**Is HIV the Cause of AIDS?**

Peter H. Duesberg and Bryan J. Ellison Respond to Their Critics

The Summer 1990 issue of Policy Review contained one of the three or four most-talked-about articles in the history of the magazine. "Is the AIDS Virus a Science Fiction?: Immunosuppressive Behavior, Not HIV, May Be the Cause of AIDS," by Professor Peter Duesberg of the University of California at Berkeley and his doctoral student Bryan Ellison, put into layman's language an argument that Duesberg, one of the world's leading retrovirus researchers, has advanced several times in scientific publications. The article elicited more letters to the editor than any in Policy Review's history, and our offices received more comments, both positive and negative, than on any other article in recent memory.

Most responses to the article have been sharply critical, as one would expect for an argument challenging the reigning paradigm of most leading scientists and doctors closely involved with AIDS, as well as of most conservatives who are deeply knowledgeable about the disease. But a large number of readers said they were absolutely fascinated by the questions Duesberg and Ellison raise about prevailing AIDS wisdom, and they wondered why Duesberg's argument has received virtually no public attention. Whether or not one agrees with Duesberg's and Ellison's arguments—and the important public policy implications if they are correct—it does seem that there has been a rush to judgment implicating HIV as the cause of AIDS and a distressing politicization in the scientific community that refuses even to entertain contrary views.

What follows is a sampling of letters we received about the Duesberg-Ellison article, together with a response from the authors.

**Fringe of Science**

Dear Sir:

I was stunned by the article by Peter H. Duesberg and Bryan J. Ellison. I felt like I had been on Mars for 50 years and had just returned home to find that everything I had come to know about AIDS was no longer true.

In the interest of brevity, I will point out only seven statements by the authors that are completely at variance with current knowledge:

1) "Evidence increasingly indicates that large numbers of people infected with HIV, probably the majority, will never develop AIDS."

2) "Koch's postulates unmet." Robin Weiss and Harold Jaffe thoroughly trounce this assertion in their commentary appearing in the June 21, 1990, issue of the British medical journal Nature, citing the etiological

3) "AIDS diseases without HIV." Duesberg and Ellison note that Kaposi's sarcoma has been diagnosed in some homosexual men who are HIV-negative. Your authors failed to mention a hypothesis gaining increasing support: that Kaposi's sarcoma may well be the result of a completely separate etiologic agent from AIDS, transmitted in homosexuals through anal intercourse, as is AIDS. Kaposi's sarcoma occurs rarely among other HIV-infected individuals, such as blood-transfusion recipients, intravenous drug users, and hemophiliacs. This observation is simply not a legitimate "flaw" in the HIV hypothesis.

4) "The syndrome began to level off in 1988." It is widely known and accepted that the leveling off in new AIDS cases among homosexual men in New York, Los Angeles, and San Francisco was the direct result of therapy with AZT, an antiviral drug that slows progression to AIDS and therefore postpones diagnosis. Cases among infected intravenous drug users, the majority of whom could not afford to take AZT prophylactically, have risen steadily, accounting for 21 percent of all cases in the United States, and 47 percent of new cases in New York City.

5) "There are no confirmed cases of AIDS among health care workers after accidental infection." This is simply false. Infections and subsequent AIDS cases resulting from needle-stick exposures are well known and documented. One physician, infected in precisely this manner in 1985 and diagnosed with AIDS in 1988, addressed the Sixth Annual AIDS Convention last June in San Francisco.

6) "The AIDS diseases seen among infants tend to be the typical pediatric diseases." This statement is disgracefully inaccurate. Pneumocystis carinii pneumonia (occurring in 40 percent of pediatric AIDS cases) is a typical childhood disease? Lymphocytic interstitial pneumonitis? Fungal infections of the esophagus or lungs? Cryptosporidiosis? Cryptococcal meningitis? Even before the advent of antibiotics and vaccines these infections were never considered routine.

7) "AIDS is a childhood disease?" Robin Weiss and Harold Jaffe thoroughly trounce this assertion in their commentary appearing in the June 21, 1990, issue of the British medical journal Nature, citing the etiological
agents of cholera, polio, and tuberculosis as well-known exceptions to the outdated postulates. They go on to explain that other researchers, following modernized versions of the postulates, have convicted HIV as the causative agent of AIDS.

7) "[For people with AIDS] the use of AZT and similar antiviral-specific drugs should be avoided." With this particular statement the authors cross over the border of science into the realm of quackery. AZT, certainly, is an imperfect drug. It is not a cure. But as numerous studies published in reputable medical journals establish, AZT is the best and only antiviral treatment currently available for HIV infection. To recommend that HIV-infected persons forgo such treatment based on "anecdotal case descriptions" is a grievous misinterpretation of scientific evidence.

I am deeply distressed about this article and its contents, which are, at best, at the fringe of science.

Elizabeth M. Whelan
President
American Council on Science and Health
New York, NY

Evidence for HIV

Dear Sir:

In their recent article Duesberg and Ellison argue that HIV is not the causal agent of AIDS and that preventive measures based on this premise are "misguided." As an epidemiologist involved in the investigation of HIV infection and AIDS, I cannot accept these assertions. Here are some of my reasons:

1) Among 386 homosexual men who were already infected by HIV in 1984 and were followed with twice-yearly examinations by my colleagues and me, 140 (36 percent) developed AIDS, and 80 died of AIDS in the ensuing five years. Among 40 homosexual men infected by HIV after entering the study, two (5 percent) developed AIDS. Among 370 homosexual men, simultaneously recruited for study from the same source and who remained uninfected during the five years of observation, none developed AIDS.

2) Among the 386 men already infected by HIV in 1984, 193 (50 percent) had T-helper cell counts below 500 per microliter of blood on initial examination, while among the 370 uninfected men, only 18 (5 percent) had T-helper cell counts lower than 500 per microliter. A deficiency of T-helper cells is the key factor causing the immune deficiency, which, in turn, is responsible for the wide spectrum of clinical manifestations of the acquired immune deficiency syndrome (AIDS).

3) In HIV-infected men, T-helper cell counts fell, on average, about 80 per microliter in each year of observation. Less than 15 percent of HIV-infected men failed to show a decline in T-helper cell counts during the follow-up period. The average T-helper cell count in uninfected men remained constant over the five years of observation.

4) In our study, and in all other studies, acquisition of infection by HIV among homosexual men was primarily associated with a particular sexual practice, receptive anal intercourse with numerous different partners. Acquisition of infection was not related to drug use, per se, but was highly correlated with needle sharing during drug use. These observations are fully consistent with an infectious mechanism of transmission.

5) The rate of infection by HIV in the 410 initially uninfected men in our study declined from an annual average of 6 percent for the period 1984-85 to less than 1 percent during 1989. This decline was associated with the adoption of recommended safe sexual practices by a large proportion of study participants.

Koch Knew His Limits

Duesberg and Ellison emphasize the failure of HIV to satisfy the criteria of Koch's postulates. However, even when he was restating criteria earlier proposed by his teacher, Jacob Henle, Robert Koch knew that certain pathogenic bacteria, in particular, the tubercle bacillus, did not fully satisfy the criteria. In modern times, established pathogens such as poliovirus do not satisfy Koch's first or third postulate, i.e., the virus cannot be isolated from all cases and only a small proportion of infected persons develop disease. Duesberg and Ellison are wrong when they claim that no medical workers, accidentally infected, have developed AIDS. Of the 27 documented cases of HIV infections acquired through accidental infection by medical workers, two have developed AIDS.

The epidemiological evidence supporting a causal role for HIV in the etiology of AIDS is overwhelming. The modes of transmission of HIV have been established and provide the basis for a rational approach to prevention. However, an understanding of the pathophysiology of HIV infection remains incomplete. As this understanding develops, many of the apparent paradoxes enumerated by Duesberg and Ellison may be resolved.

