my father's employment circumstances and academic research experience as a professor in the physical sciences. Over the years, I have had plenty of opportunity to see exactly how research expectations affect the quality of the work we produce. It is clear to me that the pressure to obtain big government grants and to publish as many papers as possible is not necessarily helping the advancement of science. Rather, academics (young ones, in particular) are pressured to choose projects that can be completed quickly and easily, so they can increase their publication list as fast as possible. As a result, quality suffers.

This lowering of scientific standards and critical thinking has been apparent in many aspects of research for some time, and it is now beginning to infiltrate the classroom—in the textbooks and the undergraduate curriculum. It is germane at this point to indicate that many of the common arguments presented in response to the queries of HIV/AIDS skeptics are essentially some form of appeal to the use of low standards. (For example, "You don't need a reference that HIV causes AIDS," "The fact that HIV and AIDS are so well correlated indicates that it must be the cause," "HIV is a new virus, and new viruses will meet new standards," "Koch's postulates are outdated and don't apply in this day and age," "We don't need to worry about the actual infectious virus,

viral markers should suffice," or "Real scientists do experiments; they don't write review articles on the literature.") All of these observations are eloquently summed up by the mathematician Mark Craddock:

> Science is about making observations and trying to fit them into a theoretical framework. Having the theoretical framework allows us to make predictions about phenomena that we can then test. HIV "science" long ago set off on a different path ... People who ask simple, straightforward questions are labeled as loonies who are dangerous to public health. (Craddock 1996)

It is this decline in scientific standards that I point to when I am asked how so many scientists and doctors could be so wrong. Given the current research atmosphere, it was almost inevitable that a very significant scientific mistake was going to be made.

Three

SCIENCE BY CONSENSUS

IF THE AIDS establishment is so convinced of the validity of what they say, they should have no fear of a public, adjudicated debate between the major orthodox and dissenting scientists and the scrutiny of such a debate by the scientific community. Yet all the major AIDS researchers have avoided such a public debate, either by claiming that the "overwhelming scientific consensus" makes such a debate superfluous, or by saying that they are "too busy saving lives." Consider the result of the 1988 *Science* fight, to date the only such debate:

> After the "Policy Forum" appeared, Peter all but begged Dan to sanction another round, to no avail. And so just when it was getting good, the bout was declared a technical draw on an inexplicable and nonappealable decision of commissioner Koshland. There was never to be a rematch. The failure to extend the discussion in the pages of *Science* was significant. Most scientists have neither time nor inclination to follow specialist literature in fields outside their own. They depend, consequently, on journals like *Science* and *Nature* to tell them what is considered important. Having read, as best they could at the time, the arguments of the Policy Forum, and then seeing nothing more than vulgar anti-Duesberg editorials in the scientific press and worse in the popular media, even a partially persuaded nonspecialist could and would eventually concur with the "overwhelming evidence" of Team

> Virus, although it has become even less overwhelming now than it was in 1988. (Bialy 2004)

In place of public debate, politically motivated documents such as the Durban Declaration remain the establishment's standard response to dissenting voices. Even a cursory reading of this document reveals it to be a statement of faith, designed to divert attention from dissenters at the very moment when they were threatening to expose the orthodoxy in South Africa in 2000. The Durban Declaration was signed by over five thousand "PhD researchers," which would lead one to assume that the signatories had at least familiarized themselves with the orthodox and dissident literature on HIV/AIDS. This is entirely misleading, as an e-mail which went out as an attachment to the solicitation to sign the declaration included the following statement: "Many of you will say that HIV/AIDS is not your area, but by now you have heard enough of the arguments" (Bialy 2004). There is nothing scientific about the Durban Declaration—it is quite obviously a piece of propaganda somehow made authoritative by the thousands of signatures attached to it.

But science is not a democracy. As much as we might like to be able to mold our results and discoveries to fit hypotheses we would like to see proven, this is not how science should proceed. If our hypotheses fail to explain and predict, we should consider other ideas.

Until such time as a causal role for HIV in the etiology of AIDS is decisively proven or disproven, we can only rely on the available evidence for policy and public health decisions. Furthermore, this evidence should *not* be gathered and formulated within the framework of the HIV hypothesis. Due to the current practice of discrimination against HIV-positives, as well as the apparent lack of any benefit of anti-HIV drugs, a causal role should *not* be assumed until proven, but this is exactly what has happened.

In order to truly understand how the HIV/AIDS connection became nearly universally accepted *without question,* one must revisit the early days of AIDS and the discovery of HIV. I will discuss the changing face of AIDS itself in a later chapter, so for the time being, let us consider the original evidence for HIV as given by one of its discoverers.

The first scientific papers claiming a definite causal role for HIV were published May 5, 1984, in the esteemed journal *Science.* Robert Gallo, late of the NIH, and his chief secondary collaborator, Mikulas

Popovic, published four papers describing the detection of HIV in a proportion of AIDS patients and the details of how HIV was detected (Gallo et al. 1984). It is amazing that in the paper purporting to have *frequently* detected HIV in AIDS patients, actual HIV could be detected in only twenty-six out of seventy-two AIDS patients and in eighteen out of twenty-one pre-AIDS patients (pre-AIDS is an obsolete term that was used to describe a collection of symptoms including persistent fever, weight loss, and generalized lymphadenopathy). Gallo claimed that the reason for such a low frequency of detection (in spite of the title using the word *frequent*) was probably due to "sample contamination." It was later determined that his samples were indeed contaminated with mold, but one wonders how it is possible to come to such fundamental scientific conclusions using contaminated evidence!

Regardless, it seems strange that finding HIV in fewer than half of AIDS and pre-AIDS patients would ever qualify a virus for a pathogenic role, and indeed in the scientific papers Gallo's team avoided using any absolute terms to indicate causation. However, he *did* use such words in the press conference that was held before the publication of these papers. By the time the supporting papers were published, the lay press had all but declared HIV to be "the AIDS virus," and debate in the scientific arena was effectively stopped.

It was sometime in 1985 that HIV mysteriously went from "the virus associated with AIDS" to "the virus that causes AIDS," squelching debate in the scientific arena. What changed? What happened to make scientists come to such certainty? If you look at the actual papers, you'll see quite clearly that the answer is: nothing.

However, the AIDS machine kept going, and the questions of dissenting scientists were rarely acknowledged, let alone answered. One of the major problems with the HIV theory has always been that very little HIV can ever be found in the blood of AIDS patients and, in spite of claims to the contrary, there is no "massive covert infection" to be found in the lymph nodes, either (Embretson et al. 1993a, Pantaleo et al. 1993, Papadopulos-Eleopulos et al. 1995a). How could a virus appearing at concentrations of one to ten infectious particles per milliliter—and sometimes unable to be found at all (Gallo et al. 1984, Piatak et al. 1993)—be considered pathogenic?

In 1995, two papers were published in the journal *Nature* that supposedly answered this question once and for all (Ho et al. 1995, Wei

et al. 1995). These papers made popular the "hit hard, hit early" and Highly Active Antiretroviral Therapy (HAART) treatment strategies, as well as the concept of viral load testing as a measure of treatment success. One of the authors, David Ho, was named *Time* magazine's "Man of the Year" in 1996. The papers have since been thoroughly discredited on both immunological and mathematical grounds (Craddock 1996, Duesberg and Bialy 1996, Roederer 1998).

The mathematical models used in these papers claimed to show that HIV replicated furiously from day one, in contrast to earlier evidence suggesting it to be quite inactive (Embretson et al. 1993a, Embretson et al. 1993b). Even now, few people are aware that these conclusions were based on very poorly constructed mathematical models. If analyzed properly, the models predict the onset of AIDS within *weeks or months* after infection by HIV, *before* antiviral immunity as evidenced by the appearance of antibodies (Craddock 1996). To make matters worse, the statistical analyses were very poorly done and the graphs were presented in such a way as to lead the reader to believe something different from what the data supported. Yet these papers were lauded at the time as groundbreaking and even "brilliant," leading to a "new mathematical understanding of how the immune system works," according to the former editor of *Nature.* In an editorial appearing in the very same issue, Sir John Maddox, the editor in chief of *Nature,* presented the papers as evidence once and for all that this HIV hypothesis was correct and that dissidents, most particularly Peter Duesberg, were wrong. Maddox even went so far as to say that, in light of the evidence presented in the Ho/Wei papers, "Now may be the time for [the Duesbergs of the world] to recant."

This example illustrates a central flaw in the HIV theory. The vast majority of the literature I've read uses what is known as circular logic—you assume that something will happen, and then you mold the definitions, models, experiments, and results to support that conclusion. Craddock describes a typical example of circular logic in the Wei paper:

> They are trying to estimate viral production rates by measuring viral loads at different times and trying to fit the numbers to their formula for free virus. But if their formula is wrong, then their estimates for viral production will be wrong too. (Craddock 1996)

Such tactics, *by definition,* are excellent at maintaining the façade of a near-perfect correlation between HIV and AIDS, and of providing seemingly convincing explanations of HIV pathogenesis. But the resultant science does little to expand our actual understanding.

As has been indicated, the Ho/Wei papers have been essentially debunked by both establishment and dissenting researchers on biological as well as mathematical grounds; they are now acknowledged to be wrong by the scientific community, and it remains a mystery how they were ever able to pass peer review in the first place. It is often asked, "Why should we care at this point? Those papers are eleven years old; our understanding has progressed since then." The short answer is that viral load and combination therapies are used to this day, despite the fact that their original justification was based on these incorrect papers. Although current therapeutic regimens have been scaled back from the "hit hard, hit early" dogma that was popular ten years ago, the fact remains that a large population of people have been, and continue to be, treated on the basis of a theory that is fundamentally unsupportable.

Yet there is another answer to this question which is even more fundamental. It is a curious fact that few HIV researchers seem to be bothered by the events surrounding the Ho/Wei papers. You might imagine that people may care at this point because of concern over the integrity of science. You might imagine that people might feel an urge to discuss the manner in which the papers got published and whether other such mistakes have happened since that time. You might imagine that the failure of the peer-review process to detect such patently inept research would send off alarm bells within the HIV-research community.

You would be wrong.

HIV researchers know the Ho/Wei papers are wrong, yet they continue along the clinical path charted by the papers. They know that the quantitative use of PCR has never been validated, yet they continue to use viral load to make clinical decisions. They know that the history of HIV/AIDS is littered with documented cases of fraud, incompetence, and poor-quality research, yet they find it almost impossible to imagine that this could be happening at the present moment. They know their predictions have never panned out, yet they keep inventing mysterious mechanisms for HIV pathogenesis. They know many therapies

of the past are now acknowledged to be mistakes (AZT monotherapy, "hit hard, hit early"), yet they never imagine that their current therapies (the ever-growing list of combination therapies) might one day be acknowledged as mistakes themselves.