Warren Winkelstein Jr.
Professor of Epidemiology
University of California at Berkeley
Berkeley, CA

Proof in the Pudding

Dear Sir:

Duesberg and Ellison repeat misleading and fallacious arguments that have been refuted many times in other journals. I shall reiterate some of these points.

Koch's postulates were a great advance a century ago. However, they no longer encompass our increased knowledge of infectious diseases. Even so, the relationship of HIV to AIDS does in fact fulfill the modern version of Koch's postulates.

Contrary to statements in the Duesberg and Ellison article, certain strains of simian immunodeficiency virus do cause an AIDS-like disease in monkeys; HIV is very different in genetic structure from most other retroviruses (it has at least five additional genes); the distributions of HIV and AIDS are similar when allowance is made for the long latent period and for differences in reporting; there is now a drastic decrease in the proportion of pediatric AIDS attributable to transfusion (there has been an increase in the number of cases because the latent period after infection until appearance of clinical illness ranges from 2 to 15 years);
there are well-established instances of health care workers with no other risk factors becoming infected with HIV and then developing clinical AIDS, as exemplified in the highly publicized recent New York City court case; and numerous examples of heterosexually transmitted AIDS directly linked to HIV seroconversion without any other risk factors (or the life-style factors claimed by Duesberg and Ellison as the cause of AIDS) have also been well documented.

**Pediatric Evidence**

Most convincingly and tragically, mothers infected with HIV pass the virus to about one-third of their offspring, although all offspring of HIV-infected mothers receive antibodies to HIV. Several studies show a large excess of AIDS and related symptoms in HIV-infected children of HIV-infected mothers compared with uninfected children of HIV-infected mothers. For example, in a study by Goedert, 15 of 16 HIV-infected children of HIV-infected mothers had AIDS or pre-AIDS symptoms, while none of 39 uninfected children of HIV-infected mothers were ill. In total, 72 percent of the HIV-infected children of HIV-infected mothers had the disease, compared with only 5 percent of the uninfected children. Duesberg and Ellison state that “the risk behavior of many of their mothers has reached these victims.” It is clear that what reached the children was HIV.

That HIV causes AIDS is well established. An anti-HIV therapy, AZT, has actually decreased the rate of appearance of new cases of AIDS. However, there are still many unanswered questions about the pathogenesis by HIV, about how to develop a safe and effective vaccine against HIV, about how to stop behavior that results in transmission of HIV, as well as how to pay for treatment of HIV-induced disease, and many others. (The majority of the federal spending on AIDS is not spent on research, but on treatment.) The only way we will stop the AIDS epidemic is through more biological and behavioral research.

**Harmful Science**

Dear Sir:

I was profoundly disappointed to learn that *Policy Review* would print anything by an individual who has been so discredited in the scientific community as Peter Duesberg. His ideas are not only wrong, but incredibly harmful.

When we formed our organization, Americans for a Sound AIDS/HIV Policy, over three years ago, we researched intensively to find the truth regarding HIV disease. We found a dramatically range of opinion on this topic, often being biased by either pro- or anti-homosexual opinions. The least biased studies have been done by the armed forces.

**Research at Walter Reed**

The military is completely thorough in its research in order to protect its personnel, since, in time of war, its soldiers serve as its front-line blood bank. Among other things, the military conducted extensive surveys in Africa and other countries (many of these unpublished), as well as screened its entire active force of over two million individuals, of whom presently more than 6,000 are infected. It has also tested all civilian military applicants since October 1985 for the HIV virus. The data generated by these extensive studies fully conclude that HIV is a progressive disease that causes a slow but relentless destruction of T-cells and eventually results in the individual succumbing to what would be otherwise non-life-threatening diseases.

Researchers at Walter Reed Army Institute of Research found a progressive decline in the average number of T-cells from time of HIV infection until symptomatic AIDS and death. This finding contradicts Duesberg’s statement that “the number of T-cells lost at any time would be roughly equivalent to the number lost from bleeding from shaving. Such losses could be sustained indefinitely without affecting the immune system because the body constantly produces new T-cells at far higher rates.”

Duesberg’s assertions that “virtually no reactivation of the virus occurs when AIDS patients develop sickness” and that “after the body produces antibodies against HIV the virus remains at low levels for the rest of that person’s life,” are equally false. As the number of T-lymphocytes declines, the volume of virus in body fluids increases. This is not theory or hypothesis. This is reality.

The progressive nature of the virus is further detailed by numerous studies showing that those infected with HIV progressively worsen through the diminution of T-cell counts. Most major clinical trials involving therapeutic drugs or treatments use T-cells as a prime marker for disease progression. This is accepted scientific practice and not some form of witchcraft as Duesberg would have readers believe.

Regarding Koch’s postulates, Duesberg is mistaken again when he writes, “until the recent advent of highly sensitive methods no direct trace of HIV could be found in the majority of AIDS cases.” Incorrect. Ninety-five percent of late-stage AIDS patients at Walter Reed Army Medical Center in Washington, D.C., could be cultured and all tested positive for HIV—at all stages. Duesberg alleges that the virus level “is typically so low” that it could not be isolated. Equally false. Duesberg maintains that “when accidentally injected into health care workers, even though the virus successfully infects those hosts,” these people didn’t develop AIDS. Again, incorrect. Two highly publicized lawsuits were settled recently by Johns Hopkins with Dr. Anoun and New York City’s Health & Hospitals Corporation with Dr. Prego. It was acknowledged that their infections occurred on the job, and both doctors now have symptomatic AIDS. There are many others.

**Epidemic Proportions**

Perhaps Duesberg’s biggest misrepresentation of all is the statement that “evidence increasingly indicates that large numbers of people infected with HIV, probably the majority, will never develop AIDS.” All evidence now shows that HIV will claim all whom it infects. The epidemic continues to increase. Annualized cases ending...
in June 1990 totaled 40,006 versus 33,512 cases reported through June 1989. The epidemic will continue to claim ever-increasing numbers and percentages of people. The Centers for Disease Control recently reported staggering HIV-infection rates, as high as 7.8 percent in certain neighborhoods in the Northeast.

In discussing civilian military applicant data, it is important to note that in the six most heavily infected counties in the country today, the ratio of young men to women infected with HIV is now one to one. That means that ultimately the ratio of men to women with AIDS in that age bracket will approach one to one. Recently reported cases of AIDS—which reflect HIV infections five to 15 years ago—have no bearing on present infections, other than to reveal that the epidemic is becoming a heterosexual epidemic among young people. It is not true, as Duesberg states, that "males with HIV are more likely than females to develop AIDS even though they have the same virus." He also states that the proportion of men to women in reported AIDS cases "has not changed since AIDS was first defined." Again, untrue. The ratio, in fact, has dropped from about 13 to 1, males to females, to about 9 to 1 today.

Duesberg would be interested to know that the CDC's AIDS case definition actually does not require a positive HIV test to qualify as AIDS. In all likelihood some individuals in the past were defined as AIDS cases and yet were not HIV-infected. This would account for some of the long-term survivors of AIDS who may never have had HIV disease, and were able to combat the other opportunistic diseases effectively.

**Heterosexuals Beware**

Duesberg's arguments about risk behavior have some validity in that this is a disease acquired through intimate sexual or intravenous contact. However, his statements are much more misleading since there are those he wouldn't classify in any "risk group." Many young, sexually active heterosexuals are now becoming infected and will be at risk for contracting this disease in the future. In fact, heterosexuals are now the fastest-growing group of reported AIDS cases.

Duesberg also claims that AZT is nearly the only treatment prescribed to people who are HIV-infected, while conventional diseases are neglected. This is also blatantly untrue. Other diseases are treated. However, because the individual no longer has a functioning immune system, these diseases in time overwhelm the body, even with medication to defend against them. Once again, Duesberg is utterly wrong when he claims that, "HIV is inactive by the time AZT is administered."

One of the author's statements is correct: that his risk hypothesis should reduce the fear of HIV infection. It certainly will do that. As a result, many will believe they aren't at risk, and will subsequently become infected and die. Having worked with many families who are suffering from this disease through all modes of acquisition of this virus, we can say that his recommendation that sexual partners of HIV-positives need not be contacted or traced is perhaps the most irresponsible position that could be taken by anyone in this epidemic.