It's time for them to wake up.

Four

WHAT IS "AIDS"?

WHAT WE NOW know as "AIDS" bears little resemblance to the original cases of AIDS, as observed in New York City, Los Angeles, and San Francisco in 1981. The *original* definition of AIDS was based upon the observation of very rare opportunistic infections in previously healthy homosexual men. This list of opportunistic infections included Kaposi's sarcoma (although it is highly debatable whether KS has anything at all to do with immune suppression), *Pneumocystis carinii* pneumonia, cytomegalovirus (CMV) infection, and severe candidiasis (CDC 1986). The status "HIV-positive" had nothing to do with a diagnosis of AIDS prior to 1984, as HIV had yet to be identified.

It is worth noting that AIDS was *not* originally conceived as a specific disease. The definition was developed as a surveillance tool to assist clinicians and epidemiologists in identifying and controlling this strange new syndrome. It remains a matter largely hidden from the public that the first cases of AIDS did not suddenly arrive all at once, but rather were sought out by an assistant professor of immunology at UCLA Medical Center named Michael Gottlieb in 1981. After searching hospitals in Los Angeles for gay men suffering from opportunistic infections, he managed to find five (Brown 2001). Upon measuring their T-cells, a subset of the immune system, he found that in all five men they were depleted. What is quite curious about this discovery is that the technology to count T-cells had only just been perfected.

The acronym AIDS was introduced to replace the previously used pejorative term GRID (Gay-Related Immune Deficiency). Regardless, AIDS remains to this day a government-defined *syndrome* with, simultaneously, no specific clinical symptoms of its own yet a myriad of indirect illnesses and symptoms supposedly "caused" by the immune suppression—really quite a clever idea, since essentially everything is a symptom.

A clinical syndrome is useful when initially attempting to better understand what might be the causative agent of said syndrome. Plainly speaking, one designates a syndrome *before* one has any knowledge of the precise molecular mechanism of pathogenesis underlying the set of symptoms. Defining the clinical syndrome enables public health authorities and physicians to narrow the scope of their investigation to factors common to all those people in the epidemiological cohort among which the syndrome is manifest. A clinical syndrome is useful when it illuminates a causative agent of a disease, and this identification ideally has the effect of narrowing the scope of the clinical syndrome. That is, as we know more about what causes the syndrome, the number of symptoms under the syndrome umbrella should become smaller as we identify and throw out those that clearly do not fit the pattern.

AIDS is peculiar historically in that the definition of the syndrome actually became *more* expansive after the alleged causative agent was identified. This is contrary to all logic and counter to the reasoning that underlies the existence and usefulness of clinical syndromes in the first place. Moreover, these expansions make it very difficult to properly analyze epidemiological data. As the definition expanded and as it became more and more clear that HIV did not do at all what it was purported to do—that is, kill CD4+ T-cells by any detectable method—researchers began to invent more and more convoluted explanations for why their theory was correct. The logical, scientific thing to have done would have been to notice that their original disease designation did not accurately identify the causative agent or agents and, rather than changing the syndrome, throw out the supposed causative agent(s) and find one that explained the observations better. As we know, this has not happened.

Even a diagnosis of HIV-positive accompanied by no clinical symptoms at all can result in an individual's inclusion under the umbrella of AIDS, which flies in the face of the very reason for the designation

of a *syndrome* as a set of clinical *symptoms*. In another major lapse of logic, the classification of HIV-free AIDS, "Idiopathic CD4+ Lymphocytopenia" or ICL for short, was introduced in 1993 to actually *exclude* from the AIDS designation people who were free of any trace of HIV but still had *symptoms that would ordinarily result in their being classified as having the syndrome AIDS* (CDC 1993).

One important feature of the original classification of AIDS was its distinction as occurring in "previously healthy" homosexuals. While recent reports have cast doubt on the presumption that these original AIDS patients were, in fact, previously healthy at all (Cochrane 2004), this distinction raises the question of why hemophiliacs were *ever* considered AIDS patients. It is well known that the immune system does not operate normally in hemophiliacs, and that clotting factor (Factor VIII) therapy is itself immunosuppressive (Papadopulos-Eleopulos et al. 1995). Furthermore, hemophiliac AIDS patients experienced clinical disease presentations very distinct from those among other risk groups (Duesberg 1992); for example, candidiasis being very common but Kaposi's sarcoma virtually unseen.

The continual redefinitions of AIDS have resulted in a syndrome today whose clinical manifestation is very different from that seen in the original AIDS cases of the early 1980s. Some of the conditions listed are not even caused by immune deficiency, whereas others are clearly politically motivated, such as the 1993 inclusion of invasive cervical cancer. One can only presume that this disease was added to correct the disparity between male and female AIDS numbers, as there is little basis for including as "AIDS-defining" a cancer that is relatively common among women with no evidence of immune suppression whatsoever. After this addition, the media began issuing alarming statements such as "women are the fastest growing group of people with AIDS," conveniently neglecting to mention that the increases were simply small percentage differences and in some case actually indicated a *decrease* in overall incidence.

Perhaps the most egregious addition was the inclusion of low T-cell numbers as qualifying a person for an AIDS diagnosis. This change came about in 1993 and resulted in the number of reported AIDS cases more than doubling overnight. The rationale for this change was as follows: the immune suppression observed in AIDS patients could be quantified by counting the number of CD4+ T-cells per cubic millimeter

of blood. CD4+ cells are those cells for which HIV possesses a receptor, and it has been stated that the normal level of CD4+ T-cells per cubic millimeter of blood in a healthy individual is about one thousand. However, it is also well established that these counts can vary *dramatically* among healthy individuals and even within the same individual under conditions as severe as illness or drug use, or as mild as over-exercise or simply taking the measurements at different times of day (Beck et al. 1985, Carney et al. 1981, Des Jarlais et al. 1987). (CD4+ T-cell counts are subject to diurnal variation, similar to variations in appetite and energy level.)

Mathematically speaking, the figure one thousand cells per cubic millimeter is a *mean* value, an average. However, the amount of variance about that average is quite high, even among the general population. The studies that do exist regarding low T-cell counts in HIV-negative patients reveal that this laboratory anomaly is common among people with infectious mononucleosis, chronic illnesses other than HIV, and even among highly trained athletes (Verde et al. 1992). Furthermore, unusually high levels of T-cells do not generally indicate health but rather an inflammatory process in the body, such as allergies or an autoimmune condition that would cause the T-cell population to remain on "high alert."

Mainstream AIDS consensus generally holds that a CD4+ count under five hundred refers to definite immune suppression (whatever that means) and a CD4+ count under two hundred qualifies a person for a diagnosis of AIDS, even in the absence of clinical symptoms (CDC 1993). Another important aspect of the "low T-cell count AIDS" definition is that the figure two hundred refers not to an average count, nor even to the most recent T-cell count, but rather to the *lowest count ever measured.* The "low T-cell count AIDS" classification is significant in part because, given the dramatic variation possible in T-cell counts within a single person, one can almost guarantee that at some point an unmedicated person will experience a low T-cell count[3] if enough measurements are taken over time, *regardless of their HIV status.*

Beyond diagnosing hundreds of thousands of Americans[4] with a deadly disease on the basis of *no clinical disease at all,* the definition change served to create the illusion that new anti-HIV therapies were dramatically lowering the number of AIDS deaths in the early 1990s. The orthodoxy has done nothing to correct that impression. The ef-

fect of introducing an entire class of "healthy AIDS patients" was, first of all, to more than double the actual number of AIDS cases and, secondly, to drastically decrease the number of those patients who actually died. It doesn't take a trained pathologist to recognize that if a person is not experiencing any illness, they are much less likely to die of any illness any time soon. Thus, the proportion of AIDS cases that resulted in death experienced a large drop in 1993–94, which the orthodoxy and the mass media were more than happy to portray as decreased mortality thanks to protease inhibitors. However, protease inhibitors were not even generally available to AIDS patients until 1996, over two years after the decline in the death rate began. In spite of the fact that there is little or no official evidence that HIV protease inhibitors extend life or decrease morbidity, they have been hailed as magic cure-alls. All one has to do is examine the disclaimers on the packet inserts for any anti-HIV medication to realize that *none* of them have been shown to prolong life; that *all* of them cause debilitating side effects, some of which are indistinguishable from the symptoms of AIDS itself; that none of them, with the exception of AZT in the disastrous clinical trials whose fraud has been thoroughly documented (Lauritsen 1990), has been tested in placebo-controlled clinical trials; and that some of them have not even been tested in clinical trials at all.

The many stories of AIDS patients rising from their deathbeds to a renewal of good health and vitality are just that—stories. Such stories, however, have been interpreted as a major thorn in the side of the dissenting argument. Since the anti-HIV drugs stop AIDS, HIV must cause AIDS—right?

It is worth noting at the outset that there are still no significant studies that actually demonstrate the statement that "anti-HIV drugs stop AIDS." There is simply no evidence, and this conclusion appears to have been reached as a matter of pure faith rather than being based on any real solid science. The majority of evidence supporting the statement that "anti-HIV drugs stop AIDS" falls into two broad categories: people who were never sick in the first place and who still aren't sick,[5] and people who were really quite ill indeed and experienced some improvement following the initiation of therapy (Atzori et al. 2000). A third category consists of people who die too quickly from adverse effects of the drugs to ever develop AIDS (Anastos et al. 2002, Heal Toronto 2002, Hosein 2002).

It constantly amazes me that HIV researchers, and HIV-drug manufacturers, can honestly and with a straight face state that since someone *who was healthy when they started therapy* happened to stay healthy for some time on the drugs, that this is some sort of credit to the medications. Since the new dosages of nucleoside analogue drugs and protease inhibitors are much lower than the massive doses of AZT that were given in the late 1980s, and that undoubtedly caused the deaths of many, it stands to reason that patients will not get sick *because of the drugs themselves* quite as quickly as they did fifteen years ago. So healthy people stay healthy for a while, and this is credited to the drugs—but there is no evidence to say that they would not have remained healthy even if they never took any medication at all. This is due to the fact that clinical trials of "anti-HIV" drugs rarely if ever use placebo controls, so there is no way to determine whether, for example, nevirapine is better than nothing. Trials are always in the form "AZT vs. nevirapine," and activists and researchers alike defend this fundamentally unscientific notion by saying that denying toxic drugs to HIV-positives is "unethical."

On the other hand, a person who is really quite sick and is experiencing opportunistic infections prior to beginning a regimen of antiretroviral therapy[6] is likely to experience a temporary reprieve for some very logical reasons. Reverse transcriptase inhibitors are nonspecific cell killers and attack all growing cells. They will naturally attack those cells that are dividing the fastest, such as the bacteria and fungi that are causing an acute illness. As a result, opportunistic infections are fairly efficiently killed by these drugs. The same is true for the protease inhibitors. Although these inhibitors are claimed to be specific to the HIV proteases, they are not *completely* specific and in the doses taken by HIV-positives they have the capacity to interfere with many non-HIV proteases. These include the proteases required for replication of bacteria, viruses and other microbes. As one example, it has been demonstrated that protease inhibitors appear to be particularly effective at controlling *Candida* (Cassone et al. 1999) and *Pneumocystis* (Atzori et al. 2000).

Putting aside all potential nonspecific benefits of anti-HIV drugs, the fact remains that the risks appear to far outweigh these supposed benefits. Simply consider that the annual mortality rate of North American HIV-positives who are treated with anti-HIV drugs—between 6.7

and 8.8 percent—is much higher than the estimated 1 to 2 percent global mortality rate of HIV-positives *if all AIDS cases were fatal in a given year* (Duesberg et al. 2003b).

It should also give us pause to note that if these drugs were truly HIV-specific, then one drug should suffice, rather than combinations of three or four of them. Mainstream researchers argue that the high mutation rate of HIV necessitates that we "confuse" or "trick" the virus with many different medications. However, this is a ridiculous assertion, as it is simply impossible for any retroviral entity to mutate that much and remain viable. (The influenza viruses, by contrast, have a segmented chromosome and are capable of mutating by recombination, or rearrangement of their genes. HIV, like other retroviruses, has only approximately nine thousand nucleotides and as such is incapable of mutation by any method other than transcription error. Such transcription errors would be expected to quickly lead to mutations that render new virus particles noninfectious.) Also, if these drugs were truly HIV-specific, much smaller doses would be necessary than those that are currently prescribed.

A rather curious addition was made to the list of AIDS-defining diseases recently. It is named Immune Reconstitution Syndrome (IRIS or simply IRS), and it consists of the development of opportunistic infections while being treated with antiretroviral therapy. Official dogma states that as the immune system is gaining strength, it becomes confused and this enables AIDS-defining opportunistic infections to take hold. In reality, it seems to be just another attempt to explain away the fact that clearly the medications are not working as they were intended—just like the invention of ICL in 1993 was a convenient way to sweep all the HIV-free AIDS cases under the rug.

Consider also that the leading cause of death among medicated HIV-positives is no longer even an AIDS-defining disease at all, but liver failure, a well-documented adverse effect of protease inhibitors. Amazingly, some people seem to think that's a good thing, as evidenced by the following comment by a blogger on LibertyPost.org, in response to an article I wrote (Culshaw 2006b):

> And worse, she claims that protease inhibitors are killing HIV patients, "And the leading cause of death in HIV-positives in the last few years has been liver failure, not an AIDS-defining disease in any way,

> but rather an acknowledged side effect of protease inhibitors, which asymptomatic individuals take in massive daily doses, for years," when that's exactly what you would hope for (mortality drastically decreasing to the point that more deaths were the result of side effects) if protease inhibitors were in fact EFFECTIVE treatment for AIDS. (Posted March 3, 2006)

Another important—and really very shocking—fact is that in some states and countries, you don't have to die of an AIDS-defining illness to die of AIDS. In Massachusetts, for example, all deaths among HIV-positives are counted as AIDS deaths, and this happens if the person died of liver failure, a heart attack, suicide, drowning, CMV infection, or a car accident, *or anything else,* AIDS-related or not (Massachusetts Department of Public Health 2002).

If it weren't bad enough that perfectly healthy asymptomatic individuals who just happen to test positive for some arguably nonspecific antibodies are pressured to begin regimens of just such drugs without being given adequate information about side effects, infants and children often have no choice at all in the matter. At least adults have the opportunity to decline such medicine and are capable of gathering sufficient information to make an informed decision—although admittedly, much of the vital information regarding toxicity is not readily available from mainstream sources. Infants born to HIV-positive mothers are in many states forced to undergo antiretroviral therapy, and since only a few drugs have been approved for children, the drugs administered are usually among the most toxic, AZT and nevirapine being foremost. Oftentimes this drug regimen begins before the baby is born, in certain cases against the wishes of the mother, and continues throughout childhood. A particularly shocking example of the lengths to which HIV-treatment activists will go to ensure no child left behind in their quest to medicate at any cost is the forced drug trials that HIV-positive children underwent in New York City's Incarnation Children's Center (ICC) recently. Investigative journalist Liam Scheff uncovered the fact that children were being force-fed HIV drugs against their will while in the custody of ICC and that if the children refused to take drugs orally, a tube was inserted into their stomach to render any treatment noncompliance impossible (Scheff 2004). This atrocity was further examined

in a BBC documentary, *Guinea Pig Kids,* which was based on Scheff's work and aired in Europe, but not in the U.S.

New CDC recommendations encourage *all* pregnant women to be tested for HIV antibodies, regardless of risk. This may seem a commonsense guideline until the evidence is examined more closely. One of the immediate problems is that pregnancy itself is an admitted common cause of false positives on the HIV test (Cordes 1995, Ng 1991, Proffitt et al. 1993, Steckelberg et al. 1988, Voevodin 1992), so there is no way of knowing if the treatment recommendations that directly follow a positive HIV result are appropriate, *even if HIV were the cause of AIDS.* This fact alone should ring alarm bells regarding the wisdom of medicating pregnant mothers with toxic antiviral drugs. Most people are aware that pregnant women are encouraged to avoid any potentially toxic substance, including caffeine, alcohol, painkillers, and antibiotics. The lessons of the Thalidomide disaster ought to have been well learned, but apparently the risk of giving birth to a child carrying HIV antibodies is greater than that of any deformity, cancer, or even stillbirth.

Current treatment protocol for pregnant women diagnosed HIV-positive is to administer a course of AZT or some other combination of anti-HIV drugs from the second trimester of pregnancy through delivery, which must be by Cesarean section, as vaginal delivery is considered too risky. The baby is then tested for HIV antibodies and given AZT as well. Often babies who test HIV-positive are simply harboring what are called ghost antibodies inherited from the mother. In the case of such ghost antibodies, they will disappear within nine to eighteen months of birth *in the absence of antiviral medication.* It is estimated that more than half of HIV-positive babies revert to negative. The wisdom of providing toxic drugs to these children is highly debatable no matter one's position on HIV and AIDS.

AZT is by no means the only drug available to treat HIV, but it is certainly one of the most toxic, and its symptoms include wasting, anemia, bone marrow suppression, and fulminating white-blood-cell death, making disease from AZT virtually indistinguishable from AIDS itself. What is particularly significant about AZT is that it is among the most common of the drugs approved to prevent mother-to-child transmission of HIV in the U.S. In other countries, nevirapine has been approved in single-dose use, presumably for administration during labor,

but it has not been approved for this purpose in the U.S., and its use has been implicated in the high-profile death of at least one mother, Joyce Ann Hafford, who died from nevirapine toxicity within days of giving birth (Farber 2006a).

The treatment of HIV-positive expectant mothers and children remains a matter of much debate, although media reports seem to insist that any fears about mutagenic or teratogenic effects must be quelled in the face of the far greater threat of delivering an HIV-positive child, or of having a child die of AIDS.

The question is: what is an AIDS death in a child? Certainly children can die of *Pneumocystis carinii* pneumonia (PCP), or of candidiasis, or any of the other traditional AIDS-defining diseases (though Kaposi's sarcoma is mysteriously absent in children with AIDS as it is in all non-homosexual risk groups). However, one disease that has been added to the AIDS definition, only for children, is "recurrent bacterial infections" (CDC 1993).

No number is given for what constitutes "recurrent." Putting aside for the moment the fact that many children suffer from recurrent bacterial infections, a more disturbing question arises. Why is this condition *not* AIDS-defining for adults? The traditional definition of an immune deficiency is the inability to fight a multitude of common bacterial infections, but this is absent in AIDS patients. The diseases that AIDS patients succumb to are commonly fungal infections such as *Pneumocystis* and *Candida,* not multiple bacterial infections at all, leading one to question whether AIDS is truly an immune deficiency in the *traditional* sense.

The chief reasons it was initially believed that AIDS is a standard immune deficiency are twofold: patients were getting sick with diseases that were previously rare in "healthy" individuals, and these patients, when tested, showed a significant depletion in the CD4+ subset of the T-cells of their immune system. A decline in CD4+ cells was purported to be the hallmark of the disease and a general barometer of the overall health of the immune system. It was for this reason that scientists focused on searching for a pathogen that was capable of infecting and damaging these very cells.

But what was also known from the beginnings of AIDS—though bizarrely, not investigated to nearly the extent that CD4+ cells have been investigated—was that AIDS patients suffered disruptions in many

subsets of their blood cells. Virtually all of these patients had elevated levels of many different types of antibodies, indicating that something had gone wrong with the "antibody arm" of the immune system. (The existence of such an unusually high level of antibodies, by the way, has been suggested as a serious confounding factor in the alleged specificity of the HIV antibody tests, and this topic will be discussed further in a later chapter.)

Significant understanding as to why AIDS patients, and to a lesser extent, a nontrivial proportion of HIV-positives, experienced highly nontraditional immune deficiencies became possible in the late 1980s, when the subset of CD4+ ("helper") T-cells was further differentiated into two subtypes, Th1 and Th2 (Mossman and Coffman 1989). The Th1 subset controls what is referred to as "cell-mediated" immunity, and is directed toward *intracellular* pathogens, such as fungi and yeasts. A depletion in the Th1 subset results in the types of opportunistic infections seen in AIDS patients. The Th2 subset is associated with antibody production and "humoral" immunity, and as such effectively directs against mainly bacterial infections. Typically seen in AIDS patients is a reduction in the Th1 subset and an increase in the Th2 subset, leading to a preponderance of opportunistic infections but very few, if any, bacterial infections. Also, an excess in the Th2 subset inevitably leads to excessive antibody production (Mossman and Coffman 1989).

Further support for what is called the Th1/Th2 switch can be found by considering where the different subsets of T-cells "live." Th1 cells are found primarily in the bloodstream, whereas Th2 cells remain in the bone marrow and the lymph nodes. Finding a low T-cell count in the bloodstream, therefore, may not mean that any depletion at all has occurred in the *total* CD4+ cell population, but rather that levels of Th1 cells are lowered and those of Th2 cells elevated. Indeed, this explains perfectly the observation that traditional bacterial immune deficiency diseases are typically not seen in AIDS patients.