Duesberg's conclusion that, "the HIV hypothesis has not yet saved a single life," is totally untrue. We personally know individuals who have been saved because their spouse learned of their infection in time to alter behaviors. We also know a number of children who tragically are losing both parents because a spouse wasn't informed. These are real people who are dying at very early ages and leaving behind fine young children whom we will all have to take care of in some way.

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**The authors' recommendation that sexual partners of HIV positives need not be contacted or traced is perhaps the most irresponsible position that could be taken by anyone in this epidemic.**

—W. Shepherd Smith Jr.

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**AIDS Virus Not a Fiction**

Dear Sir:

"Is the AIDS Virus a Science Fiction?" ask Peter H. Duesberg and Bryan J. Ellison. Had you asked some practicing physicians, or even a mining engineer with knowledge of the situation in Africa, the answer would be clearly "No."

The article sets some of its errors in large, bold-face type, e.g., "there are still no confirmed cases of AIDS among health workers after accidental infection with HIV." There is a multimillion dollar lawsuit in progress about one such case. Another is described in the first person in the September 7, 1989, issue of the New England Journal of Medicine.

Can "the combination of prolonged malnutrition with heavy use of alcohol, heroin, cocaine, and antibiotics...lead to complete immune system collapse?"? We didn't see this when I was a student or intern in inner-city hospitals. And when we see it now, the HIV test is usually positive. (Yes, all tests known to man have false negatives. And there is more than one cause of immunodeficiency.)

Public health departments may be delighted to hear that "there is no need to trace the sexual partners of HIV positives," since most of them don't do it anyway. But the prevalence of seropositivity for HIV in regular heterosexual partners of infected persons has ranged from 10 percent to 60 percent in various studies.

Nobody knows how many Africans have died of AIDS.
Some say that as few as 1 percent of actual AIDS deaths are reported. But there are armies of orphaned children, and workers refuse to go to some mining communities where the prevalence of disease is especially high.

There are too many errors in this article to cover them all. Perhaps the most obvious one is the assertion that syphilis is "difficult to test for."

Jane M. Orient, M.D.
Tucson, AZ

HIV One of Many Immunosuppressors

Dear Sir:

Lest readers of Duesberg and Ellison’s article claiming that HIV is not the cause of AIDS think that the authors are lone wolves crying in the wilderness, let me add my voice to the growing chorus. While I am not convinced that HIV is irrelevant to understanding AIDS—after all it is highly correlated with the syndrome—I am not convinced that it is any more important than other immunosuppressive agents associated with AIDS. On the contrary, I believe existing evidence demonstrates that HIV is neither necessary nor sufficient to cause AIDS.

First, data linking HIV to AIDS are nowhere near as good as the public are led to believe. Reference to the Centers for Disease Control’s own data reveals that 5 percent of AIDS patients tested for HIV never display signs of infection, and that less than 50 percent of AIDS patients have been tested for HIV.

Recently, cases of homosexual men with AIDS and without HIV infections have been verified. In response, HIV proponents are lobbying for a change in the definition of AIDS to exclude HIV-free cases. These people do not, apparently, understand two things: 1) that defining AIDS by HIV and simultaneously demonstrating that HIV causes AIDS is tautological, and therefore bankrupt, reasoning; and 2) that altering the definition of AIDS does not alter the fact that HIV-free people can and do develop the same set of opportunistic infections as those who are HIV-infected. Whether these HIV-free cases are listed as AIDS patients or not, they are still medical patients whose syndrome is in need of explanation. Logically, HIV is not, therefore, necessary to cause the development of these symptoms, and other causes of what we now call AIDS must exist.

Other Agents

My own research, which was published this summer in Perspectives in Biology and Medicine, suggests what these other causes of acquired immunosuppression may be. Briefly summarized, all of the following agents had been demonstrated to be immunosuppressive prior to the discovery of HIV, and all are highly associated with one or more AIDS risk groups: immunological response to semen following anal intercourse; the use of recreational drugs such as the nitrates (“poppers” and “snappers”); chronic antibiotic use (often associated with promiscuity); opiate drugs; multiple transfusions; anesthetics; malnutrition (whether caused by “gay bowel syndrome,” drug use, poverty, or anorexia nervosa); multiple, concurrent infections by diverse microbes; and infection by specific viruses such as cytomegalovirus, Epstein-Barr virus, and hepatitis-B virus (all of which are as highly associated with AIDS as is HIV).

Several of these agents, including cytomegalovirus, hepatitis-B virus, opiate drugs, and repeated blood transfusions, are known to cause the same sort of T-cell abnormalities that are found in AIDS, and which are usually attributed (perhaps inaccurately) to HIV infection. The other agents cause a wider spectrum of immunosuppressive responses, and probably explain why more than simply T-cells are non-functional in AIDS patients. Every AIDS patient has several of these immunosuppressive agents at work in his or her system in addition to, and sometimes in the absence of, HIV. We cannot, therefore, logically conclude that HIV is the sole or even the main cause of immunosuppression in AIDS.

19th-Century AIDS

Now, if the so-called life-style theory of AIDS is correct, one important implication is that AIDS should not be a new syndrome. It is not. I am one of only a handful of scientists who have bothered to search intensively through the back issues of medical journals for odd cases that match the CDC surveillance definition of AIDS. So far I have found hundreds of such cases, extending back to 1872 (the date when the first opportunistic disease associated with AIDS was identified). I have also scoured the medical literature for data relevant to changes in life-style risks associated with immunosuppression. What I have found is very provocative.

Whereas the Kinsey report of 1948 indicates that the average homosexual man had a sexual encounter no more frequently than once a month, by 1980, the advent of gay bars and bath houses had increased this average to dozens per month. Gay AIDS patients have often had thousands of sexual partners. Medical reports of complications arising from AIDS-associated high-risk activities such as anal intercourse and fisting are first mentioned in the medical literature only at the beginning of the 1970s, and become increasingly frequent thereafter. From 1960 to 1980, the rates of syphilis triple, gonorrhea quadruple, and diseases related to “gay bowel syndrome” quadruple. These increases were found only among gay men, but not among heterosexual men or women.

From 1960 to 1980, hepatitis-B cases rose 10-fold, in part due to sexual transmission in gay men, and in part to IV drug abuse. Arrests on opiate-related drug charges rose nearly 20-fold during the same period. There is, then, no doubt that AIDS was preceded by medically evident changes in life-style among those groups at highest risk for AIDS, and these changes are such that not only HIV, but the entire spectrum of immunosuppressive agents mentioned above became increasingly prevalent in these groups.

These data indicate to me that HIV is not sufficient to explain the manifestation of AIDS or its recent appearance. Many other factors are also at work. It is a tremendous mistake to base our policy decisions concerning AIDS on an exclusive HIV basis. Far from undermining current drug prevention and safe sex programs, the recognition of non-HIV immunosuppressive factors in AIDS suggests that these programs are failing because they are too narrow. AIDS will only be understood when we begin to explore the ways in which...
anal sex, infections, drugs, blood products, anesthetics, antibiotics, and malnutrition interact. At present, we know almost nothing about such interactions. Since increasing evidence from the laboratories of the discoverers of HIV indicates that HIV needs immunosuppressive co-factors to be active, such studies are clearly needed. In the meantime, those who wish to avoid contracting AIDS should avoid all potential causes of immunosuppression, not just HIV. And those who are HIV-positive but not ill may find that if they, too, avoid this lengthy list of immunosuppressive co-factors, they too will stay healthy.

Robert S. Root-Bernstein
Associate Professor of Physiology
Michigan State University
East Lansing, MI

Paradigm Unvisited

Dear Sir:

My concern, expressed for some years in TV programs, magazine articles, and my book AIDS: The HIV Myth, has been the subjective element in scientific research. Medical science in particular is presented to the public as a seamless body of unchallengeable knowledge, when in fact it is a complex mass of conflicting beliefs, each supported by a foundation of fact, but buttressed by the vested interests of research institutes and fashionable theories.