Another curiosity is the fact that markers for HIV expression have only been found among the Th2 cell types, and not among the Th1 cells (Maggi et al. 1994). This presents a question whose answer should be very interesting indeed: Why does HIV apparently only infect cells whose growth actually *increases* following infection?

It is currently popular to speak not of "AIDS" but of "HIV disease," a final linguistic alteration that cements the circularly derived

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. Spe-

cifically, 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.

Six

WHY THERE IS NO EVIDENCE THAT HIV CAUSES AIDS

THE ASTOUNDING LACK of evidence supporting the HIV paradigm can be summarized in both biological and epidemiological terms. For the sake of simplicity, I will present a summary of the major biological criticisms first and will follow with the epidemiological inconsistencies. Also, please notice that there is considerable overlap between this chapter and the previous one, since many of the reasons to doubt the validity of the HIV tests also cast doubt on the ability of HIV to cause any disease.

AIDS is said to be caused by a dramatic loss of the immune system's T-cells, said loss being presumably caused by HIV. However, as recently as March of this year, longtime HIV researcher Dr. Zvi Grossman stated, in a paper published in *Nature Medicine* that examined the various hypotheses of HIV-mediated T-cell depletion, and found them all wanting: "The pathogenic and physiologic processes leading to AIDS remain a conundrum" (Grossman et al. 2006).

Why is it that still no one understands the dynamics of the fundamental disease process—that is, how are T-cells actually killed by HIV? Early models assumed that HIV killed T-cells directly, by what is referred to as lysis. An infected cell lyses, or bursts, when the internal

viral burden is so high that it can no longer be contained, just like your grocery bag breaks when it's too full. This is the accepted mechanism of pathogenesis for virtually all other pathogenic viruses. But it became clear that HIV did *not* kill T-cells in this manner, and this concept was abandoned, to be replaced by various other ones, each of which resulted in very different models and, therefore, different predictions (Irwin 2001). Which model was *correct* was never clear.

There still is no consensus as to how HIV kills T-cells, although the notion of apoptosis, also known as programmed cell death, has become popular despite no real evidence of its occurrence. In laboratory experiments where apoptosis has been demonstrated in HIV-infected cell cultures, apoptosis is detected only after the addition of powerful chemical stimulants called mitogens. However, *uninfected* cultures that have been mitogenically stimulated also demonstrate apoptosis (Papadopulos-Eleopulos et al. 1995a). It is claimed that the presence of the envelope protein gp120 and its oligomer, gp41, prime CD4+ T-cells early on for a future process of programmed cell death. However, it is known that neither gp120 nor gp41 are specific to HIV, and gp41 is presumed by Luc Montagnier's group to be cellular actin, a ubiquitous component of all cells. The conundrum of how proteins that are present in normal cells could possibly induce apoptosis only in the cells of "HIV-positive" individuals has never been resolved. Furthermore, such apoptosis-inducing proteins as gp120, tat, and nef are present in other retroviruses including human endogenous retroviruses, yet these retroviruses are not thought to induce apoptosis to anywhere near the extent that HIV supposedly does.

HIV is possibly the most studied microbe in history—certainly it is the best funded—yet there is still no agreed-upon mechanism of pathogenesis. There are good reasons to believe that HIV is not pathogenic at all. One important reason is the fundamental nature of retroviruses themselves.

Retroviruses were popular in the 1970s "War on Cancer" research program as candidates for cancer-causing viruses because, unlike most pathogenic viruses, retroviruses do not kill the cells they infect. In fact, in some instances it was found that the cells infected by retroviruses actually grew at a faster than normal rate. However, despite findings that some retroviruses did seem to be associated with tumors in animals, the quest to find a cancer-causing retrovirus has been a failure (Duesberg 1987).

A retrovirus is nothing more than RNA with an outer protein shell. The shell enables it to bind to cells of the type it infects, and once it gains entry, the outer coating disappears and the RNA is transcribed to DNA and incorporated *as provirus* into the host cell's own genome. It is for this reason that retroviruses are called enveloped viruses, and it is also the reason that it is very difficult to distinguish between *exogenous* retroviruses (those that originate outside the body from a foreign invader) and *endogenous* retroviruses (those that are manufactured from our own retroviral-like genetic sequences[11] under conditions of cellular stress, including disease).

It should be clear why an enveloped virus would not kill its host cell, as it is completely dependent on the host to replicate. Instead, replication is accomplished by means of new viral particles budding from the host cell's membrane. However, this productivity is low in the case of HIV, as only approximately one in ten thousand CD4+ T-cells is ever productively infected (Duesberg 1989), which is why finding actual HIV in humans is extraordinarily difficult. It has been proposed that free HIV is not responsible for the vast majority of cellular infection, and instead that direct cell-to-cell infection is the dominant mode of transmission within the host.

If HIV really does somehow cause the destruction of an extraordinary number of CD4+ T-cells, it would be a most unorthodox virus indeed, as it would have the distinction of being the first retrovirus that caused cell destruction outside of the laboratory. (Note that a "retrovirus" is a subset of the class of "RNA viruses." I have been asked numerous times why it is that RNA viruses such as Ebola and hantavirus can cause disease, but the RNA virus HIV does not. The answer is that these are quite simply *not* the same type of virus. RNA-containing viruses that are not retroviruses are not enveloped and can indeed induce lysis, killing their host cells in the same way that "traditional" DNA-containing viruses do.)

Another conundrum is the difficulty in culturing active HIV from AIDS patients at all—and this doesn't even consider the real difficulties encountered in properly isolating HIV at all, a feat many researchers argue has never been accomplished. As has been discussed in previous chapters, before the publication of the Ho/Wei models in 1995, a major thorn in the side of the HIV hypothesis was that negligible amounts of virus were ever to be found—whether one was well, sick, or dying from

AIDS, virus titers (as measured by culturing, which generally involves at best detection of reverse transcription, or of p24, or of retroviral-like particles, none of which is specific to HIV) were so low, at about one viral particle per milliliter or even zero, as to be unable to explain HIV's allegedly ferocious pathogenesis.

The farcical concept of viral load was invented to create the illusion of correcting this embarrassing fact. However, as we discussed, viral load does not correlate with infectious viruses and thus, even according to HIV theory, cannot possibly have anything to do with illness. To best illustrate the ridiculous level of illogic some HIV scientists can display when confronted with these conundrums, I refer to the experience that Dr. David Rasnick had at a Gordon Conference on AIDS in 1997, which he attended to present a poster that disputed the hypothesis that anti-HIV drugs stop working because of the high rate of mutation of HIV.

> In the discussion period of Mellors's lecture, I decided to return to the questions that I'd wanted Markowitz to answer, about the meaning of "viral load." After all, that was the heart of the matter: Mellors's call to discard clinical endpoints [e.g. to consider only surrogate markers such as viral loads as measures of treatment success, disregarding clinical health] was only as valid as the "viral load" figures with which he wished to replace them.
>
> For starters, I wanted to compare his answers to Markowitz's. So I repeated my question about the relation between "viral load" and infectious doses. Mellors responded by proclaiming, "Viral load has nothing to do with infectivity!"
>
> Ah-ha! Now I had a second HIV big shot admitting that the "viral load" figures did not indicate infectious HIV.
>
> Assuming that "viral load" testing accurately counted HIV, and that infectious dose testing accurately counted infectious HIV, I offered my 99.8 percent figure from the Ho/Markowitz paper as the fraction of circulating HIV that was non-infectious.
>
> Non-infectious HIV, then, is the source of RNA and proteins—including protease—from which the genetics and other characteristics of HIV are derived.
>
> He agreed. (How could he not?)

Now I had him. Since non-infectious viruses have no conceivable clinical relevancy, then neither could any data derived from them.

What's the significance of all the non-infectious HIV? I asked. I had no idea how he could work himself out of this corner, but even I was stunned by his response: "The non-infectious particles [HIV] are pathogenic."

Now here was a first. I don't think that anybody's ever gone on record before proposing that non-infectious virus could cause disease.

I sat there flabbergasted, noticing the murmur that had broken out. In my astonished state I realized there was nothing else to be said.

In the meantime, the session was declared over, the time allotted for discussion having been exhausted by my cross examination, with no one else having had time to pose questions.

My God, I thought. Talk about a rich source of research opportunity. The pathogenicity of non-infectious viruses. Anybody familiar with the antibody response and the premise of vaccinations can appreciate the revolutionary nature (and implausibility) of this idea.

My sense is that the audience did, given the intense murmuring, which continued even after the lecture had been dismissed. On the way out of the room an Indian scientist grabbed my arm and asked, "Did you hear that?"

Indeed I had. AIDS was caused by a deadly army of viral corpses. (Rasnick 1997)

More perplexing is the fact that no two identical HIV genomes have ever been obtained *in vivo*—even from the same person (Papadopulos-Eleopulos et al. 1998). This observation has led some researchers to consider that HIV is a "quasi-species" of virus. Others claim that this genetic diversity is the result of HIV's alleged high mutation rate, unprecedented in the history of viruses. Another disturbing possibility that arises is that much of the genetic material attributed to HIV is in fact DNA or RNA from decaying cells, which are capable of producing retroviral-like particles when stressed or dying in large quantities. Human beings are filled with such *endogenous* retroviruses, which are expressed under conditions of cellular stress and decay. Whether one believes that this stress exacerbates the expression of an exogenous retrovirus HIV, or that it is an endogenous, noninfectious retrovirus, or

simply a "viral mirage," this information casts serious doubt on the validity of either viral load testing or of using either reverse transcriptase or retroviral-like particles or genetic sequences as markers for HIV.

The epidemiology of HIV and AIDS is puzzling and unclear as well. In spite of the fact that AIDS cases increased rapidly from their initial observation in the early 1980s and reached a peak in 1993 before declining rapidly, the number of HIV-positive individuals in the U.S. has remained virtually constant at one million since the advent of widespread HIV antibody testing, as discussed in the Introduction. Again, this cannot be due to anti-HIV therapy, since the annual mortality rate of North American HIV-positives who are treated with anti-HIV drugs is much higher, at a value somewhere between 6.7 and 8.8 percent, than would be the approximately 1 to 2 percent global mortality rate of HIV-positives, assuming all AIDS cases were fatal in a given year. This fact, as well as the disparities between HIV and AIDS in men and women, motivated Henry Bauer, emeritus dean of science at Virginia Polytechnic Institute and State University, to perform a comprehensive analysis of the CDC's own data from 1985 to the present day (Bauer 2005, Bauer 2006a, Bauer 2006b). What he found was shocking.