This has never been more true than in the case of the HIV theory. But there is another element here. There is something about the heady mix of science and sex in this theory that inspires extremes of intolerance in those who espouse it. Anyone questioning the link between HIV and AIDS is met with an unreasoning fury or an offended refusal to discuss the matter. The hysteria directed against critics of the HIV theory suggests that it is not a matter of scientific fact that is being defended here but a belief system.

This is particularly disconcerting because of the very poor quality of scientific thinking behind the HIV theory. To give some examples: Current scientific method says that to prove a theory we should actively seek information that would disprove it. It is by resisting these repeated challenges that the theory becomes stronger, or, instead, it fails and gives way to another theory more appropriate to the evidence. In fact, since HIV was declared the cause of AIDS at a press conference in April 1984 (before the scientific papers that were supposed to support it were even published), there have been no experiments designed to test the HIV theory. All the work in this multimillion dollar research project has been designed and carried out to support the HIV theory of the cause of AIDS.

No Questions Asked

One of the pillars of scientific thinking is predictive testing. As a part of policy-making, government scientists make predictions of the number of AIDS cases based on current HIV infection rates. The predictions have turned out to be wrong by orders of magnitude. There are far fewer AIDS cases than would be predicted by the number of HIV cases. Under the rules, if your prediction doesn’t come true, you re-examine the theory. What the HIV supporters do in these circumstances is to conjecture that maybe their assessment of the number of people with HIV infection was wrong, or maybe the incubation period is longer than they thought. The parameters of the predictive experiment have to be adjusted in retrospect to fit an unwelcome outcome. The only thing they will not do is re-examine the theory that HIV alone and of itself causes AIDS.

The use of mainly anecdotal "evidence" from Africa in defense of the HIV theory is particularly shocking. In 1988 and 1989 the AIDS epidemic failed to follow predictions and AIDS cases in the U.S. and Europe began to plateau without substantially exceeding the limits of the so-called at-risk population. What should have happened was a wholesale re-evaluation of the HIV theory. Instead, its supporters told us to look to Africa, which would demonstrate they had been right all along. So the two continents with the greatest capacity for collecting medical information and analyzing statistics about the epidemic were relying for confirming evidence not on their own well-funded institutions, but on information garnered from the continent with the least sophisticated health and statistical services.

Shabby Criticism

The poor quality of scientific thinking leads to shabby behavior in the conference halls and journals. A theory that is poorly grounded has to defend itself from its critics on the basis of sneer and insult, for it has no honorable weapons of debate.

Now, having failed to rise to the challenge to their theory by scientists such as Duesberg in the scientific papers, defenders of the HIV theory complain that criticism of it has been made available to the public. This will, we are told, undermine confidence in public health measures designed to protect the general population. I happen to feel that the use of clean needles and condoms is a valuable public health measure in itself without the bogeyman of HIV. But what really interests me is the way critics of the HIV theory are told to keep their doubts to themselves because if they don’t, the very theory about which there is serious doubt might lose its influence with the public. Thus doubt is placed in the service of certainty in the public interest.

Ultimately, expert advice must be evaluated by the people who are not experts—politicians, journalists, and the public. This is part of democratic life and a scientist has no more right to exclusion from public scrutiny than a treasury official. All expert advice affecting our lives must be subjected to abrasive doubt. In the field of the
HIV theory this doubt has had a struggle to thrive in the scientific community. It needs an infusion of energy from outsiders whose only interest is to ensure that hard questions are asked and the "AIDS establishment" is pinned down to answer them.

Jad Adams
London, England

A Study in Risk Behavior

Dear Sir:

Duesberg and Ellison present as thorough and balanced a review of AIDS as I have seen in print. It certainly makes a strong case for the thesis that immunsuppressive risk behavior is at least as likely as HIV to cause this complex array of diseases. I have witnessed the abuse to which Duesberg has been subjected for arguing this thesis. The coup de grace that is supposed to silence him is that AIDS cases among hemophiliacs and children of HIV-positive mothers do not exhibit the risk behavior. But the article deals effectively with those objections by showing that they, in their own way, constitute risk groups.

The authors' suggestion that controlled studies be done on HIV-positive and negative groups with equivalent risk behavior is an eminently sensible one, and I am amazed that this has not been done. This, of course, must be done using non-HIV controls undergoing equivalent risk behavior as the HIV carriers, i.e., equivalent number of anal-receptive drug-using sexual encounters, or frequency of intravenous drug use. To the best of my knowledge, such a study has not been done, much less published.

If the authors are correct, and HIV is essentially a reporter for high-risk behavior, it would be difficult indeed to find the right controls. Until it is done in a scientifically sound way, I am not willing to accept—or to completely reject—a central role for the virus in the etiology of the disease. But, since such a strong case can be made for the role of drugs, antibiotics, and related risk behavior in the origin of AIDS, it makes little sense to recommend clean needles and condoms while ignoring the behavior itself.

The one solid epidemiological fact we seem to have is that the disease in the U.S. is restricted almost entirely to certain risk groups. Regardless of the involvement of the virus, the only sure cure is to modify the behavior.

Harry Rubin
Professor of Cell and Developmental Biology and Virologist to the Virus Laboratory
University of California at Berkeley
Berkeley, CA

Mycoplasmal Agents

Dear Sir:

In the spirit of "the openness" of science we salute Peter Duesberg for his challenging and courageous voice speaking out against the present scientific establishment. His extensive experience and knowledge about retroviruses lends merit to his critical evaluation of the possible causative role of HIV in the AIDS disease.

However, there is a fundamental difference between our judgment of the AIDS disease and that of Duesberg. We believe that the disease of AIDS is an infectious process. Despite our respect for Duesberg’s expertise in retroviruses, we think his assessment that no microbe, including any mycoplasma, could possibly cause the full set of AIDS diseases is premature. It has been known for many years that microbes known as mycoplasmas can cause immune suppression, weight loss, diarrhea, and chronic debilitation in animals; but mycoplasmas were not considered fatal in humans. The recent discovery that a previously unrecognized pathogenic mycoplasma, M. incognitus, causes fatal systemic infections in experimental monkeys, has suggested that this microbe could be playing a disease-promoting role for AIDS. It is significant that mycoplasmal infection has been found in diseased brains, livers, and spleens of AIDS patients, as well as some HIV-negative patients displaying similar symptoms.

Luc Montagnier, the French discoverer of HIV, is the most famous but not the only eminent scientist who endorses the possibility that mycoplasmal agents could play a significant role in AIDS. Many mycoplasmologists worldwide have now joined the search of these microbes in patients with AIDS. We also applaud Montagnier’s courageous strong stand at the recent International Conference on AIDS that mycoplasma could be the key co-factor of AIDS disease.

There are many intriguing, but certainly not well understood, biological characteristics of M. incognitus and the infection it produces. The infection suppresses the immune system, causes immune derangement, and can be associated with chronic debilitating disease.

The biology and nature of these mycoplasmas need to be carefully reassessed, using modern technology. The rapidity of advances in understanding the significance of mycoplasmal disease in humans will be directly proportional to the amount of funds available. At present only a very small amount of money supports mycoplasma studies.

We believe the most healthy and responsible scientific
attitude in dealing with AIDS research is to explore all possible avenues. To make any conclusion lightly or prematurely, such as ruling out any possible role of microbes in AIDS, or to commit oneself exclusively to a particular agent and completely rule out any other possible role of a different microbe, may all result in a greater loss of AIDS victims.

Shyh-Ching Lo
Chief, Division of Geographic Pathology
Col. Douglas J. Wear, MC, USA
Chairman
Dept. of Infectious and Parasitic Disease Pathology
Department of Defense
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Washington, DC

AIDS or Syphilis?

Dear Sir:

While we agree with many of Duesberg and Ellison's criticisms of AIDS research, they are ignoring research (i.e., on AIDS contracted by the wives of hemophiliacs) that appears to support the HIV hypothesis.

In our experience, Duesberg and Ellison are overly simplistic in believing that abstinence from risk behaviors will avert the devastation of AIDS or that the risk-behavior theory can explain the presence of AIDS in those who have not engaged in these behaviors.

Between 1981 and 1985, our group at the Institute for Thermobaric Studies worked with over 400 individuals with AIDS, or AIDS-related complexes (ARC), or who were at risk due to contact with AIDS patients. As did Duesberg and Ellison, we saw major causes of immune suppression in the behaviors and life-styles that would make for classical (not HIV-related) acquired immune deficiency syndrome. Included in the classical causes of immune suppression were chronic inflammatory diseases (venereal diseases, hepatitis, allergies, chronic irritation, infection, or injury), chemical suppression (from any anti-inflammatory, antibiotic, or depressant drug as well as the wide range of street drugs), and malnourishment (including bulimia, anorexia, and laxative abuse).

During this time, we taught over 200 AIDS, ARC, and at-risk clients to support optimum immune competence and minimize their exposures to immune-suppressive drugs and behaviors.

As Duesberg and Ellison hypothesize, we did see a significant improvement in the general health of our clients, but not with everyone and not uniformly. Initially, we attributed this to different health status and varying degrees of dedication, discipline, or economic resources. Daily documentation was made of diet, stress, exercise, sleep, medications, and drug use. Despite the best efforts and the highest quality of care, men continued to sicken and die, although more slowly than those not engaged in our program.

Black Death

By 1984, we saw the resurgence of the opportunistic infections regardless of the quality of care and decided to take a closer look at these infections in the period prior to the antibiotic/drug era that began in 1945. We quickly found that everything we were seeing in AIDS had been seen before, most often and most profoundly in individuals who had an underlying, progressing infection of syphilis.

By 1985, the narrow focus of AIDS research and the shifting of funds out of sexually transmitted disease areas into HIV research had severely restricted any open inquiry into factors related to AIDS that did not directly promote the HIV hypothesis. We were seeing research by mandate and epidemiology by fiat. To continue our investigation, we developed BASIS, Biological Assessment of Syphilis and Immune Suppression. BASIS has been screening educated, affluent, health-conscious consumers who are not engaged in risk behaviors although they may have in the past. We continue to find a major correlation between a prior history of syphilis and the development of AIDS independent of the sensationalized behaviors or blood transfusions, and independent of whether they tested HIV-positive.

Like Duesberg and Ellison, we believe that the fundamental science to prove the HIV hypothesis has not been done. We do not, however, assert that there is no correlation between HIV and the disease syndrome we are seeing in AIDS. Long-term infections of syphilis, while causing immune suppression, also foster overgrowth of viruses, odd forms of virus, as well as other opportunistic infections. HIV may actually be a marker for an otherwise undetected, altered form of syphilis. We suspect it may be the "black syphilis" of Asian origin.

Quick and Painless

Like Duesberg and Ellison, we see the use of AZT as a political and economic solution without real medical benefit to the patients. AZT is a known immune suppressant that essentially shuts down the immune system. By administering AZT to AIDS and ARC patients, few symptoms emerge that require medical care or hospitalization until the final stage of massive system failure from multiple infections. With AZT, the insurance companies avoid the $150,000-$250,000 expenses of earlier AIDS cases where 9 to 18 months of hospital and medical care were threatening to bankrupt the companies. Hospital and health care administrators, including Medicaid officials, who saw their ruin looming as Medicaid AIDS patients filled their wards, were relieved that their financial exposure could be limited to a few weeks or months by AZT administration to patients. Politicians who were reluctant to expend more money and public resources for the care of economically and politically disenfranchised minorities could assuage concerned families and friends and the media that everything was being done that could be done medically with AZT. They promised to make AZT easier to obtain and require that all physicians seeing AIDS patients urge them to go on the drug. AZT does not stop the progression of the disease. It does not stop patients from dying. But the dying is quiet, convenient, and cheap at $5,000 to $15,000 per patient.

We believe that AIDS is the tip of an iceberg of immune-suppressive disorders in our country, which if combined with syphilis could lead to a major human die-off by the end of the century. Duesberg and Ellison do us all a disservice by continuing to promote the idea.
that normal people, with normal sexual patterns, who do not abuse drugs, are not at risk.

Joan J. McKenna
Director of Research
TBM Associates
Berkeley, CA

Break Up the HIV Monopoly

Dear Sir:

We agree with Duesberg and Ellison that the foundation of our national AIDS policy is crumbling due to its own errors and incompleteness, and feel that the whole thrust of HIV testing and research must be reconsidered.

Our experience in working with people with AIDS and those at risk clearly supports a multifactorial theory.

One urgent consideration is that tens of thousands of HIV-positive people are walking around with the fearful misunderstanding that it’s only a matter of time before they necessarily become ill and die. Many of these people feel sick solely because of this belief. Thus, the possibility that HIV is not the cause of AIDS brings up issues of psychological murder, as well as scientific error.

It is not in our best interest to allow the HIV/AIDS establishment to maintain their monopoly on the prevention and treatment of AIDS and it is long past time for us to insist on open-minded, first-rate science, rather than simply accepting the unproven assumption that HIV causes AIDS, let alone that AZT extends life.

We hope that medical doctors and the Food and Drug Administration will be held accountable for the distribution of AZT based on the poor quality of research provided in the studies, and the well-known dangers of psychological murder, as well as scientific error.

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We hope that medical doctors and the Food and Drug Administration will be held accountable for the distribution of AZT based on the poor quality of research provided in the studies, and the well-known dangers of psychological murder, as well as scientific error.

AIDS in Africa

Dear Sir:

The spread of AIDS in Africa is consistent with the bold thesis of Peter Duesberg and Bryan Ellison that AIDS is not primarily caused by HIV, but it contradicts the authors’ suggestion that the disease is caused simply by behavior. Their statement that “AIDS in Africa is evenly distributed between males and females” is quite wrong; for the majority of the 53 African countries there is a “female preponderance of AIDS,” as first noted by Dr. Neeguaye and colleagues from Ghana. Sex parity of AIDS incidence is true only of seven countries in East/Central Africa, and of one in West Africa where AIDS is in the propagation phase. For the remaining 39 sub-Saharan African countries in the introduction phase, AIDS was, until very recently, known as a female disease resulting from international prostitution.

That Certain “Something”

It is well documented that African men who use only village prostitutes are less likely to get AIDS than town prostitute users, who are less likely to get AIDS than city prostitute users. There must be “something” that the city international prostitute transmits to produce AIDS. In Arabic North Africa, for example, international prostitution is practiced only on pain of death and there is no AIDS problem. The Duesberg and Ellison hypothesis does not address this.

Another example of the international connection to AIDS is from my own Krobo tribe in Ghana, where promiscuous men who have not left the tribe do not get AIDS. The only Krobo men—three in all—who have AIDS have been the international prostitutes’ pimp-husbands who accompanied their repatriated wives home from the Ivory Coast. “Something” must have been transmitted from the prostitute wives to their non-promiscuous husbands that was not transmitted by the non-prostitute wives to their Krobo husbands who stayed at home. How then can AIDS be said to be non-infectious? And why do children born to Krobo families at home, both polygamous and non-polygamous, escape AIDS while those of families involved in the sex trade do not?

Risk Behavior Not Sufficient

Duesberg and Ellison are correct in saying that immunosuppressive behavior is a factor in AIDS, but alone it is not enough to produce AIDS. For example, the international prostitutes who repatriated from the Ivory Coast to die form the bulk of Ghana’s AIDS patients. Many of these repatriated prostitutes are in the third generation of their profession. Duesberg and Ellison’s hypothesis cannot explain why these prostitutes’ second- and first-generation relatives never got AIDS.