In this devastating analysis, Bauer points out many of the epidemiological aspects of HIV that are utterly incompatible with the hypothesis that it causes AIDS.

For instance, HIV has been present everywhere in the U.S. in every population tested, including repeat blood donors and military recruits, at a virtually constant rate since testing began in 1985. It is deeply confusing that a virus thought to have been brought to the AIDS epicenters of New York City, San Francisco, and Los Angeles in the early 1970s could possibly have spread so rapidly at first, yet have stopped spreading completely as soon as testing began.

But the centerpiece of what he noticed was that positive HIV tests show an astonishing regularity across lines of age, gender, race, and geographic location utterly unlike what one would expect from a sexually transmitted infection. Although there was a correlation between regions with high AIDS incidence and those with high HIV prevalence, AIDS incidence was nowhere near as strong an indicator for HIV-positivity as were other variables. The strongest correlate was race, with the shocking fact that black teenagers from places with very low AIDS incidence

were more than twice as likely to test HIV-positive as the average non-black teenager from places of high AIDS incidence.

Bauer shows that according to official CDC data compiled from testing facilities such as blood banks, prisons, military and job corps testing sites, hospitals, STD clinics, and more, the frequency of positive HIV tests follows the identical distribution over age and race in every group tested. This includes the lowest-risk groups—repeat blood donors and members of the Marine Corps. In every category, without exception, the frequency of positive HIV tests declines from birth into the teen years, increases steadily into middle age, and then begins to fall. The prevalence is nowhere zero, even among groups presumed to be at no risk of infection.

Furthermore, the HIV prevalence ratios in all groups could be categorized by race as follows: from lowest to highest incidence, HIV occurred in the racial categories Asian,[12] Caucasian, Native American, Hispanic, and Black.

In summary, accumulated data from years of testing indicate that the levels of HIV in the population are unchanging geographically—always higher in the East and the South than in the West and the Midwest, unchanging in number, and far too consistent over racial groups and gender to be consistent with the irregularities of AIDS in the population. All the epidemiological evidence to date strongly indicates that whatever testing HIV-positive signifies, it clearly is not a reliable indicator of the risk of ever developing AIDS.

Seven

SOCIOLOGICAL IMPLICATIONS OF AIDS

ON APRIL 23, 1984, the "probable cause of AIDS" had been identified and was announced to the world via press conference. Robert Gallo, PhD, of the NIH, and Margaret Heckler, secretary of Health and Human Services for the Reagan Administration, presented this information, which was then broadcast the world over and reported on extensively in newspapers and magazines for weeks, months, years afterward.

The story of AIDS began long before the fateful 1984 press conference. At least as early as mid-1980, reports began to surface of a small group of gay men who were dying from a strange pneumonia and a hitherto rare—and *not* previously fatal—form of skin cancer called Kaposi's sarcoma. The first five men with AIDS were patients of Michael Gottlieb who used a new technology that enabled technicians to count not just the total number of white blood cells a patient has but the number of each *subset* of T-cells. Using this new technology—which coincidentally came into existence and was patented at the beginning of the AIDS era—Gottlieb was able to determine that these men suffered from an unusually low number of the white blood cell subset known as helper T-cells.

The hunt for an agent capable of selectively targeting and depleting this subset of white blood cells was on. In the early days, all manner of

infectious and noninfectious causes were considered, but the dogged determination of the retrovirus hunters encouraged some zealous scientists to consider that the target was probably a retrovirus capable of entering the CD4+ T-cells. Robert Gallo had previously discovered two other human retroviruses, HTLV-I and HTLV-II, that were tropic for CD4+ T-cells, so when he found evidence implicating a new retrovirus in some AIDS patients (temporarily christened HTLV-III and now and forever known to the world as HIV), all questions about causation came to an abrupt halt. At the time, the retrovirus seemed to supply all the answers we needed, and thus began work on a cure and a vaccine that was promised by 1986.

Twenty years after the cure was promised to have arrived, there is none, and there likely never will be a vaccine. A massive industry has been built around T-cell testing, viral load testing, antibody testing, and drug development. Drugs have been developed to lower viral load and drugs have been developed to alleviate the sometimes horrific effects of the primary drugs. An entire plastic surgery industry has been put into place to mask the loss and redistribution of fat caused by the drugs.

What good has come of this? How many peoples' lives have actually been *improved* by an HIV-positive diagnosis? Who is better off from this campaign of psychological terror?

The nails in the coffin of the dead HIV/AIDS paradigm have been hammered long ago, by a long list of scientists and medical researchers. The AIDS orthodoxy's only counters to the points made and the questions raised consist of *ad hominem* attacks including use of the term "denialist," as well as stating that dissenting views have long since been "discredited," without any reference to exactly *where* these views have been discredited. Unfortunately, words are powerful, and personal attacks are very effective at silencing people. Even a cursory examination of the literature reveals that the "discrediting" of dissenting views takes place entirely within non-peer-reviewed outlets such as the anonymously authored NIH/NIAID document, "The Evidence That HIV Causes AIDS," and the Durban Declaration—both of which have been thoroughly refuted.

The persistence of this intellectually bankrupt theory in the public mind is thanks entirely to the campaign of fear, discrimination, and terror that has been waged aggressively by a powerful group of people whose sole motivation was and is behavior control. Yes, the money

and the vast interests of the pharmaceutical industry and government-funded scientists are important, but the seeds of the HIV/AIDS hypothesis are sowed with fear. If the fear were to end, the myth would end.

To understand the sociological motivations behind the HIV/AIDS paradigm, one must understand the racism and homophobia that has persisted in society for centuries. It is only very recently in the timeline of history that gays and blacks have been accorded equal rights under the law—rights that Caucasians and heterosexuals have enjoyed since time immemorial. To understand the racism and homophobia behind the very definition of AIDS, one only needs to consider the official party line: AIDS infected humans when Africans consumed or did strange things with monkeys, and it has been spread throughout the world by gay men and sexually promiscuous, prostitute-visiting black Africans.

This ridiculous concept is utterly intellectually bankrupt—the evidence for an African origin for HIV, much less AIDS, is slim indeed and based entirely on the hypothesis that Africans have been doing strange things with monkeys which magically permitted not one but two distinct retroviruses, HIV-1 and HIV-2, to somehow jump to humans and start causing massive immune deficiency the likes of which has never before been caused by a single—let alone two distinct—infectious agent. For this to be true, these two new retroviruses must be pretty new in monkeys, too, since nothing has changed regarding how Africans relate to monkeys in the last forty or so years, and logically, such a zoonotic jump, if it were possible, should have happened long ago. For this to be true, AIDS ought to have existed in Africa *significantly before* it existed in New York City, Los Angeles, and San Francisco, rather than *after* (1983), which is what happened.

Scientists jumped to these conclusions because they did not have any hard evidence. The first five men with AIDS were not sexually involved with one another, so why was a sexually transmitted cause considered to be so likely? And of Gallo's cohort of seventy-two homosexuals with AIDS, only twenty-six had any trace of HIV. Yet somehow HIV (and therefore AIDS) was considered sexually transmittable. This conclusion was arrived at not by the traditional method of proving an infection is indeed an STI, which involves microbial isolation and contact tracing, but rather by simply assuming sexual transmission.

Laboratory studies of "HIV," in which researchers do experiments showing things like "HIV" not being able to penetrate latex or "HIV" being able to infect monkeys when rectally injected, do not use HIV particles at all, but rather molecular biology experiments consisting of combinations of proteins that trigger an antibody reaction. So how do we know anything about what HIV really does, where it came from, and even what it is?

The answer is: we don't, anymore than we did back in 1984. Despite the fact that other viruses (cytomegalovirus and herpes virus, to give just two examples) were far more prevalent in AIDS patients than HIV ever was, the HIV train started rolling and hasn't lost momentum since. Would this have happened if the first five AIDS patients had been heterosexuals in the prime of their lives?

Many of the biggest crimes committed by the AIDS orthodoxy are psychosocial and not medical at all. People far more well versed than me have exhaustively exposed the level of iatrogenic harm that has been done to HIV-positive individuals by anti-HIV medications, and these arguments remain relevant to this day (Brink 2000, Duesberg 1996, Lauritsen 1990). However, I believe that more attention needs to be given to the discrimination that has been leveled against these people, as well as the death-cult mentality surrounding "HIV-positivity."

It is absolutely stunning that the notion that HIV=AIDS=DEATH has been so firmly entrenched in the public mindset and has been perpetuated by medical personnel and public health "educators." Virtually every other disease known to man is accompanied by some hope of recovery—not so with AIDS.

From the mail that I have received in response to articles published on Lew Rockwell's website, I can attest to the fact that there are many people living healthy lives twenty years after an HIV diagnosis and, furthermore, that there are a significant number of people healthy fifteen or twenty years after an actual *AIDS* diagnosis, without benefit of anti-HIV drugs. Why then does hope not ring eternal for AIDS patients?

Currently, "HIV disease" is classified into four stages, from asymptomatic to AIDS. "Stage 4 HIV disease" refers to a CD4+ T-cell count of less than two hundred or the presence of opportunistic infections (CDC 1993). Remarkably, it is stated in plain language that once an individual has been classified as Stage 4, they can never return to any of the lower stages, even if their CD4+ count rebounds or they recover

from illness. This is remarkable and totally unprecedented in the history of medicine. A cancer patient is allowed to recover, but an AIDS patient (whatever that means) can never recover, *by definition,* even if their health returns to normal.

The psychological effects of an HIV diagnosis are profound. Further, the psychological effects of the *fear* of an HIV diagnosis are often made manifest in physical symptoms that mimic AIDS—so much so that the terms "AIDS-phobia" and "AFRAIDS" were coined to describe a syndrome. This syndrome consists of symptoms such as weight loss, gastrointestinal disturbances, night sweats, and flu-like ailments, and it occurs in people who have had recent close contact with people they suspected might be HIV-positive—*even though the "AFRAIDS" sufferer repeatedly tested negative.*

The discrimination leveled against those given an HIV-positive diagnosis has reached a level not seen since leprosy was common. HIV-positives are the modern equivalent of lepers (and in Cuba, where they are quarantined, are even treated as such), despite the fact that all mainstream evidence reveals the infectivity of HIV, even in intimate contact, to be so negligible as to be incapable of sustaining any sort of epidemic. Although education campaigns commonly claim that "we're all at risk" and "AIDS does not discriminate," most Americans are well aware that people really do believe AIDS does discriminate.