Another example from Africa refutes Duesberg and Ellison’s risk hypothesis. I discovered two pockets of traditional male-male sex practitioners—one among the Swahili Arabs and the other among some West Africans influenced by an immigrant culture. While there is no AIDS among these rural folk, AIDS has developed among a third group of “homosexuals”—young men who roam international hotels in Africa’s largest cities practicing the same “immunosuppressive behavior” for foreign exchange. “Something” must have been transmitted to this last group who were anything but malnourished.

Along with the international link and that certain “something” in spreading AIDS, another possible factor is that a traumatic experience may hasten the onset of AIDS. Professor Quartey and I have established that virgins in Africa develop AIDS within 10 to 12 weeks after exposure. It seems that perineal trauma allows “something” to be transmitted, as happened with the 12-year-old girl who developed AIDS quickly after being raped by Ugandan rebel troops. Similarly, HIV-positive Ugandan patients with no symptoms whatever were suddenly tipped into AIDS by surgery, pregnancy, or even gynecological investigations, proving that HIV alone was not enough to produce AIDS.

Duesberg and Ellison’s case against HIV is bolstered by the many cases that defy the HIV-only theory. In Rwanda, for example, I learned of the case of an international prostitute who gave birth to twins. The seronegative baby died from AIDS, while the seropositive one lived. Similarly, a seropositive Ghanian child is still alive and well without symptoms four years after her mother died of AIDS. Duesberg and Ellison are also
correct in pointing out that malnutrition is not only immunosuppressive, but can also be present with features exactly like AIDS.

Yet despite such findings, much evidence contradicts Duesberg and Ellison’s hypothesis. Studies show that in the early propagation-phase of AIDS in East and Central African countries, the failure to screen blood for transfusions has led to AIDS. Similarly, in the West African countries of Nigeria and Ghana, where the HIV antibody rate in blood donors is very low, AIDS infection through blood transfusion is rare. The Duesberg and Ellison hypothesis fails to explain these discrepancies. Clearly, the debate over AIDS is not over.

Tribal Therapy for AIDS

The best support for Duesberg and Ellison’s hypothesis comes from the success of both tribal and non-tribal therapeutics in Africa. Traditional healers in Africa have been tackling AIDS with varying degrees of success. Two food items that were also found anecdotally to be therapeutically interested me most. Pawpaw seeds (Carica papaya), traditionally used in the Gold Coast (Ghana) for intestinal parasites, abdominal pain, and diarrhea but with no known anti-retroviral action, have been tried in Ghana in AIDS diarrhea with some encouraging results; and the winged bean (Psophocarpus tetragonolobus), also with no known anti-retroviral action, has helped Ashanti women with AIDS.

One non-tribal treatment for AIDS is the special formulation of human alpha interferon (KEMRON) that was produced through the international cooperation of experts from Kenya, the U.S., and Japan. It holds the best promise for AIDS treatment and owes its efficacy less to an anti-retroviral (HIV) effect than to an immune-enhancing capability. Indeed, drugs with specific anti-retroviral properties are less effective in treating AIDS.

In summary, there are “pluses” and “minuses” in the Duesberg and Ellison hypothesis. There is still a lot of rethinking to be done regarding HIV and its relationship with AIDS, and Professor Duesberg has been right to insist over the past four years that the debate should not be closed.

Felix Konote-Ahuulu, M.D.
Cromwell Hospital
London, England

Clinical Evidence for HIV

Dear Sir:

There are many perplexing questions regarding HIV and AIDS. Clinical observations may help understand the process. If many of the points Duesberg and Ellison make are evaluated in the context of the reality of patient care, many of the questions they pose can be answered.

HIV Destroys Immune System

The average patient, for example, has HIV disease for 10 to 15 years. At each cross-section of time, as Duesberg and Ellison highlight, only a small percentage of T-cells are found to be infected. But, these few cells appear to die earlier than expected, so over time, gradually fewer total cells are left. However, a few of those have received as their legacy HIV infection to slowly continue the T-cell-depleting effects of the disease. The fact that only a few T-cells are affected at any one time does not change the fact that ultimately, left untreated, HIV destroys the immune system.

On that natural disease course, one should superimpose an individual’s characteristics. For example, it is true that one’s immune system can be markedly depressed by use of “recreational” drugs. Substantial percentages of the earliest cases of AIDS were in drug users, as Terry Krieger and I pointed out in a Wall Street Journal article as early as 1985. Drug users appear to have been the earliest patients because they may have had a shorter HIV disease course than the average HIV patient due to drug-induced immune system dysfunction.

It is evident to clinicians that stopping substance abuse, during any disease, increases a patient’s survival time, but that should not be equated with a cure of the disease. Nor should immune-system dysfunction from drug usage be equated with immune-system depression from a disease.

Many different diseases can produce the same objective findings. Cases of pneumocystis and Kaposi’s sarcoma occur, for example, in those who are immune-suppressed from other factors than HIV, for example, from the effects of organ transplants. But the clinical history is so different that it makes good medical sense to distinguish such patients separately from those with HIV-antibody positivity and T-cell disease due to HIV.

AZT Dangerous But Effective

AZT, today’s primary HIV medicine, unfortunately is not the ideal. It does not always control HIV as measured by the P-24 HIV antigen test and does not always cause T-cells to increase as much as desired. But it does achieve these things in statistically significant numbers, and without AZT many people would have died much earlier. Any clinician involved with AIDS treatment has a “control group” of patients that for one or another reason have refused AZT. In one of our groups we reviewed 102 HIV P-24 antigen-positive patients, 77 of whom received AZT. There were 25 who did not receive AZT. These had a 36 percent death rate, contrasted to the 77 who received AZT, who had an 18 percent death rate.

AZT as a treatment for HIV disease is, of course, “dangerous,” like any chemotherapy or some antibiotics.
However, use by patients under appropriate physician monitoring, with continuous laboratory testing, markedly decreases the dangers. In fact, under such circumstances, there are only minimal side-effects from AZT evident to the patient. Further, anemia, the most significant hazard, quite often can be controlled and reversed by concomitant use of erythropoietin, indicating, in 1990, that AZT is not as “dangerous” as originally thought in the mid-1980s when it was introduced.

Cesar A. Caceres, M.D.
The Caceres Health Group
Washington, DC

Duesberg Injected

Dear Sir:

My initial reaction to anyone challenging the AIDS industry in any way is favorable, but in the case of Peter Duesberg and his co-author Bryan Ellison, I really must demur.

The authors state that HIV does not satisfy Koch's postulates that the germ must cause the sickness when injected into healthy hosts, because "HIV has not been shown to cause disease when injected experimentally into chimpanzees, nor when accidentally injected into human health care workers." In fact, HIV does cause disease in chimpanzees, but not AIDS. Rather, it causes a swelling of the lymph nodes, which is one of the earliest symptoms of human HIV infection. It is startling that the authors do not point out that pathogen-caused disease is to a great extent animal specific. The vaccinia virus, for example, causes the disease cowpox in cattle but does not cause the disease in humans. Indeed, in humans it is used as a vaccination to prevent smallpox. Further, there have been cases of health care workers becoming infected with HIV and then developing AIDS, including the case of a New York physician that recently made national news after she sued the hospital. Another doctor with whom I appeared on TV had been infected on the job and was suffering from the early stages of the disease. Duesberg and Ellison state that HIV does not satisfy another of Koch's postulates that the germ must be found in the affected tissues in all cases of the disease. By this standard few if any viruses could be considered the cause of any disease since viruses are notoriously difficult to isolate directly. (Koch, incidentally, developed his postulates before the discovery of viruses.) This is why the use of antibodies as viral markers was an accepted practice long before AIDS came along. The authors shoot themselves in the foot when they point out that "no HIV at all can be isolated from at least 10 to 20 percent of AIDS patients; until the recent advent of highly sensitive methods, no direct trace of HIV could be found in the majority of cases." Exactly. It wasn’t that the virus wasn’t there; it’s that current technology could not detect it then, and improved technology cannot always detect it now.

African Epidemic Exaggerated

The authors are correct that the African epidemic has been exaggerated, as indeed has the epidemic everywhere. But they incorrectly accept on its face the estimate of Ugandan HIV-positives (800,000) and the estimate of AIDS cases there (41,000) to assert: How could so much HIV cause so little AIDS? The number of estimated infections is so high because the political body that came up with it grossly exaggerated it, but it’s much more difficult to exaggerate case numbers. Why do the authors go to Africa where the numbers are so foggy when they can look at exact studies right at home? Two different studies of homosexual males in San Francisco have found that after an average of 10 years of infection with HIV, about 80 percent of the subjects have developed full-blown AIDS or symptoms demonstrated to be precursors to developing AIDS. A British study presented at the International AIDS Conference in San Francisco found 23 percent of hemophiliacs had HIV and 69 percent of those were symptomatic after only an average of five-and-a-half-years' infection. One study of 172 children, which appeared in the December 28, 1989, New England Journal of Medicine found that 12 percent had full-blown AIDS but all had symptoms associated with HIV infection, indicating they were on their way to AIDS. This is an extremely high correlation between infection and disease, far higher than the correlation between disease and cytomegalovirus (CMV), or poliovirus, or the bacteria that cause tuberculosis, Mycobacterium tuberculosis. But nobody argues that since CMV nor the tuberculosis bacterium always cause disease they are not responsible for those diseases occurring.

Role of Co-Factors

The authors make some nice points, but they are points that don't prove their case. Yes, co-factors do appear to play a role in developing AIDS, especially in developing Kaposi's sarcoma. But co-factors also play a role in determining whether cytomegalovirus or tuberculosis bacterium lead to disease.

Yes, the value of AZT is unknown because standard testing protocol was not followed. Indeed, the worth of AZT has been grossly overstated by an AIDS industry desperate to show results from the massive infusion of money it has received—and taken from other disease research areas. But again, this does nothing to disprove the HIV hypothesis.

The bottom line is that there is as much or more
Evidence for HIV causing AIDS as there is for numerous other pathogens to cause the diseases we've associated with them. Epidemiology reveals that those infected with HIV are getting it from the blood and semen of other HIV-infected persons. Those infected with HIV can look to cohorts of homosexuals, hemophiliacs, and transfusion recipients and assume that chances are they will get sick and die from opportunistic infections. Further—and this is what is most troublesome about the authors' hypothesis—they must assume themselves capable of infecting and ultimately killing others. If the authors convince them otherwise, they are doing a great disservice. What I would suggest, in perfect seriousness, is that before the authors write another article suggesting that it is perfectly okay for HIV-infected persons to have unprotected sex with uninfected persons or vice-versa, that they, in a public forum, inject themselves with HIV. Apparently Duesberg has hinted he may do it; I think he should go beyond that. Readers have a right to know just how much faith the authors have in their own theory.

Michael Fumento
author, The Myth of Heterosexual AIDS
Lakewood, CO

Peter H. Duesberg and Bryan J. Ellison respond:

We are gratified to find our challenge against the virus-AIDS hypothesis finally generating the debate that should have occurred within science, and among the tax-paying public, years ago. While our view, particularly the risk-AIDS hypothesis, is currently a minority view among scientists, co-factors in AIDS are being viewed as important by an increasing number of researchers; even Luc Montagnier, the discoverer of HIV, has officially joined this modified view of AIDS. And since billions of tax dollars have failed to save even a single life from AIDS, the HIV hypothesis deserves review. Simply put, there is no proven precedent for the following claims regarding HIV:

1) No virus has ever been scientifically shown to cause its disease only after being neutralized by antibodies.
2) No virus has ever been proven to cause disease typically after long latent periods, rather than when it first infects a new host.
3) No virus has ever been shown to kill thousands of times more cells than it infects.
4) No retrovirus, including HIV, has ever been demonstrated to kill systematically any of the cells it infects.
5) No virus has ever been found that causes radically different diseases in different hosts (AIDS includes diseases not caused by immune suppression).
6) No sexually transmitted disease has ever remained so rigidly confined to specific risk groups, and to males, for 10 full years.

In short, if the HIV-AIDS hypothesis were true, it would be a truly revolutionary break with all previous scientific experience.

Yet several categories of arguments are frequently marshalled in defense of the virus-AIDS hypothesis:

Cohort Studies

From the field of epidemiology, “prospective” or “longitudinal” cohort studies are often cited, in which HIV-positive patients are followed as they progress to AIDS, while the HIV-negative control group generally does not develop fatal disease. These studies can be confusing to those unaware that their purpose is not to determine whether HIV causes AIDS, but rather to find clinical symptoms that can define various stages of sickness. The HIV-negative group is usually selected from people in good health, who are then compared with the HIV-positive patients with the health risks that we believe cause AIDS. One could just as easily conduct a cohort study, comparing HIV-negatives with health risks to HIV-positives without them, and get precisely opposite results!

Controlled studies, on the other hand, would compare two groups of people, one with HIV and the other without. The sizes of both groups should be large, at least in the hundreds, so that the occasional unexplainable anecdotal cases of AIDS diseases would be averaged out between the groups. The two groups would be matched for every health risk that might possibly be involved in the various AIDS diseases. Both groups would have the same number of hemophiliacs, of the same ages, with the same degrees of hemophilia; the same number of drug addicts would be in both, and the groups would be matched for types, amounts, and history of drug use; both groups would have the same number of transfusion recipients, with the same conditions requiring transfusions; and so on. If the HIV-positive group in such a study had significantly more diseases than the negative group, HIV would be shown to play a role in causing AIDS. The type of cohort studies cited by Whelan, Winkelstein, Temin, and Fumento is in no way this sort of controlled study. Further, we wonder how Winkelstein could construct a study able to determine that “Acquisition of [HIV] infection was not related to drug use, per se, but was highly correlated with needle sharing during drug use.”

Old Diseases

The uncontrolled cohort study is also used to argue that the majority of HIV-positives eventually develop AIDS. One of the most frequently cited is the San Francisco hepatitis-B cohort, originally selected because they were already sick with hepatitis. The disease and death rates of these extremely unhealthy people were projected onto the rest of the HIV-infected population, leading to the grossly high estimate of future AIDS rates. But since only 13 percent of the estimated one million American HIV-positives have actually developed anything called AIDS over the last decade, there is no reason to believe that such cohorts in any way reflect the condition of most HIV-positives. One should expect death rates to be relatively high in health risk groups like this. Again, a controlled study is needed, not careless extrapolation.

Similar studies with biased selection methods have created the unjustified impression that AZT has extended lifespans, but no properly conducted studies have yet been published showing longer life resulting from this invariably toxic drug. Indeed, recent data shows that long-time AZT users have an incredible 50 percent rate of lymphoma, a cancer of the white blood cells. This AZT
effect is officially being blamed on HIV, since several lymphomas are listed as AIDS diseases, although the percentage of AIDS victims overall who develop lymphomas is much lower than 50 percent.

Further, those diseases that do occur in the HIV-negative group of such studies are not diagnosed as AIDS, since this syndrome is defined by the presence of indicator diseases if the patient has antibodies against HIV; tuberculosis found in the HIV-negative group is simply called tuberculosis, not AIDS. This presumptuous and misleading definition of AIDS continually generates confusion among those who do not realize that AIDS is merely a new name for old diseases.

Individual Examples

Anecdotal cases of AIDS patients seem to be most powerful in convincing people that HIV is a dangerous virus, despite the scientific worthlessness of such individual situations. For such conditions as immune suppression, individual cases can always be found in which no underlying cause is obvious to the examining physician. In past decades, such diseases as Pneumocystis carinii pneumonia and Kaposi's sarcoma have been diagnosed in individuals without visible health risks. Diseases without obvious underlying causes are typically referred to as "primary" causes of the condition, and do not in any way indicate that something profoundly new causes it. About 3 percent of AIDS cases cannot be connected to confirmed health risks, which is not surprising; to determine scientifically whether HIV causes AIDS, a controlled study using large sample sizes is required, specifically designed to average out such anecdotal cases (as described above).