Perhaps the most illustrative example of the twisted way in which HIV is viewed as the perpetrator of all evil is the ongoing story of Christine Maggiore and Eliza Jane Scovill. Christine was diagnosed HIV-positive in 1992 and volunteered for several years as an "AIDS educator" before she began to question the basis of her diagnosis. Eventually, any meaning it might have held for her was gone and she founded Alive and Well AIDS Alternatives, a support group for HIV-positive individuals who did not want to bow to conventional HIV/AIDS theories.

Christine married filmmaker Robin Scovill and gave birth to two healthy children, Charlie and Eliza Jane. Indeed, she and her family have remained so healthy that many proponents of the HIV/AIDS paradigm have put forth the hypothesis that Christine is not really HIV-positive. As Jeanne Bergman said in the *New York Press:*

> False negative tests are extremely rare, while false positives are much more common, though infrequent. This fact and all the other evidence

> available strongly indicate that Maggiore was never infected by HIV ... Most people would be thrilled to know they were uninfected, but Maggiore was unwilling to give up the spotlight. This HIV pretender twisted her good health and the marginal incidence of false positives into a lucrative[13] new racket—selling HIV denialism and bragging about her good life "without pharmaceutical treatments or fear of AIDS." But of course Maggiore has no "fear of AIDS"—she doesn't have HIV ... She has since had two children ... whom she boasted ... have never been tested ... But of course, Maggiore doesn't want them to be tested: she knows they are not at risk and their being uninfected would lead people to question her own status. (Bergman 2005)

Amazingly, last year a tragedy occurred that managed, in a moment, to change the public view of Christine's "status" from negative to positive.

In May 2005,[14] Eliza Jane, then three years old, came down with a cold that turned into an ear infection. After consultation with three doctors, she was prescribed Amoxicillin, which was taken in a dose exceeding that normally given a child her size. She began throwing up, and within twenty-four hours she stopped breathing. After several hours of attempting to resuscitate her at the hospital, she died of cardiac arrest.

Within several months, the Los Angeles County coroner was informed that Christine was HIV-positive, and an investigation was undertaken into what previously had been a death for which AIDS was not even considered as a cause. Four months after Eliza Jane's death, the Los Angeles County coroner released a report stating that she died of AIDS-related pneumonia and HIV-induced encephalitis. This finding was supported by finding *Pneumocystis* in her lungs—although this is a ubiquitous organism present in over 90 percent of humans—and the HIV-associated core protein p24 in her brain (although not in her blood). Mysteriously, no HIV test results were released, although supposedly an HIV test was performed.

The parents hired another pathologist to perform a differential diagnosis, and he did so with the conclusion that she died of an allergic reaction to Amoxicillin. Nevertheless, the debate raged on, especially on various blog sites, with people attacking the credentials of the doctor who performed the differential diagnosis and attacking Christine (though not

her husband, although presumably he had equal say in his child's health-care decisions).

Recently, a story was published in the *Los Angeles CityBeat* in which the Eliza Jane Scovill case was extensively examined. One crucial piece of information was presented: Eliza Jane had an absolute lymphocyte count that was elevated, going completely against the government's definition of AIDS as a state of HIV-induced immune suppression (Farber 2006b). That should have been the end of it, but it wasn't. The debate rages on.

This story is fascinating because it encapsulates everything that has come to characterize the AIDS debate, and all that is mysterious and ill-defined about the syndrome itself: a mother whose HIV status changes in people's minds according to what is convenient for them to think at the time; a death from PCP that exhibits absolutely no symptoms, even a day before her death; a diagnosis from AIDS made in spite of no HIV test results at all; and, saddest of all, vultures who will stop at nothing to prop up their paradigm, attacking a family who ought to be left alone and ought always to have been left alone. There is no precedent for assuming that anyone but her parents has the right to decide on her health care, and as such there is no reason for any of us to believe we have a right to vote on it.

Eight

WHERE DO WE GO FROM HERE?

> *AIDS does not inevitably lead to death, especially if you suppress the co-factors that support the disease. It is very important to tell this to people who are infected. I think we should put the same weight now on the co-factors as we have on HIV. Psychological factors are critical in supporting immune function. If you suppress this psychological support by telling someone he's condemned to die, your words alone will have condemned him.* (Luc Montagnier, co-discoverer of HIV, Wikipedia main site)

EVEN THE CO-DISCOVERER of HIV acknowledges the dangers of uncritically promoting the HIV=DEATH hypothesis. In order to prevent more deaths caused by inappropriate medical treatment and the psychological terror that accompanies an HIV diagnosis, we must fairly and honestly assess all the evidence.

There are several practical considerations. HIV tests are unacceptably unspecific, given the ramifications of a reactive result. Using proper isolation (and not just culturing methods to detect viral markers), we must rigorously verify the accuracy of these tests. The isolation experiments as proposed by prominent scientists would cost about $100,000 but despite the fact that this would be a drop in the bucket by AIDS research standards, no funding is forthcoming.

There urgently needs to be a proper debate in the scientific literature between the foremost establishment scientists and the best-credentialed dissenting ones. But the scientific ruling majority (note the intentional use of an oxymoron) refuses to even consider the possibility that they might be wrong, despite every indication to the contrary, and the top HIV scientists in the country continually refuse to participate in a debate with any "dissident."

The suppression of debate goes back to Peter Duesberg's very first criticisms of the HIV debate and Robert Gallo's refusal to entertain any such debate by quite literally running away. It continues to this day with slanderous accusations by leading scientists and a refusal to "dignify" the dissenting arguments by responding to or even acknowledging them.

Harvey Bialy recently challenged Dr. John Moore of Cornell University to a debate on the AIDS Wiki regarding the etiology of AIDS. Dr. Bialy's challenge was: "I will present one fully referenced (with PDF files that the moderator can hyperlink) challenge to your favorite and livelihood-sustaining hypothesis, and you can demolish my feeble arguments in the same fashion. We will continue this for one additional round, and then move on to the next challenge. I have maybe seven such challenges. At the end, we will have produced the first fully documented, real scientific debate on the cause of AIDS. Interesting that after twenty-five years none has ever been held before, Bob Gallo's promise in the *PNAS* in 1989 not withstanding."

Rather than accepting this debate, Moore replied by stating: "Participating in any public forum with the likes of Bialy would give him a credibility that he does not merit. The science community does not 'debate' with the AIDS denialists, it treats them with the utter contempt that they deserve and exposes them for the charlatans that they are. Kindly do not send me any further communications on this or any related matter."

Moore unwittingly exposes the true motivations of the AIDS "science community" in his reply to Bialy. It is clear that Moore and his ilk only desire to "expose charlatans" within self-defined constraints; namely, in situations in which they are protected from ever having to defend their own viewpoint and through channels that support their interests in their paradigm.

Furthermore, his choice of language is illuminating. He refers to the "scientific community," as though it were some sort of moral majority in-crowd, as though dissenters were not scientists at all—despite the fact that signatories to the petition for the Scientific Reappraisal of the HIV/AIDS Hypothesis number in the thousands and include two Nobel Prize winners and hundreds of PhDs and MDs. In Moore's view, apparently, none of these people qualifies as being a member of the "scientific community."

But HIV/AIDS research has always suffered from this sort of moral absolutism, outright discrimination, and suppression of argument. As Kary Mullis says in his book *Dancing Naked in the Mind Field,* "What people call science today is probably very similar to what was called science in 1634. Galileo was told to recant his beliefs or be excommunicated. People who refuse to accept the commandments of the AIDS establishment are basically told the same thing" (Mullis 1998).

The HIV theory has never been about science but rather about behavior modification primarily and, to a lesser extent, about money, power, and prestige. Language surrounding HIV and AIDS is infected with a sort of pious moralism that is completely inappropriate in science, and this sort of language is not restricted to the cultural and sociological aspects of AIDS. We can see it in the use of terms like "denialist" by scientists like Moore, and in the words of Dr. Mark Wainberg, who said that HIV dissenters are "perpetrators of death" and that "Peter Duesberg is the closest thing we have on this planet to a scientific psychopath" (Scovill 2004).

This same sort of science-by-majority-rule attitude can be seen in the words of an unnamed Berkeley scientist, interviewed by Celia Farber for her recent book *Serious Adverse Events: An uncensored history of AIDS:* "He did it to himself, you know. You see, he wouldn't give up an idea. He went at it with a hammer. He may well be 3,000 percent right, but he upset an awful lot of people … Nobody believed in him because what he was doing was overturning generally held views. They felt betrayed … You don't just stand up and say everybody is wrong" (Farber 2006a).

That sentence alone should illuminate just how much is wrong in HIV/AIDS science. But a society that has been so largely secularized has to believe in something with total faith, and for so many of us who

don't have the time to look into the minutiae of every issue for ourselves, that something so often is science and scientific discoveries, broadcast to us in the reassuring tones of *those who know better.* We don't question—we have faith. As Mullis says about the high priests of science: "Thank your lucky stars that they didn't bother to change their clothes or their habits. They still wear priestly white robes and they don't do heavy labor. It makes them easier to spot."

In his 1993 book *Rethinking AIDS: The Tragic Cost of Premature Consensus,* Robert Root-Bernstein wrote: "We do not understand AIDS." Fifteen years later, we still do not understand AIDS. And we will *never* understand AIDS until we acknowledge our own ignorance, but there are powerful forces at work preventing such acknowledgment.

First of all, there are tremendous financial and social interests involved. Billions of dollars in research funding, stock options, and activist budgets are predicated on the assumption that HIV causes AIDS. Entire industries of pharmaceutical drugs, diagnostic testing, and activist causes would have no reason to exist.

Second, the scientific and medical communities have a great deal of face to lose. It is not much of an exaggeration to state that when the HIV/AIDS hypothesis is finally recognized as wrong, the entire institution of science will lose the public's trust, and science itself will experience fundamental, profound, and long-lasting changes. The "scientific community" has risked its credibility by standing by the HIV theory for so long. This is why doubting the HIV hypothesis is now tantamount to doubting science itself, and this is why dissidents face excommunication.

Third, doctors have become emotionally attached to the idea of an HIV/AIDS pandemic threatening to take over the world. The HIV/AIDS "predictions" are a projection of the medical profession's self-identity, and taking away the HIV/AIDS paradigm threatens the medical profession's self-identity (Caton 1995).

Fourth, powerful psychological forces are at work. It is simply easier for most people to project our neglect of disenfranchised groups—gay men, drug users, blacks, the poor, and so on—onto a virus and accept those "infected" as sacrificial victims, than to recognize that *there is no bug.* For society, the latter would require acceptance of these disenfranchised groups as equal participants in mainstream society and culture.

However, the most significant obstacle of all is apathy. In a world full of constant distractions, most people are content to live in the public reality created by the media and advertisers. They do not want to be disturbed or provoked. Our most important goal is to make people *care.* We must reach their hearts, as well as their minds, and appeal to their inherent sense of justice and of what is right and wrong.

At that point, it is up to each person to acknowledge their own ignorance, to do their own homework, and to decide for themselves. To make that decision, all information must be available to everyone because, after all, as we have been told from the beginning by the AIDS mainstream, SILENCE=DEATH.

Appendix A:

FAILED PREDICTIONS OF THE HIV HYPOTHESIS

IN ORDER TO be considered viable, any scientific hypothesis needs to do two things—explain and predict. If a hypothesis finds itself, time and time again, making predictions that fail, it needs either to be seriously reassessed or to be considered a failed hypothesis.

Consider just a few of the predictions made by the HIV hypothesis of AIDS and decide for yourself.

HIV causes immune deficiency by killing CD4+ T-cells. In fact, it is currently not believed that HIV kills T-cells in any way, but rather, that HIV primes T-cells to commit suicide at some later time. This hypothesis has been put forward to explain the lack of evidence for any cell-killing mechanism that can be attributed to HIV.

HIV will spread rapidly throughout the population. "If the spread of AIDS continues at this rate, in 1996 there could be one billion people infected; five years later, hypothetically ten billion ... Could we be facing the threat of extinction during our lifetime?"—Theresa Crenshaw, President's AIDS Commission, 1987. Currently only *38 million* people worldwide are estimated to be HIV-positive, which is significantly fewer than was predicted at the beginning of the "epidemic." Furthermore, as the HIV prevalence curve and the CDC's own data show, at

least in the U.S., HIV has not spread at all since testing was first available.

By 1990, one in five heterosexuals may be dead of AIDS. This prediction, made in 1987, has proved catastrophically wrong. Approximately one in 250 Americans is estimated to test HIV-positive, and outside the risk groups this number drops to about one in *five thousand*—a far cry from the "one in five" figure cited nineteen years ago.

AIDS will decimate Africa. But even in the hardest-hit regions of sub-Saharan Africa, the population is growing at a rate of a few percent per year. HIV estimates are derived from extrapolations of data obtained by anonymously testing the blood of pregnant women with a single ELISA test. Since the beginning of the AIDS era, the population of Africa has increased by nearly 300 million—an increase equal to the entire population of the United States.

A cure will be available by 1986. This pronouncement was made at the Gallo-Heckler April 1984 press conference. Not only has it failed absolutely, it is now acknowledged that a cure is unlikely to ever be found. As Dr. Joe Sonnabend has said, "The notion of 'eradication' is just total science fiction. Every retrovirologist knows this. The RNA of retroviruses turns into DNA and becomes part of us. It's part of our being. You can't ever get rid of it." (Farber 2000)

A vaccine will be available by 1986. This pronouncement was made at the same press conference that so boldly predicted a cure. Not only has every vaccine trial to date been a flop, a vaccine may be impossible since HIV-positive individuals all have antibody to HIV already. There are therefore two options—either having antibodies is not protective, in which case a Jennerian vaccine is useless; or having antibodies is protective, in which case HIV is harmless and no vaccine is needed.

HIV will spread primarily by sexual transmission, needlestick injuries, and needle-sharing drug use. Since only one in a thousand unprotected sexual contacts with an HIV-positive person is estimated to transmit HIV, even a constant number of cases could not be sustained in this way. Clearly, the dominant mode of HIV transmission must be

other than sexual. However, to date fewer than one hundred needle-stick transmissions of HIV have even been reported. Additionally, studies show that users of needle-exchange programs are significantly *more* likely to test HIV-positive than are those who do not use clean needles.

If HIV is the sole cause of AIDS, it must be present at high titer in AIDS patients and conversely, AIDS will not be present in the HIV-negative. HIV has proven barely to be found in AIDS patients. In fact, according to Gallo's original research, HIV was found with higher frequency in pre-AIDS patients (at 88 percent) than in AIDS patients (at 36 percent). Viral load is only measured using PCR since many HIV-positive individuals have no evidence of virus by culture. By contrast, traditional viruses such as herpes, influenza, smallpox, etc. only cause disease at very high titer—thousands or millions of infectious unit per cubic millimeter of infected tissue. As far as finding no AIDS in the HIV-free, this idea was rendered obsolete with the addition of ICL in 1993 to explain the "HIV-free AIDS" cases that appeared and continue to appear.

AIDS will develop within one to five years from infection with HIV. This prediction, made in the mid-1980s, has had to be changed several times to avoid the embarrassment of explaining exactly why it is that AIDS rarely develops within such a short time frame. By 1998 the latent period had been estimated to be ten to fifteen years, and at the current time it is claimed to be about ten years, but little is known as to how accurate that is. Furthermore, this presents a conundrum when one considers the first AIDS cases: If AIDS takes ten years, on average, to appear, then we should expect that these original AIDS patients were all *at least* thirty or so years old. But many of the first AIDS patients were in their early twenties (ages ranged from the early twenties to the late forties among the first hundred men with AIDS), leaving an HIV believer with no other option than to consider that they became infected at twelve or thirteen years of age.

AIDS does not discriminate. However, in Europe and the U.S. AIDS remains restricted to the risk groups of homosexuals and drug abusers. The vast majority of cases affect men, and those not in the risk groups rarely develop AIDS without profoundly immunosuppressive cofac-

tors such as hemophilia or antiviral therapy. Even more damning is the fact that different risk groups exhibit different AIDS-defining diseases (Duesberg 1992).

Anti-HIV drugs stop AIDS. The annual mortality rate of HIV-positives undergoing antiviral therapy is much higher, at 7 to 9 percent, than the mortality rate of all HIV-positives worldwide, at about 1 to 2 percent per year (Duesberg 2003). Furthermore, there is ample evidence that treated HIV-positives die much faster of liver failure or cardiac failure than they would have to develop AIDS in the first place. Also, it is estimated that approximately one-third of HIV-positives, even in the U.S., do not know their status. If this is the case, there should be a huge number of people dying suddenly of AIDS, and this is not happening.

AIDS, and HIV, will spread randomly. This is clearly not the case. AIDS remains restricted largely to the risk groups, and HIV itself is dramatically more common among people of African descent than among Asians or Caucasians. HIV theorists have invented convoluted explanations for why this is so. The most popular is currently that a nontrivial proportion of Caucasians possesses a CCR-delta receptor deletion, rendering them immune to HIV. Supposedly neither Asians nor Africans possesses this mutation. This theory does nothing to explain why it is that the incidence of HIV is in fact lower among Asians than it is among Caucasians, nor does it explain why large populations of African prostitutes in high-risk areas such as Nairobi appear to be immune to HIV.

The prostitution and pornography industries will be decimated by AIDS. But prostitutes are not at risk for AIDS unless they are also drug users, and there are virtually no clients who have contracted AIDS from a prostitute. Moreover, the porn industry remains largely unaffected by AIDS despite the fact that condoms are rarely used and testing is known to be inaccurate.

Appendix B:

SUGGESTED FURTHER READING/VIEWING

Poison by Prescription: The AZT Story, John Lauritsen
The AIDS War, John Lauritsen
Infectious AIDS: Have We Been Misled? Peter Duesberg
Inventing the AIDS Virus, Peter Duesberg
AIDS: The Failure of Contemporary Science, Neville Hodgkinson
What if Everything You Thought You Knew about AIDS Was Wrong? Christine Maggiore
Oncogenes, Aneuploidy, and AIDS, Harvey Bialy
Serious Adverse Events: An Uncensored History of AIDS, Celia Farber
When AIDS Began, Michelle Cochrane
Rethinking AIDS: The Tragic Cost of Premature Consensus, Robert Root-Bernstein
Debating AZT, Anthony Brink
Dancing Naked in the Mind Field, Kary Mullis
Wrongful Death: The AIDS Trial, Stephen Davis
The Other Side of AIDS, directed by Robin Scovill
The Last Lovers on Earth, directed by Charles Ortleb

GLOSSARY

ad hominem: A form of arguing in which the strategy is to attack the person presenting the argument rather than the substance of the argument itself.

AIDS: **A**cquired **I**mmune **D**eficiency **S**yndrome, a classification consisting of any one of twenty-five to thirty different medical conditions plus positive antibody to HIV. The term AIDS replaced GRID in 1982.

AIDS-phobia: A term coined to describe the phenomenon wherein people who had recently had close contact with someone they suspected to be HIV-positive exhibited some symptoms of AIDS despite persistently testing HIV-negative.

Amoxicillin: A moderate-spectrum antibiotic used to inhibit a variety of gram-positive, and some gram-negative, bacteria.

antibody: A protein that is meant to identify and neutralize foreign objects such as viruses and bacteria.

antibody test: A laboratory test, usually performed on blood but sometimes on other bodily fluids such as saliva, that tests for the presence of antibodies to a particular organism by determining whether there is a reaction between the bodily fluid and certain antigens in the test kit. These antigens should be specific to the pathogen for which it is being tested.

antigen: A substance that initiates antibody production.

apoptosis: A type of programmed cell death, in which cells destroy themselves deliberately.

cell-mediated immunity: The branch of the immune system that handles intracellular parasites, such as viruses, fungi, and mycobacteria. Some consider cell-mediated immunity to have some involvement in cancer surveillance.

CD4+ T-cells: A subset of the lymphocytes involved in activating and directing other immune cells. Also called helper T-cells, CD4+ T-cells do not kill or destroy pathogens themselves.

correlation: A measure of the strength of the association between two or more variables. Correlation does not necessarily indicate a causal relationship between two variables.

differential diagnosis: Essentially a "second opinion"; when a person's initial diagnosis is inconsistent with clinical symptoms and a new diagnosis is sought, the new diagnosis is the differential diagnosis.

electron micrograph: A photograph or image taken through an electron microscope (a very high-powered microscope used to detect items too small to be seen via ordinary microscope) to show a magnified image of an item.

ELISA: The **e**nzyme-**l**inked **i**mmuno**s**orbent **a**ssay is a technique used to detect the presence of an antibody or antigen in a sample. It uses two antibodies, the first of which is specific to the antigen and the second of which is coupled to an enzyme (this second antibody gives the assay its "enzyme-linked" name) and will cause a chromogenic or fluorogenic substrate to produce a signal, which is seen as a color change.

endogenous: A factor or factors that originate from within an organism, e.g. the hormone estrogen is synthesized endogenously.

epidemiology: The branch of science concerned with factors affecting the health of individuals and populations.

etiology: Related to the causation of disease.

exogenous: A factor or factors that originate from outside an organism, e.g. a medication taken intravenously is exogenous.

genome: The hereditary information of an organism, encoded in either DNA or RNA.