When citing occasional AIDS cases among babies, heterosexuals not using IV drugs, or health care workers with HIV, defenders of the HIV hypothesis are generally assuming, rather than absolutely confirming, the absence of other health risk factors in each of these uncommon cases. This is particularly true of drug abuse, which can be quite difficult to verify.

Media sensationalism has also convinced people that wives of hemophiliacs or transfusion recipients, presumably, having no more health risks than the general population, often contract AIDS from their spouses. But among the thousands of wives of HIV-positive hemophiliacs in the U.S., a fair number are likely to contract the virus eventually. Since AIDS is merely, by definition, a list of old diseases that are renamed when they appear in the general population, often contract AIDS from their spouses. But among the thousands of wives of HIV-positive hemophiliacs in the U.S., a fair number are likely to contract the virus eventually. Since AIDS is merely, by definition, a list of old diseases that are renamed when the doctor develops AIDS "complications." Whether the doctor is also using the toxic drug AZT is not stated. We therefore continue our relentless search for confirmed cases of AIDS resulting from needlestick injuries.

Nothing shows the bankruptcy of the virus-AIDS hypothesis better than the claims of Temin, Winkelstein, Whelan, Orient, Smith, and Fumento that one, or possibly two, health care workers may have contracted AIDS from hypodermic needles contaminated with materials from AIDS patients. The U.S. employs some five million health care workers, treating a cumulative total of over 100,000 AIDS patients for almost 10 years; thousands of American scientists also work on HIV. None of these are vaccinated against HIV. Compare the one or two debatable needlestick cases with the consequences if the nation's health workers were instead exposed for so long to polio or hepatitis virus, also without vaccination!

A Unique Virus

One of the most mistaken impressions of HIV holds that it is in some way an unusual virus. Often statements are made about its genetic complexity and "additional genes." Retroviruses have between 5,000 and 10,000 letters, or "bases," of genetic information; HIV has nine thousand, a typically small number. And virtually any genetic sequence contains some overlapping pieces of information, the "additional genes" referred to by Temin, which can also be found in any retrovirus. In the test tube, HIV behaves no differently from other retroviruses in any observable way. In short, we would like to know where this unusual complexity of HIV is hiding.

HIV would certainly have to be an extremely unusual virus to be able to kill billions of T-cells, though little or no active virus can be found in the body (contrary to Smith's assertion, for which both sides of the virus-AIDS debate would certainly appreciate a published reference). This fatal blow to the HIV hypothesis sometimes prompts strange and creative explanations. Caceres, for example, believes that the body's T-cells could be depleted if infected cells died sooner. But the time it would take infected cells to die would not matter. In all viral diseases, the virus must reproduce faster than the host's cells if it is to overtake and deplete them. HIV never even comes close. Further, HIV does not kill infected cells: Robert Gallo has patented the HIV antibody testing procedure from virus that is produced in cell lines that grow continuously, rather than die, and Luc Montagnier has recently confirmed that HIV does not kill cells in the test tube.

When supporters of the HIV hypothesis cannot make HIV sound unusual enough, they try instead to make other viruses sound like HIV. For example, a retrovirus termed SIV is said to cause an "AIDS-like" disease in monkeys. But with no long latent period, no wide variety of diseases (no Kaposi's sarcoma or dementia), and where antibodies protect against disease, we have great difficulty calling such conditions "AIDS-like."

Changing the Rules

When all else fails, defenders of the virus-AIDS hypothesis resort to moving the goalposts; rather than bringing the hypothesis into question, paradoxes lead to painful contortions of its details. A latent period first had
to be invented, then extended to its present, and still growing, total of 10 to 11 years. Antibodies had to be used, not merely to indicate that the host carried the virus, but actually to predict future disease. When Kaposi's sarcoma no longer even remotely correlated with HIV, the Centers for Disease Control had to consider dropping it from the AIDS list, rather than questioning whether AIDS was even a single condition at all. In the past, virus-AIDS defenders continually cited Africa as proof of the hypothesis; when we ourselves began citing the actual data on Africa, opponents such as Fumento turned around and adopted our previous position, that Africa proves nothing.

And when HIV cannot meet Koch's postulates with AIDS, Whelan, Winkelstein, Temin, and other virus-AIDS supporters casually try to abandon those time-tested, commonsensical postulates. Contrary to oft-stated claims, the polio virus and the tuberculosis bacterium have indeed satisfied Koch's criteria for their respective diseases. In polio, for example, the virus can be isolated from the affected tissue in every case of polio (postulate #1); the virus has been cultured (#2); the virus causes disease in animals (apparently Winkelstein is unaware that such experimenters as Albert Sabin have caused poliomyelitis in monkeys with injected polio virus); vaccines block the virus from causing disease in humans (#3); and the virus can always be reisolated (#4). Until they are able to propose a clearly superior set of standards for determining whether a virus causes a disease, HIV-AIDS proponents are arbitrarily throwing out proven standards to accommodate HIV in AIDS.

**Alternative Explanations**

Questions are raised by Orient about whether health-risk factors brought on AIDS-type diseases in past decades. Searchers of the medical literature, such as Robert Root-Bernstein and ourselves, do reveal the association of these conditions with such risks as drug use, surgical operations, chemotherapy, and so forth. *Pneumocystis carinii* pneumonia, for example, has generally been found in precisely such risk groups; former California state legislator Paul Gann would never have been considered unusual for developing such a condition after a traumatic operation at an advanced age, but this was renamed AIDS because he was also infected with HIV. And drug use has exploded in both numbers and amount of use, so that only within the last 10 or 15 years have noticeable numbers of diseased addicts begun showing up in “inner-city hospitals.”

Many who are willing to question the HIV hypothesis are still not willing to abandon the idea that AIDS is at least an infectious disease. McKenna, Lo, Wear, and Konotey-Ahulu suggest that other infectious agents may serve as co-factors, or even as primary agents of AIDS. However, AIDS simply does not behave as any known infectious condition; it is rigidly segregated in certain very specific risk groups, which for the most part are themselves associated with drug use, and after almost a decade over 90 percent of AIDS cases in the United States continue to be found in males (over 80 percent of heavy drug users are male). No precedent exists among infectious diseases for this strange distribution. All known venereal diseases spread widely through the population, including syphilis, herpes, gonorrhea, chlamydia, etc. Mycoplasmas are also quite universal, and certainly do not confine themselves so carefully to males or special risk groups, so consistently, for a decade or more. Konotey-Ahulu's evidence for AIDS as an infectious condition, mostly regarding urban prostitutes in Africa, seems to us more probably related to drug use and other western types of risk factors that have recently increased in availability in those cities.

**How to Resolve the Debate**

A relatively simple set of tests would quickly determine, once and for all, whether HIV (or any virus) causes AIDS:

1) The virus should be chemically active in more cells than the host can generate.
2) The symptoms of the disease should occur within weeks or months after infection.
3) The disease should spread relatively randomly among its potential hosts, rather than being confined to highly specific groups.
4) Antibodies produced by the immune system should be able to fight or completely neutralize the disease.
5) A controlled study, in which a group of people with the virus should be compared to a group without, to see whether those with the virus develop the sickness. The groups should be matched for all possible health risk factors: equivalent types and amounts of drug use, use of antibiotics, use of AZT, exposure to previous diseases, hemophilia, etc.

HIV, of course, already fails points (1) through (4), and we have little trouble anticipating the result of a controlled study.

But both of us would be quite willing to carry out the Fumento test: if he will arrange for sufficient national publicity, if he would be convinced by our action, and if he will thereafter help us bring exposure to our viewpoint, we will indeed be quite happy to have ourselves publicly injected with HIV. Perhaps Fumento will also be willing to check on our health status in the year 2000, or after whatever additional time is eventually added to the virus' latent period.

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—Peter H. Duesberg and Bryan J. Ellison