GRID: The original name for AIDS, dating from 1980; the acronym stands for **G**ay-**R**elated **I**mmune **D**eficiency.

HAART: Highly **a**ctive **a**ntiretroviral **t**herapy refers to a combination of three or four antiretroviral drugs given to HIV-positives.

HIV: An acronym that stands for **H**uman **I**mmunodeficiency **V**irus. HIV replaced the American term "HTLV-III" and the French term "LAV" to describe phenomena attributed to an exogenous retrovirus, often found in AIDS patients and commonly considered the causative agent of AIDS.

humoral immunity: The branch of the immune system that handles extracellular parasites such as bacteria and worms. It is also involved in antibody production.

hypergammaglobulinemia: A condition in which an individual's immune system produces too many antibodies to both internal and external antigens.

hypothesis: A suggested explanation of some phenomena.

immune system: A system of specialized cells and organs that protect the organism from biological influences (mostly exogenous).

immunology: The branch of biochemical science that studies all aspects of the immune system in all organisms.

isolation: The separation of a biological agent from any other agent; removal of contaminants.

Koch's postulates: Four criteria published by Robert Koch in 1890, used to establish a causal relationship between organism and disease. These are (1) the organism must be found in all individuals suffering from the disease, and in no healthy individuals; (2) the organism must be isolated from a diseased individual and grown in pure culture; (3)

the cultured organism should cause disease when introduced into a healthy individual; and (4) the organism must then be reisolated from the experimentally infected individual.

lymphadenopathy: Abnormal swelling of the lymph nodes.

lymphocyte: Any of a number of white blood cells in the immune system involved in the defense against pathogens.

lymphocytopenia: Also called lymphopenia, a condition characterized by a marked depression in the number of lymphocytes.

mathematical biology: A field of study that models natural and biological processes using deterministic and stochastic predictive systems. The field includes models of population dynamics, cell biology, ecology, and physiological systems. This is not to be confused with statistical modeling, which analyzes biological systems using data.

mitogen: A chemical that prompts a cell to begin cell division (mitosis).

opportunistic infection: An infection caused by an organism that does not usually harm an individual with a healthy immune system but may cause disease in an immune-suppressed host.

positive predictive value (PPV): The PPV of a test indicates the proportion of positive tests that can be expected to indicate the true prevalence of the pathogen being tested for in the target population. For example, a 100 percent PPV means that every positive test is a true positive, whereas a 10 percent PPV means that only 10 percent of positive tests are true positives, and that 90 percent of positive tests are false positives.

protease inhibitor: A type of medication that inhibits viral protease, an enzyme used by viruses to assemble new virions.

retrovirus: An enveloped virus possessing an RNA genome, which replicates via reverse transcription. RNA is transcribed into DNA using the enzyme reverse transcriptase and is then incorporated into the host cell's genome via the integrase enzyme.

reverse transcriptase: Also known as RNA-directed DNA polymerase.

A DNA polymerase enzyme that transcribes single-strand RNA into double-strand DNA, the reverse of the way transcription normally occurs.

paradigm: A thought pattern in a scientific or epistemological context.

pathogen: A biological agent that causes disease or illness in its host.

polymerase chain reaction (PCR): A method of amplifying (mass-producing) DNA so it can be seen more easily.

prevalence: Defined to be the ratio of the number of people in a population affected by a certain disease to the total number of susceptible people in the population.

sensitivity: A measure of how likely it is that a particular test will produce a negative result when in fact the true result is positive. The higher the sensitivity, the fewer false negatives will occur.

specificity: A measure of how likely it is that a test will produce a positive reaction when in fact the true result is negative. A highly specific test will yield very few false positives.

teratogen: An adverse circumstance, including a variety of substances that cause congenital malformations in fetuses and babies.

viral load: A term meant to indicate the number of infectious viruses in a given sample of tissue, the HIV viral load uses quantitative PCR to magnify and estimate the number of HIV-associated RNA fragments in a milliliter of blood. Official estimates (Piatak et al. 1993) consider HIV viral load tests to overestimate infectious virus titers by a factor of 60,000. Viral load is not used to diagnose HIV infection.

Western Blot test (WB): Also called an immunoblot, the WB is a method used to detect a protein in a sample. The WB uses gel electrophoresis to separate proteins according to molecular weight and then determines the strength of sample reactions against these proteins individually rather than as a mixture.

ENDNOTES

[1]Although heterosexual transmission of HIV is presumed to be responsible for 70 to 80 percent of HIV infections worldwide, with the vast majority of cases occurring in Asia and sub-Saharan Africa, the actual data reported indicate the impossibility of the statement. Specifically, transmission probabilities reported for Africa (Gray et al. 2001, Hugonnet et al. 2002) are effectively identical to those in the U.S. (Padian et al. 1997), revealing the impossibility of a heterosexually transmitted epidemic anywhere in the world.

[2]African AIDS is diagnosed differently from AIDS anywhere else in the world. The so-called Bangui definition, arrived at in 1985 at a WHO meeting in Bangui, Central African Republic, consists of a set of symptoms *with no test for HIV antibodies necessary.* These symptoms are easily confused with those of tuberculosis, malaria, dysentery, cholera, and other common African diseases. Furthermore, in places where HIV testing is available, the criteria for a positive HIV test are the *least* stringent of any in the world, dramatically increasing the likelihood of cross-reactivity, particularly in a place where cross-reacting agents are common. Finally, estimates of the HIV prevalence in Africa such as those trumpeted in the world media are derived from blood tests given to pregnant women at antenatal clinics. What happens is that pregnant women are tested for syphilis as part of routine prenatal care, and some of the blood samples that are left behind are anonymously given a single ELISA HIV antibody test. The results of these tests are then extrapolated to the general population via computer simulations. The problems with this approach are many and include the fact that pregnancy itself is a source of

false positives, compounded by the fact that a single ELISA test will give an unacceptably high number of false positives.

[3] Medication increases T-cell counts almost immediately not because HIV has been attacked so effectively but rather because, in any person, artificial chemical stimulation produces an effect known as hysteresis, which means that the immune response surges to attack the chemical invader, creating an initial, and not necessarily beneficial or even meaningful, increase in T-cells.

[4] Currently, most countries do *not* use the low T-cell definition of AIDS. Canada and most of Europe do not.

[5] See http://healtoronto.com/rrsurvival.html.

[46] It is worth noting that there is no such thing as an "antiviral" drug. Drugs classified as "antiviral" in general work by changing the dynamics of the host cell to make the cell inhospitable to viral replication. There is no mechanism of drug action that can eliminate viruses from the body, and this problem is further compounded with retroviruses since the retroviral DNA is incorporated into the host cell's genome and remains a part of the host for life.

[7] It has been pointed out that there are a variety of other viruses, most notably herpes simplex, varicella zoster virus (which causes chickenpox but also shingles), and others, that can induce disease long after the establishment of antiviral immunity as evidenced by the appearance of antibodies. Such diseases are often used as arguments for HIV's apparent pathogenicity long after antibody production. What the HIV promoters consistently fail to mention is that in all the other cases, the antibody response is weakened and the virus is highly active, meaning that the symptomatic infection appeared thanks to a temporary decline in immunity that allowed for the appearance of the cold sore, the shingles rash, or what have you. HIV, in contrast, is not highly active at any point during final AIDS stages, so the comparison is not apt.

[8] Notice though that the presence of all such antibodies to latent infection merely indicate the *possibility* that the infection may later reactivate, not the certainty that it will. But with HIV, for some reason as yet never demonstrated in the literature, the presence of antibodies is taken to mean that the infection will not only later reactivate (since it is supposedly never inactive despite its activity being notoriously difficult to observe), but that it will do so in a particularly spectacular fashion, in every single case.

[9] Of course, there are a very few viruses that can only be cultured. However, these examples contain ample further evidence of pathogenicity.

[10] See http://www.healtoronto.com/durban/craddock.html#pcr.

[11] It is estimated that 3 percent of the human genome is retroviral in nature. This amount of genetic material is several hundreds of times larger than the genome of HIV.

[12] This belies the reports that one of the reasons Asian countries supposedly have higher rates of HIV and AIDS than the West is that Asians lack the alleged genetic mutation that supposedly protects some people from contracting HIV. If this were so, Asians in North America should have higher rates of infection than whites, which is not the case.

[13] One wonders how it is possible for anyone to refer to the questioning of the HIV/AIDS paradigm as "lucrative," as many people who have questioned this dogma have actually been harmed financially and career-wise.

[14] Bergman's *New York Press* article was published in June 2005, implying that it must have been submitted before Bergman was aware of Eliza Jane's death, and certainly before its cause was ever questioned, in September 2005.

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Rarely do researchers bite the hand that funds them—Rebecca Culshaw felt she had no choice.

DESPITE THE BEST EFFORTS of the medical and insurance establishments, the government, and the media to promote its acceptance, the HIV/AIDS hypothesis remains highly controversial. In *Science Sold Out,* Culshaw, a researcher who spent years constructing mathematical models of HIV's interactions with the immune system, focuses on the changing definition of AIDS and the flaws in all HIV testing. She explains how the current, government-based structure of scientific research has corrupted science in the search for truth, and offers not only scientific reasons for repudiating the HIV/AIDS theory but sociological explanations of how the theory was accepted by the media and the world so quickly. The book also presents a scathing critique of the outrageous discrimination that HIV-positives have faced since the theory was first accepted.

"An excellent account of the most shameful episode in the history of medicine. Rebecca Culshaw has pulled it all together: a history of inept and dishonest AIDS 'science,' the manifold reasons HIV cannot be the cause of AIDS, the harmfulness of AIDS drugs, the physical and psychological human suffering caused by the AIDS hoax."

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"Every mathematician knows that by changing the definition of something, you can change the entire truth about that thing. Rebecca Culshaw describes how the HIV = AIDS 'orthodoxists' have abused this idea. As in a shell game, they keep moving the definitions around, so that anything can be true and everyone will be confused. The abuse of science that has been documented here is itself very frightening. But when we learn that the standard treatment for HIV-positives—antiviral therapy—will substantially increase their risk of dying, it's even scarier."

Dan Fendel, professor of mathematics, emeritus, San Francisco State University


NATHAN SMITH

REBECCA CULSHAW, assistant professor of mathematics at The University of Texas at Tyler, came to the U.S. in 2002, after receiving her PhD in mathematics from Dalhousie University in Halifax, Canada. She has published several journal articles regarding mathematical modeling of HIV immunology, and serves on the advisory board of *Journal of Biological Systems.*

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